Comments by Michael Honeycutt, Ph.D., with the Texas Commission on Environmental Quality Regarding the Primary National Ambient Air Quality Standards for Ozone and PM, and the Utility MACT

Main Conclusions

On behalf of the Texas Commission on Environmental Quality (TCEQ), I disagree with the United States Environmental Protection Agency's (EPA) proposed range of values for the eight-hour ozone and PM standards because of uncertainties relating ambient concentrations to personal exposures and limitations of the epidemiological and clinical studies used as the basis of the revisions. The TCEQ strongly recommends that EPA use robust scientific data as the basis for the ozone and PM standards and Utility MACT, and more meaningful consideration of risk management issues in its final policy decisions.

The roles of uncertainty and bias in EPA's assessments have been severely downplayed and should be reexamined. This is particularly true in EPA's analysis of personal exposure. For ozone, EPA relies on studies that estimate personal exposure (the amount of ozone a person actually breathes) by using ambient monitoring data, which oversimplifies personal exposure by assuming that ambient monitoring data accurately reflects personal exposure. Further, EPA doesn't acknowledge or account for this potential overestimate in their standard calculations. Also, it is essential that EPA clearly discuss the uncertainties associated with adverse health effects reported in both ecological epidemiology and clinical studies. These uncertainties should also be clearly communicated in publicly accessible documents in consideration of new standards.

EPA should be more critical and conscientious in its selection of studies they use to calculate proposed numerical standards. Specifically, EPA should consider ecological epidemiology studies in a more broad, supportive context, rather than as the primary basis for calculating air quality standards. Ecological epidemiology studies are not scientifically rigorous enough to draw conclusions about the cause of health effects identified in the studies for ozone or any other pollutant and are not suitable for policy decisions. As with all observational studies, the results may provide valid areas for further inquiry and be informative, but should not be considered conclusory. EPA's criteria for the selection of key studies should emphasize not only statistical significance but also biological significance of the observed adverse health effects Furthermore, EPA should focus on the entire weight of evidence of more robust epidemiology and toxicology studies for the basis of its policy decisions.

Finally, EPA should avoid unnecessary regulation that will not improve human health. EPA's own analysis demonstrates that the Utility MACT will not have an effect on mercury levels in fish in US watersheds. EPA's claims of mercury causing lower IQ and heart disease scares the public into avoiding seafood. EPA ignores the fact that Japanese eat 10 times more fish than Americans do and have higher levels of mercury in their blood, but have lower rates of coronary heart disease and high scores on their IQ tests. To claim that a policy decision is "based on the science" without putting those decisions in appropriate context with real world implications is not just a misuse of science but causes harm to the public. It is a disservice to her citizens when government exaggerates, misstates or misleads the public about the "real" risk of environmental effects.

Ozone NAAQS

Ecological Epidemiology Studies

EPA used ecological epidemiology studies, also known as time-series analyses, as the primary basis of the most recent proposed ozone standard. Ecological epidemiology studies are observational studies designed to look for correlations. To accomplish this they examine the relationships between exposure and disease at a population-level rather than on an individual-level. These types of studies are intended to be followed up by more rigorous epidemiology studies to determine if the correlations are real. While ecological epidemiology studies are useful in evaluating potential associations between health effects and ambient exposures to environmental pollutants, they are severely limited due to their study design. Policy conclusions should not be based on ecological epidemiology studies for the following reasons:

1. *Ecological epidemiology studies are not designed to determine if ozone caused the health effects evaluated.*

The assumption that ozone caused all evaluated health effects, including aggravation of asthma and premature mortality, in ecological epidemiology studies is not well-grounded in science. Ecological epidemiology studies do not collect data on when, how long, and how much exposure occurred; if exposure occurred before the health effects; or if it makes biological sense that the chemical could cause the effect. In other words, the study designs are incomplete. Scientists agree that the incomplete study design does not provide enough information to determine the actual cause of studied effects. Ecological epidemiology studies are not supposed to be used quantitatively and they certainly are not rigorous enough to set environmental policy.

2. *Lack of personal exposure data severely limits the utility of ecological epidemiology studies.*

The issue of limited or entire absence of personal exposure data is significant. Personal exposure is a measurement of the amount of an air pollutant that a person actually breathes. In the case of air pollutants like ozone, ecological epidemiology studies rely on ambient monitoring data as a surrogate for personal exposure for percentages of people with a health issue in an area (i.e., census tract, county, or state). However, it is very unlikely that people would ever be exposed to those pollutants at concentrations measured at outdoor monitors for very long. This is partly because the average American spends 90% of his/her time indoors, especially during the heat of the summer when ozone concentrations tend to be at their highest. Ozone concentrations in most buildings are characteristically low, due to the reactive nature of ozone, the tendency of ozone to deposit on surfaces, and the ventilation systems inside buildings (McClellan et al. 2009). Other additional factors such as time spent outdoors, outdoor activity level, and weather (especially temperature and relative humidity) can dramatically change the potential for ozone exposure and the resultant estimate of risk. Therefore, ambient ozone concentrations alone do not adequately characterize, and easily overestimate, personal exposures (Sarnat et al. 2006). This position is shared by the National Academies of Science (NAS 2008) and the Clean Air Science Advisory Committee (CASAC 2006). That ecological epidemiology studies continue to derive inconsistent and vastly differing conclusions about the adverse effects of ozone is perhaps evidence of this fact.

3. *Ecological epidemiology studies frequently do not take into account the heterogeneity of regional air pollution and oversimplify their exposure analysis by relating health effects to only ozone.*

In most ecological epidemiology studies[1], exposure is estimated to be either some statistical representation (e.g., average or weighted average) of several air monitors or concentrations at the monitor with the highest readings. This assumption oversimplifies outdoor exposure because concentrations vary across a given area[2]. Moreover, few studies fully account for simultaneous exposure to multiple other pollutants, such as particulate matter, nitrogen dioxide, and sulfur dioxide. The ratios of these pollutants can vary tremendously from region to region, making it difficult to determine which effects are related to which pollutants. This complication blurs the association between health effects and ozone exposure, as documented in recent studies. Furthermore, it has been documented in studies that the association between ozone and health effects is confounded by temperature and relative humidity (which alone can cause physical stress), and population characteristics, such as age, health status, socioeconomic status, and exercise.

4. *Ecological epidemiology studies have considerable uncertainty in their identification of health effects.*

To determine prevalence of a health issue, epidemiologists frequently use readily-available information, including hospital admissions records and death certificates, or participant surveys. In some of the ecological epidemiology studies EPA used for the proposed ozone standard, death certificates for thousands of people who died at a hospital from any non-accidental cause were compared to outdoor ozone levels from up to three days before the person died. Because of the broad selection criteria, it is highly likely that many of these people died due to non-respiratory health issues unrelated to ozone exposure. This problem is compounded when paired with the lack of personal exposure data, making it impossible to know if decedents were actually well enough to be outdoors in the days preceding their deaths. In this case, patient history records from physicians would be more reliable than hospital admission records or death certificates for determining the presence and severity of any health effects potentially caused or aggravated by ozone exposure. EPA could better serve the public trust to recognize the limitations on the information and data used and to fully consider these limitations when making policy decisions.

5. *Additional statistical analysis (time-series and multi-city time-series studies) further complicates the interpretation of ecological epidemiology studies.*

The shortcomings of ecological epidemiology studies are compounded when researchers perform time-series studies, which try to correlate health effects collected from epidemiology studies and ambient ozone concentrations measured during the hours and days leading up to their hospital visit or death. Some studies compare even broader sets of data from multiple cities averaged over multiple years. In addition to the issues regarding uncertainty in the original ecological epidemiology study discussed previously, this additional analysis fails to take into account:

[1] Many ecological epidemiology studies do not look at the area immediately around a monitor but rather at a conglomeration of several cities. For example, Bell and Dominici (2008) looked at communities, which they defined as a county or contiguous counties.

[2] EPA acknowledges the variance in ozone concentrations across a region within its state implementation planning (SIP) process by its requirements to have multiple monitors within a populated region and its requirement to further analyze unmonitored areas during the planning process.

- The high degree of variability between cities, seasons, and years
- The effect of other pollutants that contribute or cause the same effects
- The inconsistent ambient air sample collection period between cities
- Socioeconomic factors such as age, access to healthcare, etc., and
- Mortality differences among cities.

In addition, further analysis of time-series data indicates the studies are highly influenced by the type of statistical model used (often, the model showing the most health effects) and publication bias (studies showing effects are more likely to get published than those showing no effects). Due to the substantial uncertainty in these studies, policy decisions should not be based on these studies and EPA should revise its study selection criteria to use studies of higher scientific quality.

6. *Results of ecological epidemiology studies are inconsistent and it remains unclear if ozone is truly related to increased health effects.*

Ecological epidemiology studies have provided vastly different conclusions regarding the effects of ozone on the population, with studies showing significant adverse effects, no effects, or even protective effects of ozone. In particular, one reanalysis of an ecological epidemiology study frequently cited by EPA identified only six out of 95 cities evaluated with a significant correlation of mortality and ozone (Smith et al. 2009). Furthermore, although it has been repeatedly hypothesized that ozone is a potent inducer of asthma attacks, Texas Inpatient Hospital Discharge data on the numbers of hospital visits for asthma between 1999 and 2001 actually showed that people were more likely to visit the hospital for asthma during winter when ozone is at its lowest than they were in the summer when ozone concentrations are high. In a relative risk sense, cold weather and pet dander are more potent inducers of asthma hospital visits than ozone. Furthermore, results from a four-year (2000-2003) air quality study conducted by Texas A&M University and Driscoll Children's Hospital indicate hospital admissions to be weakly correlated with ambient daily maximum ozone levels.

Clinical Studies

Clinical studies expose humans to a known concentration of ozone for a known period of time and monitor their health. Although these studies do not have the significant limitations of ecological epidemiology studies, there is confusion among scientists and regulators about whether subtle clinical changes documented in the studies represent adverse effects. In its ozone reassessment, EPA failed to consider key recommendations regarding this issue and conducted a reanalysis of clinical data that was not scientifically appropriate.

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

Ambiguity exists in defining what constitutes an adverse effect on exposure to air pollution. Clinical studies evaluating health effects due to ozone exposure have mainly focused on decreases in lung function as measured by forced expiratory volume in one second (FEV_1)[3] and other similar measures. Daily normal activities, exercise, and diurnal variations can themselves cause changes in the FEV_1. Within a

[3] FEV_1 is a measure of the forced expiratory volume during the first second of an active exhalation. This measurement is used to assess lung function and is often used in epidemiological or controlled clinical studies. The measurement is accomplished by having a subject inhale deeply and then exhale quickly. A significant reduction in FEV_1 may be indicative of impaired ventilation.

single day, FEV_1 in normal subjects can vary by over 5% (Pellegrino et al. 2005) and be as high as 17.6% (Medarov et al. 2008). Therefore, controlled exposure studies must properly account for normal changes by including filtered air (FA) exposures and a range of concentrations and exposure durations. The American Thoracic Society (ATS) recommends a comprehensive description of "adverse" effects by combining the loss of lung function in conjunction with respiratory symptoms, such as cough and discomfort while breathing (ATS 2000). Further, OEHHA, the TCEQ, and jointly the ATS and the European Respiratory Society (ERS) consider decrements in FEV_1 of $\leq$ 20% as "mild," not "adverse." However, in its reevaluation of the Adams (2006) study, EPA identified FEV_1 decrements of only 2.8% to be adverse effects. According to the sources listed previously and Adams himself, the decrements in the Adams (2006) study at 0.06 ppm are not of biological significance, even though they may be of statistical significance. Therefore, it is also prudent that the EPA justify the importance of key study results to indicate not just statistical significance, but also biological significance before labeling the result as an adverse effect.

2. *EPA's reanalysis of Adams (2006) data is not scientifically appropriate and should not be included as part of the final ozone policy decision.*

In addition to the issue of whether or not the decrease in FEV_1 was adverse, the EPA also conducted a highly contentious statistical reanalysis of the Adams (2006) data to show statistical significance in the absence of the effect (Brown 2007, Brown 2008). Dr. Adams himself disagreed with the EPA's reanalysis and statistical reinterpretation of his study during a teleconference on March 5, 2007, and in written comments to the EPA during the 2007 comment period. EPA's reanalysis was also criticized by other statisticians and scientists, as stated in comments submitted to EPA by Drs. RL Smith and JE Goodman. The TCEQ concurs with Dr. Adams' peer-reviewed results.

3. *EPA should consider more recent studies as part of the ozone weight of evidence.*

Recent clinical studies (Kim et al. 2011, Schelegle et al. 2009) of ozone exposure at concentrations lower than 0.08 ppm have further confirmed the Adams (2006) results, showing no adverse effects at 0.06 ppm. When compared to filtered air, Schelegle et al. (2009) reported statistically significant mean percent change in FEV_1 at 0.07 ppm (5.34%) and Kim et al (2001) reported statistically significant mean percent change in FEV_1 at 0.06 ppm (1.71%), these are not only within in the range of intra-individual variability but are also substantially less than the 20% decrease identified as adverse.

4. *EPA needs to emphasize the importance of having realistic controls for clinical studies.*

Many of the clinical studies use filtered air (no ozone) for the control groups (Schelegle et al. 2009, Kim et al. 2011), which creates an unrealistic scenario as the natural background ozone concentration in the atmosphere is around 0.04 ppm (Last et al. 2010). In its analysis of the clinical studies, EPA has not adjusted for this background factor and has not provided any justification for not doing so. Not adjusting for background can result in overestimating the severity of the observed effects as "adverse effects," when in fact the effects were "not adverse." Based on the clinical studies, it can be inferred that the weight of evidence at the lower range of exposure levels (i.e, 0.06 – 0.07 ppm) is weak and inconclusive. Thus, I can conclude that the clinical studies used to justify the lower end of the proposed range do not support lowering the ozone standard below the present NAAQS of 0.075 ppm (Adams 2002 and 2006, Schelegle et al. 2009, Kim et al. 2011). Further, these studies are conservative since they do not consider personal exposure.

Differing Roles of Policy and Science

EPA's recent attempts at using science to justify policy decisions are particularly troubling. In its reconsideration of the ozone standard, EPA attempts to establish a health basis as the need for a new, reduced standard. However, the assumption that the reduced standard would prevent up to 12,000 deaths is based on dubious studies and the use of such an analysis signals an unfortunate shift in the roles of scientists and risk managers.

1. *The basis of the theoretical number of lives saved is meaningless and unrealistic.*

EPA relied on studies that took mortality data from ecological epidemiology studies to calculate the number of theoretical deaths that would be avoided with a lower standard. Not only do these studies suffer from the severe limitations described above, but theoretical lives saved estimates are also meaningless from a scientific and practical standpoint. It is not possible to verify either the current number of deaths due to ozone exposure or the future change in deaths if the standard is lowered because there is still no conclusive evidence that ozone causes mortality at ambient concentrations.[4] There is no guarantee of increased life expectancy or degree of confidence in this estimation, since some degree of risk is present in all aspects of daily life.

2. *EPA misuses scientific studies to justify policy decisions. Scientific studies should be just one aspect of responsible policymaking.*

Rigorous scientific studies focus on expanding the knowledge of how a chemical interacts with the body at different tested doses. However, even the most extensive studies are not able to define an acceptably safe level of a chemical. In the specific case of ozone, scientific studies have still been unable to clearly identify human risk at current ambient levels and have certainly not shown if 0.065 ppm ozone is substantially more protective than 0.08 ppm. Determining what level of risk is acceptable is and should remain a decision for risk managers, not scientists.

Responsible risk managers and policymakers consider science as one of many aspects to be considered in setting policy. Science cannot determine practical issues, such as the feasibility of implementation and to what extent society would accept the trade-offs associated with the standard. For example, an overly restrictive health-based standard might be more detrimental to public health if it forces an industry out of business due to the cost of compliance and its employees are unable to find work to support their families. Studies have consistently indicated that poverty is a much better predictor for premature mortality than exposure to environmental pollutants. Public officials with a broader perspective of potential policy implications are better equipped to evaluate these important aspects.

PM NAAQS

The Proposed PM standard

EPA has proposed a new particulate matter (PM) standard that is twice as stringent as the current standard. Attainability of the proposed standard, especially in rural and agricultural areas, is impractical and even EPA staff acknowledges that the available scientific evidence supports the effectiveness of the current standard in protecting public health. There is no scientific basis supporting a reduction in the current standard, let alone a two-fold reduction.

[4] In fact, EPA has provided no data to illustrate lives saved under previous standards. All estimates of lives saved are projections, not factual.

1. *EPA based the proposed PM standard on an ecological epidemiology study.*

EPA used a study by Zanobetti and Schwartz (2009), which is an ecological epidemiology study, as a basis for the proposed PM standard. This ecological epidemiology study concludes that exposure to coarse PM is "suggestive" of a causal relationship with adverse effects. As stated above, ecological epidemiology studies are incomplete studies plagued with limitations and should not be used as the basis for policy conclusions.

2. *EPA assumes all PM composition is equal.*

Not all PM is created equally; however, EPA makes the assumption that it is. Coarse PM is produced by surface abrasion or suspension of biological material and fragments of living things. Because of this, PM in urban and industrial areas is likely to be vastly different from PM in rural and agricultural areas. Urban and industrial PM is expected to be enriched with pollutants; pollutants that are inherently more toxic than the dust predominantly found in agricultural operations and arid rural areas. EPA didn't take this scientific fact into consideration when they developed their proposed PM standard. When they assume all PM composition is the same they ignore the fact that agricultural and rural areas will likely exceed the standard due to natural occurrences rather than man-made sources.

3. *PM composition varies greatly by geographic regions.*

The PM data EPA used in their assessment for the proposed PM standard were not uniformly distributed across the United States or even within counties. Therefore, potential differences in PM composition may be reflected in the EPA estimates. Geographic variability is also strongly influenced by region-specific sources, meteorology (e.g., wind speed and direction), and topographical conditions (e.g., trees, mountains). When PM composition differs geographically, the conclusions drawn may not apply equally to all parts of a geographic region.

Utility MACT

Mercury and the Utility MACT

EPA has proposed a National Emission Standards for Hazardous Air Pollutants (NESHAP) rule for coal- and oil-fired electric utility steam generating units (EGU). This proposed NESHAP rule (the Utility MACT) would establish maximum achievable control technology (MACT) emission limits for certain hazardous air pollutants (HAP), including mercury. In EPA's analysis for mercury, they state "if U.S. EGU impacts to watersheds included in the risk assessment were zeroed-out, for a significant majority of those watersheds, total exposure would still exceed (and in most cases, significantly exceed) the RfD [Reference Dose]." In EPA's own words they are admitting control of US EGU mercury emissions will not have an effect on mercury levels in fish in US watersheds; however, they still insist on the necessity to require these controls. Concurrent with the Utility MACT, the EPA's National-Scale Mercury Risk Assessment Supporting the Appropriate and Necessary Finding for EGUs (mercury risk assessment) was released for public comment and review by EPA's Science Advisory Board Mercury Review Panel. Currently, the Mercury Review Panel's support for the mercury risk assessment is contingent upon development of a revised document that addresses numerous issues. The Panel's comments to EPA on the mercury risk assessment were finalized in September 2011, illustrating the limited time allowed for review and revisions of such an important document whose purpose was to determine whether a public health hazard is associated with US EGU emissions. One could easily conclude that the Panel's input was

merely a formality and was not intended to be seriously considered, much like EPA treats input from the States.

1. *The EPA 2000 appropriate and necessary finding[5] estimates were inaccurate.*

The risk analysis estimates of hazard quotients due to US EGU-attributable emissions of mercury have already decreased significantly between the 2005 and 2016 scenarios; mainly due to PM controls. In fact, 2010 levels of mercury emissions are already at levels predicted for 2016. In addition, the 2000 appropriate and necessary finding was based on estimates that US utility mercury emissions would increase from 46 tons in 1990 to approximately 60 tons in 2010. In reality, emissions were reduced to 29 tons in 2010.

2. *US EGU mercury emissions are insignificant compared to other sources.*

The Utility MACT preamble states that on average, US EGUs are estimated to contribute only 2% to total mercury deposition in the US. Therefore, any health benefits related to mercury reductions would pose an insignificant change in the overall risk from mercury from all sources. Only in combinations of the worst-case watersheds with fish consumption rates (e.g., 95th and 99th percentile fish consumption rates paired with the 95th and 99th percentile watersheds) did estimates of US EGU-attributable hazard quotients (HQs) exceed 1.5 (EPA considered an HQ > 1.5 to represent a potential public health hazard). US EGUs contributed insignificantly to the total risks posed by other sources of mercury; thus, regardless of this regulation, risk from mercury deposition will remain from sources other than US EGUs.

Mercury is a global pollutant. It travels beyond boundaries of states and continents. EPA modeling estimates that, on average, 83% of the mercury deposited in the US originates from international sources, excluding Canada; the remaining 17% comes from US and Canadian sources. As such, control strategies related to EGUs may not affect change in fish tissue concentrations of mercury. According to EPA (2007), "The mix of long-distance and local sources makes it difficult in some water bodies to achieve water quality standards for mercury."

3. *EPA uses a worst-case scenario for risk and does not characterize risk for realistic US populations.*

EPA should have characterized risk for the more realistic general recreational angler population to provide perspective and information to that population. Instead, the EPA's mercury assessment is essentially a worst-case scenario that focuses on subsistence fishing populations and may overestimate risk for the majority of the US population. EPA's own Science Advisory Board Mercury Review Panel states "There is scant evidence documenting the prevalence or extent of subsistence fishing in the United States."

[5] In December 2000, EPA issued a "regulatory determination" under the Clean Air Act (CAA) that it is "appropriate and necessary" to regulate mercury emissions from coal-based power plants and nickel emissions from oil-based power plants. This regulatory determination listed coal- and oil-based EGUs as a source category under section 112(c) of the CAA, the first step to setting MACT standards. On January 30, 2004, EPA proposed to remove EGUs from the 112 list based on a finding that it was neither appropriate nor necessary to regulate EGUs under this section of the CAA. On March 29, 2005, EPA issued a final revision of the appropriate and necessary finding for coal- and oil-fired EGUs and removed such units from the 112 list. The removal of EGUs from the 112 list was challenged in court. On February 8, 2008, the court determined that EPA violated the CAA by removing EGUs from the 112 list. As a result, EGUs remain a CAA section 112(c) listed source category according to EPA. The basis of the court ruling was that EPA did not follow the requirements of 112(c)(9) in removing EGUs from the 112 list. As such, the court did not reach a determination on the merits of the case.

4. *EPA states that about 7 percent of women of child-bearing age are exposed to mercury at a level capable of causing adverse effects in the developing fetus.*

Several well-conducted studies examining effects of mercury on children have been conducted, including the Seychelles Child Development Study (Seychelles) and the Faroe Island Study (Faroe). A blood mercury No Effect Level (NEL) of 85 parts per billion (ppb) was observed in the Seychelles study. Interestingly, this study also observed positive improvements on IQ as mercury levels increased; a phenomenon likely due to nutrients such as omega-3 fatty acids and selenium from high fish consumption. A blood mercury NEL of 58 ppb was observed in the Faroe study; however, these residents also consumed large quantities of whale meat and blubber that contained unsafe (according to EPA) levels of polychlorinated biphenyls (PCBs). Since neither study found effects below 58 ppb blood mercury levels, one would only expect to find health effects in children whose mothers had mercury levels *higher* than 58 ppb in their blood. EPA's safe level (the RfD) is set to prevent blood mercury levels exceeding 5.8 ppb, ten times lower than the NEL of 58 ppb from the Faroe study.

Data from the Centers for Disease Control's (CDC's) National Health and Nutrition Examination Survey (NHANES), 2003–2008, show the mean blood mercury level for pregnant women is 0.69 ppb (well below EPA's safe blood mercury level) (Jones et al. 2010). Although some individuals have blood mercury levels greater than EPA's safe blood mercury level, none have blood mercury levels above the Faroe study NEL of 58 ppb, and therefore adverse health effects would not be expected in their children. A 2005 study conducted by Texas Department of State Health Services (DSHS 2005) determined that even when subsistence fishers are eating fish from Caddo Lake with elevated mercury, women of child-bearing years did not have blood mercury levels greater than the EPA's safe blood mercury level.

On comparing US blood mercury levels to other countries, both the United Kingdom (UK) and Japan median blood mercury levels are higher. Using EPA's RfD to describe Japan's data, 66% of Japanese women are exposed to levels above EPA's safe blood level. From this the claim could be made (falsely) that 66% of Japanese children are born at risk for adverse effects. On the contrary, the Japanese population consumes ten times more fish than the US population but only shows positive outcomes; they have lower rates of coronary heart disease and high IQ scores. EPA is causing unnecessary alarm in the public with their assertions that 7% of women of child-bearing age are exposed to mercury at a level capable of causing adverse effects in the developing fetus when the evidence clearly shows this statement to be false and misleading.

5. *EPA uses an RfD that is more conservative than most other Agencies (US and World).*

The Agency for Toxic Substances and Disease Registry (ATSDR) and the US Food and Drug Administration (FDA) both have established safe levels three-fold higher than EPA's conservative RfD. The World Health Organization (WHO) recommends a level that is two times higher than EPA's RfD; Health Canada uses a value similar to the WHO recommended value. The TCEQ agrees with ATSDR and FDA that it is more appropriate to use a study that reflects US fish consumption (e.g., saltwater fish such as tuna) rather than a study based entirely on consumption of saltwater fish and mammals (e.g., whale).

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