

TEXAS COMMISSION ON ENVIRONMENTAL QUALITY'S (TCEQ) COMMENTS TO THE
U.S. ENVIRONMENTAL PROTECTION AGENCY'S (EPA)

ORAL COMMENTS FOR CASAC LEAD REVIEW PANEL PUBLIC TELECONFERENCE REGARDING
THE SECOND EXTERNAL REVIEW DRAFT OF THE INTEGRATED SCIENCE ASSESSMENT FOR
LEAD
DOCKET ID No. EPA-HQ-ORD-2011-0051

Good Morning. I am Dr. Stephanie Shirley, Toxicologist at the Toxicology Division of the Texas Commission on Environmental Quality and I will be making the following oral comments on behalf of the TCEQ. I would like to thank the CASAC for this opportunity. Written comments have already been submitted to both the EPA and the CASAC.

1. The time allowed for comment on the Second ISA is unreasonably short.

The assessment of the health hazards associated with airborne lead (Pb) has significant regulatory implications. The 60-day comment period does not allow regulatory agencies and stakeholders to provide the most thorough and meaningful comments possible based on an in-depth review and analysis of the 1,467-page second draft ISA and plethora of associated citations.

2. The draft ISA frequently lacks transparency and would benefit from a more specific and structured approach.

There is one over-riding issue that is especially problematic: associations are frequently confused for causations. Simply enumerating the ecological epidemiology studies that report associations between Pb and effects of interest does not demonstrate causality. There is little to no discussion of study quality or limitations, and data is often presented as if there is no ongoing debate within the field of study. This is especially apparent in the discussion of Pb exposure and intelligence quotient (IQ), where there continues to be uncertainties related to the frequency, timing, duration, and level of Pb exposure that may contribute to health effects. It is clear that IQ is a malleable measure of global neurological function and is determined by a complex milieu of factors including genetics, maternal IQ, environment, education, and enrichment.

3. Inhalation of Pb in ambient air is a minor source of Pb exposure compared to exposure by other routes.

Air is a very minor pathway for childhood Pb exposure. Therefore, more strictly regulating Pb in air accomplishes little in terms of real risk reduction.

Given current childhood Pb exposure through air is approximately 200 times less than normal intake from other sources (e.g., food, water, soil/dust) it is highly unlikely that significant risk reduction would result from more restrictive air regulations. Moreover, EPA's own Integrated Exposure Uptake Biokinetic (IEUBK) model for Pb in children supports this conclusion.

Summary

Current typical airborne Pb exposure for children (the sensitive population) is insignificant compared to normal exposure by other routes, and promulgation of increasingly stringent NAAQS for Pb will not significantly improve protection of public health from Pb toxicity. This is supported by results from EPA's IEUBK model for Pb in children, which show that predicted mean blood Pb levels of children are not expected to exceed the blood Pb 10 µg/dL level of concern (or even the newly recommended 5 µg/dL, which is based on the 97.5th percentile of NHANESIII dataset¹) using typical background soil/dust Pb concentrations and either a Pb NAAQS of 1.5 µg/m³ or 0.15 µg/m³.

Neurological effects such as IQ loss or poor academic performance depend on a multitude of factors. In such circumstances, epidemiology studies are limited in their ability to accurately identify and quantify adverse effects and have not adequately controlled for potential confounding by variables such as parental IQ, socioeconomic status, parent education, developmental delays, alcohol/drug use, and the home environment. Consequently, when the health outcomes of concern have complex etiologies and confounders have not been adequately adjusted for, a scientifically defensible and accurate dose-response assessment is unlikely. This is certainly the case for Pb-exposure and potential neurological effects.

¹ CDC Advisory Committee for Childhood Lead Poisoning Prevention January 2012 (<http://www.cdc.gov/nceh/lead/ACCLPP/activities.htm>)

Finally, the EPA should acknowledge that the dramatic decreases in ambient air Pb and children's blood Pb levels are inconsistent with their suggestion that Pb is a causal factor in the increased frequency of ADHD. The significant decreases in child Pb exposure are inconsistent with concurrent increases in the prevalence of ADHD. Moreover, the assertion that population-wide decline in IQ will occur if Pb levels in air are not further decreased is not supported by science, as there is a general consensus that the opposite trend is occurring (i.e., the Flynn Effect).