

Comments regarding

U. S. EPA First External Review Draft Integrated Science Assessment for Lead

[FRL-9428-5; Docket ID No. EPA-HQ-ORD-2011-0051]

Prepared by: Craig J. Boreiko, Ph.D

July 19, 2011

International Lead Zinc Research Organization

1822 East NC Highway 54, Suite 100

Durham, NC 27713, U.S.A.

Tel: 919-361-4647

Fax: 919-361-1957

cboreiko@ilzro.org

The International Lead Zinc Research Organization is pleased to offer comments regarding the May, 2011 draft of EPA's Integrated Science Assessment for Lead. ILZRO notes that substantive critical comments have been prepared by Gradient for submission by the Association of Battery Recyclers (ABR) and concurs with the concerns expressed. ILZRO's comments will attempt to avoid duplicating those submitted on behalf of ABR but will instead focus upon general aspects of the ISA which appear to be problematic and highlight specific examples of shortcomings evident in the scientific analysis contained within the Draft ISA.

EPA Framework for Causal Determination

The ISA prefaces its analysis by discussing the framework by which causality can be inferred from human observational epidemiology and other studies. Given the recent criticisms made of EPA Risk Assessment Procedures, the inclusion of such a preface in the current document is an interesting decision. The Agency appears to be proposing the application and codification of causality criteria that are less rigorous than those applied in the past. The development and application of such criteria merits significant independent scientific review and discussion. Embedding new causality criteria within the framework of the Lead ISA is problematic in that objective evaluation is difficult to conduct when presented within the context of a toxic substance which, in and of itself, has been the subject of acrimonious and contentious scientific debate. Matters are further complicated by the subsequent failure of the ISA to adhere to "weight of evidence" evaluations put forward as metrics of causality. One is thus faced with the dilemma of both weighing the merits of the causality criteria proposed and the failure of the ISA to then adhere to the weight of evidence evaluation standards being promulgated.

The rather simplistic discussion of confounding and confounder correction exemplifies the ISA's short-comings. Potential effects of confounders are noted and then indications made in Chapter 1 that they can be addressed if enough studies have been conducted under sufficiently disparate conditions to permit dissociation of confounder effects from the effects of interest etc. Subsequent discussions of confounding are included in the evaluation of studies evaluating relationships between lead exposure and health endpoints such as neurocognitive development in children, but ultimately the discussions provided are superficial and, with few exceptions, do not attempt to quantify the potential impacts of confounding or attempt to define divergent exposure scenarios that would provide opportunities to dissect the impacts of lead from that of confounders. Lead exposure remains one of the most pervasive markers of social disadvantage known and this fact continues to impede efforts to accurately define the effects of low level lead exposure. The ISA further gives scant attention to studies that have reported ubiquitous confounders (Surkan et al., 2008) that should be controlled in studies of lead effects on child development. In essence the ISA defines an (albeit simplistic) means of addressing confounding and then fails to apply its own guidelines in the most critical health endpoint evaluated – the effect of lead upon child development.

Similar short-comings are evident in the consideration of modifying factors and co-exposures. While co-exposures are noted as potentially important in Chapter 1 of the ISA, the ISA ignores the recent suggestion by Gulson (2008) that co-exposures to other environmental neurotoxicants may have influenced the outcome of some lead studies. Lead studies have seldom measured or corrected for such co-exposures, but studies of other environmental toxicants have now begun to correct for lead exposure as a confounder (Stewart, 2008; Perera et al., 2006, 2008).

Comparative Evaluation of Study Results

The ISA is said to have as its goal the undertaking of a careful evaluation of the recent scientific literature and a weight of evidence evaluation that yields scientifically robust conclusions regarding the effects of low-level lead exposure. The ISA fails in this goal because it misstates the conclusions of the studies reviewed to incorrectly claim that health effects are consistently observed at low level lead doses.

A rather blatant example of this approach is evident in studies of academic achievement and lead exposure. Table 2-2 cites the study of Miranda et al (2009) for this impact with accompanying verbiage that suggests that “since no evident threshold has yet been clearly identified for most effects, the effects of such effects at still lower blood lead levels cannot be ruled out based on available information.”

The narrative description of Miranda et al (2009) on pages 5-59 to 5-60 is consistent with this conclusion and brief note is taken on 5-61 of a study by Chandramouli et al (2009) who are said to have made similar observations of a relationship between early blood lead and later academic performance. Those who read the study of Chandramouli et al will take note that lead appears to

have an impact upon academic performance at blood lead levels less than 10 µg/dL but would likely be surprised to read the authors conclusion that the effects of lead appear to exhibit a threshold with little or no impact below a blood lead level of 5 µg/dL. The ISA further continues to characterize all low level health effects of lead upon child development as exhibiting a supralinear dose response – a conclusion that is facilitated by a failure to cite studies (e.g. Roy and Bellinger, 2009 that observe strictly linear dose response functions for lead impacts). A number of instances of selective and misleading interpretations of the published literature are present in the comments prepared by Gradient and hardly inspire confidence in the objectivity of the analysis that has been presented.

Linkages between air lead and health and ecological impacts

Although the ISA provides estimates of the dose response for different lead health effects, and also provides estimates of annual anthropogenic lead emissions, a central issue which remains unresolved is the existing contribution of air lead to contemporary U.S. blood lead levels. Given the paucity of air lead monitoring data available for most counties in the country, and the vintage nature of much of the existing air lead monitoring network, there would appear to be inherent imprecision in any estimate of the current contribution of air lead to human blood lead levels. Moreover, given that implementation of the recently adopted NAAQS for lead has only just begun, the generally limited contribution of airborne lead to human exposures would be expected to decline. In particular, the 2006 NAAQS documentation made a number of “worst case assumptions” regarding the relationship between air lead and human blood lead in estimating the impact of different air quality standards upon blood lead. In the absence of information defining the nature and extent of any exposure reductions realized as a result of the recent dramatic downward revision of the NAAQS, there is little factual information on which to make a determination of a need for further reductions.

The current ISA further estimates that anthropogenic sources contribute about 1200 tonnes of atmospheric lead per annum, but little sense is provided of the contribution of this atmospheric input to human or environmental exposures. From this perspective, it is informative to compare anthropogenic inputs to estimates of natural input of lead into the atmosphere. For example, the current estimate of anthropogenic release in the United States is about 10% of the natural global atmospheric release estimated by Nriagu (1989) and is an even smaller fraction of more recent estimates of natural North American atmospheric releases (34,000 tonnes average) made by Richardson et al. (2001). If atmospheric lead is no longer a significant contributor to human exposures in the United States - and/or natural sources of atmospheric lead are now quantitatively equal to, or greater than, anthropogenic sources – further efforts to reduce anthropogenic emissions would seem to yield limited public health or environmental benefit.

Literature cited in comments but not discussed within the ISA

Gulson, B. L. (2008). "Can some of the detrimental neurodevelopmental effects attributed to lead be due to pesticides?" The Science of the total environment **396**(2-3): 193-195.

Nriagu, J.O. (1989). "A global assessment of natural sources of atmospheric trace minerals." Nature **338**:47 – 49.

Perera, F., T. Y. Li, et al. (2008). "Benefits of Reducing Prenatal Exposure to Coal-Burning Pollutants to Children's Neurodevelopment in China." Environmental Health Perspectives **116**(10): 1396-1400.

Perera, F. P., V. Rauh, et al. (2006). "Effect of Prenatal Exposure to Airborne Polycyclic Aromatic Hydrocarbons on Neurodevelopment in the First 3 Years of Life among Inner-City Children." Environmental Health Perspectives **114**(8): 1287-1292.

Richardson, M.G., Garrett, R., Mitchell, I., Mah-Paulson, M., and Hackbarth, T. (2001). "Critical Review on Natural Global and Regional Emissions of Six Trace Metals to the Atmosphere." At:
http://echa.europa.eu/doc/trd_substances/VRAR/Copper/vrar_env/vrar_appendix_p3.pdf

Roy, A., D. Bellinger, et al. (2009). "Lead exposure and behavior among young children in Chennai, India." Environmental Health Perspectives **117**(10): 1607-1611.

Stewart, P. W., E. Lonky, et al. (2008). "The Relationship between Prenatal PCB Exposure and Intelligence (IQ) in 9-Year-Old Children." Environmental health perspectives **116**(10): 1416-1422.