Review of the Human Health Risk Assessment for the Kiln-Related Emissions at the Holcim Trident Cement Plant at Trident, Montana

Wendy Heiger-Bernays, PhD
Associate Professor of Environmental Health
Boston University School of Public Health

September 28, 2006

I have been asked by my colleague, Dr. T. Webster, to review and comment on the Draft EIS for the Holcim (US) Inc., Tire Burning Proposal, dated July 2006. Based on an initial cursory review and reading of previous documents, my comments are presented below.

As an environmental toxicologist, my practical experience is in the area of multi-pathway human health risk assessment and regulatory toxicology. Several of the risk assessments on which I have worked include heavy metals and semi-volatile compounds, such as PCBs and dioxins. I teach graduate level courses in environmental health, toxicology, and risk assessment. I have taught classes on conducting risk assessments for hazardous waste incinerators, have written guidance documents on conducting risk assessments on dioxin-contaminated sediments and am an active member of the Risk Assessment Advisory Committee for the Massachusetts DEP.

General Comments

My comments are by definition general in nature due to the lack of transparency of this assessment and lack of inclusion of formulae, justification for use of emissions assumptions and estimated emissions, toxic equivalency factors (and their application), exposure assumptions, averaging times and risk calculations.

1. Due to the absence of calculations (both baseline and cumulative) for emissions values, assumptions and variables used in the risk calculations, use of toxic equivalency factors, and documentation of IEUBK assumptions and estimates of risk for the lead model, I am unable to verify any of the conclusions drawn. These are public documents that should be transparent in process and in presentation.

2. The conclusion that the cumulative risks are nearly identical or lower than the baseline risks is inconsistent with the data showing increased emissions of dioxins/furans, PCBs, arsenic and chromium in the test burns from the comparison facilities and with the increased amounts of several heavy metals in the lead smelter slag.

3. The risks due to exposure to dioxins/furans are underestimated due to the use of the PC-MACT limit as the "default" for the TCDD equivalents, rather than the actual values calculated from the emissions inventory. By using the federal dioxin limit in both the baseline and cumulative cases, risks due to facility-related activities are essentially excluded when assessing the incremental increase in risk due to dioxin exposures. This approach is not defensible and is inconsistent with the use of test burn emissions values used for assessing non-dioxin/furan risks. It is particularly egregious since dioxins/furans are demonstrated to be the risk drivers. Since the PC-MACT limit is used as the default for the TCDD equivalents, this implies that the PCBs were not included in the analysis, again, underestimating cancer risks. Non-cancer risks are also underestimated if the PCBs are not included in the TEQ estimate.

4. Inhalation of particulate matter is dismissed from the analysis because of the presence of other sources of particles. This permit requires demonstration that the cumulative impacts from the burning of tires result in risks below Montana's negligible risk standard. Due to the hazards associated with inhalation of fine particulate matter (EPA, 2006) contribution of

particulates from this facility (baseline, incremental & cumulative) should be included in this assessment.

5. It is not clear why risks to an on-site worker are not evaluated.

6. The uncertainty analysis is insufficiently documented.

Specific Comments

1. Estimating Emissions from Kiln Upsets. There is no justification for the assigned relative percentages of particulate and gaseous partition of several HAPs. The assigned upset multiplier will most likely underestimate risks.

2. The lack of a table that presents the emissions concentrations used in the risk assessment makes it impossible to assess the accuracy of the risk assessment calculations. I am unable to re-create concentrations from the tabular HAP Emissions Study (2006), adjustments in emissions inventory (p. 18 in Appendix B) and estimated kiln emissions (Table 2 in Appendix B). Units are inconsistent within tables and between tables (lb/hr, g/sec, t/yr, lb/yr). Likewise, a list of COPCs and their concentrations in each medium should be available.

3. It is unclear whether the modeled soil concentrations used as exposure point concentrations represent the mean value for the deposition over 70 years or whether the value represents the cumulative deposition. Since many of the COPCs have lengthy half-lives, the concentrations are over-estimated initially and then underestimated.

3. Existing and potential receptors include workers, residents and recreational site users. Although consumption of fish and venison are considered for the recreational user, these pathways have been considered in isolation and have not been integrated into the total risk calculation. It is likely that residents hunt and fish in the impacted area and these exposure pathways should be included in the residential total site risk estimates. In addition, consumption of milk products and meat from a large goat dairy farm within the depositional area will contribute to the dioxin/furan risks. The risks due to

4. The description of the use of the IEUBK model states that site-specific values for food and air are used, but it is unclear whether default or site-specific values are used for soil and water. The text does not state that uptake or bioavailability default parameters were used.

5. Table 2-1. Acute Hazard Assessment. For non-worker hazard assessment, the preferred acute criteria that should be used are the ATSDR MRLs and the CalEPA RELs. For example, the peak benzene concentrations should be compared with the REL and not with the DOE TEEL.

6. Table 3-1. In selection of the COPCs, EPA Region IX PRGs do not address food pathways. The appropriate screening values are the EPA Region III PRGs that are adjusted such that a Hazard Index of 0.1 and Cancer Risk of 1×10^{-7} is used (EPA, Region III). For example, arsenic, chromium, cadmium, dioxins and PCBs should be retained, rather than screened out with these criteria.

7. The assessment evaluates “chronic” exposure to non-carcinogenic COPCs based on a 1-year annual average worst-case ground-level air concentrations, although justification for selection

of exposure durations and averaging times is not presented. The child's risk should be presented separately from the adult resident's risk. This will provide a more accurate daily dose based on the child's body weight. It is not clear whether a one-year average was also used to assess carcinogenic risks. If this correct, then cancer risks would be underestimated by at least a factor of 30.

8. The non-cancer hazards are insufficiently addressed. Mercury and PCBs, manganese and lead have all been identified as neurotoxicants, and several of these compounds are now undergoing additional toxicity testing and re-evaluation by EPA. While toxicity factors may not be readily available for many of these, the risks associated with exposure to these COPCs should be discussed qualitatively.

References

EPA, 2006. PM NAAQS Final Federal Register Notice. 40 CFR Part 50. 09-21-06.

EPA Region III, 2006. Risk-Based Concentration Table.
http://www.epa.gov/reg3hwmd/risk/human/info/cover.pdf