

Review of the Health Risk Assessment for the Holcim, Inc.
Cement Plant at Trident, Montana

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I. Introduction

I teach environmental health at Boston University School of Public Health. Among my interests are the fate, transport, and risks of dioxins. I have written several papers concerning modeling of dioxins in the food chain (e.g., Connett and Webster 1987; Webster and Connett 1990). I was an invited participant in the USEPA Science Advisory Board review of the agency's methodology for assessing health risks associated with indirect exposure to combustor emissions. I was also a member of the USEPA's Dioxin Peer Review/Risk Characterization Committee.

I have been asked by Montanans Against Toxic Burning to review the health risk assessment for the Holcim cement kiln's proposal to burn tires, presented in the *Application for Alteration to Air Quality Permit* of 10/3/01. This review has been somewhat difficult because of the changing nature of the application and its various appendices, often with insufficient documentation. Nevertheless, I have a number of comments.

As the risks due to dioxins dominate the results of the risk assessment, I have focused most of my attention there. By "dioxins" I will here mean the polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs), sometimes called dioxins and furans. Ideally, one would include other dioxin-like compounds, including a number of the PCBs (discussed more below).

II. Specific Comments

1. Scope

The very limited scope of the risk assessment is an overarching problem:

- (a) Rather than just analyzing the health risks of one option—as was done for Holcim—risk assessments should be conducted within a context of a comparison of alternatives.
- (b) Analyzing one facility at a time is inappropriate when pollutants such as dioxin are transported regionally (or globally) so that the cumulative impact of many sources becomes important.
- (c) Neither kiln residues nor risks to workers are addressed.
- (d) Population risks due to contamination of food consumed by many people, including areas to which food is exported, are ignored.
- (e) The Holcim assessment only analyzes risk from the estimated increase in dioxins due to tire combustion. Was a previous risk assessment performed for dioxins when the kiln was not burning tires? Cement kilns can be a substantial source of dioxin; it seems odd to examine only the increment. If a risk assessment is only being done now because of a change in regulatory status—i.e., to a waste burner—should not total dioxin emissions be examined?

2. Risk due to dioxins in beef and milk

Appendix J of the Holcim risk assessment shows the estimated lifetime cancer risks for various scenarios due to exposure to dioxins via a number of different routes. Inhalation produces the dominant risk; in particular, it is larger than risk due to beef and dairy ingestion. This is an unusual result for scenarios that evaluate exposure to dioxins via agriculture. Many groups find quite the reverse. For example, the risk assessment methodology discussed by the USEPA in the draft dioxin reassessment indicates that exposure via beef and milk can each greatly exceed inhalation exposure (USEPA 2000a).

As the detailed analysis in the attached appendix shows, the Holcim risk

assessment greatly underestimated total exposure to dioxins. Cancer risks due to dioxins in beef and milk can, depending on the scenario, exceed 10^{-6} .

3. Non-cancer risks of dioxins

When comparing exposure with a reference dose, one is supposed to use the *total* exposure: “background” plus increment. Otherwise, the comparison with a reference dose makes little sense. (A reference dose is a dose below which adverse effects are deemed unlikely.) The Holcim risk assessment compared the estimated *incremental* exposure of dioxin to a reference dose.¹ This is not a trivial issue. The USEPA dioxin reassessment stated that any reference dose is likely to be much *less* than the so-called “background” exposure (USEPA 2000b).² Similar issues have arisen at the World Health Organization (WHO 1998). WHO set a “tolerable” daily intake that was approximately equal to the background dose, acknowledging that subtle effects may be occurring at current levels.³ Thus, it is likely that the total dose—background plus incremental—would exceed a reasonable reference dose.

4. Upsets

The analysis of upsets is quite weak for dioxin. It only examines upsets of the electrostatic precipitator (ESP) and does not take into account upsets in combustion, temperature, and other factors that may also increase dioxin emissions (as discussed with incinerators⁴). The risk assessment does not examine the possible effect of upsets on chronic exposure and risk. I am not convinced by the upset estimates for the ESP, as it relies on one old paper for the particulate-vapor split.

5. Uncertainty

The uncertainty analysis is superficial and an understatement. McKone and Hammond (2000) list several major uncertainties associated with examining health risks of waste incineration (also discussed by a National Research Council panel): (i) the very limited data on emissions under upset and off-normal conditions; (ii) the focus on risks to local populations to the exclusion of workers and longer-range impacts (and cumulative effects); (iii) the uncertainty in food chain and other cross-media models. All of these uncertainties apply to the Holcim risk assessment.

At least two other sources of uncertainty deserve attention. (1) Substantial uncertainty is introduced by potential interactions of chemicals, a subject only superficially discussed in an appendix of the Holcim risk assessment. As an example of

¹ This reference dose is old (CAPCOA 1993), not taking into account either USEPA (2000b) or WHO (1998). See Appendix O of the Holcim risk assessment.

² The “background dose” is the exposure, primarily through food, thought to be due to cumulative impact of many sources.

³ WHO’s TDI is 1-4 pg/kg/d of TEQ (including PCBs). A TDI does not mean the same as a reference dose.

⁴ De Fré and Wevers (1998) found that standard stack gas measurement techniques—with samples collected over a 4–6 hour period—can underestimate longer-term (15-day) measurements by a factor of 30–50. See also McKone and Hammond (2000) for a discussion of upsets.

the interesting work on dioxin, Brown et al. found that a single pre-natal dose of dioxin dramatically increased the rate of mammary tumors in female rats exposed to DMBA (a carcinogen) later in life. (2) The USEPA's draft dioxin reassessment indicates that the cancer slope factor could substantially increase over the value used in the Holcim risk assessment, in part due to the incorporation of human epidemiological data (USEPA 2000b).

6. Produce

Dioxin risks due to ingestion of produce are unaccountably neglected by the Holcim risk assessment, assigned values of "Not Applicable" in Appendix J without explanation.

7. TEFs

Holcim uses toxic equivalence factors (TEFs) for PCDDs and PCDFs but does not apply established TEFs for PCBs. They employ emission estimates in TEQ from other documents without noting the TEF system of those sources or correcting for changes (TEFs have evolved over time).

8. Two items requiring clarification

Appendix V appears to analyze a residential scenario at the maximum concentration located on the Holcim boundary. Previously, a worker scenario was used. However, the risks due to dioxin in Appendix V are smaller than the worker scenario estimates in Appendix J. It is not clear why.

The October 15 submission provides a new stack concentration limit for dioxin. Given the earlier dioxin emission estimates by Holcim, a justification of an ability to achieve this value is required.

III. Conclusions

My primary conclusions regarding the Holcim health risk assessment are:

- 1) The scope of the assessment is much too narrow.
- 2) Risks from dioxin in beef and milk were substantially underestimated.
- 3) Lifetime cancer risks from dioxin exceed 10^{-6} for some scenarios.
- 4) Background exposure to dioxins is not properly taken into account in examining non-cancer health effects of dioxins.
- 5) The uncertainty of the risks is underestimated.

IV. References

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Appendix: Problems with the Holcim Risk Assessment regarding dioxin exposure via beef and milk

In the following section I discuss the Holcim health risk assessment as presented in the *Application for Alteration to Air Quality Permit* of 10/3/01 and Appendix J, submitted later. Appendix J lists risks for dioxins (and other chemicals) for adults and children at a number of different locations. To keep this review manageable, I will focus on three pathways of exposure—inhalation, ingestion of beef, ingestion of dairy products—for adults at two locations: the nearest residence and the point of maximum impact on the Holcim property boundary. These two locations were associated with χ/Q values—a normalized measure of the degree of dispersion in the atmosphere—of 0.029 and 0.661 ($\mu\text{g}/\text{m}^3$)/(g/s) (p. 54). One multiplies the χ/Q value by the emission rate to obtain the annual average air concentration. I will also use the emission rate for dioxins (measured as toxic equivalents or TEQ) assumed by the risk assessment: 2.98×10^{-8} g/s (p. 44).⁵ I do this not because I agree with the value, but because the purpose of this section is to critique the food chain model. Part of the strategy is to verify the computations made in the risk assessment or show where they are in error.

The Holcim risk assessment is unusual in that the risks for dioxins from ingestion of beef and dairy products are smaller than risks from inhalation. This is illustrated by some results from Appendix J:

Table 1. Excess lifetime cancer risk from dioxins for adults at the nearest residence

	<u>Risk</u>	<u>Ratio</u>
Inhalation	1.22×10^{-8}	1
Beef	2.77×10^{-9}	0.23
Milk	2.56×10^{-9}	0.21

For agricultural scenarios, risks (and exposure) via ingestion of beef and dairy are typically much larger than inhalation. For example, the USEPA's draft reassessment of dioxin illustrates exposure in a scenario where local agriculture is exposed to stack emissions (USEPA 2000). Exposure via beef ingestion is roughly thirty times higher than inhalation, dairy about sixteen times. Many other groups (although not all) have found similar or higher results for beef and dairy (discussed in Connett and Webster 1987, and Webster and Connett 1990). The dominance of exposure via food is not surprising given the tendency of dioxins to accumulate in food chains.

I thus suspected from the very beginning of this review that something was amiss. I cannot definitively say why the Holcim risk assessment has this problem because the document is not completely transparent: parameters and procedures are not completely spelled out and intermediate calculations (with some exceptions) are not presented. Nevertheless, I have two hypotheses: (i) there appears to be an error in unit conversion or transcription of results for pollutant concentrations in plants; (ii) the parameter describing

⁵ The value could be increased by taking upsets into account (as well as using the correct degrees of freedom for the t-statistic as discussed by other commentators).

the yield of plants (Y) is too large. Let me explain both problems and how they affect risks.

(i) Concentration of pollutants in plants: Hypothesized error in unit conversion or transcription

A clue to the first problem is given by the appendix to the Holcim health risk assessment that provides sample calculations for some pathways for *arsenic* (these are the only such sample calculations). Pages 5 and 6 of that section illustrate the estimation of exposure of cattle to arsenic via feed and pasture. These equations depend on C_f , the concentration in feed and pasture, listed in the example as:

$$C_f = 4.02 \times 10^{-7} \mu\text{g/kg} \quad (1)$$

Page 5 explains that C_f is “assumed to be equal to the average conc. in root, vine, and leaf produce, see page 8.” Page 8 provides the following computation for the concentration of arsenic in leafy crops:

$$C_f = C_{\text{depv}} * \text{BIO} + C_{\text{trans}} \quad (2)$$

$$= (7.41 \times 10^{-4} \mu\text{g/kg})(1) + (5.4 \times 10^{-5} \mu\text{g/kg}) \quad (3)$$

$$= 7.94 \times 10^{-4} \mu\text{g/kg} \quad (3)$$

$$= 7.9 \times 10^{-7} \text{mg/kg} \quad (4)$$

where C_{depv} is the concentration due to direct deposition, BIO is a bioavailability factor (equal to 1 for arsenic), and C_{trans} is the concentration due to root uptake. Note the correct unit conversion in equation (4). C_{depv} is computed on page 16:

$$C_{\text{depv}} = \text{DEP} * \text{IF} / (\text{k} * \text{Y}) * (1 - \exp[-\text{k} * \text{T}]) \quad (5)$$

$$= (4.11 \times 10^{-4} \mu\text{g/m}^2\text{-d})(0.2) / [(0.693/14 \text{ d})(2 \text{ kg/m}^2)] * [1 - \exp(-0.693/14 * 45)]$$

$$= (8.30 \times 10^{-4} \mu\text{g/kg})(.8922) = 7.41 \times 10^{-4} \mu\text{g/kg}$$

where DEP is the atmospheric deposition rate on the vegetation ($\mu\text{g/m}^2\text{-day}$), IF is the interception fraction (unitless), k is the weathering constant (days^{-1}), Y is the yield of the plant (kg/m^2), T is the growth period (days). We are not provided the sample calculations for root and vine crops, but these are easily computed. The risk assessment assumes the same parameters for all crops except the interception fraction (Table 7.5.2-8 of the risk assessment): IF = 0.2 for leafy crops, IF = 0 for root crops, IF = 0.1 for vine crops.

Using equation (5) we compute:

$$C_{\text{depv-root}} = 0 \quad \text{for root crops since IF} = 0$$

$$C_{\text{depv-vine}} = 3.70 \times 10^{-4} \mu\text{g/kg} \quad \text{for vine crops since IF} = 0.1$$

As C_{trans} was assumed equal for all three crops (pages 74–75 of the risk assessment), the total concentrations from both routes are, from equation (2):

$$C_{f-root} = 0(1) + (5.4 \times 10^{-5} \mu\text{g/kg}) = 5.4 \times 10^{-5} \mu\text{g/kg}$$

$$C_{f-vine} = (3.70 \times 10^{-4} \mu\text{g/kg})(1) + (5.4 \times 10^{-5} \mu\text{g/kg}) = 4.24 \times 10^{-4} \mu\text{g/kg}$$

Simple averaging of the concentrations in leafy, root, and vine crops yields:

$$C_{f-ave} = (7.94 \times 10^{-4} \mu\text{g/kg} + 5.4 \times 10^{-5} \mu\text{g/kg} + 4.24 \times 10^{-4} \mu\text{g/kg})/3$$

$$= 4.24 \times 10^{-4} \mu\text{g/kg} \tag{6}$$

$$= 4.24 \times 10^{-7} \text{mg/kg} \tag{7}$$

The average equals C_{f-vine} because the average interception fraction (0.1) equals the interception fraction for vine crops (all other parameters being equal). It is conceivable that some kind of weighted average of the three crops was used—the Holcim risk assessment doesn't provide any more details or provide a sample computation—but weighted averages cannot be less than the smallest value, $C_{f-root} = 5.4 \times 10^{-5} \mu\text{g/kg}$. Given the lack of discussion, it is reasonable to assume that the simple average was used.

The concentration of arsenic in feed and pasture estimated by the Holcim risk assessment should have been the value in equation (6): $4.24 \times 10^{-4} \mu\text{g/kg}$ (or at a minimum, $5.4 \times 10^{-5} \mu\text{g/kg}$). But the Holcim risk assessment actually used the number in equation (1): $4.02 \times 10^{-7} \mu\text{g/kg}$. **The Holcim value is nearly 1000 times too small.** I haven't found anything in the Holcim risk assessment to explain this huge difference. My hypothesis is that there was an error in unit conversion or transcription of results between sections so that $4.24 \times 10^{-4} \mu\text{g/kg}$ was somehow rendered as $4.02 \times 10^{-7} \mu\text{g/kg}$ (The difference between 4.24 and 4.02 is about 5% and might be due to rounding). Perhaps mg and μg were confused.

Based on the erroneous value in equation (1), Holcim's sample computations estimated lifetime cancer risk for adults at the nearest residence due to exposure to arsenic via ingestion of beef and milk: 2.31×10^{-12} and 3.6×10^{-13} , respectively (page 11). These are quite close to those provided in Appendix J: 2.32×10^{-12} and 3.66×10^{-13} . I therefore conclude that the same problem occurs in the computer program that generated Appendix J.

When I programmed the equations—including the hypothesized error of dividing C_f by 1000—and applied them to dioxins, I was able to arrive at values very similar to the numbers in Appendix J. Compare the first two columns of Table 2:

Table 2. Excess lifetime cancer risk from dioxins for adults at the nearest residence computed using various assumptions about C_f

	<u>Appendix J</u>	<u>Computed using $C_f/1000$</u>	<u>Computed using C_f</u>
Inhalation	1.22×10^{-8}	1.22×10^{-8}	1.22×10^{-8}
Beef	2.77×10^{-9}	2.78×10^{-9}	1.26×10^{-8}
Milk	2.56×10^{-9}	2.57×10^{-9}	1.17×10^{-8}

(ii) High value for the yield (Y) parameter

As Table 2 shows, the risks due to exposure to dioxins in beef and dairy are nearly the same as inhalation when I use the correct value (C_f) rather than $C_f/1000$. I argued earlier that beef and dairy risks from dioxin should be much larger than inhalation. Something else must also be going on in the Holcim risk assessment.

A second problem is caused by the type of food exposure model and the choice of parameters used by the Holcim risk assessment. At one point MDEQ asked for more justification for the selection of the CAPCOA 1993 risk model as the basis for the Holcim risk assessment. Bison Engineering responded on March 11, 2002, on behalf of Holcim. Page 5 of that document provides a table comparing some of the parameters used in the CAPCOA model with certain models by OEHHA (CalEPA) and two specific documents from USEPA. The table does not include comparisons with the model in USEPA (2000a), the dioxin reassessment. The parameters shown in the table are quite similar (interception fractions values are not however listed for the USEPA). The document says that the “applied exposure assumptions are consistent with the most conservative and health-protective recommended by OEHHA and EPA.” I disagree. The table does not discuss the produce yield parameter (Y). The value used in the Holcim risk assessment—which appears to derive from CAPCOA—is much too large for animal feed, especially pasture/grazing.⁶ The Holcim risk assessment assumes that pasture/grazing accounts for 80% of the beef and dairy cattle food (page 7.71).

The large value of Y leads to low estimated pollution concentrations in plants and animals. Equation (5), reproduced below in a slightly different form, estimates the pollution concentration in plants due to atmospheric deposition:

$$C_{\text{depv}} = \{IF/Y\} * \{DEP/k*(1-\exp[-k*T])\} \quad (8)$$

The term in the first set of brackets is plant specific; the term in the second set of brackets is not (at least here). C_{depv} is directly proportional to the *ratio* of IF to Y: the interception fraction divided by the yield. The Holcim risk assessment assumes IF = 0, 0.1, 0.2 for root, vine, and leafy crops respectively. The same value of Y of 2 kg/m² is assumed for all plants. These values lead to IF/Y ratios of 0, 0.05, and 0.1 (with units of m²/kg). The average concentration of pollutants is governed by the average value of IF/Y: 0.05. (As discussed above, we need only consider the average value of IF.)

Holcim’s IF/Y value is too small. Connett and Webster (1987) estimated IF/Y values of 2.38, 2.82, and 0.56 for hay, pasture grass, and silage in northern New York State based on Baes (1984), a commonly used reference for food chain models. The USEPA’s draft dioxin reassessment lists IF/Y values of 0.75–2.0 for hay, 1.8–2.6 for pasture grass, and 0.47–0.67 for silage.⁷ USEPA (2000a) used the following values in their example calculations: 2.3 for grass, 0.98 for hay and silage averaged. **The IF/Y**

⁶ Woodward-Clyde’s 1995 *Air Quality Health Risk Assessment Procedures/Model*, produced for MDEQ, notes on pages 4–11 that “the agricultural yield suggested in CAPCOA is 4–20 times higher than the value suggested by EPA,” referring to yet another set of EPA documents.

⁷ USEPA lists several sets of choices for Y and IF. I computed the IF/Y values. See page 4-65 of USEPA (2000a).

values employed by USEPA were 46 times larger than Holcim’s for grass and 20 times larger for hay/silage. It is unclear why CAPCOA—the apparent source of Holcim’s parameters—chose $Y = 2$ for all plants. IF is usually computed based on Y (see Baes 1984; USEPA 2000a). Y is usually the *dry* yield; since plants contain substantial amounts of water, wet weight yield can greatly exceed dry weight yield.

Table 3 provides cancer risk estimates at the nearest residence for dioxins via inhalation, beef, and dairy products computed using the same methodology as the Holcim risk assessment except for two changes: (i) correction for the hypothesized unit conversion problem discussed earlier; and (ii) use of larger IF/Y values for animal food: 2.3 and 0.98, the values used by USEPA in their sample computations for grass and hay/silage, respectively. For IF/Y = 2.3, the ratio of ingestion to inhalation risks is 37 for beef and 35 for dairy products (approximately the same value for beef as that computed by the far more sophisticated USEPA [2000] food chain model). For IF/Y = 0.98, the ratio is 16 for beef and 15 for milk, roughly half as much and therefore less credible for beef. The results in the Holcim risk assessment (Appendix J) are included for comparison. Although I only examined three pathways⁸ (and for adults only), the total risk from all pathways is likely to exceed 10^{-6} .

Table 3. Excess cancer risk from TEQ for adult receptors at the nearest residence computed with alternative values of IF/Y.

	<u>Appendix J</u>	<u>IF/Y = 2.3</u>	<u>IF/Y = 0.98</u>
Inhalation	1.22×10^{-8}	1.22×10^{-8}	1.22×10^{-8}
Beef	2.77×10^{-9}	4.56×10^{-7}	1.96×10^{-7}
Milk	2.56×10^{-9}	4.22×10^{-7}	1.81×10^{-7}

Table 4 presents similar results for the point on the Holcim boundary where the annual average air concentration was highest.⁹ The Holcim risk assessment estimated risks for this location using a worker scenario. An agricultural scenario is more appropriate: for compounds like dioxin, the location where food is produced is more important than where a person lives. (In the later Appendix V, the Holcim risk assessment agrees to use a resident scenario.) The ratios of ingestion to inhalation risks are, as expected, the same as those in Table 3. But since the estimated air concentration for Table 4 is larger, the risks for each pathway are also larger, **approximately 10^{-5} for beef ingestion alone.**

Table 4. Excess lifetime cancer risk from TEQ for adult receptors (point of highest concentration) computed with alternative values of IF/Y.

⁸ I have not discussed corrections to risks in the other animal-based foods or human milk. All of these will be increased.

⁹ $\chi/Q = 0.661 (\mu\text{g}/\text{m}^3)/(\text{g}/\text{s})$

	<u>IF/Y = 2.3</u>	<u>IF/Y = 0.98</u>
Inhalation	2.78×10^{-7}	2.78×10^{-7}
Beef	1.04×10^{-5}	4.47×10^{-6}
Milk	9.61×10^{-6}	4.13×10^{-6}

These calculations do not mean that I endorse all aspects of this particular methodology. Instead, they are a sensitivity analysis to show how correction of an apparent error and substitution of commonly used parameters for IF/Y make a very large difference in the estimated risks. Nevertheless, the risks computed using IF/Y = 2.3 produce beef to inhalation ratios roughly consistent with the modeling procedure used by USEPA (2000a). As discussed earlier, larger ratios are also reasonable.