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Competitor Assessment

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March 31, 2004

Mr. Vince Castellanos
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Dear Vince,

RE: Soil Dioxin Contribution to Daily Dioxin Intake

It was a pleasure to speak with you and to catch up on the twenty years since we met during the public meetings on the licensing of Salzburg landfill.

I finally had the opportunity to examine the issue of daily dioxin intakes by Midland Area residents, including those living near or in the floodplain. I believe that this issue was raised in a recent Community Advisory Panel meeting, but was not discussed in detail.

In the minutes of the meeting, the MDEQ indicated for areas, with TEQ levels of approximately 10 ppt, that soil-related dioxin intakes were approximately 5% of the daily intake and dietary factors accounted for the other 95%. This information is correct. The EPA provided a fair amount of information on this subject in the 2000 Draft of their Dioxin Reassessment.

The MDEQ also indicated that a Dow study stated that, with a background level of 348 ppt-TEQ, that the soil-related contribution was 35%. I'm having difficulty understanding the basis for this conclusion. Based on the EPA's modeling of soil and dietary intake, soil-related dioxin intake is directly proportional to soil dioxin levels. In order for Dow's contractor to develop the 35% contribution, significant changes to the dietary intake had to occur.

Perhaps, the table below will help to better understand the daily intake calculations.

Daily Dioxin Intake – Adults
(pg/kg/day)

Soil TEQ, ppt	<u>9.4</u>	<u>348</u>	<u>348</u>
Soil-related intake	0.031	1.148	1.148
Diet-related intake	0.559 -----	0.559 ⇐ -----	2.132 ⇐ -----
Total Daily Dioxin Intake	0.590	1.707	3.280 pg/kg/day
Soil Intake, % Total	5.3%	67%	35%

pg = picogram = 10^{-12} gm = trillionth of a gram

If soil-related dioxin intake is proportional to soil TEQ levels, the only way for Dow's contractor to estimate that a soil dioxin level of 348 ppt equates to 35% of the total daily intake is to dramatically increase the diet-related intake from 0.559 to 2.132 pg/kg/day.

If someone can provide a copy of the contractor's study, I will be glad to review it and give you my opinion as to the basis for the discrepancy.

There are a number of issues associated with dioxin exposure from contaminated soil. Let me break up the issues as follows:

1. Soil-related Dioxin Intakes
 - Children
 - Adults
2. Daily Dioxin Intake – Risk Levels
3. Dioxin Body Burden Levels – adults
4. Computer Modeling of Historic Dioxin Levels

Soil-Related Dioxin Intakes - Children

Due to their playing habits, young children ingest more soil than do adults and, therefore, would ingest higher levels of dioxins. In addition, the diet of young children is also different from that of adults and this results in a further increase of daily dioxin intakes.

Let me present some information on how daily dioxin intakes change with age. This information was included in the EPA 2000 Draft on TCDD. The intake information is based on a dioxin soil level of 9.4 ppt - TEQ.

	<u>Daily Dioxin Intake</u> (pg/kg/day)			
Age	<u>1 – 5 Yrs</u>	<u>6 – 11 Yrs</u>	<u>12 – 19 Yrs</u>	<u>Adult</u>
Soil-related Intake	0.124	0.064	0.039	0.031
Diet-related Intake	2.269	1.236	0.701	0.559
Total Daily Dioxin Intake	2.393	1.300	0.740	0.590
% Soil-related	5.2%	4.9%	5.2%	5.3%

As can be seen, daily dioxin intakes are highest for young children and then decline into adulthood.

However, the most important factor is not the percentage of soil-related dioxin, but the actual amount of dioxin ingested on a daily basis for an extended period of time.

There is only a limited amount of information as to the dioxin/furan levels in the floodplain. Even though approximately 225 locations have been sampled, based on the calculation that the floodplain has an area of approximately 727 acres, there is less than a 1% confidence level that

all locations with high concentrations of dioxins have been identified. For this reason, it is not appropriate to use "average levels" of dioxins since "average" is not yet defined.

I will based my calculations on the dioxin levels that were found at several residences along the river. This should give a reasonable estimate as to the impact of soil dioxins on daily intake for a range of dioxin levels.

Daily Dioxin Intake – Children 1 to 5 Yrs.
 (pg/kg/day)

Avg. TEQ, ppt	<u>9.4</u>	<u>203</u>	<u>413</u>	<u>940</u>	<u>2058</u>	<u>2626</u>
Soil-related Intake	0.124	2.94	5.45	12.40	27.15	34.64
Diet-related Intake	2.270	2.27	2.27	2.27	2.27	2.27
	-----	-----	-----	-----	-----	-----
Total Daily Dioxin Intake	2.393	5.21	7.72	14.67	29.42	36.91
Location	(1)	(2)	(3)	(4)(5)	(6)	(7)

- (1) US National Average, Urban soils
- (2) 2580 River Road, Samples 3-1,3 6,15
- (3) 6850 Wallace, Samples 2-1,3,6,15
- (4) 5885 Midland Road, Samples 1-1,3,6,15
- (5) 1133 St. Andrews, Samples 2-1,3
- (6) 5585 Michigan, Samples 2-1,3,6,15
- (7) 1180 River Road, Samples 3-1,3,6,15

It is recognized that young children and nursing infants are especially sensitive to dioxins. Unfortunately, these is very little scientific information that defines the exact impact of daily dioxin intakes at the levels shown above.

The Michigan Department of Community Health has stated, "Fetuses, infants, and children may be especially sensitive to dioxin because they are growing and developing rapidly. However, information on the effects of dioxin in children is limited."

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Dr. Theodore Schettler, M.D., MPH, *Human Effects of Dioxin, 2003*, made these remarks, "... in the US, a breast feeding infant consumes approximately [17 – 20 times more dioxin, TCDD equivalent basis, per day] than the average adult. Nursing infant exposures are at levels which cause abnormalities in animal studies. All studies of dioxin toxicity indicate that early development is the lifestage of greatest sensitivity to many of [dioxin's] health effects. However, since many of the adverse effects of fetal or infant dioxin exposure may be apparent only much later in life, human epidemiological studies of the results of those exposures have yet to be conducted since early exposures are impossible to estimate with accuracy."

It should be noted that Dr. Schettler's comments pertain to normal levels (~10 ppt) of dioxins in the US and not to the much higher dioxin levels being found in Midland or in the river floodplain.

Note: I have some information that does address the dioxin intake of nursing infants but I have not included it in my comments at this time. If you or any other member of the CAP would like this information, please let me know and I will provide it.

Let me make one additional comment about dioxin exposure to children. Even at the highest intake level shown above (36.91 pg/kg/day), someone may comment that this is an extremely small amount of dioxin... an amount so small that it's hard to believe that it could cause any harm.

The amount is extremely small: $36.91 \text{ pg/kg/day} = 36.91 \times 10^{-12} \text{ gm/kg/day} = 0.0000000000000813 \text{ pounds/kg/day}$.

However, consider this: if a five year old child that weighs 18 kg (~40 lbs.) is exposed to 36.91 pg/kg/day of TCDD, that child is exposed to more than one trillion molecules of TCDD each day. Actually, the number is 1,242,875,100,000 (1.242875×10^{12}) molecules. The molecular weight of TCDD is 321.96 and Avogadro's number is 6.023×10^{23} molecules per gm-mole.

Most health agencies agree that the common mode of action of TCDD and the other chlorinated dioxins and furans is to bind initially to the aryl (benzene or aromatic) hydrocarbon (Ah) receptors found in cells. The Ah receptor is an intracellular protein found in every cell of the vertebrates, including rodents and humans. Through activation of the Ah receptor, TCDD and the other dioxins induce a wide spectrum of biological responses considered to be important to the carcinogenic process. There is scientific consensus that TCDD/dioxin binding to the Ah receptor is a necessary step—but not the only step – in the inducement of cancers and other diseases in the human body.

Children have marvelously effective defense mechanisms that each day fight off a wide variety of bacteria, viruses and toxic chemicals. However, sometimes those defense mechanisms are overwhelmed and children do get sick and, sometimes, children die. Until it is absolutely confirmed that dioxin, at any level, is totally harmless, I believe that it is a very wise precaution to limit dioxin exposure to the lowest level possible.

Soil-Related Dioxin Intakes – Adults

Daily Dioxin Intakes for adults are calculated in the same manner as were the intakes of young children – soil-related intakes are proportional to soil dioxin levels with diet-related intakes added to give an estimate of daily intakes.

Let me use the same residences that I used before since there is a range of dioxin contamination levels.

Daily Dioxin Intake – Floodplain Adults
 (pg/kg/day)

Avg. TEQ, ppt	<u>9.4</u>	<u>203</u>	<u>413</u>	<u>940</u>	<u>2058</u>	<u>2626</u>
Soil-related Intake	0.031	0.735	1.363	3.10	6.79	8.66
Diet-related Intake	0.559	0.559	0.559	0.56	0.56	0.56
	-----	-----	-----	-----	-----	-----
Total Daily Dioxin Intake	0.590	1.294	1.921	3.66	7.35	9.22
Location	(1)	(2)	(3)	(4)(5)	(6)	(7)

- (2) US National Average, Urban soils
- (2) 2580 River Road, Samples 3-1,3 6,15
- (3) 6850 Wallace, Samples 2-1,3,6,15
- (8) 5885 Midland Road, Samples 1-1,3,6,15
- (9) 1133 St. Andrews, Samples 2-1,3
- (10) 5585 Michigan, Samples 2-1,3,6,15
- (11) 1180 River Road, Samples 3-1,3,6,15

I believe that you are aware that The Agency for Toxic Substances and Disease Registry (ATSDR) and the EPA have commented on “safe” levels of dioxin exposure. The ATSDR has defined a minimal risk level (MRL) for a hazardous substance (such as dioxins and furans) as the estimate of daily human exposure that is likely to be without appreciable risk of adverse noncancer health risk over a specified period of time and exposure route (oral, etc.)

I've summarized the ATSDR's minimal risk levels in the table below:

Minimal Risk Levels (MRL) - Oral Ingestion – Noncancer Health Risks
(ASTDR – 1998)

	<u>Exposure Duration</u>	<u>TCDD Exposure</u> (pg/kg/day)
Acute	< 14 days	200
Intermediate	1 – 365 days	20
Chronic	> 365 days	1

As can be seen, chronic MRL levels are easily exceeded when dioxin soil levels are greater than 200 ppt. It should also be noted that the MRL exposure estimates are based on animal exposure data and not human exposure data. The Acute MRL is based on immunological effects in female mice; the Intermediate MRL on guinea pigs; and the Chronic MRL on rhesus monkeys. In each case, uncertainty factors were applied to address animal-to-human extrapolation.

The ATSDR did not comment on the cancer risks associated with dioxin exposure, but the EPA did provide some guidance. This information is based on human exposures to TCDD and the other dioxins.

Increased Cancer Risk – TCDD Exposure
(EPA – 2000)

<u>Lifetime Cancer Risk</u>	<u>Daily Dioxin Intake</u> (pg-TCDD/kg/day)
1 in 10	300
1 in 100	30
1 in 1000	3
1 in 1 million	0.001

I've been using TCDD and TEQ interchangeably and I really should be more precise. The TEF-TEQ system is more of a regulatory convenience than it is good science. There are a number of locations in Midland and in the floodplain with almost identical TEQ's even though actual TCDD levels and total dioxin/furan levels are widely different. I'll refer you to my March 25, 2004 letter to Ms. Cheryl Howe for more information on this subject.

Ultimately, the real impact of daily exposure levels have to be examined in the context of how the higher intake levels affect the levels of dioxins in the human body. The background exposure level of 9.4 ppt in the US translates into a body burden level of 21.1 pg/gm blood serum lipid. This is equivalent to 21.1 ppt in the blood serum lipid. Lipids are the various substances of the human body that are soluble in nonpolar organic solvents (as chloroform and ether), that with proteins and carbohydrates constitute the principal structural components of living cells (including blood), and that includes fats, waxes and other related compounds. Lipids are the fatty, waxy components of whole blood.

There is reasonable consensus that, in most cases, dioxin body burdens are proportional to daily dioxin intakes. There are a number of conditions: 2 years to 3 years of reasonably uniform dioxin exposure being the most important. Using the adult daily dioxin intakes calculated above, the average dioxin body burdens for the floodplain residents are shown below:

Dioxin Body Burden Levels
(pg/gm blood serum lipid)

Soil Dioxin Levels, ppt	<u>9.4</u>	<u>203</u>	<u>413</u>	<u>940</u>	<u>2058</u>	<u>2626</u>
Daily Dioxin Intake, pg/kg/day	0.59	1.294	1.921	3.659	7.346	9.219
Dioxin Body Burden, ppt	21.1	46.3	68.7	130.8	262.7	329.7
					⌆	⌆

The highest two body burdens are of concern since they are at levels which have been correlated with elevated levels of cancer.

There are a tremendous number of factors that affect body burden levels and daily dioxin intake levels are only one of many. Without actual testing of body burden level, the above levels are only reasonable “guess-timates”.

Dioxin levels have been determined in both Midland Area soils and in the soils of Dow’s Midland plant for almost twenty years. Given the large amount of information that has been developed on the toxic effects of dioxins, it is absolutely inconceivable, at least to me, as to why some measurement of body burden levels in exposed residents and employees hasn’t been determined so far. A few measurements of actual body burdens would go a long way to confirming or denying estimates of dioxin exposure and human uptake.

I was able to find three epidemiology studies that measured dioxin body burden levels and correlated those levels with observed health effects. Consider these studies as indicators of a possible problem rather than being an accurate predictor of what will be.

<u>Study</u>	<u>Body Burden</u>	<u>Observed Health Effect</u>
NIOSH, 1991 study of 5,172 chemical workers exposed to dioxins	233 ppt	All Cancers – 15% increase Rare Cancers – 62% increase
NIOSH, 1991 study, 1520 workers with > 20 yrs of latency	462 ppt	All Cancers – 46% increase
Spanish, 1998 study of 549 chemical workers exposed to 245-T, with > 20 yrs of latency	244 ppt	All Cancers – 410% increase over non-exposed morality
German, 1998 study of 459 workers exposed to chlorophenols, phenoxy herbicides	296 ppt	All Cancers – 154% increase

It should be noted that the body burden levels in the studies were measured some period of time after dioxin exposure was stopped. It is probable that body burden levels were much

higher while exposure was ongoing. For example, the NIOSH study measured TCDD body burden levels in employees whose last exposure was between 15 years to 37 years prior to measurement. After 17 years of ongoing TCDD exposure, the average body burden level of the exposed worker was 3000 ppt. NIOSH used an estimated half-life of TCDD of 8.7 years in the study. If a body burden measurement was made 15 years after exposure, the initial body burden level would have been approximately 9900 ppt; if 37 years after exposure, the initial body burden level would have been approximately 57,200 ppt. Unfortunately, the NIOSH report could not provide any accurate measurement of the daily dioxin intakes that resulted in these body burden levels.

The data from the NIOSH report can be used as the basis for one conclusion: the longer the dioxin exposure, the higher the body burden levels. I believe that this conclusion is also valid for Midland Area residents.

Computer Modeling of Historic Dioxin Levels

The final topic is based on the fact that dioxins and furans undergo environmental degradation and that the dioxin/furans levels being found to today were much higher in prior years... the question is, "How much higher?"

I have carried out a certain amount of computer modeling to estimate the levels of dioxins and furans that were present when dioxin deposition rates were much higher than they are today. Modeling of dioxin levels in Midland soils is relatively easily for the years after Dow upgraded the 703/830 Incinerators to improve destruction efficiencies and reduce dioxin emissions. After the late 1970's, the daily deposition of new dioxins/furans would be essentially zero and calculations based on environmental half-lives is straightforward. Estimates of dioxin levels in Midland prior to the late 1970's are a little more complicated since it is probable that daily deposition rates of dioxins were much higher.

Calculations as to dioxin levels in the river floodplain are very complex. Based on the history of Dow's waste water treatment facilities, we can assume that the concentration of dioxins/furans released to the river were high before 1937 when Dow installed the Phenolic Waste Water Treatment Plant (WWTP). Dioxin concentrations were somewhat lower between 1937 and 1946 and still lower after 1946 when Dow installed the General WWTP and the majority of Dow's chemically contaminated wastes were biologically treated.

There were a few complicating effects: (1) the volume of phenolic waste waters doubled in WW II and the production of 245-T and other TCDD contaminated products began in 1942. These two factors should have resulted in an increase in the volumes of dioxins/furans being released to the river. The unlined chemical waste ponds probably began to leak heavier-than-water chemical wastes shortly after construction. Even though Dow began to biologically treat its effluent in 1937 and in 1946, the ponds continued to leak during this time period. In the 1990's, the underground collection system was completed on both sides of the river. There is still some uncertainty if the dioxins still being found in the floodplain are from a high inventory of dioxins/furans in the river or if some unrecognized, ongoing release from the plant site is still occurring. The re-distribution of dioxin inventories in the river in the springtime is a very complicating factor when trying to model historic levels.

All these factors make computer modeling of floodplain dioxin level in earlier years very difficult. If Dow has carried out this type of modeling, the company, as you know, has not released the results of the modeling to the floodplain residents.

However, there are three properties along the river where dioxin/furan modeling might be reasonably accurate. These properties were sampled at locations that were close to the homes. If we assume that substantial flooding has not been a problem close to the homes, then it is possible that current dioxin levels were the result of environmental degradation without the deposition of new dioxins being a significant factor.

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Unfortunately, to carry out accurate modeling, the actual concentration of each dioxin and furan is required and I only have the TEQ levels. I will ask the DEQ for the information and, when available, I will provide you a summary of the results of the revised modeling.

In the meantime, for discussion purposes only, I used a typical distribution of dioxins/furans found in the floodplain to do some preliminary estimates of historic dioxin levels.

River Floodplain – Historic Dioxin/Furan Levels
(TEQ-PPT)

	<u>2002</u>	<u>1992</u>	<u>1982</u>	<u>1972</u>	<u>1962</u>
5768 River Road or 2580 River Road	1,120	3,185	11,103	51,046	319,240
6850 Wallace	1,970	5,603	19,530	89,787	561,520

Information as to when the homes were constructed will provide guidance as to what year to stop modeling. Until more accurate modeling is carried out, I do not believe that it is appropriate to calculate body burden levels from these rough estimates.

Vince, this has turned into a “you asked for the time of day... I told you how to build a watch.” The issues associated with dioxin contamination are significant and varied and I thought that it was much better to give you a complete answer than a partial one. If you need any clarification, please do not hesitate to ask.

I hope that this information will be of value to you and the other members of the CAP. I may be in Midland sometime in April. Perhaps we can get together and have lunch or dinner. I will call you as soon as my travel plans are better defined.

Sincerely,

David L. Linhardt

