Petitioned Health Consultation:
Dow Chemical Company Michigan Division
Dioxin Contamination in Soil in Midland, Midland County, Michigan

EPA ID #: MID000724724

Prepared by

Michigan Department of Community Health
Under a Cooperative Agreement with
Agency for Toxic Substances and Disease Registry
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Summary

A Midland resident and two Michigan-based environmental organizations petitioned the federal Agency for Toxic Substances and Disease Registry (ATSDR) to conduct a public health assessment of dioxin and dioxin-like compound (DLC) contamination in Midland, Michigan. The present public health consultation addresses soil contamination in the Midland area. An initial draft of this health consultation was released for public comment. The data necessary to determine if DLC-contaminated soil in the Midland area poses a public health risk are not available; therefore, the site poses an indeterminate public health hazard.

The results of several preliminary soil sampling studies indicate that DLCs have been detected in soil at two locations in the Midland community at concentrations greater than the ATSDR residential action level. DLCs detected in most of the soil samples collected from the Midland community fell between the ATSDR screening level of 50 parts per trillion (ppt) and the ATSDR action level of 1,000 ppt. Therefore, the ATSDR guidance indicates that further site-specific evaluation is necessary to determine if DLC contamination in Midland soil presents a public health hazard.

The Michigan Department of Community Health (MDCH) recommends that soil-sampling plans for the Midland community be developed and implemented; including contingency plans for potential public health actions in residential areas if DLC concentrations exceed the ATSDR action level. The MDCH further recommends a comprehensive evaluation of site-specific exposure factors for the residents of the Midland area and a comprehensive assessment of exposure that includes all identifiable sources of DLC exposure. Priority should be given to people living in areas where DLC concentrations in soil exceed the ATSDR screening value.

Purpose and Statement of Health Issues

On May 1, 2001, a resident of the city of Midland and two Michigan-based environmental organizations petitioned the Agency for Toxic Substances and Disease Registry (ATSDR) to conduct a public health assessment of DLC contamination in Midland, Michigan (Attachment A). The petitioners stated that the "likely source of this contamination is the Dow Chemical Company” (Petitioners for the Dow Midland site 2001). Specific concerns noted by the petitioners included:

- DLCs had been detected in soil in Midland at concentrations above the Michigan Department of Environmental Quality (MDEQ) residential cleanup criterion. Levels of DLCs detected in soil adjacent to the eastern perimeter of the Dow plant site and along a road way (haul route) in the community exceeded 1,000 ppt.

- DLCs had been detected in fish taken from the Tittabawassee River downstream of Midland. Levels detected exceeded the State of Michigan trigger levels for fish consumption warnings. In 1985, the U.S. Environmental Protection Agency (EPA) noted that the highest national levels of DLCs in fish were found in the Tittabawassee River.
• In 1985, the EPA "called for a comprehensive health study" of DLC exposures and the resulting health effects in the Midland community (Petitioners for the Dow Midland site 2001). No such comprehensive study has ever been performed.

ATSDR and the Michigan Department of Community Health (MDCH) have a cooperative agreement for conducting public health assessments and health consultations for potential health hazards at sites of environmental contamination within the State of Michigan. MDCH staff and a representative from the ATSDR Region 5 office visited the Midland area on July 30, 2001, and toured the Dow Chemical Company (Dow) plant site. The MDCH completed a "Petition Scoping Report" and provided the information obtained from these activities to the ATSDR on August 31, 2001. A copy of the report is provided in Attachment B. The MDCH met with the petitioners on October 3, 2001, to discuss the health assessment process and to provide an opportunity for exchange of additional information.

ATSDR responded in writing (Attachment C) to the petitioners on November 2, 2001, stating that, "After reviewing the public health issues and community concerns about potential DLC contamination and the Dow Midland facility, ATSDR has found a reasonable basis to prepare public health consultations to address the concerns associated with the Dow facility" (ATSDR 2001). MDCH has agreed to address the petition through a series of health consultations. ATSDR will review MDCH's work and provide technical support as needed.

The present consultation will only address concerns related to DLC contamination in Midland soil. Additional consultations that address other contaminated media may be developed in the future with the ultimate goal of providing a full multi-media, multi-pathway public health assessment.

DLCs are a group of chlorinated chemicals with similar structures and chemical properties, including chlorinated dioxins, furans, and some polychlorinated biphenyls. For simplicity, this group of chemicals is referred to here collectively as "dioxin-like compounds (DLCs)." Where analytical concentrations are reported in this discussion, the term total dioxin TEQ (toxic equivalents) will be used. Please see the text of the consultation for an explanation of the TEQ approach for reporting DLC concentrations in the environment.

**Background**

The Dow Chemical Company (Dow), founded in 1897, operates a chemical manufacturing plant in the city of Midland, Michigan. The Dow plant encompasses approximately 1,900 acres on the southern perimeter of the city (Figure 1). The Tittabawassee River forms the southern boundary of the plant site and flows east to the Saginaw Bay of Lake Huron.

Chemicals that have been produced at the Dow plant include but are not limited to: styrene, butadiene, picric acid, mustard gas, Saran Wrap, Styrofoam, Agent Orange, and other various chemicals including chlorpyrifos (Dursban) and 2,4,5-trichlorophenoxyacetic acid (2,4,5-T). Chlorophenol production began in 1915. Wastes generated from this process were initially disposed of in 600 acres of on-site waste ponds. During high flow periods in the early 1900s,
wastes from these ponds would be intentionally released to the Tittabawassee River (Brandt 1997). Dow currently operates its own wastewater treatment plant on-site.

Two rotary kiln incinerators were formerly used for treatment of liquid and solid hazardous and non-hazardous wastes generated from manufacturing activities at the facility (SAF·Risk 2001). These incinerators were replaced with a new incinerator that came on line in 2003. The new permitted emission rate will result in a substantial reduction in DLC emissions and impacts, compared with the operation of the two previously existing incinerators. However, ambient air dispersion modeling and monitoring indicate that the northeastern quadrant of the city of Midland has been impacted by historical emissions from the incinerators. Some site refuse has been and is taken by truck from the Dow plant to local landfills, including the currently operating Salzburg Landfill and the closed Rockwell Road Landfill, via an off-site haul route named Salzburg Road (Figure 1.) The Dow property and releases from the Dow property are subject to the corrective action program administered by the MDEQ under Part 111, Hazardous Waste Management, of the Michigan Natural Resources and Environmental Protection Act (NREPA), 1994 PA 451. The EPA sampled soil in the city of Midland in the 1980s as part of the corrective action plan for the property. Samples collected during these studies were analyzed only for the most toxic dioxin congener, 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD). Concentrations of 2,3,7,8-TCDD were found at several locations and the EPA recommended additional sampling in the future to monitor levels of DLCs in the community (MDEQ 2001).

Discussion

Environmental Contamination and Other Hazards
In September 1996, the MDEQ conducted what was intended to be the first phase of a study to determine the current levels of dioxins and furans in surficial soils and sediments around Midland. Dioxins and furans are known to be impurities in some chlorinated phenolic chemicals—such as the herbicide 2,4,5-T—that were manufactured at Dow until the late 1970s. These studies were conducted to characterize sources and to identify environmental levels of dioxins and furans that were emitted from certain production, waste treatment, and combustion processes. Samples collected during the 1996 Midland community study were analyzed for all 17 of the 2,3,7,8-substituted dioxin and furan congeners that are necessary to evaluate the total toxic equivalent (TEQ) concentration of a mixture of dioxins and furans. Samples were collected at 35 locations that included schools, parks, community use and other areas, and at 15 locations on-site at the Dow plant. Sample locations were selected as a follow-up to the studies conducted in Midland during the mid-1980s by Dow and the EPA (MDEQ 1997).
A comparison of results from samples collected in the same or nearby locations generally indicated that levels of DLCs had not changed substantially, although the data suggested a decline in the concentration of 2,3,7,8-TCDD in the Midland community samples (MDEQ 1997). All of the total dioxin TEQ concentrations for the Midland community samples (Table 1) were below the 1,000 parts per trillion (ppt) action level\(^1\) for residential soils used by the ATSDR (De Rosa et al. 1997a). However, many of the samples, especially those located directly adjacent to and downwind to the northeast of the Dow plant exceeded the MDEQ residential soil direct contact criterion of 90 ppt for total dioxins TEQs (MDEQ 2000).

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**Dioxin Toxic Equivalencies**

Dioxin toxic equivalents (TEQs) are calculated by multiplying the level of a particular dioxin-like compound by its toxicity equivalency factor (see page 7 for additional information). The resulting TEQs are then added together to determine the total dioxin TEQ concentrations in a soil sample.

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**Table 1. Dioxin TEQ Concentrations Detected in Soil Samples Collected in the Midland Community and from the Dow Plant Site in 1996**

<table>
<thead>
<tr>
<th>Soil Samples Location</th>
<th>Range of TEQ Detected (ppt)</th>
<th>Number of Samples</th>
<th>DEQ Cleanup Criterion (ppt)</th>
<th>ATSDR Screening Level (ppt)</th>
<th>ATSDR Action Level (ppt)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Southeast Quadrant</td>
<td>6 - 602</td>
<td>4</td>
<td>90</td>
<td>50</td>
<td>1,000</td>
</tr>
<tr>
<td>Southwest Quadrant</td>
<td>10 - 95</td>
<td>5</td>
<td>90</td>
<td>50</td>
<td>1,000</td>
</tr>
<tr>
<td>Northeast Quadrant</td>
<td>77 – 598</td>
<td>14</td>
<td>90</td>
<td>50</td>
<td>1,000</td>
</tr>
<tr>
<td>Northwest Quadrant</td>
<td>22 – 125</td>
<td>3</td>
<td>90</td>
<td>50</td>
<td>1,000</td>
</tr>
<tr>
<td>Dow Plant Site</td>
<td>16.5 – 8,840</td>
<td>15</td>
<td>990</td>
<td>50</td>
<td>1,000</td>
</tr>
</tbody>
</table>


The highest detected total dioxin TEQ concentration in the northeast quadrant (598 ppt) was found in a soil sample taken near the intersection of South Saginaw and Bay City roads at the northeast corner of the Dow plant site (Figure 1). Dioxin TEQ concentrations up to 602 ppt (Table 1) were also detected in soil samples taken along the Salzburg Road haul route in the southeastern quadrant (Figure 1). The highest detected total dioxin TEQ concentration on the Dow plant site was 8,840 ppt in a sample taken near the incinerator complex.

Based on the levels of DLCs detected in soil in the 1996 Midland community study, the MDEQ staff recommended that additional investigations needed to be planned and completed. Pursuant to the corrective action requirements of its federal hazardous waste permit issued in 1988, Dow

\(^1\) A concentration of chemicals at which consideration of action to interdict/prevent exposure occurs (De Rosa et al. 1997a).
is responsible for conducting several dioxin/furan monitoring programs (e.g., community and Midland Plant soil, surface water sediment, incinerator stack, ambient air, dust, food chain) under the oversight of the MDEQ and the EPA. Information from these studies was expected to be used to assess the need for corrective action activities (MDEQ 2001).

In 1998, as a follow-up to the 1996 study, Dow collected samples on the Dow Corporate Center property (Dow 1999). The Dow Corporate Center is located in the northeastern quadrant of the city of Midland within the predicted area of impact for the Dow incinerators and adjacent to residential areas (Figure 1). Therefore the MDEQ agreed to accept DLC concentrations detected in soil samples taken from the Corporate Center as a surrogate for DLC concentrations that might be found in soil in the surrounding residential areas. Historical records and photographs were consulted to identify areas that had not been disturbed during construction of the buildings now located on the Corporate Center property. Soil samples were collected from four grassy areas with some trees. Detected total dioxin TEQ concentrations ranged from 0.077 to 584 ppt in surface soils (Table 2). The highest detected concentration was in a soil sample taken across the street from residential homes.

Samples were also collected from the Midland Plant site along internal plant haul routes, along the northeast plant perimeter at the intersection of South Saginaw and Bay City roads, and along the Salzburg Road haul route (Dow 1999). Table 2 presents the range of concentrations detected in these areas.

Table 2. Dioxin TEQ Concentrations Detected in Soil Samples Collected from the Northeast Perimeter of the Dow Plant Site, Salzburg Road Haul Route, the Dow Plant Site, and the Dow Corporate Center In 1998

<table>
<thead>
<tr>
<th>Soil Samples Location</th>
<th>Range of TEQ Detected</th>
<th>Number of Samples</th>
<th>MDEQ Residential Direct Contact Criterion (ppt)</th>
<th>ATSDR Screening Level (ppt)</th>
<th>ATSDR Action Level (ppt)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Northeast Perimeter</td>
<td>6 – 1,068</td>
<td>11</td>
<td>90</td>
<td>50</td>
<td>1,000</td>
</tr>
<tr>
<td>Salzburg Road</td>
<td>10 – 2,663</td>
<td>16</td>
<td>90</td>
<td>50</td>
<td>1,000</td>
</tr>
<tr>
<td>Dow Plant Site</td>
<td>170 – 17,030</td>
<td>18</td>
<td>90</td>
<td>50</td>
<td>1,000</td>
</tr>
<tr>
<td>Dow Corporate Center</td>
<td>77 - 584</td>
<td>39</td>
<td>90</td>
<td>50</td>
<td>1,000</td>
</tr>
</tbody>
</table>

De Rosa et al. 1997a; Dow, 1999; MDEQ 2000

Total dioxin TEQs were detected on the Dow plant site at concentrations ranging up to 17,000 ppt. Detected concentrations of total dioxin TEQs ranged up to 1,068 ppt on the northeast plant perimeter on the west side of South Saginaw Road (Figure 1). Properties located directly across the road are primarily commercial with residential homes located immediately behind and to the east. Detected concentrations of total dioxin TEQs ranged up to 2,663 ppt in samples taken along the Salzburg Road haul route (Figure 1). The highest concentration was found on the haul route approximately midway between Waldo and Midland County Line roads. This sample location has been remediated, however only 16 samples were taken on a 775-foot interval over a 2.6-mile length of this road. This relatively low frequency sampling identified several areas on the haul route with elevated concentrations. The level of 2,600 ppt found at one sampling
location suggests that other areas of elevated concentrations might have been missed. Most of the surrounding property is privately owned, but currently unoccupied.

No additional soil samples for dioxin and furan analysis have been collected in the Midland community since 1998. When considered together, the data gathered in the 1996 and 1998 soil studies indicate that detected total dioxin TEQ concentrations are generally very high on the Dow plant site and decrease as a function of distance from the Dow plant perimeter. However, data are available for only one community soil sample within a ¾ mile radius of the location on the northeast plant perimeter where total dioxin TEQ concentrations were detected at 1,068 ppt.

On June 12, 2003, the Department of Environmental Quality (DEQ) issued a hazardous waste management facility renewal operating license to The Dow Chemical Company (Dow), Michigan Operations for its treatment, storage, and disposal facility in Midland, Michigan, and related corrective action activities. In addition to on-site corrective action activities, the operating license also addresses major off-site corrective action activities for Midland area soils.

**Human Exposure Pathways**

To determine whether people are or could be exposed to environmental contaminants, ATSDR and MDCH evaluate the environmental and human components that lead to human exposure. An exposure pathway contains five major elements: 1) a source of contamination, 2) contaminant transport through an environmental medium, 3) a point of exposure, 4) a route of human exposure, and 5) a receptor population. An exposure pathway is considered complete if there is evidence that all five of these elements are, have been, or will be present at the property.

#### Table 3. Exposure Pathway for Dioxin-Contaminated Soil in Midland

<table>
<thead>
<tr>
<th>Source</th>
<th>Environmental Transport and Media</th>
<th>Chemicals of Concern</th>
<th>Exposure Point</th>
<th>Exposure Route</th>
<th>Exposed Population</th>
<th>Time Frame</th>
<th>Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>The Dow Chemical Company</td>
<td>Former Incentrator Emissions, Soil Erosion, Loss and Track-Out</td>
<td>Chlorinated Dioxins and Furans</td>
<td>Midland Community Soil</td>
<td>Incidental Ingestion, Dermal Contact, Inhalation</td>
<td>Residents of Midland</td>
<td>Past</td>
<td>Complete</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Current</td>
<td>Complete</td>
</tr>
</tbody>
</table>

Chlorinated dioxins and furans have been detected in soil samples taken from the Dow plant site, from the plant perimeter, from haul routes leading from the plant to near-by landfills, from the Dow Corporate Center, and in soil samples taken from parks, playgrounds and other locations within the Midland community. The likely source of these contaminants is chemical manufacturing activities on the Dow plant site. Former emissions from the on-site incinerator complex, wind blown transport of contaminated soil, and loss or track-out of contaminants during waste transport are all possible mechanisms of transport from the Dow plant site to the surrounding community. Residents of Midland and the surrounding communities could be exposed to dioxins and furans in the soil through incidental ingestion, direct dermal contact, and inhalation of soil and dust. Chlorinated dioxins and furans were produced during historical processes; therefore, exposure is likely to have occurred in the past and will continue in the absence of any remedial action.
**Demographics**
The city of Midland is the county seat of Midland County, Michigan and encompasses an area approximately 28 square miles. The population of Midland was approximately 38,090 in 1990. Twenty-five percent of the population in 1990 was children under the age of 17 years (U.S. Census Bureau 1990).

Residential neighborhoods are located in close proximity to the northeast perimeter of the Dow plant and within a quarter of a mile from a soil sampling location where total dioxin TEQs were detected at concentrations greater than the ATSDR action level of 1,000 ppt. No soil data are available for these residential neighborhoods.

**Toxicological Evaluation**

**Health Effects**

DLCs are a group of over 210 chlorinated chemicals with similar structures and chemical properties. This group of chemicals, which includes chlorinated dioxins, furans, and some polychlorinated biphenyls (PCBs), is often referred to collectively as simply "dioxins" or "dioxin-like compounds (DLCs)." When found in the environment, dioxins are usually a mixture of several of these chemicals. Most DLCs are not intentionally produced and have no known use. Not all DLCs have the same toxicity or ability to cause illness and adverse health effects. However, it is assumed that dioxins and dioxin-like compounds cause adverse health effects through a similar biological mechanism of action. Further, the available science indicates that the health effects resulting from exposure to multiple DLCs are additive, meaning that the health effects are greater than would be expected for a single compound.

The most toxic chemical in the group is 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD). Toxic equivalency factors (TEF) have been developed to compare the relative toxicity of other dioxins and dioxin-like compounds with that of 2,3,7,8-TCDD. The levels of other dioxin-like compounds measured in the environment are multiplied by a TEF to produce a 2,3,7,8-TCDD toxic equivalent (TEQ) concentration. The resulting TEQs for all dioxin-like compounds measured in a sample are then added together to determine the total dioxin TEQ concentration for that sample.

People who have been exposed to high levels of DLCs (such as those found in an industrial setting or due to a significant industrial explosion) have developed chloracne, a skin disease with severe acne-like pimples. Chloracne can persist for years, sometimes clearing only to recur several years later. Changes in blood and urine that may indicate liver damage have also been seen in some people. Exposure to high concentrations of DLCs may cause long-term alterations in glucose (blood sugar) metabolism and slight changes in hormone levels (ATSDR 1998).

Exposure to lower levels of DLCs in laboratory animals has resulted in a wide variety of adverse health effects such as cancer, liver damage, and disruption of the endocrine system. In many species of animals, DLCs weaken the immune system and cause a decrease in the system's ability to fight infection. In other animal studies, exposure to DLCs has caused reproductive damage and birth defects. Some animal species, including monkeys, exposed to DLCs during
pregnancy had miscarriages, and the offspring of animals exposed to DLCs during pregnancy often had birth defects including skeletal deformities, kidney defects, weakened immune responses, and neurodevelopmental effects (ATSDR 1998).

It is not known whether people exposed to low levels of DLCs will experience the same health effects seen in animal studies. However, based on the available information, DLCs are believed to have the potential to cause a wide range of adverse effects in humans, including cancer. The EPA (EPA 2000) has characterized the mixture of DLCs to which people are commonly exposed as "likely human carcinogens." The EPA has also characterized 2,3,7,8-TCDD as a "human carcinogen" (EPA 2000). The U.S. Department of Health and Human Services, National Toxicology Program 9th Report on Carcinogens (NTP 2001) lists 2,3,7,8-TCDD as a substance "known to be a human carcinogen." The International Agency for Research on Cancer (IARC) has determined that 2,3,7,8-TCDD is “carcinogenic to humans” based on limited human data and sufficient animal data (IARC 1997).

**ATSDR Interim Guidance**

Because of the potential for adverse health effects in human populations exposed to environmental levels of DLCs, the ATSDR has developed interim policy guidelines to assist health assessors in identifying soil concentrations of potential concern (Attachments D and E). The guidelines recommend the tiered approach shown in the table below to evaluate DLC concentrations in soil. MDCH follows these guidelines when evaluating the public health hazard posed by DLC contamination in soil.

### Table 4. ATSDR's Decision Framework for Sites Contaminated with Dioxin and Dioxin-Like Compounds.

<table>
<thead>
<tr>
<th>Screening Level</th>
<th>Evaluation Level</th>
<th>Action Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤ 50 ppt TEQs</td>
<td>&gt; 50 ppt but &lt; 1,000 ppt TEQs</td>
<td>≥ 1,000 ppt TEQs</td>
</tr>
<tr>
<td>Health effects are unlikely and further evaluation is not necessary, unless there are community health concerns.</td>
<td>Evaluation of site-specific factors, such as • Bioavailability • Ingestion rates • Pathway analysis • Soil cover • Climate • Other contaminants • Community concerns • Demographics • Background exposure</td>
<td>Potential public health actions considered, such as • Surveillance • Research • Health studies • Community education • Exposure investigations</td>
</tr>
</tbody>
</table>

(De Rosa et al. 1997a)

The screening level of 50 ppt total dioxin TEQ is the environmental media evaluation guide (EMEG) for 2,3,7,8-TCDD. The EMEG was developed from the ATSDR minimum risk level (MRL) based on neurodevelopmental effects observed in the offspring of female rhesus monkeys exposed during pregnancy and after birth through nursing (ATSDR 1998). EMEGs are very
conservative and protective values. Generally, if soil concentrations do not exceed the EMEG, ATSDR assumes that exposure is not likely to result in adverse health effects. However, if soil concentrations exceed the EMEG, this does not mean that adverse human health effects are likely. Instead, soil concentrations greater than 50 ppt total dioxin TEQ indicate further site-specific evaluation is necessary (De Rosa et al. 1997a).

The action level of 1,000 ppt TEQ is a concentration of DLC in residential soil at which various actions might be considered to prevent or limit exposure. The action level is based on the analysis by Kimbrough et al (1984) of the carcinogenic potential of 2,3,7,8-TCDD. ATSDR recommends that the action level for soil be used in full consideration of site-specific factors that could affect total exposure to DLCs through all media and exposure pathways (De Rosa et al. 1997b).

ATSDR recommends that the action level be compared to the "maximum concentrations identified at the site" (De Rosa et al. 1997b). Dioxin TEQ concentrations greater than 1,000 ppt have been detected at two locations in the Midland community: 1,068 ppt dioxin TEQ was detected on the northeast perimeter of the Dow plant site near the intersection of South Saginaw and Bay City Roads, and 2,663 ppt dioxin TEQ was detected along the Salzburg Road haul route between Waldo and Midland County Line Roads.

Soil concentrations of dioxin TEQs that fall between the screening level and the action level warrant further site-specific study (De Rosa et al. 1997b). Many factors, such as those shown in the table above, can affect how much DLCs people are exposed to, how much is absorbed into the body, and whether or not adverse health effects will result. Most of the soil samples collected from the Midland community fall within 50 and 1,000 ppt, including all the samples taken from the Dow Corporate Center property that was intended to serve as a surrogate for the surrounding residential community.

**Background Exposure to DLCs**

An important consideration when evaluating DLC levels in soil is the level of exposure from all sources of DLCs, or the "background exposure." People can be exposed to DLCs from many sources other than contaminated soil. The general population is mainly exposed to DLCs through their diet by eating plants and animals that contain DLCs. People who live near or work at hazardous waste sites containing DLCs, waste incinerators, or manufacturing facilities that produce DLCs as a by-product could have additional DLC exposures beyond their diet. When people are exposed to and absorb DLCs, the DLCs are stored in fatty body tissues where they might persist for months or years. The half-life (the time needed for the body to rid itself of half the contaminants absorbed) for DLCs in humans is 5 to 14 years. Because they remain for a long time, DLCs accumulate in the body and can cause health effects long after exposures have ended. The amount of DLCs accumulated over time is referred to as the "body burden." The best available science suggests that body burden levels of DLCs are closely associated with the likelihood of health effects. Therefore, many scientists recommend comparing DLC body burden levels in at-risk populations to those associated with health effects observed in animal and human studies (EPA 2000, De Rosa et al. 1997b).
Because people might be exposed to DLCs from a variety of sources, and because all these exposures contribute to the body burden of DLCs accumulated over time, ATSDR recommends evaluation of the contribution of soil exposures to total exposures from all sources (De Rosa et al. 1997b). Evaluation of soil exposures would require sufficient knowledge of DLC concentrations in soil, bioavailability of DLCs in site soils, and human behaviors that can affect exposures. Additionally, evaluation of all other sources of DLC exposure would be required to evaluate the incremental contribution of soil exposures to the total body burden of DLCs in the at-risk population. Much of this information is not currently available for the Midland area.

**DLC Concentrations in Michigan Soils**
In 1997 and 1998, the MDEQ collected soil samples from 68 urban and rural locations in Michigan. These samples were taken to gain an understanding of statewide DLC concentrations that have resulted from industrial activities, waste incineration, and chemical use. Analysis of these samples indicates that DLC soil background concentrations vary from less than 1.0 ppt TEQ to 35 ppt TEQ with an average of 6.0 ppt TEQ. Similar nationwide efforts by the U.S. EPA found an average DLC soil concentration of 10 ppt TEQ (MDEQ 1999).

**Community Involvement**
This document was released for public comment in March 2002. The comment period lasted for 90 days. A public meeting was held to solicit comments. The comments that were received during the comment period are addressed in Attachment F.

**ATSDR Child Health Considerations**
Children may be at greater risk than adults from certain kinds of exposure to hazardous substances at sites of environmental contamination. They engage in activities such as playing outdoors and hand-to-mouth behaviors that increase their exposure to hazardous substances. They are shorter than adults, which means they breathe dust, soil, and vapors close to the ground. Their lower body weight and higher intake rate results in a greater dose of hazardous substance per unit of body weight. The developing body systems of children can sustain permanent damage if toxic exposures are high enough during critical growth stages. Prenatal exposures and those that occur in the first few years of life are more likely to cause permanent damage.

Fetuses, infants, and children may be especially sensitive to DLC exposure because of their rapid growth and development. In animal studies, exposure to DLC has caused reproductive damage and birth defects. Some animal species exposed to DLCs during pregnancy had miscarriages, and the offspring of animals exposed to DLCs during pregnancy often had birth defects including skeletal deformities, kidney defects, weakened immune responses, and neurodevelopmental effects (ATSDR 1998).

**Conclusions**

The data necessary to determine if DLC-contaminated soil in the Midland area poses a public health risk are not available; therefore the site poses an indeterminate public health hazard.
ATSDR classifies sites of environmental contamination into the indeterminate category when the data to make a final decision are lacking.

Several soil-sampling studies have been conducted in the Midland area by the EPA, the MDEQ, and by the Dow Chemical Company. The results of these studies indicate that dioxin TEQs have been detected in soil on the Dow plant site at concentrations up to 17,000 ppt. Total dioxin TEQs above the ATSDR action level for residential soils have been detected in off-site soil at the Dow plant perimeter at concentrations up to 1,068 ppt and along the Salzburg Road haul route at concentrations up to 2,663 ppt. Total dioxin TEQs detected in most of the soil samples collected from the Midland community fall between the ATSDR screening level and the action level, including all the samples taken from the Dow Corporate Center property intended to serve as a surrogate for the surrounding residential community. Therefore, the ATSDR guidance recommends a site-specific evaluation to determine if DLC contamination in Midland soil presents a public health hazard (De Rosa et al. 1997a, 1997b).

Soil data are not available for all of the Midland community, and it is likely that some areas are more heavily contaminated than others—particularly the northeast quadrant most impacted by historical emissions from the Dow incinerators. Of particular concern are those areas of the community closest to the Dow plant site to the east and north. No data are available concerning DLC concentrations in these areas of Midland.

Additional site-specific information including an assessment of background exposures is necessary to evaluate the incremental contribution of soil exposure to the total DLC body burden for the residents of Midland. This information is not currently available.

**Recommendations**

- Develop and implement a soil-sampling plan for residential areas closest to the Dow plant site. Sampling should begin at those residential properties that are most likely to be contaminated with DLC, east and north of the Dow plant site.

- Develop a contingency plan concurrent with the recommended residential soil-sampling plan for potential public health actions if total dioxin TEQ concentrations exceed the ATSDR action level, consistent with evaluation of site-specific exposure factors.

- Allow the sampling plans for the residential areas to be reviewed and commented upon by MDCH, ATSDR, and U.S. EPA prior to finalization and implementation. Feedback from MDCH and ATSDR will be solicited to assess whether the sampling plan will be adequate to collect the information necessary to better characterize the public health implications.

- Based on the environmental sampling results, conduct an Exposure Investigation (EI) to evaluate actual exposures in the community. The EI may consider biota sampling as a means to verify exposure to site contaminants.
Public Health Action Plan

- The MDEQ will review and approve, if acceptable, Dow proposals for interim response measures and remedial investigation and actions under the corrective action program. The MDEQ should require that Dow submit an interim response measure to develop, approve, and implement soil-sampling plans for properties in Midland most likely to have elevated levels of DLC in soil.

- The MDEQ should require that a contingency plan for potential public health actions to be developed as part of the Dow operating license under the corrective action program. Public health actions should be implemented immediately if total dioxin TEQ concentrations exceed the ATSDR action level, consistent with evaluation of site-specific exposure factors.

- The MDCH will request ATSDR collaboration and support for an exposure assessment for the Midland community. The exposure assessment will consist of an evaluation of site-specific exposure factors.

- The MDCH in cooperation with the Midland County Health Department and the city of Midland will undertake health education activities to define and respond to the information needs of the Midland community.

- The MDCH in cooperation with the Midland County Health Department and the city of Midland will be available to consult on the appropriateness and efficacy of future remedial actions.

Contact Information

If any citizen has additional information or health concerns regarding the Midland, Michigan petitioned health assessment/consultation, please contact the Michigan Department of Community Health, Division of Environmental and Occupational Epidemiology, at 1-800-648-6942.
References


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CERTIFICATION

This Dow Chemical Company Midland Division Health Consultation was prepared by the Michigan Department of Community Health under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with approved methodology and procedures existing at the time the health consultation was begun.

_____________________________________________________________
Chief, Cooperative Agreement Team, SSAB, DHAC, ATSDR

The Division of Health Assessment and Consultation, ATSDR, has reviewed this public health consultation and concurs with the findings.
Comment: Fourth full sentence, paragraph 1, page 3 is imprecise and misleading insofar as it implies that the dioxin levels in the Midland residential community were “elevated” above an applicable action level.

Response: The word “elevated” has been removed from paragraph 1, page 3 of the consultation.

Comment: MDCH has mischaracterized the relevant and available data, First, numerous soil samples exists for the residential area north and east of the Dow plant site. The Dow Corporate Center is located within 3/4 of a mile of the northeast corner of the Dow plant perimeter, and it was tested extensively in 1998 as a surrogate for the community.

Response: While the Corporate Center may be within 3/4 of a mile of the northeast corner of the Dow plant site, there are other areas both north and east of the plant that have not been sampled to date. Additionally, dioxin TEQ concentrations ranging up to 584 ppt were found on the Corporate Center property. Additional sampling is needed in nearby neighborhoods to confirm if these concentrations are present on private, residential properties and to assess the resulting health risks to the residents.

Comment: The Kociba, et al, 1978 bioassay in Sprague-Dawley rats has been the basis for most quantitative and qualitative assessments of the potential carcinogenic action of TCDD. In particular, the excess of liver tumors in female rats (the most responsive tumor site) has provided the basis for EPA’s quantitative estimate of the cancer potency of TCDD. However, MDCH’s assessment of the Kociba et al., 1978 bioassay ignores many other relevant aspects of this bioassay. Three dose levels were used in that bioassay: 0.001, 0.01, and 0.1 ug/kg-day. Although the study found some increase in tumors in the highest dose group, it also found statistically significant decreases in several tumor types. The lowest dose group had fat tissue levels of 540 ppt TCDD - more than 50 times typical human TCDD fat levels - and yet experienced no increase in any tumor type. Further, total tumor burden was lower in all dose groups compared to the control rats. Thus, the picture MDCH paints of TCDD as a potent rat liver tumorigen distorts TCDD’s overall properties.

Perhaps most significantly, however, even the rat liver tumor response in this bioassay is of little predictive value for human exposure situations. Human studies do not show any statistically significant increase in liver cancers as a result of TCDD exposure. The increases in liver tumors in the bioassay occurred only at dose levels that resulted in severe liver toxicity. Mechanistic studies of the effect of TCDD on rat liver tumor growth indicate that even when administered in conjunction with a known tumor initiator, TCDD produces increases in liver tumor precursors only at the highest dose levels tested, with lower exposures producing no increase or a decrease in the indicators for liver tumors. Thus, the animal bioassay data, while providing a basis for regulatory assessments of theoretical cancer potency of TCDD under worst-case assumptions, do not provide data to indicate a cancer risk due to exposures to low levels of TCDD in animals or in humans (e.g. 10 to 20 times background soil concentrations).

Response: While the Kociba, et al, 1978 rat bioassay is used by both the U.S. EPA and the MDEQ to calculate an oral cancer slope factor for dioxins, it is not in any way the
only study of the carcinogenic effects of dioxins, nor is liver cancer the only carcinogenic
effect noted in the vast amount of literature available. In particular, the Kociba study
noted a significantly increased incidence of squamous-cell carcinomas of the tongue, hard
palate, nasal turbinates and lung in both sexes of rats. The calculated cancer slope factor
is based on the incidence of liver cancer in female rats because this effect of dioxin
exposure showed the strongest response (i.e., had the highest rate of tumors) in this study.
While it is true that endocrine-related tumours of the reproductive system and mammary
glands were lower in the group of female rats most exposed to dioxin, the lower
incidence of these tumours was likely attributable to a significant decrease in body
weight seen in this group. A similar reduction in tumor incidence was not seen in female
rats exposed to lower doses of dioxin or in male rats. So, at the highest dose used in this
study, female rats showed less tumors of the reproductive and mammary glands, but more
tumors of the liver. At these same doses, male rats showed increased tumors of the
tongue, nasal passages, and lung without showing any reduction in any other type of
tumor.

Most of the human data available to assess the carcinogenic potential of dioxins is from
adult male workers. These studies of adult male workers indicate a significant increase in
all cancers combined and in lung cancer among the more highly exposed groups. Animal
studies suggest the possibility of a protective hormonal effect of dioxins and the risk of
lung cancer in females. Conversely, adult male workers showed no evidence of
developing liver cancer even in the workers exposed for the longest time to the highest
levels of dioxin. This is consistent with the data from the Kociba, et al, 1978 bioassay,
where the liver tumors were observed only in female rats. In addition, while it is true that
female rats that developed liver cancer also showed evidence of significant liver toxicity,
the livers of male rats also showed evidence of toxicity, but did not progress to liver
cancer. The available data, viewed in its totality rather than focusing on one study or one
result in a single study, indicate that dioxins are a potent carcinogen, that the effects
appear to be hormonally mediated, and that human response to dioxin exposure cannot be
assumed to differ from the animal models.

Comment: In other recent health assessments and consultations, ATSDR has described the
health effects of dioxins in a much more balanced fashion (see excerpts from a 2000 Public
Health Assessment and a 1999 Health Consultation). MDCH should revise the passage from
page 7 of the Draft Midland Consultation warning of “adverse health effects,...including cancer”
to include, as ATSDR has in other consultations and assessments, a balanced discussion of the
current scientific uncertainty as to whether dioxin is carcinogenic to humans.

While the MDCH Draft Midland Consultation is not an appropriate forum to critique ATSDR’s
interim policy guidelines for dioxin, note that certain aspects of the De Rosa, et al., 1997b
guidelines are extremely conservative. For example, the guidelines rely on incorrect
assumptions regarding the relationship between animal studies and human health effects. In the
De Rosa guidance, “[a] n uncertainty factor of 10 was used for extrapolation from animals”.
ATSDR has recognized in other health assessments and consultations, humans are believed to be
10 to 100 times less susceptible to dioxin than animals typically used in laboratory studies - not
10 times more as De Rosa et al., suggest. Accordingly, and especially in light of the site-specific
study that has been conducted for Midland, the ATSDR action level of 1ppb should be considered highly protective of human health.

**Response:** While the potency of dioxin as a human carcinogen may be debated, the US EPA, the World Health Organization, the International Agency for Research on Cancer, and the U.S. National Toxicology Program have all concluded that 2,3,7,8-TCDD is a human carcinogen. MDCH will continue to use and cite the ATSDR dioxin policy as a basis for assessment of the human health risks of dioxin contamination in soil.

**Comments Pertaining to the Health Outcomes Data Section**

Numerous comments were received by MDCH concerning the Health Outcomes Data section of the Consultation. MDCH has removed this section from the Consultation and has not, therefore, addressed each individual comment here. Health data and a review of the available epidemiological studies will be comprehensively presented in future Public Health Assessment documents. Comments on the health outcomes section are provided below.

**Comment:** Ample studies of humans have been conducted based upon workers who have been occupationally exposed or residents who have been accidentally exposed to dioxins. These studies do not show any increased cancer risk or mortality rates among those exposed to even high levels of dioxins.

**Comment:** A comprehensive, community-approved new monitoring system should be set up to collect data on relevant health effects for the Midland and downriver communities. A special form should be designed for health practitioners specific to this region to collect data. Consideration should be given to providing a control population.

**Comment:** The Midland County Health Department claims three "studies," -- actually reviews of available databases on disease incidence -- give the community of Midland a clean bill of health. Unfortunately, the data show a more complicated picture.

- Note the birth defects reference categorized as "integument" shows a statistically significant higher incidence in Midland 1992-96 when compared to the rest of Michigan. The report characterizes it as not significant but it is. The 95% CI is 1.01-2.61. (integumentary defects are important because this is what was seen in the PCB exposed kids in Taiwan, etc). Further, 6 of the 8 birth defect categories monitored showed increases over expected numbers. Again, with a small population and rare disorders, statistical significance is hard to achieve.

- The birth defects registry is notorious for underreporting. In addition, the exposed population is relatively small and the effect would have to be many times above the background level in order to be statistically significant, given the rarity of the disorder. It is important to communicate this point to the public. Further, it is unclear that children sent to specialty clinics are properly categorized in the registry.
- Again, gross birth defects are the ones that get reported. More subtle losses or defects are not reported.

- The four primary cancer sites -- prostate, lung, breast and colo-rectal are not considered primarily dioxin-related (although a recent report in Environmental Health Perspectives\(^2\) suggests a link with breast cancer -- Breast cancer incidence went up marginally in Midland from 1985-99 (age adjusted rate 7.4-7.9/10,000). Dioxin-related cancers would be a better measure to review. However, those cancers are very rare and therefore statistical significance is difficult to achieve with such a small exposed population. Again, this point must be communicated to the public.

**Comment:** In addition, no such reviews of the data have been conducted for the potentially most exposed population along the Tittabawassee and Saginaw Rivers.

**Comment:** Although gross disorders more directly attributable to dioxin contamination are rare, and the population impacted is relatively small, it is therefore even more remarkable that Midland has experienced statistically significant elevated rates of a number of these conditions over the years. Those conditions include soft tissue sarcomas, cleft palate, and other dioxin exposure related birth defects. Diane Hebert notes these in her comments, and we incorporate those citations by reference here.

**Comment:** Media reports have pointed to more than 20 “studies” purporting to show no health effects, primarily to workers, from dioxin. A close review of Dow’s web site, which includes over 20 “papers” shows these papers are actually a mix of scientific papers and letters to the editor or commentaries. Only ten are actually journal articles, primarily from the same cohort. All of the papers discuss only cancer mortality, not incidence, or other relevant health effects related to dioxin (with the exception of chloracne, a condition associated with very high levels of exposure).

**Comment:** These papers are not new data and have been reviewed and considered by the EPA in their reassessment of dioxin, and by other expert bodies when weighing evidence on the hazards of dioxin. Even with Dow’s data, those expert bodies have agreed dioxin is a potent toxin to humans, and dangerous for workers.

**Comment:** Further, the papers do not include a more recent review by Dow, which did find additional excess cancers, although that study’s results were initially characterized otherwise. Dow has indicated they have submitted the recent paper for publication but it was rejected. It is unclear whether Dow has submitted that paper to ATSDR/MDCH for review, but it certainly should be reviewed as part of the health consultation.

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\(^2\) Environmental Health Perspectives Volume 110, Number 7, July 2002

Serum Dioxin Concentrations and Breast Cancer Risk in the Seveso Women’s Health Study
Marcella Warner,1 Brenda Eskenazi,1 Paolo Mocarelli,2 Pier Mario Gerthoux,2 Steven Samuels,1,3 Larry Needham,4 Donald Patterson,4 and Paolo Brambilla2
Comment: Given that dioxin’s toxicity can be more likened to the contaminant lead (in the importance of non-cancer effects and developmental and functional losses), Dow’s overwhelming emphasis on cancer (and specifically, cancer mortality) in its comments is strange. They unscientifically take issue with the findings of several international agencies that dioxin is carcinogenic, yet point to their worker cancer mortality studies as evidence of dioxin’s safety. Dow does seem to agree that cancers with strong associations to dioxin are rare, and their rarity makes statistical significance difficult.

Comment: Numerous reports have surfaced from surgeons, pediatricians, and other health professionals about the anomalous health effects from Midland area residents. One report from a surgeon in Saginaw suggested more surgery for cleft palate (associated with dioxin exposure) in the area than he had ever encountered in a practice that spanned a number of regions. Numerous reports from physicians at Mott Children’s Hospital in Ann Arbor suggest Midland children are over-represented in the state with rare conditions. These reports have persisted over the years. Attempts to quantify these problems are frustrated by deficiencies in data collection and the likelihood that rare conditions are often not treated in the Midland area. Anecdotal reports of elevated rates of disease conducted by independent citizen reviews have also been reported. Those comments seem to warrant an investigation of the reports that do not appear to have ever been compiled and evaluated.

Comment: Anecdotal reports from veterinarians suggest anomalous birth defects and other problems in farm animals born downwind of Midland. Small populations, and lack of data tracking impede an understanding of any trends or anomalies here.

Comment: While anecdotal reports are, by definition, not rigorous reviews, and can be misleading, they can also be early warning systems for problems that may be escaping official data collection systems for a variety of reasons.

Comment: There is more than ample evidence that Midland and downriver residents have been exposed to elevated levels of dioxin. Biological sampling of the population, however, has not been done to answer the most important question – do the residents of Midland have elevated levels of dioxin in the bodies and breast milk. We urge biological sampling of wildlife and humans to determine if area residents have elevated levels of dioxin. Sampling protocols and plans should be developed in consultation with the community. Sampling of humans should focus on those most likely to have elevated levels, including fishers, long-term residents, those in closest proximity to contaminated soils, and those directly downwind from dioxin sources. Sampling should include breast milk.

Comment: If biological sampling reveals elevated levels of dioxin, further public health interventions should be considered. For instance, additional screening to determine if thyroid levels in women of childbearing age have been altered will be critical in order to determine if routine screening should be part of regular prenatal care. Dioxin is known to alter thyroid levels in those exposed, even at relatively low levels of exposure. Thyroid is critical to development for the fetus. A simple test for pregnant women can determine if thyroid levels are adequate for fetal health. Interventions to address imbalances are possible. Ongoing monitoring of this program, and regular evaluation, can serve to determine its usefulness.
Comment: A new Environmental Health Perspectives article indicates that circulating levels of thyroid might not be the best predictor of thyroid function, so appropriate tests may need to be found, but in any case, this should be further explored.

Comment: There may be other proactive public health interventions to address the impacts of dioxin exposure that should be explored.

Comment: Please consider an independent comprehensive study of the overall health of Midland before issuing a new report.

Comment: I believe the Midland area needs a comprehensive health study done. I do not believe there are any studies to date regarding the synergistic effects of dioxin with other chemicals such as PBB, marathon or any of the numerous other chemicals with which Midland residents are bombarded, permitted and intermittently.

Comment: “I know that soil was moved to new subdivisions from flood plain areas and fields, so soil may have been moved back and forth between the two zip codes.” (Comment addressing the zip code study – may explain why it seems inconsistent with expectations)

Comment: The MDCH study is incomplete, misleading and incorrect – omits previous soil and epidemiological studies. One error example - the zip code areas that MDCH report indicates a potential health concern are upwind from the Dow facility.

Comment: The MDCH report ignores 20 years of past studies concluding that Midland residents are as healthy or healthier than the average person in the state of Michigan.

Comment: Since it is already known that cancer incidence and birth defect rates in the Midland area are the same or lower than the rest of the state, what is the MDCH expecting to find from its study?

Comment: The 48640 zip code area was shown not to be as healthy as the 48642 area. Since the 48640 area is comprised of lower income residents, doesn’t this have to be taken into account when making a comparative health assessment?

Comment: The zip code with the higher cancer rate (48640) has less dioxin than 48642.

Comment: Since Midland is a community with a high degree of population turnover, it would be important to study the health effects of lifelong residents. Previous studies that report Midland residents are healthier than the average state resident do not take the duration of time an individual has resided in Midland into account.

Comment: The Midland report should include a detailed discussion of specific, relevant health findings, such as the number of incidences of soft-tissue, prostate and lung cancer, as well as birth defects data, compared to State and county rates. Even findings that are not “statistically
significant” may be indicators of potential problems that could be investigated in a future health evaluation.

**Comment:** The public should be told that there are high incidences of certain cancers and birth defects in Midland and Saginaw.

**Comment:** Any evaluation should include all Midland zip codes that touch the plant site, not just the ones immediately north and south of the plant (48640 and 48642). Since the prevailing winds are west and northwest, the east and southeast zip codes would be relevant to the study.

**Comment:** An evaluation should also include all zip codes in downriver communities. These areas could be downwind and downriver so health problems could be air or water related.

**Comment:** Recommendations should include a means of identifying disease clusters, such as going door-to-door, to determine what diseases are present in the immediate population.

**Comment:** Impacts to children and females vs. males should be considered to determine differential effects.

**Comment:** Local medical and mental health professionals should be trained in the diagnosis, treatment, and reporting of health effects expected in this environment of contaminated air and soil.

**Comment:** The “Zip Code Study” dated July 23, 2001 reports that the 1998 rate for invasive bone and soft-tissue cancer for the 48640 zip code were significantly high but this is not noted in the Health Consultation.

**Comment:** Midland is the only county in the state in the “significantly high” category for chromosomal anomalies.

**Comment:** It would be helpful to know the Midland incidence of non-Hodgkin’s lymphoma, as soft-tissue sarcomas are rare but elevated in the presence of dioxins.

**Comment:** How does our health data compare with other similarly contaminated communities?

**Comment:** The health data indicates that there could be a problem. The report should clearly state this.

**Comment:** I suggest that a map of the Midland area, emphasizing ZIP code areas, be included in the soil consultation report and the language be changed as follows: On page 10, 2nd paragraph under “Cancer Incidence for the Midland area”, 2nd sentence “Zip code 48640 encompasses the southwest area of Midland including the Dow plant site, urban areas north and northwest of Dow, and a portion of Salzburg Road just east of Dow, while Zip code 48642 includes property to the north and northeast of the Dow plant site.”
Comment: I would recommend that you delete the Dow study (occupational) from the Health Consultation until it has been submitted for peer review and published.

Comment: The Midland community needs to be told that there has never been a comprehensive health study in Midland and therefore it is impossible to suggest that Midland health has not been adversely impacted by dioxin and other chemicals.

Comment: Health issues that need to be studied or followed-up include soft tissue sarcoma, cleft palate, all other birth defects- please identify “other”-, and autism.

Comment: If this is such a huge health problem, why aren’t some of the health effects showing up very clearly in public health data? Are the people in Midland healthy because the worst effects have been washed downstream into Saginaw?

Comment: Current and previous residents in any contaminated area should be interviewed about possible health problems that might have been caused by dioxin and tested for concentrations in their bodies.

Comment: Zip Code Study: MDCH imprudently relied on a “zip code study” that it knew to be flawed and, in fact, demonstrates an inverse correlation between the amount of dioxin in residential soils and incidence of cancer.

Comment: Epidemiology Studies: MDCH did not meaningfully address important epidemiology studies which found no consistent adverse health effects among Dow workers occupationally exposed to concentrations hundreds or thousands of times greater than the theoretical exposure from residential soils.

Comment: Please consider an independent comprehensive study of the overall health of Midland before issuing a new report.

Comment: I believe the Midland area needs a comprehensive health study done. I do not believe there are any studies to date regarding the synergistic effects of Dioxin with other chemicals such as PBZ, malathion or any of the numerous other chemicals with which Midland residents are bombarded, permitted and unpermitted.

Comment: In the Summary section, the report suggests that there are no data regarding health risks in Midland Community. This is untrue and the summary section should be modified with this suggested language inserted after the second sentence as a replacement for third sentence.

"Over the years there have two birth defects studies, cancer studies, including a soft tissue sarcoma study and numerous studies by the Dow Chemical Company of their employees. None of these studies identified any health problems in Midland in excess of what one might in any other community in Michigan. Although no problems were identified in any health studies, because these were population based studies, some elevated problems may not have been detected from these studies. Therefore, it is our opinion that the site poses an indeterminate public health risk."
Comment: The use of the cancer incidence study conducted by the MDCH is disingenuous. "The greatest number of Midland residents that would be effected [sic] by dioxin live North East [sic] of the plant in zip code 48642 which had cancer incidence rates lower than 48640. The Plant location is on the Eastern [sic] edge of zip code 48640 boundary with the majority of the population in 48640 living north and West [sic] of the Plant.

"The only relevance of this study [is] to show that even with soil dioxin levels elevated above the ATSDR Screening level, there was no evidence of higher cancer rates in those areas. The cancer rates in the elevated dioxin areas were lower than the other comparables. Recommend if this study has little relevance to the issues of soil dioxins in Midland, it should be removed from the report. If it is included, a more complete description of its relevance and interpretation of the study results is warranted."

Comment: "The Report briefly mentions and quickly dismisses a Birth Defects Study conducted in 1999 by the MDCH as being irrelevant and unreliable. There have been two birth defects studies conducted by MDCH at the request of the Midland County Health Department in addressing the concern of possible elevated birth defects due to dioxins present in our community. In both studies no elevated birth defect rates were observed."

Comment: All current information we have about the Midland community is that our health status is similar to what one might find in any community in the State of Michigan. Our cancer rates and birth defects rates show no increased health problems as a result of dioxin in the soils in Midland.

Comment: August 6, 2001 – Midland County Health Department was contacted by the Dow Chemical Company requesting information about a cancer zip code study being conducted in Midland. MDCH had not contacted, involved or even informed the Midland County Health Department that the zip code study was being conducted.

Comment: August 30, 2001 - The Midland County Health Department...pointed out several flaws in the (zip code) study that still have not been addressed. MDCH representatives noted that what the study showed in that there is not indication of health problems in Midland because the higher soil dioxin areas had lower cancers.

Comment: Dow has commissioned numerous, extensive studies regarding the health effects of Midland workers who were occupationally exposed to hundreds or thousands of times as much dioxin as is the general Midland population from residential soil. Those studies have not found any adverse health affects at low-level exposures of the type to which certain Midland residents could potentially be exposed, and the results of studies of Dow and other workers exposed to very high levels of dioxin are inconsistent at best. In light of the extensive research of more exposed populations, including more highly exposed Midland workers, there is no scientific basis to believe that Midland residents exposed to orders if magnitude lower levels of dioxin in soils would face any discernible health effects whatsoever. MDCH should correct the Draft Health Consultation to reflect this reality.
Comment: As noted above, more than ample data exist to find the Midland residents face no apparent health risk based upon the small concentrations of dioxin above background levels found in Midland residential soils. MDCH should amend this statement accordingly, and in light of the extensive studies of the Dow worker cohort, including substantial data and analyses that MDCH did not consider, which show that there is no basis to believe Midland residents would experience any adverse health effects as a result of exposure to Midland residential soils.

Comment: MDCH should delete the entire discussion that comes under the heading “Cancer Incidence for the Midland Area” because it is based upon a highly unreliable “zip code study” that MDCH has acknowledged is of little or no scientific value. Indeed, ATSDR has noted the severe limitations of zip code studies under even the best of circumstances. Moreover, as explained more fully in Dow’s main comments (see Section VI.A), the Midland zip code study is seriously flawed because: (i) it relies on census data from 1990, even though the relevant zip code boundaries changed on July 1, 1996; (ii) some of the cancer causes in the registry likely have been misclassified based on post office boxes or places of treatment, as opposed to place of residence; and (iii) although MDCH mentions that the study found slightly elevated levels of cancer in zip code area 48640, it fails to note the proximity of the Dow plant site to the 48642 zip code area, the fact that the prevailing winds carry emissions in the direction of 48642, and the fact that the zip code (48642) with the higher dioxin levels (whatever their cause) has the lower cancer rates.

Accordingly, MDCH should delete all reference to the zip code study in its revised Health Consultation. If MDCH insists on including the zip code study, it would be irresponsible not to acknowledge all the substantial shortcomings of the study (including those noted by ATSDR with respect to an early zip code study) in revised Health Consultation. See Section VI.A.

Comment: MDCH improperly dismisses one of the Dow study’s key findings in above-quoted passage. The finding is significant, however, insofar as the Dow worker cohort showed no association between exposure to high levels of dioxin and mortality rates. Moreover, total mortality rates for all causes of death were lower among Dow workers studies compared with white male cancer mortality rates for the general population in the U.S. (See Section VII.) MDCH should replace the sentence that suggests the lower death rate is “not remarkable” and acknowledge the significance of these data in a revised Health Consultation.

Comment: MDCH fails to mention that the data show fewer than expected deaths with respect to a roughly equal number of other cancer sites (such as lung, liver, and brain). Moreover, all cancers combined are at expected levels. Further, the 245 workers in this study with chloracne- and presumably very high dioxin exposure- had a deficit of cancer. Rather than highlighting only those cancers where the incidence is slightly greater than expected, MDCH should present all of the foregoing data in a revised Midland Health Consultation. See Section VII.B.

Comment: As explained more fully in Dow’s main comments, this statement is inconsistent with (i) the findings of the International Agency for Research on Cancer’s (“IARC”) recent review of dioxin health effect studies, (ii) the confounding factors present in the studies MDCH references, (iii) serum lipid level comparison; and (iv) the study of accidentally exposed residents in Seveso, Italy. MDCH should revise the above-quoted reference, as the body of
available scientific evidence does not demonstrate any “consistent” elevation of any particular cancers due to dioxin exposure

MDCH: *There were two cases of soft-tissue sarcoma in the cohort, an excess also reported in some other studies.* (Dow 1997,1998)

**Comment:** As explained more fully in Dow’s main comments, MDCH has failed to note that the slightly higher than expected incidence of soft tissue sarcomas is not statistically significant. Moreover, the most recent update found that the SMR for these sub-cohorts had decreased compared with the original study, which indicates a decreased likelihood of a casual connection between dioxin exposure and STSs. See Section VII.B.2. MDCH should address these more recent data, and the statistically insignificant nature of the earlier data upon which it relied, in a revised Midland Consultation.

MDCH: *There are significant limitations in generalizing from the Dow study to the general population. First, the study is limited by the size of the exposed cohort, which is not sufficient to detect moderate increases in incidence rates for many of the cancer types of concern. Second, the individuals included in the cohort were all males. The effect of dioxin exposure on exclusively female cancer types cannot, therefore, be considered. Lastly, and of most importance, the assignment of workers to the “exposed” and “unexposed” groups and to the assumed level of exposure was based on job history, that is the work time spent in areas of the Dow plant where there was potential for dioxin exposure.*

**Comment:** These criticisms are unjustified. First, with respect to the size of the study, the Dow cohort of 2,187 workers represents the largest single plant mortality study of dioxin workers ever reported. Second, with respect to data on females, although admittedly not statistically significant, the Dow study did include five female workers with dioxin exposure, and all five are still alive, as reported in the most recent update. Third, with respect to the dioxin exposure characterization, this aspect of the study was based on comprehensive and reliable indicators of dioxin exposure, including industrial hygiene monitoring, analysis of historical plant operations, and detailed work histories of all cohort members.
Comment: According to the ATSDR Toxicological Profile for Chlorinated Dibenzo-p-Dioxins, the following populations may have higher exposures than the general population. Those groups include those who were the victims of industrial accidents, those living in proximity to sites where CDD’s are produced as chemical by-products and those where the chemicals are disposed, those living near hazardous waste incinerators, or sites of environmental contamination. Interestingly, the Midland area has all of these things. Dow has uncontrolled contamination sites on site containing dioxin laden waste, a landfill off-site they use to dispose of dioxin-contaminated waste, a manufacturing site with production of compounds known to generate dioxin as a byproduct, two hazardous waste incinerators with dioxin emissions, old industrial accidents which potentially contained dioxin contaminated wastes, and recent incidents involving the loss of containment of dioxin-laden dried soils. Further, measured levels of dioxin in the community and downriver and orders of magnitude above background levels.

ATSDR also suggests that other populations may be at risk because of dietary behavior, including recreational and subsistence fishers, subsistence hunters, infants and children who are breast fed, and subsistence farmers and their families living in areas with elevated dioxin levels that also consume home grown foods. Note that fishers, hunters and breast-fed children are at risk without even living in contaminated areas, and represent the most at-risk populations in the Midland/downriver area. The Midland/Saginaw area has representatives of each of these populations (however only anecdotal reports of subsistence farmers available).

Area residents may have elevated rates of PCB’s in their bodies because of major PCB contamination in the Saginaw area. Michigan fish are also widely contaminated with PCB’s. Michigan was also the site of a serious and widespread PBB industrial incident that resulted in elevated levels of PBB’s in many Michiganders. While TEQ’s have not been defined for PBB’s, some are dioxin-like in their activity and must be considered as additive to other exposures. In order to be complete and accurate, all dioxin-like compounds must be considered when assessing potential health impacts, not only those likely to be present in the general population, but also those specific to this region. Historically, there have been fish consumption bans based on PBB contamination in the Chippewa River, the Pine River, the Saginaw River and the Tittabawassee River. There have been numerous PCB and dioxin fish advisories as well (see Toxic Chemicals in the Great Lakes and Associated Effects, Environment Canada, page 230).

Local residents are often simultaneously exposed to various airborne chemicals as well as exposure to dioxin-contaminated soils. What are the combined effects and what is the potential for synergism? (Lists several examples of reported releases of ammonia, vinyl chloride, benzene, cyclohexane, and others as example)

Response: Comments noted. These issues will be addressed in future documents.

Comment: The draft dioxin assessment report by US EPA “Exposure and Human Health Reassessment of 2,3,7,8-(TCDD) and Related Compounds” was not cited.
Response: The EPA dioxin reassessment was not cited because it is not yet final. The draft EPA document carries a notation of “Do not cite or quote.” MDCH will cite the dioxin reassessment in the future if EPA removes the notation.

Comment: Have people with soft tissue cancers or children with learning difficulties been tested for exposure to dioxin?

Response: Neither the MDCH nor the ATSDR is aware of any testing of Midland residents for dioxin exposure other than testing which may have been conducted by Dow for its worker studies.

Comment: The State needs to notify people about growing zucchini, yellow squash and pumpkins on dioxin-contaminated soil as these absorb higher levels (than other produce).

Response: This comment was conveyed to the Michigan Department of Agriculture. The Michigan Department of Agriculture addressed this issue at a public meeting in October 2002 by indicated it was safe to eat squash as part of a normal diet. The Department of Agriculture has also put a fact sheet on its website entitled, “Food, Farming and Gardening Guidelines for Minimizing Dioxin Exposure.”

Comment: The chances of getting cancer from the general environment are almost nothing. The EPA has overestimated the dioxin danger by about a factor of 10.

Response: The EPA’s dioxin reassessment is not yet final and is undergoing review both internally and within several federal agencies. The appropriateness of EPA’s assessment has not yet been determined.

Comment: Studies have proven that grass cover provides a reasonable barrier to dioxin exposure from soils.

Response: Grass cover does indeed provide a barrier to underlying soil contamination. However, grass cover is easily disturbed when people landscape, garden, or even when children play. For this reason, the MDEQ generally recommends a minimum of 6 inches of clean soil be placed over contaminated soil before grass cover is established.

Comment: At what depths were the soil and sediments collected?

Response: The depth of soil samples varies depending on where they were collected. In Midland, soil samples were generally taken from the top 1 inch of soil, just under any vegetative cover.

Comment: Are you using TEQ and TEF data from the WHO scheme (1998) or the EPA (ATSDR) recommended in De Rosa et al., 1997? Does it make any difference in interpretation?

Response: The TEQs reported in the March 2002 health consultation were based on the toxicity equivalency factors (TEFs) recommended in the De Rosa et al., 1997 citation.
Currently the MDEQ and the MDCH are using the TEFs recommended in 1998 by the World Health Organization (WHO). Although the revised TEFs differ slightly from the previous recommendations, it makes little difference in calculation of the TEQs for Midland because the TEFs have not changed for the congeners found most abundantly in Midland soils.

**Comment:** There has been inadequate sampling to date to determine hot spots in Midland and downriver. Extensive sampling will be required to determine exactly where dioxin is elevated, in order to remediate contaminated soils and sediments. Further, extensive sampling of indoor dust in order to quantify that source is necessary. There may be areas where water samples may also be necessary, although dioxin's tendency to adhere to particles should be a consideration when selecting water-testing sites. All sampling plans should be developed in consultation with the community and the most impacted residents.

**Response:** The MDEQ is responsible for developing sampling plans and/or approving any sampling plans developed by Dow. The MDEQ has formed a “Community Advisory Panel” or CAP to provide an opportunity for the community to comment on these plans.

**Comment:** The ATSDR's 1 ppb action level was derived more than 15 years ago and has been referred to as a policy-based level, rather than a risk or health-based level. The number does not incorporate the newest synthesis of dioxin science in the dioxin reassessment. Further, the ATSDR and MDCH in numerous communications recognize a variety of other relevant numbers to consider when evaluating the potential health impacts from contaminated soils.

With regard to the policy derived cutoff of 1 ppb: 1 ppb is much higher than the levels found to cause adverse health effects. In fact, a “no effect” level has not been scientifically established. Those who have suggested that levels below 1 ppb are “safe” are misleading the public.

**Response:** ATSDR's Policy Guidance on Dioxin in Soil concludes that "the action level of 1 ppb (TEQ) for dioxin and dioxin-like compounds, when coupled to a site-specific context of evaluation for the range >50 ppt to < 1 ppb TEQs in residential soils, is protective of public health and continues to represent a level at which consideration of health action to interdict exposure, including cleanup should occur." However, in recognition of the recent advances in understanding the toxicity of dioxin, the ATSDR is currently reconsidering the 1,000 ppt action level and the accompanying dioxin policy.

**Comment:** Michigan residential and industrial soil cleanup levels compared with some other EPA Regions are not protective. Both EPA Region III and EPA Region IX have cleanup standards far more protective than those promulgated in Michigan. The residential standard in Region III is 4.3 ppt and the industrial standard is 38 ppt. The residential standard in Region IX is 3.9 ppt and 27 ppt for industrial sites. A recent review by the MDEQ of the Michigan methodology suggests that the cleanup standard should actually be lowered when information from the dioxin reassessment is considered.

**Response:** Comment noted, however, the MDCH does not develop MDEQ cleanup criteria.
**Comment:** Midland and downriver soils are significantly elevated compared to state and national averages. The average of dioxin soil testing in the state is 6 ppt. The national average is between 8 and 10 ppt, although those “background” levels include measurements from the Midland community (158 ppt) which substantially skew the findings. Absent Midland levels, the background dioxin levels are more likely 1 ppt (see Lester, Background Levels of Dioxin in Soil). These levels are in dramatic contrast to those found in the Midland community and downriver. Why would Michigan want to increase its toleration of this highly toxic, known carcinogen to 150 or 1,500 ppt?

**Response:** The MDEQ has collected and analyzed soil samples from areas with no known source of dioxin contamination. The range of total dioxin TEQ concentrations is from 1 to 35 ppt. No samples from Midland are included in this range. A discussion of dioxin background data for Michigan has been added to the consultation. Concentrations of environmental contaminants that exceed background levels do not necessarily represent an exposure risk. Soil cleanup criteria or action levels consider the inherent toxicity of a chemical as well as the potential for people to be exposed to the contaminant in soil or other environmental media. The ATSDR screening level of 50 ppt, as well as the MDEQ criteria of 90 ppt, are based on these considerations.

**Comment:** I am not convinced that further sampling is a cost effective use of taxpayer money, there is a health effect concern for the residents of Midland, or any level of analysis or remediation would satisfy the concerns of the activists who petitioned for the study. The absence of data does not, in and of itself, justify another study.

**Response:** The MDCH has recommended further soil sampling in Midland in neighborhoods in closest proximity to the Dow plant site. No soil dioxin data are available for these areas. Some samples taken nearby, but just inside the fence line on the Dow property had levels of total dioxin TEQs that exceeded the ATSDR action level of 1,000 ppt. It is possible that people living in these areas may be experiencing higher levels of dioxin exposure that cannot be known for certain without additional soil sampling.

**Comment:** If public policy requires additional sampling, there should be a cost-benefit analysis to determine whether or not the overall effort is justified in light of the resources consumed, social impact on the community and projected improvements in the quality of life for Midland residents.

**Response:** Neither federal nor Michigan law requires that a cost-benefit analysis be conducted before environmental contamination is investigated. Such assessments generally come after the nature and extent of contamination is identified, and options to clean up or remediate the contamination are being explored.

**Comment:** There is some general concern that Dow’s brine well system is being used to dispose of potentially hazardous chemicals.
Response: Comment noted. The MDCH will look into this issue and it will be addressed in future documents if necessary.

Comment: Ambient Air Monitoring: MDCH failed to discuss ambient air monitoring conducted in 1997-98 which showed the level of dioxins in ambient air is similar to that in rural, non-industrialized parts of the US.

Response: The current consultation focuses on soil dioxin contamination only. Other media will be addressed in future documents.

Comment: Dust Control Program: MDCH failed to acknowledge that Dow has had an extensive dust-control program in place since 1980.

Response: Comment noted, however dioxin levels in Midland soils remain above the MDEQ residential soil direct contact criteria and the ATSDR screening level.

Comment: Residential Soil Sampling Data – MDCH failed to acknowledge that more than 100 soil samples have been collected in residential areas of Midland and not one exceeded the ATSDR action level of 1ppb. Two samples that did exceed 1ppb were in areas zoned for industrial use.

Response: Soil sampling results for the city of Midland are discussed in the Environmental Contamination and Other Hazards section on pages 3-5 of the consultation. Table 1 on page 4 provides the results of residential soil sampling as well as the Dow plant site. Total dioxin TEQs were detected at concentrations ranging up to 2,663 ppt along Salzburg Road. This area is a road right-of-way and public access is not restricted. Total dioxin TEQs were also detected at concentrations up to 1,068 at the northeast perimeter of the Dow plant site. Commercial and residential properties are located immediately across Saginaw Road and these areas have not been sampled to determine if the elevated concentrations extend off the Dow property.

Comment: Won’t excavation or other remediation efforts increase airborne exposure?

Response: The MDCH has not proposed any remedial methods in the city of Midland. Rather, we have recommended additional sampling to define the extent of dioxin soil contamination.

Comment: Is the MDCH trying to impose unnecessarily stricter limits (than the EPA) on potentially harmful substances in our community?

Response: The MDCH is not a regulatory agency and cannot impose contaminant limits. MDCH recommends that State of Michigan law and cleanup criteria be applied in the city of Midland.

Comment: Please follow up on the soil reports for Central Middle School and the Central Park areas as well as the elementary schools near the plant. Further sampling was proposed years ago
but never addressed. The number of children with learning disabilities in these school attendance areas seems out of proportion with those in other parts of town.

Response: This comment has been noted and will be taken into consideration during any sampling activities that occur within the City of Midland.

Comment: Consideration should be given to the fact that Love Canal, Hyde Park and Syntex soils capped at 50 ppt. Given the background contamination in the food supply, communities can tolerate little additional accumulation from soil.

Response: This comment has been noted. As indicated in the health consultation, additional data and information is needed to evaluate this situation.

Comment: Consideration should be given to the total impact/exposure risk imposed on the surrounding communities due to historic and current chemical manufacturing and waste incineration. Air and soil contamination should be considered as well as exposure to other pollutants.

Response: This comment has been noted. The goal is to develop several health consultations and then a comprehensive public health assessment.

Comment: The report says, “Levels of dioxins detected in soil adjacent to the eastern perimeter of the Dow plant site and along a road way (haul route) in the community exceeded 1,000 ppt.” I would like to see this modified to clearly identify the sites. What cross streets or what bounded areas are impacted?

Response: Cross roads have been added to the description of these sample locations on pages 4 and 5 of the consultation.

Comment: There is information that this is a follow-up to the studies of the mid-1980s. Is this routine monitoring?

Response: No, the 1996 MDEQ sampling investigation and the 1998 Dow investigation would not be considered "routine monitoring." Routine environmental monitoring programs typically involve sampling the same location(s) for the same parameters at periodic fixed, interval(s). The 1996 and 1998 studies were in follow up to earlier Dow and U.S. EPA studies that were conducted in the early to mid-1980s. These earlier studies did not provide the information necessary to determine if the dioxin and furan levels in the Midland Area soils are below the applicable regulatory criterion that have been developed to be protective of human health. Most of the data collected from these earlier studies was limited to one dioxin congener, 2,3,7,8-TCDD. It is now known that there are 17 dioxin and furan congeners that are important in determining dioxin-like toxicity. The 1980s data are incomplete in this regard, and provide only a partial estimate of dioxin-like toxicity, resulting in the need to conduct further investigation. The MDEQ conducted the initial follow up study in 1996 because that is when Michigan was
delegated the responsibility to oversee corrective action at hazardous waste facilities by the U.S. EPA.

Comment: I ask that you continue to include and consider skin and inhalation exposures in the evaluations in addition to the oral pathway which seems to be the center of discussion. These other pathways should not be ignored, especially with regard to children.

Response: Comment noted. MDCH is not ignoring these pathways of exposure. However, the soil ingestion route of exposure has a greater impact on overall exposure than does either dermal (skin) or inhalation exposure. A comprehensive public health assessment will incorporate these exposure pathways.

Comment: Soil samples should be taken all over town as contaminated soil may have been moved to a new construction area.

Response: Comment noted. The MDCH will consult with the MDEQ as soil-sampling plans for the city of Midland are developed.

Comment: Suggested correction for your report conclusion: “The data necessary to determine if dioxin-contaminated soil in the Midland area poses a public health risk are not available; therefore the site poses an indeterminate public health hazard.” The correct way to write the first part of the conclusion above is: “Based on a study of all available data, there is no current evidence of a public health risk in the Midland area due to dioxin-contaminated soil.”

Response: MDCH does not agree with the suggested language. The conclusions are based on the lack of adequate environmental data, not human health data. No change has been made in the consultation.

Comment: "The wording in the second paragraph under the Summary should be modified to more accurately reflect the soil dioxin levels in community"

Response: The second paragraph of the Summary was modified by adding numeric values for the ATSDR screening and action levels for dioxin to provide a frame of reference.

Comment: The summary section should reflect the current status of the mitigation efforts imposed by the MDEQ and how they impact on further exposure in the community.

Response: MDCH does not agree that mitigation efforts should be addressed in the health consultation. Remedial activities to be performed by Dow under its Corrective Action obligations have not yet been determined at the time of this writing.

Comment: "The last paragraph in the summary section discusses a "comprehensive exposure assessment" that should be implemented in any areas where soil levels exceed the ATSDR screening level. This statement is in direct conflict with the ATSDR Decision Framework that
states an Exposure Assessment is appropriate when the soil levels reach or exceed 1,000 ppt action level." The comment continues by suggesting revised language.

**Response:** The summary section has been revised for clarity.

**Comment:** "On the top of page 2 reference is made to a 1985 EPA study and provides a single quote about a comprehensive health study. This "quote selection" gives a slanted view of the conditions in Midland." This section should be amended.

**Response:** The bulleted item referred to by the commenter is taken directly from the petition letter dated May 1, 2001, and is included as the third bullet in a list of three that provide the concerns of the petitioners listed in the letter. The reader is advised to read the previous 3 paragraphs to put this item into appropriate context. No change is warranted in the consultation.

**Comment:** "In the 2nd paragraph page 2, the report references "community concerns about potential dioxin contamination and the Dow Midland facility." The process used to develop this consultation ... have not in anyway engaged the Midland community. This section of the report should be modified by (1) removing reference to "community" in the 2nd sentence of the 2nd paragraph and interesting "petitioner" in its place; and (2) add "the community has not yet been engaged in the development, review, or comment on this report."

**Response:** The language the commenter objects to is a direct quote from a letter dated November 2, 2001 from ATSDR to the petitioners. Language in direct quotes is not generally subject to change: if changed, the language is no longer a quote. The current process of providing a public comment period is intended to engage the community in review and comment.

**Comment:** The "Report" significantly understates the decline in dioxin concentrations between the EPA 1986 sampling and the 1996 study. The community soil levels were nearly 50% lower (without mitigation) which was probably due to the natural decay of the dioxins.

**Response:** MDCH disagrees with this statement. The 1998 Dow Corporate Center Study that was conducted in follow-up to the 1996 MDEQ study demonstrated that there is a high degree of spatial variability in dioxin concentrations between sampling points that are relatively close to each other. Minor disturbances of the upper portion of the soil horizon from soil disturbances such as landscaping appear to substantially influence dioxin concentrations. Therefore, based on the results of the 1998 Dow study, it is inappropriate to conclude that there has been a measurable decline in dioxin soil concentrations from the 1980s to the 1990s. There is no basis to conclude that any significant "natural decay" of dioxins and furans is occurring in Midland soils.

**Comment:** "Table 2, Page 5 – Data for the non-residential areas should be separated from residential areas and compared to ATSDR (or EPA) recommendations for Industrial or other appropriate designations."
Response: ATSDR does not recommend a specific level of dioxin in soil for industrial land use. Rather, the ATSDR Decision Framework recommends site-specific evaluation of exposure pathways that would include identification of appropriate receptors and the level of exposure that may occur on non-residential property. Table 2 has been revised to show the MDEQ Industrial soil direct contact criterion of 990 ppt as the appropriate comparison value for soil samples taken from the Dow plant site.

Comments: The following language is recommended for the last sentence of the first paragraph on page 6: Chlorinated dioxins and furans are very persistent in soil, and community exposure to levels in the ATSDR screening range of 50 –1000 ppt may have occurred in the past. There is no evidence the community has been exposed to levels that exceed the ATSDR action level of 1,000 ppt. Further, there is not current evidence that these exposures have resulted in health problems in the Midland population."

Response: The suggested language implies that adequate environmental data are available and have been evaluated to determine that exposures to soil concentrations greater than 1,000 ppt have not occurred. Further, the suggested language implies that adequate health effects data are available, and proper evaluation has shown no health problems in the Midland population. Both implications are unfounded and the suggested language will not be incorporated into the consultation.

Comment: "Under the ATSDR Interim Guidance section on page 8, last sentence, the report states "...and whether or not adverse health effects will result". This is very controversial, as the writer of the report cannot state that health effects will occur as a result of exposures to these very low levels of dioxin. These statements reflect on the credibility of the Consultations again conclusions are being drawn before site-specific evaluations have been conducted. If health effects will occur, why are we not seeing health effects in the community?"

Response: The sentence when read in context conveys the uncertainty surrounding dioxin exposure, uptake, and the potential health effects that could result. The language will not be changed. The consultation does not state that health effects will occur, rather it states that many factors may affect whether or not health effects will occur.

Comment: “In the ATSDR Child Health Initiative section, the last paragraph discusses animal studies as evidence of health concerns regarding birth defects. However, in these laboratory studies the defects observed were from substantially higher doses of dioxin that [sic] is being seen in soils testing from the community. Recommend modifying the report by inserting "high" before the work exposures in the next to last sentence on page 9."

Response: The MDCH does not agree that high levels of dioxin were administered to animals whose offspring showed adverse health effects in laboratory studies. "High" is an imprecise term and several studies have shown adverse health effects in the offspring of animals fed a diet that contained as little as 50 parts per trillion in-feed. The section will not be modified.
Comment: The report should make it clear that the intent is to use ATSDR's Decision Framework to guide further study of the community.

Response: Explicit language has been added to the ATSDR Interim Guidance section to make this intent clear.

Comment: The commenter recommends the following actions for the Midland community:

- Develop a soil-sampling plan for the residential area immediately north and east of the Plant.
- Soil dioxin levels that exceed 1,000 ppt should be mitigated.
- Exposure Investigations to measure actual exposure in the community should proceed in areas where soil sampling exceeds 1,000 ppt. Exposure Assessments should include a comprehensive Human Health Study that is scientifically sound and peer reviewed.
- Require Public Health actions be implemented if the ATSDR Action Level of 1,000 ppt is exceeded.
- For area of the Midland community that have soil dioxin levels between 50 and 1,000 ppt; the ATSDR Evaluation of site-specific factors should be conducted.
- The Midland County Health Department expects to be included as a full partner in the development and implementation of any additional soil testing, exposure investigations, community surveys, Human Health Studies or community education efforts.

Response: The response is provided below, point-by-point.

- Sampling plans are under development for the areas north and east of the plant indicated by air dispersion modeling to be the most highly impacted by air-born emissions from the Dow plant incinerators.
- MDCH agrees that soil dioxin levels that exceed 1,000 ppt may require mitigation.
- MDCH agrees that exposure investigations should be conducted for areas where soil dioxin levels exceed the ATSDR action level of 1,000 ppt. However, evaluation of site-specific exposure factors may also be needed where soil dioxin levels are detected between 50 and 1,000 ppt. Human health studies are generally the next step if an exposure investigation indicates that people are exposed and are absorbing contaminants into their bodies. An epidemiological health study is not conducted as part of an exposure investigation.
- MDCH agrees that public health actions should be implemented if soil dioxin levels exceed the ATSDR action level.
- MDCH has, on several occasions, in person and in writing, asked the Midland County Health Department to participate in the health consultation process.

Comment: Analysis of blood lipid levels in studies of other occupationally exposed workers and a credible estimation of such levels in Midland residents, shows that the most highly exposed occupational workers had lifetime average serum lipid levels 1,000 to nearly 10,000 times greater than the estimated contribution to serum lipid levels of the most highly contaminated Midland soils. Accordingly, based on the consistent findings in the occupational studies that
low levels of dioxin exposure do not indicate any increased risk of adverse effects, there is no reason to believe limited dioxin intake such as that from residential Midland soils, would have adverse health effects on Midland residents.

Response: Exposure to dioxins in soil and the resulting incremental addition to a person’s total dioxin body burden cannot be addressed without consideration of other exposures. If, as many scientists believe, exposure to dioxins in the food supply is already at a level that may cause health effects, then any additional exposure is undesirable.

Comment: Ample data exist to show that the northeast quadrant of Midland, including the residential areas located therein, are not impacted by dioxin anywhere near the ATSDR action level of 1ppb.

Response: Neither MDCH nor MDEQ agree that ample data exist for the City of Midland. Additional sampling is required under the hazardous waste management facility renewal operating license issued by the MDEQ to the Dow Chemical Company on June 12, 2003.

Comment: A contingency plan for public health actions if total dioxin TEQ concentrations in soil samples exceeds the ATSDR action level is not necessary because none of the previous samples have shown dioxin levels above 1,000 ppt.

Response: A contingency plan, developed prior to soil sampling, is necessary to prevent undue delay in taking actions to protect public health should dioxin soil levels be found in excess of the ATSDR action level.

Comment: All interested stakeholders should be allowed to review sampling plans, not just the MDCH, ATSDR and the U.S. EPA.

Response: The recommendation that MDCH, ATSDR and the U.S. EPA be allowed to review any sampling plans is not exclusive of other stakeholders. The MDEQ has formed a Community Advisory Panel to allow the public to review and comment on actions planned for the city of Midland under the hazardous waste operating license issued to Dow.

Comment: The recommendation to provide the National Institute for Occupational Safety and Health (NIOSH) with copies of this consultation since dioxin contamination is located on an operating facility is gratuitous (i.e., unnecessary).

Response: Agreed. This recommendation has been removed from the Consultation.

Comment: An Exposure Investigation is not necessary because there is no scientific basis to find that Midland soils pose any potential public health hazard.
Response: The consultation concludes that there is an indeterminate public health hazard because the scientific evidence is not available to determine whether or not dioxin in Midland soil poses a hazard. An exposure investigation is necessary to determine if people in Midland who are exposed to contaminated soil are being affected.

Comment: June 27, 2001 - "The Midland Community Leader Panel on Dioxin learned that ATSDR was petitioned to conduct a health assessment by several environmental groups. Michigan Department of Community Health (MDCH) initiates a public health assessment of Midland without contacting or seeking involvement of the Midland County Health Department (MCHD)." The Midland County Health Department was denied the opportunity to review and provide input into the MDCH report before it was issued.

Response: The MCHD was notified on June 28, 2001, by way of a letter addressed to Mr. Charles Newell that (1) ATSDR had received a petition for a public health assessment in Midland, (2) that ATSDR had a cooperative agreement with the MDCH to conduct public health assessments in Michigan, and (3) that MDCH would begin collecting the available information and would use this information to determine IF a public health assessment was warranted for Midland. The letter stated in part that "MDCH will keep you informed of the progress on this project and welcomes the involvement of the Midland County Health Department." The letter was on MDCH letterhead and signed by Linda D. Dykema (then Larsen), Section Manager of the MDCH Toxicology and Response Section.

Comment: "July 19, 2001 – MDCH staff contact the Midland County Health Department and inform them they are making a tour of the Dow Plant facility and of the community. The Health Officer stated he would like to be a part of the community tour. A few days letter the MDCH called and noted they stated [sic] the Midland County Health Department could not be a part of the tour."

Response: Two members of the MDCH staff, an ATSDR representative, and a DEQ staff person met with Dow representatives, and toured Midland and the Dow Plant Site on July 30, 2001. MDCH had received requests from local Midland officials, Dow representatives, and the petitioners all of whom wanted to take part in the tour. When it became apparent that 20 or more individuals wanted to be included, MDCH made the decision that this would not be a productive use of staff time. Because not all parties could be accommodated, the decision was made to disinvite ALL those who had made requests. Dow representatives were, of course, present during the tour of the plant site.

Comment: November 8, 2001 - Midland County Health Department received notice from ATSDR that a reasonable basis to proceed with a public health assessment for the Midland Community and the MDCH was under contract to conduct the study. Midland County Health Department was given no information by MDCH.

Response: MCHD was notified by a letter addressed to Mr. Charles Newell dated November 8, 2001 that the ATSDR had found a reasonable basis to proceed with a public health assessment in Midland. The letter was on MDCH letterhead and signed by Linda
D. Dykema (then Larsen), Section Manager of the MDCH Toxicology and Response Section. The letter invites Mr. Newell to contact Dr. Dykema with any questions or additional information he'd like to provide.

**Comment:** The MDCH met with the petitioners on October 3, 2001, to discuss the health assessment process and to provide an opportunity for exchange of additional information. In contrast to the cooperative relationship described above - in which MDCH apparently solicited information from, and gave information to, the petitioners - MDCH met with Dow only belatedly and briefly. Moreover, MDCH ignored vital information that Dow summarized and presented.

**Response:** Page 2, 2nd paragraph of the consultation states that MDCH and a representative of the ATSDR Region 5 office met with Dow and toured the Dow facility on July 30, 2001 **before** meeting with the petitioners. MDCH spent several hours with Dow officials and considered all information presented.

**Comment:** Napalm has never been produced in Midland. In addition, Dursban is Chlorpyrifos and therefore they should not be both listed. Moreover, the selection of the above-listed items, among the hundreds of beneficial chemicals and products Dow produces or has produced in Midland, distorts the reality of the vital role these chemicals play in agriculture, pharmaceuticals, consumer products and various other sectors. The statement should be replaced with something more objective and neutral. For example: “Early products include bromides, chlorine, caustic soda, carbon tetrachloride, chloroform, arsenical insecticides, magnesium metal, and indigo. The product line expanded rapidly to include monochlorobenzene, salicylic acid, calcium chloride, phenol, ethylene dibromide, and later styrene, vinyl, and vinylidene chloride. Currently, more than 500 chemicals are produced, including industrial and specialty chemicals, plastics, herbicides and pesticides, pharmaceuticals, and consumer products.”

**Response:** The first sentence in paragraph 6, page 2 of the consultation has been revised to read, “Chemicals that have been produced at the Dow plant include but are not limited to: styrene, butadiene, picric acid, mustard gas, Saran Wrap, Styrofoam, Agent Orange, and various pesticides including Chlorpyrifos (i.e., Dursban) and 2,4,5-trichlorophenol (2,4,5-T).”

**Comment:** Extensive air monitoring conducted in 1997-98 has shown that the dioxin levels in Midland’s ambient air are not elevated. Moreover, extensive and effective dioxin emission controls are in place at Dow’s facility, and therefore current emissions from Dow’s plant site are not contributing to any increase in dioxin levels in Midland residential areas. Absent other dioxin sources, dioxin levels in Midland soils should not increase appreciably. MDCH should describe the current level of strict emissions control on Dow’s incinerator to provide the proper context for this statement, and should note that any dioxin concentrations attributable to Dow are from historic, not current, operations at the Midland plant site.

**Response:** Since public comment release of the consultation in March 2002, the two former Dow incinerators have been replaced. Paragraph 7, page 2 of the consultation has been revised.
Attachment F

Response to Public Comment Summary
Responsiveness Summary

MDCH provided an initial 60-day comment period that was extended an additional 30 days at the request of several commenters. The comment period closed on June 14, 2002. Comments received after June 14, 2002 will not be addressed here.

Some commenters expressed concerns related to comments made by other individuals, companies, or organizations. These comments are not germane to the Public Health Consultation and will not be addressed further here.

Where possible, several related comments have been combined to reduce redundancy.

Several commenters provided journal articles, research papers, and other reference materials and the MDCH thanks the contributors for this information. Several commenters also provided historical background information concerning previous studies of dioxin contamination in the city of Midland, contamination in media other than soil, or other contaminants of concern in Midland. MDCH thanks the contributors and will retain this information in our files for use in developing a comprehensive Public Health Assessment for the city of Midland.

Dioxins are a group of chlorinated chemicals with similar structures and chemical properties including chlorinated dioxins, furans, and some polychlorinated biphenyls. For simplicity, this group of chemicals is referred to here collectively as "dioxins." Where analytical concentrations are reported in this discussion, the term total dioxin TEQ will be used. Please see the text of the consultation for an explanation of the TEQ approach for reporting dioxin concentrations in the environment.

Comment: The MDEQ “residential cleanup criterion” is not relevant to MDCH’s analysis, which should be based on the ATSDR action level of 1ppb. The MDEQ generic 90ppt cleanup criterion is invalid as a matter of law because it has not been properly promulgated or calculated and is being re-evaluated. Therefore, MDCH should delete all references to the MDEQ “cleanup criterion” and should analyze soil samples only in the context of the relevant action level of 1ppb. At a minimum, should MDCH insist on maintaining a reference to the 90ppt proposed standard, MDCH should describe it more accurately as a “proposed generic residential soil direct contact criterion.”

Response: The MDEQ residential soil direct contact criterion is legally promulgated within the Administrative Rules for Part 201, Environmental Remediation, of the Natural Resources and Environmental Protection Act, 1994 PA 451, as amended. It should also be noted that the ATSDR screening level of 50 ppt must also be considered when assessing the public health hazard posed by dioxin contamination in soil. MDCH considers all relevant environmental standards and criteria when assessing public health risks.

Comment: Site Specific Assessment – MDCH failed to mention a comprehensive site-specific assessment of the Midland area that concluded that a soil criterion of 1.48 ppb dioxin in residential soils would be fully protective of human health in Midland. The assessment,
performed by a leading expert on dioxin, determined that a soil criterion of 1.48 ppb PCDD/F TEQ would be fully protective of public health in Midland, based upon site-specific considerations. The methods used in that report are consistent with ATSDR’s guidelines. Moreover, criterion was developed pursuant to, and is consistent with, the Michigan Natural Resources and Environmental Protection Act. Given that extensive soil sampling has already been conducted and not a single residential sample even approaches the ATSDR action level, let alone the site-specific soil criterion, no further-site specific evaluation is necessary to determine that dioxin in Midland soil does not present any apparent public health hazard. MDCH should amend the above-quoted statement to reflect the findings of the assessment.

Pursuant to ATSDR guidance, the only action that is appropriate where, as here, the ATSDR screening level but not the ATSDR action level has been exceeded is the performance of a site-specific exposure assessment. An assessment taking into account the site-specific exposure factors typically employed by ATSDR for this purpose has already been conducted for Midland. Because a scientifically sound site-specific assessment has already been conducted for Midland and extensive sampling has shown that residential soils do not exceed the ATSDR action level or the site-specific criterion, no further action is necessary.

Response: A report entitled “Calculation of a Site-specific Soil Criterion for Midland, Michigan” was prepared by Exponent for the Dow Chemical Company. This document was submitted to the MDEQ on April 9, 2002—several weeks after the MDCH draft health consultation was released on March 4, 2002, therefore, a discussion of the assessment contained in this document could not have been included in the Draft Health Consultation. The site-specific criterion and supporting documentation was reviewed by MDEQ and MDCH staff and deficiencies in the assessment were noted in a memo dated September 9, 2002 from the MDEQ Toxics Steering Group Dioxin Subcommittee to then MDEQ Deputy Director Arthur Nash. The 12-page memo details the regulatory and scientific deficiencies noted in the approach taken by Exponent in the development of the proposed site-specific criterion. Based on the noted deficiencies in the assessment, a lower site-specific criterion of 831 ppt was proposed in the draft Corrective Action Consent Order for the Dow Chemical Company dated November 6, 2002. This Consent Order was withdrawn on December 27, 2003 and no further proposal for a site-specific criterion or a site-specific risk assessment has been submitted to any state agency. Therefore, the consultation cannot consider or reference a site-specific criterion developed for the city of Midland.

Comment: The report fails to mention that a bioavailability study was conducted last year and is currently under review.

Response: The study referred to was, in fact, an in vitro (i.e., in glass) bioaccessibility laboratory study intended to approximate the bioaccessibility of dioxin in soil in living animals. In vitro studies are not conducted in living animals and it is not known as yet whether this study will adequately mimic the dioxin availability in whole animals. In March of 2002, the bioaccessibility study was undergoing review by the Michigan Department of Environmental Quality. It has since been determined that further study in live animals (in vivo) is needed to validate the results of the previous in vitro study.
results of the in vivo study cannot be incorporated into the consultation process until they have undergone review, and are determined to be adequate to represent the availability of dioxin in Midland soil. At the time of this writing, all bioaccessibility studies are on hold pending resolution of regulatory issues associated with the Dow Correction Action requirements in the city of Midland.

**Comment:** The relevant standard for the Dow plant site is the EPA preliminary remediation goal for industrial sites, which is 5-20 ppb.

**Response:** The relevant and applicable standard for the Dow plant site is the legally promulgated MDEQ Industrial soil direct contact criterion of 990 ppt (i.e., 0.99 ppb). Table 1 has been revised accordingly in the consultation.

**Comment:** The maximum concentration at the Corporate Center was 513 ppt TEQ, not 583 ppt.

**Response:** The concentration reported by Dow at the Corporate Center sampling location with the highest detected concentration was 476 ppt TEQ. A duplicate sample taken at the same location showed 614 ppt TEQ. The average of these two concentrations is 545. However, Dow reported these concentrations on a “wet weight” basis that must be adjusted for the percent of solids in the sample. The correct adjusted concentration detected at this location is 583.51 ppt TEQ. MDCH mistakenly rounded this value to 583, when it should have been rounded to 584. This error has been corrected in the consultation.

**Comment:** Given the levels of dioxin in TEQ in Midland soils, any exposure from soils is not consequential with respect to health effects. As Dow has discussed elsewhere, the maximum known concentration in the Midland community to which the general population may be exposed is 0.513 ppb. Thirty years of daily exposure to this maximum concentration in Midland would correspond to a daily intake of 0.069 picograms per kilogram per day (pg/Kg/day), or approximately 7% of the daily EPA estimated “background” intake. Thirty years of exposure to the average concentration (0.0999 ppb) yields an intake of 0.0135 pg/Kg/day, or about 1 percent of the overall daily dietary intake – which is consistent with the conclusion reached by EPA in the dioxin reassessment. In short, even if individuals were exposed on a daily basis for 30 years to the maximum PCDD/F TEQ concentration measured in Midland soils (a highly unlikely occurrence), the soil-related dose would be a tiny fraction of the “background” dose associated with the diet. MDEQ should revise this passage to reflect the facts regarding the percentage intake that Midland residents face as a result of exposure to the levels of dioxin in Midland soils. MDCH should acknowledge: (i) the relative exposure impact of the food supply compared with soils; (ii) the fact that the overwhelming majority of the dioxin intake of Midland residents is no different from that of any other U.S. resident since there is no commercial farming in Midland, and therefore, (iii) that there is no scientific basis to conclude that residential soil exposure at levels well below 1 ppb will have any health effect on Midland residents.

**Response:** As previously stated, the maximum detected concentration of dioxins on the Dow Corporate Center was 584 ppt. MDEQ and MDCH toxicologists have developed calculations to determine the contribution of exposure to dioxins at varying soil
concentrations to daily intake rates. Using national average dioxin concentrations in various foods coupled with intake levels provided in the U.S. EPA Exposure Factors Handbook, and assuming soil concentrations of 10 ppt, total average adult exposures to dioxins from all sources would equal 0.61 pg/Kg-day. Soil exposures would constitute 0.6 percent of that total. However, exposure to soil dioxin TEQ concentrations of 584 ppt will result in a daily dioxin intake of 0.21 pg/Kg-day from soil alone, or 26% of the total daily intake of 0.80 pg/Kg-day from all sources combined including diet. This estimate assumes that residents in Midland are exposed to dioxins in their diet at average concentrations for the United States and that 50% of the dioxins in soil will be absorbed into the body. If, however, consumption of locally caught fish containing 24 ppt TEQ is assumed, total daily intake rises to 1.24 pg/Kg-d and the relative contribution of soil exposure drops to 17%. If this same fish-eating average adult were exposed to dioxins in soil at concentrations of 1,000 ppt, the total daily intake rises to 1.4 pg/Kg-day with the contribution of soil exposures again rising to 26% of the total. These calculations do not support the position that exposure to dioxin-contaminated soils in Midland constitutes only a “tiny fraction” of diet-associated exposures. Further, these estimated daily intake rates for average adults exceed the ATSDR minimal risk level (MRL) of 1 pg/Kg-day indicating the need for further site-specific evaluation.

**Comment:** The "Report" contains conflicting comments regarding dioxin levels in soils in the community. The "Report" states; "The results of several preliminary soil sampling studies indicate that dioxins have been detected in soil at two locations in the Midland community at concentrations greater than the ATSDR action level." Again the Report on pg 8, states, "Dioxin TEQ concentrations greater than 1,000 ppt have been detected at two locations in the Midland community...referencing the Plant perimeter and the Salzburg haul route." The Plant perimeter and the Salzburg haul routes should not be included as community samples because they are not residential areas. These samples are not located in residential areas, are highly localized, and have little or no bearing on residential exposure. Indeed, one of the two sample sites is located inside the Dow fence line and is inaccessible to the public. The other sample site, which was located along an industrial haul route, has since been remediated.

**Response:** The Dow plant site is located in the Midland community. The samples taken at the Northeast Perimeter of the Dow plant site were taken just within the fence line. Samples from this area were taken at either the base of the fence or within 10 feet of the fence. The 1998 sampling that was conducted in this area was in response to the elevated level of dioxin in soils in the traffic island at Saginaw and Bay City Roads. Three of the 11 samples from the Dow fence line exhibited concentrations of dioxins and furans near 1 ppb: NEPP-S-02 and its replicate (.930 and .473 ppb); NEPP-S-10 (.923 ppb); and adjacent sample NEPP-S-11 (1.07 ppb). In addition, it should be noted that samples NEPP-S-10 and NEPP-S-11 are located directly across Saginaw Road from the Corning Lane neighborhood. The chain link fence at the perimeter of the Dow facility does not provide a barrier to the movement of contaminants and there is no reason to think that the concentrations of dioxins and furans would be significantly different on the outside of the fence line vs. the inside of the fence line.
The Salzburg Road haul route is also located in the Midland community. This area is unrestricted to public access. Sixteen samples were taken on a 775-foot interval over a 2.6-mile length of this road. This relatively low frequency sampling identified several areas on the haul route with elevated concentrations. A dioxin level of 2.6 ppb (2600 ppt) found at one sampling location suggests that other areas of elevated concentrations may have been missed and further sampling and evaluation should be conducted.

Comment: We suggest educational materials be designed that are accurate, comprehensive, and informative for community members who must make choices about their behavior and living situations in an area with elevated levels of dioxin. Any educational materials must point out the limitations of current data collection in answering critical questions such as- has Midland or Saginaw residents’ health been affected by exposure to dioxin.

Response: Comments noted.

Comment: We urge cooperative agreements between local and state public health officials in order that accurate statements are being provided to the community.

Response: Comment noted.

Comment: Why were the public meetings scheduled in Saginaw for May 1st and Midland for May 2nd cancelled for unknown reasons?

Response: MDCH had some difficulty identifying available and appropriate locations to hold public meetings. When suitable locations were identified, MDCH postponed the meetings for a few days to allow the public adequate notification time.

Comment: MDCH ignored the conclusion of the US EPA that current levels of dioxin do not pose an unacceptable public health risk (based on EPA’s own testing).

Response: The EPA “Risk Assessment for Dioxin Contamination, Midland, Michigan” was released in March of 1988. MDCH reviewed the EPA risk assessment, however, scientific advancements in understanding the potential health risks of exposure to dioxin suggest that an updated risk assessment is necessary. For example, the EPA based its risk assessment only on detected concentrations of 2,3,7,8 tetrachlorodibenzo-p-dioxin (TCDD) and did not account for the dioxin-like toxicity of other dioxin and furan congeners detected in Midland soil samples. The MDEQ estimates that the levels of TCDD found by the EPA in the 1980’s accounts for only one-third of the total dioxin like toxicity associated with dioxin TEQ concentrations in Midland soil.

Comment: How does dioxin biodegrade and at what rate? Do dioxins degenerate to furans? Is there any chance that the analytical process itself can cause dioxins to become furans?

Response: Dioxins degrade very slowly in the environment as a result of exposure to sunlight (photolysis) or by the action of microorganisms in the soil. The ability of a chemical to degrade is generally described in terms of the chemical half-life, or the time
it would take for one-half of the amount of chemical present to degrade. Estimates of the half-life of dioxins range from 9 to 15 years at the soil surface, to 25 to 100 years in subsurface soils. Some microorganisms are capable of removing chlorine atoms from dioxin and furan molecules, but the rate of degradation varies for each congener. It is possible that dechlorination by microorganisms could convert highly chlorinated and less toxic congeners to potentially more toxic compounds (ATSDR1998). Dioxins do not degenerate or degrade into furans, either in the environment or in the laboratory.

Comment: Dioxin congeners found downriver indicate dioxin-contaminated PCB’s as the source.

Response: During the 2002 Phase II sampling in the flood plain of the Tittabawassee River, the MDEQ initially conducted PCB analysis on soil samples taken both upstream and downstream of Midland. PCBs were present in only very low concentrations at all locations and contributed very little to the total dioxin-like toxicity of the samples. Therefore, it does not appear likely that PCBs are a source of the dioxin contamination found either in Midland or in the flood plain of the Tittabawassee River. The Phase II data will be discussed in future public health assessment documents.

Comment: Is dioxin being released into the air from the Dow plant and at what rate? (Alternately, is this dioxin contamination historical or ongoing?)

Response: The dioxin contamination in soil in the city of Midland is primarily historical. In 2001, MDEQ issued a permit to Dow Chemical to install and operate a new hazardous waste incinerator under stringent conditions. State and federal standards required a maximum achievable degree of emission reductions for dioxins and a thorough multipathway risk assessment was performed as part of the permit application and review process. The risk assessment accounted for the existing locally elevated dioxin levels in soil and fish, as well as the incremental future impacts of emissions. The new permitted emission rate will result in a substantial reduction in dioxin emissions and impacts compared to the operation of the two previously existing incinerators. The multipathway risk assessment demonstrated that the additional potential exposures and risks associated with the new incinerator were acceptably low. The new incinerator is currently operational and early testing indicates that it will meet all emission standards specified in the permit.

Comment: The table below suggests the kinds of health effects likely to be experienced by residents of Midland and downriver areas exposed to elevated levels of dioxin. Data on these effects are generally not reliably collected or not collected at all (with the exception of genital malformations). Most would be impossible to measure at the clinical level and are only measurable at the population level. Yet they represent the most important risk to area residents.
<table>
<thead>
<tr>
<th>Body Burden (ng/kg)</th>
<th>Species</th>
<th>Health effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>83</td>
<td><strong>Human</strong></td>
<td>Decreased testosterone</td>
</tr>
<tr>
<td>73</td>
<td>Rats</td>
<td>Genital malformations (female)</td>
</tr>
<tr>
<td>64</td>
<td>Rats</td>
<td>Decreased sperm count</td>
</tr>
<tr>
<td>54</td>
<td>Monkeys</td>
<td>Endometriosis</td>
</tr>
<tr>
<td>50</td>
<td>Rats</td>
<td>Immune suppression</td>
</tr>
<tr>
<td>42</td>
<td>Monkeys</td>
<td>Endometriosis</td>
</tr>
<tr>
<td>42</td>
<td>Monkeys</td>
<td>Object learning</td>
</tr>
<tr>
<td>28</td>
<td>Rats</td>
<td>Decrease in sperm counts</td>
</tr>
<tr>
<td>19</td>
<td>Monkeys</td>
<td>Learning disability</td>
</tr>
<tr>
<td>14</td>
<td>Human</td>
<td>Decreased testes size</td>
</tr>
<tr>
<td>14</td>
<td>Human</td>
<td>Altered glucose tolerance</td>
</tr>
<tr>
<td>10</td>
<td>Mice</td>
<td>Adult immune suppression</td>
</tr>
<tr>
<td>7</td>
<td>Mice</td>
<td>Increased susceptibility to virus</td>
</tr>
<tr>
<td>7</td>
<td>Monkeys</td>
<td>Altered immune response</td>
</tr>
<tr>
<td>10</td>
<td></td>
<td>Current background body burden nationally (It is not known whether Midland and Saginaw residents have body burdens above the national average, but it is certainly a strong possibility. In addition, the average masks considerable variation among individuals.)</td>
</tr>
</tbody>
</table>

**Response:**  Comment noted. MDCH will consider these potential effects if any health studies are conducted in the future.

**Comment:** Levels of dioxin in indoor dust must be considered as part of the health consultation. A German study of dioxin levels in dust near contaminated areas found significant levels of dioxin in indoor dust. It is important to consider dioxin-laden dust as an important source of exposure in the health consultation.

**Response:** MDCH agrees and is developing a protocol for indoor dust sampling.

**Comment:** If the EPA estimates over 94% of exposure to dioxins come from food, why is the MDCH not focusing its efforts on eating habits?

**Response:** The EPA estimates of dioxin exposure cited in the comment are based on the assumption that most people are exposed to dioxin in soil at levels of 1 to 10 ppt total dioxin TEQs. Under this scenario, soil dioxin accounts for less than one percent of a person’s total dioxin exposure. However, if a person is exposed to soils with dioxin levels of 1,000 ppt total dioxin TEQ, their total exposure is increased and soil exposure accounts for about 37 percent of their total dioxin exposure.
While very low levels of dioxin in soil occur naturally as a result of forest fires and other types of combustion, most dioxin is released to the environment by human activity. Dioxin contamination is present in food because it is present in the environment. While the MDCH recommends that people eat a diet low in animal fat and follow the Michigan Fish Advisory to reduce dioxin exposures, it also advocates for environmental clean up to begin to reduce the amount of dioxin in these foods. Fish consumption advisories are in place for the Tittabawassee River. In addition, MDCH is working to complete a health consultation involving eggs from chickens raised on the Tittabawassee floodplain.

**Comment:** Dioxin at its worst is a very weak carcinogen and in all probability is not carcinogenic at all in humans (paraphrased quote from Dr. Vernon Houk circa 1990, the Assistant US Surgeon General and Director of the Center for Environmental Health and Injury Control, CDC).

**Response:** Dr. Houk’s statements were made prior to the EPA’s reassessment of the health effects of dioxin. Contrary to his opinions at the time, the EPA and the International Agency for Research in Cancer have since concluded that 2,3,7,8-TCDD is a human carcinogen. The EPA has concluded that the mixture of dioxin and furans typically found in the environment is a likely human carcinogen.

**Comment:** The report addresses only cancer and reproductive/teratogenic effects – what about other potential health effects? (e.g. endometriosis, diabetes, immune system, neurological and respiratory effects, endocrine disruption, etc.)

**Response:** Several other adverse health effects associated with dioxin exposure in humans or animals were addressed in the consultation including: chloracne, liver damage, alterations in glucose metabolism, changes in hormone levels, disruption of the endocrine system, weakening of the immune system, and birth defects. Please see the Toxicological Evaluation section on pages 6 and 7. We have insufficient data to evaluate the likelihood of specific toxicological effects at this site. The discussion in the text is provided for general information. It is not all inclusive. Other efforts, as suggested by the comment, have been reported in the literature.

**Comment:** Wildlife data must be considered in the health consultation, as often this data provides important information about extent of the contamination, historical trends, and potential health effects in humans. It is possible to conduct studies on wildlife that cannot be done on humans. Further, wildlife data include studies of subtle functional losses, immune system problems, etc. that are relevant to the human population, and that come from organisms living in the same environment and eating the same fish as humans in the watershed. The health consultation should include a thorough review of the literature for these purposes.

**Response:** Wildlife studies are currently being designed and, in some cases, are already underway. An aquatic eco-assessment was completed in September 2003 by an independent contractor for the MDEQ and a study report is pending. Additional studies may be conducted by the MDEQ, or by Dow with oversight by the MDEQ. The MDCH will report on these findings as appropriate in future public health assessment documents.
APPENDIX 3 - MRLs AND EMEGs FOR TCDD

CURRENT MRLs

ATSDR published the *Toxicological Profile for TCDD* (ATSDR, 1989). Minimal risk levels (MRLs) listed in the profile were for acute, intermediate-duration, and chronic oral exposures (see Table 3-1).

*Acute Oral MRL*

The acute oral MRL of 100 pg/kg/day was based on hepatotoxic effects in guinea pigs that were observed following administration of a single gavage dose of 0.1 µg/kg TCDD (Turner and Collins, 1983).

An uncertainty factor of 10 was used for extrapolation from animals to humans, a factor of 10 for human variability, and a factor of 10 for the use of a lowest-observed-adverse-effect level (LOAEL).

*Intermediate Oral MRL*

The LOAEL of 0.001 µg/kg/day was considered for derivation of the intermediate-duration oral MRL of 1 pg/kg/day. At this exposure level, dilated pelvises and changes in gestational index were observed in rats (Murray et al., 1979) and abortions were reported in monkeys (Allen et al., 1979).

An uncertainty factor of 10 was used for extrapolation from animals to humans, a factor of 10 for human variability, and a factor of 10 for the use of a LOAEL.

*Chronic Oral MRL*

The intermediate-duration oral MRL of 1 pg/kg/day was also adopted as the chronic oral MRL.

PROPOSED MRLs

The *Toxicological Profile for CDDs* was in a draft stage in 1993/1994. The internal MRL workgroup proposed oral MRLs for TCDD (see Table 3-1).

*Acute Oral MRL*

The acute oral MRL of 20 pg/kg/day was based on the LOAEL of 0.01 µg/kg/day TCDD that induced suppressed serum complement activity in B6C3F1 mice exposed to 14 daily doses administered by gavage-in-oil vehicle (White et al., 1986).

An uncertainty factor of 10 was used for extrapolation from animals to humans, a factor of 10 for human variability, and a factor of 10 for the use of a LOAEL. Furthermore, a modifying factor of 0.5 was applied to adjust for the difference in higher bioavailability of TCDD from gavage-in-oil vehicle than from food or soil.
Intermediate Oral MRL
The intermediate-duration oral MRL of 7 pg/kg/day was based on a no-observed-adverse-effect level (NOAEL) of 0.0007 µg/kg/day TCDD for decreased thymus weight in guinea pigs exposed for 90 days in their feed (DeCaprio et al., 1986). The LOAEL in the study was 0.005 µg/kg/day.

An uncertainty factor of 10 was used for interspecies extrapolation and a factor of 10 for human variability. The NOAEL for deriving an intermediate-duration exposure MRL is also supported by the same level NOAEL for liver effects in the DeCaprio et al. study. The liver effects reported at higher levels consisted of hepatocellular inclusions and hypertriglyceridemia.

Chronic Oral MRL
A chronic oral MRL of 0.7 pg/kg/day was based on a LOAEL of 0.0002 µg/kg/day TCDD in the feed of monkeys that resulted in mild learning and behavioral impairment in their offspring (Bowman et al., 1989).

An uncertainty factor of 3 was used for the use of a minimal LOAEL, a factor of 10 was used for interspecies extrapolation, and a factor of 10 for human variability.

Environmental media evaluation guides (EMEGs) are media-specific comparison values that are used to select contaminants of concern at hazardous waste sites.

EMEGs are derived for air, water, and soil environmental media. They are based on inhalation and oral MRLs for air and water/soil exposures, respectively. The methodology and formula for derivation of EMEGs are described in ATSDR's Public Health Assessment Guidance Manual (ATSDR, 1992).

EMEGs are estimates of external dose. They do not provide data on how much of the dose is actually absorbed. No EMEGs are available for the dermal exposure route.

EMEGs based on these MRLs are presented in Tables 3-2a and 3-2b.
# TABLE 3-1. MRLs' for TCDD

<table>
<thead>
<tr>
<th>Year</th>
<th>Exposure duration</th>
<th>MRL' in pg/kg/day</th>
<th>UF LOAEL/NOAEL</th>
<th>UF interspecies</th>
<th>UF sensitivity</th>
<th>MF''</th>
<th>End point</th>
<th>Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>1989</td>
<td>acute</td>
<td>100</td>
<td>10</td>
<td>10</td>
<td>10</td>
<td></td>
<td>LOAEL for hepatotoxicity guinea pigs</td>
<td>Turner and Collins, 1983</td>
</tr>
<tr>
<td></td>
<td>1989 intermediate</td>
<td>1</td>
<td>10</td>
<td>10</td>
<td>10</td>
<td></td>
<td>LOAEL for abortions and other reproductive, developmental effects rats, monkeys</td>
<td>Murray et al., 1979 Allen et al., 1979</td>
</tr>
<tr>
<td></td>
<td>1989 chronic</td>
<td>1</td>
<td>10</td>
<td>10</td>
<td>10</td>
<td></td>
<td>LOAEL for abortions and other reproductive, developmental effects rats, monkeys</td>
<td>Murray et al., 1979 Allen et al., 1979</td>
</tr>
<tr>
<td></td>
<td>1994 acute</td>
<td>20</td>
<td>10</td>
<td>10</td>
<td>10</td>
<td>0.5</td>
<td>LOAEL for suppressed serum complement activity mice</td>
<td>White et al., 1986</td>
</tr>
<tr>
<td></td>
<td>1994 intermediate</td>
<td>7</td>
<td>10</td>
<td>10</td>
<td></td>
<td></td>
<td>NOAEL for decreased thymus weight; liver toxicity guinea pigs</td>
<td>DeCaprio et al., 1986</td>
</tr>
<tr>
<td></td>
<td>1994 chronic</td>
<td>0.7</td>
<td>3</td>
<td>10</td>
<td>10</td>
<td></td>
<td>LOAEL for mild learning and behavioral impairment monkey offspring</td>
<td>Bowman et al., 1989</td>
</tr>
</tbody>
</table>

*The MRL is calculated as MRL = (NOAEL or LOAEL)/(UF x MF), where MRL = minimal risk level (mg/kg/day), NOAEL = no-observed-adverse-effect level (mg/kg/day), LOAEL = lowest-observed-adverse-effect level (mg/kg/day), UF = uncertainty factor (unitless), MF = modifying factor (unitless) **MF for bioavailability was used in the derivation of an acute MRL (1994)

# TABLE 3-2a. EMEGs (in ppb) Based on 1989 TCDD MRLs

<table>
<thead>
<tr>
<th>Exposure duration</th>
<th>Child</th>
<th>Adult</th>
</tr>
</thead>
<tbody>
<tr>
<td>acute</td>
<td>5</td>
<td>70</td>
</tr>
<tr>
<td>intermediate</td>
<td>0.05</td>
<td>0.7</td>
</tr>
<tr>
<td>chronic</td>
<td>0.05</td>
<td>0.7</td>
</tr>
</tbody>
</table>

# TABLE 3-2b. EMEGs (in ppb) Based on 1994 TCDD MRLs

<table>
<thead>
<tr>
<th>Exposure duration</th>
<th>Child</th>
<th>Adult</th>
</tr>
</thead>
<tbody>
<tr>
<td>acute</td>
<td>1</td>
<td>14</td>
</tr>
<tr>
<td>intermediate</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>chronic</td>
<td>0.04</td>
<td>0.5</td>
</tr>
</tbody>
</table>

*The EMEG is calculated as EMEG = (MRL x BW)/IR. where EMEG = environmental media evaluation guide (mg/kg), BW = body weight in kg (adult = 70 kg; child = 10 kg), IR = soil ingestion rate (mg/day) (adult = 100 mg/day; child = 200 mg/day)
APPENDIX 4 - RECENT HEALTH EFFECTS STUDIES

Introduction
A significant number of toxicological studies have been conducted since the development of the 1 ppb action level for dioxin and dioxin-like compounds in residential soil. Many of these studies have examined human health effects after known or suspected exposure. In addition, in these intervening years, analytical techniques have been perfected to permit determination of very low levels of dioxin and dioxin-like compounds in environmental and biologic media. Significant advances have also been made in assessing possible health effects associated with exposure. This appendix is a synopsis of this more recent information.

Mechanism of Action
Recent studies have indicated that dioxin and dioxin-like compounds act through the same mechanism of action mediated by the Ah receptor, and that responses to their toxicity have been shown to be similar in several species (Birnbaum, 1994; DeVito et al., 1995).

Human Studies
Direct exposure information is generally not available in human studies, and so body burden is used as a surrogate. In this approach, the exposure is estimated from measured body burden, the elimination rate for humans, and the time since the exposure incident. Positive correlations have been observed between dioxin exposure and cancer (Fingerhut et al., 1991; Zober et al., 1990; Manz et al., 1991). More recent studies on cohorts investigated previously confirmed the association between dioxin exposure and higher cancer mortality (Flesch-Janys et al., 1995; Becher et al., 1996; Ott and Zober, 1996). The correlation was dose-dependent and increased with the latency period. IARC (1997) classified TCDD as a Group 1 carcinogen (carcinogenic to humans).

For health end points other than cancer, epidemiologic studies suggest a positive correlation between exposure to TCDD and development of chloracne (Mocarelli et al., 1986; Pazderova-Vejlupkova et al., 1981; Reggiani, 1980; Zober et al., 1990), dermal hyperpigmentation and hirsutism (Poland et al., 1971; Jirasek et al., 1974), elevated hepatic enzyme levels, mainly γ-glutamyl transferase (Mocarelli et al., 1986; May, 1982), and increased risk of diabetes (Sweeney et al., 1992; Table 4-1).

Other studies showed an association between development of subtle health effects (e.g., lower vitamin K levels, mild changes in liver enzymes, decreased neurologic optimality, and subtle changes in hormonal levels) in infants and their exposure to dioxin and dioxin-like chemicals from maternal milk (Pluim et al., 1992, 1994a, 1994b; Huisman et al., 1995; Koopman-Esseboom et al., 1994; Table 4-2). It is important to note that in reviewing the issues surrounding breastfeeding, the World Health Organization has concluded that the risks to infants do not outweigh the positive biologic and psychologic aspects of breastfeeding (Johnson, 1992a).
It has been suggested that dioxin and dioxin-like chemicals have the ability to disrupt endocrine function at low levels of exposure. A recent study of the cohort of people exposed during the Seveso accident indicated an alteration of the human sex ratio in their offspring (Mocarelli et al., 1996). In the 7-year period following the exposure, 26 males versus 48 females were born, but the study was limited by not providing information on sex-related spontaneous abortions in the cohort. A study of occupationally exposed workers reported altered reproductive hormone levels (Egeland et al., 1992). Other studies indicate low-exposure contamination of maternal milk with dioxin and dioxin-like compounds may have an impact on the hypothalamic-pituitary-thyroid regulatory system in newborns (Plium et al., 1992; Koopman-Esseboom et al., 1994).

Animal Studies
Studies in animals demonstrated a wide range of effects associated with CDDs exposure including mortality, cancer, wasting, and hepatic, immunologic, neurologic, reproductive, and developmental effects (ATSDR, 1989). In support of the findings that showed endocrine system disruption in humans, studies in animals reported that TCDD affects the adrenal (DiBartolomeis et al., 1987; Gorski et al., 1988a, 1988b) and thyroid glands (Hermansky et al., 1988; Hong et al., 1987; Lu et al., 1986; Henry and Gasiewicz, 1987; Rozman et al., 1983) and also alters estradiol (Umbret et al., 1987), testosterone, and dihydrotestosterone levels (Mebus et al., 1987; Moore et al., 1985). TCDD decreased responsiveness of the ventral prostate to testosterone in male offspring of exposed female rats and inhibited sexual differentiation in the central nervous system without altering sexual dimorphism in estrogen-receptor concentrations (Bjerke et al., 1994; Bjerke and Peterson, 1994). In animal studies, effects have been seen with the lowest doses evaluated, with the most sensitive end point being neurobehavioral changes in the offspring of dioxin-exposed monkeys (Schantz et al., 1992). A summary of critical study results and observed effect levels is presented in Table 4-3.

Body Burdens and Associated Health Effects
Health effects reported from human studies and associated body burdens of TCDD are listed in Table 4-1; these body burdens range from concentrations of 18 to 2,357 ng/kg. As can be seen from a comparison of animal and human studies shown in Table 4-3, body burden concentrations calculated for effect dosage rates in animal studies are in the same range as body burden concentrations associated with health effects in human studies. These results underscore the need for research to elucidate the toxicity of dioxin at low doses to human populations (CCEHRP, 1992) and to evaluate exposures in at-risk populations in view of total body burdens of dioxin and dioxin-like compounds.

Based on this review of more recent data, ATSDR has determined that its MRL of 1 pg/kg/day for TCDD is approximately two orders of magnitude below the health effect levels observed in recent studies. This is also true of cancer effect levels (Kociba et al., 1978). Independently, the Health Council of the Netherlands (1996) reassessed the risk associated with dioxin and dioxin-like compounds based on recent studies and recommended a health-based exposure limit equal to 1 pg/kg/day total TEQs.
ATSDR concludes that the chronic oral MRL of 1 pg/kg/day TCDD is protective of public health based on the fact that the MRL is approximately two orders of magnitude below the effect levels demonstrated experimentally and in epidemiologic studies.

**TABLE 4-1. Health Effects Associated with Exposure to TCDD and Body Burdens in Humans**

<table>
<thead>
<tr>
<th>Duration of exposure</th>
<th>System</th>
<th>Effect</th>
<th>Body burdens ng/kg body weight</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 1 year</td>
<td>Dermal</td>
<td>Chloracne in children</td>
<td>2357&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Mocarelli et al., 1991</td>
</tr>
<tr>
<td>&lt; 1 year</td>
<td>Reproductive</td>
<td>No increased risk of spontaneous abortion</td>
<td>&gt; 24&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Wolfe et al., 1995</td>
</tr>
<tr>
<td>≥ 15 years</td>
<td>Gastrointestinal</td>
<td>No increased risk of clinical gastrointestinal disease</td>
<td>418&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Calvert et al., 1992</td>
</tr>
<tr>
<td>≥ 15 years</td>
<td>Hepatic</td>
<td>No increased risk of clinical hepatic disease</td>
<td>418&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Calvert et al., 1992</td>
</tr>
<tr>
<td>Not specified</td>
<td>Dermal</td>
<td>Chloracne in 577 subjects</td>
<td>80.5&lt;sup&gt;d&lt;/sup&gt; 18&lt;sup&gt;e&lt;/sup&gt;</td>
<td>Schecter et al., 1993</td>
</tr>
<tr>
<td>11 years</td>
<td>Dermal</td>
<td>Chloracne</td>
<td>646&lt;sup&gt;f&lt;/sup&gt;</td>
<td>Jansing and Korff, 1994</td>
</tr>
<tr>
<td>6.5 years</td>
<td>Immunologic</td>
<td>Immunosuppression</td>
<td>156–176&lt;sup&gt;g&lt;/sup&gt;</td>
<td>Tonn et al., 1996</td>
</tr>
<tr>
<td>≥ 15 years</td>
<td>Neurologic</td>
<td>No increased risk for peripheral neuropathy</td>
<td>418&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Sweeney et al., 1993</td>
</tr>
<tr>
<td>≥ 15 years</td>
<td>Reproductive</td>
<td>Increased prevalence of high luteinizing hormone and low testosterone levels</td>
<td>31&lt;sup&gt;h&lt;/sup&gt;</td>
<td>Egeland et al., 1994</td>
</tr>
<tr>
<td>Not specified</td>
<td>Genotoxicity</td>
<td>No chromosome aberrations or sister chromatid exchanges</td>
<td>63-833&lt;sup&gt;i&lt;/sup&gt;</td>
<td>Zober et al., 1993</td>
</tr>
<tr>
<td>≥ 1 year</td>
<td>Cancer</td>
<td>Increased cancer mortality risk</td>
<td>124–459&lt;sup&gt;j&lt;/sup&gt;</td>
<td>Fingerhut et al., 1991</td>
</tr>
<tr>
<td>≥ 20 years</td>
<td>Cancer</td>
<td>Increased cancer mortality rate</td>
<td>69–461&lt;sup&gt;k&lt;/sup&gt;</td>
<td>Manz et al., 1991</td>
</tr>
</tbody>
</table>
TABLE 4-1. Health Effects Associated with Exposure to TCDD and Body Burdens in Humans (cont'd)

<table>
<thead>
<tr>
<th>Description</th>
<th>TCDD Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calculated using serum TCDD levels measured shortly after exposure. Body burdens were calculated using body weights of 13 kg for 1-3 year olds, 20 kg for 4-6 year olds, 28 kg for 7-10 year olds, 45 kg for 11-year-old males, and 55 kg for 16-year-old females and body fat percentages of 15% for 0-10 year olds, 15% for 11-year-old males, and 20% for 16-year-old females (ICRP, 1981).</td>
<td>&gt; 110 ppt</td>
</tr>
<tr>
<td>Calculated using the reported mean half-life adjusted serum TCDD level of &gt; 110 ppt and assuming the average worker weighed 70 kg with 22% body fat (DeVito et al., 1995). The authors calculated the half-life adjusted serum TCDD level using a half-life of 7.1 years.</td>
<td></td>
</tr>
<tr>
<td>Calculated using the reported mean half-life adjusted serum TCDD level of 1900 pg/g lipid and assuming the average worker weighed 70 kg with 22% body fat (DeVito et al., 1995).</td>
<td></td>
</tr>
<tr>
<td>Calculated by averaging the reported individual body burdens divided by the reference body weight of 75 kg for males and 65 kg for females. The authors calculated half-life adjusted serum TCDD levels using the assumption of 75 kg and 65 kg body weights for male and female workers, respectively, and a half-life of 5 years.</td>
<td></td>
</tr>
<tr>
<td>Same as footnoted, but using a half-life of 10 years.</td>
<td></td>
</tr>
<tr>
<td>Calculated using the reported mean half-life adjusted serum TCDD level of 2935 pg/g blood fat and assuming the average worker weighed 70 kg with 22% body fat (DeVito et al., 1995). The authors calculated the half-life adjusted serum TCDD level using a half-life of 7 years.</td>
<td></td>
</tr>
<tr>
<td>Calculated using the reported mean current serum TCDD level of 329.5 pg/g blood lipid. Half-life adjusted serum TCDD level was calculated using a half-life of 11.6 years (Wolfe et al., 1994), background TCDD concentration of 5 ng/kg lipid, and 13-15 years elapsed time. Body burdens were calculated assuming the average worker weighed 70 kg with 22% body fat (DeVito et al., 1995).</td>
<td></td>
</tr>
<tr>
<td>Calculated using the reported mean current serum TCDD level of 15 ppt. Half-life adjusted serum TCDD levels were calculated using a half-life of 11.6 years (Wolfe et al., 1994), background TCDD concentration of 5 ng/kg lipid, and 34 years of elapsed time. Body burdens were calculated assuming the average worker weighed 70 kg with 22% body fat (DeVito et al., 1995).</td>
<td></td>
</tr>
<tr>
<td>Calculated using the reported mean of current serum TCDD levels of 340-472 ppt (based on lipid content of blood). Half-life adjusted serum TCDD levels were calculated using a half-life of 11.6 years (Wolfe et al., 1994), background TCDD concentration of 5 ng/kg lipid, and 35 years of elapsed time. Body burdens were calculated assuming the average worker weighed 70 kg with 22% body fat (DeVito et al., 1995).</td>
<td></td>
</tr>
<tr>
<td>Calculated using the reported mean current serum TCDD level of 233 pg/g lipid. Half-life adjusted serum TCDD levels were calculated using a half-life of 11.6 years (Wolfe et al., 1994), background TCDD concentration of 5 ng/kg lipid, and 35 years of elapsed time. Body burdens were calculated assuming the average worker weighed 70 kg with 22% body fat (DeVito et al., 1995).</td>
<td></td>
</tr>
<tr>
<td>Calculated using the reported mean current adipose tissue TCDD level of 296 ng/kg. Half-life adjusted adipose TCDD levels were calculated using a half-life of 11.6 years (Wolfe et al., 1994), background TCDD concentration of 5 ng/kg lipid, and 1-33 years of elapsed time.</td>
<td></td>
</tr>
<tr>
<td>Number of Children</td>
<td>Breast milk levels (mean levels in pg of TEQs per g of milk fat)</td>
</tr>
<tr>
<td>--------------------</td>
<td>---------------------------------------------------------------</td>
</tr>
<tr>
<td>17</td>
<td>(29.85–92.88)</td>
</tr>
<tr>
<td>32</td>
<td>29.4 (13.7–62.6)</td>
</tr>
<tr>
<td>78</td>
<td>&gt; 30.75</td>
</tr>
<tr>
<td>104</td>
<td>30.19</td>
</tr>
<tr>
<td>48</td>
<td>not specified</td>
</tr>
<tr>
<td>35</td>
<td>28.1 (8.7–62.7)</td>
</tr>
<tr>
<td>19</td>
<td>37.5 (29.2–62.7) high exposure group</td>
</tr>
<tr>
<td>19</td>
<td>18.6 (8.7–28.0) low exposure group</td>
</tr>
</tbody>
</table>

AST = aspartate aminotransferase; ALT = alanine aminotransferase; TEQs = toxicity equivalents; TSH = thyroid-stimulating hormone
<table>
<thead>
<tr>
<th>Duration of exposure</th>
<th>System</th>
<th>Effect</th>
<th>Body burdens ng/kg body weight</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Studies in humans</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 1 year</td>
<td>Dermal</td>
<td>Chloracine in children</td>
<td>2357⁷</td>
<td>Mocarelli et al., 1991</td>
</tr>
<tr>
<td>Not specified</td>
<td>Dermal</td>
<td>Chloracine in 5/7 subjects</td>
<td>80.5⁸ 18⁹</td>
<td>Schecter et al., 1993</td>
</tr>
<tr>
<td>11 years</td>
<td>Dermal</td>
<td>Chloracine</td>
<td>646⁵</td>
<td>Jansing and Korff, 1994</td>
</tr>
<tr>
<td>6.5 years</td>
<td>Immunologic</td>
<td>Immunosuppression</td>
<td>156-176⁴</td>
<td>Tonn et al., 1996</td>
</tr>
<tr>
<td>≥ 15 years</td>
<td>Reproductive</td>
<td>Increased prevalence of high luteinizing hormone and low testosterone levels</td>
<td>31¹</td>
<td>Egeland et al., 1994</td>
</tr>
<tr>
<td>≥ 1 year</td>
<td>Cancer</td>
<td>Increased cancer mortality risk</td>
<td>124-459⁴</td>
<td>Fingerhut et al., 1991</td>
</tr>
<tr>
<td>&gt; 20 years</td>
<td>Cancer</td>
<td>Increased cancer mortality rate</td>
<td>69-461⁸</td>
<td>Manz et al., 1991</td>
</tr>
<tr>
<td>Studies in animals</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>14 days</td>
<td>Immunologic</td>
<td>Suppressed serum complement in mice</td>
<td>74⁴</td>
<td>'White et al., 1986</td>
</tr>
<tr>
<td>90 days</td>
<td>Reproductive</td>
<td>Decreased litter size in rats</td>
<td>26⁶</td>
<td>'Murray et al., 1979</td>
</tr>
<tr>
<td>90 days</td>
<td>Immunologic</td>
<td>Decreased thymus weight in guinea pigs</td>
<td>164⁴</td>
<td>'DeCaprio et al., 1986</td>
</tr>
<tr>
<td>16 months</td>
<td>Developmental</td>
<td>Behavioral alterations in offspring in monkeys</td>
<td>32¹</td>
<td>Schantz et al., 1992</td>
</tr>
<tr>
<td>2 years</td>
<td>Cancer</td>
<td>Liver, lung carcinoma in rats</td>
<td>2976⁶⁷</td>
<td>Kociba et al., 1978</td>
</tr>
<tr>
<td>2 years</td>
<td>Cancer</td>
<td>Liver carcinoma in mice</td>
<td>944⁸</td>
<td>NTP, 1972</td>
</tr>
</tbody>
</table>
TABLE 4-3. Human Body Burdens and Animal Body Burdens Associated with Health Effects (cont’d)

*Studies which serve as the basis for ATSDR’s health guidance values  
*Calculated using serum TCDD levels measured shortly after exposure. Body burdens were calculated using body weights of 13 kg for 1-3 year olds, 20 kg for 4-6 year olds, 28 kg for 7-10 year olds, 45 kg for 11-year-old males, and 55 kg for 16-year-old females and body fat percentages of 15% for 0-10 year olds, 15% for 11-year-old males, and 20% for 16-year-old females (ICRP, 1981).  
*Calculated by averaging the reported individual body burdens divided by the reference body weight of 75 kg for males and 65 kg for females. The authors calculated half-life adjusted serum TCDD levels using the assumption of 75 kg and 65 kg body weights for male and female workers, respectively, and a half-life of 5 years.  
*Same as footnote d but using a half-life of 10 years.  
*Calculated using the reported mean half-life adjusted serum TCDD level of 2935 pg/g blood fat and assuming the average worker weighed 70 kg with 22% body fat (DeVito et al., 1995). The authors calculated the half-life adjusted serum TCDD level using a half-life of 7 years.  
*Calculated using the reported mean current serum TCDD level of 329.5 pg/g blood lipid. Half-life adjusted serum TCDD level was calculated using a half-life of 11.6 years (Wolfe et al., 1994), background TCDD concentration of 5 ng/kg lipid, and 13-15 years elapsed time. Body burdens were calculated assuming the average worker weighed 70 kg with 22% body fat (DeVito et al., 1995).  
*Calculated using the reported half-life adjusted serum TCDD level of >140 pg/g blood lipid and assuming the average worker weighed 70 kg with 22% body fat (DeVito et al., 1995). The authors calculated the adjusted serum dioxin level using a dioxin half-life of 7.1 years and background dioxin level of 6.08 pg/g blood lipid.  
*Calculated using the reported mean current serum TCDD level of 233 pg/g lipid. Half-life adjusted serum TCDD levels were calculated using a half-life of 11.6 years (Wolfe et al., 1994), background TCDD concentration of 5 ng/kg lipid, and 35 years of elapsed time. Body burdens were calculated assuming the average worker weighed 70 kg with 22% body fat (DeVito et al., 1995).  
*Calculated using the reported mean current adipose tissue TCDD level of 296 ng/kg. Half-life adjusted adipose TCDD levels were calculated using a half-life of 11.6 years (Wolfe et al., 1994), background TCDD concentration of 5 ng/kg lipid, and 1-33 years of elapsed time. Body burdens were calculated assuming the average worker weighed 70 kg with 22% body fat (DeVito et al., 1995).  
*Acute exposure study in mice (White et al., 1986). Assumed parameter values a: 0.8 (Curtis et al., 1990). t_{1/2} = 11 days (Birmbaum, 1986).  
*Intermediate-duration exposure study in rats (Murray et al., 1979). Assumed parameter values a: 0.8 (Curtis et al., 1990). t_{1/2} = 24 days (Van den Berg et al., 1994).  
*Assumed parameter values for guinea pigs in Decaprio et al. (1981) study: a = 0.5 (Van den Berg et al., 1994). t_{1/2} = 94 days (Olson, 1986).  
*The lowest effect level in the current database for chronic-duration exposure. Assumed parameter values for monkeys in Schantz et al. (1992) study: a = 0.8 (value for rats from Van den Berg et al., 1994), t_{1/2} = 391 days (Bowman et al., 1989).  
*mA cancer study in rats. Body burdens calculated in DeVito et al., 1995.  
*nA cancer study in mice. Body burdens calculated in DeVito et al., 1995.
APPENDIX 5 - CHRONOLOGY FOR DIOXIN AND DIOXIN-LIKE COMPOUNDS: HEALTH GUIDANCE VALUES AND POLICY STATEMENTS

1984

R. Kimbrough, H. Falk, and P. Stehr (1984) recommended 1 ppb of TCDD in soil as a level of concern for human health. They also concluded that “One ppb of 2,3,7,8-TCDD in soil is a reasonable level at which to begin consideration of action to limit human exposure for contaminated soil” (emphasis added) (p. 47). However, the authors cautioned not to use this level for every site, but rather to estimate the risk associated with each site according to specific circumstances at the site.

The estimated risk dose was 1.4 pg/kg/day TCDD (a 95% upper bound for a one-in-a-million risk estimate for cancer). The calculations were based on cancer studies in laboratory animals.

1985

EPA derived oral slope factor, \( q_1^* \), of 1.56x10\(^5\) (mg/kg/day\(^{-1}\)) for TCDD (EPA, 1985) that represents the mean 95% upper-limit carcinogenic potency factor for humans. Based on this factor, a risk-specific dose of 0.006 pg/kg/day TCDD was calculated.

1989

ATSDR published the Toxicological Profile for TCDD. The profile describes the use of toxicity equivalents (TEQs) for assessing exposure to dioxin and dioxin-like compounds. MRLs for TCDD listed in the profile for the acute, intermediate-duration, and chronic exposures were 100 pg/kg/day, 1 pg/kg/day, and 1 pg/kg/day, respectively. Developmental and reproductive endpoints were the bases for intermediate and chronic duration MRLs. Based on the chronic MRL, the EMEG of 50 ppt is typically used in public health assessments for dioxin contaminated soil.

1990

The Food and Drug Administration (1990) introduced a risk-specific dose of 0.057 pg/kg/day TCDD (a 95% upper bound for a one-in-a-million risk estimate for cancer). The number was based on a linear low-dose extrapolation from the Kociba et al. (1978) cancer study in rats. The value applied to consumption of contaminated food, specifically fish.

1992

The Public Health Service Committee to Coordinate Environmental Health and Related Programs (CCEHRP) recommended, in the Interim Statement on Dioxins, to adopt the FDA risk-specific dose (0.057 pg/kg/day) as the risk-specific level for TCDD equivalents (TEQs).

1992

In a memo to ATSDR senior management, B. Johnson stated that “The Interim Statement, while mentioning FDA’s tolerable daily intake of dioxin as 0.057 pg/kg/day, should not be understood to supplant ATSDR’s position of 1 ppb of dioxin in residential soil as a soil action level.” Consistent with the CCEHRP statement, ATSDR’s practice incorporates the TEQ approach.
The *Toxicological Profile for CDDs* was in a draft stage. The internal MRL workgroup met with representatives of other ATSDR divisions and proposed MRLs for TCDD for the acute, intermediate-duration, and chronic exposures as 20 pg/kg/day, 7 pg/kg/day, and 0.7 pg/kg/day, respectively. Developmental effects were the bases for derivation of the chronic MRL.

Pohl et al. (1995) published the “Public health assessment for dioxins exposure from soil” paper.

This paper reviewed more recent findings on the potential health effects of dioxin. Based upon this review, Pohl et al. presented a proposed chronic MRL for TCDD of 0.7 pg/kg/day and a corresponding EMEG of 40 ppt for children.

From a health risk assessment perspective, the EMEG of 40 ppt is not substantially different from the current EMEG of 50 ppt based on the 1 pg/kg/day MRL (ATSDR, 1989). The MRL of 1 pg/kg/day is approximately two orders of magnitude below effect levels demonstrated experimentally or in epidemiologic studies.
APPENDIX 6 - REFERENCES


JOHNSON, B.L. (1992a). Testimony before the Subcommittee on Human Resources and Interghovernmental Relations, Committee on Government Operations, Washington, DC.


U.S. ENVIRONMENTAL PROTECTION AGENCY (EPA) (1994). METHOD 8290: Polychlorinated Dibenzodioxins (PCDDs) and Polychlorinated Dibenzofurans (PCDFs) by High-Resolution Gas Chromatography/High-Resolution Mass Spectrometry (HRGC/HRMS).


Attachment E

Dioxin and Dioxin-Like Compounds in Soil,
DIOXIN AND DIOXIN-LIKE COMPOUNDS IN SOIL, PART II: TECHNICAL SUPPORT DOCUMENT FOR ATSDR INTERIM POLICY GUIDELINE

CHRISTOPHER T. DE ROSA, DAVID BROWN,* ROSALINE DHARA, WOODROW GARRETT, HUGH HANSEN, JAMES HOLLER, DENNIS JONES, DENISE JORDAN-IZAGUIRRE, RALPH O'CONNOR, HANA POHL, AND CHARLES XINTARAS

Agency for Toxic Substances and Disease Registry
U.S. Department of Health and Human Services
Atlanta, Georgia

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2. Abbreviations: ATSDR, Agency for Toxic Substances and Disease Registry; AUC, area under the curve; CDDs, chlorinated dibenzo-p-dioxins; BDDs, brominated dibenzo-p-dioxins; BDFs, brominated dibenzofurans; CCEHRP, The Public Health Service Committee to Coordinate Environmental Health and Related Programs; CDDs, chlorinated dibenzo-p-dioxins; CDFs, chlorinated dibenzofurans; EMEGs, environmental media evaluation guides; EPA, U.S. Environmental Protection Agency; FDA, U.S. Food and Drug Administration; LOAEL, lowest-observed-adverse-effect level; MRL, minimal risk level; NOAEL, no-observed-adverse-effect level; PBBS, polybrominated biphenyls; PCBs, polychlorinated biphenyls; TCDD, 2,3,7,8-tetrachlorodibenzo-p-dioxins; TEF, toxicity equivalent factor; TEQs, toxicity equivalents.

3. Key words: dioxin, human exposure, risk assessment, soil levels, TCDD, TEQs.

4. Note: 65 Bulkley Avenue North, Westport, CT 06880.


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INTRODUCTION

Dioxin remains at the forefront of public health concerns in the United States and throughout the world. Over the past 20 years, a wide range of federal agencies and other organizations have been involved in developing policy statements, strategies, and assessment methods to address the public health implications of dioxin exposure. These positions were developed in response to issues confronted by those organizations in pursuing their missions, often as a direct function of legislative mandates. Because of distinct differences in perspective, policy, and practice, dictated by the mandated activities of these organizations and the evolving understanding of dioxin toxicity, apparently divergent positions may be reflected in their conclusions.

In pursuing its mandated responsibilities, the Agency for Toxic Substances and Disease Registry (ATSDR) must address public health concerns associated with exposure to dioxin and dioxin-like compounds in the context of all available relevant information. This information includes both technical data and science policy positions adopted by ATSDR and others that are germane to the public health assessment of dioxin and dioxin-like compounds.

The issues outlined previously, coupled with requests from the public, other agencies, the private sector, and agency staff for a statement reflecting the agency’s position on science and science policy issues related to dioxin and dioxin-like compounds, prompted the development of this technical support document. This document is intended to serve as technical background and support for the agency interim policy guideline on dioxin and dioxin-like compounds in soil and to harmonize such efforts with those of other federal agencies and relevant organizations to the extent practicable. This document reflects an assessment of current practice within the agency and defines the appropriate roles of professional judgment and emerging scientific principles in ATSDR’s public health assessments of exposures to dioxin and dioxin-like compounds.

This document is not intended to supplant the Environmental Protection Agency’s (EPA) ongoing reassessment of dioxin and dioxin-like compounds or ATSDR’s toxicological profile on chlorinated dibenzo-p-dioxins (CDDs). But it will provide technical background support for ATSDR’s public health practice at sites contaminated by dioxin and dioxin-like compounds. A central theme of this document is the use of health guidance values in the broader context of biomedical and other scientific judgment to define exposures of concern rather than single numerical conclusions that may convey an artificial sense of precision (ATSDR, 1993; CEQ, 1989).
After reviewing the previously cited issues, ATSDR further considered three specific issues:

- **Issue 1**: The relationship between the ATSDR action level of 1 part per billion (ppb) dioxin and dioxin-like compounds in residential soil and ATSDR’s environmental media evaluation guides (EMEGs)

- **Issue 2**: That current analytic and sampling techniques employed for soil contaminated with dioxin and dioxin-like compounds may not be sufficiently sensitive

- **Issue 3**: That ATSDR’s action level of 1 ppb dioxin and dioxin-like compounds in residential soil is too high.

Each of these issues is addressed in subsequent sections of this paper. To facilitate its review of these issues ATSDR has

- developed a glossary of critical terms and concepts to facilitate a consistent use and understanding of terminology in this support document (Appendix 1)

- identified and evaluated key assumptions underlying the review and evaluation of the ATSDR action level of 1 ppb of dioxin and dioxin-like compounds in residential soil, the ATSDR minimal risk level (MRL), and the ATSDR EMEG (Appendix 2)

- reviewed and evaluated the documentation for the ATSDR action level of 1 ppb for dioxin and dioxin-like compounds in residential soils, the MRL of 1 picogram/kilogram/day (pg/kg/day) 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), and the EMEG of 50 parts per trillion (ppt) (Appendix 3)

- reviewed and evaluated ATSDR’s use of an action level of 1 ppb (HazDat) for dioxin and dioxin-like compounds, given recent insights into the toxicologic and human health effects of such compounds, particularly those associated with reproductive and developmental toxicities (Appendix 4).

**DISCUSSION**

**Issue I: Relationship between ATSDR’s action level and EMEGs**

*Comparison Values*

EMEGs are comparison values used by ATSDR health assessors to select contaminants for further evaluation based on concerns about endpoints other than cancer. As such, EMEGs represent a starting point for the health assessor to make an initial determination of whether or not a specific contamination level merits further evaluation as a potential health concern. EMEGs are based on ATSDR’s MRLs or analogous health guidance values that are thought to be without appreciable risk for a given route and duration of exposure.
Generally, if a concentration of a chemical at a site is less than the EMEG, ATSDR assumes there is little likelihood that the chemical presents a health hazard at the site via a particular environmental medium. In some instances, ATSDR may further consider contaminants present at levels below the EMEG, based on community health concerns. However, if the concentration of a chemical meets or exceeds the EMEG, this does not mean there is a chemical health hazard; instead, this means that the situation merits further evaluation of site-specific information (for example, bioavailability, demographics, on-site activities, climatic conditions, or soil cover). Follow-up evaluation of all available site-specific information may reveal that there is no health threat at the site even though the media concentrations may exceed the EMEG.

**Exposure Evaluation and Interdiction Strategies**

Levels greater than the EMEG of 50 ppt (0.05 ppb) TCDD in soil are used to determine whether further site-specific evaluation for dioxin is to occur. Because the toxicity of dioxin and dioxin-like compounds is assumed to be elaborated through a common receptor-mediated mechanism, the EMEG is expressed in total toxicity equivalents (total TEQs). An action level of 1 ppb (also expressed as total TEQs) is used to determine the need for public health actions on a site-specific basis and on the basis of the maximum concentration identified at the site.

For these reasons, ATSDR considers source-specific contributions to total exposure and associated body burdens of dioxin and dioxin-like compounds expressed as TEQs in evaluating sites. This requires insight into not only contamination levels in soil, but also into other media as well. In this way the contribution of each potential source of exposure is evaluated and viewed in the context of total exposure and associated body burdens for a given at-risk population.

ATSDR also evaluates exposure levels and potential body burdens in at-risk populations in the context of current knowledge regarding effect levels as identified in both experimental studies and epidemiologic investigations (DeVito et al., 1995; Appendix 4). A full range of strategies to interdict exposures and reduce overall body burden are then considered. These exposure interdiction strategies include restricted land use and access, health education, relocation, and remediation to reduce incremental contributions to body burdens and risks of potential health effects.

**Action Levels, EMEGs, and MRLs**

ATSDR’s health guidance values for dioxin or dioxin-like compounds (MRLs, EMEG, action level) each have their distinct application corresponding to screening, evaluation, or consideration of potential public health actions (Table 1). The use of such a hierarchy or framework of quantifiable conclusions for purposes of screening, evaluation, and consideration of action is not intended to serve as a surrogate for professional judgment. Parameters of exposure and toxicity that may serve to either increase or decrease health concerns for at-risk populations should be considered on a site-specific basis. ATSDR’s approach is consistent with recommendations of the National Research Council (NRC, 1994) that a tiered or iterative approach be used in health assessment efforts, beginning with relatively conservative screening techniques and subsequently relying on more rigorous data-intensive efforts as suggested by public health concerns.
Limitations, Assumptions, and Uncertainties

Health guidance values reflect the application of a range of default assumptions that are conservative (i.e., protective) and which are believed, in aggregate, to result in protective health guidance values. These assumptions include bioavailability of dioxin and dioxin-like compounds from test vehicles, soil ingestion rates for different at-risk populations (i.e., children, geophagic children, adults), and the use of animal data in the absence of adequate epidemiologic data addressing the health effects in human populations (Appendix 2). Additionally, to account for recognized areas of uncertainty regarding species variability in effect(s) and effect levels, sensitive human populations, and low-dose extrapolation, uncertainty factors are used in developing health guidance values. The application of such uncertainty factors contributes further to the protective nature of health guidance values.

The limitations, assumptions, and uncertainties inherent in health risk assessment are addressed in the National Academy of Sciences report “Science and Judgment in Risk Assessment” (NRC, 1994). In this report, the Academy states that “uncertainty analysis should be an iterative process, moving from the identification of generic uncertainties” to more refined analyses for chemical-specific or industrial plant-specific uncertainties. Implicit in this scenario are site-specific applications addressed in this support document. ATSDR’s practice in evaluating sites contaminated with dioxin and dioxin-like compounds is consistent with the position of the National Academy of Sciences (NRC, 1994) in terms of uncertainty analysis.

Issue 2: Analytic and sampling techniques

Analytic Techniques

The EPA 8280 method is currently unable to provide analytical data for levels between the screening level of 50 ppt and the action level of 1 ppb TEQs (EPA, 1995). The EPA 8290 method can provide analytical data in the range of 50 ppt to 1 ppb. The detection limit of Method 8290 has a range of 1–5 ppt. Thus, in those instances where the health assessor has determined that it is necessary to evaluate the site-specific public health implications of exposure to soil levels of dioxin and dioxin-like compounds between 50 ppt and 1 ppb, it may be appropriate to implement the EPA 8290 (EPA, 1994) soil analytic method with the more sensitive detection limit. This decision should be made on a site-specific basis.

Sampling Techniques

ATSDR’s position regarding soil sampling strategies is germane to the discussions in this document. ATSDR recommends that appropriate soil sampling methods be determined on a site-specific basis (Emmett and Jordan-Izaguirre, 1994).
### TABLE 1. ATSDR’s Decision Framework for Sites Contaminated with Dioxin and Dioxin-Like Compounds

Because the toxicity of dioxin and dioxin-like compounds is assumed to be elaborated through a common receptor-mediated mechanism, levels greater than 50 ppt (0.05 ppb) TEQs* are used to determine whether further site-specific evaluation for dioxins is to occur based on the maximum soil concentrations identified at the site. A level of 1 ppb TEQs is used to determine the potential need for public health actions on a site-specific basis and on the basis of adequate sampling and measured or projected human exposure—past, present, or future—as determined by the health assessor.

<table>
<thead>
<tr>
<th>SCREENING LEVEL</th>
<th>EVALUATION LEVELS</th>
<th>ACTION LEVEL&quot;</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤ 50 ppt (0.05 ppb) TEQs</td>
<td>&gt; 0.05 ppb but &lt; 1 ppb TEQs</td>
<td>≥ 1 ppb TEQs</td>
</tr>
</tbody>
</table>

- The EMEN for TCDD is 50 ppt
- This is based on an MRL of 1 pg/kg/day for TCDD (ATSDR, 1989).
- For screening purposes 50 ppt TCDD is assumed to be equivalent to 50 ppt TEQs

- Evaluation of site-specific factors, such as:
  - Bioavailability
  - Ingestion rates
  - Pathway analysis
  - Soil cover
  - Climate
  - Other contaminants
  - Community concerns
  - Demographics
  - Background Exposures

Potential public health actions considered, such as:
- Surveillance
- Research
- Health studies
- Community education
- Physician education
- Exposure investigations

---

*The toxicity equivalent (TEQ) of TCDD is calculated by multiplying the exposure level of a particular dioxin-like compound by its toxicity equivalency factor (TEF). TEFs are based on congener-specific data and the assumption that Ah receptor-mediated toxicity of dioxin-like chemicals is additive. The TEF scheme compares the relative toxicity of individual dioxin-like compounds to that of TCDD, which is the most toxic halogenated aromatic hydrocarbon.

**A concentration of chemicals at which consideration of action to interdict/prevent exposure occurs, such as surveillance, research, health studies, community education, physician education, or exposure investigations. Alternatively, based on the evaluation by the health assessor, none of these actions may be necessary.

### Issue 3: One part per billion of dioxin and dioxin-like compounds as an action level for cleanup

The decision to derive standard action levels for individual chemicals and to further use these values to drive clean-up activities is an EPA risk management decision. Risk management issues are outside the direct mandates of ATSDR.

### Historical Background

The 1 ppb level for dioxin has been described as a “reasonable level to begin consideration of action to limit exposure” (Kimbrough et al., 1984); “a level of concern” (Kimbrough et al., 1984; Pohl et al., 1995); and “a soil action level” (Johnson, 1992b). This action level of 1 ppb was originally used in reference to TCDD in soil (see Appendix 5 for a complete chronology regarding the use and application of these terms). More recently, it has been used in reference to TCDD toxicity equivalents or TEQs (CCEHRP, 1992). The TEQ approach is based on the assumption of a common receptor-mediated mechanism of toxic action for dioxin and dioxin-like compounds (Birnbaum, 1994; DeVito et al., 1995).
Limitations of Soil Action Level

A key limitation inherent in the use of any soil action level is the incomplete understanding of how such a soil action level would contribute to body burdens in at-risk populations. The extent of contribution of soil dioxin and dioxin-like compounds to body burdens of dioxin is a function of all media-specific levels of the contamination at a given site. Accordingly, a 1 ppb level of dioxin and dioxin-like compounds in residential soil could result in distinctly different contributions to overall body burdens in different populations. For this reason, ATSDR’s use of 1 ppb has always been coupled with the recommendation that full consideration be given to site-specific factors such as demographics, on-site activities, climatic conditions, and soil cover.

These site-specific factors provide health assessors with valuable insight into how closely the assumptions associated with health guidance values actually reflect real site conditions. Moreover, such insight and understanding are essential to the determination of whether a site-specific action level other than 1 ppb might be appropriate. As noted by Kimbrough et al. (1984), exposure assessments used to project risk contain assumptions that are unlikely to be actually encountered. These assumptions include uniform levels of contamination, uniform land use patterns, lifetime exposure, and no degradation of dioxin and dioxin-like compounds.

Carcinogenic Versus Other Health Outcomes

A significant point to be considered in regard to 1 ppb as an action level for dioxin and dioxin-like compounds in residential soil is the issue of carcinogenic versus other health outcomes. As discussed previously, 1 ppb dioxin in residential soil was identified by Kimbrough et al. (1984) as a “level of concern,” and was recommended as “a reasonable level to begin consideration of action to limit exposure.” It is important to note that Kimbrough et al.’s (1984) conclusions were derived in part from an evaluation of the carcinogenic potential of TCDD, based on a 2-year oral chronic toxicity and oncogenicity study in rats (Kociba et al., 1978).

The Kociba et al. (1978) study also served as the basis for the Food and Drug Administration’s (FDA’s) derivation of a risk-specific dose of 0.057 pg/kg/day dioxin for a 1 in a million (10^-6) upper-bound risk estimate for cancer (FDA, 1990). Using typical default values of 70 kilograms (kg) for average body weight, and 100 milligrams/day (mg/day) for soil consumption, FDA’s 0.057 pg/kg/day risk-specific dose corresponds to a soil concentration of 40 ppt, a value marginally lower than, but essentially equivalent to (from a risk assessment perspective), the ATSDR screening EMEG of 50 ppt (0.05 ppb). EPA’s 0.006 pg/kg/day risk-specific dose corresponds to a soil concentration of 4 ppt, a value about one order of magnitude below the FDA level. In contrast, Paustenbach et al. (1992) reexamined human exposure to dioxin and dioxin-like compounds from soil. In residential areas, soils containing 20 ppb of TCDD were calculated to pose a lifetime cancer risk no greater than 1 in 10^-3. Assumptions used for estimating exposure from soil differed from previous evaluations of soil ingestion, dermal contact, dust inhalation, fish consumption, and in the cancer slope factor for TCDD. Exposure through dermal contact was discussed.

As noted previously, ATSDR’s EMEG is based on the MRL of 1 pg/kg/day TCDD, which is approximately two orders of magnitude below any human effect levels demonstrated either experimentally or in epidemiologic studies for both cancer and noncancer health end points. The
conservative (i.e., protective) nature of both the MRL and the EMEG reflects adjustments made for recognized areas of uncertainty perhaps spanning two to three orders of magnitude (Appendix 2). As such, the EMEG and the MRL (on which the EMEG is based) are below levels of exposures associated with demonstrated health effects and are therefore considered protective of human health. A 1000-fold uncertainty factor was used in the derivation of the MRL, reflecting the range of currently recognized areas of scientific uncertainty. The EMEG of 50 ppt is at the low end of this range, which is approximately 50–50 000 ppt (0.05–50 ppb). The level calculated by Paustenbach of 20 000 ppt (20 ppb) is closer to the mid-point of the range of scientific uncertainty.

In the case of the FDA’s risk-specific dose, it should be noted that this dose is based on an upper-bound estimate of risk in the 95% confidence limit sense. This means that there is a 95% chance that actual risk is less (CCHEHRP, 1992) and could be as low as zero. This places the low end of ATSDR’s range of evaluation (> 0.05 ppb but < 1 ppb TEQs) approximately two orders of magnitude below health effect levels demonstrated experimentally or in epidemiologic studies.

CONCLUSIONS

Protection of Public Health
The issues discussed previously indicate that (1) ATSDR’s EMEG and MRL are approximately two orders of magnitude below effect levels in experimental and epidemiologic studies, (2) cancer risk-specific doses and screening values for end points other than cancer are essentially equivalent from a risk assessment perspective, (3) ATSDR’s EMEG of 50 ppt (0.05 ppb) and action level of 1 ppb are not inconsistent, and (4) a 1 ppb action level for dioxin and dioxin-like compounds in residential soil, when coupled to a site-specific context of evaluation for the range of greater than 50 ppt to less than 1 ppb (TEQs) in residential soil, is protective of public health. Similarly, a cleanup level of 1 ppb (TEQs) for dioxin and dioxin-like compounds in residential soil is considered to be generally protective of human health if coupled with a full evaluation of site specific factors.

Site-Specific Parameters
A range of site-specific parameters, e.g., soil type, soil cover, media-specific contamination levels, and demographics, affect body burdens of dioxin and dioxin-like compounds in at-risk populations. Because these parameters vary on a site-specific basis, it is not currently feasible to identify, for all sites, a single numerical value to appropriately guide cleanup or other public health actions. For this reason, ATSDR uses a hierarchy of health guidance values (Table 1) for purposes of screening, evaluation, and consideration of the potential need for further action to interdict exposures, extending to and possibly including cleanup. Alternative actions may include, but are not limited to, health education, restricted access, deed restrictions, and relocation.

Evaluation of Recent Literature
Based on ATSDR’s evaluation of more recent literature (Appendix 4), ATSDR has determined that the agency’s MRL of 1 pg/kg/day (ATSDR, 1989) is approximately two orders of magnitude below effect levels in experimental and epidemiologic studies. Accordingly, ATSDR concludes that this MRL and the EMEG of 50 ppt, which is based on the MRL, continue to be reasonable and protective, although geophagic children and those with elevated body burdens of dioxin and
dioxin-like compounds may represent special at-risk populations. Such an approach is consistent with the current public health conclusions and practices reflected in a recent publication by the Health Council of the Netherlands (1996), in which a health-based exposure limit of 1 pg/kg/day dioxin and dioxin-like compounds was also recommended based on the council's own independent reassessment of dioxin.

With specific reference to the issues outlined in this paper, ATSDR further concludes the following:

- ATSDR's action level of 1 ppb of dioxin and dioxin-like compounds (TEQs) in residential soil is consistent with ATSDR's EMEG. These values are used for distinctly different purposes in the evaluation of dioxin-contaminated sites (Table 1).

- Currently used soil analytic methods may not be sufficiently sensitive. Determination of an appropriate analytic method should be made on a site-specific basis. Specific knowledge of different dioxin-like compounds at a given site is required to evaluate the adequacy of a soil sampling protocol.

- ATSDR's action level of 1 ppb for dioxin and dioxin-like compounds (TEQs) in residential soil is not too high. Whether to use the 1 ppb action level should be decided on a site-specific basis in which residential soil levels greater than 50 ppt and less than 1 ppb are further evaluated in the context of site-specific parameters.

Health Guidance Values
While health guidance values represent an important frame of reference in public health assessment, they are not surrogates for biomedical and other technical judgments in public health assessments. For this reason, health guidance values, including those used for screening, evaluation, and consideration of action, are used by ATSDR in the context of all relevant site-specific parameters. In this site-specific context of evaluation for levels of dioxins in soil greater than 50 ppt and less than 1 ppb, ATSDR concludes that the 1 ppb level in residential soil continues to represent a level at which consideration of health action to limit exposure should occur. ATSDR considers this action level to be both reasonable and protective.

The identification of a threshold body burden/blood serum level, below which adverse health effects are not anticipated, would help to better define potential health risks at sites contaminated with dioxin and dioxin-like compounds. However, since significant uncertainties remain regarding such levels, especially for at-risk populations by virtue of exposure or physiologic sensitivity, a threshold level cannot be identified at present.

RECOMMENDATIONS

Evaluation of Hazardous Waste Sites
ATSDR's approach to the evaluation of hazardous waste sites, including those contaminated with dioxin and dioxin-like compounds, places preeminent emphasis on biomedical and other technical judgment. In the exercise of such a judgment, health guidance values serve as a frame
of reference to guide agency practice at sites. In this frame of reference, values of \( \leq 50 \text{ ppt (0.05 ppb)} \) TEQs, \( > 50 \text{ ppt (0.05 ppb)} \) but \( < 1 \text{ ppb TEQs} \), and \( \geq 1 \text{ ppb TEQs} \) continue to be the agency's best estimate of appropriate health guidance values for purposes of screening, evaluation, and consideration of health action to limit exposure, respectively (Table 1).

Based on the foregoing frame of reference, the dioxin workgroup's recommendations are as follows:

**Issue 1: Relationship between ATSDR's action level and EMEGs**

- That ATSDR continue to use the EMEG of 50 ppt as TEQs for soil contaminated with dioxin and dioxin-like compounds for purposes of screening

- That 1 ppb dioxin and dioxin-like compounds expressed as TEQs in soil continue to be used by ATSDR as an "action level" (Johnson, 1992b), which has been characterized as "a reasonable level to begin consideration of action to limit exposure" (Kimbrough et al., 1984) to dioxin from residential soil.

**Issue 2: Analytic and sampling techniques**

- That ATSDR and EPA continue their efforts to assure earlier consultation at sites

- That the adequacy of analytic and sampling techniques be assessed on a site-specific basis.

**Issue 3: One part per billion of dioxin and dioxin-like compounds as an action level for cleanup**

- That ATSDR continue to consult with EPA regarding the appropriateness of 1 ppb of dioxin and dioxin-like compounds as an action level for cleanup or other actions to interdict exposure and protect human health on a site-specific basis.

*ATSDR Draft Profile for CDDs*

It is recommended that ATSDR complete its draft profile on CDDs in coordination with EPA's dioxin reassessment.

*Further Evaluation of Dioxin and Dioxin-Like Compounds*

Finally, once ATSDR's toxicological profile has been completed, the health guidance values for dioxin and dioxin-like compounds should be further evaluated when new information becomes available.
# APPENDICES FOR TECHNICAL SUPPORT DOCUMENT
## FOR ATSDR INTERIM POLICY GUIDELINE

## APPENDIX 1 - GLOSSARY

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Action level</td>
<td>A concentration of chemicals at which consideration of action to interdict/prevent exposure occurs, such as surveillance, research, health studies, community education, physician education, or exposure investigations. Alternatively, based on the evaluation by the health assessor, none of these actions may be necessary.</td>
</tr>
<tr>
<td>&quot;At-risk&quot; population</td>
<td>A population at a potentially elevated risk due to physiological sensitivity and/or increased exposure to a hazardous chemical.</td>
</tr>
<tr>
<td>BDDs</td>
<td>Brominated dibenzo-(p)-dioxins</td>
</tr>
<tr>
<td>BDFs</td>
<td>Brominated dibenzofurans</td>
</tr>
<tr>
<td>CDDs</td>
<td>Chlorinated dibenzo-(p)-dioxins</td>
</tr>
<tr>
<td>CDFs</td>
<td>Chlorinated dibenzofurans</td>
</tr>
<tr>
<td>Comparison value</td>
<td>A concentration used to select contaminants of concern at hazardous waste sites that are taken forward in the health assessment process for further evaluation (The terms comparison value and screening level are often used synonymously.)</td>
</tr>
<tr>
<td>Dioxin</td>
<td>A term used interchangeably with 2,3,7,8-tetrachlorodibenzo-(p)-dioxin or TCDD</td>
</tr>
<tr>
<td>Dioxin-like compounds</td>
<td>Compounds from a group of halogenated aromatic hydrocarbons that have molecules shaped like TCDD and produce similar toxic effects, such as certain other chlorinated dibenzo-(p)-dioxins (CDDs) and certain chlorinated dibenzofurans (CDFs), polychlorinated biphenyls (PCBs), polybrominated biphenyls (PBBs), brominated dibenzo-(p)-dioxins (BDDs), and brominated dibenzofurans (BDFs).</td>
</tr>
<tr>
<td>Dioxins</td>
<td>A term used interchangeably with chlorinated dibenzo-(p)-dioxins</td>
</tr>
<tr>
<td>EMEG</td>
<td>An environmental media evaluation guide (EMEG) is a media-specific comparison value that is used to select contaminants of concern at hazardous waste sites.</td>
</tr>
<tr>
<td>HazDat</td>
<td>ATSDR’s Hazardous Substance Release/Health Effects Database</td>
</tr>
</tbody>
</table>
MRL

A minimal risk level (MRL) is an estimate of the daily human exposure to a hazardous substance that is likely to be without an appreciable risk of adverse noncancer health effects over a specified route and duration of exposure.

PBBs

Polybrominated biphenyls

PCBs

Polychlorinated biphenyls

Screening

The process of initially identifying potentially important chemical contaminants and exposure pathways by eliminating those of known lesser significance.

TCDD

2,3,7,8-Tetrachlorodibenzo-p-dioxin

TEFs

Toxicity equivalency factors (TEFs) are based on congener-specific data and the assumption that the toxicity of dioxin and dioxin-like compounds is mediated by the Ah receptor and is additive. The TEF scheme compares the relative toxicity of individual dioxin-like compounds to that of TCDD, which is the most toxic halogenated aromatic hydrocarbon.

TEQs

Toxicity equivalent (TEQ) is defined as the product of the concentration, \( C_i \), of an individual “dioxin-like compound” in a complex environmental mixture and the corresponding TCDD toxicity equivalency factor (TEF) for that compound. The total TEQs is the sum of the TEQs for each of the congeners in a given mixture:

\[
\text{Total TEQs} = \sum_{i=1}^{n}(C_i \cdot \text{TEF}_i)
\]
APPENDIX 2 - ASSUMPTIONS, LIMITATIONS, AND UNCERTAINTIES IN DEVELOPING HEALTH GUIDANCE VALUES

Regulatory and policy decisions regarding contaminant levels must constantly be made in the face of scientific and technical uncertainties. In establishing health-based benchmarks such as minimal risk levels (MRLs) and environmental media evaluation guides (EMEGs), multiple assumptions are made about the nature of these uncertainties, depending on the specific question or issue being addressed. In interpreting and using health-based benchmarks to make general and/or site-specific decisions, these assumptions must be identified and addressed to avoid underestimating or overestimating actual risks. Some of these assumptions are made routinely during the development of health-based guidance values, and the conservatism they introduce into the final estimate is explicitly prescribed in the appropriate guidance documents.

Minimal Risk Level
An ATSDR MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse noncancer health effects for a specified route and duration of exposure. These substance-specific estimates, which are intended to serve as screening levels, are used by ATSDR health assessors and other responders to identify contaminants and potential health effects that may be of concern at hazardous waste sites. It is important to note that MRLs are not intended to define clean-up or action levels for ATSDR or other agencies.

MRLs are intended to serve as a screening tool to help public health professionals decide where to further evaluate the potential for health effects. They may also be viewed as a mechanism to identify those hazardous waste sites that are not expected to cause adverse health effects. MRLs contain some degree of uncertainty because of the lack of precise toxicological information on the people who might be most sensitive (e.g., infants, elderly, individuals with liver disease, and nutritionally or immunologically compromised) to the effects of hazardous substances. ATSDR uses a conservative (i.e., protective) approach to address these uncertainties consistent with the public health principle of prevention. Although human data are preferred, MRLs often must be based on animal studies because relevant human studies are lacking. In the absence of evidence to the contrary, ATSDR assumes that humans are more sensitive than animals to the effects of hazardous substances and that certain persons may be particularly sensitive. Thus, the resulting MRL may be as much as two orders of magnitude below levels shown to be effect levels in laboratory animals.

Environmental Media Evaluation Guide
The EMEG is a media-specific concentration below which exposure is unlikely to pose a health threat. The EMEG is calculated by multiplying the MRL by the body weight and dividing by the ingestion rate. No site-specific assumptions are used in deriving the EMEGs. Because they are not site-specific, they are not clean-up levels.
Assumptions used in developing the ATSDR EMEGs include (1) exposure occurs 24 hours a day for every day of the exposure period, (2) body weight, 10 kilograms for a child (22 pounds) and 70 kilograms for an adult (154 pounds), (3) ingestion rate for drinking water is 2 liters per day for adults and 1 liter for children, and (4) ingestion rate for soil is 100 milligrams per day for adults, 200 milligrams per day for children, and 5 grams per day for the geophagic child.

EMEGs should not be used to suggest or predict adverse health effects or to set clean-up levels. Their purpose is to provide health assessors with a means of selecting environmental contaminants for further evaluation (ATSDR, 1992).

**Exposure to Dioxin-like Compounds**

Dioxin-like compounds or "related chemicals" are other compounds containing chlorine or bromine whose molecules are shaped like TCDD and produce similar toxic effects, including some other dioxin congeners, some furan compounds, some polychlorinated biphenyls (PCBs), and some polybrominated biphenyls (PBBs) (Schierow, 1995). (See also Table 2-1 and Table 2-2.) As explained in Appendix 1, TEQs are used to estimate toxicity of dioxin-like compounds.

**TABLE 2-1. Recommended Toxicity Equivalency Factors (TEFs) for CDDs and CDFs**

<table>
<thead>
<tr>
<th>CDDs</th>
<th>EPA current recommended values</th>
<th>CDFs</th>
<th>EPA current recommended values</th>
</tr>
</thead>
<tbody>
<tr>
<td>monoCDDs</td>
<td>0</td>
<td>monoCDFs</td>
<td>0</td>
</tr>
<tr>
<td>diCDDs</td>
<td>0</td>
<td>diCDFs</td>
<td>0</td>
</tr>
<tr>
<td>triCDDs</td>
<td>0</td>
<td>triCDFs</td>
<td>0</td>
</tr>
<tr>
<td>2,3,7,8-TCDD</td>
<td>1</td>
<td>2,3,7,8-tetraCDF</td>
<td>0.1</td>
</tr>
<tr>
<td>other tetraCDDs</td>
<td>0</td>
<td>other tetraCDFs</td>
<td>0</td>
</tr>
<tr>
<td>2,3,7,8-pentaCDD&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.5</td>
<td>1.2,3,7,8-pentaCDF</td>
<td>0.05</td>
</tr>
<tr>
<td>other pentaCDDs</td>
<td>0</td>
<td>2.3,4,7,8-pentaCDF</td>
<td>0.5</td>
</tr>
<tr>
<td>2,3,7,8-hexaCDD&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.1</td>
<td>2.3,7,8-hexaCDF&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.1</td>
</tr>
<tr>
<td>other hexaCDDs</td>
<td>0</td>
<td>other hexaCDFs</td>
<td>0</td>
</tr>
<tr>
<td>2,3,7,8-heptaCDD&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.01</td>
<td>2.3,7,8-heptaCDF&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.01</td>
</tr>
<tr>
<td>other heptaCDDs</td>
<td>0</td>
<td>other heptaCDFs</td>
<td>0</td>
</tr>
<tr>
<td>octaCDF</td>
<td>0.001</td>
<td>octaCDF</td>
<td>0.001</td>
</tr>
</tbody>
</table>

<sup>a</sup>Any isomer that contains chlorine in the 2,3,7,8-positions

CDDs = chlorinated dibenz-p-dioxins; CDFs = chlorinated dibenzofurans;

TCDD = tetrachlorodibenzo-p-dioxin.


Some of the assumptions for using the TEQ approach include a well-defined group of chemicals, a broad database of information, consistency across end points, additivity of the effects, and a common mechanism of action (EPA, 1989a). According to EPA guidelines for risk assessment of complex mixtures, potency-weighted additivity is assumed for mixtures in the absence of information to the contrary (EPA, 1987).
The limitations associated with the use of TEQs must be considered in developing health guidance values. TEQs are derived using toxicity equivalency factors (TEFs) that are constants determined from experimental studies for each congener. Although TEFs are considered a constant, they are dependent on the specific study (end point, dose, and duration of exposure). As defined, TEQs are assumed to be additive and not synergistic or antagonistic. In actual mixtures of dioxin and dioxin-like compounds, competitive inhibition may occur at sufficiently high doses. As with MRLs and EMEGs, biomedical judgment must be used in considering site-specific conditions that would reasonably modify estimates applicable for an individual site.

**TABLE 2-2. Recommended Toxicity Equivalency Factors (TEFs) for Dioxin-Like PCBs**

<table>
<thead>
<tr>
<th>PCB</th>
<th>WHO proposed interim values</th>
<th>PCB</th>
<th>WHO proposed interim values³</th>
</tr>
</thead>
<tbody>
<tr>
<td>3,3',4,4'-TCB</td>
<td>0.0005</td>
<td>2,3,3',4,4',5-HxCB</td>
<td>0.0005</td>
</tr>
<tr>
<td>3,3',4,4',5-PeCB</td>
<td>0.1</td>
<td>2,2,3',4,4',5'-HxCB</td>
<td>0.0005</td>
</tr>
<tr>
<td>3,3',4,4',5,5'-HxCB</td>
<td>0.01</td>
<td>2,3',4,4',5,5'-HxCB</td>
<td>0.00001</td>
</tr>
<tr>
<td>2,3,3',4,4'-PeCB</td>
<td>0.0001</td>
<td>2,3,3',4,4',5,5'-HpCB</td>
<td>0.0001</td>
</tr>
<tr>
<td>2,3,4,4',5-PeCB</td>
<td>0.0005</td>
<td>2,2',3,3',4,4',5-HxCB</td>
<td>0.0001</td>
</tr>
<tr>
<td>2,3',4,4',5,5'-PeCB</td>
<td>0.0001</td>
<td>2,2',3,4,4',5,5'-HpCB</td>
<td>0.00001</td>
</tr>
<tr>
<td>2,3,4,4',4,4'-PeCB</td>
<td>0.0001</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

³Interim values proposed by World Health Organization/International Programme on Chemical Safety.

PCB = polychlorinated biphenyl; TCB = tetrachlorinated biphenyl; PeCB = pentachlorinated biphenyl; HxCB = hexachlorinated biphenyl; HpCB = heptachlorinated biphenyl.

Source: derived from Ahlborg et al. (1994).

**Bioavailability**

Bioavailability is an integral factor in the estimation of the internal dose (or dose at target tissue) of the chemical. The gastrointestinal absorption of TCDD and related compounds is variable, incomplete, and congener- and vehicle-specific. More lipid-soluble congeners, such as 2,3,7,8-tetrachlorodibenzofuran, are almost completely absorbed, while the extremely insoluble octachlorodibenzodioxin is less well absorbed depending on the dosing regimen; high doses may be absorbed at a lower rate, whereas low repetitive doses may be absorbed at a greater rate. The only study of TCDD bioavailability in humans was reported by Poiger and Schlatter (1986) and was based on a single male in which the gastrointestinal absorption was > 87% when TCDD was administered in corn oil.

Laboratory data suggest that there are no major interspecies differences in the gastrointestinal absorption of CDDs and CDFs. However, absorption of TCDD is dependent on conditions and characteristics of the soil medium; in animals, absorption of TCDD from different soils ranged from 0.5% (Umbreit et al., 1986a, 1986b) to 50% (Lucier et al., 1986). Absorption from a diet was 50% to 60% in rats (Fries and Marrow, 1975). Therefore, exposure with food as a vehicle, rather than with oil as a vehicle, relates more closely to exposure from soil. Bioavailability has to be considered when calculating the hypothetical ingestion dose.
If assumed that 100% of TCDD is bioavailable, risk may be overestimated. The health assessor should recognize that others used various assumptions in their calculations. Kimbrough et al. (1984) assumed 30% bioavailability from ingestion of soil, but pointed out that animal studies with contaminated Missouri soil indicated absorption up to 30% to 50% (McConnell et al., 1984). Pohl et al. (1995) assumed 40% bioavailability from soil. In contrast, Paustenbach et al. (1986) estimated bioavailability of 10% to 30%. Unless toxicokinetic studies that use soil samples from the specific site are available, it is difficult to speculate on how much TCDD and related compounds will be absorbed. Therefore, the estimate of the actual intake has limitations.

The chronic MRL is based on studies where food was the vehicle. Results from animal studies indicate that bioavailability of TCDD from soil varies between sites because dioxin and dioxin-like compounds bind tightly to soil, and increasingly so with the passage of time (Gough, 1991) and clay content of soil. Therefore, TCDD content alone may not be indicative of the potential for human health hazard from contaminated environmental materials, and site-specific evaluation is essential.

Soil Ingestion
Soil ingestion rates are assumptions included in the derivation of EMEGs (see previous section). ATSDR (1992) uses assumptions based on consumption of 100 mg/day for adults and 200 mg/day for children. The soil ingestion for children is based on a number of studies (Binder et al., 1986; Clausing et al., 1987) estimating the average soil ingestion in populations of normal children. Kimbrough et al. (1984) assumed in their calculations that children between 1.5 and 3.5 years of age ingest about 10 g of soil daily, and their risk assessment was based on “extreme total daily dose estimates.” This estimate was later disputed, and several studies were conducted to evaluate the daily intake of soil by children. One of the reports suggested that an average child ingests only about 25–40 mg of soil daily (Gough, 1991). However, about 1% to 2% of children are geophagic and ingest from 5 g to 10 g of soil daily (EPA, 1989b). Uncertainties associated with this issue are acknowledged, but ATSDR (1992) views ingestion rates of 100 mg/day and 200 mg/day for adults and children, respectively, to be reasonable. In the event that geophagic children are at risk, ATSDR considers this issue further in the public health assessment.

Background Exposure
EMEGs represent an estimation of exposure dose from one source only. All relevant sources of exposure from the hazardous waste site and all possible background exposures should be included in the final evaluation of actual exposure.

Dioxin and dioxin-like compounds are known to readily enter the food chain. It has been estimated that about 98% of exposure occurs through food. It should be noted that the average background intake of dioxin and dioxin-like compounds and of all TEQs of TCDD for adults in the general population were estimated as 0.35 pg/kg/day and 1.9 pg/kg/day, respectively (WHO, 1991).

Further, it is important to consider the background level of dioxin and dioxin-like compounds in contaminated soil. The U.S. background TCDD soil levels ranged from nondetected to 10 ppt in industrialized areas of groups of midwestern and mid-Atlantic states (Nestrick et al., 1986).
Exposure from Soil by Different Routes
Kimbrough et al. (1984) estimated that the lifetime uptake of TCDD from soil will consist of 95% from soil ingestion, 3% from soil dermal exposure (assuming 1% dermal absorption), and 2% from inhalation. Paustenbach et al. (1986) indicated that the 1% dermal absorption proposed for TCDD-contaminated soil may be too high. Similarly, he further lowered the estimates of inhalation intake, speculating that 2% from inhalation may be too high.

Unless indicated otherwise by the specific on-site circumstances, exposure by routes other than oral can be considered insignificant.

Use of Body Burdens to Compare Health Effects in Humans and Animals
Levels of exposure to dioxin and dioxin-like compounds that produce toxicity in experimental animals cannot be directly compared with levels associated with adverse health effects in humans because most epidemiologic studies do not provide adequate data to estimate the exposures in the studied cohort. However, body burden history can sometimes be estimated from reported serum or adipose concentrations and empirically based assumptions regarding whole-body elimination kinetics. Comparisons between estimated body burdens of dioxin and dioxin-like compounds associated with adverse health effects in experimental animals and humans have shown that humans and animals appear to respond to similar body burdens (DeVito et al., 1995).

By definition, the body burden of a chemical is the total amount of chemical present in the whole body at a particular time (Hodgson et al., 1988). Body burden of a chemical is determined by its toxicokinetics. It has been demonstrated that absorption, distribution, and elimination of dioxin and dioxin-like compounds are congener-specific (Flesch-Janys et al., 1996; Van den Berg et al., 1994). Further, parameters such as increased age of the exposed individual, increased body fat, and smoking may influence toxicokinetics (Flesch-Janys et al., 1996). Assumptions made regarding toxicokinetics of dioxin and dioxin-like compounds may result in limitations of the body burden method.

ATSDR acknowledges that other approaches may be used to estimate internal dose such as the area-under-the-curve (AUC) approach (Aylward et al., 1996). AUC is the total area under the curve that describes the concentration of a chemical in the systemic circulation as a function of time (from zero to infinity). AUC is equal to external dose divided by clearance (i.e., elimination rate divided by concentration in body fluid). As some authors have speculated (DeVito et al., 1995), it is possible that, in addition to dose and body burden, length of exposure may also play a significant role in the toxicity of dioxin and dioxin-like compounds. As such, it may be advantageous in some instances to use the AUC method. However, since information about length of exposure and external dose is often missing or inaccurate, the use of body burdens remains the method of choice to describe dose-response relationship. The body burden approach is employed by other ATSDR programs (e.g., in epidemiologic studies executed by the Division of Health Studies), by other U.S. governmental agencies (EPA, FDA), and by international agencies (WHO, IARC).
Attachment A

Petition Letter to ATSDR dated May 1, 2001
Dr. Henry Falk
Assistant Administrator
Agency for Toxic Substances and Disease Registry (ATSDR)
1600 Clifton Road, NE (E28)
Atlanta, GA 30333

Dear Dr. Falk,

We are writing on behalf of our organizations to petition for a public health assessment of the population of Midland, Michigan, because of chronic and serious dioxin contamination. The primary source of this contamination is the Dow Chemical Company.

Our petition is prompted by the following facts, which have emerged over the last two decades:

- Results of soil sampling by the state Department of Environmental Quality (DEQ) in Midland have been surprising, and alarming. Although the state’s residential cleanup criteria for dioxins in soil is 90 parts per trillion, of 37 samples taken in the community in 1996, almost a third had dioxin above that level. The areas that exceeded the residential cleanup standard included two elementary schools, an intermediate school, a high school and parks. These are areas where the public has access and children play. After release of these results, DEQ promised to resample community areas and determine potential human exposures.

- Instead of carrying through on its commitment, DEQ agreed with Dow’s suggestion to use soils inside Dow’s corporate center as a “surrogate” for the community. Levels found in 1998 sampling at the corporate center ranged from 66 to 476 parts per trillion, with an average of 136 parts per trillion. One particularly high dioxin hot spot was adjacent to a residential area east of the Dow facility.

- DEQ and Dow still refuse to keep commitments to characterize human exposures to dioxins and take appropriate protective actions – more than four years after the first sampling was done.

- There are other routes of exposure to dioxins in Midland. Dioxin contamination of fish in the Tittabawassee River below Midland is chronically high. According to the Michigan Fish Contaminant Monitoring Program: 2000 Annual Report issued by DEQ this winter, dioxin TEQ concentrations exceeded the “trigger level” for fish consumption warnings in all 10 carp collected in 1999 and in all 11 carp collected since 1992, and in 2 of 5 smallmouth bass. As a result, the state has tightened its advisory to warn against eating more than 1 meal per week.
for smallmouth bass from the river due to dioxin, and advises that women and children eat no smallmouth basis from the river due to elevated levels of dioxins and PCBs.

This data is simply the latest in a long line of disclosures about dioxin contamination of the community. For example, a 1985 multi-media risk assessment by the U.S. Environmental Protection Agency pointed to birth defects and cancer data suggesting elevated health effects in the Midland community, noted that the highest levels of dioxin in the nation were found in Tittabawassee River fish, and called for a comprehensive health study. To date, no such study has ever been done. Further, rather than taking action to protect the public from the serious soil contamination documented in the two most recent rounds of soil testing, the State of Michigan has continued to engage in private discussions with Dow Chemical Company about how to manage public relations. Despite repeated requests from our organizations for an independently-funded, state-commissioned health study and a plan to protect citizens from exposure to excessive levels of dioxins, the state has taken no such action.

It is abundantly clear that significant levels of dioxins and other hazardous materials, including PCBs, are present in the Midland community and in adjacent communities, where contaminants are transported from Dow via water and air. These contaminants may be ingested through fish, consumed in other food, absorbed through dermal contact with soils, and inhaled. The science supporting the link between dioxins and human health effects is strong and growing. It is time for a public health assessment by ATSDR and appropriate protective actions by federal, state and local agencies to prevent further exposures to dioxin and to study health impacts in the community.

Sincerely,
Attachment B

Petition Scoping Report for Dow Chemical Company Midland
Petition Scoping Report  
August 31, 2001

Site/City/State: Dow Chemical Company/Midland/Michigan
Region: 5
Scoping Team:

A. Petitioner’s Concern(s)

- Dioxins, reported as total equivalent concentrations (TEQs) of 2,3,7,8-TCDD, have been detected in soil in Midland at concentrations above the Michigan Department of Environmental Quality residential cleanup criterion. Levels of dioxins detected in soil adjacent to the eastern perimeter of the Dow plant site and along a road-way (haul route) in the community exceed 1 ppb.

- Dioxins have been detected in fish taken from the Tittabawassee River. Levels detected exceed the State of Michigan trigger levels for fish consumption warnings. In 1985, the U.S. EPA noted that the highest national levels of dioxins in fish were found in the Tittabawassee River.

- In 1985, the U.S. EPA “called for a comprehensive health study” of dioxin exposures and the resulting health effects in the Midland community. No such comprehensive study has ever been performed.

B. Brief Site Background

The Dow Chemical, founded in 1897, operates a chemical manufacturing facility in the city of Midland, Michigan. The facility encompasses approximately 1900 acres on the southern perimeter of the city. The Tittabawassee River forms the southern boundary of the facility and flows east to the Saginaw Bay of Lake Huron.

Chemicals produced at the Midland plant include: styrene, butadiene, picric acid, mustard gas, Saran wrap, Styrofoam, Agent Orange, napalm, and various pesticides including Dursban and 2,4,5-T. Chlorophenol production began in 1915. Wastes generated from this process were initially disposed of in 600 acres of on-site waste ponds. During high flow periods in the early 1900’s, wastes from these ponds would be released to the Tittabawassee River. Dow currently operates it’s own wastewater treatment plant on-site.

Two rotary kiln incinicators are used for treatment of liquid and solid hazardous and non-hazardous wastes generated from manufacturing activities at the facility. Ambient air dispersion modeling and monitoring indicates that the north-eastern quadrant of the city of Midland have been affected by emissions from the incinicators.

C. Key Previous Actions Related To The Site

The Dow property is currently part of the RCRA corrective action program delegated by the U.S. EPA to the MDEQ Waste Management Division. The EPA sampled soil in the city of Midland in the 1980's and found elevated concentrations of 2,3,7,8-TCDD. The EPA recommended additional sampling in the future to monitor levels of dioxins in the community. In 1996, the MDEQ took additional soil samples from public properties such as parks and school yards and found total dioxins (TEQ) at concentrations exceeding the Michigan residential cleanup criterion for soil. The Dow plant site, it’s northeastern perimeter, and a community road-way leading from the Dow plant to a landfill were also sampled. Concentrations at the plant perimeter and on the road-way exceeded 1 ppb. In 1998, as a follow-up alternative to additional community sampling, the Dow Company and the DEQ agreed to sample the Dow Corporate Center property as a surrogate for the community. Levels of TEQs detected at the Corporate Center ranged from 77-583 parts per trillion (ppt).
No public health assessment has been conducted by either the MDCH or the ATSDR.

D. Public Health Issue(s)

Table 1. Levels of total dioxins detected in soil in the city of Midland.

<table>
<thead>
<tr>
<th>Soil Samples Location</th>
<th>Range of TEQ detected in ppt</th>
<th>DEQ Cleanup Criterion in ppt</th>
<th>ATSDR Chronic Soil EMEMG for a-Child in ppt</th>
<th>ATSDR Chronic Soil EMEMG for an Adult in ppt</th>
<th>ATSDR Action Level for TCDD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Northeast Plant Perimeter</td>
<td>6 - 1068</td>
<td>90</td>
<td>50</td>
<td>700</td>
<td>1000</td>
</tr>
<tr>
<td>Road-Way (Haul Route)</td>
<td>10 - 2663</td>
<td>90</td>
<td>50</td>
<td>700</td>
<td>1000</td>
</tr>
<tr>
<td>Dow Corporate Center</td>
<td>77 - 583</td>
<td>90</td>
<td>50</td>
<td>700</td>
<td>1000</td>
</tr>
<tr>
<td>Northeast Quadrant of Midland</td>
<td>22 - 598</td>
<td>90</td>
<td>50</td>
<td>700</td>
<td>1000</td>
</tr>
</tbody>
</table>

Table 2. Levels of total dioxins (TEQ) detected in fish in the Tittabawassee River downstream of Midland.

<table>
<thead>
<tr>
<th>Date</th>
<th>Range of 2,3,7,8-TCDD in ppt</th>
<th>Range of TEQ in ppt</th>
<th>MDCH Advisory Trigger in ppt</th>
</tr>
</thead>
<tbody>
<tr>
<td>1976 - 1980</td>
<td>3 - 695</td>
<td>NA</td>
<td>10</td>
</tr>
<tr>
<td>1983 - 1989</td>
<td>1.1 - 530</td>
<td>1.11 - 10.91</td>
<td>10</td>
</tr>
<tr>
<td>1990 - 1999</td>
<td>1 - 209</td>
<td>4.56 - 73.90</td>
<td>10</td>
</tr>
<tr>
<td>2000 -</td>
<td>Data pending</td>
<td>Data pending</td>
<td>10</td>
</tr>
</tbody>
</table>

Demographics

The city of Midland is the county seat of Midland County, Michigan and encompasses an area approximately 28 square miles. The population of Midland was approximately 38,090 in 1990. Twenty five percent of the population in 1990 were children under the age of 17 years.

Previous Health Studies

At the request of the MDEQ Air Quality Division (AQD) and in support of the AQD review of an application made by the Dow Company for an air quality permit for a new incinerator, the MDCH performed a statistical analysis of cancer incidence for zip codes 48640 and 48642, Midland County, and Bay County. This analysis showed that the 1994 through 1998 age-adjusted incidence rate for all cancers combined in zip code 48640, which includes the Dow plant site, was significantly higher than the corresponding rates for all white residents in Midland County, Bay County, and the State of Michigan. Incident rates were also elevated in this zip code for lung and prostate cancer. No elevations in cancer rates were indicated for zip code 48642. (see attachment)
E. Exposure Pathways

<table>
<thead>
<tr>
<th>Pathway</th>
<th>Source of Contaminant</th>
<th>Contaminant and Level</th>
<th>Environmental Media</th>
<th>Exposure Point</th>
<th>Exposure Route</th>
<th>Exposed Population</th>
<th>Time Frame</th>
</tr>
</thead>
<tbody>
<tr>
<td>Direct Contact with Soil</td>
<td>Incinerator Emissions</td>
<td>Dioxins and Furans 6 - 2663 ppt</td>
<td>Soil</td>
<td>Soil in the city of Midland</td>
<td>Incidental Ingestion Dermal Contact Particulate Inhalation</td>
<td>Residents of Midland</td>
<td>Past</td>
</tr>
<tr>
<td>Fish Consumption</td>
<td>Release to Surface Water</td>
<td>Dioxins and Furans 1.1 - 73.9 ppt</td>
<td>Fish Tittabawassee River</td>
<td>Ingestion</td>
<td>Anglers and their families</td>
<td>Future</td>
<td>Past</td>
</tr>
</tbody>
</table>

Level of Community Interest (Difficult to gauge at this time. The Michigan Environmental Council, a well-organized environmental group is one of the petitioners. However, Midland is a corporate town and support for the company is high)

- **X** High
  Large numbers of inquiries about the site/release; well attended meetings about a site/release; the involvement of national, state, and local environmental activist groups, and community groups that are well-organized; extensive environmental, health and/or political interest and extensive national, state and local media coverage.

- **X** Medium
  Involvement of the petitioner and community groups without the involvement of national, and state environmental activist groups; some national or state environmental, health and/or political interest; only local media coverage.

- **Low**
  Involvement of the petitioner; no community, environmental, health, or political interest; no media coverage.

F. Decision Criteria

2.1 Are the location, concentration, and toxicity of the hazardous substances related to the petition, site, or release possibly of public health concern?

Yes, levels of dioxins detected in soil in the city of Midland and in fish in the Tittabawassee River downstream of Midland exceed health-based comparison values. Dioxin and related compounds are believed to cause both carcinogenic and noncarcinogenic human health effects at extremely low levels of exposure.

2.2 Is there an exposed or potentially exposed population as indicated in the petition and as determined by evaluating the human exposure pathways for the hazardous substance release(s)?

Yes, dioxins are present in soil throughout the city of Midland. More than 38,000 people live in the city, 25% of which are children under the age of 17. The Tittabawassee River is a valuable State of Michigan fishery resource and is heavily utilized both by the residents of Midland and by other communities down river.

2.3 Is there a plausible relationship between possible human exposure to a release of hazardous substances and community health concerns or adverse health outcomes?
Yes, the U.S. EPA, the International Agency for Research on Cancer, and the National Toxicology Program have determined that exposure to dioxins is associated with elevated rates of all cancers combined as well as several particular cancers including lung and soft tissue sarcoma. Elevated incidence rates of all cancers combined, lung and prostate cancer have been detected in the city of Midland. Additional information is needed to determine if exposure to dioxins is related to other health effects in the population of Midland and the surrounding communities.

G. **Recommendation:**

The scoping team recommends:

<table>
<thead>
<tr>
<th></th>
<th>Action</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>X</td>
<td>No further action</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>Further characterization</td>
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</tr>
<tr>
<td>X</td>
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<td>X</td>
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<td>Exposure Investigation</td>
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</tr>
</tbody>
</table>

*Refer as a Public Health Consultation to a toxicologist, epidemiologist or physician for determination of plausible relationship between possible human exposure to a release of hazardous substances and community health concerns or adverse health outcomes, if relationships are not readily available utilizing the tox. profiles.*

**Referral to:**
Attachment C

ATSDR Letter to Petitioners for Dow Midland site dated November 2, 2001
In May 2001, you wrote to the Agency for Toxic Substances and Disease Registry (ATSDR), about the Dow Chemical Company, Midland, Michigan and dioxin contamination. ATSDR acknowledged your letter to be a petition for a public health assessment. The following outlines ATSDR's response to your petition.

After reviewing the public health issues and community concerns about potential dioxin contamination and the Dow Midland facility, ATSDR has found a reasonable basis to prepare public health consultations to address the concerns associated with the Dow facility. The public health consultations will review and summarize the existing environmental and health data for dioxin concentrations in soils in the Midland community and in fish found in community streams. The consultations will evaluate possible ways that people could be exposed to harmful substances, document and evaluate community health concerns, state health-based conclusions, and make recommendations. We believe that the health consultations will provide timely, appropriate responses to the concerns.

ATSDR maintains a cooperative agreement with the Michigan Department of Community Health (MDCH), under which MDCH conducts public health assessments and other environmental health activities in Michigan. ATSDR has requested that MDCH complete the public health consultations and release them for public review in fiscal year 2002, which began October 1, 2001. MDCH has a talented staff with a good track record for successfully conducting public health evaluations, and they understand the needs of Michigan communities. ATSDR will review MDCH's work and provide technical support as needed. We have enclosed a fact sheet about public health consultations.

Thank you for referring your concerns to ATSDR. We welcome your comments about this response and the planned public health consultations. If you have questions about our proposed plan of action, please contact Dr. Mark Johnson, ATSDR Senior Regional Representative, at telephone (312) 886-0840, or Alan Yarbrough, ATSDR Technical Project Officer, at telephone (404) 498-0427. Dr. Linda Larsen, MDOH, may be contacted at (517-335-8566). Community members may also contact ATSDR by calling our toll-free telephone number, 1-888-42-ATSDR (1-888-422-8737).

Sincerely yours,

Robert C. Williams, P.E., DEE
Assistant Surgeon General
Director, Division of Health Assessment and Consultation

Enclosure

cc:
Linda D. Larsen, Ph.D.
Michigan Department of Community Health
Attachment D

Dioxin and Dioxin-Like Compounds in Soil,
Part 1: ATSDR Interim Policy Guideline
DIOXIN AND DIOXIN-LIKE COMPOUNDS IN SOIL, PART I:
ATSDR INTERIM POLICY GUIDELINE

CHRISTOPHER T. DE ROSA, DAVID BROWN, ROSALINE DHARA,
WOODROW GARRETT, HUGH HANSEN, JAMES HOLLER, DENNIS JONES,
DENISE JORDAN-IZAGUIRRE, RALPH O'CONNOR, HANA POHL, AND
CHARLES XINTARAS

Agency for Toxic Substances and Disease Registry
U.S. Department of Health and Human Services
Atlanta, Georgia

PURPOSE

The Agency for Toxic Substances and Disease Registry (ATSDR) has adopted this interim policy
guideline to assess the public health implications of dioxin and dioxin-like compounds in residential
soils near or on hazardous waste sites. These compounds include

- 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD)
- Related chlorinated dibenzo-p-dioxins (CDDs)
- Chlorinated dibenzofurans (CDFs)
- Other structurally related groups of chemicals from the family of halogenated aromatic
  hydrocarbons.

These substances are defined under the Comprehensive Environmental Response, Compensation,
and Liability Act of 1980 (CERCLA), as amended, commonly known as Superfund. This interim
policy guideline will provide a clear and consistent understanding of ATSDR's current approaches
and judgments regarding hazards posed by the presence of TCDD and its less toxic dioxin-like
congeners, the CDDs and CDFs, in residential soils. Likely users of this interim policy guideline
include:

- ATSDR and state-based health assessors
- ATSDR partners including relevant federal, state, and local health and environmental
  entities
- Concerned community groups.

1. Address all correspondence to: Christopher T. De Rosa, Ph.D., Director, Division of Toxicology,
Agency for Toxic Substances and Disease Registry, Mailstop E-29, 1600 Clifton Road, NE, Atlanta, GA
30333. Tel.: (404) 639-6300. Fax: (404) 639-6315. E-mail: cyrd0@cdc.gov.
2. Abbreviations: ATSDR, Agency for Toxic Substances and Disease Registry; CDDs, chlorinated dibenzo-
p-dioxins; CDFs, chlorinated dibenzofurans; CERCLA, Comprehensive Environmental Response,
Compensation, and Liability Act of 1980; EMEG, environmental media evaluation guide; FDA, U.S. Food
and Drug Administration; MRL, minimal risk level; TCDD, 2,3,7,8-tetrachlorodibenzo-p-dioxin; TEF,
toxicity equivalency factor; TEQs, toxicity equivalency.
3. Key words: dioxin, human exposure, risk assessment, soil levels, TCDD, TEQs.
4. Note: "65 Bulkley Avenue North, Westport, CT 06880.
INTERIM POLICY GUIDELINE

This interim policy guideline is based on a current understanding of the toxicology and epidemiology associated with TCDD and its congeners (see “Background” section) and on exposure potential when soil is the primary medium of interest.

This guidance is consistent with the Dioxin and Dioxin-Like Compounds in Soil, Part II: Technical Support Document for ATSDR Interim Policy Guideline (De Rosa et al., 1997) and with the ATSDR Public Health Assessment Guidance Manual (ATSDR, 1992). They explain how to use comparison values to select contaminants for further evaluation and then draw conclusions about the public health implications of the contaminants. Assessments of public health implications are based on considerations of site-specific factors affecting the extent and characteristics of exposure and on the toxicology and epidemiology of the compounds selected for evaluation.

This guidance for dioxin and dioxin-like compounds is unique because of the potency of TCDD itself, and the need to consider the total potency of all dioxin and dioxin-like compounds detected in soil. The toxicity of a dioxin-like compound is commonly referred to in terms of its dioxin toxicity equivalency factor (TEF). See “Background” section for further information.

These guidelines and procedures apply to human exposure for direct ingestion of soils contaminated with dioxin and dioxin-like compounds in residential areas and may not be appropriate for other exposure scenarios. The guidance will be evaluated in view of new data that may become available. The science basis for the guidance is outlined in the “Background” discussion.

Step 1. Screening for contaminants of concern

Review soil sampling data and compare levels against dioxin comparison values (environmental media evaluation guide or EMEG for children) that are not site-specific. If one or more soil sampling values exceed the screening value of 50 parts per trillion (ppt) of toxicity equivalents (TEQs), further site-specific evaluations are needed as described next and in Table 1.

If samples exceed this screening value, then ATSDR generally assumes that further evaluation is required. However, even if samples are below these values, ATSDR policy states that it may still be necessary to conduct a more detailed site-specific evaluation under the following conditions:

- community health concerns
- health assessor’s concerns about other combinations of contaminants.
Step 2. Evaluating potential exposure pathways

Further evaluation includes the most critical aspect of health hazard evaluations, that is, the determination of likelihood, extent, and duration of exposure of populations. Thus, the health assessor uses the following to determine the existence of a potential or completed exposure pathway—past, present, or future:

- site visits and observations
- detailed review of data packages for land use scenarios, contaminant locations, and site locations
- evaluation of receptor populations and potential points of contact.

If a completed or potentially completed exposure pathway is identified, then the extent of exposure and public health implications are further evaluated.

Site-specific exposure scenarios based on site-specific factors are evaluated in conjunction with relevant toxicologic, epidemiologic, and medical information. This involves assessing site-specific information about the likelihood, frequency, routes, and levels of exposure to contaminants, and the populations that are likely to be exposed.

Where estimated levels of exposure in soil fall in the range of greater than 50 ppt to less than 1 part per billion (ppb) TEQs (Table 1), a weight-of-evidence approach is recommended to evaluate the exposure and the public health implications of the exposure.

Health assessors must ask the following questions:

- How extensive is the contamination?
- Is the contamination isolated or widespread?
- Is the contamination in surface soils or areas easily accessible to children or adults? Is it in areas with no vegetation or in any other areas?
- At this site, how often (daily, weekly, monthly) and for what length of time (months, years, lifetimes) would exposures be likely to occur?

Many of these estimates depend on professional judgment and experience regarding the likelihood of exposures from soils in different kinds of sites. For further information on the evaluation process see ATSDR (1992).

Interpretation of Health Guidance Values

The policy incorporates information on exposure potential from residential soils and residential exposure scenarios. It should be noted that the levels (in TEQs) ≤ 50 ppt (0.05 ppb), > 0.05 ppb but < 1 ppb, and ≥ 1 ppb in residential soils are guidance values and should not be construed to indicate that actual health effects will occur. The policy provides a protective framework for evaluating the health implications of exposures to dioxin and dioxin-like compounds in residential soils on a site-specific basis.
TABLE 1. ATSDR's Decision Framework for Sites Contaminated with Dioxin and Dioxin-Like Compounds

Because the toxicity of dioxin and dioxin-like compounds is assumed to be elaborated through a common receptor-mediated mechanism, levels greater than 50 ppt (0.05 ppb) TEQs* are used to determine whether further site-specific evaluation for dioxins is to occur based on the maximum soil concentrations identified at the site. A level of 1 ppb TEQs is used to determine the potential need for public health actions on a site-specific basis and on the basis of adequate sampling and measured or projected human exposure—past, present, or future—as determined by the health assessor.

<table>
<thead>
<tr>
<th>SCREENING LEVEL</th>
<th>EVALUATION LEVELS</th>
<th>ACTION LEVEL*</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤ 50 ppt (0.05 ppb) TEQs</td>
<td>&gt; 0.05 ppb but &lt; 1 ppb TEQs</td>
<td>≥ 1 ppb TEQs</td>
</tr>
<tr>
<td>• The EMEG for TCDD is 50 ppt</td>
<td>Evaluation of site-specific factors, such as:</td>
<td>Potential public health actions considered, such as:</td>
</tr>
<tr>
<td>• This is based on an MRL of 1 pg/kg/day for TCDD (ATSDR, 1989).</td>
<td>• Bioavailability</td>
<td>• Surveillance</td>
</tr>
<tr>
<td>• For screening purposes 50 ppt TCDD is assumed to be equivalent to 50 ppt TEQs</td>
<td>• Ingestion rates</td>
<td>• Research</td>
</tr>
<tr>
<td></td>
<td>• Pathway analysis</td>
<td>• Health studies</td>
</tr>
<tr>
<td></td>
<td>• Soil cover</td>
<td>• Community education</td>
</tr>
<tr>
<td></td>
<td>• Climate</td>
<td>• Physician education</td>
</tr>
<tr>
<td></td>
<td>• Other contaminants</td>
<td>• Exposure investigations</td>
</tr>
<tr>
<td></td>
<td>• Community concerns</td>
<td>• Background Exposures</td>
</tr>
<tr>
<td></td>
<td>• Demographics</td>
<td></td>
</tr>
</tbody>
</table>

The toxicity equivalent (TEQ) of TCDD is calculated by multiplying the exposure level of a particular dioxin-like compound by its toxicity equivalency factor (TEF). TEFs are based on congener-specific data and the assumption that Ah receptor-mediated toxicity of dioxin-like chemicals is additive. The TEF scheme compares the relative toxicity of individual dioxin-like compounds to that of TCDD, which is the most toxic halogenated aromatic hydrocarbon.

*A concentration of chemicals at which consideration of action to interdict/prevent exposure occurs, such as surveillance, research, health studies, community education, physician education, or exposure investigations. Alternatively, based on the evaluation by the health assessor, none of these actions may be necessary.

Step 3. Defining public health implications/actions

Where exposures to concentrations in residential soils exceeding 1 ppb TEQs are significant, ATSDR health assessors should consider judging the site a public health hazard and consider site-specific public health recommendations/actions to prevent or interdict exposures (Table 1).

BACKGROUND FOR INTERIM POLICY GUIDELINE

Dioxin and Dioxin-Like Compounds

Dioxin and dioxin-like compounds are structurally related groups of chemicals from the family of halogenated aromatic hydrocarbons. Depending on the number of chlorine-substituted positions, there are several congeners in each group. The most toxic and the most studied congener is TCDD.
TEFs were developed to compare the relative toxicity of individual dioxin-like compounds to that of TCDD (Tables 2 and 3). This comparison is based on the assumption that dioxin and dioxin-like compounds act through the same mechanism of action. The TEF for TCDD is defined as one, whereas TEF values for all other dioxin-like compounds are less than one. TEQs are used to assess the risk of exposure to a mixture of dioxin-like compounds. A TEQ is defined as the product of the concentration, $C_i$, of an individual “dioxin-like compound” in a complex environmental mixture and the corresponding TCDD TEF, for that compound. The total TEQs is the sum of the TEQs for each of the congeners in a given mixture:

$$\text{Total TEQs} = \sum_{i=1}^{n} (C_i \cdot \text{TEF}_i)$$

Adverse Health Effects

Studies in animals demonstrated a wide range of effects associated with dioxin exposure including death, cancer, and wasting, as well as hepatic, immunologic, neurologic, reproductive, and developmental effects. In contrast to laboratory results, direct exposure information is not available in human studies; therefore, body burden is used as a surrogate. Body burdens in some animal studies were in the same range as those associated with adverse health effects in human studies. For more information, see Technical Support Document for ATSDR Interim Policy Guideline: Dioxin and Dioxin-Like Compounds in Soil (ATSDR, 1997). These results underscore the need for research to elucidate the toxicity at low doses to human populations and to evaluate exposures in at-risk populations (see Appendix 1) in view of total body burdens of dioxin and dioxin-like compounds.

**TABLE 2. Recommended Toxicity Equivalency Factors (TEFs) for CDDs and CDFs**

<table>
<thead>
<tr>
<th>CDDs</th>
<th>EPA current recommended values</th>
<th>CDFs</th>
<th>EPA current recommended values</th>
</tr>
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<tr>
<td>monoCDDs</td>
<td>0</td>
<td>monoCDFs</td>
<td>0</td>
</tr>
<tr>
<td>diCDDs</td>
<td>0</td>
<td>diCDFs</td>
<td>0</td>
</tr>
<tr>
<td>triCDDs</td>
<td>0</td>
<td>triCDFs</td>
<td>0</td>
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<tr>
<td>2,3,7,8-TCDD</td>
<td>1</td>
<td>2,3,7,8-tetraCDF</td>
<td>0.1</td>
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<tr>
<td>other tetraCDDs</td>
<td>0</td>
<td>other tetraCDFs</td>
<td>0</td>
</tr>
<tr>
<td>2,3,7,8-pentaCDF&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.5</td>
<td>1,2,3,7,8-pentaCDF</td>
<td>0.05</td>
</tr>
<tr>
<td>other pentaCDDs</td>
<td>0</td>
<td>2,3,4,7,8-pentaCDF</td>
<td>0.5</td>
</tr>
<tr>
<td>2,3,7,8-hexaCDD&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.1</td>
<td>other pentaCDFs</td>
<td>0</td>
</tr>
<tr>
<td>other hexaCDDs</td>
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<td>2,3,7,8-hexaCDF&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.1</td>
</tr>
<tr>
<td>2,3,7,8-heptaCDD&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.01</td>
<td>other hexaCDFs</td>
<td>0</td>
</tr>
<tr>
<td>other heptaCDDs</td>
<td>0</td>
<td>2,3,7,8-heptaCDF&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.01</td>
</tr>
<tr>
<td>octaCDD</td>
<td>0.001</td>
<td>octaCDF</td>
<td>0.001</td>
</tr>
</tbody>
</table>

<sup>a</sup>Any isomer that contains chlorine in the 2,3,7,8-positions

CDDs = chlorinated dibenzo-p-dioxins; CDFs = chlorinated dibenzofurans; TCDD = tetrachlorodibenzo-p-dioxins.

TABLE 3. Recommended Toxicity Equivalency Factors (TEFs) for Dioxin-Like PCBs

<table>
<thead>
<tr>
<th>PCB</th>
<th>WHO proposed interim values*</th>
<th>PCB</th>
<th>WHO proposed interim values*</th>
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</thead>
<tbody>
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<td>3,3'4,4'-TCB</td>
<td>0.0005</td>
<td>2,3,3'4,4',5-HxCB</td>
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<td>3,3',4,4',5-PCB</td>
<td>0.1</td>
<td>2,2,3,4,4',5-HxCB</td>
<td>0.0005</td>
</tr>
<tr>
<td>3,3',4,4',5,5'-HxCB</td>
<td>0.01</td>
<td>2,3',4,4',5,5'-HxCB</td>
<td>0.000001</td>
</tr>
<tr>
<td>2,3',4,4'-PeCB</td>
<td>0.00001</td>
<td>2,3',4,4',5,5'-HpCB</td>
<td>0.0001</td>
</tr>
<tr>
<td>2,3',4,4',5-PeCB</td>
<td>0.0005</td>
<td>2,2',3,3',4,4',5-HpCB</td>
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<tr>
<td>2',3',4,4',5-PeCB</td>
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<tr>
<td>2',3',4,4',5-PeCB</td>
<td>0.00001</td>
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</tbody>
</table>

*Interim values proposed by World Health Organization/International Programme on Chemical Safety
PCB = polychlorinated biphenyl; TCB = tetrachlorinated biphenyl; PeCB = pentachlorinated biphenyl;
HxCB = hexachlorinated biphenyl; HpCB = heptachlorinated biphenyl
Source: derived from Ahlborg et al. (1994).

Screening Level for Dioxin and Dioxin-Like Compounds in Soil
While identifying levels of potential concern to human health, ATSDR considers a spectrum of contaminant concentrations. In general, screening levels are concentrations used to select contaminants of concern at hazardous waste sites that are taken forward in the health assessment process for further evaluation (screening levels are also called comparison values; see Appendix 1 - Glossary).

A minimal risk level (MRL) is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse noncancer health effects over a specified duration and route of exposure. These substance-specific estimates, which are intended to serve as screening levels, are used by ATSDR health assessors and others to identify contaminants and potential health effects that may be of concern at hazardous waste sites. The intermediate-duration oral MRL of 1 picogram/kilogram/day or pg/kg/day for TCDD (ATSDR, 1989) was based on reproductive effects in rats. The intermediate-duration oral MRL was also adopted as a chronic oral MRL. Based on this value, an EMEG of 50 ppt (0.05 ppb) TCDD, which is equivalent to 50 ppt (0.05 ppb) TEQs, was derived for exposure from contaminated soil. Uncertainty factors of 1000 (total) were used in the calculations of the MRL (for further details, see Appendix 3 of the Technical Support Document). Based on a review of more recent literature, ATSDR scientists conclude that the MRL of 1 pg/kg/day is approximately two orders of magnitude below the noncancer health effect levels observed in recent studies. This is also true for cancer effect levels.

Evaluation Levels for Dioxin and Dioxin-Like Compounds in Soil
Evaluation levels are concentrations > 50 ppt (0.05 ppb) but < 1 ppb TEQs at which site-specific factors, including, but not limited to, bioavailability, ingestion rates, pathway analysis, soil cover, climate, other contaminants, community concerns, demographics, and background exposure, are considered in a deliberative process to assess the nature and extent of contamination and its impact on the community. Such an evaluation process may prompt further assessment at the next level where actions are considered. The evaluation levels are to be used as a framework to guide procedures for that judgment process. Thus, judgments in the evaluative phase are linked to actions where consideration is given to interventions from a public health perspective.
Action Level for Dioxin and Dioxin-Like Compounds in Soil

Action levels are concentrations of chemicals at which consideration of action to interdict exposure occurs: 1 ppb TCDD in residential soil was identified by Kimbrough et al. (1984) as a "level of concern," and recommended as "a reasonable level to begin consideration of action to limit exposure." Kimbrough et al.'s (1984) conclusions were derived in part from an evaluation of the carcinogenic potential of TCDD, based on a 2-year oral chronic toxicity and oncogenicity study in rats (Kociba et al., 1978). With the advancement of knowledge about dioxin-like chemicals and their assumed common mechanism of toxicity, the TEQs were introduced into the risk assessment process. Since then, 1 ppb of total dioxins (expressed as TEQs) in soil has been used as an action level by ATSDR.

The Kociba et al. (1978) study also served as the basis for the Food and Drug Administration's (FDA's) derivation of a risk-specific dose of 0.057 pg/kg/day dioxin for a l in a million (10^-6) upper-bound risk estimate for cancer (FDA, 1990). Using a typical default value of 70 kg for average body weight and 100 milligrams/day (mg/day) for soil consumption, FDA's 0.057 pg/kg/day risk-specific dose corresponds to a soil concentration of 40 ppt. This value is marginally lower, but from a risk assessment perspective, it is essentially equivalent to the ATSDR media-specific screening level/comparison value (EMEG) of 50 ppt.

As noted previously, ATSDR's EMEG is based on the MRL of 1 pg/kg/day TCDD, which is approximately two orders of magnitude below any health effect levels demonstrated either experimentally or in epidemiologic studies for both cancer and noncancer health endpoints. The conservative (i.e., protective) nature of both the MRL and the EMEG reflects adjustments made for recognized areas of uncertainty, perhaps spanning two to three orders of magnitude. As such, the EMEG and the MRL, on which the EMEG is based, are below levels of exposures associated with demonstrated health effects and are therefore considered to be protective of human health. The EMEG of 50 ppt (0.05 ppb) is at the low end of the range reflecting currently recognized areas of scientific uncertainty; this range is 50–50 000 ppt (or 0.05–50 ppb), which is based on the 1000-fold uncertainty factor used to derive the MRL.

CONCLUSIONS

ATSDR concludes that the action level of 1 ppb (TEQ) for dioxin and dioxin-like compounds, when coupled to a site-specific context of evaluation for the range > 50 ppt (0.05 ppb) to < 1 ppb TEQs in residential soil, is protective of public health and continues to represent a level at which consideration of health action to interdict exposure, including cleanup, should occur. This conclusion is based on ATSDR's review and evaluation of

- more recent experimental and epidemiologic research findings
- ATSDR's historical use of the term "action level"
- the range of health guidance values developed by ATSDR including the MRL and EMEG
- the limitations and uncertainties of ATSDR's health guidance values and the scientific data on which these values are based.
ATSDR considers this action level to be both reasonable and protective for the following reasons:

- ATSDR's MRL is approximately two orders of magnitude below effect levels in experimental and epidemiologic studies.
- Cancer risk-specific doses and screening values for end points other than cancer are essentially equivalent from a risk assessment perspective.

WHERE TO FIND MORE INFORMATION

For more information on the historical and scientific background of dioxin in soil values, their proper use, and data on limitations associated with these numbers, please refer to Dioxin and Dioxin-Like Compounds in Soil, Part II: Technical Support Document for ATSDR Interim Policy Guideline (De Rosa et al., 1997).

REFERENCES


APPENDIX 1 - GLOSSARY

**Action level**

A concentration of chemicals at which consideration of action to interdict/prevent exposure occurs, such as surveillance, research, health studies, community education, physician education, or exposure investigations. Alternatively, based on the evaluation by the health assessor, none of these actions may be necessary.
“At-risk” population A population at a potentially elevated risk due to physiological sensitivity and/or increased exposure to a hazardous chemical.

BDDs Brominated dibenzo-p-dioxins

BDFs Brominated dibenzofurans

CDDs Chlorinated dibenzo-p-dioxins

CDFs Chlorinated dibenzofurans

Comparison value A concentration used to select contaminants of concern at hazardous waste sites that are taken forward in the health assessment process for further evaluation. (The terms comparison value and screening level are often used synonymously.)

Dioxin A term used interchangeably with 2,3,7,8-tetrachlorodibenzo-p-dioxin or TCDD

Dioxin-like compounds Compounds from a group of halogenated aromatic hydrocarbons that have molecules shaped like TCDD and produce similar toxic effects, such as certain other chlorinated dibenzo-p-dioxins (CDDs) and certain chlorinated dibenzofurans (CDFs), polychlorinated biphenyls (PCBs), polybrominated biphenyls (PBBs), brominated dibenzo-p-dioxins (BDDs), and brominated dibenzofurans (BDFs).

Dioxins A term used interchangeably with chlorinated dibenzo-p-dioxins

EMEG An environmental media evaluation guide (EMEG) is a media-specific comparison value that is used to select contaminants of concern at hazardous waste sites.

HazDat ATSDR’s Hazardous Substance Release/Health Effects Database

MRL A minimal risk level (MRL) is an estimate of the daily human exposure to a hazardous substance that is likely to be without an appreciable risk of adverse noncancer health effects over a specified route and duration of exposure.

PBBs Polybrominated biphenyls

PCBs Polychlorinated biphenyls
Screening

The process of initially identifying potentially important chemical contaminants and exposure pathways by eliminating those of known lesser significance.

TCDD

2,3,7,8-Tetrachlorodibenzo-p-dioxin

TEFs

Toxicity equivalency factors (TEFs) are based on congener-specific data and the assumption that the toxicity of dioxin and dioxin-like compounds is mediated by the Ah receptor and is additive. The TEF scheme compares the relative toxicity of individual dioxin-like compounds to that of TCDD, which is the most toxic halogenated aromatic hydrocarbon.

TEQs

Toxicity equivalent (TEQ) is defined as the product of the concentration, $C_i$, of an individual "dioxin-like compound" in a complex environmental mixture and the corresponding TCDD toxicity equivalency factor (TEF) for that compound. The total TEQs is the sum of the TEQs for each of the congeners in a given mixture:

$$\text{Total TEQs} = \sum_{i=1}^{a}(C_i \cdot \text{TEF}_i).$$