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Risk Assessment for Dioxin Contamination Midland, Michigan

FINAL

RISK ASSESSMENT FOR DIOXIN CONTAMINATION AT MIDLAND, MICHIGAN

Second Edition

April 1988

REGION V
U.S. ENVIRONMENTAL PROTECTION AGENCY
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PREFACE

The second edition is identical to the first, which was dated March 1988, except for the following corrections:

Explanatory footnotes have been added on pages III-106 and III-107, and footnotes d and f to Table III-31 on page III-109, have been amended accordingly.

PART I

INTRODUCTION

Chlorinated dibenzo-p-dioxins and dibenzofurans (CDDs and CDFs, respectively; see Appendix C for discussion of nomenclature used in this report) are families of toxic and persistent organic chemicals which are formed as side products in certain commercially significant chemical reactions, and during high temperature decomposition and combustion of certain chlorinated organic chemicals. Over the past decade, the United States Environmental Protection Agency (USEPA) has become increasingly concerned about the presence and significance of environmental levels of CDDs/CDFs. USEPA's initial concern, in the 1970's, was focused on 2,3,7,8-tetrachlorodibenzo-p-dioxin (2378-TCDD) (known to be an impurity in certain chlorinated phenolic chemicals, including the herbicide, 2,4,5-T) which had demonstrated reproductive toxicity and carcinogenic activity in animal systems at very low doses. Many later studies (see Part II) have shown that other compounds sharing the same general structure (dibenzo-p-dioxin or dibenzofuran substituted with two or more chlorine atoms) also share the same general toxic properties of 2378-TCDD, although their degree of toxicity is lower.

In the late 1970's, evidence of environmental contamination with CDDs/CDFs in the Midland area came to light, prompting a series of investigations of the sources and levels of contamination and analyses of the potential risks to the health of individuals living in and around Midland. The investigations were undertaken by the USEPA and the Michigan Departments of Natural Resources

(MDNR) and Public Health (MDPH). The Dow Chemical Company, which operates a large chemical production facility in Midland (the Michigan Division of Dow Chemical U.S.A., hereafter referred to as the Dow Midland facility), was suspected of being the major source of the contamination. Dow Chemical also conducted investigations of dioxins in and around the Dow Midland facility. This report presents the results of analyses conducted by the USEPA of the potential risks associated with exposure to CDDs/CDFs in the environment in and around Midland. In conducting this analysis, the authors have drawn on studies performed by USEPA and its contractors, the State of Michigan, and Dow, in an attempt to synthesize all the available data regarding risks and exposures and to present the most comprehensive assessment possible.

A. History of CDD/CDF Contamination at Midland

The Dow Midland facility is a large chemical manufacturing complex encompassing about 1,500 acres along both banks of the Tittabawassee River at Midland, Michigan (Figure I-1). Throughout its history, Dow Chemical has manufactured over 1,000 different inorganic and organic chemicals at the Midland facility, including cyclic intermediates, industrial organic and inorganic chemicals, plastic materials, synthetic resins, nonvulcanized elastomers, medicinal chemicals, surface active agents, finishing agents, sulfonated oils, insecticides, herbicides, and formulated pesticides. The manufacture of chlorinated phenols and herbicides, and the formulation of pesticides and other products derived from them have been major operations at the Dow Midland facility for many years. Commercial production of chlorinated

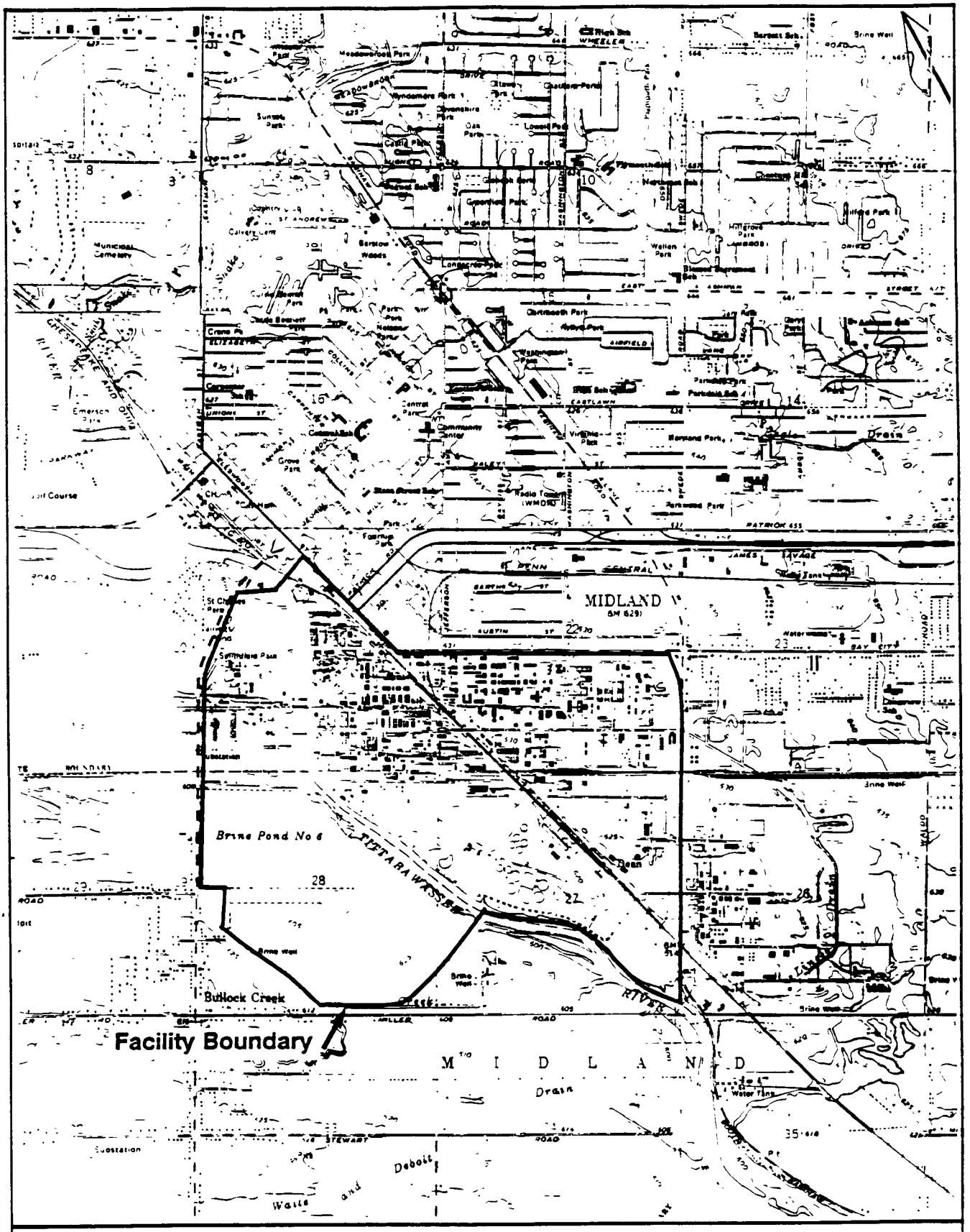

Figure I–1

Midland, Michigan Area and Dow Midland Facility

Sources: USGS (1973), Dow (1984)

phenols began in the 1930's and continued at substantial levels into the 1970's. Dow Chemical reports that only two chlorinated phenolic products are currently manufactured (Dow 1984):

- 2,4-dichlorophenol, and
- 2,4-dichlorophenoxyacetic acid (2,4-D).

Production of all other chlorinated phenolic intermediates and products was terminated in the late 1970's. A list of chlorinated phenolic compounds that have been produced at the Dow Midland facility is presented in Table I-1. The Dow-Corning plant (not known to have been involved in the production of chlorinated phenols or their derivatives) is adjacent to the Dow Midland facility to the east. The main residential and commercial areas of the city are located to the north and the northeast of the Dow Midland facility.

In June 1978, the Dow Midland facility informed the MDNR that rainbow trout exposed to a mixture of Dow Chemical's treated effluent prior to discharge from outfall 031 to the Tittabawassee River accumulated significant levels of 2378-TCDD, the most toxic of the CDD/CDF compounds. Supplemental analyses of edible portions of Tittabawassee River native catfish, previously collected in 1976 downstream of the discharge from the Dow Chemical facility, showed concentrations of 2378-TCDD ranging from 70 to 230 parts per trillion (ppt). Fish collected upstream of the facility, above Dow Dam, did not contain detectable levels of 2378-TCDD. The results of these studies prompted the Michigan Department of Public Health to issue a formal advisory in June 1978 warning against consumption of any fish collected from the Tittabawassee River

TABLE I-1

A COMPILATION OF THE COMMERCIALLY SIGNIFICANT CHLOROPHENOLIC COMPOUNDS REPORTED TO HAVE BEEN MANUFACTURED AT THE DOW MIDLAND FACILITY

Chlorophenols
2-chlorophenol
4-chlorophenol
2,4-dichlorophenol[a]
2,4,5-trichlorophenol
sodium 2,4,5-trichlorophenate
zinc 2,4,5-trichlorophenate
2,4,6-trichlorophenol
sodium tetrachlorophenate
2,3,4,6-tetrachlorophenol
pentachlorophenol
sodium pentachlorophenate
Chlorophenoxy Derivatives[b]
2,4-dichlorophenoxyacetic acid (2,4-D)[a]
2-(2,4-dichlorophenoxy)-propanoic acid
2-methyl-4-chlorophenoxyacetic acid
2,4,5-trichlorophenoxyacetic acid (2,4,5-T)
2-(2,4,5-trichlorophenoxy)-propanoic acid
Other Chlorophenol Derivatives
2-(2,4,5-trichlorophenoxy) ethanol
2-(2,4,5-trichlorophenoxy)-ethyl-2,2-dichloropropanoate
O,O-dimethyl-O-(2,3,5-trichlorophenyl) phosphorothioate
2-cyclopentyl-4-chlorophenol
4-t-butyl-2-chlorophenol
4-t-butyl-2-chlorophenyl-methyl-N-methyl-phosphoramidate
chlorinated phenylphenols
chlorinated diphenyl oxide derivatives

Source: Dow 1984.

[a]2,4-dichlorophenol and 2,4-D are the only compounds from this list that are currently being manufactured on the Midland plant site.

[b]These chlorophenoxy acid derivatives have also been converted into various water soluble salts.

downstream of Dow Dam. (The advisory remained in effect until March 1986, when the Department of Health modified it to apply only to catfish and carp.)

In response to the Dow Chemical findings, the MDNR and USEPA, Region V undertook a number of investigations during the period 1978-1981 to determine whether, or to what extent, the Dow Chemical operations at Midland contributed to 2378-TCDD contamination in Tittabawassee River fish. These investigations included a caged fish bioaccumulation study and an experimental large volume wastewater effluent sampling program conducted in September 1981. The results of those studies conclusively demonstrated that the Dow Chemical wastewater effluent was a significant source of 2378-TCDD to the Tittabawassee River. The preliminary results from those studies were released in March 1983 with a series of recommendations for more comprehensive dioxin studies in Midland and elsewhere. Most of those recommendations were subsequently incorporated into USEPA's Dioxin Strategy and National Dioxin Study (USEPA 1983b, 1987a)

Also, in March 1983, the State of Michigan made a formal request to the then acting administrator of USEPA for assistance in conducting a comprehensive multi-media investigation of dioxin emissions and discharges from Dow Chemical and dioxin contamination in the Midland area. In the spring and summer of 1983, Region V collaborated with the Michigan Departments of Agriculture, Natural Resources, and Public Health, and the Michigan Attorney General's Office in planning for the requested studies. At about the same time local environmental groups petitioned USEPA pursuant to Section 8(e) of the Toxic Substances Control Act for broad scale toxic pollutant investigations of an eight-county area in mid-Michigan including Midland County. Although USEPA

subsequently denied that petition, some of the requested investigations were within the scope of those being planned by Region V and the state agencies.

The studies conducted by USEPA and the State were formally called the Michigan Dioxin Studies and included the following major programs:

1. Supplemental native fish and sediment sampling in the Tittabawassee River.
2. Surface soil sampling at the Dow Chemical facility, in the City of Midland, and in comparison sites.
3. Evaluation of public and private potable water supplies and Dow Chemical brine operations.
4. Supplemental Dow Chemical wastewater and sewer system sampling.
5. Incinerator emissions and limited ambient air monitoring.

These investigations included analyses of dioxins and other toxic pollutants that might be present, and were consistent with the then-evolving USEPA Dioxin Strategy. Since the Dow Chemical Plant was considered to have operations within Tiers 1, 2, 3, 4, and 6 of the Dioxin Strategy, funding for the studies was provided principally through the CERCLA (or Superfund) program. All Tier 1 and 2 facilities in the Dioxin Strategy were studied through Superfund.

In 1983, Dow Chemical initiated its own independent point source investigation of dioxins at the Midland Plant. That work included comprehensive surface soil sampling at the plant, untreated and treated process wastewater sampling, incinerator emissions testing and limited ambient air

monitoring. Dow Chemical has also conducted supplemental incinerator emissions testing in 1987, supplemental monitoring of Tittabawassee River fish in response to a consent order with USEPA, and twice monthly monitoring for 2378-TCDD in the process wastewater discharge to the Tittabawassee River.

Studies by Dow and USEPA revealed widespread contamination of the surface soil at the Midland facility (average of 0.5 ppb 2378-TCDD). Several small areas within the facility were found to be highly contaminated (2-50 ppb). USEPA studies indicated lower level contamination of the soils throughout the community, with CDDs/CDFs (average <0.1 ppb 2378-TCDD). Since these studies were undertaken, Dow has been ordered to remediate areas of high onsite contamination to prevent the spread of contaminated soil. The source of the on-site soil contamination appears to have been a combination of leaks or fugitive emissions from one or more of the production processes discussed above and fallout from the waste incinerator. The off-site soil contamination has been attributed to airborne emissions of CDDs/CDFs from the waste incinerator, wind-borne transport of contaminated soil from the facility, and possibly past fugitive emissions from production operations.

B. Risk Assessment Structure and Methods

The USEPA has compiled the data from its testing program (USEPA 1985a, Barna and Amendola 1985, Amendola and Barna 1986, Trembly and Amendola 1987) and, in this document, presents its assessment of these data as they reflect on

the risks posed by the CDDs/CDFs in the areas sampled. This report builds on preliminary studies prepared by other offices (Clark 1985, Cleverly 1986).

This document follows the conceptual framework for risk assessment articulated by the USEPA Guidelines for Carcinogen Risk Assessment (USEPA 1986a) and Guidelines for Estimating Exposures (USEPA 1986b). It is also consistent with the 1983 report of the National Academy of Sciences (NAS), "Risk Assessment in the Federal Government: Managing the Process" (NRC 1983). As envisioned by the NAS and USEPA Guidelines, risk assessment contains four parts: hazard identification, dose-response assessment, exposure assessment, and risk characterization. In keeping with this scheme, Part II of this report contains a brief summary of the Hazard Identification and Dose-Response Assessment of CDDs/CDFs, referring to other agency documents for elaboration. Part III describes the site-specific Exposure Assessment for each of the contaminated media, based on the data derived from the USEPA field investigations in Midland, and Part IV, Risk Characterization, integrates the information from Parts II and III to develop quantitative estimates of risks faced by the exposed populations. Part III also includes a discussion of the uncertainties associated with the estimates of exposure, and Part IV includes a discussion of the uncertainties associated with the estimates of risk.

PART II

HAZARD IDENTIFICATION AND DOSE-RESPONSE ASSESSMENT

The USEPA and other organizations have compiled and evaluated the existing toxicological data on CDDs and CDFs (e.g., USEPA 1984a, 1985b, Ontario Ministry of the Environment (Ontario) 1984). Although there is extensive literature on some of these compounds, the toxicological information on these families of more than 200 compounds is far from complete. Nevertheless, a growing body of information on mechanisms of action and structure-activity relationships within these families of compounds makes it possible, with reasonable confidence, to infer information where data are missing.

Among the 210 congeners of CDDs and CDFs, the compound that appears to be the most toxic and has generally raised the greatest health concerns is 2,3,7,8-tetrachlorodibenzo-p-dioxin, abbreviated as 2378-TCDD. Experimental studies with 2378-TCDD in animal systems have demonstrated a variety of toxic effects resulting from exposure to this compound (USEPA 1985b). These effects include carcinogenesis, cancer promotion, reproductive and teratogenic effects, immunotoxic effects, thymus atrophy, liver damage, and effects on the skin and thyroid. Acute exposures of sensitive species of animals to 2378-TCDD result in a characteristic "wasting syndrome," followed by death. Extensive experimental studies have revealed marked variations among species in both the array of effects caused by 2378-TCDD and the dose levels at which these effects are elicited (USEPA 1985b, Pitot et al. 1986). Limited toxicological testing of other CDDs/CDFs has demonstrated that several of these compounds cause similar toxicological effects, but that higher doses of these compounds are

generally required to cause effects of comparable magnitude to those induced by 2378-TCDD.

The nature and extent of effects in humans exposed to 2378-TCDD are not nearly so well defined (USEPA 1985b, Ontario 1984, Pitot et al. 1986). There is a consensus that exposure of humans to 2378-TCDD can result in a skin condition known as chloracne, an acne-like lesion which, while not life-threatening, can be disfiguring, persistent, and refractory to treatment. Several studies of human populations exposed to chemical mixtures containing 2378-TCDD have suggested increased frequencies of certain cancers (e.g., Hardell and Sandstrom 1979, Hardell et al. 1981, Thiess et al. 1982, MDPH 1983a, Hoar et al. 1986), but inconsistencies among the studies and incomplete characterization of exposure make the evidence, taken as a whole, inconclusive (USEPA 1985b, Blair 1986). Evidence for reproductive impairment in humans exposed to 2378-TCDD (including one study conducted in Midland County: MDPH 1983b) is inconclusive for similar reasons (USEPA 1985b, Kimbrough 1986). Other effects in humans that have been more clearly associated with exposure to mixtures containing 2378-TCDD include disturbances in lipid metabolism (Moses et al. 1984, Suskind and Hertzberg 1984) and increased frequency of gastric ulcers (Bond et al. 1983, Suskind and Hertzberg 1984).

More specific and quantitative information is available on toxic effects of CDFs in humans, as a result of two large-scale poisoning incidents in Japan and Taiwan (Kuratsune and Shapiro 1984). The affected persons ingested, over periods of weeks to months, food contaminated with a mixture of CDFs, polychlorinated biphenyls (PCBs) and polychlorinated quaterphenyls (PCQs).

Comparative toxicological studies have indicated that CDFs were the primary toxic agents in these poisoning incidents and that 23478-PeCDF was probably the most important single compound (Masuda and Yoshimura 1984, Kunita et al. 1984, 1985, Bandiera et al. 1982, Masuda et al. 1985, Chen et al. 1985, Miyata et al. 1985). The most prominent toxic signs were skin eruptions similar to those of chloracne, along with skin pigmentation and eye abnormalities (Lu and Wong 1984, Urabe and Asahi 1985). Other effects reported include impairments in lipid metabolism and immune function (Okumura et al. 1974, Chang et al. 1980, 1982) and persistent respiratory symptoms (Nakanishi et al. 1985). A preliminary report by Kuratsune et al. (1987) indicates a significant excess frequency of liver cancer and possibly lung cancer among male victims within 15 years after exposure. Reported effects on reproduction include menstrual disturbances (Kusuda 1971), skin hyperpigmentation in infants (Yamashita and Hayashi 1985, Hsu et al. 1985), and perinatal mortality (Hsu et al. 1985). These effects observed in humans are qualitatively similar to those reported in animals exposed to CDFs and CDDs (McNulty 1985); this provides support for the use of animal data as the basis for hazard assessment for other members of these families of compounds.

USEPA has determined that the critical end points of concern for purposes of assessing risk associated with exposure to CDDs/CDFs from the Midland facility are cancer and reproductive and teratogenic effects. This portion of the Risk Assessment briefly summarizes the evidence for these effects for 2378-TCDD (and, in a few cases, other CDDs or CDFs) and discusses how these data have been used to generate quantitative measures of toxic potency for use in Dose-Response Assessment. The concluding section discusses how these

results have been extended to include other CDDs/CDFs which have not been as extensively tested.

A. Cancer

1. Hazard Identification for 2378-TCDD

The USEPA Health Assessment Document on CDDs (USEPA 1985b) summarized the results of several long-term animal studies in which 2378-TCDD has been investigated as a possible carcinogen. The principal studies provide clear evidence for the conclusion that 2378-TCDD is an animal carcinogen (Kociba et al. 1978, NTP 1982a, NTP 1982b). These data show that exposure of rats and mice to 2378-TCDD at very low doses is related to the development of tumors at a variety of sites, principally and most consistently in the liver.

On the basis of these animal studies and associated factors, such as short-term tests and structure/activity considerations, USEPA has concluded that 2378-TCDD should be regarded as a potential human carcinogen (USEPA 1985b). This substance has been assigned a designation of "B2" in USEPA's scheme of categories for qualitative weight-of-evidence of carcinogenic potential. This designation is given to agents for which there is "sufficient" evidence of carcinogenicity based on animal studies and "inadequate" data regarding carcinogenicity from human epidemiologic studies (USEPA 1986a).

2. Dose-Response Assessment for 2378-TCDD

USEPA (1985b) has developed a Dose-Response Assessment for 2378-TCDD based upon data from the study by Kociba et al. (1978). The procedures used by USEPA are in keeping with its recently published Cancer Risk Assessment Guidelines (USEPA 1986a) which are consistent with the Cancer Principles laid out by the Office of Science and Technology Policy in 1985 (OSTP 1985). Briefly, USEPA employed the linearized multi-stage (LMS) model to estimate an upper bound on the excess lifetime cancer risk at doses below those used in the animal experiment. In order to extrapolate from dose-response data in animals to predict human risk, USEPA used its standard procedure of adjusting relative doses on a body surface area basis, reflective of relative metabolic rate (USEPA 1985b).

Applying these procedures, USEPA (1985b) used the experimental animal data to estimate an upper bound on the cancer potency factor for 2378-TCDD. The cancer potency factor is equivalent to the slope of the projected linear dose-response curve in the low-dose region, adjusted to apply to humans. The upper bound on the cancer potency factor estimated for 2378-TCDD (designated and referred to as q_1^*) is 1.6×10^5 $(mg/kg\text{-}d)^{-1}$. The actual potency is not likely to exceed this upper bound estimate, formally referred to as the upper 95% confidence limit (UCL).

In recent years, several alternative approaches to carcinogenic risk assessment for 2378-TCDD have been presented by scientists or regulatory agencies, both in the U.S. (e.g., Miller 1983, Kimbrough et al. 1984, Portier

et al. 1984, MDH 1985, MDPH 1986, Hoel 1986, Sielken 1987, Shu et al. 1987, Thorslund et al. 1987) and in other countries (e.g., Ontario 1984, FRG 1984). Most of these assessments remain unpublished and have not been peer-reviewed. In general, they differ from the USEPA dose-response assessment in one or both of two respects:

- Several assessments that utilized the linearized multi-stage model incorporated different data or made different assumptions about the way in which the data should be used. Examples include the use of different sets of tumor data as the basis for extrapolation (Kimbrough et al. 1984, Portier et al. 1984), the use of tissue concentrations as measures of dose (Portier et al. 1984), the use of mg/kg body-weight scaling (Miller 1983, Kimbrough et al. 1984, MDH 1985, MDPH 1986), or the use of different ways of averaging lifetime dose (Kimbrough et al. 1984). The most important of these differences is the use of mg/kg body-weight scaling, which results in a human cancer potency factor about 6 times lower than that derived from body-surface-area scaling. Primarily for this reason, estimates of cancer potency developed by other U.S. agencies (including the Centers for Disease Control, the Food and Drug Administration, and the States of Michigan and Minnesota) have ranged from a value near to the USEPA value to a value about one order of magnitude less potent (Kimbrough et al. 1984, FDA 1983, MDH 1985, MDPH 1986). Although the selection of an interspecies scaling factor is a matter for scientific judgment, the greater retention time of 2378-TCDD in humans than in rats provides a rationale for the selection of the more "conservative" body-surface-area scaling factor used by USEPA.

- Several assessments have been based on the assumption that 2378-TCDD acts primarily as a cancer promoter, and on the further assumption that cancer promotion is a reversible phenomenon with a threshold-type dose-response relationship. On the basis of these assumptions, "acceptable" daily intakes for 2378-TCDD have been proposed by applying "Uncertainty Factors" to dose-levels thought to be "Lowest-Observed-Adverse-Effect-Levels" (Ontario 1984, FRG 1984, Shu et al. 1986). Although there is evidence that 2378-TCDD is a potent promoter and has little propensity to interact with DNA in the manner of a classical cancer initiator (Pitot et al. 1986), currently available evidence on mechanisms of cancer promotion does not support the assumption that promoting activity would be reversible and have a threshold-type dose-response relationship (Upton et al. 1985, Weinstein 1984, 1987, Yamasaki and Weinstein 1985, Gallagher 1986). Goodrow et al. (1986) have reported that cancer promotion by 2378-TCDD is associated with its binding to receptors associated with the Ah gene locus and receptors for epidermal growth factor. Other studies have suggested that binding to one or both of these receptors results in activation of certain genes (Israel and Whitlock 1984, Whitlock et al. 1984, Jones et al. 1985, 1986a,b). There is no

evidence that these molecular mechanisms would necessarily be reversible and would display threshold-type dose-response relationships. Even if receptor binding is assumed to be reversible, the fact that 2378-TCDD is more strongly retained in human tissues than in those of other animals would have to be taken into account (Hoel 1986). Finally, the promoting effects of 2378-TCDD might augment risks resulting from prior human exposure to initiating carcinogens. At present, there are no accepted models that can be used to predict low-dose risks resulting from these effects of 2378-TCDD. Thorslund et al. (1987) have presented preliminary results of a model in which 2378-TCDD is assumed to act by causing proliferation of initiated cells, but it has not been demonstrated that this approach accurately reflects the biochemical mode of action of 2378-TCDD in cancer causation.

For the above reasons, it remains appropriate to use the dose-response assessment for 2378-TCDD derived by USEPA (1985b), based on the linearized multistage model (LMS) with body-surface-area scaling. Portier et al. (1984) have reported that available dose-response data fit a linear model if tissue concentration is used as a measure of dose. USEPA recognizes, however, that use of the LMS model is controversial at the present time; dose-response assessment for carcinogenic effects of 2378-TCDD is currently under review by the Agency, and this review may lead to revision of the cancer potency factor.

Ongoing work on mechanisms of action (Jones et al. 1986a,b, Goodrow et al. 1986), pharmacokinetics (Leung et al. 1987, Van den Berg and Poiger 1987), and mathematical modeling (Thorslund et al. 1987) will eventually help to resolve the controversies surrounding cancer risk estimates for 2378-TCDD. Pending this resolution, it should be recognized that these features of the biological activity of 2378-TCDD add substantial uncertainty to risk estimates derived from the LMS model. These estimates are intended to represent upper bounds on risk and will be reported as such. Even as upper bounds, however, they could be too high (e.g., if the dose-response relationship is strongly non-linear) or

too low (e.g., if CDDs/CDFs act to promote cancers initiated by other widespread environmental carcinogens).

B. Reproductive and Teratogenic effects

1. Hazard Identification for 2378-TCDD

2378-TCDD has been shown to be teratogenic in all strains of mice which have been tested. Further, this compound has produced teratogenic and fetotoxic effects in all strains of rats tested. Reproductive effects have been demonstrated in other species as well, including subhuman primates (USEPA 1985b).

For reproductive effects, USEPA has focused on a three-generation rat feeding study (Murray et al. 1979) as the critical study for estimating the non-cancer risk posed by 2378-TCDD. The Centers for Disease Control (CDC) have cited a reproductive study in monkeys (Allen et al. 1979) as the critical study (Kimbrough et al. 1984). USEPA (1985b) also cited this study, as well as another report on the same research (Schantz et al. 1979) in support of their findings. For teratogenic effects, the critical study is a study in rats treated with 2378-TCDD, administered daily by gavage on days 6-15 of gestation (Sparschu et al. 1971).

2. Dose-Response Assessment for 2378-TCDD

In assessing toxic effects produced by non-carcinogens (i.e., "systemic toxicants"), USEPA has adopted the concept of the Reference Dose (RfD) (USEPA 1987b). The RfD is operationally defined as the "no observed adverse effect level (NOAEL)" (i.e., the highest dose level at which no adverse effects were observed in an experiment using an adequate number of test animals) determined in the critical toxicological study, divided by an "Uncertainty Factor (UF)" which is selected on the basis of specific attributes of the data base. (In some cases, an additional modifying factor (MF) is introduced to account for peculiarities in the data base.) The RfD can be defined as an estimate (with uncertainty spanning perhaps an order of magnitude) of the daily exposure to the human population (including sensitive subpopulations) that is likely to be without an appreciable risk of deleterious effect during a lifetime. The RfD supersedes, and is generally equivalent to, the Acceptable Daily Intake (ADI) values previously used by USEPA and other agencies to define dose levels for non-cancer endpoints.

There has been some debate as to whether or not a dose of as little as 1 ng/kg-d (1000 pg/kg-d) of 2378-TCDD was a NOAEL in the three-generation reproductive study in the rat (Murray et al. 1979, Nisbet and Paxton 1982, Kimbrough et al. 1984, USEPA 1985b). USEPA has examined this study in detail and selected a combined UF of 1000, (including subfactors of 10 because the lowest administered dose was not a NOAEL, 10 to account for possible interspecies differences in sensitivity, and 10 to account for possible intraspecies differences in sensitivity) such that an RfD of 1 pg/kg-d is

derived (USEPA 1987c). USEPA (1985b, 1987c) also placed weight on the study by Schantz et al. (1979), which reported adverse reproductive effects in rhesus monkeys exposed to 2378-TCDD at about 1.5 ng/kg-d, leading to a similar value for the RfD. As noted above, the CDC selected a different critical study in deriving their functional equivalent of the RfD, but the CDC scientists obtained essentially the same value as USEPA, i.e., 1-2 pg/kg-d (Kimbrough et al. 1984).

In addition to effects associated with low level, long-term exposures to CDDs and CDFs, USEPA is also concerned about relatively large doses which pregnant women might ingest at a critical time of organogenesis in the development of the fetus. A rat gavage study (Sparschu et al. 1971) yielded a NOAEL of 30 ng/kg-d for these teratogenic effects. USEPA has adopted procedures to issue "health advisories" (HAs) for exposures associated with such less-than-lifetime situations. These standard procedures lead to selection of an UF of 100 (Kimmel 1987), and hence to a "health advisory" dose-level of 300 pg/kg-d for protection against teratogenic effects. This HA dose-level is appropriate for comparison with single-dose or single-day intakes, whereas the RfD of 1 pg/kg-d is more appropriate for comparison with long-term or lifetime exposures (see below for further discussion).

Dose-response data for reproductive/teratogenic effects of 2378-TCDD are well established, and there is a reasonably sound basis for the establishment of the RfD and the HA. The reports of reproductive impairment in humans exposed to CDFs add qualitative support for the use of these data in risk assessment. However, one additional factor may need to be considered.

Bowman et al. (1987a,b) have reported apparently adverse reproductive effects in rhesus monkeys exposed to 2378-TCDD at a dose rate of about 0.125 ng/kg-day, a factor of 8-10 lower than the previous LOAELs. These reports are presently available only in abstract form; if confirmed, they may require downward revision of the RfD.

3. Other Toxic Effects

Although USEPA has determined that reproductive/teratogenic effects are the critical, noncarcinogenic toxic effects for dose-response assessment of 2378-TCDD, some uncertainty may arise if dose-response data for such effects are used in risk assessment for persons of non-reproductive age (e.g., for children or post-menopausal women) or persons who are not reproducing for other reasons. The RfD is probably applicable to children, both because 2378-TCDD is retained for periods of years in the body and hence may exert effects long after exposure occurs, and because germ cells in females are subject to exposure at any time after they are formed during embryonic development. However, it is not clear that the HA based on teratogenic effects can be applied directly to any population group except pregnant women. To reduce such uncertainty, it is desirable to consider data on other toxic end points of 2378-TCDD. This section briefly considers dose-response data for other toxic effects of 2378-TCDD, based on the literature review by USEPA (1985b).

Chronic toxicity studies have been conducted in non-human primates, and in rats and mice. In studies in rhesus monkeys, exposure to a dietary concentration of 50 ppt 2378-TCDD (about 1.5 ng/kg-day) resulted in hair loss,

edema and pancytopenia (Schantz et al. 1979). In one study in mice, exposure by gavage to doses of 10 ng/kg-week 2378-TCDD resulted in a significant increase in the incidence of toxic hepatitis (NTP 1982a); in another study in mice, exposure by gavage to doses of 7 ng/kg-week 2378-TCDD resulted in skin lesions and amyloidosis of the kidney, liver, and spleen (Toth et al. 1978, 1979). All three of these dose-levels were Lowest-Observed-Adverse-Effect Levels (LOAELs); No-Observed-Adverse-Effect Levels (NOAELs) have not been reported for chronic exposure to 2378-TCDD in these species (USEPA 1985b). In rats, USEPA (1985b) reported that 1 ng/kg-day was a NOAEL, but the study on which this conclusion was based (Kociba et al. 1978) actually reported a statistically significant increase in foci or larger areas of slight hepatocellular alterations in female rats at this dose level. On the basis of the studies cited in this paragraph, doses in the range 1-1.5 ng/kg-day should be regarded as LOAELs for these effects of 2378-TCDD in animals. This is the same range of doses as that cited above as LOAELs for reproductive effects in animals. Hence, it is appropriate to apply the RfD of 1 pg/kg-day to all individuals in the human population, regardless of their reproductive status.

For acute or subchronic effects of 2378-TCDD, USEPA (1987c) has cited a study by Turner and Collins (1983) as the critical study for dose-response assessment. In this study, a single dose of 100 ng/kg administered to female guinea pigs was a LOAEL, causing histopathologic changes in the liver. USEPA (1987c) used this LOAEL to derive One-day and Ten-day Health Advisories, by applying Uncertainty Factors of 100 and 1,000, respectively. These SAB-reviewed HAs are equivalent to intakes of 280 pg/kg (single dose) and 28 pg/kg-day (for 10 days), respectively. The former HA is very close to that derived

above on the basis of teratogenic effects, justifying the application of this HA to population segments other than pregnant women. The latter HA provides a basis for risk assessment for subchronic exposures (ranging in duration from a few days to a few weeks).

In general, RfDs are based on studies involving lifetime exposure of animals and are formally defined for comparison with lifetime average dose rates in humans (USEPA 1987b). In the case of 2378-TCDD, the RfD is based on a three-generation reproductive study in which rats were exposed for two reproductive cycles, and another study in which rhesus monkeys were exposed for only 7 months yielded a similar LOAEL (see above). Hence, it is appropriate to compare this RfD with dose-rates for less-than-lifetime exposure in humans. In Chapter IV of this report, the RfD for 2378-TCDD will be compared with average dose rates for human exposure lasting for several months or longer; the 10-day HA will be compared with average dose-rates for human exposure lasting for a few days to a few weeks; the 1-day HA will be compared with single-dose or single-day intakes.

Although dose-response data for liver effects yield HAs very similar to those for reproductive/teratogenic effects, one additional factor should be considered. Immunotoxic effects of 2378-TCDD have been reported at very low doses (1 ng/kg-week) by Clark et al. (1983). Reports of immune system impairment in humans exposed to CDFs (Chang et al. 1982a,b) and perhaps in humans exposed to 2378-TCDD (Hoffman et al. 1986, but see Evans et al. 1987) suggest that the findings of Clark et al. (1983) are relevant to assessment of potential human risks. However, there is no precedent or accepted procedure

for the use of immunotoxicity data in establishing RfDs or HAs. Along with the preliminary data reported by Bowman et al. (1987a,b), the results of Clark et al. (1983) suggest that there may be some risk resulting from dose rates at or even below the RfD or HAs for other toxic effects.

C. Hazard Identification and Dose-Response Assessments for Mixtures of CDDs/CDFs, Including 2378-TCDD

While the toxicological properties of 2378-TCDD have been reasonably well characterized, the toxicological data base for the other CDDs and CDFs is limited. This section summarizes the limited testing of other CDDs and CDFs for carcinogenicity, cancer promotion and/or teratogenicity.

1. Carcinogenicity

Only six CDD/CDF congeners other than 2378-TCDD have been tested for carcinogenic activity. In a study reported by NCI (1980), a mixture of the two 2378-substituted-HxCDDs induced liver tumors in rats and mice. Based on this study, USEPA (1985b) assigned this mixture to the category "B2" in USEPA's qualitative weight-of-evidence scheme (see definition above) and calculated a cancer potency factor of 3.9×10^4 $(mg/kg\text{-}d)^{-1}$. In another study conducted by NCI (1979), suggestive evidence was found for the carcinogenicity of 27-DCDD when administered at high doses to male mice. In a two-stage bioassay for cancer promotion on the skin of hairless mice, 2378-TCDF was found to be a potent cancer promoter, but was about 20 times less potent than 2378-TCDD which

was tested in the same study (Poland and Knutson 1982, Poland et al. 1983). In a two-stage bioassay for cancer promotion in rat liver, 23478-PeCDF and 123478-HxCDF were found to be potent cancer promoters, the former being more potent (Nishizumi and Masuda 1986).

2. Reproductive/Teratogenic Effects

Only limited testing for teratogenic effects and no testing for other reproductive effects has been conducted with other CDDs and CDFs. Several studies have shown that 2378-TCDF induces cleft palates and hydronephrosis in fetal mice when administered on days 10-13 of gestation (Weber et al. 1984, Hassoun et al. 1984, Krowke 1986). Krowke (1986) reported that 12378-PeCDD and 123478-HxCDD also caused cleft palates in mice exposed in utero. Birnbaum et al. (1987a,b) reported that 12378-PeCDF, 23478-PeCDF, and 123478-HxCDF also caused cleft palates and hydronephrosis in mice exposed in utero. All these effects were similar to those induced by 2378-TCDD in the same or in parallel experiments, but 2378-TCDD was the most potent of the compounds tested in all these respects.

3. Other Toxic Effects

A somewhat larger number of CDDs and CDFs has been tested for acute and subacute toxic effects, primarily on the liver and thymus (McKinney and McConnell 1982, Mason et al. 1985, 1986a,b, Safe 1986). These studies have generally shown that most CDDs and CDFs cause similar effects to those caused by 2378-TCDD in the same bioassay systems, but that 2378-TCDD is the most

potent congener among those tested to date in all systems. Further, these studies have shown structure-activity relationships within both families of compounds, with a general parallelism between relative potencies in in vivo and in vitro bioassays (Safe 1986). The results of these studies have suggested a general approach to risk assessment for these compounds, which can be applied to complex mixtures of the type commonly found in the environment.

4. Toxicity Equivalence Factors

Given the lack of information on most of the CDDs and CDFs at a time when reports of these compounds in the environment increasingly call for some type of interpretation, USEPA has adopted an interim science policy position for assessing risks of CDDs/CDFs other than 2378-TCDD (USEPA 1987d, Thomas 1987). The procedure is called the "toxicity equivalence factor (TEF)" approach and, in the process of gaining USEPA acceptance, underwent internal and external USEPA review, including examination by the USEPA's Science Advisory Board (SAB 1986). It has been adopted by USEPA as an interim procedure to be used until sufficient additional data are available to derive a more accurate procedure that can be scientifically validated. The TEF approach is based on the similarity of structure and activity seen in the behavior of members of the CDD/CDF family. This similarity is used as the basis for estimating the toxicity, with regard to both carcinogenic and non-carcinogenic endpoints, of any CDD/CDF mixture in terms of an equivalent amount of 2378-TCDD. Within each homologue group, distinction is made between those CDD/CDF congeners which are "2378-substituted" (i.e., substituted with chlorine at the lateral 2, 3, 7, and

8 positions; see Appendix C) and those which are not, and a common TEF is applied to all congeners in each category as shown in Table II-1. Structure-activity studies have shown that 2378-substituted congeners are more potent in a number of assays than non-2378-substituted congeners (Poland et al. 1979, Mason et al. 1985, 1986a,b, Safe 1986, USEPA 1985b) and the former are assigned much higher TEFs (USEPA 1987d). In cases where analytical procedures identify CDDs/CDFs only to the homologue level and do not distinguish between 2378-substituted and non-2378-substituted congeners, the USEPA's interim procedure (USEPA 1987d) proposes two alternative procedures:

A. Assume that all CDDs/CDFs are 2378-substituted and apply the TEF for 2378-substituted congeners to the total quantity of each homologue reported; or

B. Assume that all CDD/CDF congeners are equally likely to occur and allocate congeners to 2378-substituted and non-2378-substituted categories in proportion to the numbers of each type of congener within each homologue group.

The "A-method" yields an "upper bound" estimate of risk. The "B-method" typically leads to an estimate of risk about an order of magnitude lower, depending upon the exact mixture of congeners and the quality of the data regarding the amounts of specific congeners present (USEPA 1987d); it is appropriate as an alternative method of risk estimation, but does not necessarily yield a reliable point estimate or "most likely" case because conditions of formation, persistence, or bioaccumulation may favor unequal distributions of congeners.

The TEF approach is used in Part III of this report to convert reported quantities of CDDs/CDFs in environmental samples to equivalent quantities of 2378-TCDD. The resulting concentrations of "2378-TCDD toxicity equivalents"

TABLE II-1

TOXICITY EQUIVALENCE FACTORS (TEFs) FOR CDDs/CDFs

Congener Group[a]	TEF	Proportion of Homologue[b]
Total TCDDs	1	1
2378-TCDDs	1	0.05
other TCDDs	0.01	0.95
Total PeCDDs	0.5	1
2378-PeCDDs	0.5	0.07
other PeCDDs	0.005	0.93
Total HxCDDs	0.04	1
2378-HxCDDs	0.04	0.3
other HxCDDs	0.0004	0.7
Total HpCDDs	0.001	1
2378-HpCDDs	0.001	0.5
other HpCDDs	0.00001	0.5
Total TCDFs	0.1	1
2378-TCDFs	0.1	0.03
other TCDFs	0.001	0.97
Total PeCDFs	0.1	1
2378-PeCDFs	0.1	0.07
other PeCDFs	0.001	0.93
Total HxCDFs	0.01	1
2378-HxCDFs	0.01	0.25
other HxCDFs	0.0001	0.75
Total HpCDFs	0.001	1
2378-HpCDFs	0.001	0.50
other HpCDFs	0.00001	0.50

[a]TEFs for all congener groups not listed here are zero.

[b]Proportion of congeners within the homologue group that falls into this subgroup; this proportion is used in calculating TEQs by the "B-method" (see text).

SOURCE: USEPA 1987d.

(TEQs) are summed over all congeners present in the mixture, and are then treated as if they were concentrations of 2378-TCDD itself. The "A-method" is used to yield maximum estimates of TEQs; the "B-method" is also applied (except for exposure via fish) for comparative purposes.

The TEF procedure incorporates a number of assumptions with varying scientific basis and degree of validation; these assumptions are listed below with comments on their basis and limitations.

1. All CDD/CDF congeners have the same mechanism of action and cause the same spectrum of toxic effects; there is an extensive empirical basis for this assumption, at least for mechanisms of action and acute toxic effects (Safe 1986, USEPA 1987d).

2. The relative potencies of the CDD/CDF congeners are similar for different toxic effects, so that measures of relative potency derived from in vitro or short-term in vivo tests can be used to predict relative potencies for the critical toxic effects used in risk assessment; there is a fairly extensive empirical basis for similarity in relative potencies between in vitro and short-term in vivo measures of activity (Safe 1986); only a few CDD/CDF congeners have been tested for carcinogenicity and teratogenicity, but the results of these tests are consistent with the assumption (see references cited above).

3. The effects of different CDD/CDF congeners are additive; two in vitro studies (Sawyer et al. 1983, Safe et al. 1986) and one teratogenicity study (Krowke 1986) provide very limited support for this assumption, although two other teratogenicity studies (Weber et al. 1985, Birnbaum et al. 1987b) suggested synergistic action.

4. Within each congener group, all 2378-substituted congeners have similar relative potencies; however, available studies actually suggest moderate variability, sometimes by an order of magnitude (Poland et al. 1979, Knutson and Poland 1981, Mason et al. 1985, 1986a,b, Safe 1986).

5. All CDD/CDF congeners with 1-3 chlorine atoms substituted at any position have negligible biological activity; available studies suggest a low level of activity, at least for 237-substituted congeners (NCI 1979, Knutson and Poland 1981, Mason et al. 1985).

6. In cases where congener-specific analyses are not available, the "A-method" provides a reasonable upper bound estimate on TEQ, and the "B-method" provides an informative alternative; this assumption has been discussed above.

Because of the limited validation available for these assumptions, the TEF procedure is recognized to yield risk estimates with a substantial degree of uncertainty; however, it is believed that the estimates of TEQ are generally reliable to within at least to order of magnitude (USEPA 1987d). Uncertainties arising from specific features of the data for the Midland site will be discussed in Part IV.

D. Risk Assessments of CDD/CDF Mixtures

When applied to analytical data on a CDD/CDF mixture, the TEF procedure yields an estimate of TEQ, i.e., the Toxicity Equivalent Quantity of 2378-TCDD. This is then combined with dose-response data on 2378-TCDD (specifically, the cancer potency factor and the RfD and HAs) and exposure estimates to yield estimates of health risks faced by individuals exposed to the mixture. In interpreting the results of this procedure, several additional factors should be taken into account:

1. The dose-response assessment for 2378-TCDD incorporates certain assumptions about scaling of doses between species; specifically, it uses body-surface-area scaling for carcinogenic effects and a scaling factor (incorporated into the uncertainty factors) of 10 for reproductive, teratogenic, and other toxic effects. There are no dose-response data for 2378-TCDD with which these factors can be validated. However, for oculodermatological effects of CDFs, effective doses reported for humans (Hayabuchi et al. 1979, Hsu et al. 1985) are similar to those reported for rhesus monkeys (McNulty et al. 1981, Yoshimura et al. 1981).

2. The dose scaling factors and uncertainty factors used in the dose-response assessments for 2378-TCDD take no account of differential pharmacokinetics. In fact, preliminary data suggest that biological half-lives for 2378-TCDD and other CDDs/CDFs are much longer (on the order of years) in humans than in laboratory animals (on the order of weeks) (Poiger and Schlatter 1986, Kunita et al. 1984, Neal et al. 1982). This suggests that humans may be subject to proportionately larger internal exposure, so that risks may be greater than those predicted using the current dose-response assessments for 2378-TCDD.

3. Recent data suggest that the U.S. population has substantial body burdens of CDDs/CDFs, including 2378-TCDD (Rappe et al. 1986, USEPA 1986c, Ryan 1986, Graham et al. 1986). Additional site-related exposures would be expected to add to these pre-existing internal exposures, and this should be taken into account in interpreting the risk assessments. For carcinogenic effects, site-related risks will be additive to those resulting from other sources of exposure. For noncarcinogenic effects, the RfD approach incorporates the concept of a threshold exposure level; in the presence of pre-existing body burdens, smaller incremental exposures will be required to exceed the threshold, so that doses below the RfD may give rise to adverse effects. In other words, the RfD should be compared with total exposure (background exposure and site-specific exposure) for purposes of risk assessment.

Taken together, these factors suggest that USEPA's procedures for risk assessments of CDDs/CDFs may not be particularly "conservative." These procedures incorporate upper bounds on carcinogenic risk, body-surface-area scaling factors, and relatively high Uncertainty Factors, which are generally designed to avoid underestimation of risks in areas of uncertainty (USEPA 1986b, 1987b). In the case of CDDs/CDFs, however, the factors discussed above may offset to some degree the "conservatism" of USEPA's standard risk assessment procedures.

PART III

EXPOSURE ASSESSMENT

A. Introduction

This part presents a qualitative and quantitative assessment of human exposure to CDDs/CDFs in the Midland area. Successive sections evaluate the potential for exposure via each of the media whose contamination has been investigated: air (Section B), soil (Section C), water (Section D), and fish (Section E). Section F briefly considers other routes of exposure which may be significant but which have not been investigated directly. Appendix A characterizes the populations at risk of exposure by the various routes.

Each section in this chapter summarizes the information available on levels of contamination of the medium under consideration with CDDs/CDFs (and, in a few cases, other contaminants) and derives estimates of average concentrations in the medium at points of exposure. These are combined with estimates of rates at which humans contact the medium (breathing rates, fish consumption rates, etc.) and with data on bioavailability, to yield estimates of rates of intake of CDDs/CDFs into the body. To the extent possible, estimates of exposure are expressed as TEQs, to take account of likely exposure to complex mixtures of CDDs/CDFs. Each section also characterizes the populations at risk of exposure via the medium under consideration.

In keeping with USEPA's guidelines on exposure assessment (USEPA 1986b), this chapter generates two or more sets of exposure assessments for each route

of exposure, using different assumptions, as appropriate, about environmental levels of CDDs/CDFs present, congener distributions, durations of exposure, and other factors affecting the intake of CDDs/CDFs. These estimates are intended to span the range of exposure which could plausibly occur under the given circumstances of exposure and to provide an assessment of the magnitude of uncertainty introduced into the exposure assessment by specific analytical assumptions.

The exposure estimates derived in this chapter are most directly applicable to the period at which the data were collected (in most cases, 1984-85, although data on CDD/CDF levels in fish obtained in 1987 are also included). Information on changes in manufacturing processes and waste treatment practices at the plant, combined with limited data on emission rates, suggests that emissions of CDDs/CDFs had been much higher prior to 1984. Limited data available suggest that a reduction in emissions to the air from the waste incinerator has occurred since 1983 (see Section III.B, below) and several areas of soil contamination in the facility have been remediated. However, because some CDDs/CDFs are still being released from the plant and because the existing environmental contamination with CDDs/CDFs is likely to persist, exposure is likely to continue into the indefinite future. Although it is expected that ambient concentrations of CDDs/CDFs will eventually decline, no information is yet available from which the rate of such decline could be predicted. For this reason, this exposure assessment takes no account of any declines in exposure during the period for which risks are calculated; more data would be required before any such changes could be assessed.

B. Exposures to CDDs/CDFs in Air

1. Background

In September of 1984, USEPA conducted air sampling for CDDs/CDFs at four locations near the Dow Midland facility. Significant amounts of CDDs/CDFs were detected in air at all four locations (one upwind[1] of the facility, three downwind[1]) on all 3 days during which 8-hour, high-volume samples were collected. These observations provide direct evidence that CDD/CDF exposure to the general population outside the facility boundary could be occurring through exposure to ambient air. Two of the downwind sampling locations were in light industrial areas near the facility boundary; the third was in a residential area about 1 mile from the facility boundary, and about 1.8 miles from the waste incinerator. The observed ambient air contamination with CDDs/CDFs was thought to be related to operations at the Dow Midland facility, for several reasons:

- Historical data indicate that the Dow Midland facility engaged in the production of chlorinated benzenes, chlorinated phenols, and chlorinated diphenyl ethers and their derivatives in various combinations since the 1930s (Dow 1984). Many of the processes used to produce these materials are known to result in the generation of CDDs or CDFs in varying amounts. Fugitive and stack emissions from these operations may have resulted in releases of CDDs/CDFs.

- Wastes at the facility, including those generated by the operations just discussed, have been burned at a waste incinerator that has been operating on the site since 1971. Studies of the emissions from the incinerator stack (Bumb et al. 1980; Dow 1984, 1987a; Trembly and Amendola 1987) have repeatedly shown that CDDs/CDFs are being released by the incinerator into the air.

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[1]In accordance with the prevailing wind directions derived from recorded meteorological data for the area and, except as noted, during the sampling.

- Several areas of near-surface and surface soil contamination have been detected at locations in the Dow Midland facility. Two have been remediated (capped with limestone and paved over), and another is currently being remediated. It is possible that CDD/CDF-contaminated dust from these and possibly other areas at the facility has been transported as wind-borne particulate to contribute to observed ambient air contamination off-site.

In the section that follows, the available data regarding air contamination in the vicinity of the Dow Midland facility will be reviewed, along with data concerning the nature and amounts of CDDs/CDFs released in the incinerator stack emissions, which represent a possible primary source of airborne CDD/CDF contamination. Quantitative assessments of CDD/CDF exposures in air for populations near the Dow Midland facility are developed, based on the ambient monitoring data, and the uncertainties and limitations associated with the exposure estimates are discussed. The reasons for using the ambient data rather than the incinerator stack emissions as the basis for exposure estimates are also discussed.

2. Ambient Monitoring Data

As detailed by Trembly and Amendola (1987), specially equipped ambient air samplers were installed at four different locations in and around Midland to permit collection of simultaneous samples on three separate days (September 8, 12, and 22, 1984). Figure III-1 shows the locations of the sampling sites in relation to the incinerator building, plant boundaries, and chlorophenol production areas. Site 1 was located across the Tittabawassee River from the facility, about 1.2 miles to the west of the waste incinerator. Site 2 was located at the fence line of the facility at the (then) intersection of Ball Street and Bay City Road in a light industrial area about 0.9 mile northeast of

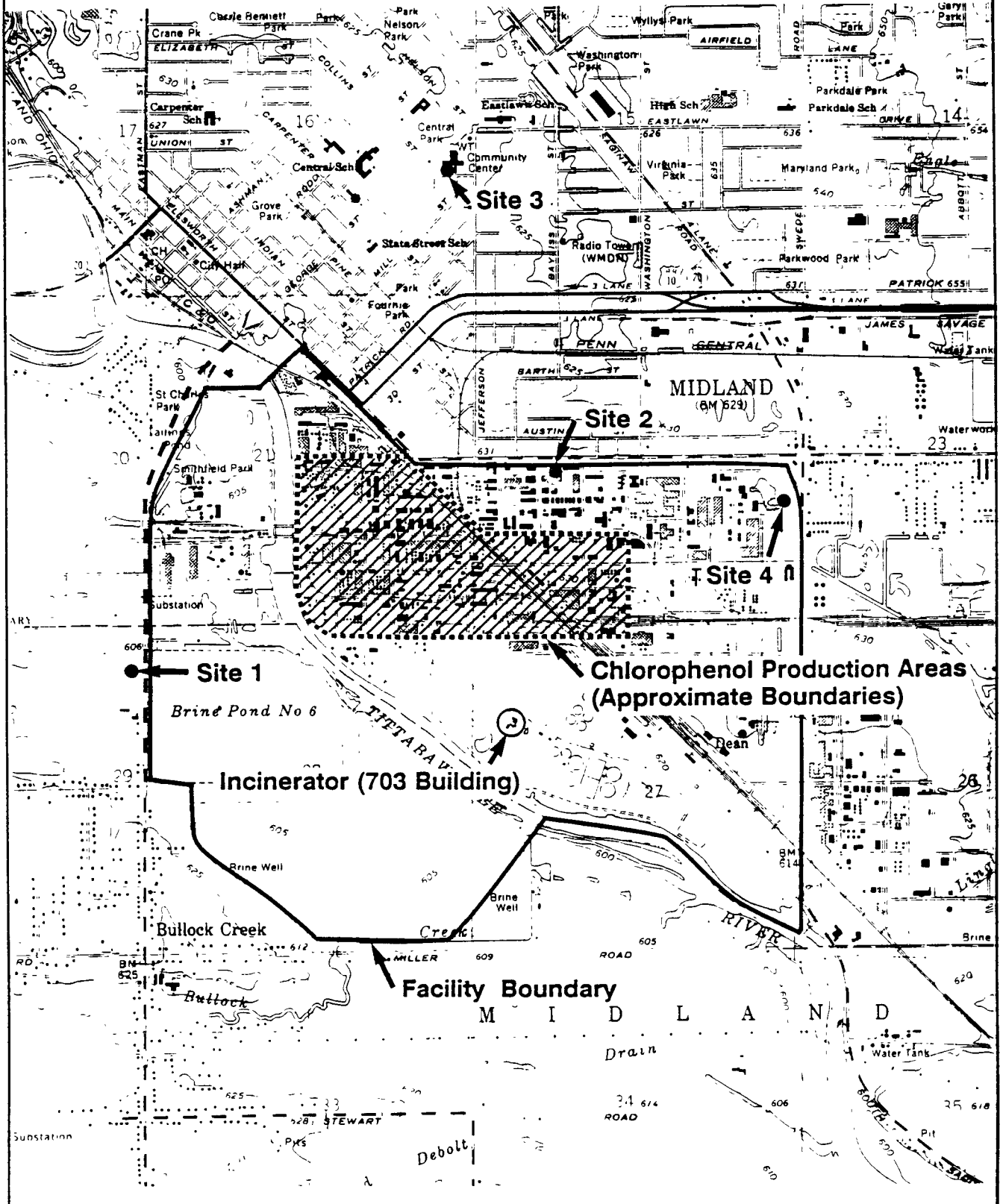


Figure III-1

Dow Midland Facility Boundaries, Chlorophenol Production Areas Incinerator Building, and Ambient Air Monitoring Locations.

Sources: USGS (1973), Dow (1984)

the incinerator. Site 3 was atop the Midland Community Center in a residential area about 1.9 miles north of the facility. Site 4 was also at the fenceline, about 1.2 miles northeast of the incinerator. Total airborne particulate levels were not measured at any of the ambient monitoring sites.

The Dow Midland facility waste incinerator located in the 703 Building was in operation on all 3 days on which ambient air samples were collected. The mean wind direction and observed variability in wind direction on these 3 days are summarized in Table III-1. On all 3 days, sampling location No. 1 was upwind from the incinerator. ("Upwind" is defined as being more than two standard deviations away from the mean downwind direction during the sampling periods). It was also in a generally upwind direction from the major production areas at the Dow facility during the three sampling events. Sampling locations 2, 3, and 4 were all located within two standard deviations of the mean downwind direction from the incinerator on all three sampling days, with the exception of location 3, which would have been slightly to the west of a narrowly dispersed plume emanating from the incinerator on September 22. Locations 2, 3, and 4 were also generally downwind of the major production areas at the facility, with the exception of location 3, which was slightly to the west of some of them (see Figure III-1). It should be pointed out that local meteorologic conditions and normal diffusion and advection processes may have substantially broadened plumes of CDD/CDF-laden particulate from the incinerator stack or from other sources that may have existed on site on any or all days during which ambient monitoring took place. Thus, it is unlikely that either monitoring station 2 or 4 actually experienced the CDD/CDF levels equal to those that would be calculated for the centerline of a theoretically modeled

TABLE III-1

WIND DATA - AMBIENT AIR SAMPLING PROGRAM
MIDLAND, MICHIGAN - SEPTEMBER 7-27, 1984

Run dates	CCA Run No.	EPA Run No.	Wind Direction		Wind Speed	
			Mean, degrees	Std. deviation	Mean, mph	Std. deviation
9/7-8	3	84ET08	184	12	5.9	1.5
9/8-9	4	84ET09	199	14	6.2	2.1
9/11-12	5	84ET10	329	91	3.8	0.9
9/12-13	6	84ET11	191	40	5.6	1.3
9/13-14	7	84ET12	309	32	3.8	1.3
9/14-15	8	84ET13	331	25	6.6	1.6
9/15-16	9	84ET14	296	62	4.9	2.8
9/16-17	10	84ET15	257	38	3.3	2.4
9/17-18	11	84ET16	212	9	4.1	1.5
9/18-19	12	84ET17	235	30	4.0	2.0
9/19-20	13	84ET18	250	44	4.1	1.5
9/20-21	14	84ET19	334	41	3.7	2.0
9/21-22	15	84ET20	12	134	4.1	1.7
9/22-23	16	84ET21	212	15	4.9	2.1
9/23-24	17	84ET22	197	42	2.6	1.1
9/24-25	18	84ET23	195	25	4.9	1.4
9/25-26	19	84ET24	284	25	6.1	1.9
9/26-27	20	84ET25	293	31	2.7	1.4

Source: Trembly and Amendola 1987

plume for the entire monitoring period. It is also probable that, on September 22, a small amount of CDDs/CDFs could have been expected to reach monitoring station 3 under reasonable assumptions about atmospheric conditions and plume dispersion, despite the fact that it was not directly "downwind" from some or all of the likely sources. As will be discussed further below, it also appears likely that directly transported emissions from the incinerator stack are not the only source of CDDs/CDFs detected in air at the ambient monitoring locations.

Modified high-volume samplers were used to collect the CDD/CDF samples (Trembly and Amendola 1987), with analysis of both the first-stage particulate filters and polyurethane foam (PUF) backing for CDDs/CDFs. Samples were extracted, "cleaned up" by solvent partitioning and liquid chromatography, and analyzed using standard gas chromatographic/mass spectrometry (GC/MS) methods. The analyses were conducted by Midwest Research Institute, with QA/QC oversight by the USEPA Sample Management Office and USEPA Region V Central Regional Laboratory. Two samples were reanalyzed by the USEPA Environmental Monitoring and Support Laboratory (EMSL) at Research Triangle Park, North Carolina.

The results of the analysis are summarized for sampling locations 1-4 in Tables III-2 through III-5, respectively. While less evident in these data than in other cases in this investigation where samples sizes were larger (e.g., soil and fish samples), the statistical distribution of CDD/CDF levels tended to be positively skewed, with a few values much higher than the arithmetic mean or median. In such circumstances, the arithmetic mean is dominated by these high values, and is higher than the geometric mean or

TABLE III-2

CONCENTRATIONS (pg/m^3) OF CDDs/CDFs DETECTED
IN MIDLAND AMBIENT AIR
SEPTEMBER 8, 12, AND 22, 1984[a]

SITE 1

Compound	9/8	9/12	9/22	Mean[b]
2378-TCDD	(--)	(0.19)	(0.06)	0.0
Total TCDDs	0.99	0.13	(--)	0.33
PeCDDs	(--)	(0.38)	(0.24)	0.0
HxCDDs	0.95	(1.0)	(0.18)	0.32
HpCDDs	0.81	0.69	(0.69)	0.50
OCDD	1.2	1.7	0.30	1.1
2378-TCDF	(--)	(0.18)	(0.11)	0.0
Total TCDFs	0.86	14.5	(--)	5.1
PeCDFs	(--)	(2.9)	0.13	0.04
HxCDFs	(--)	(0.62)	(0.26)	0.0
HpCDFs	(--)	(2.2)	(0.83)	0.0
OCDF	(--)	0.99	0.13	0.37

NOTES:

Source: Trembly and Amendola, 1987 and Appendices

[a]Values in parentheses indicate the substance in question was not detected (ND), and denote detection limits; where no value is given, no detection limit was available.

[b]Means are calculated counting "NDs" as zero. Arithmetic means are calculated for the reasons stated in the text (pp. III-8 and III-13).

TABLE III-3

CONCENTRATIONS (pg/m^3) OF CDDs/CDFs DETECTED
IN MIDLAND AMBIENT AIR
SEPTEMBER 8, 12, AND 22, 1984[a]

SITE 2

Compound	9/8	9/12	9/22	Mean[b]
2378-TCDD	(0.85)	(0.24)	(0.05)	0.0
Total TCDDs	44.8	(--)	22.4	22.4
PeCDDs	9.3	(0.43)	(0.32)	3.1
HxCDDs	(0.84)	(2.6)	0.55	0.2
HpCDDs	2.1	(3.5)	2.7	1.6
OCDDs	7.7	(6.7)	14.3	7.3
2378-TCDF	(0.84)	(0.24)	(0.99)	0.0
Total TCDFs	250	14.5	156	140
PeCDFs	30	(1.1)	7.5	12.5
HxCDFs	4.2	(1.0)	4.5	2.9
HpCDFs	5.0	(1.9)	2.9	2.6
OCDFs	3.4	(3.3)	1.6	1.7

NOTES:

Source: Trembly and Amendola, 1987 and Appendices

[a]Values in parentheses indicate the substance in question was not detected (ND), and denote detection limits; where no value is given, no detection limit was available.

[b]Means are calculated counting "NDs" as zero. Arithmetic means are calculated for the reasons stated in the text (pp. III-8 and III-13).

TABLE III-4

CONCENTRATIONS (pg/m^3) OF CDDs/CDFs DETECTED
IN MIDLAND AMBIENT AIR
SEPTEMBER 8, 12, AND 22, 1984[a]

SITE 3

Compound	9/8	9/12	9/22	Mean[b]
2378-TCDD	(0.22)	(1.1)	(0.08)	0.0
Total TCDDs	2.4	3.3	0.59	2.10
PeCDDs	(0.46)	(0.80)	(0.48)	0.0
HxCDDs	(0.32)	(1.2)	(0.39)	0.0
HpCDDs	2.1	0.65	0.55	1.10
OCDDs	7.9	5.1	2.7	5.2
2378-TCDF	(0.34)	(0.24)	(0.12)	0.0
Total TCDFs	15	45	2.1	20.7
PeCDFs	4.4	2.2	(0.23)	2.2
HxCDFs	(0.37)	(1.3)	(0.15)	0.0
HpCDFs	(0.79)	(1.2)	(0.80)	0.0
OCDFs	(1.4)	0.81	0.70	0.50

NOTES:

Source: Trembly and Amendola, 1987 and Appendices

[a]Values in parentheses indicate the substance in question was not detected (ND), and denote detection limits; where no value is given, no detection limit was available.

[b]Means are calculated counting "NDs" as zero. Arithmetic means are calculated for the reasons stated in the text (pp. III-8 and III-13).

TABLE III-5

CONCENTRATIONS (pg/m^3) OF CDDs/CDFs DETECTED
IN MIDLAND AMBIENT AIR
SEPTEMBER 8, 12, AND 22, 1984[a]

SITE 4

Compound	9/8	9/12	9/22	Mean[b]
2378-TCDD	(0.09)	(0.15)	(1.6)	0.0
Total TCDDs	0.86	0.38	74	25.1
PeCDDs	(0.09)	(0.15)	1.4	0.47
HxCDDs	0.86	2.9	0.28	1.4
HpCDDs	1.0	1.5	1.1	1.2
OCDDs	2.7	6.8	4.0	4.5
2378-TCDF	(0.12)	(0.20)	(1.6)	0.0
Total TCDFs	1.5	14	375	130
PeCDFs	1.2	1.1	37	13.1
HxCDFs	(0.65)	(1.3)	3.0	1.0
HpCDFs	(0.52)	(0.90)	3.0	1.0
OCDFs	1.7	2.7	4.6	3.0

NOTES:

Source: Trembly and Amendola, 1987 and Appendices

[a]Values in parentheses indicate the substance in question was not detected (ND), and denote detection limits; where no value is given, no detection limit was available.

[b]Means are calculated counting "NDs" as zero. Arithmetic means are calculated for the reasons stated in the text (pp. III-8 and III-13).

median. For purposes of statistical characterization or statistical testing with skewed distributions, the geometric mean or median are more appropriate measures of central tendency than is the arithmetic mean. For exposure assessment, however, the arithmetic mean is the appropriate measure of average population exposure, because population exposure is determined by the total quality of the contaminant that is contacted. In the linearized risk model used in this study, carcinogenic risks are proportional to exposure, and hence most of the population risk results from contact with locally high residue levels. In the threshold risk model used in this report, non-carcinogenic risks result from exposure above the individual's threshold level, and hence may result exclusively from contact with locally high residue levels. For these reasons, arithmetic means are calculated throughout Chapter III and are used as the basis for risk assessment in Chapter IV.

Several features of these data are of interest. Given the scarcity of analytical standards for the dozens of congeners which could be present in the samples, USEPA analysts followed current scientific practice in using a limited number of standards and reporting, primarily, homologue-specific data. The first three columns of figures in Tables III-2 through III-5 contain the estimated concentrations for two specific congeners (2378-TCDD and 2378-TCDF) and for the homologues tetra-, penta-, hexa-, hepta- and octa-CDDs/CDFs for each day of monitoring. The fourth column of figures is the arithmetic average of the concentrations over the three days of sampling, where non-detectable (ND) levels are treated as zero values. As was the case for the ambient data, these results are reported on a homologue-specific basis. In addition, no 2378-TCDD or 2378-TCDF were found above detection limits at any sampling

location on any day. There is, however, some evidence that low levels of these congeners were, in fact, present in some of the samples. The two samples that were reanalyzed by EMSL (extracts of filters and PUF plugs from locations 2 and 3 on September 8) did show levels of these congeners (0.49 pg/m^3 2378-TCDD at location 2, 0.49 pg/m^3 2378-TCDF at location 3) below the detection limits achieved by MRI (Trembly and Amendola 1987). For purposes of comparison, however, CDD/CDF values tabulated in Tables III-2 through III-5 count "non-detects" for 2378-TCDD/TCDF and other homologues as zero values. Detection limits will, however, be factored into the development of exposure estimates, as described below.

The second interesting feature of the ambient air data is the generally consistent pattern of lower levels of total CDD/CDFs at the upwind sampling location 1 than at the three "downwind" locations. The 3-day average total CDD/CDF level at location 1 (8.37 pg/m^3) was substantially lower than the 3-day average total levels at the other locations (196.68, 32.3, 180.86, respectively, for the downwind locations 2, 3, and 4), and the daily total CDD/CDF levels at location 1 were lower than the total daily levels of CDD/CDFs at all of the other three locations on all three sampling days. As will be discussed below, these relationships also hold true when the specific congener patterns detailed at each site are converted to TEQs. This finding is consistent with the hypothesis that the Dow Midland facility is a primary source of the CDD/CDF air contamination in the Midland area. The pattern of total CDD/CDF levels found at location 3 is also consistent with this hypothesis, in that the observed CDD/CDF levels were substantially lower on

September 22, a day on which this location was not directly downwind from the waste incinerator and not downwind from the majority of the production areas.

It is also important to note, however, that significant levels of CDDs/CDFs were detected at the upwind location on two of the three sampling days (see Table III-2). On the third day, only very low levels of CDDs/CDFs were detected, and the predominant homologues were HpCDDs (0.81 pg/m^3) and OCDD (1.2 pg/m^3). These findings suggest that there may be other sources of CDD/CDFs in the Midland area outside the Dow facility boundaries, such as deposits of contaminated dust or soil, which are contributing to the observed air contamination. Whether these sources were originally related to operations at the plant cannot be proven conclusively with the available data.

Another finding of importance with regard to the ambient air data is the observed congener/homologue pattern. At most sampling locations on most days, the octa-substituted homologues (OCDD, OCDF) accounted for a substantial portion of the total CDD/CDF contamination observed. As is discussed in more detail below, OCDD and OCDF were detected only at very low levels in the incinerator stack emissions compared with the other homologues, providing additional evidence that the waste incinerator may not be the only source of the observed air contamination.

The quality and limitations of the ambient air sampling data will be discussed in more detail below.

3. Stack Emissions Data

Characterization of the CDD/CDF emissions from the waste incinerator is important because the incinerator may be a major point source of CDD/CDF emissions. Recently, an assessment has been performed using data from USEPA's 1984 sampling efforts as inputs to an atmospheric transport model (Cleverly 1986), which generated quantitative estimates of exposures of Midland residents to airborne CDDs/CDFs. The results of this assessment are discussed in more detail in this section.

Several efforts have been undertaken to measure the levels of CDDs/CDFs in the stack emissions from the chemical waste incinerator at the Dow Midland facility. Prior to 1987, the only set of data available which measured a nearly comprehensive range of congeners/homologues was that gathered by USEPA on 3 days in the fall of 1984 (Trembly and Amendola 1987). Prior to this effort, other studies (Bumb et al. 1980, Dow 1984) had measured only selected subsets of CDD/CDF compounds or used analytical methods that have been superseded by more modern approaches.

Recently Dow has submitted additional data, on a preliminary basis, describing the CDD/CDF emissions from the incinerator during operations on June 25, 1987 (Dow 1987a). In addition, Dow (1984) reported stack emissions data gathered in 1983, when water pollution control sludges known to contain significant amounts of CDDs/CDFs and their parent compounds were still being burned in the incinerator, a practice that was ended in early 1984. The 1983

data also did not include analytical results for any CDF congeners or homologues.

Since the USEPA data represent the most complete, thoroughly documented, and validated set of information regarding the nature and amount of CDD/CDF emissions from the waste incinerator when wastewater treatment sludges were not being burned, and since these data were gathered at roughly the same time as the ambient monitoring samples discussed previously, they are now briefly reviewed to help provide insight into the development of the quantitative exposure assessment. Both these and the 1987 Dow data will be used later to develop rough exposure estimates for purposes of comparison with the exposure estimates derived from the ambient monitoring results.

Stack emission samples were gathered on August 28 and 30 and on September 5, 1984. Samples were collected using a USEPA Modified Method 5 sampling train, XAD resin absorbent and polyurethane foam (PUF) plug supports, each of which was analyzed for CDDs/CDFs. Chemical analyses for CDDs/CDFs were conducted by Brehm Laboratory of Wright State University with QA oversight by USEPA Sample Management Office and USEPA Region V Central Regional Laboratory.

Waste feeds to the incinerator were also analyzed on the same days as the stack emissions were monitored. The wastes contained very high levels of volatile chlorinated organics (up to 450,000 mg/kg carbon tetrachloride, up to 18,000 mg/kg chlorobenzene) and appreciable amounts of semi-volatile organics, including 1,2-dichlorobenzene (up to 1,570 mg/kg), 2,4,5-trichlorophenol (up to

4,690 mg/kg), and 2,4,6-trichlorophenol (up to 8,320 mg/kg). Also present were CDDs/CDFs at total levels up to 147 μg/kg (Trembly and Amendola 1986).

The results of the USEPA stack emissions analyses from 1984 are summarized in Table III-6. Estimates are given of the concentrations of CDDs/CDFs which were in the stack emissions during each of the three separate days on which sampling was conducted.

The results of the USEPA study revealed no detectable emissions of 2378-TCDD. However, Dow conducted similar tests on the same facility, but on different days, and detected the presence of 2378-TCDD in the emissions on all 3 days at levels up to 0.71 ng/m^3 (Dow 1984). Possible reasons for the qualitative differences between the USEPA and Dow results on the question of the presence of 2378-TCDD include the following:

- The sampling was performed on different days; therefore, differences in incinerator feed, operation conditions, etc., could have resulted in different rates of 2378-TCDD emission.
- The detection limits of the Dow investigators were lower than those of the USEPA investigators. (Only on Day 3 of USEPA's study were the detection limits sufficiently low that any of the 2378-TCDD levels reported by Dow would have been detected.)
- The experiments were performed by different researchers, using somewhat different techniques.

The more recent Dow data (Table III-7) also indicate the presence of 2378-TCDD in stack emissions (Dow 1987a). These data, while they have not been fully validated, can, along with the data from the 1984 USEPA sampling, be used to help elucidate and compare patterns of incinerator stack emissions with the ambient data discussed above. The basic pattern of homologue/congener emissions is similar in the 1984 and 1987 sampling results, with relatively

TABLE III-6

CONCENTRATIONS (ng/m^3) OF CDDs/CDFs IN CHEMICAL WASTE INCINERATOR EMISSIONS[a]
AUGUST/SEPTEMBER 1984

Compound	August 28	August 30	September 5	Mean[b]
2378-TCDD	(0.72)	(3.08)	(0.17)	0
Total TCDDs	46	44	4.9	31.5
PeCDDs	5.5	1.9	(0.85)	2.81
HxCDDs	0.88	0.37	(0.72)	0.42
HpCDDs	0.21	0.84	(0.19)	0.35
OCDDs	0.93	2.5	(0.82)	1.15
2378-TCDF	1.5	1.7	(.21)	1.06
Total TCDFs	81	77	125	94
PeCDFs	13	4.3	0.07	5.8
HxCDFs	2.5	2.0	(0.48)	1.5
HpCDFs	0.26	0.55	(0.87)	0.27
OCDFs	0.06	0.17	(4.6)	0.08

NOTES:

Source: Trembly and Amendola, 1987 and Appendices

[a]Value in parentheses are non-detect levels.

[b]In calculating means, "non-detects" are counted as zero values. Arithmetic means are calculated for the reasons stated in the text (pp. III-8 and III-13).

TABLE III-7

SUMMARY OF HAZARD INDICES FOR NON-CANCER EFFECTS
FROM EXPOSURE TO CDD/CDF CONTAMINATION IN MIDLAND, MICHIGAN

Exposure Route	Exposure Scenario	Hazard Index (HI)[a]		
		Long-Term	Short-Term	Single Meal
Fish[b]	Plausible maximum consumer	50	5	8
	High sports fisherman	20	2	0.5
	Median sports fisherman	9	0.7	0.2
	General consumer	0.7	0.4	0.2
Soil	Upper estimate young child			
	-- with pica	6	0.2	--
	-- normal	0.6	<0.1	--
	Lower estimate young child	<0.1	<0.1	--
	Upper estimate adult	<0.1	<0.1	--
Air	Infant at fenceline[c]	3	0.1	--
	Child at fenceline	1	<0.1	--
	Child in residential area	0.3	<0.1	--
	Adult in residential area	<0.1	<0.1	--

[a]Hazard Index is the ratio of intake dose to:

-- RfD (1 pg/kg/day) for long-term exposures (several months or more)
-- 10-day HA (28 pg/kg/day) for short-term exposures (few days to few weeks)
-- Single-dose HA (300 pg/kg/day) for single-meal or single-day exposures

[b]Small child could be at 2-3 times higher risk than adult. Breast-fed infant could be at 10-times higher risk than mother. Other contaminants such as PCBs, found in the fish, add to the toxicity (see Appendix B of the Risk Assessment).

[c]Includes exposures from breast-feeding.

high levels of tetra- and penta-substituted CDDs/CDFs predominating, and only low levels of OCDD and OCDF present. As will be discussed later, however, the 1987 Dow results show a considerably higher ratio of TCDFs to TCDDs than the 1984 USEPA results, and the total TEQ values calculated for the 1987 Dow data are somewhat lower than the TEQs calculated for the 1984 EPA data. The more recent data also show a higher ratio of TCDFs to total CDDs/CDFs and a correspondingly lower ratio of TCDD to total CDDs/CDFs than the earlier data. It is not clear that either of these differences indicate a permanent or systematic difference in incinerator emissions between 1984 and 1987; the patterns may merely reflect sample-to-sample variability. The pattern of CDD/CDF homologue in the incinerator emissions in both the 1984 and 1987 samples, particularly the low levels of OCDD/OCDF, is strikingly different from the pattern observed in the ambient air samples (Tables III-2 through III-5). These issues are discussed in more detail below.

4. Comparison of Stack Emissions and Ambient Air Sampling Results.

Figures III-2 through III-6 display profiles of the congener/homologue distributions of the CDDs/CDFs found in the ambient air at locations 1 through 4 and during 1984 USEPA and 1987 Dow sampling of the stack emissions, respectively. It can be seen that there are some striking differences between the congener patterns observed in the ambient air and those found in the stack emissions. These differences may be interpreted as combinations of two basic patterns, represented, respectively, by that observed at the "upwind" ambient monitoring site 1 on all three days, and the pattern observed in the stack emissions. The former pattern ("pattern 1") is characterized by the

FIGURE III-2 : Profile of CDD's/CDF's Detected In Midland, MI Ambient Air-Site 1

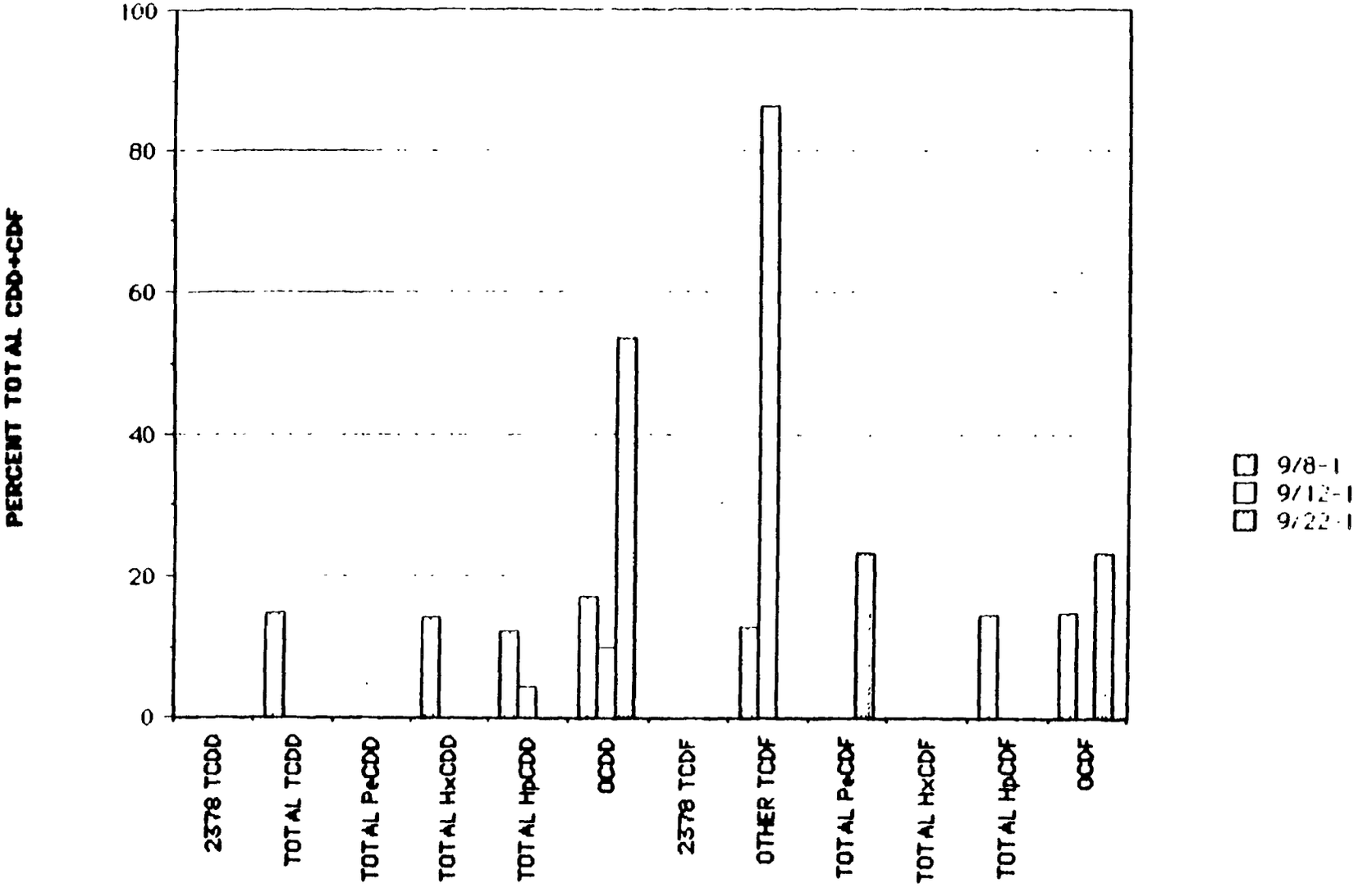


FIGURE III-3 : Profile of CDD's/CDF's Detected In Midland, MI Ambient Air- Site 2

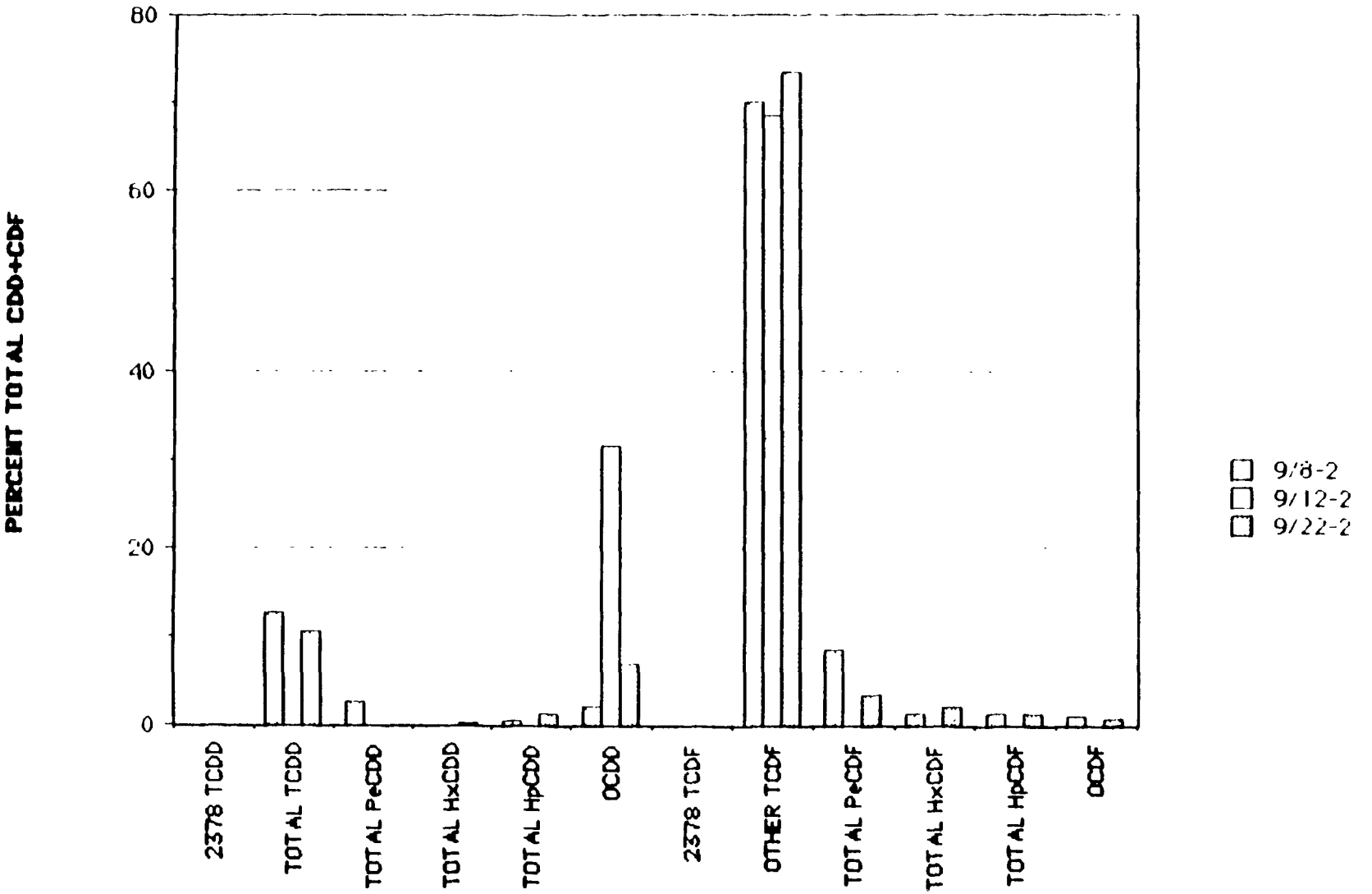


FIGURE III-4 : Profile of CDD's/CDF's Detected in Midland, MI Ambient Air- Site 3

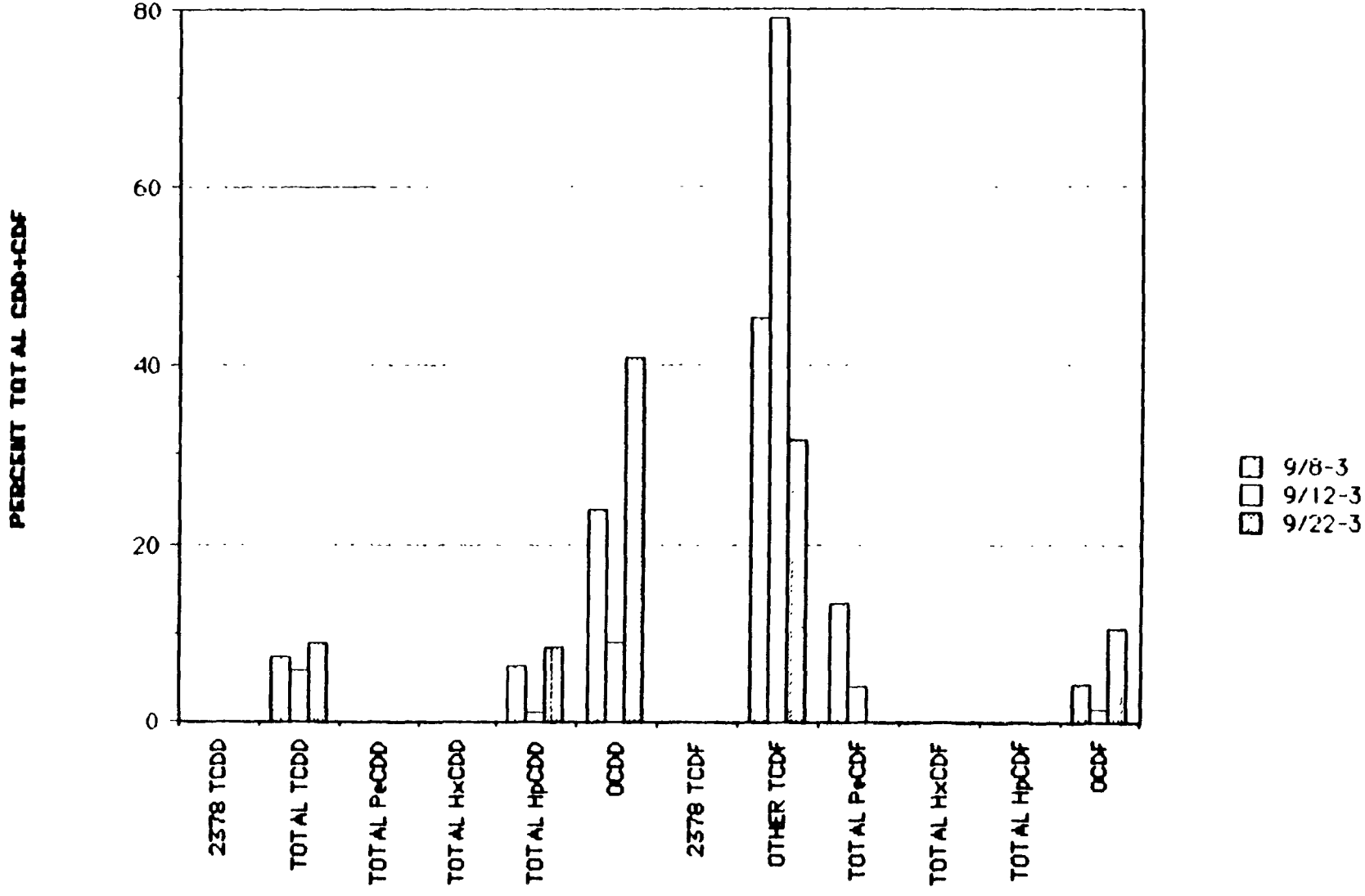


FIGURE III-5 : Profile of CDD's/CDF's Detected in Midland, MI Ambient Air- Site 4

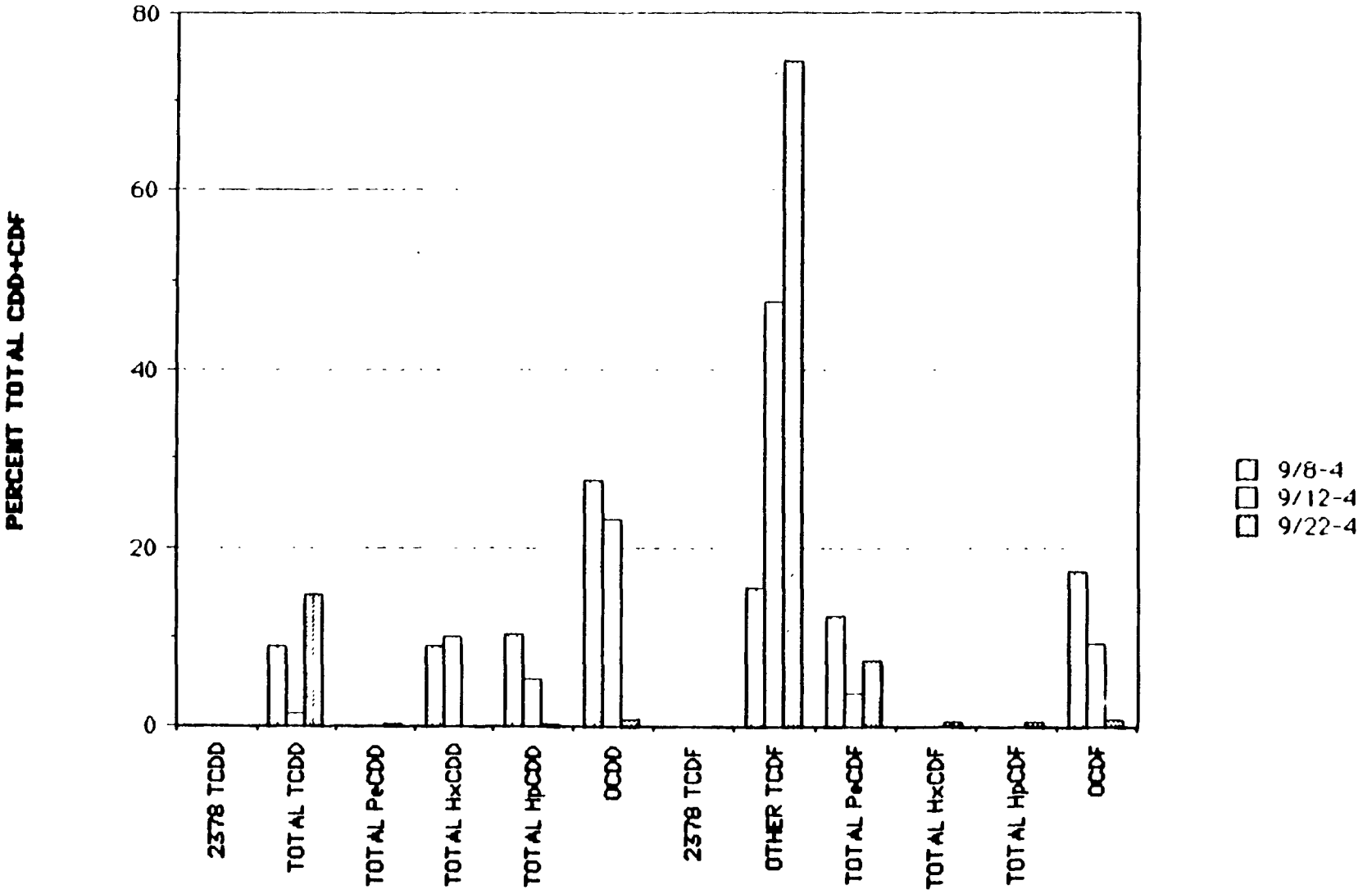
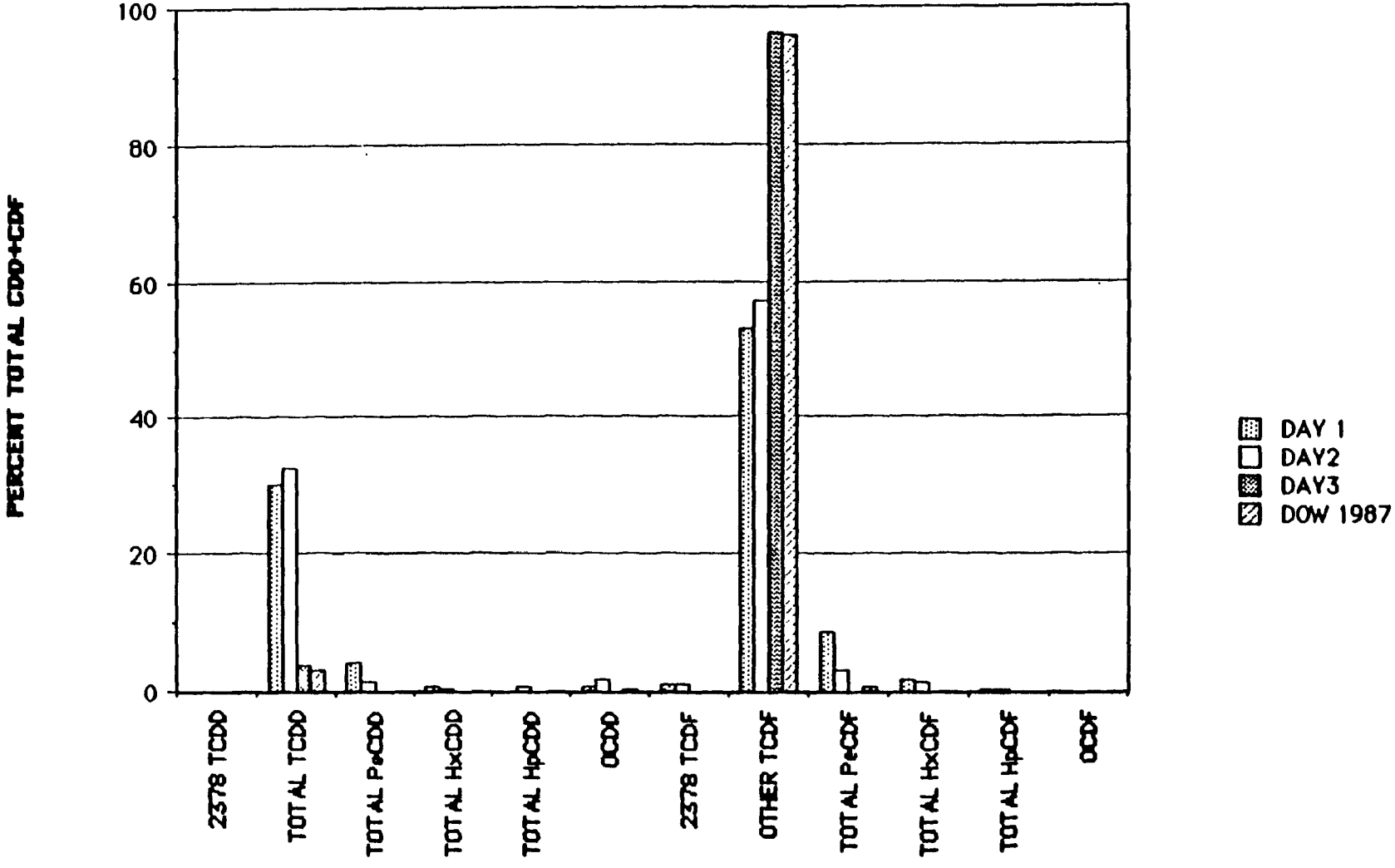


FIGURE III-6: Profile of CDD's/CDF's In Chemical Waste Incinerator Emissions



predominance of highly substituted congeners, particularly HpCDDs, OCDD and OCDF. The latter pattern ("pattern 2"), from the incinerator emissions, is characterized by comparatively high levels of TCDDs and particularly TCDFs, and the almost total absence of OCDD or OCDF. The patterns are illustrated numerically in Table III-8, where the ratios of TCDDs, TCDFs, and OCDD to total CDDs + CDFs are tabulated for each ambient sampling location, for the 1984 (and 1987) stack emissions data, and for soil samples from Midland and non-industrialized areas of Minnesota.

The "downwind" ambient sampling stations (sites 2 and 3), exhibit various patterns of congener distribution on different days, with variations among sites as well as between OCDD and TCDD levels observed on different days. Sites 2 and 4, near the facility fenceline, relatively close to the incinerator and more or less directly downwind from it on all three sampling days, in general display a pattern that is a mixture of the two basic patterns described above, with high levels of TCDDs and TCDFs observed on all three days, but significant levels of OCDD also being observed in most of the samples. The OCDD to total CDD/CDF ratios in the ambient samples exceed those in the stack emissions by at least one order of magnitude. At site 3, the pattern was also "mixed" on September 8 and 22 when the site was downwind from the incinerator, but on September 28, when the sampling station may have been outside of a narrow incinerator stack plume, the pattern of congeners observed at site 3 was much more similar to the "upwind" pattern 1.

There are a number of possible explanations for the observed differences in the patterns of congener distribution between the stack and ambient air.

TABLE III-8

RATIOS OF SELECTED HOMOLOGUES TO TOTAL CDD + CDF LEVELS IN MIDLAND AMBIENT AIR, INCINERATOR STACK EMISSIONS, AND SOIL DATA FROM MIDLAND PUBLIC AREAS AND MINNESOTA NATURAL AREAS

	Proportion of Total CDDs + CDFs Accounted for By:		
Sample	TCDDs	TCDFs	OCDD
Incinerator Emissions			
1984 USEPA Study	0.22	0.54	0.006
1987 Dow Study	0.04	0.95	0.003
Ambient Air			
1984 USEPA Study			
Site 1 Day 1	0.21	0.18	0.25
Day 2	0.007	0.81	0.09
Day 3	0.0	0.0	0.70
Mass-Weighted Average[1]	0.04	0.66	0.014
Site 2 Day 1	0.13	0.70	0.021
Day 2	0.00	1.00	0.0
Day 3	0.11	0.71	0.07
Mass-Weighted Average	0.12	0.72	0.03
Site 3 Day 1	0.08	0.45	0.25
Day 2	0.06	0.79	0.09
Day 3	0.09	0.32	0.41
Mass-Weighted Average	0.07	0.64	0.16
Site 4 Day 1	0.09	0.15	0.27
Day 2	0.01	0.48	0.23
Day 3	0.15	0.75	0.008
Mass-Weighted Average	0.14	0.72	0.02
Midland Public Areas (soil, ave.)	0.02	NA	0.70
Minnesota Natural Areas (soil, ave.)	0.02	NA	0.72

Sources: Calculated from Trembly and Amendola 1987 and Appendices, Amendola 1987, and preliminary data submitted by Dow Chemical Company (Dow 1987a).

[1]Mass-weighted average values do not equal the means of the daily average values because the levels and ratios of CDD/CDF congeners varied widely from day to day at some sampling sites and observations from one or two days may dominate the mass-weighted average.

NA = not analyzed.

One possibility is that the incinerator is sole source of the observed airborne CDD/CDFs, but that feed wastes were different on the days during which stack emissions were sampled than on days during which the ambient air was sampled. This appears unlikely, given the consistent pattern of appearance of one group of congeners (OCDD/OCDF) in the air on all three ambient sampling days.

Another possibility is that there is an additional source or sources of CDD/CDF air contamination in the Midland area. Sources could include fugitive CDD/CDF emissions from activities in the facility, resuspended soil or particulate contaminated with CDDs/CDFs from the facility area, or additional sources of CDDs/CDFs outside the facility.

In addition, the results could be interpreted to show that the collection efficiencies were different for different congeners for the stack and ambient sampling methods. There is, however, no reported evidence to suggest that either the stack or ambient sampling methods employed are selectively more efficient for specific congeners or homologues.

A final possibility is that emissions from the stack were, in fact, the major source of the observed ambient air contamination, but that the differences in congener/homologue distribution between the stack and ambient samples are the results of environmental processes which changed congener distribution between the time the emission entered the stack and the time they arrived at the sampling stations. Postulated mechanisms could include either a selective decomposition of the less-substituted congeners or the addition of chlorine to the less-substituted congeners to give the observed higher

OCDD/CDD+CDF ratios. Neither of these mechanisms seems very plausible on kinetic or mechanistic grounds and neither would explain the observed upwind CDD/CDF air contamination.

It is significant to note that "pattern 1" is very similar to the homologue distribution observed in soil samples taken from natural (non-industrialized) areas of Minnesota (USEPA 1985a, see Table III-8). The results at the upwind ambient station could thus be interpreted to be evidence of some kind of "background" contamination, although the ultimate source of this "background" cannot be identified, and could be facility-related. For example, contaminated soil to the south-west of the facility and sampling location 1 (see section III.C), could be the source of the observed CDD/CDF contamination in the "upwind" ambient air samples. Additional evidence for the facility-relatedness of the observed contamination, however, is the consistent pattern of much higher CDD/CDF levels in the downwind air samples. This holds true even for the OCDD/OCDF homologues.

There are thus several plausible hypotheses that could explain the observed pattern of ambient air contamination. They could, for example, be the result of direct airborne transport of incinerator emissions and fugitive emissions or resuspended soils from the facility (the soil presumably being selectively contaminated with OCDD/OCDF from past releases or depleted of TCDFs/TCDDs by selective volatilization or degradation of these congeners). Alternatively, the downwind air contamination could be the result of a combination of directly transported incinerator emissions and resuspended soil or dust from the downwind sampling areas outside the facility (again, these

soils would have been selectively contaminated with the more highly substituted or depleted of the less-substituted homologues, as discussed above).

Whatever the specific source or sources of ambient air contamination, the available data do strongly suggest that the waste incinerator is not the sole source of this contamination. For this reason, it appears advisable not to rely on the incinerator emission data, coupled to air transport models, as the basis for exposure estimation for ambient air exposures. Rather, the ambient air data provide the more reliable guide as to exposures likely to be experienced by populations residing near the facility, and therefore they are used as the basis of the central exposure estimation effort in this analysis.

5. Populations at Risk of Ambient Air Exposure.

The population at risk of exposure to CDD/CDF air contamination is taken to include all individuals residing and/or working near the Dow Midland facility. On-site and occupational exposure to the facility itself is not considered in the analysis. Exposure of populations outside the city and county of Midland is not considered, because levels of CDDs/CDFs are expected to decline rapidly with distance due to mixing with ambient air. However, it should be recognized that residents just outside the city and county lines are also exposed to airborne contaminants, at levels lower than those occurring within the city.

As discussed in Appendix A, there are approximately 32,000 people living in the Midland area, of which approximately 26,000 live in areas within 3 miles

of the facility boundary in a generally downwind direction. The closest residences to the facility to the north-east of the facility are about 0.25 miles from the fenceline, the intervening distance being devoted largely to light industrial uses. To the northwest and east of the facility, there are a number of residences directly adjacent to the facility boundary, but not adjacent to currently used production areas. Most of the population of the Midland area resides in residential areas to the north of the facility at distances between 0.5 and 3.5 miles from the facility boundary.

6. Exposure Estimation.

In this section, quantitative estimates are developed for inhalation exposure to CDDs/CDFs for persons residing in the Midland area in the vicinity of the Dow Midland facility. Because of the limited amount of data available, in order to provide some quantitative measure of the range of exposures that may actually occur, and to provide an illustration of the impact of different assumptions on the exposure estimates, two exposure scenarios are developed. The first "fenceline case" incorporates assumptions consistent with long-term exposures at the Dow facility fence line near the two "downwind" monitoring locations. The other "residential area case" employs assumptions corresponding more closely to exposures occurring in residential areas of Midland, further from the facility. As noted previously, data on CDD/CDF levels in ambient air near the facility are used to provide the quantitative basis for the exposure estimates. Data from the four sampling locations are used to construct the two exposure scenarios as described below.

a. Exposure Scenario 1: Fenceline Case

The fenceline case scenario attempts to simulate the long-term exposures received by a hypothetical individual living near the northern facility boundary. Pooled data from ambient monitoring sites 2 and 4 are used to provide an estimate of the average lifetime inhalation exposures for this scenario. Observations on CDD/CDF levels from the two sites are averaged (see Table III-9) using values equal to the detection limits for homologues and congeners that were not detected at these locations. The rationale for doing so is that it is likely that at least some of the congeners not detected were, in fact, present at levels lower than the detection limits during the sampling, and that using the detection limit to fill the data gaps gives an upper-bound on the levels of the non-detected congeners that were actually present. Also, as discussed previously, reanalysis of two of the ambient air samples by USEPA's EMSL laboratory did, in fact, detect 2378-TCDD and 2378-TCDF (the most toxicologically significant of the "non-detect" congeners) at levels comparable to the MRI detection limits at site 2 on one day.

In the last column of Table III-9, the averaged air concentrations of the CDDs/CDFs detected at sites 2 and 4 are summed using the Toxicity Equivalence Factor (TEF) approach described in Chapter II. Both the "A-method" (assuming all congeners among the penta- through hepta-substituted homologues are 2378-substituted) and "B-method" (assuming a uniform statistical distribution of congeners within each of these homologues) are used to develop total Toxicity Equivalents (TEQs) for the averaged site 2 and 4 data. This approach, again, helps to ensure that the exposure estimates span the range of likely values,

TABLE III-9

AVERAGE CDD/CDF LEVELS IN AIR AND TOXICITY EQUIVALENTS FOR MONITORING SITES 2 AND 4

Compound	TEF	Proportionality Factor	Average Air Level (pg/m^3)	TEQ (pg/m^3) Method A	TEQ (pg/m^3) Method B
Total TCDD	1.00	1.00	23.74	--	--
2378	1.00	0.05	[a](0.50)	0.500	0.500
Other	0.01	0.95	23.24	0.232	0.232
Total PeCDD	0.50	1.00	(1.95)	0.975	--
2378	0.50	0.07	NA	--	0.068
Other	0.005	0.93	NA	--	0.009
Total HxCDD	0.04	1.00	(1.34)	0.054	--
2378	0.04	0.30	NA	--	0.016
Other	0.0004	0.70	NA	--	0
Total HpCDD	0.001	1.00	(1.99)	0.002	--
2378	0.001	0.50	NA	--	0.001
Other	0.00001	0.50	NA	--	0
Total OCDD	0	1.00	7.03	0	0
Total TCDF	0.100	1.00	135.13	--	--
2378	0.100	0.03	[a](0.67)	0.067	0.067
Other	0.001	0.97	134.46	0.134	0.134
Total PeCDF	0.100	1.00	(12.95)	1.295	--
2378	0.100	0.07	NA	--	0.091
Other	0.001	0.93	NA	--	0.012
Total HxCDF	0.01	1.00	(2.44)	0.024	--
2378	0.01	0.50	NA	--	0.006
Other	0.0001	0.50	NA	--	0
Total HpCDF	0.001	1.00	(2.38)	0.002	--
2378	0.001	0.50	NA	--	0.001
Other	0.00001	0.50	NA	--	0
Total OCDF	0	1.00	(2.88)	0	0
Total TEQs	--	--	--	3.285	1.137
Total TEQs from "Non-Detects"	--	--	--	0.703	0.584

Source: Calculated from Trembly and Amendola (1987) and appendices.

NOTE: Figures in parentheses represent average observed levels where one or more "non-detects" were included in the averaging process. Non-detect values were counted as observations at the calculated detection limits.

[a]All the data for the indicated congener/homologues were derived from non-detect values.

[b]Arithmetic means are calculated for the reasons stated in Section III.C (footnote to p. III-59).

and provides information concerning the magnitude of the impacts of specific assumptions on the exposure estimates.

As can be seen in Table III-9, use of the "A-method" results in a higher total average TEQ (3.29 pg/m^3 TEQ) than use of "B-method" (1.14 pg/m^3 TEQ). The proportion of the calculated total TEQ for the site 2 and 4 data that is derived from the use of the detection limits in place of "non-detects" is 21 percent for the "A-method" and 51 percent for the "B-method". Thus, using one-half the detection limits for "non-detect" values (a possible alternative approach) would reduce the calculated total exposure during downwind exposures by only 11% using "A-method", and 25% using the "B-method" to calculate TEQs. Neither of these reductions would be significant, given the level of uncertainty inherent in other aspects of the exposure assessment.

In order to take into account, at least in a crude fashion, the presumed facility-relatedness of the observed CDD/CDF concentrations in the fenceline case scenario, an adjustment needs to be made for the proportion of the time that the hypothetical exposure point (at the northern facility boundary, between sampling locations 2 and 4) would be downwind from the incinerator and from other potential CDD/CDF sources at the facility. For the purpose of this assessment, it is assumed that this would be the case any time the exposure point is downwind from an appreciable proportion of the Dow Midland (but not Dow Corning) facility, that is, when the wind blows from any direction between south-southeast (157°) and west-northwest (293°). Using meteorologic data for the Consumer's Power Nuclear Plant (USEPA 1985a), it is estimated that the wind will blow from these directions about 58% of the time, on average. During the

time that the exposure point is not downwind of the major production areas and the incinerator, it is assumed that the ambient air levels are the same as those measured at the "upwind" site 1. From the data in Table III-2, it can be calculated that the average total TEQs measured at site 1 for the three sampling days were 0.34 and 0.12 pg TEQ/m^3, using the "A" and "B" methods, respectively. These calculations were again made using detection limit values for non-detects (where detection limits were available) and zero when no detection limits were available. For the "background" (site 1) monitoring results, TEQs derived from nondetect results for particular congeners account for 91% and 77% of the total TEQs calculated using the "A" and "B" methods, respectively.

The total exposure levels calculated for the fenceline scenario using the time-averaged "downwind" and "background" exposure levels are 2.1 pg/m^3 TEQs ("A" method) and 0.71 pg/m^3 TEQs ("B" method). As expected, the contribution of the "background" CDD/CDF levels represent only a small proportion (<8%) of the total TEQ levels calculated using either method. For the overall exposure estimates, "NDs" account for 26% and 52% of the total TEQs, using the "A" and "B" methods, respectively.

As discussed previously, this approach only approximates actual meteorologic conditions; natural atmospheric instability and normal plume dispersion would actually result in less than theoretical peak exposures during periods when the exposure point was "downwind" of the facility, and also could result in some appreciable levels of CDD/CDF air contamination from the facility reaching the exposure point even when it was not nominally downwind of

likely contaminant sources. Also, given the large area of the facility, it is not possible to make a precise, conclusive judgment as to when the exposure points might actually be in a downwind direction from sources other than the waste incinerator. Again, the effect of utilizing this assumption is small relative to the other sources of uncertainty in the exposure assessment.

b. Exposure Scenario 2: Residential Area Case

The "residential area case" exposure scenario is defined as occurring near ambient monitoring site 3, in an area of higher population density than the "fenceline case" scenario, further away from the Dow Midland facility, and less directly downwind of the incinerator and major production areas. The average CDD/CDF levels measured in ambient air at site 3 are used to calculate ground-level inhalation exposures for a population residing in this area (Table III-10). Again, both methods of calculating TEQs are employed, although in this scenario, "non-detect" values were replaced by values equal to one-half the detection limit for the congeners/homologues in question. The rationale for doing so is similar to that employed in adjusting the "non-detect" values for the fenceline case scenario, except that one-half the detection limit is in the middle of the possible range of values for the non-detected congeners and is less likely to represent upper-bound estimates for the non-detected compounds.

As shown in the last column of Table III-10, the estimated ground-level exposures for the residential area scenario during periods when the exposure point is downwind from the facility are 0.67 pg/m^3 TEQs and 0.32 pg/m^3 TEQs, when using the "A" or "B" methods, respectively. Unlike the scenario previously described, the "non-detect" values make a major contribution to the

TABLE III-10

AVERAGE CDD/CDF LEVELS IN AIR AND TOXICITY EQUIVALENTS FOR MONITORING SITE 3

	TEF	Proportionality Factor	Average Air Level (pg/m^3)	TEQ (pg/m^3) Method A	TEQ (pg/m^3) Method B
Total TCDD	1.00	1.00	2.10	--	--
2378	1.00	0.05	[a](0.23)	0.230	0.230
Other	0.01	0.95	1.870	0.019	0.019
Total PeCDD	0.50	1.00	[a](0.290)	0.145	--
2378	0.50	0.07	NA	--	0.010
Other	0.005	0.93	NA	--	0.001
Total HxCDD	0.04	1.00	[a](0.32)	0.013	--
2378	0.04	0.30	NA	--	0.004
Other	0.0004	0.70	NA	--	0
Total HpCDD	0.001	1.00	1.10	0.001	--
2378	0.001	0.50	NA	--	0.001
Other	0.00001	0.50	NA	--	0
Total OCDD	0	1.00	5.23	0	0
Total TCDF	0.100	1.00	20.70	--	--
2378	0.10	0.03	[a](0.12)	0.012	0.012
Other	0.001	0.97	20.58	0.020	0.020
Total PeCDF	0.100	1.00	(2.24)	0.224	--
2378	0.100	0.07	NA	--	0.016
Other	0.001	0.93	NA	--	0.002
Total HxCDF	0.01	1.00	[a](0.30)	0.003	--
2378	0.01	0.50	NA	--	0.001
Other	0.00001	0.50	NA	--	0
Total HpCDF	0.001	1.00	0.47	0	--
2378	0.001	0.50	--	--	0
Other	0.00001	0.50	--	--	0
Total OCDF	0	1.00	0.97	0	0
Total TEQs	--	--	--	0.667	0.316
Total TEQs from "Non-Detects"	--	--	--	0.420	0.277

Source: Calculated from Trembly and Amendola (1987) and appendices.

NOTE: Figures in parentheses represent average observed levels where one or more "non-detects" were included in the averaging process. Non-detect values were counted as observations at one-half of the calculated detection limits.

[a]All the data for the indicated congener/homologues were derived from non-detect values.

[b]Arithmetic means are calculated for the reasons stated in Section III.C (footnote to p. III-59).

total exposure at this exposure point. Substitution of one-half detection limit values for "non-detects" accounts for 63% of the total TEQ calculated using the "A-method" and 88% of the TEQ calculated using the "B-method". This occurs primarily because all of the information at monitoring site 3 regarding the CDD congeners with the highest toxicity (2378-TCDD and PeCDDs) is derived from "non-detects". This suggests that the exposure estimates for this scenario are substantially more uncertain, and more dependent on the quality of the analytical results, than those for the fenceline case scenario.

A long-term exposure level for the residential area scenario is calculated using a factor to convert for the proportion of the time the exposure point would be downwind of possible on-site CDD/CDF sources, with "background" level again assumed to be occurring at times when the exposure point was not "downwind," just as was done for the fenceline case scenario. For this scenario, it is estimated that the exposure point will be downwind of potential facility-related sources whenever the wind blows from any direction between southwest (235°) and southeast (135°), or about 33% of the time (USEPA 1985a). "Background" exposure levels, calculated using the "A" and "B" methods and the data from monitoring site 1 (counting nondetects as being equal to one-half the detection limit for this scenario) are 0.16 and 0.070 pg/m^3 TEQs, respectively. TEQ estimates derived from nondetect values account for 80% and 84% of the total "A" method and "B" method background TEQs, respectively.

The time-weighted average long-term exposure levels for the residential area case are calculated to be 0.34 pg/m^3 TEQ ("A" method) and 0.15 pg/m^3 TEQ ("B" method). Overall, TEQs derived from "NDs" account for 72% and 87% of the

total exposure for this scenario calculated using the "A" and "B" methods, respectively. In this scenario, "background" exposures account for a significant proportion of the total exposures, 42% for the "A" method and 39% for the "B" method. This is not unexpected, in that the residential area exposure point is assumed to be "downwind" from the Dow facility less than one-third of the time and because measured CDD/CDF levels at monitoring site 3 are lower than at the fenceline exposure point and only moderately higher than those measured at the "upwind" site 1.

c. Intake Assumptions

The last step in the development of quantitative exposure estimates for the two exposure scenarios is to define a set of assumptions which characterize the relationships between the long-term average air levels and the doses of CDDs/CDFs to exposed populations that would be associated with these levels. For the purpose of this analysis, a number of assumptions are made, the most important of which are the following.

For both exposure scenarios, it is assumed that the exposed individuals live their entire lifespan at the hypothetical exposure points. Exposures are assumed to occur 24 hours per day to the long-term average ambient air levels calculated above. It is assumed that indoor exposure will be neither higher nor lower than outdoor exposure, thus discounting either a protective effect of being indoors, or an increase in indoor exposure levels due to exposure to contaminated household dust. Dose levels are calculated for infants (age less than 1 year), children aged 1-6 and 6-12, and adults (age 12-70). Values for

the physiological parameters used to calculate inhalation intake of CDD/CDFs for each age group are summarized in Table III-11. Following the approach of Schaum (USEPA 1984b), it is assumed that 27% of the inhaled CDD/CDF is retained in the body.

Applying the assumptions just described to the long-term air levels of CDDs/CDFs calculated for the two exposure scenarios results in the calculated doses for the two exposure scenarios which are summarized in Table III-12. As expected, the average daily intakes are greatest on a mg/kg-day basis for small children who have relatively low body weights and high metabolic rates and respiratory volumes.

As will be discussed in Part IV, the intake values developed here represent estimates of the absolute amounts of CDDs/CDFs taken into the body of the exposed individuals. When these intake estimates are compared in Part IV with dose-response data for 2378-TCDD, it will be necessary to taken into account the fact that the RfD and HAs are derived from the results of experiments in which 2378-TCDD was administered to animals in feed and are expressed in terms of <u>administered</u> dose. Because the bioavailability of 2378-TCDD from feed is less than 100%, the administered dose in these experiments was greater than the absorbed dose. In Chapter IV, an additional adjustment will be applied to the intake estimates derived above to make them commensurable with the administered doses that form the basis of the RfD and HAs.

TABLE III-11

PHYSIOLOGIC PARAMETERS FOR INHALATION INTAKE ESTIMATION

Age Group	Body Weight (kg)[1]	24-Hour Respiratory[2] Volume (m^3)
Infants (age 0-1 years)	9	3.6
Young children (ages 1-6)	15	16
Older children (ages 6-12)	31	23
Adults (ages 12+)	70	20

Source: Anderson, et al. (1984) USEPA (1984b, 1985c,d).

[1]Body weights for children and infants are calculated using 50th-percentile age-group data from the NHANES survey, as cited in Anderson, et al. (1984)

[2]Respiratory volumes for children were calculated using age-group-specific rates from Anderson, et al. (1984), adjusting for mean body surface area when data for a specific age group were not available, assuming 40% rest, 30% light activity, 20% moderate activity, 10% heavy activity.

TABLE III-12

EXPOSURE LEVELS AND DOSES OF CDD/CDF TOXICOLOGIC EQUIVALENTS (TEQs) CALCULATED FOR AMBIENT AIR EXPOSURE SCENARIOS

Exposure Scenarios	Long-Term Average Air Concentrations[a] (pg/m^3 TEQs)		Dose to Receptors[b] (pg/kg/day TEQs)	
	Method A	Method B	Method A	Method B
Scenario 1--Fenceline Case	2.1	0.71	--	--
Infants (0-1 year)	--	--	0.22	0.077
Children:				
1-6 years	--	--	0.59	0.20
6-12 years	--	--	0.41	0.14
Adults (12-70 years)	--	--	0.16	0.055
Lifetime (0-70 years)	--	--	0.21	0.073
Scenario 2--Residential Area Case	0.34	0.15	--	--
Infants (0-1 year)	--	--	0.037	0.017
Children:				
1-6 years	--	--	0.098	0.044
6-12 years	--	--	0.068	0.031
Adults (12-70 years)	--	--	0.026	0.012
Lifetime (0-70 years)	--	--	0.035	0.016

Source: Calculated from ambient monitoring results as described in the text.

[a]In Scenario 1, the receptor is assumed to be downwind of source(s) 58% of the time, and receive "background" exposure when not downwind. In Scenario 2, the receptor is assumed to be downwind 33% of the time. The long-term average air concentration is calculated as the time-weighted average of the "upwind" (site 1) air concentration and either the averaged sites 2 and 4 air concentration (fenceline case) or site 3 air concentration (residential case).

[b]Assumes lifetime 24 hr/day exposures, respiratory volumes and body weights as described in Table III-11, 27% absorption of inhaled CDDs/CDFs by all age groups. The long-term average dose (D, pg/kg/day) due to air exposure is calculated as:

$$D = \frac{C(RV)(F_I)}{BW},$$

where

C = the long-term average air concentration (pg/m^3 TEQs),
RV = the average-specific respiratory volume (m^3/day), and
BW = the age-specific body weight (kg) for the exposed population.

7. Exposure Estimates from Incinerator Emissions Data

As discussed previously, there are several reasons why the available incinerator emissions data are less than ideal for use in developing exposure estimates. Aside from the small number of observations, and the variations in the quality and completeness of data gathered at various times by Dow and USEPA, it is clear that the patterns of CDD/CDF congeners found in the stack emissions during all of the sampling events are significantly different from the patterns observed in the ambient monitoring results (see Table III-8 and Figures III-2 through III-6. In addition, while the ambient data provide direct estimates of CDD/CDF levels at specific locations of interest for the exposure assessment, developing exposure estimates using the emissions data requires the use of air transport models, which add significantly to the level of uncertainty in the exposure estimates.

Despite these uncertainties, it is possible to develop exposure estimates for pollutants emitted by the incinerator in the manner just described. In the discussion that follows, the results of a recent study which employed the 1984 USEPA stack emissions data and the USEPA Human Exposure Modeling System (HEMS) to generate exposure estimates for CDDs/CDFs in Midland will be briefly reviewed for purposes of comparison with the exposure estimates generated using the ambient data, as described above. In addition, the 1987 Dow data will be used in a similar manner to develop exposure estimates that will help to illustrate how changes in emissions since 1984 may have affected incinerator-related exposure levels.

Cleverly (1986) used the 1984 USEPA stack emissions data to provide inputs to HEMS and developed estimates of the maximum total TEQ exposures associated with incinerator emissions. Where data for a specific congener was not available, e.g., 2378-TCDD, the corresponding average value for the 1984 Dow results was used. Using meteorologic data from 5 years of observations at Midland and an average concentration of 3.80 ng/m^3 TEQ in the incinerator stack gases ("A" method), it was estimated that the maximum annual ground-level CDD/CDF concentrations of 0.101 pg/m^3 TEQ would be achieved at points 0.6 miles north and northeast of the incinerator. These points lie in the same general direction from the incinerator as the fenceline scenario exposure point but are slightly closer to it. The modeled TEQ level (0.101 pg/m^3) is lower than the estimated exposures at the fenceline site (2.1 or 0.71 pg/m^3 TEQ, "A" or "B" method, respectively), and CDD/CDF intakes estimated at the site using the modeling results would also be correspondingly lower.

If, however, it is assumed that there is a "background" CDD/CDF level in the Midland area, which contributes to the total CDD/CDF levels observed downwind from the incinerator, then the model predictions are essentially the same as the observed levels for the residential sampling site. When the site 1 "background" is added to the model predictions, the total annual maximum predicted CDD/CDF level for the highest-concentration downwind location becomes 0.45 or 0.22 pg/m^3 ("A" or "B" method, respectively). These levels, while nearly the same as those measured at the residential site, are still somewhat lower than those measured at the fenceline sites. However, the difference between the two values is probably well within the expected range of variation for the model predictions and the measured CDD/CDF levels.

Substituting the 1987 Dow data into the HEMS model also gives lower exposure levels than estimated using ambient data. From the data in Table III-7, it can be calculated that the total TEQ levels measured by Dow in 1987 (0.606 pg/m^3 and 0.371 pg/m^3, "A" and "B" methods, respectively) would yield estimates of the maximum annual downwind concentration of 0.016 pg/m^3 TEQ and 0.010 pg/m^3 TEQ, respectively. These estimates assume conditions identical to those used by Cleverly to estimate downwind exposures; most importantly, perhaps, it assumes the same flow rate in the stack of the incinerator. In fact, data regarding stack flow rates for 1987 are not available at this time, thus adding to the uncertainty in this estimate. These levels would not add significantly to a "background" CDD/CDF level equivalent to that found at monitoring site 1.

The maximum ground-level concentration modeled using the HEMS system and either the 1984 USEPA or 1987 Dow data are thus lower than both the measured ambient concentrations at the closest corresponding monitoring locations (2 and 4) and the estimated long-term exposures derived from these data. Implications of these results will be discussed below.

8. Limitations of Air Exposure Assessment

Many factors contribute to the uncertainty surrounding the exposure estimates just discussed. The two major sources are the uncertainties and limitations associated with the ambient air data for CDDs/CDFs and the

uncertainties associated with the methods and models used to derive the exposure estimates from these data.

a. Data Limitations

A number of practical difficulties are inherent in the collection and analysis of samples for the detection and quantification of the wide range of CDDs/CDFs at the low levels encountered in this study. The collection efficiency of the high volume samplers used to gather these data has not, for example, been measured on an absolute basis for all the specific compounds being analyzed (Trembly and Amendola 1987). Analysis of similar compounds (specifically DDT) suggests that some of the lower molecular weight CDDs/CDFs may pass through the XAD resin cartridge to the backup PUF filter. Most of the ambient air samples were within satisfactory analytical recovery and precision targets on all three sampling days. Nine of the 45 samples had percent recoveries exceeding the 150% upper-bound target for $^{37}Cl_4$-HpCDD, indicating that sampling results for the hexa- and hepta-CDDs and CDFs, although deemed acceptable for this risk analysis, may actually be overestimated. The consistent patterns of monitoring results at the various monitoring sites suggest that the loss of lower-substituted CDDs/CDFs could not explain the differences in observed congener/homologue profiles at the different sites.

There is, however, some ambiguity in the definition of detection limits for the various homologues in the ambient air sampling data. Detection limits were set for each sample, following standard USEPA procedures, using the highest detection limit of all of the elements in the sampling train that were

analyzed (filter, XAD, PUF plug, etc.). This approach may add to the uncertainty in the exposure estimates, in that the detection limit for the sampling train as a whole could, in theory, be different from that calculated using the least sensitive element of the train.

The number of analytical chemistry standards available for use in this study was also limited. Therefore, estimates of the concentrations of some of the homologues have been made without benefit of homologue-specific, let alone congener-specific, standards. Only one isomer was used for a calibration standard for all isomers within a homologue series. This practice is based on the assumption that the response factors for all isomers in a homologous series are equal to that of the calibration isomer. On the whole, the available data do not appear to support the existence of any systematic error or bias in the analytical results for the ambient air sampling, although it is clear that these results are subject to a large degree of uncertainty.

The extent to which these data are representative of the actual CDD/CDF levels in ambient air at the location monitored depends upon many factors, including the meteorologic conditions during the sampling, variations in incinerator feed materials and operating parameters, conditions governing releases from other possible sources, and any other local conditions affecting CDD/CDF transport, persistence, and transformation in the air. With the exception of the prevailing wind direction, it is not possible, due to a lack of data, to incorporate quantitative considerations of any of these factors into the exposure assessment.

b. Limitations of Models and Methods Used to Estimate Exposures

The scenarios and assessment methods used to develop quantitative exposure estimates are designed, to the extent possible, to combine the available data with plausible, realistic assumptions, and to elucidate the impacts of various key assumptions by providing ranges of estimates where more than one assumption appeared reasonable. The exposure scenarios themselves are designed to reflect the exposure experience of two different potentially exposed populations, one living near the facility fenceline, in a direction directly downwind from the facility, the other further away and less directly downwind, based on the prevailing wind direction. The first represents an attempt to combine prudently but realistically conservative assumptions to derive an exposure estimate unlikely to be lower than that received by anyone near the facility. In fact, there are only a small number of residences as near to the incinerator and production areas and downwind from them as the two sampling locations which provided data for the assessment.

The second scenario is designed to more closely reflect conditions at a location closer to the center of population density in Midland, although still relatively near the facility. The monitoring station which provided the data for this assessment is, in fact, in a residential area, not far from the center of population density in the Midland area.

For both scenarios, the conservative assumption is made of full lifetime exposure. In addition, full 24-hour per day inhalation exposure is assumed. This assumption could be "conservative" or not, depending upon the proportion

of time spent by exposed individuals in other less (or more) heavily contaminated areas, and the extent to which exposures to potentially contaminated household dust were also occurring (Section III.F).

The exposure estimates were also developed using a model which assumed direct windborne transport of pollutants from sources at the Dow Midland facility to receptors near the various exposure points. Corrections were made, using site-specific meteorologic data, for seasonal variations in wind direction, based on the assumption that the observed air contamination was, in fact, either due to releases directly from the facility or due to "background" contamination represented by the monitoring results from site 1. If this assumption is not correct (if some of the CDD/CDF contamination observed at the monitoring locations comes from sources other than the facility, or from resuspension of contaminated soils near the specific monitoring sites), then the method used to correct for wind direction may underestimate exposures. If, as is possible, resuspended soil is contaminated primarily with the less toxic hepta-and octa-substituted congeners, then this factor may not introduce any significant bias into the analysis. Short-term variations in meteorologic patterns and local meteorologic conditions could also affect ambient air levels in ways not taken into account in this analysis.

Another important factor in developing exposure assessments for the two scenarios is the treatment of "non-detect" values in quantifying exposures. As noted previously, "non-detect" values were counted as being observations equal to the detection limits for the fenceline scenario and equal to one-half the detection limits for the residential area scenario. These approaches are often

employed in risk analysis when, as is the case here, the measured analytes are at or near analytical detection limits and there is additional reason for suspecting the presence of the non-detected pollutants. The basis for suspecting the presence of the non-detected congeners is well established in this case. The most toxicologically significant non-detected congeners/homologues (2378-TCDD and 2378-TCDF and, in one case, PeCDDs) are known to be constituents of the stack emissions, a major suspected source of the observed contamination. Also, 2378-TCDD and 2378-TCDF were detected in the samples from monitoring site 2 on September 8, 1984, which were reanalyzed by EMSL, at levels (0.49 pg/m^3) comparable to the detection limit obtained at that and other sites during the ambient sampling. The use of the two scenarios helps to illustrate the degree of uncertainty associated with the lack of knowledge about the actual levels of CDDs/CDFs present in the "non-detect" samples.

As discussed previously, "non-detects" account for less than 53% of the total TEQ exposures predicted for the fence-line scenario. They do account, however, for up to 87% of the TEQ exposure for the residential area exposure scenario calculated using the "B" method. This suggests that as a significant source of potential bias in the exposure estimates, the use of detection limit values for the scenario is not likely to be important, while for the residential scenario, the use of one-half the detection limit for "non-detects" could add appreciably to the uncertainty of the estimated exposures.

Taken together, the factors just discussed may exert a combined influence which would cause exposure estimates to err slightly on the side of

conservatism. That is, they could cause the estimated exposure to be slightly higher than values derived using other, less conservative methods. On the whole, however, it is not expected that there would be any major systematic bias in the estimates, since factors which contribute to conservatism (use of detection limits, one-half detection limits, assumption of full lifetime exposure) are at least partially counterbalanced by those factors (lack of congener- and some homologue-specific standards for the analytical data, assumptions of no indoor or non-site related exposures) which could result in underestimation of exposures.

The differences between the exposure levels estimated using the ambient data and those derived using the HEMS model and the incinerator stack emissions are not great enough to call either approach seriously into question. The difference between the predicted and measured levels is probably within the range of uncertainty associated with the modeling process, and in the chemical analyses, especially if any reasonable "background" level of CDD/CDF contamination in the Midland air is taken into account. These data should certainly not be interpreted to indicate that the incinerator accounts for only a small proportion of the total ambient exposures. While there is good reason to believe that there are sources of airborne CDDs/CDFs in the Midland area other than the Dow incinerator stack, the modeling results themselves do not provide conclusive evidence as to the identity or relative importance of these sources.

C. Soil

Surface soil sampling was conducted in the City of Midland during the period October 10-20, 1983, and at the Dow Midland facility on December 1, 1983 (USEPA 1985a). Samples obtained from the grounds of the Dow Midland facility were composites obtained from grids established at each sampling location. Both composite and grab samples were taken from around the inside perimeter of the Dow Midland facility. All samples were composites of soil obtained within 1 inch (25 mm) of the surface.

Sampling in the City of Midland was focused on the outside perimeter of the Dow Midland facility, and in public use and residential areas throughout the city. The residential sampling program included both open yard composite samples and composite samples taken at roof gutter downspouts or roof driplines. The downspout and dripline samples were obtained to help define the degree to which atmospheric deposition contributes to surface soil contamination in the Midland area.

1. CDD/CDF Concentrations in Soils

Two separate analytical programs were conducted by USEPA. Selected samples were analyzed for CDD and CDF homologues, and the results are presented in Tables III-13 and III-14. The majority of the samples were analyzed for 2378-TCDD only. These results are presented in Tables III-15, III-16, and III-17. Figure III-7 shows the locations that are referred to in the tables.

TABLE III-13

PCDDs and PCDFs
SITE #1 - Midland, Michigan Area
Surface Soil Samples

	Upwind		Dow Chemical In-Plant	
Sample No.:	13401	13395	13406	13412
Field ID.:	UPW-2-L	UPW-4-L	Station 5	Station 14
Location:	Pleasant View School	4853 W. Kent	Incinerator	West of 934 Building
PCDDs (DL)				
2378-TCDD	ND (0.004)	ND (0.004)	3.5 (0.039)	0.27 (0.007)
Total iso TCDDs	ND (0.004)			0.32
Total penta CDDs	ND (0.024)	ND (0.023)		0.24 (0.067)
Total hexa CDDs	ND (0.024)	ND (0.023)		4.0 (0.067)
Total hepta CDDs	0.15 (0.024)	0.17 (0.034)		75.0 (0.9)
OCDD	0.34 (0.026)	0.33 (0.034)		375.0 (1.3)
PCDFs (DL)				
2378-TCDF	ND (0.004)	ND (0.004)	0.45 (0.06)	0.027 (0.007)
Total TCDFs				
Total penta CDFs	ND (0.008)	ND (0.008)		0.90 (0.14)
Total hexa CDFs	ND (0.022)	ND (0.023)		3.1 (0.13)
Total hepta CDFs	ND (0.031)	ND (0.028)		15.4 (0.38)
OCDF	ND (0.051)	ND (0.045)		8.6 (0.48)

Notes: (1) Concentrations of PCDDs, PCDFs, and detection levels (DL) reported in parts per billion (ppb).

Source: Soil Survey (U.S. EPA 1985a)

TABLE III-14

PCDDs and PCDFs
SITE #1 - Midland, Michigan Area
Surface Soil Samples

	Public Use Areas					
Sample No.:	13374	13392	13393	13375	13391	13394
Field ID.:	P-5-L	P-6-L	P-7-L	P-9-L	P-10-L	P-11-L
Location:	County Line Rd.	Mapleton School	Longview School	Virginia Park	Central School (ball diamond)	Bullock School
PCDDs (DL)						
2378-TCDD	0.003 (0.001)	0.015 (0.003)	0.078 (0.003)	0.076 (0.003)	0.012 (0.003)	0.11 (0.002)
Total iso TCDDs	ND (0.001)	0.040	0.17	0.29	0.040	0.22
Total penta CDDs	ND (0.014)	ND (0.035)	interference	0.10 (0.018)	ND (0.034)	0.12 (0.022)
Total hexa CDDs	0.067 (0.007)	0.063 (0.035)	0.34 (0.02)	0.24 (0.018)	0.086 (0.034)	0.41 (0.022)
Total hepta CDDs	0.35 (0.013)	0.38 (0.028)	2.3 (0.055)	0.41 (0.093)	0.35 (0.031)	2.4 (0.042)
OCDD	3.1 (0.096)	0.86 (0.027)	7.0 (0.068)	12.0 (1.5)	0.68 (0.031)	7.0 (0.052)
PCDFs (DL)						
2378-TCDF	ND (0.002)	ND (0.005)	0.013 (0.007)	0.013 (0.002)	ND (0.005)	0.015 (0.003)
Total TCDFs	ND (0.002)					
Total penta CDFs	ND (0.01)	ND (0.008)	ND (0.025)	0.040 (0.01)	ND (0.007)	0.11 (0.017)
Total hexa CDFs	ND (0.01)	ND (0.024)	0.26 (0.036)	0.064 (0.01)	ND (0.029)	0.17 (0.037)
Total hepta CDFs	0.065 (0.02)	0.14 (0.043)	0.72 (0.021)	0.50 (0.034)	0.16 (0.062)	0.82 (0.045)
OCDF	0.044 (0.023)	0.10 (0.071)	0.64 (0.037)	0.37 (0.049)	0.11 (0.070)	0.66 (0.045)

Notes: (1) Concentrations of PCDDs, PCDFs, and detection levels (DL) reported in parts per billion (ppb).

Source: Soil Survey (U.S. EPA 1985a)

TABLE III-15

2378-TCDD
Dow Chemical - Midland Plant
In-Plant Surface Soil Samples

Sample Number	Field Identification	Location	2378-TCDD (DL) (ppb)		% Recovery	% Solids
13404	Station 1	South of 492 Building	0.018	(0.003)	71	92.9
14176	Station 2	South of 1005 Building; Southwest of 703 Building	0.074	(0.005)	52	96.2
14190	Station 3	South of 703 Building	0.42	(0.013)	90	96.1
14192	Station 4	West of 703 Building	0.020	(0.003)	63	95.0
13406	Station 5	Southwest of 956 Building; East of 703 Building	3.50	(0.039)	100	96.4
14180	Station 6	Northwest of 1159 Building; North of Shot Pond	0.13	(0.013)	103	98.5
14178	Station 7	11th and J Streets - Northwest Corner	4.60	(0.083)	101	96.3
14193	Station 8	8th and G Streets - Northwest Corner at Steam Pipeline	0.15	(0.010)	65	99.1
14194	Station 9	Northwest of 1050 Building at F Street	0.010	(0.003)	61	90.2
13405	Station 10	South of 543 Building; West of 14th Street	0.045	(0.005)	80	99.3
14187	Station 11	16th and G Streets - Southwest Corner	0.44	(0.016)	119	99.6
14182	Station 12	16th and G Streets - Northwest Corner	0.46	(0.012)	94	96.0
13407	Station 13	17th and G Streets - Northwest Corner	0.22	(0.007)	51	99.3
13412	Station 14	West of 934 Building	0.27	(0.007)	100	100.0
13413	Station 15	South and East of 874 Building North of RR Tracks	25.0	(0.50)	66	85.5
			[36.0]R	(0.28)		

Notes: (1) 2378-TCDD concentrations and detection levels (DL) reported in parts per billion (ppb).
(2) % Recovery - Recovery of internal standard (Cl^{37} 2378-TCDD or ^{13}C 2378-TCDD) expressed as percent.
(3) % Solids - Solids content of sample determined after sample homogenization, expressed as percent. Analytical results not adjusted for moisture content.
(4) [ ]R = Repeat analysis of same sample.

Source: Soil Survey (U.S. EPA 1985a)

TABLE III-16

2378-TCDD
SITE #1 - Midland, Michigan Area
Surface Soil Samples

Sample Number	Field Identification	2378-TCDD (DL) (ppb)	% Recovery	% Solids
Upwind Areas				
13354	UPW-1-L	ND (0.002)	82%	94.9%
13343	UPW-1-1	0.006 (0.002)	76%	98.6%
13401	UPW-2-L	ND (0.004)	90%	94.0%
13395	UPW-4-L	ND (0.004)	84%	98.1%
13342	UPW-4-D	0.009 (0.001)	84%	84.2%
Track Out and Perimeter Samples - Dow Chemical-Midland Plant				
13353	TO-4-G	0.011 (0.003)	52%	99.3%
13360	TO-6-G	0.25 (0.018)*	21%	99.0%
13367	TO-9-G	0.014 (0.002)	68%	99.0%
14188	PER-2-L	0.31 (0.014)	84%	82.8%
14177	PER-2-1	0.069 (0.003)	55%	99.0%
14191	PER-5-L	0.21 (0.008)	62%	99.2%
13402	PER-8-L	0.010 (0.005)	104%	99.0%
13389	PER-9-G	2.03 (0.042)	72%	95.6%
14181	PER-10-L	0.040 (0.002)	73%	97.1%
Public Use Areas				
13362	P-1-L	0.019 (0.002)	64%	99.5%
13364	P-2-L	0.028 (0.002)	64%	89.7%
13374	P-5-L	0.003 (0.001)	104%	99.7%
13392	P-6-L	0.015 (0.003)	86%	80.2%
13393	P-7-L	0.078 (0.003)	88%	98.8%
13340	P-8-L	0.17 (0.006)	94%	96.5%
13375	P-9-L	0.076 (0.003)	58%	89.2%
13391	P-10-L	0.012 (0.003)	92%	96.6%
13394	P-11-L	0.108 (0.002)	81%	100.0%

Source: Soil Survey (U.S. EPA 1985a)

TABLE III-17

2378-TCDD
SITE #1 - Midland, Michigan Area
Surface Soil Samples

Sample Number	Field Identification	2378-TCDD (DL) (ppb)	% Recovery	% Solids
Residential Areas				
13007	A-1-L	0.075 (0.006)	102%	83.0%
13008	A-1-1	0.090 (0.008)	52%	89.7%
13101	A-3-L	0.009 (0.002)	102%	88.5%
13102	A-3-1	0.112 (0.008)	92%	76.5%
13328	B-1-L	0.076 (0.007)	100%	93.2%
13305	B-1-1	0.16 (0.006)	63%	97.0%
13306	B-3-L	0.020 (0.001)	71%	97.8%
13325	B-3-1	0.27 (0.013)	74%	87.1%
13103	B-4-L	0.019 (0.002)	88%	84.7%
13104	B-4-1	0.028 (0.004)	54%	88.1%
13317	C-1-L	0.026 (0.001)	86%	78.7%
13303	C-1-1	0.054 (0.003)	64%	84.4%
13314	C-3-L	0.012 (0.001)	100%	98.6%
13307	C-3-1	0.24 (0.015)	71%	98.1%
13105	C-4-L	0.024 (0.002)	86%	91.0%
13106	C-4-1	0.032 (0.005)	88%	88.9%
13318	D-1-L	ND (0.001)	94%	84.0%
13331	D-1-D	0.024 (0.001)	100%	99.5%
13319	D-2-6	0.028 (0.001)	92%	94.9%
13316	D-3-L	0.018 (0.001)	100%	90.9%
13329	D-3-1	0.031 (0.004)	86%	99.8%
13312	E-1-L	0.026 (0.003)	96%	97.5%
13330	E-1-1	0.049 (0.001)	80%	97.0%
13301	E-3-L	0.009 (0.001)	75%	97.6%
13304	E-3-1	0.020 (0.001)	70%	99.1%
13302	F-1-L	0.013 (0.001)	73%	99.0%
13313	F-1-1	0.013 (0.001)	92%	96.8%
Miscellaneous				
14175	Sludge	0.021 (0.008)*	49%	88.7%

TABLE III-17 (Continued)

2378-TCDD
SITE #1 - Midland, Michigan Area
Surface Soil Samples

Notes: (1) 2378-TCDD concentrations and detection levels (DL) reported in parts pe
billion (ppb).
(2) % Recovery - Recovery of internal standard (Cl^{37} 2378-TCDD or ^{13}C 2378-TCDD
expressed as percent.
(3) % Solids - Solids content of sample determined after sample homogenization
expressed as percent. Analytical results not adjusted for moisture content
(4) Field identification of samples:

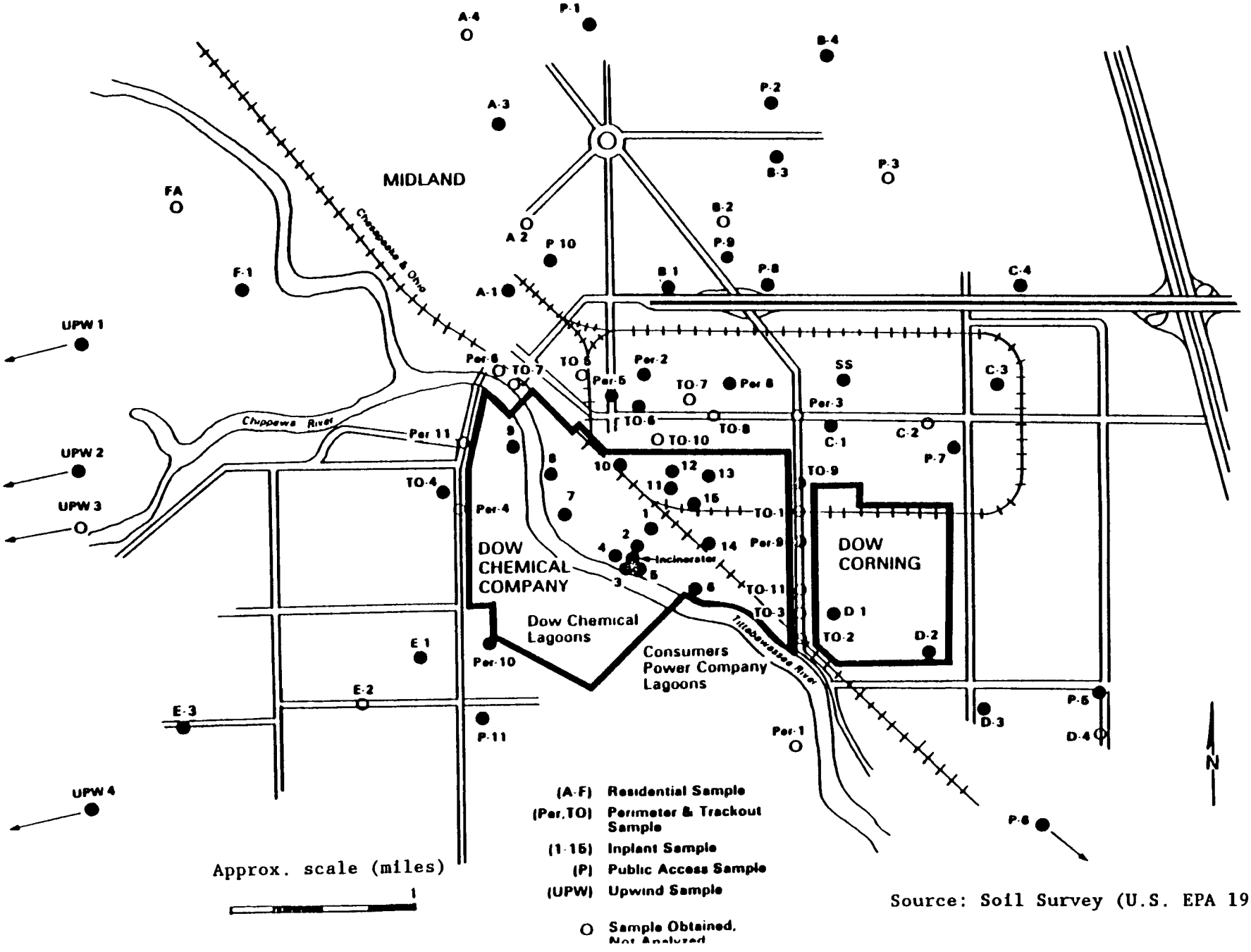
Location		Type	
UPW	- Upwind	L	- Yard, lawn, or open area composite
TO	- Track Out	1 or D	- Downspout or dripline composite
PER	- Perimeter	G	- Open area grab sample
P	- Public Use		
A-K	- Residential		

* Data not valid. Quality assurance objective not achieved.

Source: Soil Survey (U.S. EPA 1985a)

FIGURE III-7

SURFACE SOIL SAMPLING LOCATIONS: MIDLAND, MICHIGAN



Source: Soil Survey (U.S. EPA 1985a)

The upwind residential samples were located west-southwest of the Dow Midland facility. Five composite samples were analyzed for 2378-TCDD. The three samples obtained from the lawns did not contain detectable levels (detection limits ranged from 2-4 ppt). The two samples collected near downspouts or roof driplines contained 6 and 9 ppt of 2378-TCDD. Two upwind lawn samples were analyzed for CDD/CDF homologues. The soils contained HpCDDs ranging from 150 to 170 ppt and OCDD ranging from 330 to 340 ppt. No other CDD homologues and no CDF homologues were detected. The results are displayed graphically in Figure III-8.

Results of analyses for 2378-TCDD of 15 surface soil samples obtained from inside the Dow Midland facility revealed concentrations ranging from 10 ppt to 30,000 ppt (mean 2,700 ppt; for discussion of use of arithmetic means, see p. III-8). All samples contained detectable concentrations of 2378-TCDD. Eight samples were obtained from the perimeter of the facility, and 2378-TCDD concentrations ranged from 10 ppt to 2,030 ppt (mean 340 ppt). All eight samples were positive for the presence of 2378-TCDD. Concentrations of CDD/CDF homologues were determined in one soil sample. All homologues were detected, except TCDFs, for which an analysis was not performed. The results are presented graphically in Figure III-9, and differ noticeably from the upwind samples presented in Figure III-8 due to the presence of CDFs and less chlorinated CDD congeners.

Public use areas of Midland downwind (north-northeast) of the Dow Midland facility were sampled, and all samples contained detectable levels of 2378-TCDD. Nine samples analyzed for 2378-TCDD contained concentrations ranging

FIGURE III-8

PATTERN OF CDDs/CDFs DETECTED IN SOILS UPWIND OF THE DOW MIDLAND FACILITY

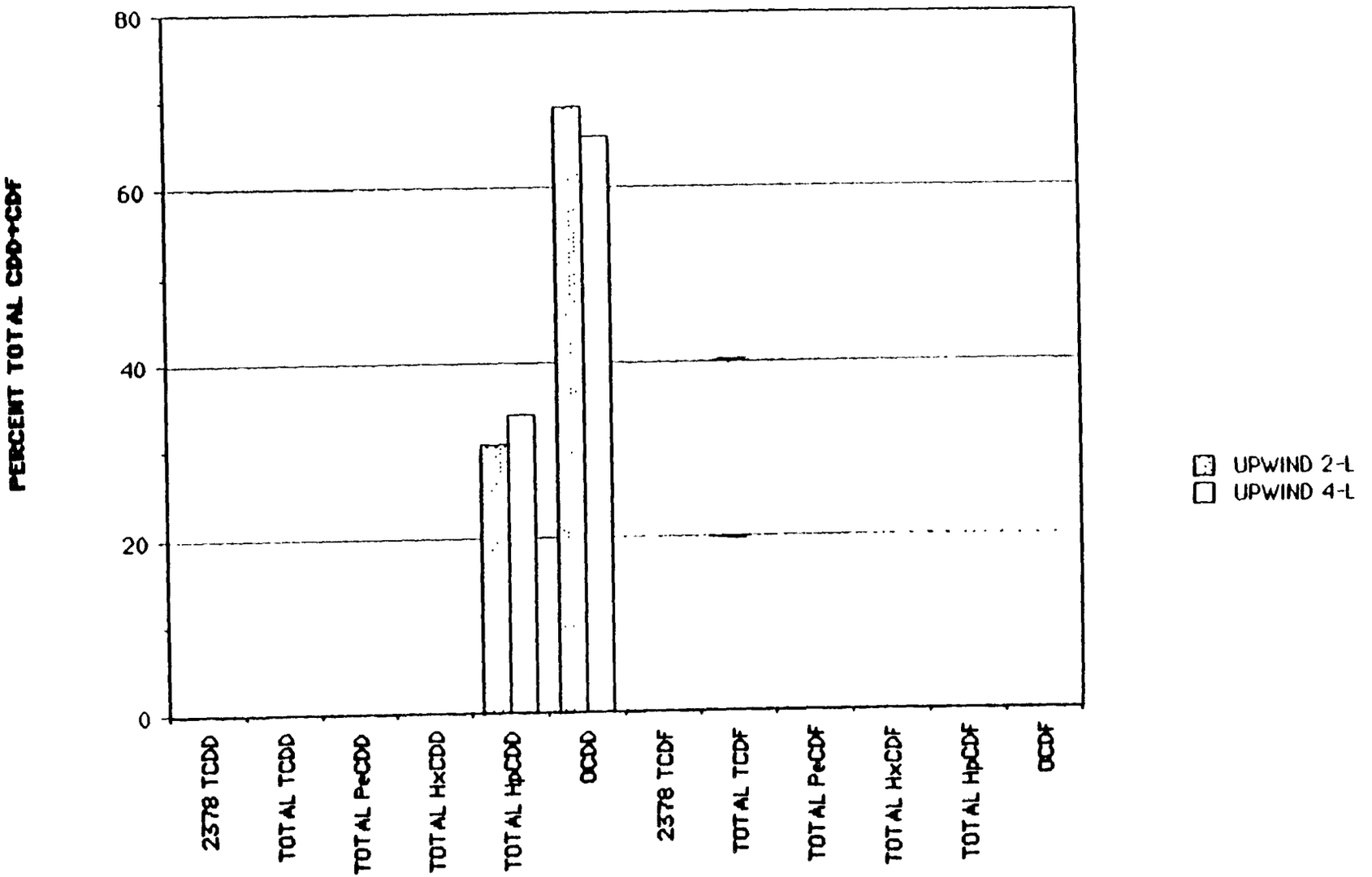
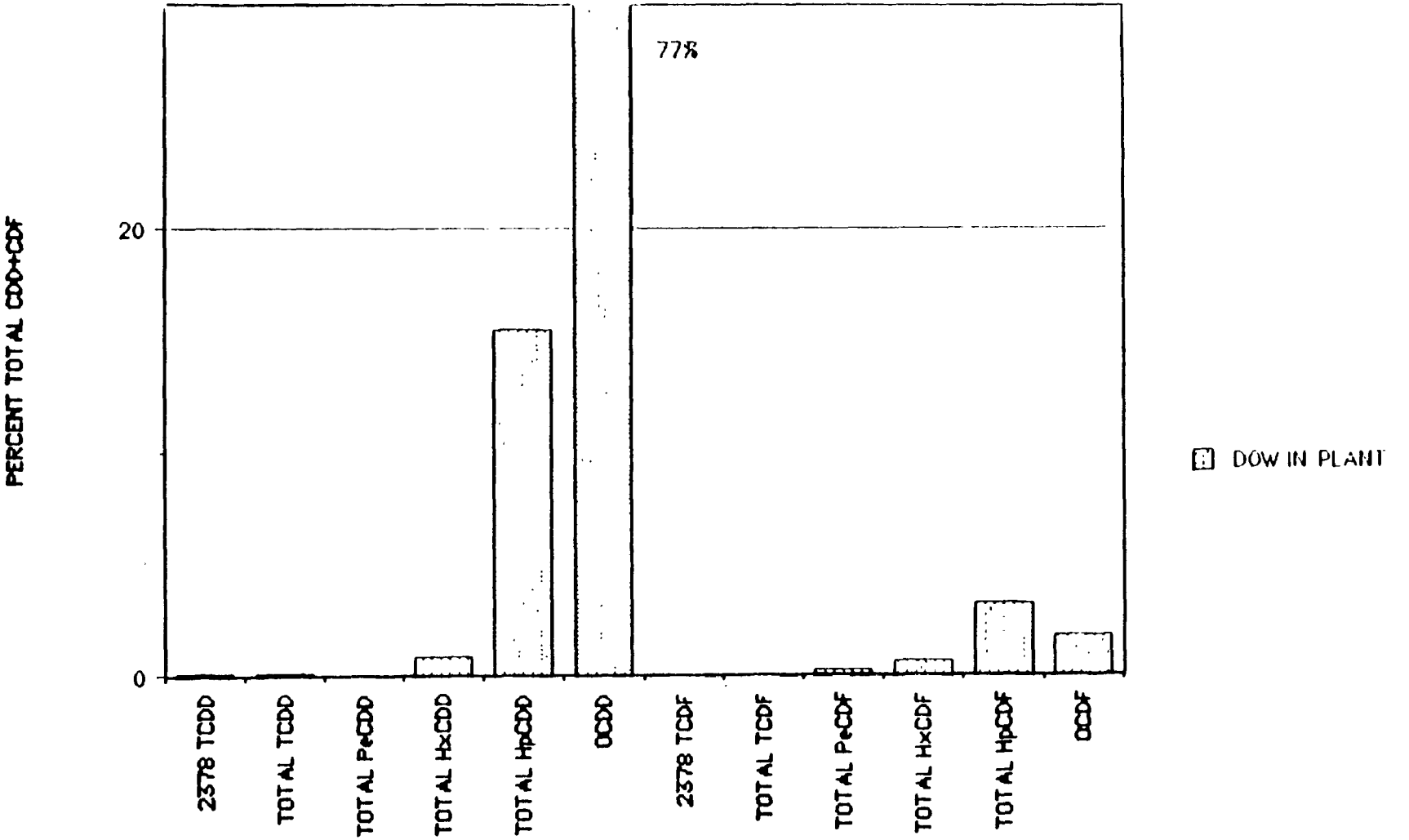


FIGURE III-9

PATTERN OF CDDs/CDFs DETECTED IN SOILS OF THE DOW MIDLAND FACILITY



from 3 ppt to 170 ppt (mean 57 ppt). Of the above samples, six were analyzed for CDD/CDF homologues, and the results are presented in Figure III-10. The pattern of CDD/CDF congeners detected in the public use areas is similar to that in soil samples obtained from the Dow Midland facility.

In the residential areas of Midland downwind of the Dow Midland facility, lawn and downspout/dripline samples were obtained. Twelve of thirteen lawn composite samples analyzed were positive for the presence of 2378-TCDD, and concentrations ranged from undetected to 76 ppt (mean of 25 ppt, assigning one-half of the detection limit to the one "not detected" report). All of the 13 downspout/dripline samples contained detectable concentrations of 2378-TCDD, ranging from 13 ppt to 270 ppt (mean of 86 ppt). With one exception (sample F-1), all of the downspout/dripline samples contained higher concentrations of 2378-TCDD than did the lawn samples. Analyses for CDD/CDF homologues were not performed on the residential samples.

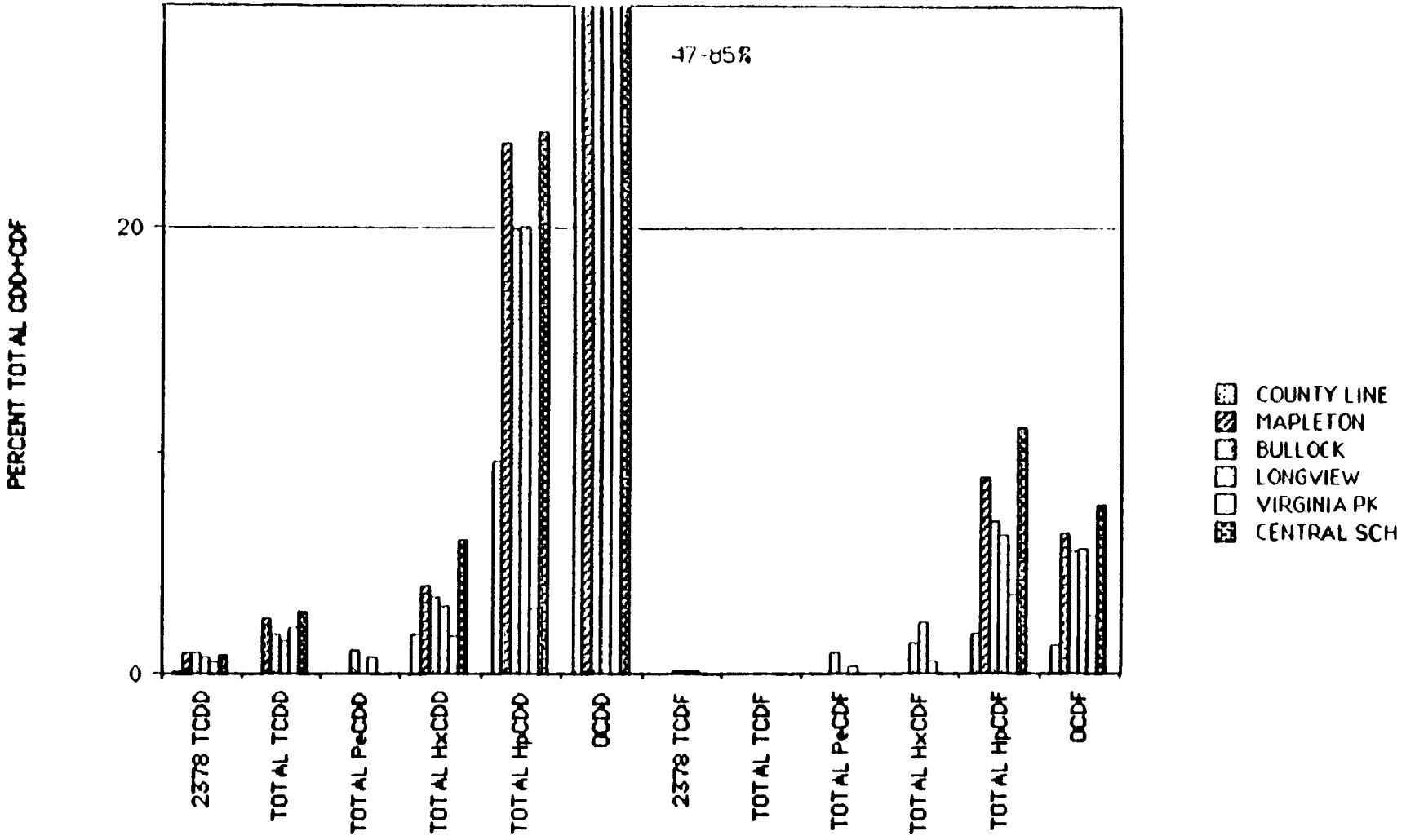
In general, the analytical results of the surface soil sampling program are consistent with the hypothesis that the Dow Midland facility is the primary source of CDD/CDF compounds detected in environmental media in the Midland area. The following results contribute to this hypothesis.

- Upwind, residential lawn samples contained primarily HpCDDs and OCDD, a pattern similar to that seen in natural areas and in cities that do not contain extensive chemical manufacturing facilities (EPA 1985a). Soil samples from upwind lawns did not contain detectable quantities of 2378-TCDD. The 2378-TCDD isomer could be detected only at locations that received the composite, concentrated products of atmospheric deposition on roof surfaces, such as roof downspouts and driplines.

- All samples obtained from the grounds of the Dow Midland facility contained detectable concentrations of 2378-TCDD and also contained detectable concentrations of all CDD/CDF homologues for which

FIGURE III-10

PATTERN OF CDDs/CDFs DETECTED IN MIDLAND PUBLIC USE AREA
SOILS DOWNWIND OF THE DOW MIDLAND FACILITY



analysis was performed. The HpCDD and OCDD congeners that were dominant in upwind samples were present at the Dow Midland facility at concentrations that were 2-3 orders of magnitude higher than the upwind samples.

- All samples, except one, from the public use and residential areas of Midland downwind from the Dow Midland facility contained detectable concentrations of 2378-TCDD. The downspout/dripline samples generally contained higher concentrations of 2378-TCDD than did the lawn samples. The concentrations of the CDD/CDF homologues in the public use areas were about 1-2 orders of magnitude less than the concentrations in the Dow Midland facility soils. However, the downwind public use-area samples contained concentrations of HpCDDs and OCDD that were an order of magnitude higher than in samples that were upwind of the Dow Midland facility. The downwind samples also contained CDD/CDF homologues that were not detected in upwind samples, but were detected in the Dow Midland facility soils.

Table III-18 presents calculated TEQs (toxicity equivalents of 2378-TCDD; see Section II) for the CDDs/CDFs measured at the locations discussed above. These calculations use the "B-method" (USEPA 1987d), in which it is assumed that all CDD/CDF congeners are equally likely to occur and congeners are allocated to 2378-substituted and non-2378-substituted categories in proportion to the number of each type of congener within each homologue group (except TCDDs, for which these results are available). Based upon the limited number of samples that received homologue-specific analyses, a significant contribution to the TEQs comes from 2378-TCDD in the Dow Midland facility and Midland public use area samples. The TEQs were greater than the 2378-TCDD concentrations by a factor of 1.4 in the Dow Midland facility samples and 1.1 in the Midland public use area samples. Because 2378-TCDD contributes such a large fraction of the TEQs, use of the "A-method" gives very similar estimates and the results are not presented separately here.

The second section of Table III-18 shows estimates of TEQs based on all of the soil samples, including those which are analyzed only for 2378-TCDD. As in Clark (1985), these estimates were derived by assuming that the ratio of TEQ/2378-TCDD in these latter samples would have been approximately the same as that derived for the soil samples in the first section of Table III-18. These ratios are then multiplied by the mean concentration of 2378-TCDD in the soil samples from the same general location to yield the estimates of TEQ listed in the right-hand column. "Average" residential soil concentrations of 2378-TCDD and TEQs are calculated by assuming that the downspout/dripline-contaminated areas represent 10 percent of the area of the yard (Clark 1985). The TEQs calculated in Table III-18 will be used in the subsequent exposure assessment.

2. Populations at Risk and Exposure Assumptions

Children may ingest soil by playing or crawling on their hands and then placing their hands in their mouths. Some children even directly eat soil, a behavior known as pica. Older children are less likely to exhibit this behavior. Adults may be exposed to soil contaminants by inadvertent ingestion of soils resulting from smoking or eating with contaminated hands. Exposure to soil is difficult to quantify due to the uncertainties caused by individual behavioral differences. As a result, the exposure assumptions will be defined as ranges, incorporating an upper and lower estimate of exposure.

LaGoy (1987) discusses the assumptions used to estimate the quantities of soil that could be inadvertently ingested by various age groups. The most likely population at risk is younger children. For 1 to 6-year-old children,

TABLE III-18

2378-TCDD TOXICITY EQUIVALENTS (TEQs)
SURFACE SOIL SAMPLES
(ppt)

A. Isomer/Homologue-Specific Analysis

Sample	Number of Samples	Mean Concentration of 2378-TCDD	TEQ	Ratio of TEQ/ 2378-TCDD
Upwind lawn	2	ND (2)[1]	ND (3)	(1.5)
Dow-Midland facility	1	270	390	1.4
Downwind public use area	6	49	53	1.1

B. Estimates of TEQs From Analyses For 2378-TCDD

Sample	Number of Samples	Mean Concentration of 2378-TCDD	Ratio of TEQ/ 2378-TCDD[2]	Estimated TEQ[3]
Upwind				
Lawn	3	ND (2)[1]	(1.5)	ND (3)
Downspout/dripline	2	8	1.5	12
Dow Facility				
Plant	15[5]	2,700[4]	1.4	3,800
Perimeter	8[5]	340	1.4	480
Downwind				
Public use area	9[6]	57	1.1	63
Residential lawn	13[6]	25	1.1	28
Residential downspout	13[6]	86	1.1	95
Residential Average[7]	-	31	1.1	34

[1] One-half of the detection limit used to estimate concentrations.
[2] From Part A of the table.
[3] Product of third and fourth columns.
[4] Includes samples from locations that have since been remediated.
[5] Sample TO-6-G rejected for QA/QC reasons.
[6] Sample D-2-6 could not be identified as to type of sample and was not included.
[7] "Average" residential soil concentrations of 2378-TCDD are calculated by the method of Clark (1985), assuming that the downspout/dripline-contaminated areas represent 10% of the area of the yard.

incidental ingestion of 500 and 100 mg/day of soil are the upper and lower estimates, respectively. For 0 to 1 and 6 to 12-year-old children, ingestion rates of 250 and 50 mg/day are assumed. For older children and adults, ingestion rates of 100 and 25 mg/day are estimated.

Although children who exhibit pica may ingest significantly higher quantities of soil, these children are assumed to comprise a small percentage of the children in the age group of concern. Dermal absorption of CDDs/CDFs from soil and dust is not considered since the absorption and therefore dose received by this route are expected to be one or two orders of magnitude below the exposure from soil ingestion (Poiger and Schlatter 1980).

The actual duration of exposure to outdoor soils varies, but a "severe worst-case" for most situations has been developed by USEPA (1984b) as 247 days/year by assuming that, on the average, soil in the northern United States remains frozen 118 days each year. For the purposes of this assessment, it will be assumed that children (less than 12 years old) will be exposed 250 days/year as the upper estimate, and about half this number, or 125 days/year, for the lower estimate. Adults are estimated to be exposed via yard work approximately once each week during the months of May-October (25 days/yr), or, for avid gardeners, 4 days per week (100 days/year) during the same period.

It is also assumed that the most probable location of periodic exposure of younger children and adults is the individual's residence. Residential samples collected downwind of the Dow Midland facility did not receive isomer/homologue-specific analyses for CDDs/CDFs. The estimated TEQs from

Table III-18 for downwind "average" residential yards are used to estimate chemical intakes resulting from exposure of younger children and adults to soils. For older children (6-12 years), it is assumed that they are more likely to play in parks, and be exposed to soils in locations other than their residences. Chemical intakes resulting from exposure of older children to soils will be calculated using the average of the "public use" TEQs and the "average" residential TEQs.

McConnell et al. (1984) and Rumbaugh et al. (1984) investigated the absorption of 2378-TCDD from soil after ingestion by administering contaminated soil from Times Beach and Minker-Stout, Missouri, to guinea pigs and rats. The soil was suspended in water and administered by gavage. The toxic responses (death in guinea pigs, AHH induction in rats) and tissue residues were compared with those observed when similar quantities of 2378-TCDD were administered in corn oil. The relative responses suggested that about one-third as much 2378-TCDD was absorbed from soil as from corn oil by guinea pigs, and 50-100 percent (mean 84%) as much 2378-TCDD was absorbed from soil as from corn oil by rats. Other studies have suggested that when 2378-TCDD is administered to rats in feed (Fries and Marrow 1975) or in ethanol (Poiger and Schlatter 1980), between 50% and 70% is absorbed into the body. Hence these results suggest that percentage absorption from ingested Times Beach soil is about 20% by guinea pigs and about 50 percent by rats.

Umbreit et al. (1985) fed contaminated soil from an industrial site in New Jersey to guinea pigs and found no deaths or toxic signs; their data suggest that considerably less than half as much 2378-TCDD was absorbed from

contaminated soils collected in the field as from soils to which 2378-TCDD was added in the laboratory. Umbreit et al. (1986) fed 2378-TCDD-contaminated soil from Newark, New Jersey and Times Beach, Missouri to guinea pigs. Missouri soils were observed to be toxic (LD_{50} <10 ug/kg) while Newark soils were not toxic at comparable concentrations. USEPA (1984b) recommended a range of 20-26 percent for absorption of 2378-TCDD from ingested soils based on data of Poiger and Schlatter (1980). However, all the studies cited above (McConnell et al. 1984, Rumbaugh et al. 1984, Umbreit et al. 1985, 1986, Poiger and Schlatter 1980) were of soils in which 2378-TCDD was present in mixtures with other organic compounds or soils to which 2378-TCDD had been added in solution. It is not clear that such studies will provide reliable measures of bioavailability in circumstances such as those prevailing at Midland, where most of the CDDs/CDFs would have been deposited onto the soil from the air, probably mostly attached to fly ash particulates. It is not known to what extent the CDDs/CDFs would subsequently have been desorbed from the fly ash particulates and resorbed onto the soil, but it is likely that at least some of the CDDs/CDFs would remain attached to fly ash.

Bonaccorsi et al. (1983) performed an experiment with contaminated soil from Seveso, Italy, and found that 32% as much 2378-TCDD was absorbed from the soil as from an alcohol-water solution after ingestion by rabbits. The soil at Seveso was contaminated by deposition from the air, but this took place in a "toxic cloud" released in an industrial accident, and is unlikely to be representative of deposition on fly ash.

The most relevant study of bioavailability of CDDs/CDFs from fly ash is that of Van den Berg et al. (1983). These authors prepared diets containing either fly ash from a municipal incinerator or toluene extracts of this fly ash. These diets were fed to rats for 19 days, at which time the rats were killed and their livers were analyzed for CDD/CDF homologues and for selected congeners. Although Van den Berg et al. (1983) did not report estimates of bioavailability, such estimates can be derived from their Table 6, which reports percentages of 11 congeners (mostly 2378-substituted) retained in livers of the rats. These data suggest that, relative to the rats fed food containing toluene extracts of the fly ash, rats fed food containing whole fly ash accumulated about 24% of the TCDDs and TCDFs, 10% of the PeCDDs and PeCDFs, and 7% of the HxCDDs and HxCDFs. These percentages are estimates of relative bioavailability; the data of Fries and Marrow (1975) indicated that rats fed food contaminated with 2378-TCDD via a solvent absorbed between 50 and 70 percent (mean, 55 percent) of the quantity administered. Assuming the same range of values would apply to other CDDs/CDFs, estimates of absolute bioavailability from fly ash would be about 13% for TCDDs/TCDFs, 5% for PeCDDs/PeCDFs, and 4% for HxCDDs/HxCDFs. Since TCDDs and TCDFs accounted for most of the TEQs for soil samples at Midland (Tables III-13 and III-18), an overall estimate of bioavailability for the TEQs found in soil would be about 12%. This is about half the range of values (20-26%) suggested for bioavailability from soil by USEPA (1984b). Since it is not clear to what extent the CDDs/CDFs would have been desorbed from fly ash particles and resorbed onto soil paricles at Midland, this report uses an intermediate value of 18% as an estimate of bioavailability for the "lower estimate" exposure scenario. For the "upper estimate" exposure scenario, a value of 40% is used,

based on the higher value derived for Times Beach soil (see above and USEPA 1984b). These assumptions are listed in Table III-19.

Intake estimates for soil ingestion are calculated as follows:

$$\text{pg/kg/day} = \frac{(C_s)(I)(E)(X)(A)}{(BW)(D)}$$

where

C_s = Chemical concentration in soil (pg/g or ppt)
I_s = Amount of soil ingested (mg/day)
E = Number of days of exposure (days/yr)
X = Conversion factor ($g/10^3$ mg)
A = Relative absorption rate (percent/100)
BW = Average body weight (kg)
D = 365 days/yr

Table III-20 presents the amounts of 2378-TCDD and estimated TEQs ingested under the assumptions discussed above for soil concentrations and ingestion rates.

The estimates of intake tabulated in Table III-20 represent estimates of the absolute amounts of CDDs/CDFs taken into the body of the exposed individuals. As discussed earlier in Section III.B.6, these estimates require further adjustment when they are compared with the RfD and HAs, because the latter are expressed in terms of <u>adminstered</u> dose. In Chapter IV, an additional factor will be applied to the estimates of <u>absorbed</u> dose tabulated in Table III-20, to make them commensurable with the RfD and HAs.

TABLE III-19

ASSUMPTIONS USED WHEN CALCULATING INTAKES OF CDDs/CDFs
BY RESIDENTS EXPOSED TO SOILS

Parameter	Lower Estimate	Upper Estimate
Exposure events		
Children (0-12 years)	125 days/yr	250 days/yr
Adults (12-70 years)	25 days/yr	100 days/yr
Period of exposure:		
Children aged 0 to 1 year	1 year	1 year
Children aged 1 to 6 years	5 years	5 years
Children aged 6 to 12 years	6 years	6 years
Ages 12 and greater	58 years	58 years
Average weight over period of exposure:		
Children aged 0 to 1 year	8 kg	8 kg
Children aged 1 to 6 years	15 kg	15 kg
Children aged 6 to 12 years	30 kg	30 kg
Ages 12 and greater	70 kg	70 kg
Incidental ingestion of soil:		
Children aged 0 to 1 year	50 mg/day	250 mg/day
Children aged 1 to 6 years	100 mg/day	500 mg/day
Children aged 6 to 12 years	50 mg/day	250 mg/day
Ages 12 and greater	25 mg/day	100 mg/day
Concentration of CDDs/CDFs in soil:		
Younger Children (0-6) and Adults (12-70)	34 ppt (Downwind Residential Average TEQs)	34 ppt (Downwind Residential Average TEQs)
Older Children (6-12)	49 ppt (50% Downwind Residential Average TEQs and 50% Public Use Area TEQs)	49 ppt (50% Downwind Residential Average TEQs and 50% Public Use Area TEQs)
Fraction of CDDs/CDFs absorbed from ingested soils	0.18	0.40

TABLE III-20

INTAKES OF CDDs/CDFs ASSOCIATED WITH EXPOSURE OF RESIDENTS TO SOILS DOWNWIND OF THE DOW MIDLAND FACILITY

Age Group (Years)	Assumed Body Weight (kg)	Soil Ingested (mg/day)		Frequency of Exposure (days/yr)		Soil Concentration (pg/g)		Dose Rate (pg/kg-day)			
								2378-TCDD		TEQ	
		Lower Estimate	Upper Estimate	Lower Estimate	Upper Estimate	2378-TCDD	TEQ	Lower Estimate	Upper Estimate	Lower Estimate	Upper Estimate
0 to 1	8	50	250	125	250	31	34	0.012	0.27	0.013	0.29
1 to 6	15	100	500	125	250	31	34	0.013	0.28	0.014	0.31
6 to 12	30	50	250	125	250	45	49	0.0045	0.10	0.0050	0.11
12 to 70	70	25	100	25	100	31	34	0.00014	0.0049	0.00015	0.0053
Child average (0-12 yrs)								0.0087	0 19	0.0094	0.21
Lifetime Average (0-70 yrs)								0.0016	0.036	0.0017	0.040

3. Data Limitations

The soil samples obtained from residences downwind of the Dow Midland facility were analyzed for the 2378-TCDD isomer only. The soil exposure assessment was therefore performed using measured concentrations of 2378-TCDD and estimated concentrations of TEQs. A review of the data shows that the large majority of the estimated TEQ for lawn, downspout, and public use area samples is contributed by the measured 2378-TCDD, however.

No information is available on time trends in soil contamination, i.e., whether the concentrations of CDDs/CDFs are increasing or decreasing with time. The available data were collected October-December 1983. Limited data summarized in section III.A (above) suggest that emissions of CDDs/CDFs from the Dow Midland facility waste incinerator were much higher on days for which sampling was conducted in 1983 than in 1984 or 1987. Hence, it is likely that atmospheric inputs of CDDs/CDFs into Midland soils have decreased since 1983. However, the rate of decrease of the soil concentrations of CDDs/CDFs, if any, will be determined by the rate at which the CDDs/CDFs in the soil will be lost by degradation or other processes.

The persistence of CDDs/CDFs in Midland soils is poorly characterized but likely to be variable. Concentrations of CDDs/CDFs in the top few millimeters of soil or on the surfaces of plants, buildings, etc. can be expected to be depleted due to environmental degradation processes (i.e., photolysis, resuspension, volatilization) (Thibodeaux and Lipsky 1985). The CDDs/CDFs that are in deeper layers of soil are more resistant to degradation. In addition,

activities such as landscaping, gardening, or tilling may mix soils at different depths and, hence, reduce the surface concentrations.

A major source of uncertainty in exposure estimates is the lack of information on the vertical distribution of CDDs/CDFs in soil. Available data from Midland represent composites of the topmost inch (25 mm) of the soil column. Most exposure of children is likely to be to soil in the topmost few millimeters. If the soil has not been disturbed or well mixed, CDDs/CDFs may be concentrated in this surface sublayer because atmospheric deposition is the primary route of input. On the other hand, if the soil has been mixed periodically, CDFs may be depleted in the surface sublayer through volatilization, photodegradation, or other loss processes that occur when soil is exposed at the surface (Thibodeaux and Lipsky 1985). Adults may be exposed to soil from the same surface sublayer if their exposure derives primarily from casual contact, but may be exposed to soil from depths greater than 25 mm if their exposure is primarily from gardening.

In the absence of data on trends in atmospheric deposition, persistence of CDDs/CDFs in soil, or vertical distribution of residues, it is assumed in this exposure assessment that the concentrations measured in residential soils in 1983 are representative of those to which people are currently exposed and will remain essentially constant, at least for the 12-year period during which most lifetime exposure takes place (Table III-20). It should be recognized, however, that the estimates of intakes presented in Table III-20 may be too high or too low, depending on the factors discussed above.

Another source of uncertainty in exposure estimates is the limited body of information on soil ingestion rates. The available information is discussed by LaGoy (1987). Although the "upper" and "lower" rates of soil ingestion listed in Table III-19 are reasonable, each could be either too high or too low for conditions in the Midland area. In particular, children with pica may ingest soil at a rate one order of magnitude higher than the maximum listed in Table III-19 (LaGoy 1987); such children could have intakes of CDDs/CDFs an order of magnitude higher than those listed in Table III-20.

A final source of uncertainty in exposure estimates is the estimates used for bioavailability. The values listed in Table III-19 are derived from a study of fly ash containing CDDs/CDFs, and from a study of Missouri soils, which had been contaminated with 2378-TCDD by application of contaminated oil 12 years earlier. Most CDDs/CDFs in Midland soil will presumably have been deposited on airborne particulates and have been retained in soil from varying periods. It is not clear to what extent the CDDs/CDFs would have been desorbed from fly ash particles and resorbed onto soil particles. Bioavailability of CDDs/CDFs from these soils may be higher or lower than indicated by the values used in this exposure assessment.

Overall, as indicated by the discussion in this section, the exposure estimates derived in this section are subject to substantial uncertainty. Although the assumptions and parameters used in this assessment are reasonable, the estimates of intake listed in Table III-20 could be either too high or too low, possibly by a large factor in either direction. This range of uncertainty will be taken into account in the risk assessment in Part IV of this report.

D. Water

Samples of potable groundwater, potable surface water, and brine from the Dow Midland facility's brine operations were obtained by USEPA between August 1984 and September 1985. The results of the sampling and analyses were reported in December 1985 (Barna and Amendola 1985). This section of the exposure assessment presents the results of that analysis.

1. CDD/CDF Concentrations in Water

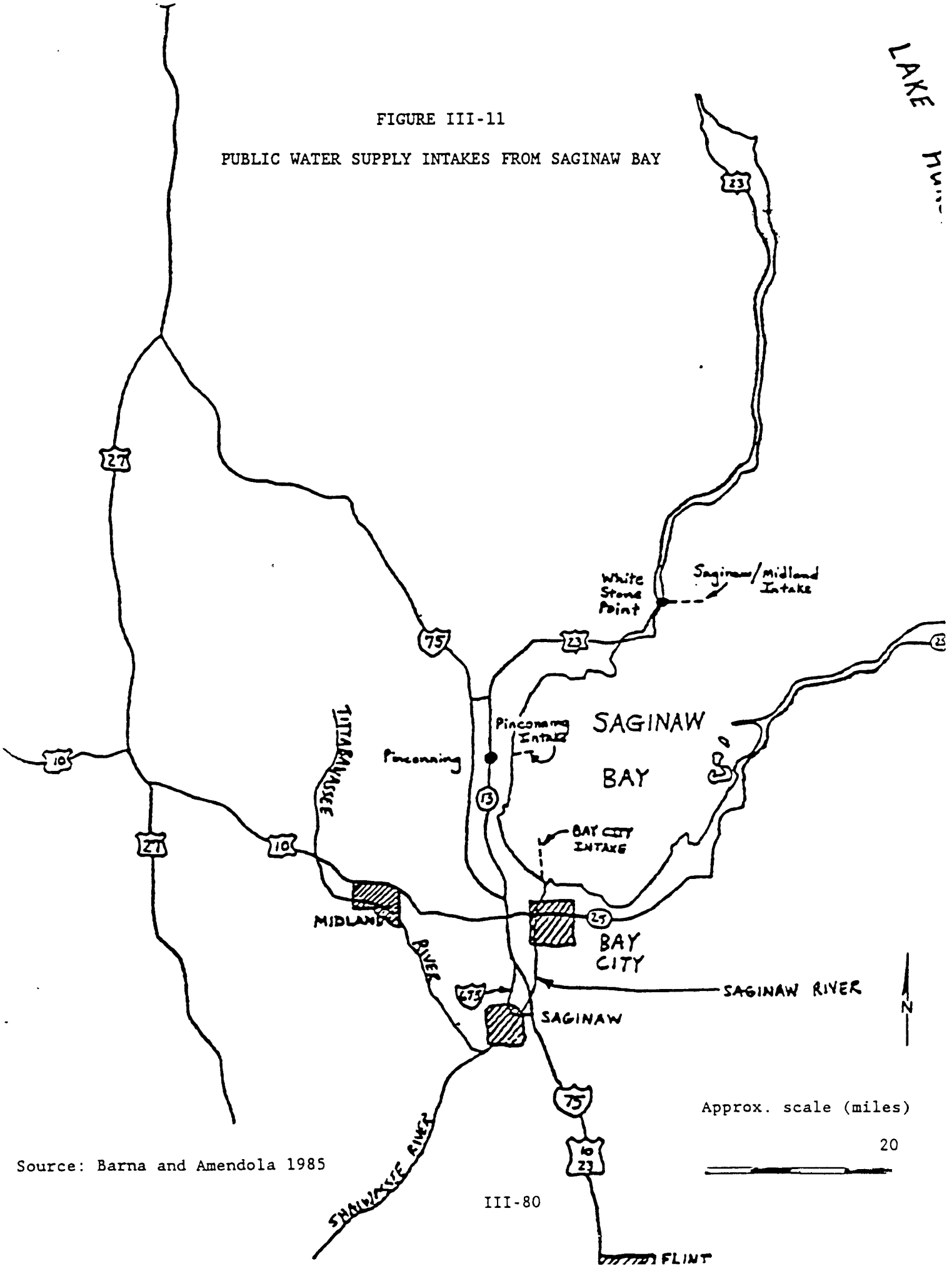
a. Surface Water Supplies.

Three intakes in Saginaw Bay are used as raw water supplies for four Michigan communities. The Saginaw/Midland intake extends about 2 miles into Saginaw Bay east-northeast of Whitestone Point. The Bay City intake extends about 3-1/2 miles into the bay from shore in a northerly direction. The Pinconning intake extends about one mile into the bay in an east-northeast direction. Figure III-11 is a location map for these intakes.

Raw water samples were collected from taps in the pump buildings for the above cities on August 5, 1984, and December 3-5, 1984. A sample from the Saginaw River, identified as a standby water intake for the City of Saginaw, and a sample of finished tap water from the City of Midland were also taken, for a total of five samples.

FIGURE III-11

PUBLIC WATER SUPPLY INTAKES FROM SAGINAW BAY



Source: Barna and Amendola 1985

2378-TCDD was not detected in any of these samples. Detection limits ranged from 2-10 parts per quadrillion (ppq) or picograms/liter (pq/l). These samples were not analyzed for other CDDs and CDFs. The detection of 2378-TCDD in surface water intakes was not expected given the low documented discharge levels of CDDs/CDFs from the Dow Midland facility, the low water solubility and strong tendency of CDDs/CDFs to adsorb to particulate matter, and the considerable dilution afforded by the distance from the point of discharge from the Dow Midland facility to the respective water intakes (Barna and Amendola 1985).

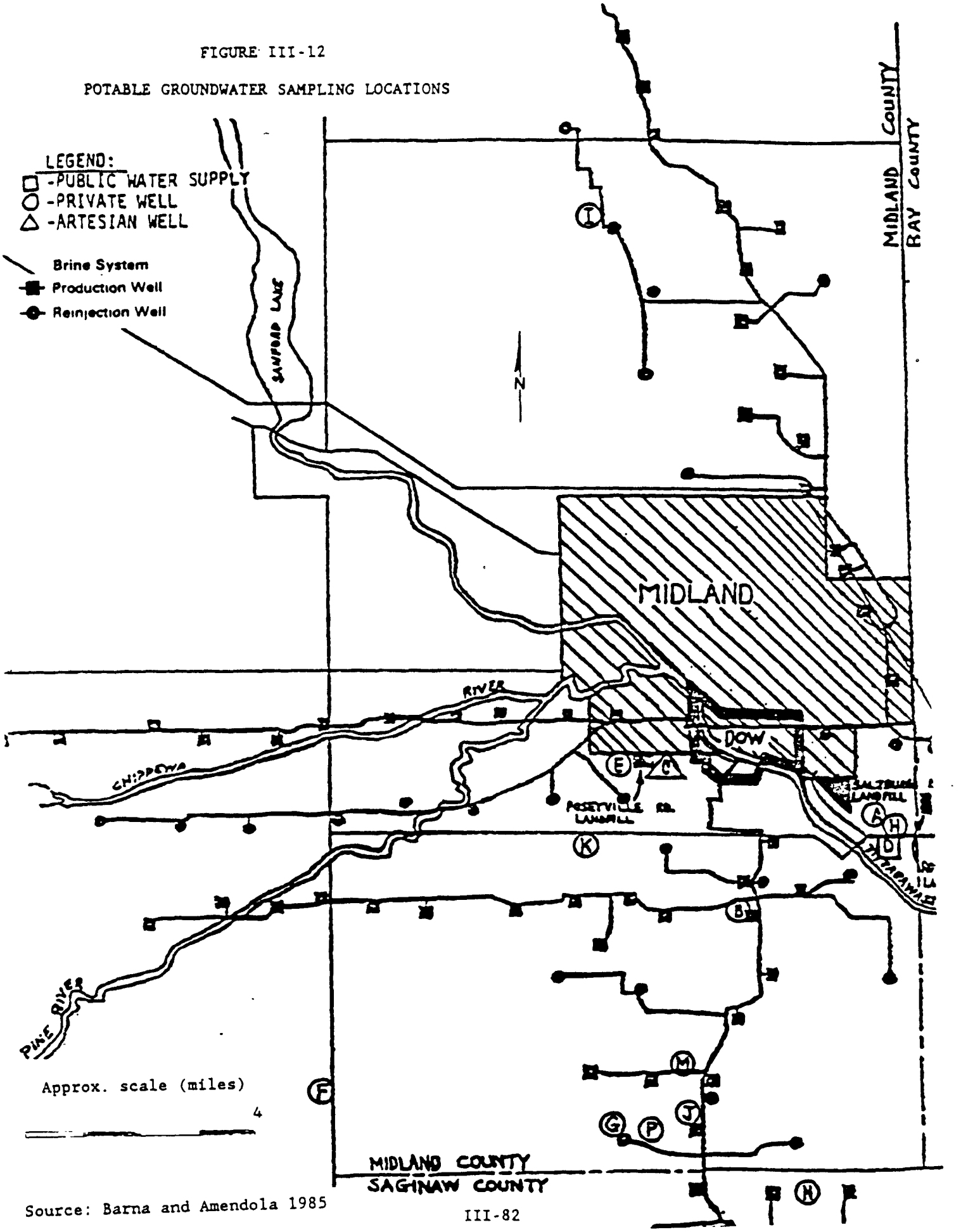
b. Potable Groundwater Supplies.

One public groundwater supply, 14 private groundwater supplies generally located near Dow Midland brine operations and landfills, and one artesian well reportedly used as a source of drinking water were sampled. The locations of the sampled wells are presented in Figure III-12.

Groundwater samples were obtained on December 3-5, 1984. 2378-TCDD was not detected in any of the samples, with limits of detection ranging from 4-50 ppq (Table III-21). Five supplemental samples were obtained on June 12, 1985, and apparent positive findings of 2378-TCDD were found in two samples (Table III-22). However, these findings were not confirmed by subsequent split sample analyses (detection limit 6-10 ppq) or by analyses of additional samples obtained on August 2, 1985, at those two locations (detection limit 1-10 ppq) as shown in Table III-22.

FIGURE III-12

POTABLE GROUNDWATER SAMPLING LOCATIONS



Source: Barna and Amendola 1985

TABLE III-21

Midland Area Ground Water Samples
2378-TCDD -- December 3-5, 1984

(parts per quadrillion)

Well Location	2378-TCDD	(DL)
A	ND	(4)
B	ND	(7)
C	ND	(4)
D	ND	(50)
E	ND	(4)
F	ND	(5)
G	ND	(12)
H	ND	(4)
I	ND	(2)
L	ND	(6)
M	ND	(12)
N	ND	(4)
P	ND	(7)

Notes: (1) Samples analyzed by Midwest Research Institute (MRI).
(2) ND - Not detected.
(3) Detection level - ( ).

Source: Barna and Amendola 1985

TABLE III-22

Midland Area Ground Water Samples
2378-TCDD -- June 12, 1985

(Results in parts per quadrillion (ppq).)

Sample Number	Location	NWQL		Brehm Laboratory		Dow Chemical	
		2378-TCDD	(DL)	2378-TCDD	(DL)	2378-TCDD	(DL)
Samples Collected on June 12, 1985							
DE017601	Mapleton	20	(10)	--	--	ND	(6)
602	Mapleton (Dup)	ND	(10)	ND	(8)	--	--
603	Artesian	ND*	(10)	--	--	ND	(8)
604	Private	ND	(10)	--	--	ND	(7)
605	Private	ND	(10)	ND	(9)	--	--
606	Private	30-40	(10)	ND	(10)	--	--
607	Field Blank	ND	(10)	ND	(9)	--	--
--	NWQL Lab Blank	ND	(10)	--	--	--	--
--	Brehm Laboratory Reagent Blank	--	--	ND	(6)	--	--
--	Dow Chemical Reagent Blank	--	--	--	--	ND	(5)

*ND (10) at resolution 9000; 40 (10) at resolution 5000.
All other samples analyzed at resolution 5000.

Samples Collected on August 2, 1985							
85EG09S02	Mapleton	ND	(7)	--	--	ND	(1)
S01	Artesian	ND	(10)	--	--	ND	(1)
D01	Artesian (Dup)	ND	(6)	--	--	--	--
	Field Blank	ND	(3)	--	--	--	--
	NWQL Lab Blank	ND	(3)	--	--	--	--

Notes: 1. DL - Detection level.
2. ND - Not detected at stated detection level.
3. Screening analyses for PCDDs and PCDFs by NWQL for samples collected on June 12, 1985, showed no detectable PCDDs or PCDFs.

To ensure that 2378-TCDD was not present in groundwater from these wells, USEPA initiated a follow-up survey which involved analyses of six potable water samples. Split samples analyzed by three laboratories show that groundwater at these locations did not contain detectable concentrations of 2378-TCDD (detection limits of 0.2-3.6 ppq), as shown in Table III-23.

One set of analyses for other CDDs/CDFs in potable well water samples was invalidated due to in-lab contamination problems with TCDDs, OCDD, and OCDF. Screening analyses for CDDs/CDFs in subsequent samples collected on June 12, 1985, showed no detectable CDDs or CDFs.

c. Dow Midland Brine Operations.

The Dow Midland facility was founded in 1897 as a producer of brine chemicals. Naturally occurring brine was pumped from the Sylvania aquifer, a sandstone formation about 5,000 feet below the surface. After removal of salts and minerals, the spent brine was sent to Brine Pond No. 6 for holding prior to filtration and pressure injection to the same formation through return wells. The brine operations are being shut down as part of a consent order with the Michigan Department of Natural Resources (Barna and Amendola 1985). The Dow Midland facility brine operations are shown in Figure III-13.

The north, south-southwest, combined raw brine main lines, and production well 29 were sampled for the presence of CDDs/CDFs in raw brine liquid. Brine pond sediments were sampled at three locations in Brine Pond 6: near the inlet

TABLE III-23

Midland Area Ground Water Samples
2378-TCDD -- September 3, 1985

EPA Sample Number	USEPA - NHQL: 2378-TCDD ppq (pg/kg)	USEPA - NHQL: Method Efficiency at 125 pg/kg	Brehm Laboratory Wright State University: 2378-TCDD ppq (pg/kg)	Brehm Laboratory Wright State University: Method Efficiency at 125 pg/kg	Dow Chemical: 2378-TCDD (ppq)	Dow Chemical: ^{13}C 2378-TCDD % Recovery[a]	Dow Chemical: pg 2378-TCDD Observed[b]
Field Samples:							
DE 017901	ND (1.0)	68%	ND (1.4)/ND (1.2)	52%/59%	ND (1)	56%	---
DE 017902	ND (0.2)	65%	ND (1.5)/ND (1.8)	59%/50%	ND (0.9)	87%	---
DE 017903**	ND (0.3)	68%	ND (1.6)/ND (2.0)	64%/45%*	ND (1)	82%	---
DE 017904	ND (1.0)	66%	ND (1.3)/ND (1.1)	73%/51%	ND (0.8)	63%	---
DE 017908	ND (1.3)	17%*	ND (1.6)/ND (1.7)	68%/61%	ND (1)	83%	---
DE 017909	ND (3.6)	39%*	ND/(1.9)	55%	ND (0.8)	85%	---
QA Samples:							
Field Blank (DE 017905)	ND (0.3)	78%	ND (1.2)/ND (2.0)	56%/48%*	ND (0.8)	81%	---
Field Sample Spiked (DE 017906)	ND (11)	27%*	11.6/9.9	55%/59%	10 (0.8)	83%	35 pg
Field Blank Spiked (DE 017907)	9 (1.5)	73%	13.9/10.5	61%/51%	12 (0.8)	67%	40 pg

Notes:

- Blank water - 39%*, ND(0.2)
- Spiked water[1] - 65%,29(1.7)
- Sample DE 017906 - positive signals observed for M/E 320 and 322; did not meet ion-ratio-criteria.

*Method efficiency below acceptable level for national dioxin study.

[1]Spike level 26 pg/kg (ppq).

Notes:

*Method efficiency below acceptable level for national dioxin study.

Notes:

Reagent Blank #	Analyses Set #	2378-TCDD (pg)	^{13}C 2378-TCDD % Recovery[a]
#1	Set #1	ND (3)	68%
#2	Set #2	ND (3)	71%
#3	Set #3	ND (3)	79%

[a] Fortification level = 5.0 ng ^{13}C 2378-TCDD per sample.

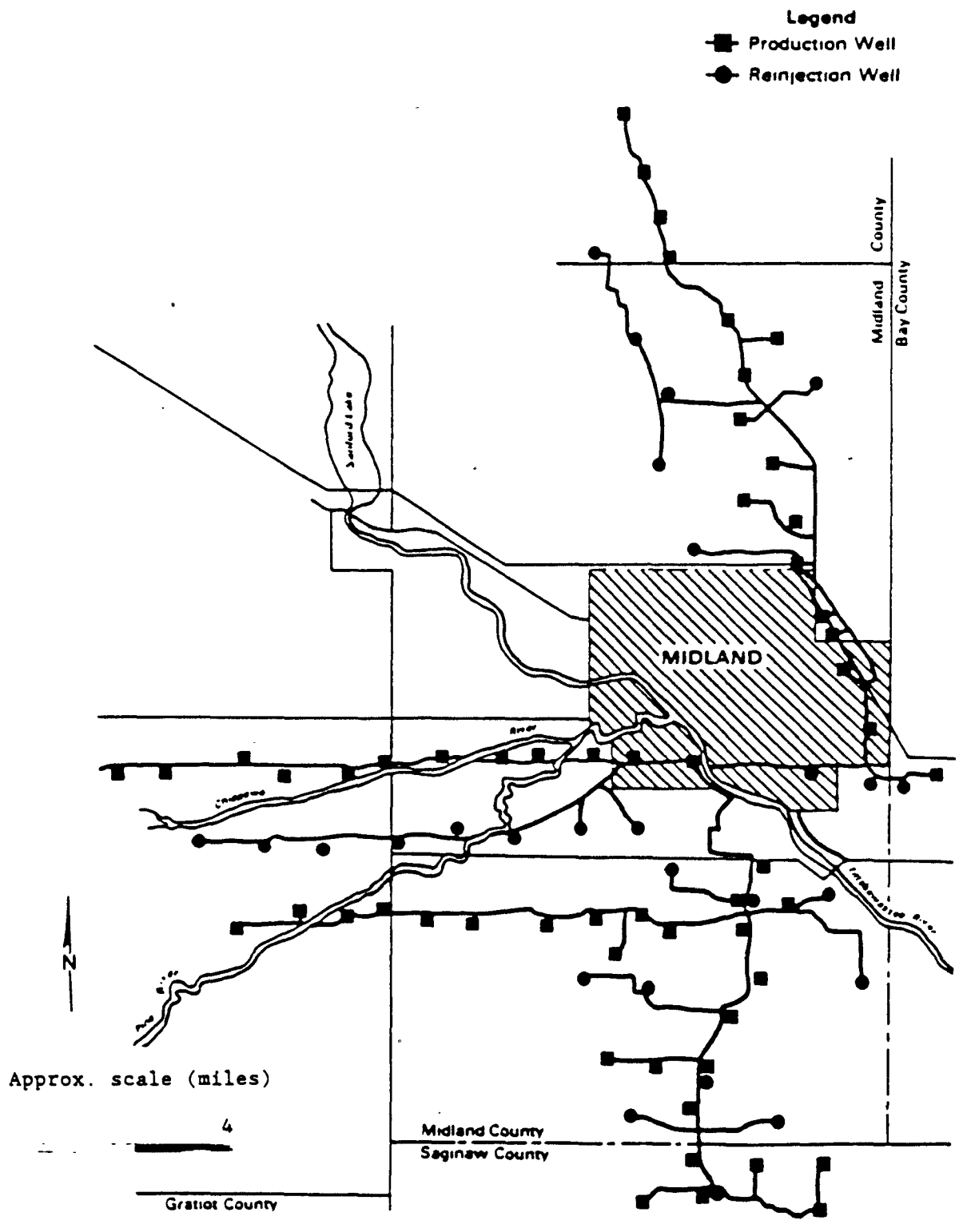
[b] Two samples were spiked with 40 pg native 2378-TCDD.

**Field sample spiked DE 017906 corresponds to field sample DE 017903.

Source: Barna and Amendola 1985

FIGURE III-13

DOW MIDLAND FACILITY BRINE SYSTEM



Source: Barna and Amendola 1985

and outlet and at one intermediate location. Samples were collected on August 13-14, 1984; October 22, 1984, and December 3, 1984.

2378-TCDD was not detected in any of the liquid brine samples. Detection limits ranged from 2-54 ppq. Analyses for other CDD/CDF homologues in the liquid brine samples were invalidated due to the presence of CDDs/CDFs in laboratory method blank samples (Barna and Amendola 1985).

In the brine pond sediments, 2378-TCDD was not detected, with detection limits ranging from 6.9 to 15.7 ppt. However, CDD/CDF homologues were positively detected in the three brine pond sediment samples, as shown in Table III-24. A possible source of the CDDs/CDFs may be deposition from atmospheric emissions from the Dow Midland facility (Barna and Amendola 1985).

2. Populations at Risk

Analyses of public and private water supplies from both surface water and groundwater sources did not detect CDDs or CDFs at detection limits as low as 0.2 ppq. Analyses of the water samples indicate that there is little likelihood of a public health concern associated with ingestion of water from the sampled surface and groundwater sources. As a result, exposure to CDDs/CDFs in potable water is not quantified.

The brine pond sediments contain ppt levels of CDDs and CDFs. There is no reasonable likelihood of direct public exposure to these sediments, and the

TABLE III-24

CDDs/CDFs DETECTED IN BRINE POND SEDIMENTS

CDDs	Range (ppb)	CDFs	Range (ppb)
Total TCDDs	ND-0.016	2378-TCDF	0.03-0.11
Total PeCDDs	ND-0.15	Total TCDFs	0.04-0.21
Total HxCDDs	ND-0.07	Total PeCDFs	ND
Total HpCDDs	0.19-0.21	Total HxCDFs	ND-3.5
OCDD	1.5-3.8	Total HpCDFs	ND-2.8
		OCDF	0.5-5.8

presence of CDDs and CDFs in the brine pond sediments is not likely to pose a public health threat (Barna and Amendola 1985). Exposure to CDDs/CDFs in brine pond sediments is also not quantified.

E. Fish

In 1978, the Dow Chemical Company submitted a report to the USEPA which indicated that detectable levels of 2378-TCDD had been found in Tittabawassee River fish taken downstream, but not upstream, of the plant process outfalls and that these results had been corroborated by fish bioaccumulation studies, indicating that the outfall was a likely source of 2378-TCDD to the river (Dow 1978).

In subsequent years, a number of additional studies of 2378-TCDD contamination of fish from the river have been conducted by the industry and by State and Federal governments. In addition, there have been investigations of a wide range of chemical contamination in river sediment and various process streams within the manufacturing facility. In the summer of 1986, USEPA Region V produced a report which summarizes this information (Amendola and Barna 1986).

The fish studies have used one of two approaches: 1) harvest native fish upstream and downstream of the Dow Midland facility or 2) place caged fish upstream and downstream of the plant for a period of 30 days. In either case, the collected fish were subsequently subjected to extraction of CDDs/CDFs, "clean-up" (to isolate the CDDs/CDFs from other compounds which could potentially interfere with the final analysis), and analysis by gas chromatography/mass spectrometry (GC/MS).

The Tittabawassee River is not a commercial fishery. However, the river is used by sports fishermen, from above Midland to its confluence with the Saginaw River some 30 miles downstream. Stocking of walleye in the Tittabawassee River has resulted in a popular sports fishery, with reports of fishermen catching fish even within the plant's process outfall mixing zone.

1. CDD/CDF residue levels

Table III-25 (Table 38 from Amendola and Barna 1986) summarizes data on concentrations of 2378-TCDD in fish collected between 1978 and 1985. Figure III-14 (Figure 11 from Amendola and Barna 1986) shows the locations referred to in the Table. Note that Dublin Road and Emerson Park are identified in footnotes (4) and (5) to the Table as sites which are upstream from the Dow Midland facility.

Three different types of samples have been analyzed: whole fish, fillets with the skin on, and fillets with the skin off. Concentrations measured in whole fish include contributions from contaminants in the viscera, generally not eaten by humans and possibly containing contaminated ingested material, e.g., sediment particulates. Fish skin is often fattier than the remainder of the fillet and may for this reason contain higher concentrations of contaminants, but is often eaten by humans. The only direct comparison possible within the data tabulated in Table III-25 is for carp in 1983: the average concentration in whole fish was about 4 times higher than that in fillets with skin off, but fell within the observed range for these fillets.

TABLE III-25

Tittabawassee River Native Fish Collections
2378-TCDD
1978-1985

(parts per trillion)

Study	Location/Species	Whole Fish No.	Whole Fish Range	Whole Fish Average	Filet - Skin On No.	Filet - Skin On Range	Filet - Skin On Average	Filet - Skin Off No.	Filet - Skin Off Range	Filet - Skin Off Average
1978 USEPA	Dow Dam to Center Road									
	Carp							6	ND-93	41
	Channel Catfish							3	42-695	337
	Yellow Perch							3[a]	ND-20	10
	Dublin Road									
	Carp							1		ND
1980 MDNR/ USEPA	Dow Dam to Center Road									
	Carp	5	33-142	89.6						
	White Sucker	3	3-10	7.0						
	Emerson Park									
	Carp	3	7-62	40.7						
1983 MDNR/ USEPA	Smiths Crossing Road									
	Carp	1[b]		190				25	12-530	50
	Catfish							1[b]		75
	Smallmouth Bass				1[b]		5.1			
	Walleye				5	2.8-5.1	3.9			

Notes: (1) a - includes two, 2-fish composites
(2) b - five-fish composite
(3) ND - not detected
(4) The Dublin Road sampling site is located upstream of the Dow Chemical - Midland Plant.
(5) The Emerson Park sampling site is located upstream of the Dow Chemical - Midland Plant.

TABLE III-25

Tittabawassee River Native Fish Collections
2378-TCDD
1978-1985

(parts per trillion)

Study	Location/Species	Whole Fish No.	Whole Fish Range	Whole Fish Average	Filet - Skin On No.	Filet - Skin On Range	Filet - Skin On Average	Filet - Skin Off No.	Filet - Skin Off Range	Filet - Skin Off Average
1985 MDNR/ MDPH/FDA/Dow Chemical	Dow Dam to Smith's Crossing Road									
	Walleye-Spring Run				8	2.5- 7.6	4.4			
	Walleye-Summer Resident				6	2.6-14.0	6.5			
	Crappie				3[c]	2.8- 4.5	3.9			
	Northern Pike				3	6.1-15.0	9.5			
	White Bass				4[d]	5.7-15.0	8.2			
	Smallmouth Bass				3	2.8- 6.4	5.0			
1985 Dow Chemical	Smith's Crossing Road									
	Carp							2	3.8-54	28.9
	Catfish							1		39
	Walleye				5	ND-3.6	2.5			
	Dublin Road									
	Carp							3	ND-24	8.6

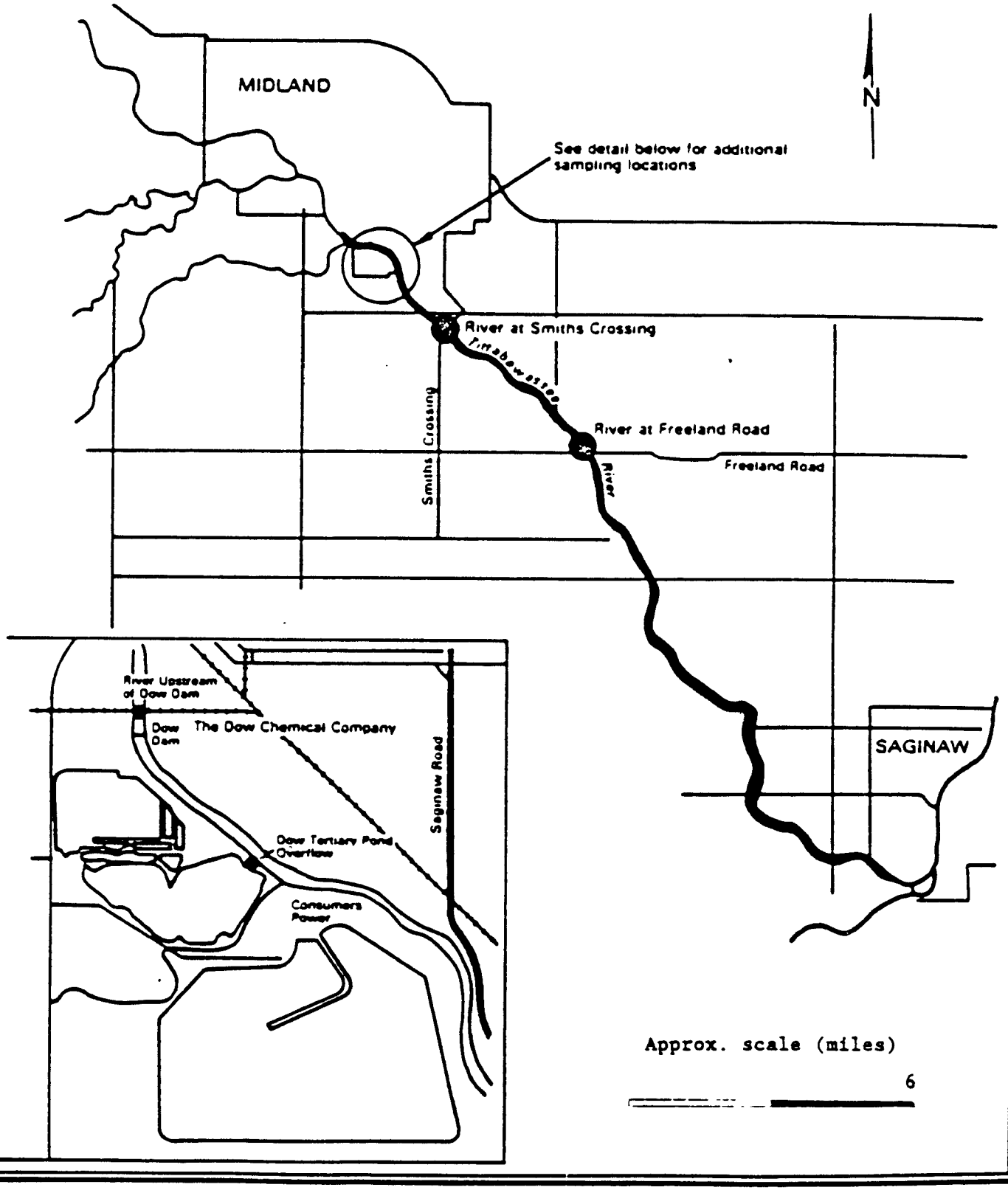
Notes: (1) c - three-fish composite for each measurement
(2) d - includes three 3-fish composite and one 4-fish composite
(3) ND - not detected
(4) The Dublin Road sampling site is located upstream of the Dow Chemical - Midland Plant.

Source: Amendola and Barna 1986

FIGURE III-14

Fish Sampling Locations

Tittabawassee River



Source: Amendola and Barna 1986

Table III-26 (Table 39 from Amendola and Barna 1986) and Figure III-15 (Figure 21 from Amendola and Barna 1986) present the same data by species and year of harvest. Carp and catfish, two fatty bottom-feeders, consistently show the highest concentrations of 2378-TCDD. In fact, the levels of 2378-TCDD in generally non-migratory, relatively fatty, bottom-feeding fish such as carp and catfish caught in an area with contaminated sediment are higher than those in sport fish caught in the same area by roughly an order of magnitude.

In general, Tittabawassee River game species (walleye, smallmouth bass, crappie, northern pike, and yellow perch), when analyzed on a skin-on fillet basis, are contaminated with 2378-TCDD at average levels ranging from ND-15 ppt, with an overall average that is close to 5 ppt (Amendola and Barna 1986).

Two studies have been conducted (by Dow Chemical under a consent agreement with USEPA) to determine the presence of CDDs/CDFs other than 2378-TCDD in fish from the Tittabawassee River. PeCDDs and CDFs other than 2378-TCDF were not analyzed for in these studies. The results from these investigations are found in Table III-27. The bottom feeders had generally higher concentrations of CDDs/CDFs than the game fish. These data indicate the possibility of a downward trend in the fish tissue concentrations of CDDs/CDFs, but a firm conclusion is not possible at this time, given the small number of fish tested and the wide variation among fish observed in previous studies.

Recently, limited research by USEPA has indicated that, in a single walleye specimen analyzed, concentrations of 2378-TCDD and 2378-TCDF in the

TABLE III-26

Tittabawassee River Native Fish Collections
Trends in 2378-TCDD Concentrations

	2378-TCDD (ppt)		
Year	Number	Range	Average
Carp - Whole Fish			
1980	5	33-142	89.6
1983	5 (comp)	--	190
Carp - Skin-off Filet			
1978	6	ND-93	41
1983	25	12-530	50
1985	2	3.8-54	28.9
Catfish - Skin-off Filet			
1978	3	42-695	337
1983	5 (comp)	--	75
1985	1	--	39
Walleye - Skin-on Filet			
1983-summer	5	2.8-5.1	3.9
1985-spring	8	2.5-7.6	4.4
-summer	6	2.6-14.0	6.5
-fall	5	ND-3.6	2.3
Smallmouth Bass - Skin-on Filet			
1983	5 (comp)	--	5.1
1985	3	2.8-6.4	5.0

Source: Amendola and Barna 1986

FIGURE III-15

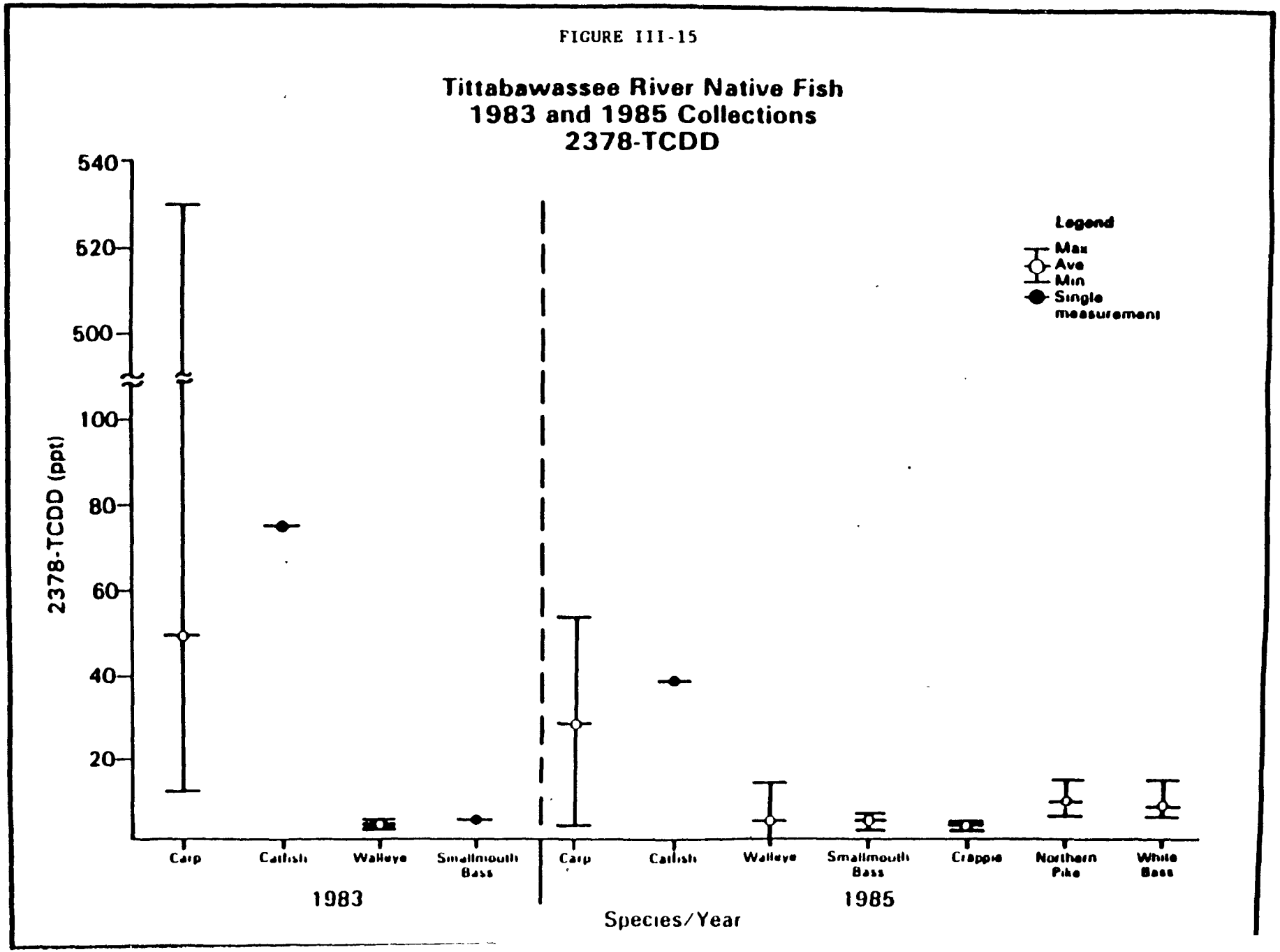


TABLE III-27

PCDDs and PCDFs
NATIVE FISH COLLECTION
TITTABAWASSEE RIVER, 1985 and 1987
(parts per trillion)

Species	Date Taken	Location Taken	2378-TCDD	2378-TCDF	Total TCDDs	Total HxCDDs	Total HpCDDs	OCDDs
Walleye	8/22/85	Smith's Crossing	2.5	34	2 8	36	34	95
Walleye	8/22/85	Smith's Crossing	2.6	24	1.9	19	26	55
Walleye	8/22/85	Smith's Crossing	3.0	28	17	5.6	6.2	16
Walleye	8/22/85	Smith's Crossing	3 6	40	ND(1.4)	ND(2.7)	ND(11)	15
Walleye (a)	8/22/85	Smith's Crossing	ND(1.8)	11	ND(1.5)	ND(2.5)	ND(2.8)	6.4
Composite of Walleye Viscera (b)			22	300	36	ND(5.3)	ND(3.9)	29
Carp (c)	8/22/85	Smith's Crossing	3.8	8.7	7.8	6.8	9.3	15
Carp	8/22/85	Smith's Crossing	54	94	59	ND(9.1)	26	26
Catfish (d)	8/30/85	Smith's Crossing	39	28	92	23	27	43
Carp	10/21/85	Dublin Road	ND(1.7)	ND(4.4)	ND(2.3)	ND(2.0)	ND(3.7)	3.8
Carp	10/21/85	Dublin Road	1.9	3.3	ND(8.8)	ND(3.8)	5.1	8.5
Carp	10/21/85	Dublin Road	24	83	ND(1.6)	15	15	14
Walleye	9/25/87	Dow Dam	1.4	40	ND(0.5)	ND(2)	6	8
Walleye	9/25/87	Dow Dam	1.1	13	ND(0.4)	ND(2)	8	9
Walleye (e)	9/25/87	Dow Dam	1.5	33	ND(0.4)	ND(2)	5	8
Walleye Viscera (f)			16	520	1.7	13	10	10
Carp	9/15/87	Dublin Road	1.6	4.7	ND(0.7)	ND(1)	3	5
Carp	9/15/87	Dublin Road	8.1	17	ND(0.8)	11	10	6
Carp	9/15/87	Dublin Road	23	10	ND(0.7)	35	23	11
Carp (g)	9/25/87	Smith's Crossing	9.0	14	ND(0.7)	5.7	5.9	5
Carp	9/25/87	Smith's Crossing	4.7	21	ND(0.6)	4	8.3	12
Carp	9/25/87	Smith's Crossing	4.0	27	ND(0 6)	5	4	5
Carp Viscera (h)			19	26	0.6	17	14	13

Notes: (1) Walleye -- skin-on fillet.
(2) Carp and catfish -- skin-off fillet.
(3) Total TCDDs do not include 2378-TCDD.
(4) Samples collected and analyzed by Dow Chemical Company pursuant to settlement agreement in Civil Action No. 83-CV7011BC (United States vs. The Dow Chemical Company).
(5) The Dublin Road sampling site is located upstream of the Dow Chemical Midland Plant.
(6) Percent lipid content for selected 1985 samples are as follows (average of 10 replicate 2 g samples):
(a) Walleye filet -- 1.9% ± 0 4%
(b) Walleye viscera composite -- 14.6% ± 0.8%
(c) Carp fillet -- 3.0% ± 0.4%
(d) Catfish fillet -- 9.1% ± 2.7%
(7) Percent lipid content for selected 1987 samples are as follows (average of 3 replicate 2 g samples):
(e) Walleye fillet -- 2.48 ± 0.05
(f) Walleye viscera -- 15.12 ± 0.52
(g) Carp fillet -- 7 54 ± 0.31
(h) Carp viscera -- 13.54 ± 0.86

Source: Amendola and Barna 1986, Dow 1987b

visceral fat were at least 10 times higher than the concentrations found in the fillet (Naumann 1986). Table III-27 also suggests that viscera of walleye contained concentrations of 2378-TCDD and 2378-TCDF about 10 times higher than those in the fillet. However, this difference was not found for HxCDDs, HpCDDs, or OCDD. More limited support derives from the data in Table III-26, which show that the average concentration of 2378-TCDD in whole carp was greater than that in skin-off fillets from the same species. Since CDDs/CDFs tend to concentrate in the fatty tissues, and several fish species have a fatty layer just below the skin, the skin-on fillets would be expected to reflect higher concentrations of CDDs/CDFs than comparable skin-off fillet samples. However, no direct comparisons are available within the data listed in Table III-25. Since humans often eat fish skin, exposure assessments in this section are based on contaminant concentrations measured in both skin-on and skin-off fillets, pooling all available data.

Table III-28 presents calculated TEQs (toxicity equivalents of 2378-TCDD: see Section II) for the CDDs/CDFs measured in each fish listed in Table III-27. This calculation uses the "A-method" (see Section II), in which all the HxCDDs and HpCDDs are assumed to be 2378-substituted and TEFs for 2378-substituted congeners are applied. Use of the "A-method" is reasonable in this case, since 2378-CDDs/CDFs are selectively absorbed and/or retained in fish (Kuehl et al. 1985, 1987). Based upon the limited data in Table III-28, the TEQs are greater than the 2378-TCDD residue concentrations by an average factor of 2.6 in the game fish and 1.3 in the bottom feeders. A significant contribution to the TEQs comes from 2378-TCDF. Note that analyses were not available for PeCDDs or any CDFs other than 2378-TCDF; for this reason, the TEQs are referred to in

TABLE III-28

TITTABAWASSEE RIVER FISH, 1985 AND 1987
2378-TCDD TOXICITY EQUIVALENTS (PARTIAL TEQs)
(parts per trillion)

	Sample	2378-TCDD	Partial TEQ	Ratio Partial TEQ: 2378-TCDD
1. Game Fish				
1985	Walleye	2.5	7.4	3.0
	Walleye	2.6	5.8	2.2
	Walleye	3.0	6.2	2.1
	Walleye	3.6	7.7	2.1
	Walleye	0.9	2.1	2.3
	Mean (n=5)	2.5	5.8	2.3
1987	Walleye	1.4	5.4	3.9
	Walleye	1.1	2.4	2.2
	Walleye	1.5	4.8	3.2
	Mean (n=3)	1.3	4.2	3.1
Both Years	Mean (n=8)			2.6
2. Bottom Feeder				
1985	Carp	3.8	5.0	1.3
	Carp	54	64	1.2
	Catfish	39	44	1.1
	Mean (n=3)	32	38	1.2
1987	Carp	9.0	10.6	1.2
	Carp	4.7	7.0	1.5
	Carp	4.0	6.9	1.7
	Mean (n=3)	5.9	8.2	1.5
Both Years	Mean (n=6)			1.3

NOTES: (1) Data from Amendola & Barna 1986, Table 38, and Dow 1987b. Downstream, fillet data only. Assumes all hexa- and hepta-CDDs are 2378-substituted. ND values are treated as equal to 1/2 the level of detection. Data for penta-CDDs and all CDFs other than 2378-TCDF are not available.

Table III-28 as "partial TEQs." Given the historical inventory of chemicals which were manufactured in the area and contaminated with CDFs (e.g., pentachlorophenol), the Dow incinerator as a source of PeCDDs and CDFs (see Section III.A above), and the persistence of PeCDDs and CDFs in the environment, the presence of residues of 12378-PeCDD and higher-chlorinated 2378-substituted-CDFs in the Tittabawassee fish would not be surprising. Omission of these congeners from the analysis is likely to have led to underestimation of the TEQs, since PeCDDs and PeCDFs contribute substantially to the TEQs for air emissions (see Section III.B).

Table III-29 shows estimates of TEQs based on all the fish fillet samples for 1983 through 1987, including those listed in Table III-25, which were analyzed only for 2378-TCDD. (Data for fish collected in earlier years showed higher concentrations of 2378-TCDD and may not have been representative of the current situation; they are not used in the exposure assessment.) These estimates are derived by assuming that the average ratio TEQ:2378-TCDD in these latter samples would have been the same as that derived for the same class of fish in Table III-28. These average ratios are then multiplied by the mean concentration of 2378-TCDD in the available fillet samples for the same class of fish, to yield the estimates of TEQ listed in the right-hand column. (Note that these estimates of TEQ, like those in Table III-28, are "partial TEQs," and are likely to be underestimates of total TEQ for the reasons discussed in the previous paragraph.) The overall averages -- 13 ppt for game fish and 58 ppt for bottom feeders -- are used in the subsequent exposure assessment. Repetition of the calculation using the detection limits in cases where

TABLE III-29

TITTABAWASSEE RIVER FISH DOWNSTREAM OF DOW CHEMICAL PLANT
1983-1987 DATA[a]
2378-TCDD AND PARTIAL TEQs
(parts per trillion)

Year	Species	N[b]	TCDD (pg/g) Range	TCDD (pg/g) Mean	Partial TEQs[c] (pg/g) Range	Partial TEQs[c] (pg/g) Mean
Bottom-Feeders						
1983	Carp	25	12-530	50	16-690	65
	Catfish	1	--	75	--	98
1985	Carp (Dow)[d]	2	3.8-54	29	5.0-64	35
	Catfish (Dow)[d]	1	--	39	--	43
1987	Carp (Dow)[d]	3	4.0-9.0	5.9	6.9-11	8.2
	Totals	32	3.8-530		5.0-690	
	Weighted Means			45		58
Game Fish						
1983	Smallmouth Bass	1	--	5.1	--	13
	Walleye	5	2.8-5.1	3.9	7.3-13	10
1985	Walleye (Spring)	8	2.5-7.6	4.4	6.5-20	11
	Walleye (Summer)	6	2.6-14	6.5	6.8-36	17
	Crappie	3	2.8-4.5	3.9	7.3-12	10
	Northern Pike	3	6.1-15	9.5	16-39	25
	White Bass	4	5.7-15	8.2	15-39	21
	Smallmouth Bass	3	2.8-6.4	5.0	7.3-17	13
	Walleye (Dow)[d]	5	ND(1.8)-3.6	2.5	ND(4.1)-7.7	5.8
1987	Walleye (Dow)[d]	3	1.1-1.5	1.3	2.4-5.4	4.2
	Totals	41	ND(1.8)-15		ND(4.1)-39	
	Weighted Means			5.0		13

[a]Fillet data only. From Amendola and Barna 1986, Dow 1987b.

[b]Number of samples analyzed (some are composites of several fish).

[c]Includes 2378-TCDD, other TCDDs, HxCDDs, HpCDDs, and 2378-TCDF only. Units are pg/g (parts per trillion). Except as indicated, partial TEQs estimated from 2378-TCDD values by multiplying by 1.3 (Bottom) or 2.6 (Game), the average ratios of mean partial TEQ to mean 2378-TCDD.

[d]Actual data used for calculating partial TEQs (Table III-28).

congeners or homologues were not detected gave virtually identical estimates, and the results are not presented separately here.

Table III-30 shows the results of averaging partial TEQs for Tittabawassee River fish over different years of collection. As discussed earlier, the average partial TEQs for the fish collected in 1987 was smaller than those for earlier years, but the sample sizes (3 individual fish from each class) were so small that it is not possible to conclude reliably that an overall decrease has taken place. Pending better evidence for a decreasing trend, the average concentrations for 1983-87 (58 ppt partial TEQs for bottom-feeding fish, 13 ppt partial TEQs for game fish) will be used for subsequent exposure assessment. The data in Table III-30 show that comparable averages are derived from all other averaging schemes except that limited to the 6 fish analyzed in 1987.

2. Populations at Risk and Exposure Assumptions

Although the Tittabawassee River does not serve as a commercial fishery, it has been a source of recreational fishing over the years. It has been reported that catches resulting from these activities can make a significant contribution to the diet of some people, particularly certain local residents who fish the river regularly (Smith and Thompson 1984).

Given the wide variety of fishermen on the river, it is likely that there is a wide variety of consumption patterns for Tittabawassee fish. USEPA has cited 6.5 g/day as an average level of fish and shellfish consumption in the U.S. (USEPA 1980). In a more recent survey, USDA (1982) found that 14.5% of

TABLE III-30

TITTABAWASSEE RIVER FISH[a]
COMPARISON OF PARTIAL TEQs[b] AVERAGED OVER DIFFERENT YEARS
(parts per trillion)

	Bottom-Feeding Fish			Game Fish		
Years	N[c]	Range	Mean[d]	N[c]	Range	Mean[d]
1978-87	41	5.0-900	86	44	2.4-39	14
1978-85	38	5.0-900	92	41	6.5-39	14
1985 only	3	5.0-64	38	32	6.5-39	14
1987 only	3	6.9-11	8.2	3	2.4-5.4	4.2
1985-87	6	5.0-64	23	35	2.4-39	13
1983-87	32	5.0-690	58	41	2.4-39	13

[a]Fillet data only. From Amendola and Barna 1986, Dow 1987b.

[b]Includes 2378-TCDD, other TCDDS, HxCDDs, HpCDDs, and 2378-TCDF only. Units are pg/g (parts per trillion). Except for 1987, partial TEQs are estimated from 2378-TCDD values by multiplying by 1.3 (Bottom) or 2.6 (Game), the average ratios of mean partial TEQ to mean 2378-TCDD (Table III-28). NDs not included in ranges.

[c]Number of samples analyzed (some are composites of several fish).

[d]ND values treated as equal to 1/2 the detection limit.

37,874 individuals surveyed throughout the U.S. consumed "finfish other than canned, dried, and raw" during a 3-day period, and that the average quantity eaten by these consumers was 54 g/day; this indicates an overall average consumption of 7.8 g/day for the population surveyed. However, these overall averages include a large proportion of individuals who eat no fresh-water fish at all, and thus may not be appropriate for assessing exposure of many local consumers such as Tittabawassee River fishermen and their families.

The Food and Drug Administration (FDA) has estimated an upper 90th percentile ingestion rate of 16 g/day of freshwater fish in the Great Lakes area (USEPA 1984a). The FDA further assumed that the concentrations of 2378-TCDD were higher in bottom-feeding fish and that these fish constitute about 10% of the freshwater fish in the diet. Jacobson et al. (1984) found roughly the same in a sample of more than 8000 pregnant women surveyed about their consumption of Lake Michigan fish. An Ontario study (Cox et al. 1985) determined an 80th percentile ingestion rate of 40 g/day for sports fish. Humphrey et al. (1976) studied active sports fishermen* around Lake Michigan and found their median consumption rate to be about 44 g/day (35 lb/yr), and the 90th percentile consumption rate to be about 100 g/day (81 lb/yr).

In a more recent survey of a larger population of sports fishermen*, Humphrey (1983) reported a median consumption rate of 48 g/day (38.5 lb/yr); he did not report percentiles for this population. These data are the most relevant to fishermen (and their families) in the Midland area who fish the Tittabawassee River. There are indications that sports fishing, and hence the size of the sports fish consuming public, is increasing along the Tittabawassee

*Cohorts comprised of sports fishermen living in the vicinity of Lake Michigan who reported consuming at least 24 lbs of fish per year.

River, although river-specific data are not available. A study is currently underway to obtain more specific data on fishing populations along the Tittabawassee River.

Estimates of the size of single meal servings range from roughly 1/4 to 1/2 lb (113-227 g). USDA (1982) found that the average quantity of finfish consumed per eating occasion was 145 g, with a median of 113 g, a 90th percentile of 255 g, and a 95th percentile of 340 g. Humphrey (1983) reported a median meal size of 319 g (340 g for men and 227 g for women) among Michigan sports fishermen*. Humphrey did not report percentiles in this study.

Four scenarios for long-term fish exposure have been developed based on the data just discussed. The first is for a "general consumer" who eats an average of 7.8 g/day of fish (about 1 quarter-pound meal every two weeks), equivalent to the overall average for the U.S. (USDA 1982), of which half is assumed to be game fish from the Tittabawassee and the other half fish from other sources free of CDD/CDF contamination. In the remaining three scenarios, all of the fish eaten are assumed to come from the Tittabawassee. In the second scenario, a "median sports fisherman" is assumed to eat an average of 48 g/day of game fish (3.0 quarter-pound meals per week), equal to the median fish consumption among Michigan sports fishermen* (Humphrey 1983). In the third scenario, a "high sports fisherman" is assumed to eat an average of 100 g/day of game fish (about 3 half-pound meals per week), equal to the 90th percentile for Michigan sports fishermen* (Humphrey et al. 1976). In the final scenario, the "plausible maximum consumer" also is assumed to eat an average of 100 g/day, but 50 percent is assumed to be game fish and the other 50 percent

*See footnote on previous page.

are assumed to be the more highly-contaminated bottom fish. Table III-31 presents the scenarios for consumption of fish from the Tittabawassee River.

Table III-31 shows the amounts of 2378-TCDD and estimated TEQs ingested under the assumptions listed above for fish tissue concentrations and ingestion rates on a long-term average daily basis. In order to provide an idea of the potential for adverse health effects associated with short-term consumption of Tittabawassee River fish, estimates of single-meal fish (and TEQ) ingestion have also been developed, as shown in Table III-32. These estimates were based on the median and 90th percentile of single-meal fish consumption as reported by USDA (1982), using the mean and maximum TEQ levels found in game and bottom fish, as shown in Table III-29.

The final step in the exposure assessment is to explore how CDD/CDF intake could vary among age groups, especially for children. Table III-33 presents data on the fish consumption of children in three age-groups. Per unit of body mass, children ingest more fish than adults and hence would ingest more CDDs/CDFs. The largest mass-specific exposures would be to children under 5 years old, whose doses (on an average and single-meal basis) would be about 2.2 times higher than that of adults.

3. Other Contaminants

This exposure assessment focuses on 2378-TCDD and on the estimated TEQs. Other carcinogenic contaminants, such as PCBs, have been detected in Tittabawassee River fish, and, while their potencies are several orders of

TABLE III-31

SCENARIOS FOR EXPOSURE TO CDDs/CDFs FROM CONSUMPTION OF TITTABAWASSEE FISH

Exposure Scenario	Fish Consumption		Concentration[a]		Dose Rate[b]	
	Mean (g/day)	Meal Size (g)	2378-TCDD (pg/g)	Partial TEQs[c] (pg/g)	2378-TCDD (pg/kg/day)	Partial TEQs[c] (pg/kg/day)
Plausible Maximum Consumer (50% game + 50% bottom fish; 90th percentile MI sports fisherman)	100[d]	255[e]	25	36	36	51
High Sports Fisherman (100% game fish; 90th percentile MI sports fisherman)	100[d]	255[e]	5.0	13	7.1	19
Median Sports Fisherman (100% game fish; median MI sports fisherman)	48[f]	113[g]	5.0	13	3.4	8.9
General Consumer (50% game + 50% clean fish; USDA average consumer)	7.8[h]	113[g]	2.5	6.5	0.28	0.72

[a]Parts per trillion (ppt), from Table III-29. All fish are assumed to be from the Tittabawassee River, except "clean" fish which are assumed to be free of CDD/CDF contamination.

[b]For a 70 kg human.

[c]Includes 2378-TCDD, other TCDDs, HxCDDs, HpCDDs, and 2378-TCDF only.

[d]90th percentile consumption rate for a cohort of Lake Michigan sports fishermen consuming at least 24 lbs/yr of fish (Humphrey et al. 1976).

[e]90th percentile fish meal size (USDA 1982).

[f]Median for a cohort of Lake Michigan sports fishermen consuming at least 24 lbs/yr of fish (Humphrey 1983).

[g]Median fish meal size (USDA 1982).

[h]Overall average consumption of "finfish other than canned, dried, and raw" by U.S. population (USDA 1982).

TABLE III-32

SINGLE-MEAL (BOLUS) INTAKES OF CDDs/CDFs FROM CONSUMPTION OF TITTABAWASSEE RIVER FISH

	Amount Fish Tissue Consumed (g)	Fish Tissue Concentration (pg/g) [1]				Bolus Dose (pg/kg-d) [2]			
		2378-TCDD		Partial TEQ[3]		2378-TCDD		Partial TEQ[3]	
		Mean	Max.	Mean	Max.	Mean	Max.	Mean	Max.
Median Meal Sizes [4]									
Game Fish	113	5.0	15	13	39	8.1	24	21	63
Bottom Feeder	113	45	530	58	690	73	860	94	1,100
90th Percentile Meal Sizes [4]									
Game Fish	255	5.0	15	13	39	18	55	47	140
Bottom-Feeder	255	45	530	58	690	160	1,900	210	2,500

NOTES:
1. Parts per trillion (ppt); based on all fish collected in 1983-87.
2. For 70 kg human.
3. Includes 2378-TCDD. Does not include penta-CDDs or any CDFs other than 2378-TCDF.
4. From USDA (1982); data of Humphrey (1983) suggest larger meal sizes for sports fishermen and their families.

TABLE III-33

RELATIVE INTAKES OF FISH BY CHILDREN AND ADULTS[a]

	Age Group				
	<1	1-5	6-14	>14[b]	All
Mean body mass (kg)[c]	9	15	35	71	62
Average fish intake:					
g/day	15	28	37	58	54
g/kg-day	1.7	1.8	1.1	0.82	0.88
Ratio to adult	2.0	2.3	1.3	1.0	1.1
Average meal size:					
g	44	76	107	153	145
g/kg	4.9	5.0	3.1	2.2	2.4
Ratio to adult	2.3	2.3	1.4	1.0	1.1
95th percentile meal size:					
g	91[d]	157	240	328	340
g/kg	10	10	6.9	4.6	5.5
Ratio to adult	2.2	2.2	1.5	1.0	1.2

[a]Source: USDA (1982), Table 7.20: "Finfish other than canned, dried, and raw." All averages and percentiles refer to individuals who ate fish at least once during the 3-day period of the survey.

[b]Adult.

[c]From USEPA (1985d).

[d]Maximum reported.

magnitude lower than that of 2378-TCDD, their concentrations in the fish are higher (Amendola and Barna 1986). A summary of the concentrations of these contaminants in Tittabawassee River fish and of the cancer potency factors for those known to be carcinogenic is presented in Appendix C. Possible contributions of these contaminants to the overall risks posed by consumption of Tittabawassee River fish (e.g., through additive toxicity or initiation-promotion interactions with CDDs/CDFs) are discussed in Appendix C.

4. Data Limitations

a. Fish

The number of samples and analytes in each of these studies is small compared to the number desirable for reaching statistically precise conclusions regarding fish contamination levels. The high cost of analysis for CDDs/CDFs, the scarcity of analytical standards, and competing priorities for the same analytical services are contributing causes for the limited data set. It should be noted, however, that the number of analyses of fish taken from the Tittabawassee River is large compared to environmental investigations of CDD/CDF contamination conducted in most other locations. The data available in this case provide a reasonable basis for estimating exposure.

As part of its National Dioxin Study, USEPA collected and analyzed whole fish composite samples (generally bottom-feeders) from approximately 400 sites representing a wide variety of land use patterns across the country (USEPA 1987a). 2378-TCDD was detected in the samples from 28 percent of the sites.

At sites where 2378-TCDD contamination was found in the whole bottom-feeders, game fish also were analyzed. The measured whole fish concentrations at two-thirds of those sites where 2378-TCDD was detected were below 5 ppt, and concentrations for all fillet samples from the study were below the average concentration for carp fillet samples from the Tittabawassee River.

It would be useful to establish time trends in these residue data; i.e., to determine whether the 2378-TCDD contamination in the fish is increasing or decreasing with time. The data in hand and their inherent limitations do not lend themselves to addressing this question easily. On the one hand, given past production, wastewater treatment, and incineration operations at the Dow Midland facility, it is likely that wastewater discharges and atmospheric emissions of CDDs and CDFs were significantly higher in the past than at present. With continuing efforts to control emissions and discharges, future releases can be expected to decrease further. On the other hand, because of the distribution of CDDs/CDFs in Midland area soils and the persistence of these contaminants in the environment, and the continuing finite--albeit reduced--levels of CDD/CDF emissions, Tittabawassee River fish may not exhibit significantly lower levels of CDDs/CDFs in the near future.

On balance, it is likely that the levels of CDDs and CDFs in fish will decrease at some slow, undetermined rate in the future. For conservatism (i.e., to avoid understating risks), however, the risk assessment in Section IV will make the assumption that the CDD/CDF levels in the fish will remain constant at current levels. Should the possible downward trend suggested by

the most recent data be confirmed, the estimated risks can be revised accordingly.

The fish analyses reported to date reflect levels in uncooked fish. From a human health point of view, one is most concerned about the levels of contaminants in food "as eaten"; i.e., after fish have been broiled, baked, fried, or otherwise cooked. There are no data currently available on the effect of cooking procedures on the CDD/CDF levels in fish. Humphrey (1983) found no marked differences between concentrations of PCBs and organochlorine pesticides in raw and cooked fish in a study conducted in Michigan. Studies to investigate the effects of preparation and cooking on CDD/CDF levels in fish are currently under way in Michigan.

Pending results of the current study, the Great Lakes States have agreed that the effects of cooking should not be considered in their derivation of fish consumption advisories, although some of the advisories do inform consumers that certain cooking procedures which allow fats to be drained away may reduce the levels of fat-soluble contaminants (such as CDDs/CDFs) in the portion eaten. For purposes of the risk assessment in Section IV, any effects associated with cooking will not be considered, since (1) the potential reduction from cooking procedures is not known, (2) there is no way to estimate the extent to which any given procedure may actually be followed, and (3) other factors associated with cooking could conceivably increase the level of CDDs/CDFs in the portion eaten.

b. Analyses for CDDs/CDFs.

The major limitation of the existing data on CDDs/CDFs in Tittabawassee River fish is the limited body of data on CDDs/CDFs other than 2378-TCDD, and the total lack of data on PeCDDs and CDFs other than 2378-TCDF. To overcome the first limitation, this exposure assessment makes the assumption that the ratio TEQ:2378-TCDD in all Tittabawassee River fish is similar to those in the fish that have been more completely analyzed (Table III-29). This procedure is reasonable when average exposures are being calculated (Table III-31), but may lead to underestimation of high single-dose exposures (Table III-32). The second limitation is more serious, because the air emissions data (Section II-A) indicate that PeCDDs and PeCDFs may contribute substantially to total TEQs. It should be recognized that the exposure estimates in Tables III-31 and III-32 represent partial TEQs only, and may underestimate total exposure to 2378-TCDD toxicity equivalents.

c. Populations at Risk

While it is generally acknowledged that sport fishing can contribute significantly to the diet of some people, estimates of the size of that contribution and the size of the population vary, as noted above. Also referred to above is a local subpopulation, of undetermined size, in the Midland area, some members of which may regularly supplement their diet through extensive fishing on the Tittabawassee. As a plausible maximum assumption, the exposure assessment assumes that these local fishermen and their families

consume at higher rates than most sports fishermen and eat a significant percentage (50%) of bottom-feeders in addition to game fish.

In the absence of data specific to the Tittabawassee River, this risk assessment employs data obtained from other studies, most of which relate to the Great Lakes area. It is likely, but by no means certain, that these data are reasonable approximations of the consumption patterns along the Tittabawassee.

F. Other routes of exposure

This section briefly considers other potential routes of exposure to CDDs/CDFs derived from the Dow Midland facility. These routes of exposure are considered because they have been identified as possibly significant in studies conducted elsewhere. Lack of data precludes development of quantitative estimates of exposure via these routes in the Midland area. The purpose of this section is to discuss each of these routes in a qualitative manner and to identify whether any is sufficiently likely to be important to justify further investigation.

1. Exposure to Indoor Dust

Thibodeaux and Lipsky (1985) have identified indoor dust as a potentially significant route of exposure to 2378-TCDD carried on airborne particulates. In circumstances where CDDs/CDFs are emitted in association with airborne particulates (e.g., from incinerators such as that at the Dow Midland facility), mass concentrations of CDDs/CDFs on these particulates may be relatively high. Thus, although only a fraction of the airborne particulates is expected to penetrate into houses and to be deposited onto surfaces, the resulting mass concentrations of CDDs/CDFs in house dust may be relatively high. In specific cases modeled by Thibodeaux and Lipsky (1985), based on empirical data for airborne lead, concentrations of 2378-TCDD in indoor dust can be much higher than those in outdoor soil, because of the much greater mass of soil into which deposited airborne particulates are mixed outdoors. Once CDDs/CDFs are deposited in indoor dust, there is a potential for human exposure

via inhalation and inadvertent ingestion (primarily by small children who crawl on the floor). It is not possible to model such exposure for the Midland area, because no data are available on the fraction of CDDs/CDFs attached to particulates, on the size distribution or rate of deposition of these particulates, or on the rates of ingestion and inhalation of indoor particulates. Hawley (1985) proposed the same rates of ingestion for indoor dust as for outdoor soil, but this seems implausible because the opportunity for bulk ingestion is much greater outdoors. Even so, the calculations presented by Thibodeaux and Lipsky (1985) show that ingestion of CDDs/CDFs by children in indoor dust can, under some assumptions, substantially exceed that in outdoor soil. Direct measurements of CDD/CDF concentrations in indoor dust in Midland residences are needed to determine whether such exposures may be significant.

2. Ingestion of Vegetables Grown in Contaminated Soils

Another potential route of exposure to CDDs/CDFs in the Midland area is via ingestion of vegetables grown in domestic gardens. Limited data suggest that highly lipophilic chemicals such as CDDs/CDFs are not translocated significantly from contaminated soils into the edible parts of plants (Briggs et al. 1982). However, there is a potential for human exposure to CDDs/CDFs adsorbed to airborne particulates that are deposited on external plant surfaces, particularly if the CDDs/CDFs are absorbed into the waxy coatings found on the surfaces of many plants (Hattemer-Frey and Travis 1987). Uptake of CDDs/CDFs has also been observed into the edible portions of some root crops (Cocucci et al. 1979). Insufficient data are available to model human exposure

via this route in the Midland area, but a study of CDD/CDF concentrations in vegetables is currently underway.

3. Ingestion of Meat and Dairy Products

Cattle and other domestic animals can ingest chemicals such as CDDs/CDFs through ingestion of grass contaminated with airborne particulates, or through inadvertent ingestion of soil while grazing (Kimbrough et al. 1984). Because CDDs/CDFs are lipophilic and strongly retained in the body, they concentrate in fatty tissues and are excreted in milk. Several studies have identified meat and milk as potential routes of exposure of humans to CDDs/CDFs (Kimbrough et al. 1984, Rappe et al. 1985, USEPA 1984b, Hattemer-Frey and Travis 1987). In the Midland area, the potential for significant exposure is limited because there are few, if any, beef or dairy farms close to the Dow Midland facility. However, evaluation of potential exposure would require information on the fraction of CDDs/CDFs attached to airborne particulates, particle size distributions or measurements of dustfall rates, and parameters required to model uptake by cattle, retention in fat and excretion in milk. None of this information is presently available.

4. Exposure of Infants via Breast Milk

Several studies have identified relatively high levels of CDDs/CDFs in human breast milk (Rappe et al. 1986, Tarkowski and Yrjanheikki 1986). Breast-feeding is a potentially significant route of exposure of infants to CDDs/CDFs (and other lipophilic compounds) because these compounds are retained in the

fatty tissues of the mother, are readily excreted in the lipid-rich milk, and are ingested by infants who depend on the milk for most or all of their nutrition. Exposure of breast-fed infants may be estimated using a simple pharmacokinetic model presented by Smith (1987). Smith's model suggests that exposure of infants of nursing mothers who have substantial long-term exposure themselves, may be an order of magnitude higher. It should be noted, however, that this model for exposure is a highly simplified representation of complex physiological processes and its predictions, therefore, are accompanied by very large uncertainties (as will be discussed in Chapter IV).

PART IV

RISK CHARACTERIZATION

A. Introduction

In this section, information from the Hazard Identification, Dose-Response Assessment and Exposure Assessment sections are combined in order to generate qualitative and quantitative estimates of the risks associated with exposure to the chemicals in question. In the case of the CDDs/CDFs around Midland, the Agency focuses on the risks of cancer and reproductive/teratogenic effects, with limited consideration of other toxic effects (see Part II). A discussion of the qualitative features of the case and of the uncertainties associated with quantitative risk estimates is also included as an integral part of the overall assessment.

B. Summary of Hazard Identification and Dose-Response Assessment for CDDs/CDFs

Hazard identification and dose-response assessment for CDDs/CDFs are presented in Part II, based in part on findings of peer-reviewed USEPA assessments. Despite some data limitations, the non-human in vivo and in vitro toxicity data on CDDs/CDFs and particularly 2378-TCDD clearly indicate that these substances are highly toxic materials. Comparative toxicity studies and in vitro structure-activity studies have provided a framework for understanding the relative potencies of the CDDs and CDFs and for identifying the more potent congeners. Despite extensive studies of human populations exposed to phenoxy

herbicides and other mixtures putatively containing 2378-TCDD, data on effects of 2378-TCDD itself in humans remain conflicting and inconclusive. However, experience derived from the Yusho and Yucheng incidents provides strong evidence that humans are sensitive to at least some of the toxic effects of CDFs. This experience, with the other structure-activity data which are embodied in the principles of the TEF approach, provides a reasonable basis for the use of animal toxicology data on CDDs/CDFs, including 2378-TCDD, to predict human risks resulting from exposure to CDD/CDF mixtures.

1. Cancer Risk Assessment

USEPA (1985b) has evaluated the data from long-term animal studies with 2378-TCDD and a mixture of 2378-substituted HxCDDs and has concluded that these materials are carcinogenic in rats and mice. On this basis, USEPA (1985b, 1986d) has concluded that it is prudent to treat these substances (and, via the TEF approach, the rest of the 2378-substituted CDDs/CDFs) as if they could cause cancer in humans as well; hence, the weight-of-evidence designation of "B2". See Section II.A above for a more complete discussion.

USEPA has conducted, following its Cancer Risk Assessment Guidelines (USEPA 1986a), a quantitative dose-response assessment for the carcinogenic effects of 2378-TCDD and the mixture of 2378-substituted HxCDDs. Using "conservative" assumptions about the shape of the dose-response curve in the low dose region (e.g., linear, non-threshold behavior) and a "conservative" assumption for extrapolating from animal data to predict human risk (body-surface-area scaling), USEPA (1985b) has generated upper-bound potency factors

(i.e., upper bounds on the estimates of excess lifetime risk of contracting cancer per unit dose) of 1.6×10^5 $(mg/kg\text{-}d)^{-1}$ [B2] and 6.3×10^3 $(mg/kg\text{-}d)^{-1}$ [B2] for 2378-TCDD and the 2378-HxCDDs, respectively. According to the TEF approach, the former cancer potency factor should be applied to the lifetime average intake of 2378-TCDD equivalents (i.e., TEQs), expressed in mg/kg-d, to provide an estimate of lifetime cancer risk for exposed individuals. Substantial uncertainties result from the application of the linearized multi-stage (LMS) model to data on 2378-TCDD and from use of the TEF approach, and a review of the potency factor is currently under way by USEPA. These uncertainties are discussed at length in Part II.

2. Non-Cancer Risk Assessment

In Part II of this report, Hazard Identification and Dose-Response Assessments were also presented for the other toxic effects caused by low-dose exposure to CDDs/CDFs, especially 2378-TCDD and 2378-TCDF. Among the effects most thoroughly studied to date are the reproductive and teratogenic effects (e.g., Courtney and Moore 1971, Allen et al. 1979, Murray et al. 1979, Weber et al. 1985, Birnbaum et al. 1985, 1987a,b; see reviews by Nisbet and Paxton 1982 and USEPA 1985b). Data from the Yusho and Yucheng incidents provide limited evidence that CDFs can cause reproductive impairment in humans (Kusuda 1971, Yamashita and Hayashi 1985, Hsu et al. 1985), although there is little evidence for teratogenic effects other than skin hyperpigmentation (Hsu et al. 1985). This provides support for the inference that other CDDs/CDFs, including 2378-TCDD, are likely to cause reproductive and/or teratogenic effects in humans. Accordingly, USEPA has adopted the standard toxicological procedure of using

animal dose-response data on 2378-TCDD to evaluate risks posed to humans exposed to that compound (and, via the TEF approach, to the other CDDs/CDFs as well).

In general, reproductive and teratogenic effects are thought to follow "threshold"-type dose-response relationships, in contrast to carcinogenesis, which is assumed by USEPA to follow a "non-threshold"-type dose-response relationship. That is, there is assumed to be a dose of the reproductive and teratogenic toxicant which is so low (a "threshold" dose) that there is no risk of the adverse effects appearing in an exposed individual. "Threshold" doses are likely to vary among individuals; the "threshold" dose for a given population is the lowest "threshold" dose for an individual in that population. Operationally, "threshold" doses cannot be measured exactly within limited groups of experimental animals; they are estimated from "No-Observed-Adverse-Effect-Levels" (NOAELs).

NOAELs determined in experimental studies with animals are used to generate Reference Doses (RfDs) or Health Advisories (HAs) by applying appropriate Uncertainty Factors (UFs). UFs are intended to take account of differences in sensitivity between animals and humans, variability in susceptibility among members of the human population, and other factors. As explained in Part II, RfDs are estimated daily exposures for the human population (including sensitive subpopulations) that are likely to be without appreciable risk of deleterious effect during a lifetime. HAs are corresponding estimates of exposures that can occur daily for shorter periods without the expectation that adverse health effects will occur.

Part II summarized the derivation of an RfD and HAs for the reproductive/teratogenic effects of 2378-TCDD (and, via the TEF approach, the rest of the CDDs/CDFs). Part II also discussed dose-response data for other toxic effects of 2378-TCDD and showed that an RfD and HAs derived on the basis of liver toxicity were similar to those derived on the basis of reproductive/teratogenic effects. The RfD derived for 2378-TCDD is 1 pg/kg-d, while HAs are in the range 280-300 pg/kg (single-dose) and 28 pg/kg-d (10-day exposure). While an argument can be made that the RfD does not apply to exposures lasting less than a lifetime, USEPA has chosen, for the purposes of this risk assessment, to use the following conservative guidelines for evaluating the potential for adverse health effects other than cancer (making it unlikely that adverse health effects will actually occur in populations exposed at the reference levels for the specified periods):

Period of Exposure	Reference Level for Comparison
Single dose/Single day	280 pg/kg/day (HA for single dose)
Few days to few weeks	28 pg/kg/day (HA for 10-day exposure)
Several months or longer	1 pg/kg/day (RfD for lifetime)

To evaluate the likelihood that adverse effects may occur, estimates of daily intake are compared with the RfD or HA, depending on the anticipated duration of exposure. The ratio between the estimated daily intake and the RfD (for chronic exposure) or the HA (for one-day or subchronic exposure) is referred to as the "Hazard Index" (USEPA 1986e). These Hazard Indices are calculated and presented in tables in the following sections. Hazard Indices greater than 1

indicate that the estimated dose rate exceeds the RfD or HA for the route of exposure and population in question. Hazard Indices for populations receiving exposure by more than one route can be calculated by summing the calculated intake values for each route of exposure.

C. Risks Associated with Exposure to CDD/CDF-Contaminated Air.

Estimates of intakes of CDDs/CDFs by inhalation of ambient air for the two exposure scenarios are summarized in Table III-12. As discussed at the end of Section III.B.6, these estimates incorporate a correction for bioavailability and thus represent absorbed doses of CDDs/CDFs. The cancer potency factor, RfD, and HAs, however, are derived from studies in which rats were exposed to 2378-TCDD in the diet, and are expressed in terms of administered dose. Studies by Fries and Marrow (1975) suggest that only 50-70% (mean, about 55%) of 2378-TCDD administered to rats in the diet is absorbed into the body. Accordingly, when estimates of absorbed dose are compared with the cancer potency factor, RfD, or HAs, it is necessary to multiply the absorbed doses by 1.8 (1/0.55) to yield estimates of equivalent administered dose. This factor (referred to as a "correction for oral bioavailability") is applied to all the estimates in Table III-12.

These adjusted estimates are now multiplied by the cancer potency factor to yield upper-bound estimates of lifetime cancer risk, and divided by the RfD to yield estimates of the long-term Hazard Index. These comparisons are made for both the exposure scenarios considered in Section III.B.6, and for both the

"A-Method" and the "B-Method" of calculating TEQs. The results of these calculations are tabulated in Table IV-1. Estimated upper-bound cancer risks are in the range $2x10^{-5}$ to $6x10^{-5}$ for the "fenceline case" and in the range $5x10^{-6}$ to $1x10^{-5}$ for adults for the "residential area case." Cancer risks for infants and children are not calculated because lifetime intakes for children exposed at these levels are expected to approximate those of the adults when averaged over a complete 70-year lifespan. Hazard indices for the non-cancer effects are all less than or equal to 1 while shorter exposures (a few days to a few weeks) would yield hazard indices no higher than 0.04.

In interpreting these estimates, the following should be borne in mind:

(1) The exposure estimates are subject to a number of limitations that have been discussed in Section III.B.8. Specifically, the estimates are based on measurements of ambient concentrations on only 3 days; the residential area exposure scenario is based on data from only one sampling station, which may have been outside the main contaminant plume on one day; estimates of TEQs were based in part (21-50%) on "non-detects" at the fenceline station and largely (72-77%) on "non-detects" at the residential station; the intake calculation assumes 24-hour daily exposure to outdoor concentrations and 70-year residence at the exposure points.

(2) The cancer risk estimates are "upper bound" estimates of lifetime risk. That is, the actual risk is not likely to be greater than these levels; the actual risks could be significantly lower. Because of uncertainties about the mechanisms of action of 2378-TCDD and the implications of these mechanisms for dose-response modeling, these estimates are subject to additional uncertainties, as discussed above.

(3) The hazard indices calculated for children are based on an RfD originally defined for reproductive effects, which are expressed only during adulthood. This approach to defining the non-cancer risk levels for children was adopted because, as discussed in Section II, RfDs or other critical toxicity values which could be derived for other non-cancer endpoints (e.g., liver and immunotoxicity) are also

TABLE IV-1

RISK CHARACTERIZATION FOR INHALATION OF CDDs/CDFs
IN AMBIENT AIR IN MIDLAND

Exposure Scenario[a]	Upper-Bound Cancer Risk[b]		Hazard Index[c] (Long-Term)	
	A-Method[d]	B-Method[d]	A-Method	B-Method[d]
1. Fenceline Case:				
Infants 0-1 year	--	--	0.4	0.1
Children:				
1-6 years	--	--	1	0.4
6-12 years	--	--	0.7	0.3
Adults (12-70)	--	--	0.3	0.1
Lifetime	6×10^{-5} ["B2"]	2×10^{-5} ["B2"]	--	--
2. Residential Area				
Infants 0-1 year	--	--	0.05	0.02
Children:				
1-6 years	--	--	0.2	0.08
6-12 years	--	--	0.1	0.06
Adults (12-70)	--	--	0.05	0.02
Lifetime	1×10^{-5} ["B2"]	5×10^{-6} ["B2"]	--	--

[a]From Section II.B.6. All exposure estimates assume 24 hr/day exposure to outdoor concentrations, long-term residence (lifetime for cancer risks).

[b]Upper-bound estimate of lifetime cancer risk, obtained by multiplying exposure estimate in Table III-12 by cancer potency factor of 1.6×10^{-4} $(pg/kg\text{-}day)^{-1}$ and multiplying by correction for oral bioavailability of 1.8 (see Section IV.C).

[c]Ratio of exposure estimate in Table III-12 to RfD of 1 pg/kg/day, multiplied by correction for oral bioavailability of 1.8, for exposures lasting several months or more. Shorter exposures (a few days to a few weeks) would yield indices about 28-times lower.

[d]A-Method assumes all Pe-, Hx- and Hp-CDDs and CDFs are 2378-substituted. B-Method assumes all congeners within these groups are equally prevalent (see Part II).

on the order of 1 pg/kg-d. In addition, if the reproductive effects of CDD/CDF exposure are the result of damage to germ cell lines (precursors to sperm and ova), such damage could arise from exposure during infancy and childhood, as well as during adulthood.

(4) 2378-TCDD and HxCDDs were detected only in trace quantities in ambient air. Most of the risk calculated by means of the "A-Method"and much of the risk calculated by means of the "B-Method" is derived by applying TEFs to measurements or estimates of other congeners. For this reason, the cancer risks estimated and tabulated in Table IV-1 are dominated by risks ascribed, through use of the TEF procedure, to exposure to compounds whose potential carcinogenicity has never been investigated directly. Although the TEF procedure is reasonable and has been widely accepted in the scientific community, the indirect basis for inferring these cancer risks should be recognized as contributing additional uncertainty to the risk estimates. To emphasize this uncertainty, the weight-of-evidence designation of B2 is placed in quotation marks.

(5) There is a question about the collection efficiency of the sampling procedures used in these studies which could mean that the measured amounts of CDDs/CDFs were underestimated. USEPA is currently studying this question.

For the above reasons, the values tabulated in Table IV-1 are subject to substantial uncertainties. They are best regarded as order-of magnitude estimates of risk, and will be so treated in the integrated characterization at the end of this Part.

D. Risks Associated with Exposure to CDD/CDF Contaminated Soil.

Estimates of intakes of CDDs/CDFs by ingestion of soil are presented in Table III-20. These estimates are of absorbed dose and require adjustment for oral bioavailability as discussed in the previous section. For this reason, all these estimates are adjusted for oral bioavailability by multiplying by 1.8 before comparison with the cancer potency factor or the RfD.

The adjusted estimates of lifetime average intake are now multiplied by the cancer potency factor to yield upper-bound estimates of lifetime cancer risk. The adjusted estimates of intake for the various age groups being considered are divided by the RfD to yield estimates of long-term Hazard Index. These calculations are performed for both the exposure scenarios detailed in Table III-20. The results of these calculations are presented in Table IV-2. The estimates of upper-bound cancer risk are about $5x10^{-7}$ ["B2"] for the "lower estimate" case, and $1x10^{-5}$ ["B2"] for the "upper estimate" case. Hazard Indices are below 1 in both exposure scenarios for all age groups, but could approach 1 in the "upper estimate" case when exposure of small children is considered and could exceed 1 for children with pica.

In interpreting these estimates, the following should be borne in mind:

(1) The exposure estimates are subject to a number of limitations that have been discussed in Section III.C.3. The most important of these limitations is probably the lack of information on the vertical distribution of the CDDs/CDFs in soil: the concentrations in soil that is actually ingested could be either higher or lower than the measured average in the top 25 mm of the soil column. Estimates of soil ingestion rates are based on limited data, as discussed by LaGoy (1987). The estimates of bioavailability are derived from studies of soil and fly ash which yielded widely varying estimates of bioavailability, and it is not clear which of these studies is most predictive of the bioavailability of CDDs/CDFs from Midland soil.

(2) The cancer risk estimates are "upper bound" estimates of lifetime risk. That is, the actual risk is not likely to be greater than these levels; the actual risks could be significantly lower. Because of uncertainties about the mechanisms of action of 2378-TCDD and the implications of these mechanisms for dose-response modeling, these estimates are subject to additional uncertainties, as discussed above.

(3) As was the case for the air risk estimates, non-cancer Hazard Indices are developed using RfDs originally derived for reproductive effects. These values are relevant to risk assessments for infants and children for the reasons discussed in Section IV.C.

TABLE IV-2

RISK CHARACTERIZATION FOR INGESTION OF CDDs/CDFs
IN SOIL IN MIDLAND

Exposure Scenario[a]	Upper-Bound Lifetime Cancer Risk[b]	Hazard Index[c] (Long-Term)
Lifetime Average Exposure:		
1. Lower Estimate:		
Infants 0-1 year	--	0.02
Children:		
1-6 years	--	0.03
6-12 years	--	0.009
Adults (12-70)	--	0.0003
Lifetime average	5×10^{-7} ["B2"]	--
2. Upper Estimate:		
Infants 0-1 year	--	0.5
Children:		
1-6 years	--	0.6
6-12 years	--	0.2
Adults (12-70)	--	0.01
Lifetime average	1×10^{-5} ["B2"]	--

[a]Assumptions and parameters are listed in Table III-19. Note that the upper estimate does not include individuals with pica. Individuals with this disorder could incur risks 10-fold higher.

[b]Upper-bound estimate of lifetime cancer risk, obtained by multiplying lifetime average TEQ dose rate from Table III-20 by cancer potency factor of 1.6×10^{-4} $(pg/kg\text{-}day)^{-1}$ and multiplying by adjustment for oral bioavailability of 1.8 (see Section IV.C).

[c]Ratio of adult TEQ dose rate from Table III-20 to RfD of 1 pg/kg-day, multiplied by adjustment for oral bioavailability of 1.8 for exposures lasting several months or more. Shorter exposures (a few days to a few weeks) would yield indices about 28-times lower.

(4) See note (4) in Section C above.

An alternative approach to assessment of risks posed by 2378-TCDD in residential soils has been developed by the Centers for Disease Control (Kimbrough et al. 1984). This approach followed the same general methodology as that used in this report for estimating cancer risks, except that the CDC scientists used higher values for soil intake rates, lower values for the cancer potency factor, and a different method of averaging lifetime dose rates. These differences tended to offset each other, so that the overall result of the CDC analysis is comparable to that presented in this report. Specifically, the CDC report identified a concentration of 1 ppb 2378-TCDD in residential soil as the level at which to begin consideration of action to limit human exposure. According to the methodology developed in this report, a concentration of 1 ppb TEQ would yield an upper-bound lifetime cancer risk of about $1x10^{-5}$ in the lower estimate scenario and about $3x10^{-4}$ in the upper estimate scenario. The former estimate is consistent with the range estimated by the CDC method (upper-bound about 10^{-5}) for similar exposures.

In June, 1985, the Agency released its report on the levels of 2378-TCDD in the soils in the Midland area (USEPA, 1985a). Based upon these data, the Centers for Disease Control (CDC) concluded that the monitored levels did not pose an unacceptable public health risk. The Agency's Chlorinated Dioxins Work Group concurred in this assessment. The results of the present assessment are generally consistent with these conclusions, with the upper estimate scenario yielding an upper-bound cancer risk of about 10^{-5}.

E. Risks Associated with Exposure to Water and Brine Sediments

The information summarized in Section III.D provides no plausible evidence of human exposure to CDDs/CDFs either in drinking water or through contact with the brine pond sediments.

F. Risks Associated with Consumption of Fish

Estimates of adult intakes of CDDs/CDFs by ingestion of contaminated fish are summarized in Tables III-31 and III-32. These are estimates of quantities ingested and hence do not need adjustment for oral bioavailability. The estimates of average rates of ingestion are multiplied by the cancer potency factor to yield upper-bound estimates of lifetime cancer risks, and are divided by the RfD to yield estimates of the Hazard Indices for long-term exposure. In addition, the estimates of single-meal (bolus) intakes are compared with the single-dose or 1-day HA to yield estimates of the Hazard Indices for single exposures. All these calculations are performed for four sets of assumptions about rates of consumption of fish, as specified in Tables III-31 and III-32. The results of these calculations are presented in Table IV-3.

Estimates of upper-bound cancer risks resulting from ingestion of fish range from 10^{-4} for the "general consumer" to about 10^{-2} (one percent) for the "plausible maximum consumer." Figure IV-1 shows the relationship between average consumption of Tittibawassee River fish and the resulting upper-bound estimate of lifetime cancer risk, based on the calculations presented in

TABLE IV-3

RISK CHARACTERIZATION FOR INGESTION OF CDDs/CDFs[a]
IN FISH FROM THE TITTABAWASSEE RIVER

Exposure Scenario[c]	Upper-Bound Cancer Risk[d,e]	Hazard Index[b] (Ratio of Dose to RfD or HA)		
		Long-Term[e,f]	Single Meal[e,g]	
			Mean	Maximum
Plausible Maximum Consumer (bottom + game fish)	$1x10^{-2}$ ["B2"]	50	0.7[h]	8[i]
High Sports Fisherman (game fish only)	$4x10^{-3}$ ["B2"]	20	0.2	0.5
Median Sports Fisherman (game fish only)	$2x10^{-3}$ ["B2"]	9	0.07	0.2
General Consumer (game + clean fish)	$1x10^{-4}$ ["B2"]	0.7	0.04[j]	0.2

[a]Other contaminants, such as PCBs, found in the fish could add to the risks (see Appendix B).

[b]Note that Hazard Indices will be about 2-3 times higher for small children (Table III-33). Hazard Indices for breast-fed infants could be 10 times higher than those of their mothers.

[c]From data in Section III.E.2 and Tables III-31 and III-32.

[d]Upper-bound estimate of lifetime cancer risk, obtained by multiplying dose rate from Table III-31 by a cancer potency factor of $1.6x10^{-4}$ $(pg/kg\text{-}d)^{-1}$ and multiplying by a factor of 1.3 to incorporate contribution of higher intakes in childhood to average lifetime intake in pg/kg-day (from data in Table III-33).

[e]Note that all estimates of intake are "partial TEQs," including only 2378-TCDD, other TCDDs, HxCDDs, HpCDDs, and 2378-TCDF.

[f]Ratio of dose rate from Table III-31 to RfD of 1 pg/kg-day, for exposures lasting several months or longer.

[g]Ratio of bolus dose from Table III-32 to single-dose HA of 280 pg/kg-day.

[h]Includes some meals of bottom feeders.

[i]Bottom feeders only.

[j]Includes some clean fish.

Figure IV.1

Upper-Bound Cancer Risks Associated With Consumption of CDD/CDF Contaminated Fish From The Tittabawassee River

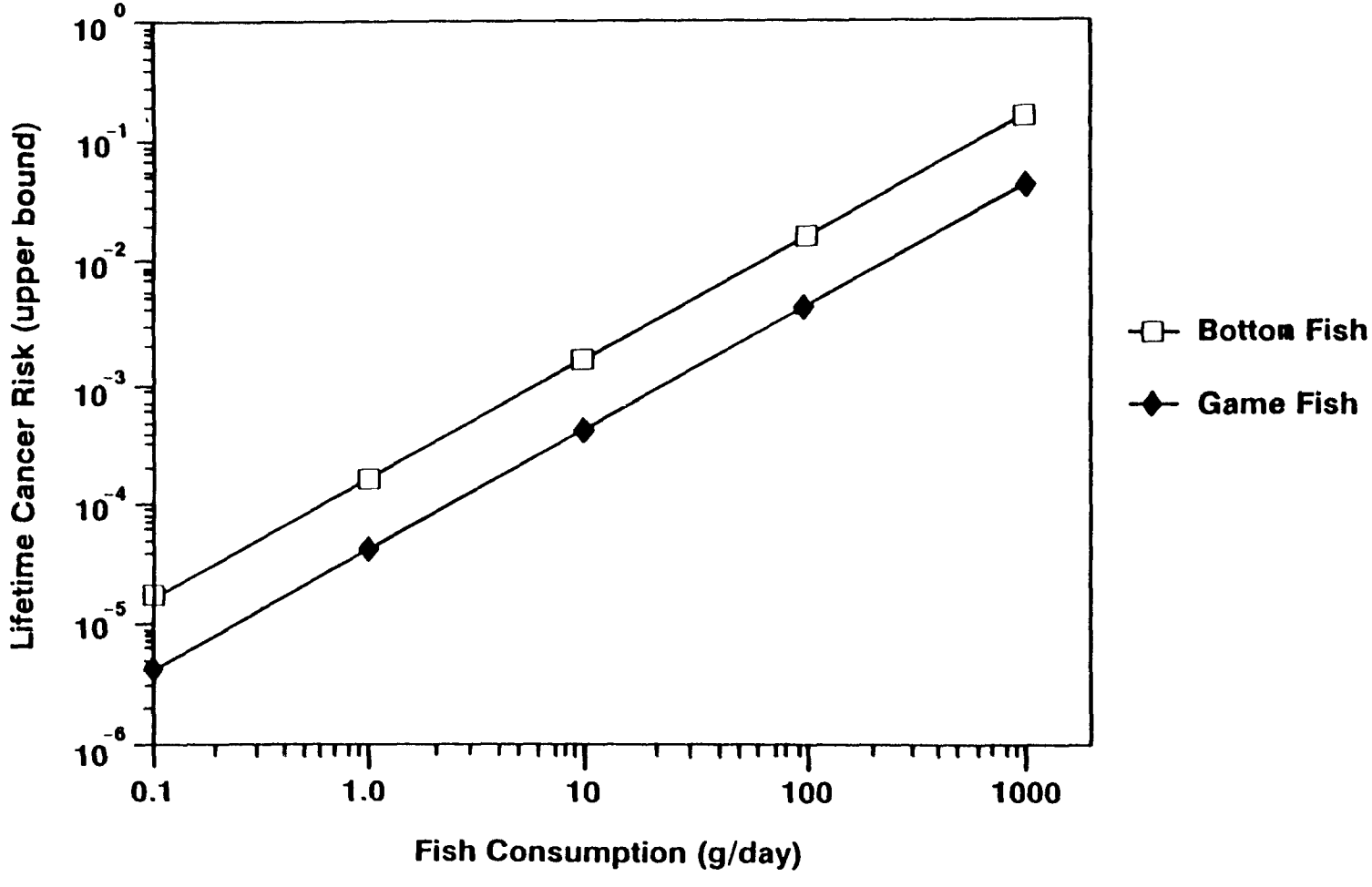


Table IV-3. Hazard Indices for long-term exposure (ratios of estimated intakes to RfD) range from 0.7 for the "general consumer" to 50 for the "plausible maximum consumer," with young children and breast-fed infants possibly 2 to 3 times and 10 times higher, respectively. Hazard Indices (ratios of estimated intake to HA) for single exposures are less than 1 for the "general consumer," the "median sports fisherman," and the "high sports fisherman" but range up to 8 for adults in the "plausible maximum consumer" case (the higher values being associated with consumption of bottom feeders). Again, young children and breast-fed infants could have higher HIs, as discussed above (breast-fed infants would be partially protected against the effects of high single intakes by their mothers because of pharmacokinetic averaging in the mothers' tissues).

Finally, in order to evaluate the potential risks of non-carcinogenic effects from short-term exposures, two consumption levels over a two-week period for each of the four scenarios are compared to the 10-day HA (see Table IV-4). The HIs for the higher level of consumption range from 0.4 for the "general consumer" to 5 for the "plausible maximum consumer." Thus, the HIs are in the same range as those resulting from the single-meal or one-day exposures.

In interpreting these estimates, the following should be borne in mind:

- The exposure estimates are subject to a number of limitations that have been discussed in Section III.E.4. The most important of these limitations is the lack of direct data on the consumption habits of Tittabawassee River fishermen; the data of Humphrey (1983), although relevant, refer to fishermen who fished in Lake Michigan.

TABLE IV-4

RISK CHARACTERIZATION FOR INGESTION OF CDDs/CDFs
IN FISH FROM THE TITTABAWASSEE RIVER

(Short-term exposures[a])

Exposure Scenario		Short-Term Hazard Index[b]
General Consumer (Each meal = 113 g of game fish or "clean" fish)		
Long-term average:	2 meals/month (1 of game fish) -- 1 meal of game fish/2 weeks	0.05
Plausible maximum:	14 meals of game fish/2 weeks (7 of game fish)	0.4
Median Sports Fisherman (Each meal = 113 g of game fish)		
Long-term average:	6 meals/2 weeks	0.3
Plausible maximum:	14 meals/2 weeks	0.7
High Sports Fisherman (Each meal = 255 g of game fish)		
Long-term average:	6 meals/2 weeks	0.7
Plausible maximum:	14 meals/2 weeks	2
Plausible Maximum Consumer (Each meal = 255 g of game fish or bottom feeders)		
Long-term average:	6 meals/2 weeks (3 of bottom feeders)	2
Plausible maximum:	14 meals/2 weeks (7 of bottom feeders)	5

[a]Exposures resulting from consumption of fish over a period of a few days to a few weeks.

[b]Average daily intake (pg/kg-day) divided by the 10-day HA of 28 pg/kg-day. Hazard indices for young children may be 2-3 times higher than the hazard indices for adults that are tabulated in this table.

- The cancer risk estimates are "upper bound" estimates of lifetime risk. That is, the actual risk is not likely to be greater than these levels; the actual risks could be significantly lower. Because of uncertainties about the mechanisms of action of 2378-TCDD and the implications of these mechanisms for dose-response modeling, these estimates are subject to additional uncertainties, as discussed above.

G. Estimates of Risks from Other Routes of Exposure

Other potential routes of exposure are discussed briefly in Section III.F. No quantitative estimates of exposure via ingestion or inhalation of indoor dust, or via ingestion of vegetables, meat, or milk, are possible, although each of these routes could be significant under appropriate circumstances. Breast-fed infants are likely to ingest CDDs/CDFs at dose rates (expressed in units of pg/kg-day) at least one order of magnitude greater than those of their mothers. Although these relatively high rates of intake by infants at a critical stage of development are of major concern, it is difficult to factor them into formal risk assessments, for the following reasons. First, USEPA's recommended procedure for assessing cancer risks resulting from time-varying exposures is to calculate the time-weighted average dose rate in mg/kg-day (USEPA 1986a). Infants are typically breast-fed for only about 1 percent of their lifetimes; hence, even if their dose rates are elevated 10-fold, this would lead only to a 1.1-fold increase in calculated lifetime risk. However, the validity of the averaging procedure is subject to question when the time-pattern of exposure is as extreme as this. Second, the HAs for CDDs/CDFs were derived from studies of adult animals, and the RfD from multigeneration feeding studies, none of which are good models for the short-term exposures to infants which are being discussed here. In the next section, risks to

breast-fed infants are characterized by assuming that their intakes are one order of magnitude higher than those of their mothers, and that the cancer potency factor, RfD, and HAs are applicable to them. However, the uncertainty of these assumptions should be recognized.

H. Integrated Risk Characterization

Because of the many limitations of the exposure and dose-response estimates that have been discussed in the preceding paragraphs, the estimates of upper-bound cancer risks and non-cancer hazard indices that are tabulated in Tables IV-1, IV-2, IV-3, and IV-4 should be regarded as reliable only to order of magnitude, i.e., to within about a factor of ten in either direction. To facilitate comparison of risks posed by exposures via different routes, Table IV-5 summarizes the order-of-magnitude upper-bound estimates of lifetime cancer risks, while Table IV-6 summarizes the estimates of Hazard Indices for other (non-cancer) toxic effects.

Tables IV-5 and IV-6 show that consumers of fish from the Tittabawassee River are at much higher risks than other residents in the Midland area. Under the assumptions listed in Part III (which include long-term consumption of fish at current levels of contamination), additional lifetime cancer risks would be in the range of 1 in 10,000 to 1 in 100; even consumers of game fish only could experience cancer risks above 1 in 1,000 and could exceed the maximum recommended long-term dose for non-cancer effects by 20-fold. Any consumer of bottom fish at current (1983-87) levels of contamination would exceed the

TABLE IV-5

SUMMARY OF UPPER-BOUND ESTIMATES OF CANCER RISK FROM EXPOSURE TO CDD/CDF CONTAMINATION IN MIDLAND, MICHIGAN

Exposure Route	Upper-Bound Cancer Risk (Exposure Scenario)	
	Higher Estimate	Lower Estimate
Fish	10^{-2} (plausible maximum consumer)	10^{-3} (median sports fishman)
	10^{-3} (high sports fisherman)	10^{-4} (general consumer)
Soil	10^{-5} (upper estimate)	10^{-6} (lower estimate)
	10^{-4} (child with pica)	--
Air	10^{-4} (fenceline)	10-5 (residential area)

NOTES:

(1) 10^{-2}, 10^{-3}, 10^{-4}, etc., indicate risks of roughly 1 in 100, 1 in 1,000, 1 in 10,000, etc.

(2) Other contaminants, such as PCBs, found in the fish add to the risk from that exposure route (see Appendix B).

Sources: Tables IV-1, IV-2, and IV-3.

TABLE IV-6

SUMMARY OF HAZARD INDICES FOR NON-CANCER EFFECTS
FROM EXPOSURE TO CDD/CDF CONTAMINATION IN MIDLAND, MICHIGAN

Exposure Route	Exposure Scenario	Hazard Index (HI)[a]		
		Long-Term	Short-Term	Single Meal
Fish[b]	Plausible maximum consumer	50	5	8
	High sports fisherman	20	2	0.5
	Median sports fisherman	9	0.7	0.2
	General consumer	0.7	0.4	0.2
Soil	Upper estimate young child			
	-- with pica	6	0.2	--
	-- normal	0.6	<0.1	--
	Lower estimate young child	<0.1	<0.1	--
	Upper estimate adult	<0.1	<0.1	--
Air[c]	Infant at fenceline	4	0.1	--
	Child at fenceline	1	<0.1	--
	Child in residential area	0.2	<0.1	--
	Adult in residential area	<0.1	<0.1	--

[a]Hazard Index is the ratio of intake dose to:

-- RfD (1 pg/kg/day) for long-term exposures (several months or more)
-- 10-day HA (28 pg/kg/day) for short-term exposures (few days to few weeks)
-- Single-dose HA (300 pg/kg/day) for single-meal or single-day exposures

[b]Small child could be at 2-3 times higher risk than adult. Breast-fed infant could be at 10-times higher risk than mother. Other contaminants such as PCBs, found in the fish, add to the toxicity (see Appendix B of the Risk Assessment).

[c]All HI values calculated using the "A method." Infant exposure includes exposure from breast-feeding.

single-meal and short-term health advisory intakes by 5- to 8-fold. These risks would be experienced by regular consumers of fish from the Tittabawassee River (including fishermen and their families). This population has still not been fully characterized, but an ongoing study is expected to provide more information about its size and other characteristics. In all exposure scenarios, small children of fishermen would be at greater risk than their parents, and breast-fed infants would be at highest risk.

According to the findings summarized in Tables IV-5 and IV-6, exposure via air and soil would not result in HIs greater than 10 for non-cancer effects (even in the unlikely case of an infant breast-fed by a woman resident at the fenceline, or in the case of a young child with pica). However, both air and soil exposures could pose upper-bound cancer risks exceeding 1 in 100,000. According to the exposure scenarios developed in Sections III.B and III.C, most long-term residents in the area north and east of the Dow Midland facility (i.e., about two-thirds of the population of the city--see Appendix A) would be subject to risks on the order of those associated with the "residential area" (air) and "lower exposure" (soil) scenarios. Residents nearer to the facility would be subject to risks nearer to those of the "fenceline" (air) exposure scenario.

According to the assumptions listed in the development of the exposure scenarios, most residents would be exposed to CDDs/CDFs via both the air and soil routes. Accordingly, exposures, and hence cancer risks, via these routes would be additive. However, adding the risks tabulated in Tables IV-1 and Table IV-2 would not change the general orders of magnitude indicated in

Table IV-5. Only an unusual combination of exposure circumstances (e.g., a breast-fed infant who later developed pica and remained in the area for most of the rest of his or her lifetime) would lead to an upper-bound estimate of cancer risk on the order of 1 in 10,000. Such risks are experienced by regular consumers of fish from the Tittabawassee River (Table IV-5).

In overall summary, this risk assessment indicates that the greatest health risks posed by CDDs/CDFs to residents of the Midland area result from the consumption of contaminated fish. Even individuals who limit their consumption to game fish can experience additional cancer risks exceeding one in a thousand and risks of reproductive effects and liver damage substantially above recommended levels. Exposure of city residents via contaminated air and soil poses smaller (but greater than one in a million) additional risks of cancer. All these conclusions should be interpreted keeping in mind the discussion of sources of uncertainty in earlier sections of this chapter.

PART V

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APPENDIX A

CHARACTERIZATION OF POTENTIALLY EXPOSED POPULATION

APPENDIX A

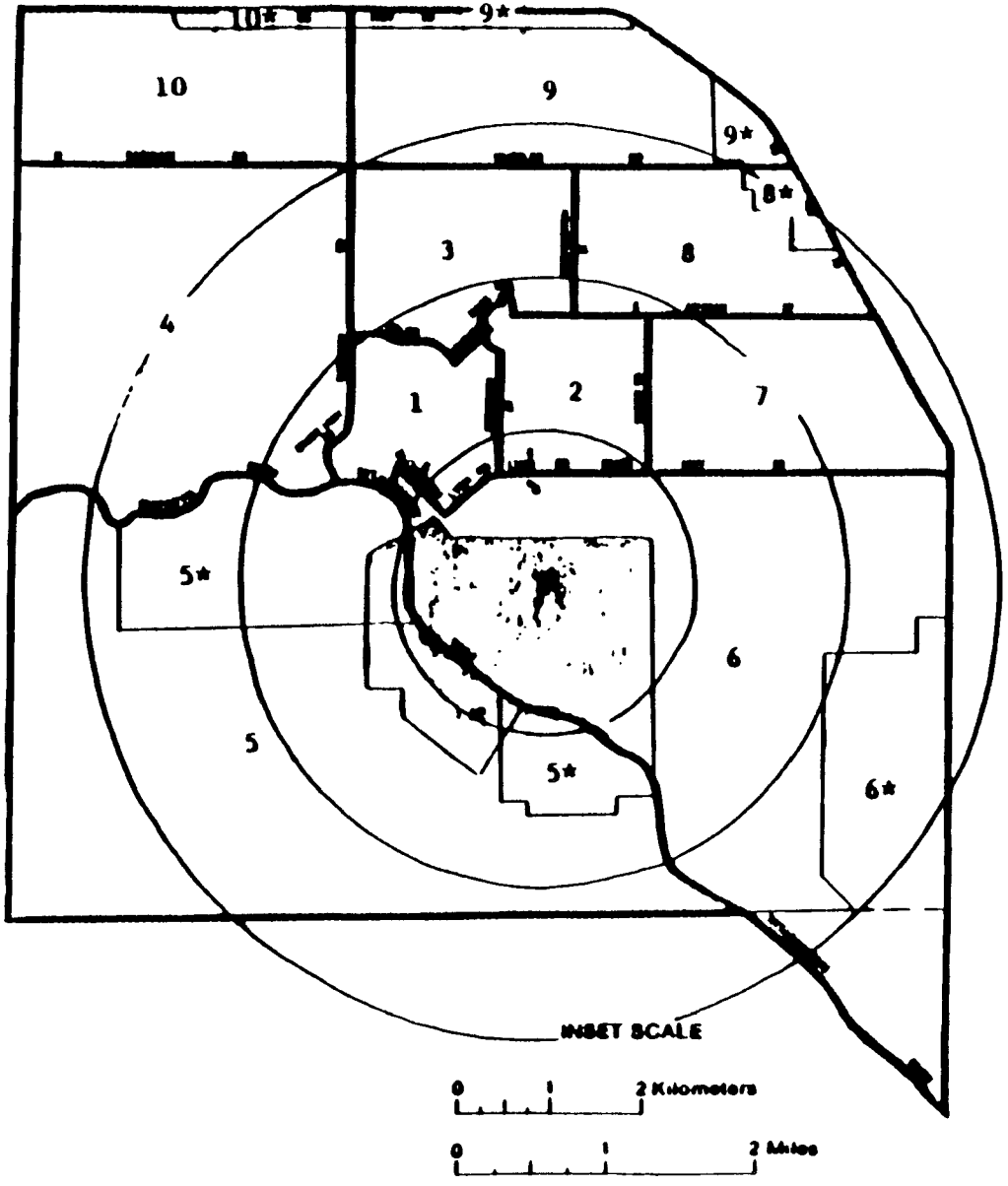
CHARACTERIZATION OF POTENTIALLY EXPOSED POPULATION

The Dow Chemical Company operates a major chemical processing facility within the town of Midland, Michigan (population 37,016). A map of the Midland area, divided into 1980 census tracts, is shown in Figure A-1. The population within each tract is shown at the right of the figure. Some tracts, namely 5, 6, 8, 9, and 10, have areas inside and outside of the Midland city boundary. The populations in these areas were tabulated separately. Areas inside the city limits are denoted by the census tract number only, while those outside are denoted by the census tract number with an asterisk. Also shown on the map are concentric circles marking 1,2, and 3 miles from the Dow Midland facility incinerator.

The Dow plant covers about 1,500 acres on the southwestern side of town along the banks of the Tittabawassee River. To the north of the plant lies the most densely populated area of Midland: the downtown area, the commercial district, and residential areas with schools, parks, playfields, and shopping malls. Moving east of the plant, the area remains residential but the population density decreases slightly. To the south and west of the plant, the population is the least dense and the area is predominantly rural/farming. The population is of normal age distribution and is very stable with 77% of the population having lived in the county for at least 5 years.

Chemical processing and hazardous waste storage and incineration practices at the plant have resulted in the release of CDDs/CDFs onto plant property and

FIGURE A-1: Map and populations of census tracts in Midland County, Michigan



Census Tract	Population
1	4031
2	3871
3	4612
4	3108
5	145
5*	1931
6	2521
6*	117
7	2887
8	5816
8*	19
9	5148
9*	13
10	4469
10*	91

into the ambient air (Trembly and Amendola 1987, USEPA 1985a). Inhalation of the ambient air around the plant is a potential route of exposure to CDDs/CDFs for Midland residents, especially those living near the plant perimeter or downwind of the incinerator. To the west and south of the incinerator, the plant boundaries extend more than a mile but to the north and east, toward the residential area, approximately 2,000 people can be found living within 1 mile of the incinerator (Figure A-1). The closest residences are approximately 0.5 mile from the plant incinerator and 0.1 mile or less from the plant boundary. In many areas, light industry and commerce begin at the plant boundary. An additional 11,000 people live within a two mile radius of the incinerator and 13,000 more within three miles. The majority of these populations are also concentrated in the residential/commercial areas to the north and east of the plant. As the prevailing wind is from the southwest, the downwind area includes the eastern edge of the most densely populated northern section and most of the less densely populated northeastern areas of town. An arc drawn 45 degrees to either side of the most prevalent wind direction and three miles from the incinerator encompasses approximately 12,000 people.

Inhalation of volatilized soil contaminants or suspended particulate matter is another component of the ambient air exposure. Since the highest concentrations of contamination have been found on plant property, the highest levels of exposure may be expected for those persons living nearest the plant perimeter or downwind of the plant property (USEPA 1985a). However, since soil samples taken from several residential and public use areas within the town also show contamination with CDDs/CDFs (USEPA 1985a), the actual number of residents with exposures above background is probably larger. The majority of

the city's residents may be exposed not only through inhalation pathways but additionally through ingestion of vegetables grown in gardens containing contaminated soil or of dust that has settled on eating vessels. There may be direct ingestion of contaminated soil by children and pregnant women, and regular use of contaminated public areas such as parks and playing fields may result in additional exposure for such users.

There has been no CDD/CDF contamination of surface water or potable water sources reported (Barna and Amendola 1985); therefore, this medium does not currently pose a significant route of exposure. However, the Dow Midland facility has several landfills and brine pond sediments contaminated with CDDs/CDFs (Amendola and Barna 1986), and the eventual contamination of ground or surface water could potentially place some residents at risk. While the majority of Midland residents receive their drinking water from Saginaw Bay, and the inlets are far removed from the plant area, an unknown number of residents draw their drinking water from at least one public and fourteen private ground water supplies and one artesian well in the vicinity of the Dow Midland brine operations and landfills (Amendola and Barna 1986).

Contaminated wastewater from the Dow Midland facility has been, and continues to be, released into the Tittabawassee River. Fish tend to accumulate CDDs/CDFs, and fish taken from the Tittabawassee have been found to be contaminated (Amendola and Barna 1986). Although the river is not fished commercially, it is fished recreationally, and the regular consumption of contaminated fish may be a significant route of exposure for recreational fishermen and their families. Specific data for the Tittabawassee fishermen

are unavailable, and the number of sport fishermen is unknown. There is evidence that the popularity of sport fishing is on the rise, and, with the stocking of the Tittabawassee, sport fishermen may represent a significant and increasing portion of the population.

Most of the residents of Midland are at risk of exposure to CDDs/CDFs through at least one of the above routes. Certain subpopulations may be more subject to exposure than others. Portions of the population at higher risk include women of child bearing age. Fifty percent of the Midland population is female and forty percent of the females are between the ages of 20 and 44 (1980 Census). Another sensitive subpopulation is disadvantaged residents. Many disadvantaged residents are thought to rely heavily on fish in their diet and may be consuming far more fish than the projected value for a typical sports fisherman. An estimate of the number of individuals in this category in the Midland area is unavailable; however, it may be significant since a recent survey of 128 Tittabawassee fishermen indicated that 72% were unemployed (Smith and Thompson 1984).

APPENDIX B

OTHER TOXIC POLLUTANTS PRESENT IN FISH

APPENDIX B

OTHER TOXIC POLLUTANTS PRESENT IN FISH

Limited sampling of fish from the Tittabawassee River has indicated that these fish may be contaminated with a variety of toxic pollutants in addition to CDDs/CDFs (Amendola and Barna 1986). Table B-1 summarizes concentrations of 9 pollutants (or groups of pollutants) detected in walleye specimens collected in 1985; this is the most systematic set of data available for comparison with concentrations of CDDs/CDFs. Seven of the 9 pollutants listed in Table B-1 are known to be carcinogenic, and their cancer potency factors (q_1^*) as determined by USEPA (1986d) are also listed in Table B-1. To compare the potential cancer risks posed by these pollutants with those posed by CDDs/CDFs, the right-hand column of Table B-1 presents a measure of the Relative Hazard, i.e., the product of the average concentration c and the cancer potency factor q_1^*. This Relative Hazard indicates the relative risks posed by the various pollutants, for the following reason.

The upper bound on the lifetime cancer risk R posed to a person of body weight W kg eating f grams of fish daily for a lifetime is given by the formula:

$$R = \frac{(f)(c)(q_1^*)}{(1,000)(W)},$$

where 1,000 is a unit conversion factor (kg/g). Thus, for any individual consumer of fish (with given values of W and f), R is proportional to the Relative Hazard ($c \times q_1^*$).

TABLE B-1

TOXIC ORGANIC POLLUTANTS
NATIVE FISH COLLECTION
TITTABAWASSEE RIVER 1985

	Walleye				
		Concentration (mg/kg)			
Compound	Number of Analyses	Range	Average	Potency Factor $(mg/kg\text{-}d)^{-1}$	Relative Hazard[5]
% Fat (hexane (extractables)	14	0.70-3.2	2.1		
PCTs (5432, 5442)	14	ND-0.500	0.093	NA[6]	-
PCBs (1254)	14	0.197-1.653	0.588	7.7	4.5
Chlordane[1]	14	ND-0.036	.009	1.3	0.01
DDT[2]	14	ND-0.212	.054	0.34	0.02
Dieldrin	14	ND-0.007	.001	30.	0.03
Hexachlorobenzene	14	0.002-0.038	.009	1.69	0.02
Toxaphene	14	ND-0.222	.097	1.1	0.11
Octachlorostyrene	14	ND-0.003	.001	NA[6]	-
Heptachlor epoxide	14	ND-0.005	.002	2.6	0.05
CDDs/CDFs (TEQ) [3]	14	2.5-15[4]	13[4]	1.6×10^{5}	2.1

1. Chlordane includes alpha-Chlordane, gamma-Chlordane, Oxychlordane, Cis-nonachlor and trans-nonachlor.

2. DDT includes p,p'-DDD, p,p'-DDE and p,p'-DDT

3. From Table III-27

4. Concentrations in ng/kg

5. Product of two previous columns

6. No cancer potency factor derived

Source: Amendola and Barna (1986).

Table B-1 indicates that for 6 of the 7 carcinogenic pollutants found in fish, upper-bound cancer risks posed by consumption of walleye from the Tittabawassee River would be 1-2 orders of magnitude lower than those posed by the CDDs/CDFs. For PCBs, however, upper-bound cancer risks posed by consumption of walleye from the Tittabawassee River would be similar to those posed by the CDDs/CDFs. Specifically, consumption of walleye by a 70-kg person at the median rate for sports fishermen of 48 g/day (see Section III.E) would lead to an upper-bound lifetime cancer risk of 3×10^{-3}. Since PCBs and CDDs/CDFs are similar in their environmental behavior and tend to concentrate in sediments and fish in the same way, it is likely that similar conclusions would hold for bottom-feeding fish also.

A similar comparative analysis of potential noncarcinogenic effects of the pollutants listed in Table B-1 indicates that PCBs may also be of comparable or greater concern than CDDs/CDFs at the relative levels found in the walleye from the Tittabawassee River. In a series of experiments reported by Allen et al. (1979), the LOAEL for adverse reproductive effects of PCBs (Aroclor 1248) in rhesus monkeys was 7 ug/kg-day (0.5 ppm in the diet administered 3 days/week); this was about 5,000 times higher than the LOAEL for 2378-TCDD (50 ppt in the diet, or about 1.5 ng/kg-day) in parallel experiments conducted in the same laboratory (Allen et al. 1979). In Tittabawassee River walleye, PCBs were present at concentrations about 110,000 times higher than those of CDDs/CDFs (TEQs) (Table B-1). Thus, at the comsumption rates discussed in Section III.E, PCBs would be judged to pose reproductive hazards in addition to those posed by CDDs/CDFs. None of the

other toxic pollutants listed in Table B-1 would pose reproductive hazards at the levels of contamination and rates of intake listed.

Although the source of PCBs in the Tittabawassee River is not known and is not believed to be related to the Dow Midland facility, it is necessary, for the reasons given in the two previous paragraphs, to consider whether the presence of PCBs in the Tittabawassee River fish may augment the hazards attributable to CDDs/CDFs. PCBs are complex mixtures, some of whose components act by the same mechanisms as 2378-TCDD and cause similar toxic effects, although with much lower potencies (Poland and Knutson 1982, Safe et al. 1985a,b). Unfortunately, few studies of the joint effects of PCBs and CDDs/CDFs have been reported. Birnbaum et al. (1985) reported that one PCB component appeared to act synergistically with 2378-TCDD in inducing cleft palates in mice. However, Haake et al. (1987) and Bannister et al. (1987) have recently reported that co-administration of a PCB mixture (Aroclor 1254) protected mice against the immunotoxic and teratogenic effects of 2378-TCDD. All these studies involved acute administration of PCBs and 2378-TCDD, and their relevance to chronic exposures is uncertain. Also, the protective effect reported by Safe et al. (1987) was observed only for relative doses (PCBs:2378-TCDD) in a range lower than that occurring in Tittabawassee River fish. Much more study is needed before definitive conclusions can be drawn about effects of joint exposure to PCBs and CDDs/CDFs.

Another hypothetical set of interactions between CDDs/CDFs and other toxic pollutants that needs to be considered is initiation/promotion interactions. 2378-TCDD is known to act as a potent late stage carcinogen, or

promoter of carcinogenesis initiated by other carcinogens (Pitot 1986). 2378-TCDF has also been reported to act as a promoter (Poland et al. 1983, Poland and Knutson 1982), and by inference, other CDDs/CDFs are likely to have similar activity. Hypothetically, therefore, CDDs/CDFs ingested in fish might act to promote cancers initiated by other carcinogens, thus augmenting the risks posed by either group of chemicals considered in isolation. In fact, however, none of the other carcinogens listed in Table B-1 is known to act as a cancer initiator, and several are known or suspected to act primarily as late stage carcinogens. Thus, it is not clear that initiation/promotion interactions would significantly augment the risks posed by CDDs/CDFs under the circumstances of exposure prevailing in the Tittabawassee River.

APPENDIX C

NOMENCLATURE FOR CHLORINATED DIBENZO-p-DIOXINS AND DIBENZOFURANS

APPENDIX C

NOMENCLATURE FOR CHLORINATED
DIBENZO-p-DIOXINS AND DIBENZOFURANS

The following terminology and abbreviations are used in this document:

1. The term "congener" refers to any one particular member of the same chemical family: e.g., there are 75 congeners of chlorinated dibenzo-p-dioxins.
2. The term "homologue" refers to a group of structurally related chemicals which have the same degree of chlorination. For example, there are eight homologues of CDDs, monochlorinated through octachlorinated.
3. The term "isomer" refers to substances which belong to the same homologous class. For example, there are 22 isomers that constitute the homologue of TCDDs.
4. A specific congener is denoted by unique chemical notation. Commas are omitted for brevity. For example, 2,4,8,9-tetrachlorodibenzofuran is referred to as 2489-TCDF.
5. Notation for homologous classes is as follows:

Dibenzo-p-dioxin	D
Dibenzofuran	F

No. of Halogens	Acronym	Example
2	D	24-DCDD
3	Tr	
4	T	2378-TCDD
5	Pe	12378-PeCDF
6	Hx	123478-HxCDD
7	Hp	
8	O	
1 through 8	CDDs and CDFs	

6. Dibenzo-p-dioxins and dibenzofurans that are chlorinated at the 2,3,7, and 8 positions are denoted as 2378-substituted congeners; e.g., 12378-PeCDF and 23478-PeCDF are both referred to as "2378-substituted-PeCDFs".

APPENDIX D

BROMINATED COMPOUNDS

APPENDIX D

BROMINATED COMPOUNDS

Recent studies have indicated that combustion of wastes containing brominated compounds can give rise to brominated dibenzo-p-dioxins and dibenzofurans as well as mixed brominated/chlorinated dibenzo-p-dioxins and dibenzofurans (Rappe et al. 1979, Buser 1987, Hutzinger and Thoma 1987). These bromine-containing compounds (collectively referred to as BrDDs/BrDFs) have at least some of the biochemical activity of CDDs/CDFs and some of them are thought to have toxic potencies approaching those of CDDs/CDFs (Mason et al. 1986a, Mason et al. 1987). Dow Chemical has manufactured brominated organic compounds at the Midland facility until 1987, and it is likely that some bromine-containing wastes have been sent to the waste incinerator. Hence, it is likely that some BrDDs/BrDFs have been emitted from the plant. However, no investigations of their possible presence in the Midland environment have been conducted.

APPENDIX E

POSSIBLE HAZARDS TO WILDLIFE

APPENDIX E

POSSIBLE HAZARDS TO WILDLIFE

Little information is available to serve as the basis for assessment of potential hazards to wildlife posed by residues of CDDs/CDFs in the Midland area. As documented in Section III.E, residues of CDDs/CDFs (and other contaminants, including PCBs) have been detected in fish in the Tittabawassee River. It can therefore be presumed that other aquatic organisms in the river, along with consumers of aquatic life, such as fish-eating mammals and fish-eating birds, are exposed to these contaminants. It is also likely that contaminated sediments have been transported downstream as far as Saginaw Bay, raising the possibility that aquatic wildlife there may be exposed to CDDs/CDFs. For example, Stalling et al. (1983) reported the presence of CDD/CDF isomers in the tissues of fish and fish-eating birds collected from the vicinity of Saginaw Bay.

Several studies have suggested that fish are adversely affected by 2378-TCDD at water concentrations as low as 100 ppq (Yokim et al. 1978). These and other studies (Miller et al. 1979, Branson et al. 1985) suggest that adverse effects on fish are associated with whole-body concentrations of 2378-TCDD in the range of 1-2 ppb or higher. Fish tissue concentrations of up to 700 ppt 2378-TCDD (equivalent to about 900 ppt TEQ) have been reported in fish in the Tittabawassee River (Table III-25). This approaches the lowest concentration reported as associated with adverse effects. However, in view of the limited range of species tested and the short duration of the experimental studies (up

to 114 days), it is not possible to determine whether or not adverse effects may be occurring.

Virtually no information is available on the toxicity of CDDs/CDFs to fish-eating mammals or birds, except that the mink is extremely sensitive. In a paper presently available only as an abstract, Hochstein et al. (1986) reported that the 128-day dietary LC_{50} for 2378-TCDD in mink was 0.85 ppb, only slightly higher than the highest concentration reported in Tittabawassee River fish. Field studies in Green Bay, Wisconsin, have shown reproductive impairment in fish-eating birds associated with residues of CDDs/CDFs and PCBs (Hoffman et al. 1987, Kubiak et al. 1987). An unpublished report by Kurita et al. (1987) documents reproductive impairment in the same species (Forster's tern) in Saginaw Bay in 1987. This raises the possibility that residues of CDDs/CDFs (and/or other pollutants) may have accumulated in the Saginaw Bay ecosystem to levels that pose chronic hazards to wildlife.