DIOXIN CASE STUDY

Prepared in Conjunction with the

Commission for Environmental Cooperation's

Continental Pollutant Pathways Project

May 1997

(Note: This PDF Version was created November 2000, and is slightly different than the May 1997 original version. The only change is Figure 6, and its description in the text, which has been updated, to allow new electronic graphic images to be used in the document)

Case Study

Dioxin

Authors:

Mark Cohen, CBNS, Queens College, Flushing, NY, USA
Barry Commoner, CBNS, Queens College, Flushing, NY, USA
Alfonso Espitia Cabrera, Universidad Autonoma Metropolitana, Mexico City, Mexico
Derek Muir, Freshwater Institute, Environment Canada, Winnepeg, Manitoba, Canada
Carlos Santos Burgoa, Instituto de Salud, Ambiente y Trabajo, Mexico City, Mexico

TABLE of CONTENTS

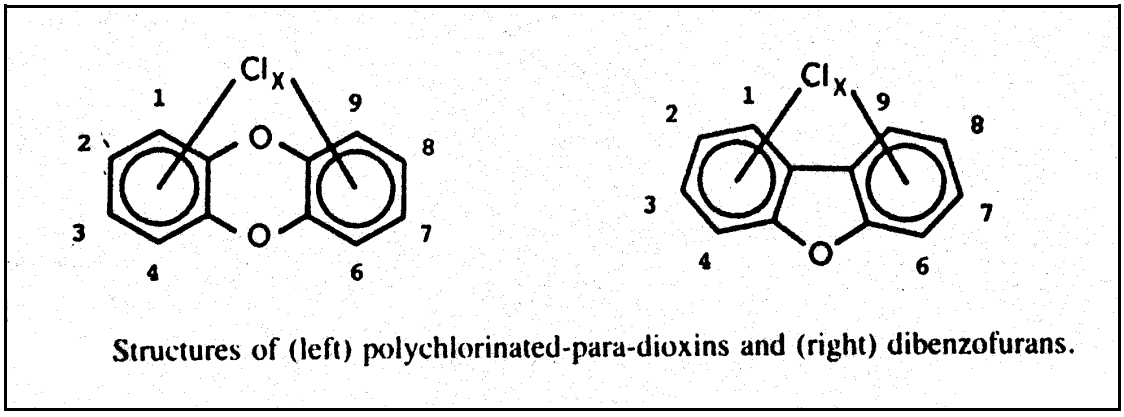
1. List of Tables and Figures

2. Introduction

"Dioxin" is the term commonly given to a class of compounds known chemically as polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans (PCDD/F's). As discussed below, there are other compounds beside PCDD/F's that exhibit dioxin-like toxicity. Evidence suggests that human exposure to PCDD/F's and dioxin-like compounds can lead to various forms of cancer and various developmental and endocrine-disruption effects.

There are 210 different chlorinated dioxin and furan molecules or "congeners." Each different congener has its chlorines in different positions in the structure. The general structure of PCDD/F molecules is shown in the diagrams below; also, as examples, the specific congeners 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD) and 2,3,7,8-tetrachlorodibenzofuran (2,3,7,8-TCDF) are shown.

It has been found that only the 17 PCDD/F congeners with chlorines in at least the 2,3,7,8 positions show evidence of dioxin-like toxicity. The toxicity of each congener can be expressed in terms of its estimated, approximate potency relative to that of the most toxic congener (2,3,7,8-tetrachlorodibenzo-*p*-dioxin) by multiplying by its estimated "Toxic Equivalency Factor" or TEF. Values of the TEF's commonly used are given in Table 1 below. The overall PCDD/F toxic potency of a mixture of dioxin and furan congeners is often summarized by multiplying the amount of each congener by its TEF and then summing over all the congeners to get the total Toxic Equivalents (TEQ) in the mixture. The meaning of a result expressed as "TEQ" is that the total overall toxicity of the mixture is presumed to be equivalent to the stated amount of "pure" 2,3,7,8-TCDD.



Structures of (left) polychlorinated-para-dioxins and (right) dibenzofurans.

Table 1. Toxic Equivalency Factors (TEF's) for PCDD/F Congeners

(U.S.EPA 1994a,b)

Dibenzo-*p*-dioxins		**Dibenzofurans**	
Congener(s)	**TEF**	**Congener(s)**	**TEF**
dibenzo-*p*-dioxin molecules with three or less chorine atoms (27 total)	0	dibenzofuran molecules with three or less chlorine atoms (59 total)	0
2,3,7,8-TCDD	**1**	**2,3,7,8-TCDF**	**0.1**
all other TCDD's (21 total)	0	all other TCDF's (37 total)	0
1,2,3,7,8-PeCDD	**0.5**	**2,3,4,7,8-PeCDF** **1,2,3,7,8-PeCDF**	**0.5** **0.05**
all other PeCDD's (13 total)	0	all other PeCDF's (26 total)	0
1,2,3,4,7,8-HxCDD **1,2,3,6,7,8-HxCDD** **1,2,3,7,8,9-HxCDD**	**0.1** **0.1** **0.1**	**1,2,3,4,7,8-HxCDF** **1,2,3,6,7,8-HxCDF** **1,2,3,7,8,9-HxCDF** **2,3,4,6,7,8-HxCDF**	**0.01** **0.01** **0.01** **0.01**
all other HxCDD's (7 total)	0	all other HxCDF's (12 total)	0
1,2,3,4,6,7,8-HpCDD	**0.01**	**1,2,3,4,6,7,8-HpCDF** **1,2,3,4,7,8,9-HpCDF**	**0.01** **0.01**
all other HpCDD's (1 total)	0	all other HpCDF's (2 total)	0
OCDD	**0.001**	**OCDF**	**0.001**

Abbreviations:

TCDD = Tetrachlorodibenzo-*p*-dioxin
PeCDD = Pentachlorodibenzo-*p*-dioxin
HxCDD = Hexachlorodibenzo-*p*-dioxin
HpCDD = Heptachlorodibenzo-*p*-dioxin
OCDD = Octachlorodibenzo-*p*-dioxin

Abbreviations:

TCDF = Tetrachlorodibenzofuran
PeCDF = Pentachlorodibenzofuran
HxCDF = Hexachlorodibenzofuran
HpCDF = Heptachlorodibenzofuran
OCDF = Octachlorodibenzofuran

As mentioned above, there are other persistent organic pollutants with a similar chemical structure to 2,3,7,8-substituted PCDD/F congeners which exhibit dioxin-like toxic effects. For example, there are at least 13 PCB congeners that are considered to exhibit dioxin-like toxicity (3 non-*ortho* PCB's, 8 mono-*ortho* PCB's, and 2 di-*ortho* PCB's[1]). Other compounds, such as chlorinated naphthalenes, chlorinated diphenyl ethers, and a range of brominated analogs, may also exhibit dioxin-like toxicity.

TEF values for the dioxin-like PCB congeners have been estimated (e.g., Ahlborg *et al*., 1994), although, as with the PCDD/F TEF's, there is some uncertainty in these estimates (e.g., Wenning and Iannuzzi, 1995). With estimated TEF values, the contribution of dioxin-like compounds to the overall dioxin-like potency of a mixture can be assessed relative to the CDD/F congeners present in the mixture.

There have been relatively few side-by-side measurements of PCB's and PCDD/F's in environmental media or tissue samples. The relative importance of PCDD/F's and PCB's to overall dioxin-like potency varies from study to study.

In studies reporting measurements of cow milk, PCDD/F's and PCB's appear to contribute roughly comparable dioxin-like potencies, e.g.:

- the Netherlands: Slob *et al.* (1995) and van der Velde *et al*. (1994);
- England: Krokos *et al.* (1996); Sewart and Jones (1996);

---

[1]. Polychlorinated biphenyls are molecules which have two benzene rings bonded together by a single chemical bond. On each of the benzene rings, there can be chlorine atoms attached in any position. Thus, there are 209 possible PCB "congeners", ranging from biphenyl (with no chlorines) to decachlorobiphenyl (with 10 chlorines). The positions on the benzene rings adjacent to the chemical bond joining the two rings are called the "*ortho*" positions; there are two such *ortho* positions on each benzene ring, each ring has one *ortho* position on each side of the joining bond. If there are no chlorines occupying an ortho position, the two benzene rings exist in essentially the same plane, i.e., they are coplanar. This coplanar, polychlorinated configuration (with at least two chlorines on each ring) is similar to the structure of 2,3,7,8-TCDD. Due to steric considerations, if there *are* chlorines in one or more *ortho* positions, the benzene rings are unable to exist in the same plane (the chlorine atom(s) get in the way); thus, these are somewhat less like 2,3,7,8-TCDD, although mono-*ortho* PCB's and a few di-*ortho* PCB's have been found to exhibit dioxin-like toxicity. Based on the commonly used set of estimates by Ahlborg *et al.* (1994), 3,3',4,4',5-PCB (PCB-126) and 3,3',4,4',5,5'-PCB (PCB-169) are considered to have approximate TEF's (their dioxin-like potency relative to 2,3,7,8-TCDD) of 0.1 and 0.01, respectively; all other dioxin-like PCB's are believed to have TEF's of 0.0005 or less. In some cases, even these less toxic congeners are present in sufficiently high concentrations that they contribute significantly to the overall dioxin-like toxicity in a given situation.

The same rough equivalence has been found in many studies of human milk, e.g.:

- Japan: Hirakawa *et al.* (1995);
- the Netherlands: Liem *et al.* (1993); Tuinistra et al (1994,1995);
- England: Duarte-Davidson *et al.* (1992).

In a comprehensive study of food in the Netherlands, PCDD/F's and PCB's were estimated to contribute approximately the same total dioxin-like potency in the average diet (Theelen *et al.*, 1993).

In measurements in aquatic ecosystems, it has been frequently found that the dioxin-like PCB's are responsible for the majority of the dioxin-like potency measured, e.g.:

- Great Lakes region (fish and fish eating birds): Smith *et al.* (1990);
- St. Lawrence River (fish): Brochu *et al.* (1995);
- the Netherlands (porpoise): Van Scheppingen *et al.* (1996)

A similar enhanced significance of PCB's (relative to PCDD/F's) appears to hold for many of the measurements made in arctic ecosystems, e.g.:

- Dewaily et al., 1994 (milk samples from Inuit women in arctic Quebec);
- Ayotte et al., 1995 (blood plasma from Inuit in arctic Quebec);
- Hebert et al., 1996 (Canadian arctic caribou fat and other tissues);
- Muir et al., 1996 (a range of Canadian arctic species)],

Pollutant levels in the arctic are discussed in much greater detail in the case study on Persistent Organic Contaminants in the Canadian Arctic (presented elsewhere in this Volume).

In addition, the case study on Persistent Organic Pollutants (POP's) (presented elsewhere in this Volume) provides detailed information about a range dioxin-like POP's and other POP's.

Much remains to be learned about PCDD/F's and other dioxin-like compounds. This case study will focus primarily on PCDD/F's, as an example.[2]

[2]. The narrowed focus of this particular case study on PCDD/F's is not meant to imply that these compounds are necessarily the most important of the dioxin-like POP's.

3. Sources

PCDD/F's have never been produced intentionally (except in extremely small quantities for use in research). Unfortunately, however, it is formed as an unwanted byproduct in many different types of processes.

The two broad classes of formation situations which are the generally regarded as being the most important are:

(1) combustion processes (e.g., waste incineration, fuel combustion, metallurgical processes);

(2) solution-phase processes (e.g., pulp bleaching with chlorine or chlorine containing compounds, chlorinated organic chemical manufacturing, chlorine production)

Formation of PCDD/F's in any given source results from a complex set of chemical reactions which are influenced by many factors, including, but not limited to, the individual concentrations of many potentially relevant reactants, temperature, and inter- and intra-phase mass transfer processes. PCDD/F formation can be enhanced when certain precursors are present (e.g., chlorophenols; *un*chlorinated dibenzo-p-dioxin), but, significant quantities can also be formed in the absence of the introduction of any known precursors. The relative proportions of different PCDD/F congeners created in different formation situations can be highly variable. While the formation of PCDD/F's in combustion and solution phase processes is exceedingly complex and only poorly understood at the present time, it is obvious that chlorine and/or another chlorine-containing compound is essential to their formation.

3.1 Emissions to Air

As mentioned above, PCDD/F's can be formed in many types of combustion systems (e.g., Commoner *et al*., 1987; Addink and Olie, 1995; Karasek, 1995). In these systems, PCDD/F formation appears to be most favored in the temperature range from about 300 - 450 ^{o}C. This is, of course, cooler than most combustion chambers; PCDD/F's are believed to form as the combustion exhaust gases cool *after* they leave the combustion chamber. PCDD/F's can form in the gas phase or on the surface of aerosol particles in the cooling stack gases. Certain compounds (e.g., copper) appear to act as a catalyst in surface-formation reactions.

There have been many attempts to determine the influence that chlorine -- in its various forms -- has on the formation of PCDD/F's in combustion systems. In a broad overview of a range of combustion-process emissions of PCDD/F's, Thomas and Spiro (1995) found a general increase in emissions as the level of chlorine in the material being combusted increased.

In certain situations, a positive correlation appears to be conclusively established. One such situation is the combustion of gasoline in the internal combustion engines of automobiles. When leaded gasoline is used, chlorinated and brominated substances (e.g., ethylene dichloride and ethylene dibromide) are added to the fuel. This is done so that the lead in the gasoline does not build up in the engine and thereby cause damage; during combustion, the chlorinated additives convert most of the lead in the fuel to relatively volatile lead chlorides which are able to escape from the engine. (The fact that the lead escapes from the engine may be good for the engine, but obviously, is not so good for the environment.) Experimental measurements have shown that PCDD/F emissions from automobiles burning leaded gasoline are significantly higher than those from unleaded gasoline (e.g.: Hagenmaier *et al*. 1990; Bingham *et al*. 1989; U.S. EPA 1994a; Marklund *et al*. 1987,1990). These measurements provide strong evidence that chlorinated substances are linked to PCDD/F formation.

In incineration processes, the situation is considerably less straightforward. A recent summary of experimental investigations involving particular combustion situations was provided by Wikstrom and coworkers (1996). These researchers also reported a new set of experimental data concerning this issue. They burned a simulated municipal waste in a laboratory-scale combustor and found that increasing levels of chlorine in the waste led, generally, to increasing PCDD/F emissions in the exhaust gases. In this experiment, inorganic chlorine (calcium chloride) and organic chlorine (polyvinyl chloride) was equally effective in leading to the formation of PCDD/F's.

In general, some studies have found a positive correlation between PCDD/F emissions from incineration and chlorine input, and some studies have not. In one recent review of available data, it was concluded that the available experimental evidence does *not* support the idea that PCDD/F formation generally increases with the amount of chlorine or chlorine-containing substances involved in the process (Rigo *et al*., 1995). These conclusions have been challenged in a recent review which concludes that there is a significant relationship between chlorine input and PCDD/F emissions (Costner, 1997).

In any given combustion setting, there may be a threshold effect in which PCDD/F formation begins to increase with increasing chlorine input *above a certain level of chlorine input*. This has been observed in several experiments (as summarized by Wikstrom *et al*. 1996). Given the complexity of the physical and chemical processes occurring, it is not surprising that the apparent threshold level appears to vary among different types of incinerators, combustion conditions, and fuels. Due to the complexity of the situation and a lack of data, the relative contribution of various chlorinated substances in the formation of PCDD/F's in many types of combustion systems remains controversial and poorly understood.

Pollution control equipment can capture some of the PCDD/F that has been formed and prevent its immediate escape from the system. The PCDD/F captured in pollution control systems for combustion devices is associated with the captured flyash. A portion of the captured PCDD/F may be re-released to the environment during handling, transport or from its ultimate

disposal site (e.g., an ashfill) to the extent that any of the following processes occur: (a) ash particles can become airborne through the action of wind or mechanical action; (b) PCDD/F's can volatilize from the ash; or (c) PCDD/F's or PCDD/F-containing ash particles can be washed into surface or ground water by precipitation.

Certain pollution control systems can actually increase the amount of PCDD/F's formed. For example, some electrostatic precipitators (ESP's) operate at a relatively high temperature — in the range in which PCDD/F formation is favored — and it has been found that more PCDD/F leaves these devices than enters them. Emissions of PCDD/F from municipal waste incinerators equipped with these so-called "hot-sided" ESP's are believed to be extremely large (e.g.: U.S. EPA 1994a; Webster and Connett, 1997).

Quantitative estimates of the amounts of PCDD/F's emitted by various sources are fraught with difficulty for several reasons.

First, for any given type of source (e.g., medical waste incinerators), very few individual facilities have actually been tested for emissions of PCDD/F's.

Second, the tests that have been done show that PCDD/F emissions can vary greatly from test to test on the same facility and from facility to facility of the same type (i.e., similar process and pollution control characteristics).

Third, overall emissions inventories and emissions estimates for given facilities are often made by using the few available tests to estimate emissions factors for a given type of source, with a given type of pollution control equipment. The use of emissions factors to estimate emissions is obviously an approximation. In order to even use such factors, however, one must be able to assemble a complete list of the sources of a given type, with details of each facility's emissions-relevant process characteristics, pollution control devices, and throughput (e.g., tons/day burned in an incinerator). Unfortunately, this does not appear to be possible for many types of sources, even those that are relatively significant.

For example, there are many uncertainties in currently available inventories of medical waste incinerators in the United States. Most State agencies are currently unable or unwilling to assemble an accurate list with relevant details (e.g., location, throughput, pollution control equipment). Because of this basic lack of knowledge, confusion and controversy surrounds the estimate of emissions from medical waste incinerators in the United States. A similar lack of information appears to exist in Canada for medical waste incinerators. The state of knowledge about medical waste incinerators appears to be more advanced in Mexico, with estimates available for the throughput and locations for each of the 10 incinerators known to exist.

Several general PCDD/F emissions summaries and/or emissions inventories have been presented; examples, in alphabetical order, include the following: (i) Cohen M., B. Commoner, *et al*. (1995); (ii) Fiedler, H., *et al*. (1990,1992); (iii) Harrad, S., *et al*. (1992); (v) Thomas and

Spiro (1995, 1996); (vi) Thornton (1994) and Thornton and Weinberg (1995); (iv) USEPA (1994a, 1995, 1996a); (vii) Zook and Rappe (1994)].

Currently, there is not enough information in Canada, Mexico or the United States to develop an accurate emissions inventory for PCDD/F's. For most source classes in each country, the situation is as follows: (a) very few (if any) emissions tests have been made, and so even the development of rough emissions factors is difficult; (b) even if emissions factors were available, adequate facility-level details about location, throughput, process, and pollution control equipment either have not been assembled or are being kept confidential by government or industry groups. Efforts are underway in each country by government agencies and/or non-governmental organizations to improve the accuracy of PCDD/F emissions inventories, although it would appear that more resources need to be allocated to these efforts.

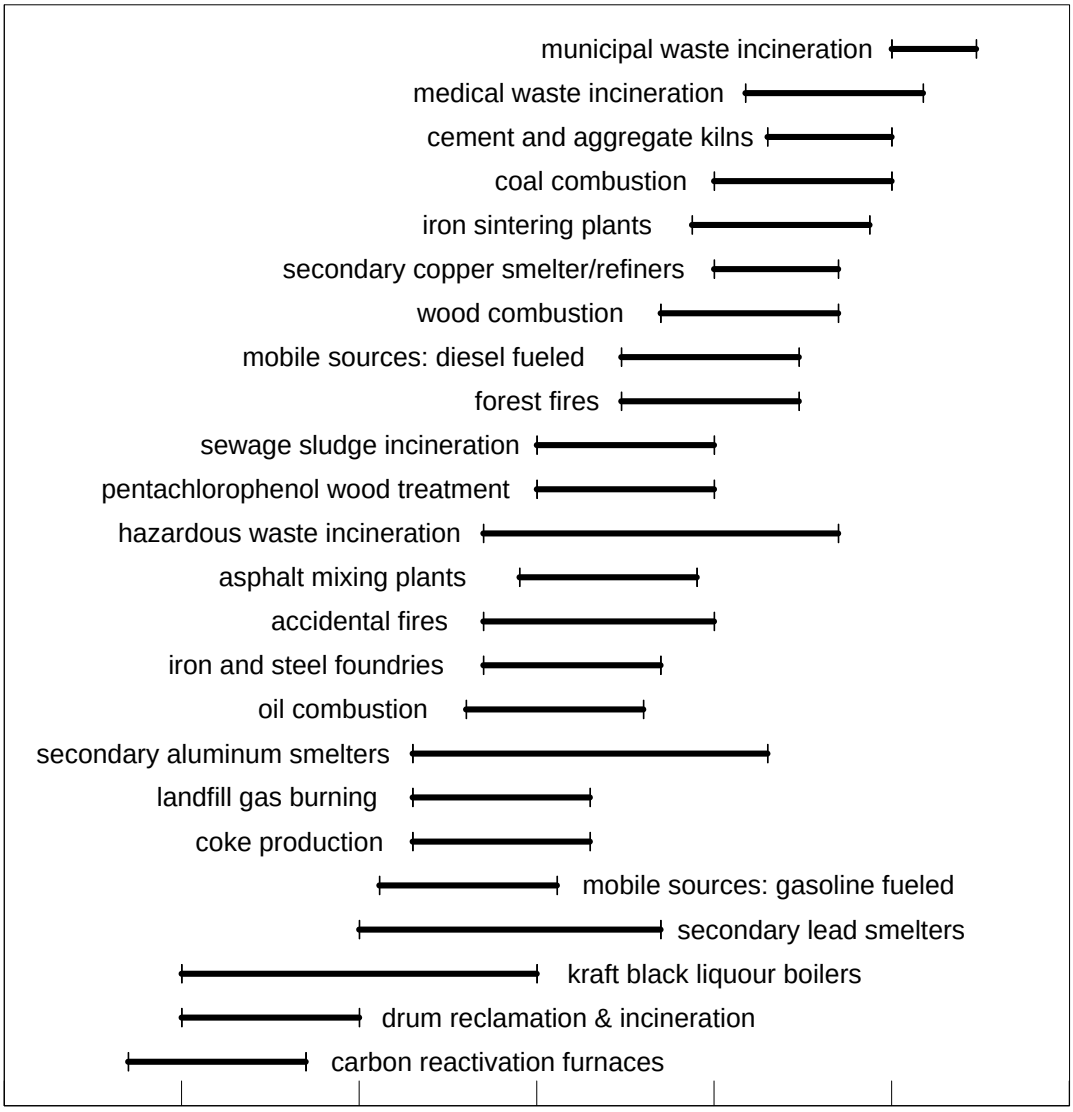
In light of the above, one must be cautious in presenting estimates for PCDD/F emissions. As a rough guide, a *qualitative* summary of approximate, estimated PCDD/F air emissions from various sources in the United States is given in Figure 1. For many of the sources in this Figure, the uncertainties in emissions are very large.

In Figure 1, no estimate was made — due to an extremely high level of uncertainty — for emissions from in-use pentachlorophenol (PCP)-treated wood (e.g., PCP-treated telephone poles). PCP is contaminated with PCDD/F, as an unintentional byproduct of its chemical synthesis, and a portion of this PCDD/F may be emitted during the lifetime of the treated wood. PCP use is currently restricted in the U.S. and Canada, but, it is still used extensively used as a wood preservative in certain applications (Feldman and Shistar, 1997). Emissions may be significant even if only a small fraction of PCP's dioxin contamination was emitted.

In the U.S., there are several ongoing PCDD/F inventory efforts, including (but not necessarily limited to) those by the U.S. EPA Office of Air Quality Planning and Standards (U.S. EPA 1995, 1996a), the U.S. EPA's National Center for Environmental Assessment (Cleverly, 1996), and an inventory currently being developed at the Center for the Biology of Natural Systems at Queens College.

Many of the sources shown qualitatively in Figure 1 for the U.S. also exist in Canada and Mexico. However, there may be significant differences in the relative importance of different sources in Canada and Mexico, compared to the situation in the U.S.

Figure 1. Emissions of PCDD/F to the Air in the United States: Qualitative Summary



"TEQ" = 2,3,7,8-TCDD Toxic Equivalents
The Indicated "Ranges" for Each Source Show the Approximate Uncertainty in the Emissions Estimate

In Mexico, initial data for a PCDD/F emissions inventory is being assembled. Initial consideration is being given to the following potential sources currently operating in Mexico: (a) medical waste incinerators; (b) hazardous waste incinerators[3]; (c) the petrochemical industry; (d) cement kilns; (e) combustion of leaded gasoline in automobiles[4]; (f) metallurgical processes (Mexico is a major copper producer); and (g) brick production facilities (where uncontrolled waste combustion is typically used as a heat source). Future work will attempt to estimate the relative significance of the above sources, and the role of other PCDD/F sources in Mexico.

Many of the sources in the U.S. also exist in Canada, although their relative importance is expected to be somewhat different. A preliminary emissions inventory for Canada was developed by the Center for the Biology of Natural Systems (Cohen *et al.*, 1995). There is a current effort led by the government-sponsored Federal/Provincial Task Force on PCDDs/PCDFs to create a nationwide emissions inventory (Morcos, 1997). A stakeholder workshop about the inventory development is to be held on March 26, 1997 in Ottawa.

Based on their chemical similarity, essentially all of the sources that emit PCDD/F's to the air will also emit other dioxin-like compounds (e.g., dioxin-like PCB's). Although combustion is not thought to be an important source of PCB's, there is evidence that some congeners (e.g., 3,4,3',4'-tetrachlorobiphenyl) can be emitted from combustion sources (Brown and Silkworth, 1995). Emissions data for these other dioxin-like compounds is even more scarce than that for PCDD/F's, however. Congener-specific emissions measurements from sources are practically non-existent. PCB's were extensively used in a range of applications until the 1970's, and residues from these earlier uses are ubiquitous (e.g., PCB-contaminated soil). PCB emissions from these residues of past uses are still occurring. While PCB use is being phased out, there are still ongoing "closed-system" applications (e.g., old electrical transformers). Large quantities of PCB oils from past uses are currently be stored, awaiting destruction. There have also been unintentional, accidental releases from storage/waste treatment sites as well as from transformer fires.

---

[3]. A new initiative in Mexico has begun in which a system of Integral Centers for the Management and Utilization of Industrial Residues ("Centros Integrales para el Manejo y Aprovechamiento de Residuos Industriales - CIMARI'S") is to be developed. These Centers are expected to be involved in the following aspects of industrial waste management: (a) recycling of oils and solvents; (b) energy recovery from certain combustibles; (c) sludge treatment; (d) treatment of residues with heavy metals; (e) treatment of residues with organic substances; (f) thermal oxidation; and (g) solidified and stabilized waste confinement.

[4]. Although the use of leaded gasoline is being phased out, it is still used somewhat. Thus, emissions from automobiles may play a relatively greater current role as a PCDD/F source in Mexico than it does, for example, in the U.S. and Canada. It should be noted, however, that leaded gasoline is still used as an aviation fuel in the U.S.

Thus, the construction of an accurate emissions inventory for PCB's (including dioxin-like PCB's) is perhaps even more complicated than that for PCDD/F's. As a consequence, we are perhaps further away from an accurate emissions inventory for PCB's than we are for PCDD/F's. As an example, in a recent draft PCB emissions inventory for the United States (U.S. EPA 1996a), no congener-specific emissions estimates could be made, emissions from only a few of the source categories considered in Figure 1 could be included, and, no estimates could be made for emissions from PCB-contaminated sites, accidental releases from transformer leaks and fires, or environmental residues from past contamination.

3.2 Emissions to Water

PCDD/F emissions to water have been a major environmental concern over the past 15 years in Canada and the U.S. In some cases, the sources of PCDD/Fs are the same as those for the atmosphere, such as runoff from or disposal of combustion-related wastes, leakage of PCB oils, PCP use (e.g. treated wood pilings). Wet and dry deposition of airborne PCDD/Fs can be important sources to water bodies (Hites, 1990; Cohen *et al.*, 1995), either directly or through runoff. Sources unique to water involve unintentional chlorination of natural dibenzo-p-dioxin or dibenzofurans, and condensation of chlorophenols and chlorinated hydroxydiphenyl ethers, under aqueous conditions.

Chlorine bleach kraft pulp and paper mills (BKM) are perhaps the most thoroughly studied source. PCDDs/Fs were first detected in the late 1980's in the pulp, effluent, and sludge of BKM's which used elemental chlorine (Keuhl *et al.*, 1987; Merriman *et al*., 1991; Swanson *et al.*, 1988; Amendola *et al.*, 1989; Clement *et al.*, 1989; Safe, 1991) and in many pulp products such as paper, coffee filters, and diapers (Safe, 1991). The predominant PCDD/Fs identified in the effluent of BKM's were 2,3,7,8-TCDD, 2,3,7,8-TCDF, and 1,2,7,8-TCDF. The congener profile of BKM emissions is unique and can be distinguished from other PCDD/F sources such as combustion and PCP's (Yunker and Cretney 1996). Beginning in 1989, the pulp and paper industry in the U.S. and Canada has reduced emissions of PCDD/Fs through process changes, including the use of other bleaching agents such as chlorine dioxide, ozone and hydrogen peroxide (Swanson *et al.*, 1988; Craig *et al.*, 1990).

Sewage treatment plants, especially those using elemental chlorine for disinfection are also sources of PCDD/Fs, due to aqueous chlorination reactions that may be similar to those in kraft pulp mills using chlorine bleaching. Atmospheric deposition may play a role as well, as urban runoff — which may be contaminated with PCDD/F from wet and dry deposition onto urban surfaces — is often routed to sewage treatment plants. In addition, industrial sources and leaching from dioxin-contaminated PCP applications may contribute PCDD/F's to sewage treatment plants, a portion of which may ultimately be discharged from the plant into receiving surface waters.

3.3 Emissions to Land

In some instances, PCDD/F-containing material is disposed of on land. Current examples of this include disposal of incinerator ash, sewage sludge, and sludge from pulp and paper manufacturing involving chlorine bleaching. In the past, the practice of land disposal of hazardous waste from the chemical manufacturing industry was significant, but, is now restricted. A dramatic example is the Hyde Park landfill in Niagara, New York, about 2000 feet east of the Niagara River, in which an estimated 0.7 - 1.6 tons of 2,3,7,8-TCDD was dumped between 1954 and 1975 (U.S. EPA, 1990). It has been speculated that this site has the most PCDD/F in any single location anywhere on earth[5]. PCDD/F's from this and other sites can be emitted to the air through volatilization and dust erosion processes (wind and mechanical), and to surface water or groundwater. Field and laboratory measurements suggest that PCDD/F's are relatively stable in the soil environment; half-lives appear to be at least on the order of 10 years or longer (Brzuzy and Hites, 1995; Freeman and Schroy, 1989; Hagenmaier *et al*., 1992; Kapila *et al*., 1989; Orazio *et al*., 1992; Puri *et al*., 1989). The reasons for this stability are the following: PCDD/F's (a) bind relatively tightly to soil particles, (b) are not very water soluble, and (c) are relatively resistant to degradation by microorganisms or by abiotic processes (e.g., photolysis) in the soil environment. Thus, the only way that PCDD/F's can efficiently "leave" the soil environment is when soil particles become airborne (i.e., windblown dust) or waterborne (e.g., soil erosion).

As mentioned above, leachate and erosion from contaminated waste sites may be an important source of PCDD/F to surface and ground water. While there is unfortunately very little data to make quantitative estimates, waste sites along the Niagara River may be very significant current contributors to the PCDD/F loading of Lake Ontario.

4. Exposure Pathways

PCDD/F's are a ubiquitous contaminant of food, water, air, soil, vegetation and commercial products. Thus, we are exposed to PCDD/F's through the food we eat, the air we breathe, the water we drink, and through dermal (skin) exposure to any contaminated material. Human exposure is defined as the contact of an environmental agent with the human body. Exposure is not equal to dose, but is a realistic approximation to it (Johnson, 1992). Currently in health research, exposure assessment has become a key element of analysis, and for environmental exposures with a long term effect, retrospective exposure assessment is now being developed (Correa *et al.*, 1994). Research has demonstrated the need to consider all the routes of human exposure (e.g., Ott, 1985; Smith, 1988). The most important route of human exposure

---

[5]. To put this amount in perspective, the total amount of PCDD/F emitted to the air in the U.S. is on the order of 5,000 - 10,000 grams TEQ/year — about 10 - 20 pounds TEQ per year. Thus, the amount of dioxin estimated to be in the Hyde Park landfill is the equivalent to approximately 70 - 320 years of current U.S air emissions of PCDD/F.

to PCDD/F is believed to be through food consumption (USEPA 1994a; Zook and Rappe, 1994; Startin, 1994; Schecter, 1994bc).

4.1 Food - Aquatic and Terrestrial

As mentioned above, the most important PCDD/F exposure pathway of most people is believed to be through food, although there have been very few measurements of the PCDD/F levels in various foods. Based on the limited available data, the most important foods contributing to exposure appear to be milk, dairy products, beef[6] and fish (USEPA 1994a; Zook and Rappe, 1994; Startin, 1994; Schecter, 1994bc). Startin (1994) has recently reviewed PCDD/F levels in different foods.

A very approximate summary of the relative PCDD/F contributions of different foods to the diet in the U.S. (from Schecter *et al*., 1994c) and Canada (from Birmingham *et al*., 1989) — both based on extremely limited data — is given in Figure 2. Similar results have been found in food surveys done in Germany (Beck *et al*., 1989; Furst *et al*., 1990), Britain (Startin 1994), and the Netherlands (Theelan *et al*., 1993).

For adults in the U.S., two recent estimates of the total estimated background exposure due to food consumption are 116 pg/day (U.S. EPA 1994a) and 18 - 192 pg/day (Schecter *et al*., 1994c). Assuming an average adult weight of 65 kilograms, these estimates are equivalent to an exposure of about 2 and 0.3 - 3 pg/kg of body weight, respectively.

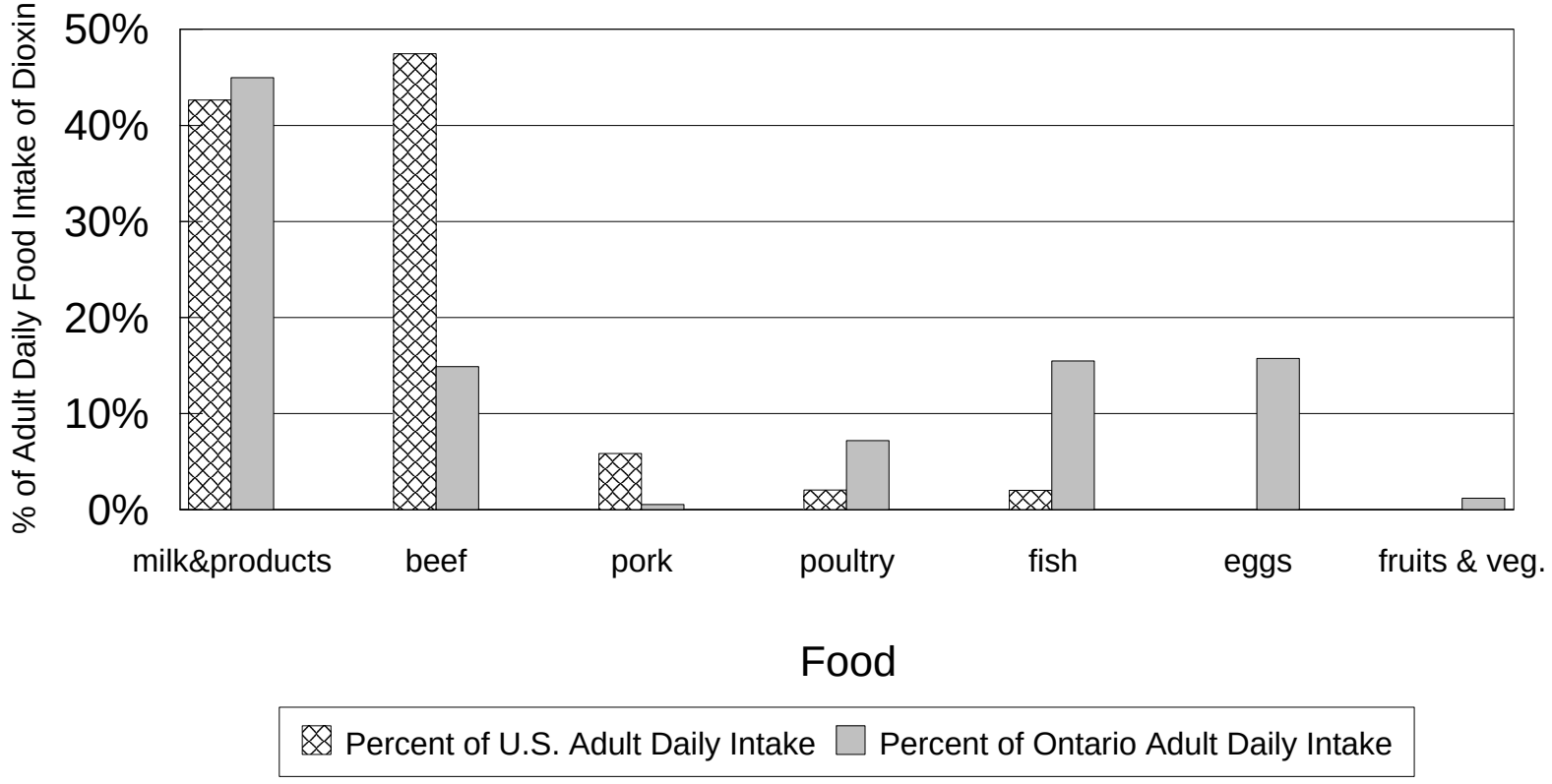
For infants, exposure through breast feeding may result in a higher intake of PCDD/F than adults. On a per-kilogram of body weight basis, a breast feeding infant's exposure appears to be *significantly* higher than adults. For infants exposed through breast milk, the exposure has been estimated to be on the order of 35 - 53 pg/kg of body weight (Schecter *et al*., 1994c).

---

[6]. Other animal products, e.g., eggs, chicken, pork, are also somewhat significant contributors as well, although in the U.S., these are considered to be of lesser importance. A Canadian study estimated that eggs contributed about as much PCDD/F to the diet as beef (Birmingham *et al*., 1989).

Figure 2. Relative Importance of Dioxin Exposure from Different Foods

(Estimates are based on extremely limited data and should be regarded as preliminary approximations only)



Note: Exposure from Egg Consumption Not Included in U.S. Data, Because of Lack of Data

U.S. Data: Schecter et al. (1994), Envr Health Persp 102(11): 962-966 (geometric mean of low & high estm's used)

Ontario Data: Birmingham et al. (1989), Chemosphere 19(1-6):507-512.

4.1.1 PCDD/F in Terrestrial Food Chains

Based on their analysis of available data, the EPA estimated that nearly all of the general population's exposure to PCDD/F is through food, of which milk and dairy products account for about 36% and beef for about 32% (USEPA 1994a). These estimates are consistent with those of Schecter *et al*. (1994c), who estimated that dairy products and beef contributed approximately 43% and 48%, respectively, of PCDD/F intake from food for adults[7].

Measurements of PCDD/F's in terrestrial food chains include measurements in beef cattle in the U.S. (Lorber *et al.*, 1996), Caribou in the Canadian arctic (Hebert *et al.*, 1996), and a range of milk and milk products as discussed below.

Given their importance to human exposure, the levels and origin of PCDD/F in dairy products will be discussed here in some detail.

The following measurements have been reported for PCDD/F's in cow's milk in the United States:

(a) a study in the vicinity of the municipal solid waste incinerator in Rutland, Vermont (USEPA 1991), before and after the incinerator began operating, in which a total of 11 milk samples from 4 different sites were analyzed for PCDD/F content;

(b) a study conducted by ENSR at a farm near the Elk River Generating Station in Elk River, Minnesota (ENSR, 1989), in which duplicate samples of cow's milk were collected from a single farm, once in the summer and once in the winter.

(c) a study conducted for the California Air Resources Board (Stanley and Bauer, 1989) in which 8 packaged milk samples were analyzed;

(d) a study by LaFleur *et al*. (1990) in which one sample of milk was analyzed for 2,3,7,8-TCDD, 2,3,7,8-TCDF and 1,2,7,8-TCDF before exposure to bleached paperboard milk cartons and after varying periods of exposure;

(e) a study conducted by the Food and Drug Administration (Glidden *et al.*, 1990) in which a total of approximately 20 milk samples were taken from 15 dairies in 6 states; samples of milk were taken both before and after the milk was placed in cartons;

---

[7]. Schecter *et al*. (1994c) report "low" and "high" estimated exposure rates from food consumption, based on a range of contamination levels in foods. Because the low and high estimates are more than an order of magnitude apart, a geometric (rather than arithmetic) average was used in summarizing this analysis.

(f) a study conducted in New York State (Schecter, *et al*., 1989) in which 3 samples (whole milk, 2% fat milk, and heavy cream) were obtained from a grocery store in Binghamton, New York;

(g) a study conducted at five farms in Connecticut (Eitzer, 1995) in the vicinity of a new municipal solid waste incinerator, before and after the incinerator began operating, in which a total of 29 samples were analyzed.

In Canada, retail milk products purchased between 1985 and 1988 in six different Canadian cities were analyzed for PCDD/F (Ryan *et al.*, 1991). It was found that PCDD/F's from the chlorine-bleached plastic-coated paperboard containers appeared to be leaching into the milk products, adding significantly to the levels of PCDD/F's in the milk. The substitution of chlorine dioxide for elemental chlorine as a bleaching agent at many paper mills has led to significant reductions in the levels of PCDD/F's in paperboard containers.

No data could be found on the PCDD/F content of milk in Mexico.

There have been several studies conducted in Europe of PCDD/F in cow's milk, including: Fanelli *et al*. (1980), Rappe *et al*. (1987), Riss *et al*. (1990), Rappe *et al*. (1990a), Beck *et al*. (1990a,b), Ryan *et al*. (1990 & 1991), Schmid and Schlatter (1992), Furst *et al*. (1992 & 1993), DeJong *et al*. (1993), Lassek *et al*. (1993), Slob *et al*. (1995), and Harrison *et al*. (1996).

The U.S. EPA (1994a) estimated the U.S. background PCDD/F concentration in milk using data from LaFleur *et al*. (1990) and the Rutland study (USEPA 1991). These estimates, coupled with estimates of PCDD/F content of other foodstuffs, air, and water, were used to estimate the total exposure of the general population.

The U.S. EPA is currently conducting a nationwide survey of PCDD/F levels in cow's milk, using a nationwide sampling network originally designed to monitor milk contamination by radioactive fallout. Samples are being collected from dairy facilities serving at least one large metropolitan area in each state. The analysis for PCDD/F may be done on regionally-pooled composite samples. In addition, the U.S. Food and Drug Administration is conducting a market-basket survey of PCDD/F in milk, dairy products and fish. These studies will provide much-needed additional data on PCDD/F levels in food in the U.S.

How do PCDD/F's get into dairy and animal products? The major route of exposure of dairy cows, beef cattle and other animals to PCDD/F is thought to be their consumption of PCDD/F-contaminated feed crops (McLachlan *et al*., 1990; Furst *et al*., 1993; Stevens and Gerbec, 1988). PCDD/F's enter crops largely from the air; they are not generally taken up by plants from soil or groundwater (McCrady *et al*., 1990). Dry deposition of vapor-phase material may be the dominant mechanism by which they enter vegetation (Welsch-Pausch *et al*., 1995). As discussed below, the more toxic tetra- and penta-chloro PCDD/F congeners (i.e., TCDD/F

and PeCDD/F) are relatively volatile and exist to a greater extent in the vapor phase in the atmosphere than do the less toxic higher-chlorinated congeners. Thus, it is "unfortunate" that vapor-phase PCDD/F's appear to be more efficiently transferred to vegetation.

Ingestion of contaminated soil is normally of minor importance but can be significant in some situations (Fries 1995ab). Soil contamination by PCDD/F can arise from atmospheric deposition and from the application of PCDD/F-contaminated fertilizers and soil amendment products (e.g., sewage sludge, animal manure, etc.). The use of PCDD/F-contaminated pesticides *may* be another contributor to the levels of PCDD/F's in animal feed crops, although it is probably of lesser importance.

The 2,3,7,8-substituted PCDD/F's are not broken down efficiently by metabolic pathways in most animals; for example, approximately 20% of the PCDD/F ingested by dairy cows is excreted in their milk (McLachlan *et al*., 1990). Recent reviews of PCDD/F's in agricultural systems include those by Fries (1995ab), Hattemer-Frey and Travis (1991), Lorber *et al*. (1994), and McLachlan (1996).

A collaborative project — involving the Center for the Biology of Natural Systems at Queens College, the New England Environmental Policy Center (director: Jean Richardson), the University of Wisconsin's Department of Dairy Science, and others — is currently being conducted in which PCDD/F is being measured and modeled in the air, crops, and cow's milk of eight dairy farms in Wisconsin and Vermont. It is hoped that this study will lead to an increased understanding of the modes of entry, relative levels, and fate of PCDD/F in dairy ecosystems.

4.1.2 PCDD/F in Aquatic Food Chains

The primary pathway of human exposure as a result of PCDD/F contamination of aquatic ecosystems is through the consumption of contaminated fish. PCDD/F's can enter aquatic ecosystems as a result of:

- sources which discharge PCDD/F's directly to the water (e.g, a pulp and paper mill using chlorine or a chlorine-containing compound for pulp bleaching);

- sources which discharge PCDD/F's to the air, a portion of which may enter a given water body as a result of atmospheric deposition.

In the aquatic environment, PCDD/Fs with four or more chlorines are found mainly associated with particulate organic carbon (POC) and dissolved organic carbon (DOC) (Broman *et al.*, 1991; Merriman *et al.*, 1991; Pastershank and Muir, 1995). The smallest particle sizes (< 1.0 um) generally contain the highest organic carbon and are potentially good carriers of bound PCDD/F's (Broman *et al.* 1991; Muir *et al.* 1992a). Particle-bound PCDD/F's are removed from

the water column by sedimentation but are also recycled within the water column, especially in large, dynamic, water bodies (Broman *et al.* 1991).

The fate and distribution of PCDD/F's in aquatic ecosystems has been examined in a series of studies in an experimental mesocosm using radiolabelled compounds (Corbet *et al.*, 1983; Servos *et al.*, 1992ab; Friesen *et al.*, 1995; Segstro *et al.*, 1995). These studies have shown that, upon entry into aquatic systems, PCDD/Fs partition rapidly to POC. Degradation of PCDD/F's in sediments could not be detected over a five-year monitoring period. PCDD/F's were slowly lost via diffusion from sediment pore waters into the water column, as well as via burial of sedimenting particles, and were also accumulated by mussels and crayfish and exported from mesocosms by emerging insects (Segstro et al. 1995; Fairchild et al. 1992).

There have been several studies of PCDD/Fs in aquatic sediments in Canadian waters (Macdonald *et al.* 1992; Evans *et al.*, 1996; Bourbinierre *et al.*, 1996) and in the Great Lakes (Czcuzwa and Hites 1984; 1986). The sediment studies are very useful for identifying trends in sources and historical inputs. Results from Great Slave Lake (Evans *et al.*, 1996) and Lake Athabasca (Bourbonniere *et al.*, 1996) suggest a small pre-industrial historical input of OCDD, presumably due to forest fires.

PCDD/F's bioaccumulate in the aquatic food chain and can reach significant levels, even though they are rarely (if ever) found above detection levels in the water column. Recent reviews of PCDD/F's in aquatic ecosystems include those by Cook *et al*. (1991), Fletcher and McKay (1993), and Walker and Peterson (1994). Clarke *et al*. (1996) have recently reviewed the levels of PCDD/F's found in fish in the United States and international waterways. Their review indicated that PCDD/F contamination in fish is ubiquitous, with quantifiable levels found worldwide.

A long-term monitoring program of the eggs of herring gulls (a fish eating bird species) has provided useful information on the spatial and temporal variation of PCDD/F's in the Great Lakes regional aquatic ecosystem (e.g., Hebert *et al.*, 1994). Measurements showed a general decline in PCDD/F levels between 1981 and 1984, but no discernable trend between 1984 and 1991. Birds eating fish in Saginaw Bay (Lake Huron) and Lake Ontario showed significantly higher levels of PCDD/F in their eggs than those in other colonies in the Great Lakes region.

4.2 Other Exposure Pathways

Under typical "background" conditions, i.e., removed from any strong local source of PCDD/F contamination, exposure pathways other than food consumption are believed to be of minor significance.

Using an estimated background drinking water PCDD/F contamination level of 0.0056 pg TEQ/liter (based on 214 measurements) and a typical adult water consumption rate of 1.4

liters/day, the U.S. EPA (1994a) estimated that exposure from water consumption was on the order of 0.008 pg TEQ/day, about 0.01% of the typical exposure due to food consumption.

Using an estimated background air concentration of 0.095 pg TEQ/m^3 (based on 84 measurements) and a typical adult inhalation rate of 23 m^3/day, the U.S. EPA (1994a) estimated that exposure from air inhalation was on the order of 2 pg TEQ/day, perhaps accounting for a few percent of the typical background exposure due to food consumption. A similarly small amount (~ 1 pg/day) was estimated for soil ingestion (U.S. EPA 1994a), under background conditions.[8]

Dermal exposure is believed to be important in some circumstances for certain pollutants (e.g., pesticide exposure of agricultural workers involved in applying pesticides (Granjean, 1990). Investigations into dermal exposure to PCDD/F's have shown that the skin is not an effective barrier against PCDD/F (e.g., see discussion in Montague, 1989). Under background conditions, however, dermal exposure to PCDD/F is not expected to constitute a significant exposure pathway.

Under certain "non-background" situations, however, these other PCDD/F exposure pathways may be important.

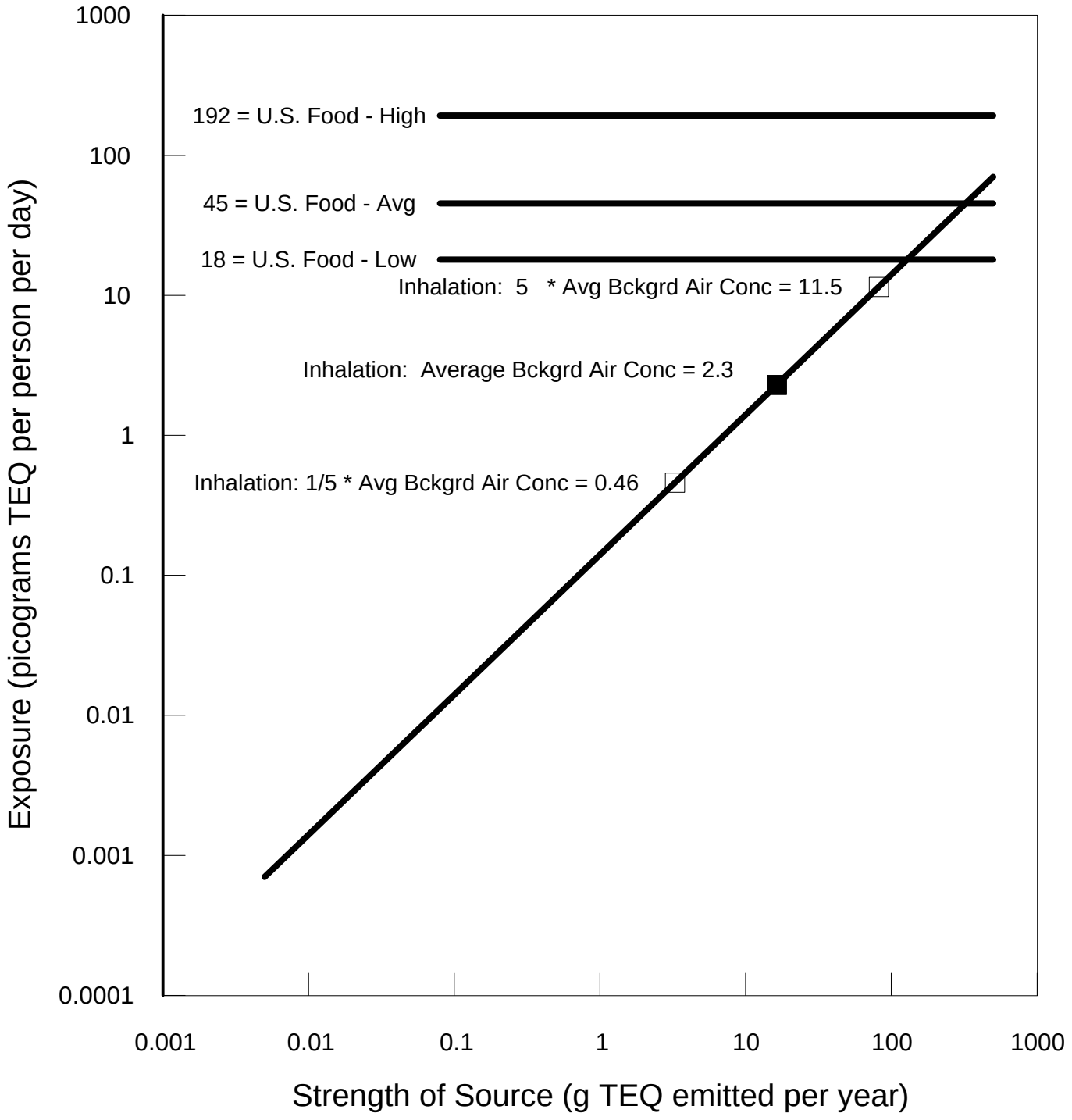
In some occupational settings, for example, exposure to PCDD/F (presumably through air and dust inhalation) may be significant. Any occupational environment associated with a significant PCDD/F source (see Figure 1) could involve significant occupational exposure. An example is municipal waste incinerator operation, where it has been found, for some plants, that workers show evidence of a higher level of exposure than the general population (presumably as a result of exposure to PCDD/F- contaminated air and dust) (Schecter 1994b).

Even for some non-occupational situations, non-food exposure routes *may* be important. Ground level concentrations downwind of facilities which emit very large quantities of PCDD/F's can reach significant levels, and people who live or work near such a facility might be significantly exposed through inhalation or dust ingestion.

As an illustration of this, consider the data presented in Figure 3, below. Here, the inhalation exposures predicted for an adult living and working downwind of PCDD/F sources of varying magnitudes are compared against the range of background food exposures as presented by Schecter (1994c). The inhalation exposure estimates in this figure are based on air dispersion calculations presented by the U.S. EPA (1994a) for the annual average ground-level air concentrations (in the direction of maximum impact) resulting from emissions from a

[8]. Soil ingestion (called "pica") is a more significant exposure route for children than it is for adults (Hayes and Rodenbeck, 1992).

Figure 3. Exposure to PCDD/F's from Food vs. Inhalation (U.S. Adults)



Food Data from Schecter et al., 1994c; Background Air Conc. from USEPA 1994a; Assumed Inhalation Rate: 23 m3/day; Inhalation 1 km from "Source" based on Dispersion Modeling in USEPA 1994a (stack height = 30.5 meters)

hypothetical facility[9]. It can be seen from this example that persons in close proximity to sources or source complexes emitting on the order of 100 g TEQ per year or larger may be exposed to significant amounts of PCDD/F (i.e., rivaling or surpassing the exposure from food consumption).

Are there any facilities with such large PCDD/F emissions? Certain municipal waste incinerators with “hot-sided” electrostatic precipitators (as discussed above in the section on sources) may have emissions of this magnitude. For example, during a 1992 stack test of a municipal waste incinerator in Columbus, Ohio, the emissions rate was estimated to have been on the order of 1000 g TEQ per year. When the facility was tested more recently, the level fell to approximately 200 grams TEQ per year, possibly the result of "special" waste preparation: the waste was allegedly pre-selected, dried, and plastics were removed (Connett, 1994)[10]. Other similar incinerators have been found to emit PCDD/F at rates of 100 g TEQ/yr or more (Webster and Connett, 1997). Certain large iron sintering plants may emit PCDD/F at a similarly high rate (based on emissions factors extrapolated from European facilities), but, data on their emissions in North America are almost non-existent.

4.3 Summary of Exposure Pathways

While atmospheric and aquatic transport widely disseminate PCDD/F’s from their numerous sources, the environmental concentrations can vary significantly from place to place, depending in detail on the proximity to sources. Also, depending on the specific physical-chemical properties of the different PCDD/F congeners and the materials they interact with, different materials exposed to even the same concentrations will exhibit different levels of contamination. Thus, for example, all things being equal (i.e., if no other sources were significant contributors), the concentrations of PCDD/F in the air a kilometer or two immediately downwind of an incinerator will be higher than those tens of kilometers away. In a similar way, the concentration of PCDD/F in cow’s milk will be higher at a farm on which dairy feed was grown under higher atmospheric concentrations of PCDD/F (due to relative proximity to sources) than at a farm with feed grown under lower concentrations. Moreover, different crops at any given farm may have different average concentrations of PCDD/F in them, even though they are

---

[9]. The hypothetical facility was modeled after an actual waste incinerator burning 200 metric tons per day. In this example, the facility is surrounded by flat terrain and has a 30.5 meter tall stack. Other important parameters for the simulation include the following: (a) the stack diameter is assumed to be 1.52 meters; (b) the exhaust gas temperature is assumed to be 127 ^{o}C; and (c) the exhaust gas exit velocity is assumed to be 8.9 meters/sec. The distance from the source at which the maximum annual-average ground-level concentration was predicted to be approximately 900 meters. A distance of 1 kilometer (with virtually the same annual average concentration as at 900 meters) was chosen for the calculations shown in Figure 3.

[10]. This facility was recently closed down.

exposed to the same air concentrations. Thus, human exposure to PCDD/F will vary from person to person, depending on where they live and work, on what they eat and drink, and in general, on many aspects of their lives.

The following general picture of human exposure to PCDD/F emerges. First, consumption of milk, dairy products and beef are probably the most significant sources of exposure for the general population. The presence of PCDD/F in these foods arises as a result its incorporation into feed-crop vegetation, primarily from the air. Thus, air emissions sources are of crucial importance to the exposure of humans to PCDD/F.

A significant fraction of the PCDD/F emitted to the air from a given source will eventually be deposited back to the earth's surface — even though it may be dispersed over 100's or 1000's — and deposition to vegetation is a relatively significant pathway. A portion of the PCDD/F is so-deposited onto and incorporated into animal feed crops. Dairy cows, beef cattle, and other animals eat these contaminated feed crops, and PCDD/F bioaccumulates in their milk and/or fatty tissues. In this way, the properties of PCDD/F, its atmospheric fate and transport, and the characteristics of agricultural ecosystems combine to efficiently capture PCDD/F emitted to the air and transmit it to the human population.

Other exposure pathways can be important in certain situations, including: (a) consumption of fish contaminated by direct PCDD/F discharges and/or atmospheric deposition to surface waters; (b) inhalation in close proximity to very large air emissions sources; and (c) certain occupational exposures.

As discussed in detail in the Case Study on Persistent Organic Contaminants in the Canadian Arctic presented elsewhere in this Volume, the primary exposure pathway of dioxin-like compounds *in Arctic communities* is also through food consumption. In these ecosystems, aboriginal peoples are very dependent on wild foods of terrestrial (e.g., plants, caribou) or marine (fish, marine mammal) origin. The principal food contributing to dioxin-like toxic exposure appears to be marine mammals, and the principal compounds in marine mammals contributing to the dioxin-like toxic exposure of humans in Arctic ecosystems appear to be the dioxin-like PCB's, rather than PCDD/F's. The contamination of the arctic food chain by PCB's arises as a result of long range transport via the atmosphere and ocean currents.

5. Exposure and Health Effects

PCDD/F's are regarded as extremely toxic substances because their effects on living things may be evident at unusually low concentrations — ranging from parts per billion to parts per trillion. Human exposure to higher concentrations occurs only in accidents and industrial situations.[11] These are important sources of direct information about the effects of PCDD/F's on humans, but, as noted above, exposure of the general population is largely the result of the passage through the atmosphere of low, environmental concentrations through the agricultural food chain to humans.

Since the early 1970s, when PCDD/F's were first discovered as an environmental contaminant, and especially in the last decade, they have been the subject of an extraordinary number of scientific investigations. The most comprehensive evaluation of exposure to low environmental concentrations and the resultant health effects is a health assessment document for PCDD/F's and dioxin-like compounds, published in draft form by the U.S. EPA in September 1994 — and still in the process of editorial revision (U.S. EPA, 1994b). Other recent overviews of the toxicological effects of exposure to PCDD/F's include DeVito and Birnbaum (1994), Silbergeld and DeFur (1994), Tritscher *et al*. (1994), and Webster and Commoner (1994).[12]

While PCDD/F's are now some of the most intensely studied chemical pollutants, many uncertainties remain, and some results are subject to divergent interpretations. Nevertheless, especially as a result of the U.S. EPA comprehensive health assessment (U.S. EPA, 1994b), a useful set of generally accepted information has emerged. This may be summarized as follows:

5.1 The Level of Exposure of the General Population

This can be estimated in two ways:

- Since the main route of human exposure is food, largely milk, dairy products and beef, the daily intake can be estimated by measuring the PCDD/F content of a typical daily "market basket" of food. In the United States this amounts to 1-3 pg TEQ per kg of body

---

[11]. Couture et al (1990) have shown consistent effects of PCDD/F's on body weight, fetal mortality, cleft palate, and hydronephrosis in all animal models. In humans, well documented studies have shown high perinatal mortality (Hsu, 1985) and developmental toxicity in babies born to exposed mothers (Rogan 1989). Fetal death, growth retardation, structural malformations and organ system dysfunction has been shown in exposure incidents (Yamashita 1985, Rogan 1989).

[12]. Additional details on biological effects of PCDD/F's, particularly of 2,3,7,8-TCDD, are given in the Case Study on Persistent Organic Pollutants elsewhere in this Volume.

weight per day, or 3-6 pg TEQ/kg/day if dioxin-like PCBs are included. In Canada, an intake of 0.5 - 2 pg TEQ/kg/day from food consumption has been estimated (CEPA 1993). No comparable estimate has been assembled for dietary exposure to PCDD/F in Mexico.

- The daily exposure to PCDD/F results in its accumulation in lipid of tissues, blood and mothers' milk. A U.S. EPA study of the PCDD/F content of body fat in a representative sample of the general U.S. population in 1982 showed that this had reached a level of 7,000-9,000 pg/kg of body weight. Investigations of the relationship between PCDD/F exposure and the resulting content of tissue indicate that this body burden can be accounted for by a daily intake of approximately 2 pg TEQ/kg/day -- in good agreement with the "market basket" estimate of 1-3 pg TEQ/day (Commoner, Webster & Shapiro, 1986).

European data are generally comparable to the U.S. data, which appear to be typical of industrialized countries. Exposure levels of populations that eat relatively highly contaminated marine organisms (for example, Inuit living in the Canadian arctic) may be considerably greater than the U.S. average.

5.2 The Health Effects of Environmental Exposure

5.2.1 Cancer

Based on clear-cut evidence that exposure of laboratory animals to PCDD/F results in a dose-dependent increase in the incidence of cancer, the U.S. EPA (1994b) dioxin reassessment has classified PCDD/F as a "Class B probable human carcinogen" While accidental and industrial exposure to relatively high levels PCDD/F have resulted in a somewhat increased incidence of cancer in humans, the data were regarded as insufficient to justify a more certain level of classification. Nevertheless, a study of industrially exposed workers showed that a body burden as low as 109,000 pg/kg -- only an order of magnitude above the level in the general U.S. population -- led to a small but statistically significant increase in cancer incidence (DeVito *et al.*, 1995).

In U.S. EPA practice, regulation of PCDD/F emissions have been based on estimates of the cancer risk to exposed populations -- generally expressed as the risk per million of an increased incidence of cancer in a person exposed over a 70-year lifetime. A risk of one per million is generally regarded as "acceptable." The general U.S. population's daily intake of 2 pg TEQ/kg/day creates a cancer risk of 330 per million, well above the "acceptable" level (Commoner, Webster & Shapiro, 1986). If exposure to dioxin-like PCBs is included as well, the risk is 500-1000 per million, according to the U.S. EPA 1994 dioxin assessment (U.S. EPA, 1994b).

5.2.2 Effects on Reproduction and Development

Numerous experiments show that laboratory animals exposed to low (i.e., environmental) levels of PCDD/F's exhibit a number of reproductive and developmental abnormalities, generally due to disruptive effects on the relevant hormone systems. These abnormalities include reduced sperm count, reduced fertility, decreased birth weight, reduced levels of sex hormones, and birth defects[13] (e.g., cleft palate in mice). Similar effects have been observed in wildlife exposed to environmental levels of PCDD/F, for example defective egg development in fish-eating birds in the Great Lakes (U.S. EPA, 1994b).

Some of the effects observed in laboratory animals occur in humans as well. Thus, testosterone levels are reduced in rats exposed experimentally to PCDD/F, and in workers exposed in industrial situations as well (U.S. EPA, 1994b; DeVito *et al*., 1995). Similarly, maternal exposure to PCDD/F is associated with the decreased birth weight of offspring in both mice and humans (DeVito *et al*., 1995). Such effects generally occur at considerably lower levels of exposure than the levels that induce increased cancer incidence. Thus, while increased incidence of cancer among laboratory animals has been observed at exposure levels of 944-137,000 pg/kg body weight, non-cancer developmental effects occur at body burdens of 10-12,500 pg/kg -- more than an order of magnitude lower (U.S. EPA, 1994b; DeVito *et al*., 1995).

5.2.3 Other Non-Cancer Effects

The immune system appears to be particularly sensitive to the disruptive effects of PCDD/F's, leading, for example, to increased susceptibility to viral infections in mice. Nervous system defects may also occur; for example, low level exposure of pregnant monkeys to PCDD/F's results in defective learning behavior. Endometriosis, the proliferation of cells that line the uterus outside that organ occurs at an enhanced rate in monkeys exposed to low doses of PCDD/F's (DeVito *et al*., 1995).

In spite of all that has been learned about the range of non-cancer health consequences of exposure to PCDD/F and other dioxin-like substances, there are still many uncertainties.

---

[13]. Environmental, nutritional and genetic factors are related to birth defects (Cordero, 1994), and birth defects are being monitored through active and passive regional surveillance systems (Santos-Burgoa *et al*., 1996) in areas with high levels of pollution. Causal relationships have not yet been developed, but, this research may lead result in an increased understanding of the toxic effects of PCDD/F and other pollutants.

6. Continental Pollutant Pathways

There are many pathways through which human exposure to PCDD/F's can result from transport of material over continental distances.

6.1 Long Range Atmospheric Transport of PCDD/F's

The ability of different PCDD/F congeners to be transported long distances in the atmosphere depends on their atmospheric "lifetimes." Many different phenomena influence the fate of PCDD/F congeners in the atmosphere. The primary mechanisms which limit the atmospheric lifetime (and hence transport) of PCDD/F's are believed to be:

- chemical transformation, including photolytic destruction by ultraviolet light and chemical reaction (e.g., with hydroxyl radical); and

- fallout (also called "deposition") as a result of precipitation ("wet deposition") and as a result of the impaction of particles and the adsorption/absorption of gases at the earth's surface ("dry deposition").

6.1.1 Vapor/Particle Partitioning

The fate of "semivolatile" pollutants (such as PCDD/F's) at any given location in the atmosphere depends intimately on whether the pollutant is in the vapor or particle phase (Junge, 1978; Bidleman, 1988). The extent of vapor/particle partitioning depends intimately on the physical-chemical properties of the pollutant, the ambient temperature, and the detailed nature of the local atmospheric aerosol.

Because of particularly low volatility, PCDD/F congeners with six, seven, and eight chlorines tend to exist primarily in the particle phase in the atmosphere. Those with four and five chlorines — including some of the most toxic congeners — are believed to exist in significant proportions in both the vapor and particle phase in the atmosphere, based on both theoretical considerations and experimental evidence.

These tetra-chloro (TCDD/F) and penta-chloro (PeCDD/F) congeners may move relatively freely between the two phases, depending on the local atmospheric conditions (i.e., they may be "exchangeable.") There is some experimental evidence which suggests that atmospheric PCDD/F is exchangeable. Eitzer and Hites (1989a and 1989c) measured the amounts of vapor-phase and particulate-phase PCDD/F in the atmosphere of Bloomington, Indiana and found that while there was no temperature-related effect on the total concentration of PCDD/F in the atmosphere, the proportions in the two phases were dependent on the ambient temperature at the time of the measurement. Furthermore, in agreement with theory, it was found

that the vapor/particle partitioning of each of the congeners was, in general, critically dependent on each congener's subcooled liquid vapor pressure (Eitzer and Hites, 1989a).

6.1.2 Chemical Transformation in the Atmosphere

When a PCDD/F molecule is in the particle phase, it is believed to be much less vulnerable to photolytic destruction by ultraviolet light (Koester and Hites, 1992a) and chemical reactions in the atmosphere than when it is in the vapor phase. In the vapor phase, the rates of destruction by ultraviolet light and chemical reaction depend, or course, on the detailed characteristics of the atmospheric micro-environment at any given location, and, will be different for different congeners at the same location. It is not possible to predict the rate of photolytic destruction or chemical reaction of a vapor-phase (or particle-phase) PCDD/F congener at any given location in the atmosphere with great accuracy at the present time, *even* if details about the concentrations of reactants and electromagnetic spectrum were known at that location. Rough approximations can be made, however. Atkinson and coworkers (Atkinson, 1991; Kwok *et al*., 1994; Kwok *et al*., 1995) have considered available theoretical and experimental evidence and have concluded the following:

- Reactions of gas-phase PCDD/F's with ozone (O_3), nitrate radical (NO_3), or the HO_2 radical are not expected to be important atmospheric loss mechanisms;
- Reaction of gas-phase PCDD/F's with hydroxyl radical (OH) is an important atmospheric loss mechanism, with reaction rates varying among different congeners;
- Photolysis:
  - *may* be somewhat important for vapor-phase tetrachloro-dibenzo-p-dioxins (TCDD's), although less important than reaction with hydroxyl radical;
  - is probably not important for pentachloro-dibenzo-p-dioxins (PeCDD's);
  - because OH radical reactions are predicted to be slower with PCDF's than with PCDD's, photolysis may be relatively more important for PCDF's.

Building upon the work of Atkinson and coworkers (e.g., Kwok and Atkinson, 1995 and references cited therein), Meylan and Howard (1993; 1996ab) have developed an improved estimation methodology for the prediction of hydroxyl radical reaction rates with gas-phase PCDD/F's and other compounds. Using reaction rates estimated with their methodology, together with an estimated average hydroxyl radical concentration of 1.5×10^6 molecules per cm^3, atmospheric vapor-phase half-lives (relative only to hydroxyl radical attack) for 2,3,7,8-TCDD,

2,3,7,8-TCDF, 1,2,3,7,8-PeCDD, 1,2,3,7,8-PeCDF, and 2,3,4,7,8-PeCDF are estimated to be 5.3, 65.0, 6.2, 143.5, and 143.5 days, respectively.

Given a lack of data, it is difficult to make quantitative estimates of the half-lives of vapor-phase PCDD/F molecules with respect to photolysis (i.e., destruction by ultraviolet light). Based on experimental measurements, Dulin *et al*. (1986) estimated the half-life of 2,3,7,8-TCDD *in water* to be on the order of 4-5 days in the summer at a 40^{o} latitude. Extrapolation of solution-phase photolysis rates to the vapor phase is not straightforward, as the processes occurring in solution can be more complex than the "simple" photolytic phenomena in the vapor phase. Nevertheless, as assumed by Atkinson (1991) the vapor-phase half-life of 2,3,7,8-TCDD relative to photolysis *may* be similar to that in the aqueous phase; if so, the rate of photolytic destruction of vapor-phase 2,3,7,8-TCDD would be of the same order as the destruction due to reaction with hydroxyl radical.

Photolysis rates for different PCDD/F congeners appear to decrease, generally, with increasing numbers of chlorines (e.g., Choudry and Webster, 1989; Sivils *et al*., 1994), and so, photolysis of PeCDD/F congeners (those with five chlorines) would be expected to be slower than TCDD/F congeners (with four chlorines). In solutions, it has been found that OCDF is more vulnerable to photolytic destruction than OCDD (Wagenaar *et al*, 1995). If this relative vulnerability is true for other dibenzofurans, the vapor-phase photolysis of 2,3,7,8-TCDF may be more rapid than that of 2,3,7,8-TCDD, and the rate for PeCDF's may be more rapid than that for PeCDD's.

A large area of uncertainty concerns the products of the atmospheric degradation of PCDD/F's. If the atmospheric degradation of a polychlorinated PCDD/F molecule results in the same compound with one less chlorine, then the toxicity can be increased (as can be seen in Table 1 at the beginning of this case study, toxicities of PCDD/F compounds generally increase as the number of chlorines decrease). Degradative dechlorination has been observed in the environment (e.g., Miller *et al*., 1989).

It has been observed that while the emissions of PCDD/F's are relatively equally distributed among the different homologue groups, the atmospheric concentrations and deposition tend to be heavily skewed towards the higher chlorinated congeners. This suggests that the dechlorination of higher-chlorinated congeners leading to the more toxic tetra- and penta-chlorinated congeners is not, ultimately, of major importance.

Perhaps the reason for this is that the rate of destruction increases as the number of chlorines falls, so, once the degradation starts, there is a cascade of dechlorination events that proceeds relatively quickly. In other words, once the conditions in the local atmosphere are "severe" enough to degrade a higher-chlorinated congener to a lower-chlorinated congener, they are more than sufficient to degrade the resulting lower-chlorinated congener. Thus, in these circumstances, the potentially more toxic intermediates may have very short lifetimes.

6.1.3 Atmospheric Deposition of PCDD/F's

The discussion regarding atmospheric deposition of PCDD/F's will be divided into vapor-phase processes and particle phase processes.

For vapor-phase PCDD/F's, dry deposition at the earth's surface is a relatively important loss process, but, this process does not deplete the total atmospheric burden of PCDD/F very rapidly. The atmospheric lifetime relative to vapor-phase dry deposition may be on the order of approximately 1 week. Due to very limited water solubility, wet deposition of *vapor phase* PCDD/F's is not expected to be an important fate pathway.

Thus, based on the chemical transformation mechanisms discussed above, even if they existed completely in the vapor phase (which they don't) the tetra- and penta-chloro dibenzo-p-dioxin and dibenzofuran congeners are expected to have atmospheric lifetimes of 1-2 days or longer.

Portions of the tetra- and penta- congeners and essentially all of the hexa-, hepta- and octa-chloro congeners are associated with particles in the atmosphere, and fate processes affecting particulate pollutants will govern their atmospheric lifetime.

Particles in the atmosphere are generally classified into three separate size ranges: the "nucleation range", comprising particles with diameters less than approximately 0.05 μm (1 μm = $1x10^{-6}$ meters); the "accumulation range", comprising particles with diameters roughly between 0.05 μm and about 2 μm; and the "coarse particle range", consisting of particles with diameters greater than about 2 μm in diameter (e.g., Lodge, 1981; Prospero *et al*., 1983; Seinfeld, 1986; Pacyna, 1995). Particles in the two smallest ranges are sometimes grouped together in a "fine particle" category.

Particles in the nucleation range are subject to coagulation and incorporation into larger particles; thus, the material contained in them is generally transferred to larger particles, generally in the accumulation range. The time scale for such processes is on the order of hours (Pacyna, 1995). Particles greater than about 10 μm -- generated primarily by mechanical actions and wind erosion -- have relatively fast sedimentation rates, and can be removed relatively quickly by dry deposition from the atmosphere. Typical residence times for such large particles are on the order of a few hours. Particles in the accumulation range, on the order of 1 μm in size, do not efficiently coagulate into larger particles, and do not have large sedimentation velocities. They can be removed by wet and dry deposition processes, but, have atmospheric lifetimes on the order of 7 - 10 days (Pacyna, 1995; Prospero, 1983). Since the fate of nucleation range particles is often to end up quickly incorporated into the accumulation range, the atmospheric lifetime of the *pollutants* associated with these small particles is also on the order of 7-10 days. Manchester-Neesvig and Andren (1989) used an average residence time for atmospheric particles in the Northern Hemisphere of 6 days in their estimation of the particle-mediated atmospheric lifetime of particle-bound PCB's.

Windblown soil particles are often relatively large, and fall into the coarse particle range. However, a fraction of such aerosols are fine particles (e.g., Pacyna, 1995).

Particles emitted from combustion and incineration processes will generally fall into the fine-particle category. Vapor/particle partitioning is often considered a surface phenomenon, and partitioning to particles is often assumed to scale with particle surface area. That is, when vapor-phase material condenses onto particles, it is considered to do so relatively equally onto each available surface, on a per-area basis. The most significant portion of the surface area available for adsorption is with the fine particles. Thus, for exchangeable, semivolatile compounds that partition between the particle and vapor phases, a significant fraction of their particle-associated mass will lie in the fine-particle range.

Measurements of the size of particles to which semivolatile compounds in the atmosphere are absorbed are difficult, and many factors can bias the results (e.g., Poster *et al*., 1995). For PCDD/F's, such a measurement was attempted by Kaupp *et al*. (1994). In this study, three samples were collected in a rural area (at the University of Bayreuth, Germany), during periods with mean ambient temperatures from 17.4 - 21.9 ^{o}C. PCDD/F's were found in both the vapor- and particle phase. Approximately 90% of particle-phase PCDD/F's were associated with particles less than 1.35 µm in diameter; approximately 50 - 60% of the particle-phase PCDD/F's were associated with particles less than 0.45 µm in diameter. It was found that smaller particles tended to have higher concentrations of PCDD/F's than bigger particles, consistent with a surface-area-related partitioning phenomena.

Thus, to the extent that they are associated with particles, PCDD/F's are expected — on the basis of theoretical and experimental considerations — to exist predominantly in the fine particle range, with sizes less than approximately 2 µm.

The atmospheric lifetime of pollution associated with such particles will be highly variable depending on meteorological conditions, source characteristics, and particle characteristics, but, average atmospheric lifetimes of such particle-associated pollution will be on the order of 1 week (or more). Depending on the meteorological conditions, such particles could travel hundreds to thousands of kilometers in the atmosphere before they are removed by wet or dry deposition processes.

6.1.4 Summary: Atmospheric Lifetime of PCDD/F's

Based on the foregoing considerations, higher chlorinated PCDD/F congeners (i.e., those with 6, 7 and 8 chlorines) are expected to exist largely associated with small particles in the atmosphere, and will have atmospheric lifetimes of roughly one week. Significant fractions of the more toxic tetra- and penta-chloro dioxin congeners are expected to exist in both the vapor and particle phases in the atmosphere. Their atmospheric lifetimes are expected to be on the

order of a few days to a week. Over such periods, PCDD/F's can be transported in the atmosphere over regional and continental scales.

6.1.5 Modeling the Long-Range Atmospheric Transport of PCDD/F's

In an air pollution model, an attempt is made to mathematically express and combine all the relevant factors to quantitatively estimate the fate and transport of atmospheric pollutants. One useful type of information that can be obtained from modeling is information about source-receptor relationships; i.e., estimates of the relative contribution of specific sources or source regions to the concentrations or deposition at a given receptor. If an eventual policy goal is to lower the impact at the receptor, then the sources contributing to that impact must first be characterized.

There have been few attempts to model the regional and long-range transport of PCDD/F's in the atmosphere. These include the following:

- Rappe *et al*. (1989) and Tysklind *et al*. (1993) compared measured atmospheric concentrations of PCDD/F at locations in Sweden to modeled back-trajectories of the air masses sampled, and found evidence that long-range atmospheric transport of these compounds was occurring.

- Van Jaarsveld and Schutter (1993) modeled regional and long-range transport and deposition of PCDD/F's in portions of Europe by adapting a Lagrangian model developed originally for analysis of acidic deposition. Specific source-receptor relationships were not reported.

- In a current study, the short-, medium-, and long-range transport of PCDD/F's from specific sources and source regions to dairy farms in Wisconsin and Vermont is being modeled. Measurements of PCDD/F's in the air above the farms, in the crops, and in the milk are also being made and will be used to test the validity of the model.[14]

- Currently, the U.S. EPA is attempting to simulate the long-range transport of PCDD/F's using the RELMAP air pollution model (Cleverly, 1996).

---

[14]. This ongoing work is a collaborative project with the principal involvement of the following people: *Mark Cohen, Barry Commoner,* and others at the Center for the Biology of Natural Systems at Queens College; *Jean Richardson* of the New England Environmental Policy Center and the University of Vermont; *Sarah Flack* of Farm Management Consulting in Vermont; *Randy Shaver, Janet Reisterer* and others at the University of Wisconsin-Madison Department of Dairy Science; *Bruce Maisel* of ENSR Consulting; *Mark Horrigan, John Stanley* and others at the Midwest Research Institute.

! In a study of the sources of PCDD/F's to the Great Lakes (Cohen *et al*. 1995), the regional and long-range atmospheric transport of PCDD/F's from air emissions sources in the United States and Canada was analyzed using a model developed by the U.S. National Oceanic and Atmospheric Administration (Draxler 1991, 1992, 1994). A few details of the results of this study will be described below.

In the Great Lakes study, algorithms were added to the NOAA model (HYSPLIT) to simulate the atmospheric vapor/particle partitioning behavior of PCDD/F's. Relatively good agreement was found between predicted and measured concentrations of atmospheric PCDD/F at Dorset, Ontario.

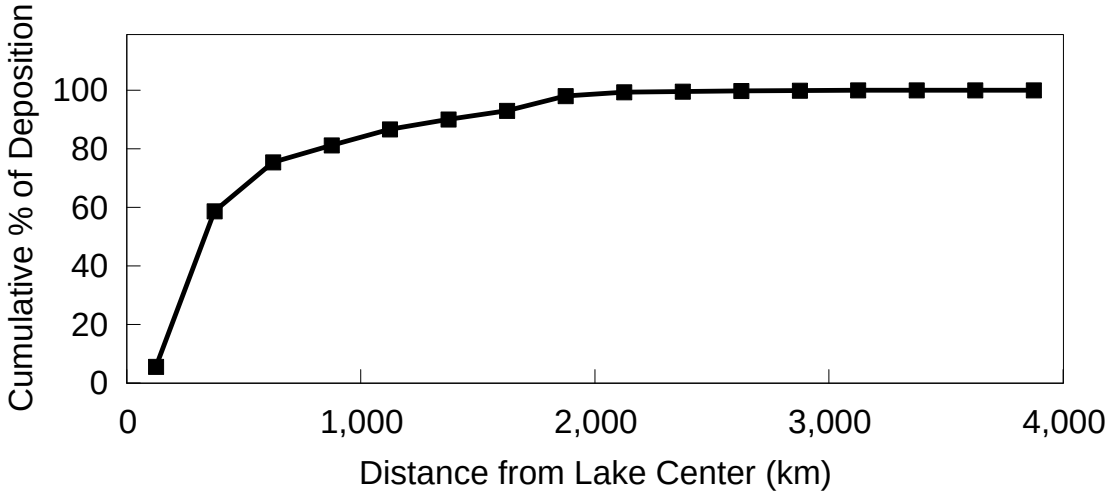
An attempt to characterize source-receptor relationships was made in this analysis. Approximately half of the predicted atmospheric deposition of PCDD/F's (on a TEQ basis) was linked to sources in states and provinces immediately adjoining the Great Lakes; the other half was associated with sources in the rest of the U.S. and Canada. Sources in Mexico and the rest of the world were not considered; if they had been included, it is expected that they would have made a lesser but perhaps not insignificant contribution to the total predicted loadings.

In Figure 4, an example of the cumulative relationship between the sources' distance from a particular Lake (in this case Lake Michigan) and the relative PCDD/F contribution (on a total TEQ basis) is shown. For this lake (similar to other Lakes), it was found that about half of the cumulative PCDD/F deposition comes from sources about 300 miles (480 kilometers) or less from the center of the Lake — that is, located in the U.S. states and the province of Ontario that border the Lake. The remaining half of the total deposition comes from sources as far as 1,500 miles (2,400 kilometers) away.

The extent to which the different sources contribute to the PCDD/F deposited in the Great Lakes was found to depend not only on the amounts they emit and their distances from the lakes, but also on their geographic location. A higher percentage of the PCDD/F emitted from sources to the south and west of the lakes is deposited in them than from the sources to the north and east. Figure 5 illustrates this effect in the case of Lake Michigan, again, on a total TEQ basis.

Another illustration of the effect of the weather pattern in influencing the efficiency with which a given source contributes PCDD/F to a given receptor is given in Figure 6, again with Lake Michigan as the example. For this purpose the entire U.S. and Southern Canada area was divided into 20,000 squares (each 270 square miles in area). Using the air pollution model, the percent of individual PCDD/F congeners emitted from each square's center point that would be transported through the atmosphere and deposited in Lake Michigan was estimated (i.e., the "air transfer coefficient" was estimated for a series of potential source locations to a given receptor, Lake Michigan). In Figure 6, the results for a typically emitted PCDD/F mixture are shown, summarized on a TEQ basis. The figure maps the geographic distribution of eight successive ranges of air transfer coefficient. It shows that PCDD/F transport and eventual deposition to Lake Michigan is most efficient for sources to the west and southwest of the Lake, and least

Figure 4. The Effect of Distance of Sources on Cumulative Deposition of Dioxin in Lake Michigan



(Data plotted are PCDD/F Toxic Equivalents (TEQ) estimated to be deposited from sources in 1993)

Figure 5. The Relation Between the Directional Orientation of Dioxin Sources and their Emissions and Deposition in Lake Michigan

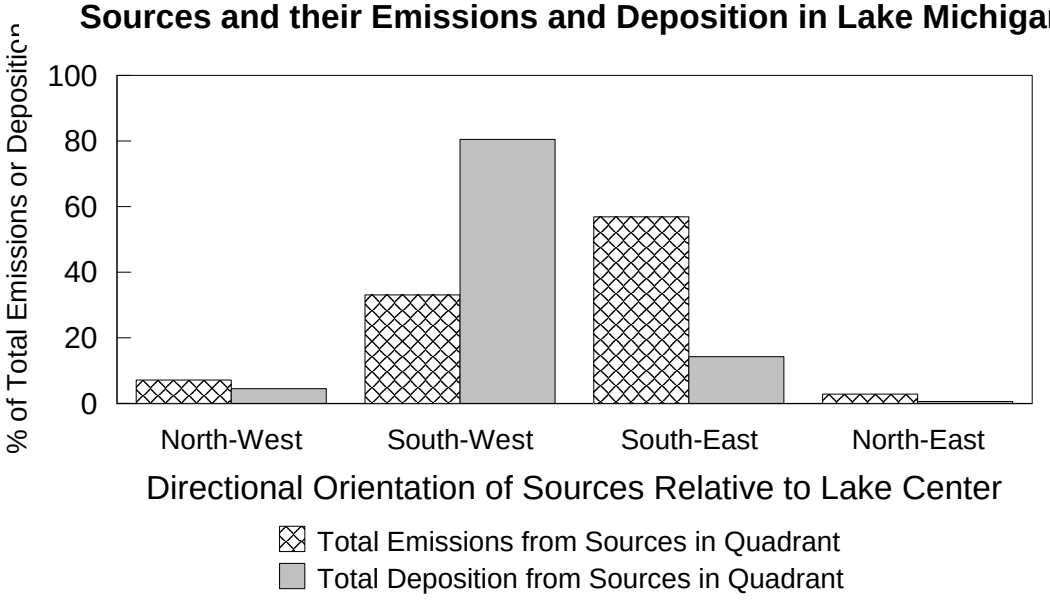
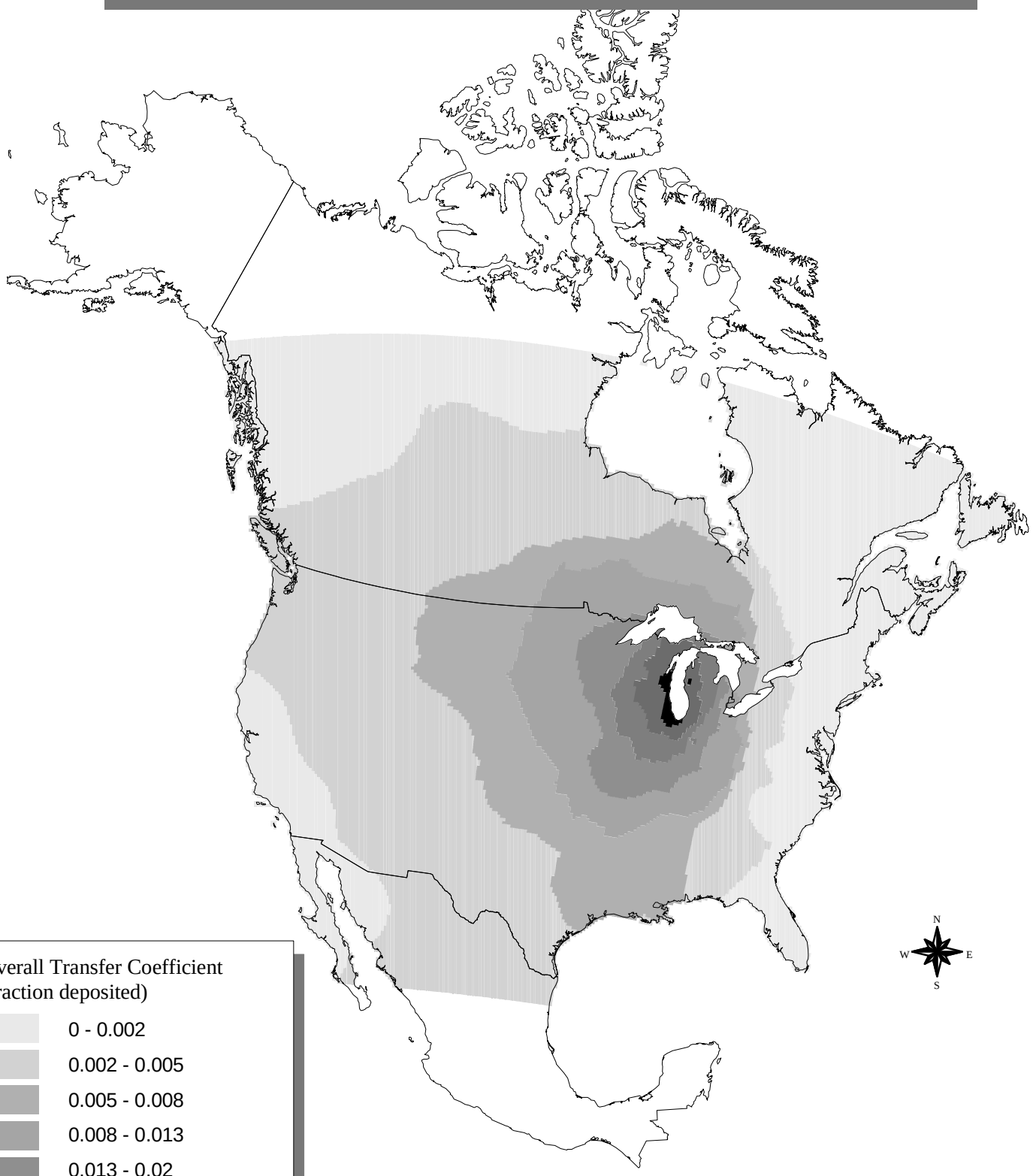


Figure 6: Fraction of 1996 Dioxin Emissions Deposited in Lake Michigan
(grams TEQ deposited per year/grams TEQ emitted per year)



Overall Transfer Coefficient
(fraction deposited)

- 0 - 0.002
- 0.002 - 0.005
- 0.005 - 0.008
- 0.008 - 0.013
- 0.013 - 0.02
- 0.02 - 0.03
- 0.03 - 0.055
- 0.055 - 0.1
- outside of modeling domain

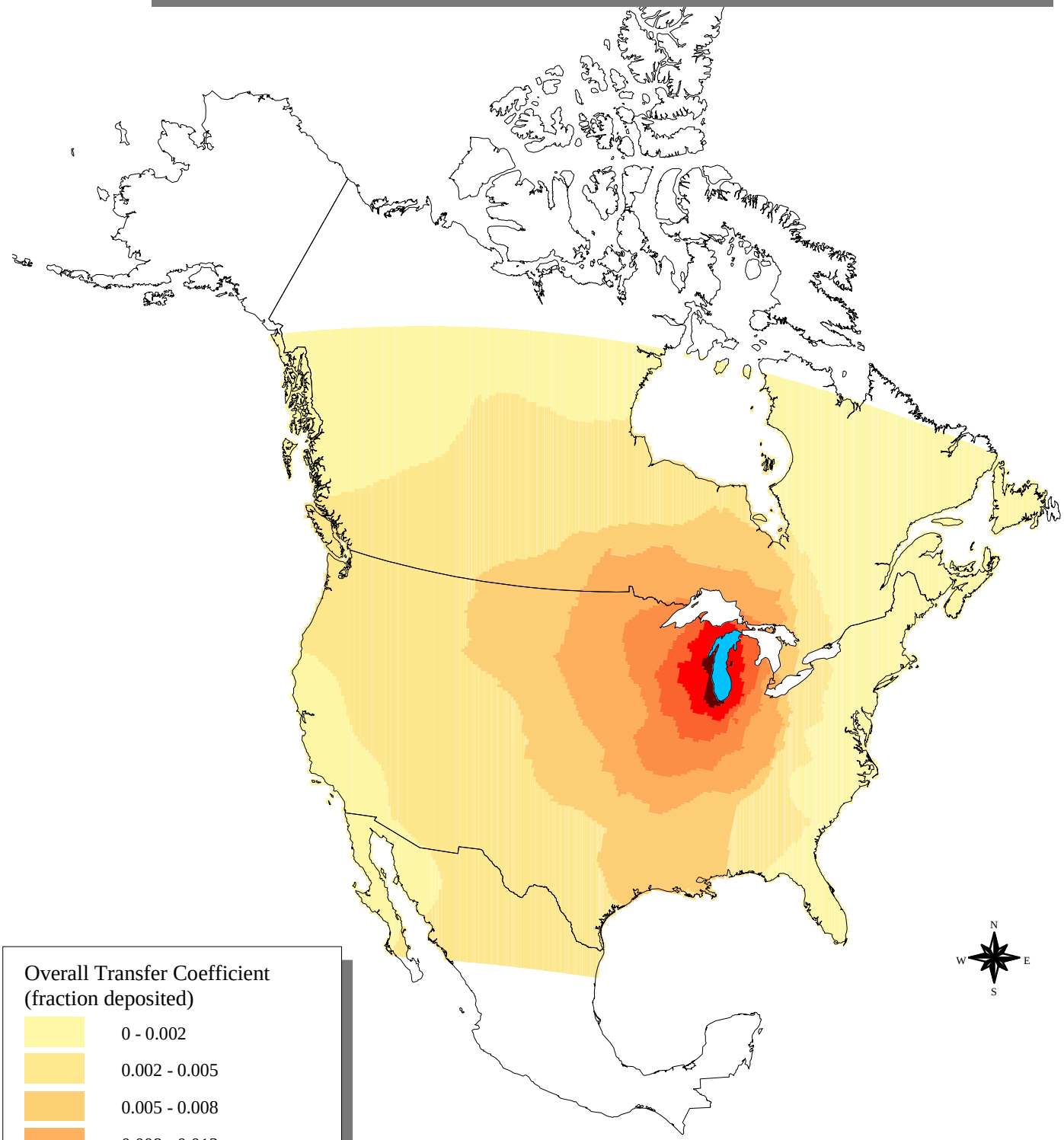
300 0 300 600 Kilometers

300 0 300 600 Miles

Projection: Lambert Conformal Conic

These estimates- also called Transfer Coefficients- were developed using NOAA's HYSPLIT atmospheric fate and transport model.

Figure 6: Fraction of 1996 Dioxin Emissions Deposited in Lake Michigan (grams TEQ deposited per year/grams TEQ emitted per year)



Projection: Lambert Conformal Conic

These estimates- also called Transfer Coefficients- were developed using NOAA's HYSPLIT atmospheric fate and transport model.

efficient for sources to the northeast and southeast of the lake. This finding is reflective of the general southwest-to-northeast and west-to-east prevailing weather patterns in the region.

6.2 Ocean Transport Pathways for PCDD/F's

There have been relatively few measurements of PCDD/F levels in ocean water, sediments, or biota, and most of the measurements are for near-coastal waters and estuaries (e.g., Hagen *et al*., 1995; Rappe *et al*., 1990b; Hashimoto *et al*., 1995; Jarman *et al*., 1996). Most measurements of persistent toxic compounds in marine ecosystems have involved PCB's and pesticides (e.g., Iwata *et al*., 1993; Tatsukawa, 1992).

Broman et al. (1991) reported detailed measurements of PCDD/F's in Baltic Sea waters. They detected 2,3,7,8-TCDD at 4 or 8 sites at an average concentration of 0.22 pg/m^3. Other 2,3,7,8-substituted congeners were also detected at one or more sites. TCDD (as a homologue group of all tetrachlorodibenzo-p-dioxin congeners) was detected at all sites, with an average concentration of 7 pg/m^3; other PCDD/F homologue groups were also detected at most or all of the sites. The recent measurements of Matsumura and coworkers (1995) in the western North Pacific may be the first reported measurements of PCDD/F's in open ocean seawater. At the one site for which sample data was given, the homologue groups TCDD, PeCDD, HxCDD, HpCDD and OCDD were detected at levels of 40, 60, 210, 350, and 390 pg/m^3, respectively.

PCDD/F's have been found in ocean-fish-eating animals, including polar bears and ringed seals in the arctic (Muir *et al*., 1992b; Norstrom and Muir, 1994) and albatross at Midway Atoll, stated to have an extensive feeding range covering much of the subtropical and northern Pacific ocean (Jones *et al*., 1996).

The ocean may play a role in transporting PCDD/F's over continental distances. First, there are direct discharges of PCDD/F's from industrial sources to rivers leading to an ocean (tributary rivers) or directly to an ocean. PCDD/F contamination in the ocean can be dispersed and transported by currents over large distances. In a similar manner, PCDD/F's discharged by sewage treatment plants to tributary rivers or oceans — as a result of discharges to the facility or possibly even as a result of the formation of PCDD/F's in the wastewater chlorination process — may be transported over long distances in the oceans. Finally, there are many pathways by which air emissions may find their way into tributary rivers or oceans. A portion of the PCDD/F emitted to the air is deposited directly onto tributary rivers or oceans; a portion of the PCDD/F emitted to the air is deposited by precipitation which finds its way to tributary rivers or oceans; and finally, a portion of the PCDD/F emitted to the air is deposited to the earth's surface even without precipitation ("dry deposition") but can be delivered to tributary rivers or oceans by runoff or wind.

Unfortunately, little is known about the relative importance of different PCDD/F sources to ocean contamination, although it is likely that both atmospheric deposition and coastal water

effluent discharges are important. Moreover, little is known about the fate and transport of PCDD/F's in ocean ecosystems. Thus, it is difficult to assess the overall significance of ocean transport of PCDD/F's *over continental distances*.

PCDD/F's in marine water are expected to partition significantly to suspended sediment, similar to its expected behavior in fresh water, although there are few data on this phenomenon available. Based on measurements and theoretical considerations, Broman and coworkers (1991) estimated that approximately 25% of the PCDD/F in the surface layers of the Baltic Sea was truly dissolved, and 75% was associated with suspended particles. Thus, sedimentation may be a dominant fate process for PCDD/F's emitted from coastal liquid effluent discharges. Thus, for PCDD/F in coastal liquid effluent discharges (i.e., from sewage treatment plants), sedimentation may be a dominant fate process and may limit the long-range transport in the ocean.

Fish feeding in a coastal area contaminated with PCDD/F can become contaminated with PCDD/F. If some of these fish then migrate to areas 100's or 1000's of kilometers away, then this would constitute a potential pathway for PCDD/F transport over continental distances. Salmon, shad, sturgeon, orange roughy, mackerel, and tuna are examples of fish widely consumed by humans that migrate long distances in the ocean.

The principal human health consequence of contamination of the oceans by PCDD/F and the possible widespread dispersal of such contamination would probably be increased dietary exposure to PCDD/F's through consumption of fish (and possibly sea vegetables).

6.3 The Potential Impact of Trade and Commerce

Discussion of the continental transport of PCDD/F reflects concerns that an action taken in one place -- for example, operation of a trash-burning incinerator -- may result in the increased exposure of the population of another region or country. This issue can arise through processes other than the actual transport of airborne or waterborne PCDD/F from one place to another. It may arise as well through trade in industrial facilities that emit PCDD/F's; through commerce in PCDD/F-contaminated food products; and even as a result of commerce in commodities, such as organochlorine hazardous waste, chlorinated plastics, or leaded gasoline, which, when burned generate PCDD/F emissions.

The following example illustrates the potential effect of international trade in industrial facilities on PCDD/F exposure. As a result of citizen pressure, the concerns of the medical community and the initiatives of regulatory agencies, PCDD/F emissions from medical waste incinerators have been under increasing scrutiny in the United States. In the last few years increasing numbers of existing incinerators have been shut down and investment in new ones has decreased. One result may be increased export of incinerators from the United States to Mexico. If 100 such newly imported incinerators were to operate there, they would emit roughly 10-100 g

TEQ of PCDD/F per year[15], contributing significantly to the exposure of the Mexican population[16]. On the other hand, if the transfer of medical waste disposal technology between the United States and Mexico were based on autoclaves rather than incinerators, the trade could have a beneficial effect on the Mexican environment.

As indicated above, ingestion of dairy products (such as cheese) and beef represent important paths of exposure to PCDD/F. Such products (in contrast with milk) are readily transported as items of national[17] and international trade, efficiently exposing the population of one region to PCDD/F generated in another region. Thus, in North America, the exposure of the respective populations of Mexico, the United States and Canada to some degree will reflect the balance of trade in such foods among the three countries[18], as well as their relative levels of PCDD/F emissions. The typical person's exposure to PCDD/F's may depend more on the level of contamination at a dairy farm 100's or 1000's of kilometers away (producing milk for cheese) than on any local source of PCDD/F's near where they live or work.

---

[15]. A typical medical waste incinerator's PCDD/F emission rate is on the order of 0.1 to 1 gram TEQ/year.

[16]. How large is this transferred emission amount relative to the long-range transport of PCDD/F through the environment (e.g., the atmosphere)? In order to answer this question, a detailed modeling study, for example, of the air transport of PCDD/F between sources in the U.S. and receptors in Mexico would need to be carried out. Such a study has not yet been done. A very rough, qualitative comparison can be made, however, in the following way. It has been estimated that the total air emissions of PCDD/F in the United States is on the order of 5,000 - 10,000 g TEQ/year (with very large uncertainties). Of these emissions, it was estimated that on the order of 40 g TEQ/year (~ 1% of the emissions) were transported to and deposited in the Great Lakes (Cohen *et al*. 1995). Given the general nature of prevailing weather patterns and the geographical distribution of PCDD/F sources, it is *not* likely that the total transport and deposition of PCDD/F through the air *from the U.S. to Mexico* would be much higher than this, and, may in fact be much lower than this. This amount is on the same order of magnitude as the amount emitted by the exported sources in this illustration.

[17]. It has been estimated that in the United States, the average food product travels on the order of 1000 - 1500 km between the site of original production and eventual human consumption.

[18]. U.S. Exports to Mexico during fiscal year 1996 were reported to include:
(a) 4,675 metric tons of cheese ($13.4 million); $120 million, total, of dairy products;
(b) 81,000 live cattle & calves ($42 million); 47,000 tonnes of beef & veal ($130 million)
Exports from Mexico to the U.S. for fiscal year 1996 were reported to include:
(a) $18 million, total, of dairy products; (b) 600,000 live cattle and calves ($180 million).
[Source of data: Foreign Agric. Trade of the U.S., Economic Research Service, USDA]

Finally, trade in materials that can serve as precursors to the generation of PCDD/F's may also carry with it an effect on the exposure of the population of the importing country. For example, in the United States the disposal of hazardous waste, which if burned emits significant amount of PCDD/F to the air, has become an important item of commerce. One result is that hazardous waste produced in the U.S. has become a major fuel in the cement industry, and, if exported to the Mexican or Canadian cement industry, would to some degree become a component in these countries' environmental PCDD/F levels. A similar issue arises when disposable polyvinyl chloride products -- major items of commerce -- produced in one country and exported another country are eventually discarded and burned in local incinerators, contributing to environmental levels of PCDD/F.

However complex and subject to debate, such processes should be considered in efforts to remedy the impact of PCDD/F's on health and environmental quality.

7. Policy Implications

7.1 The Need for Remedial Action

In U.S. EPA regulatory procedures, action to remedy the effects of an environmental pollutant is generally based on the establishment of standards that govern the emissions of the relevant classes of sources. (In the case of PCDD/F's, incinerators for example.) At present, PCDD/F standards are based on an assessment of the risk of cancer. A lifetime (70 year) risk of one in a million has been generally regarded as "acceptable" in siting individual facilities, although risk levels 10 times greater have sometimes been allowed.

As noted above, data on the PCDD/F content of food intake and measurements of the PCDD/F body burden lead to the conclusion that the general U.S. population is exposed to a cancer risk of several hundred per million, or -- if the effect of dioxin-like PCBs is included -- 500-1000 per million (U.S. EPA 1994b). It seems reasonable to conclude, therefore, that action to sharply reduce the exposure to PCDD/F's and other dioxin-like substances is mandated by the observed levels. As noted earlier, non-cancer effects due to exposure to PCDD/F's are likely to occur at levels at least an order of magnitude below those that give rise to the increased risk of cancer, intensifying the need for remedial action.

The Canadian government's guideline for human exposure is 10 pg TEQ per kg of body weight per day. This guideline is several times greater than the general level of exposure, ~1-2 pg TEQ/kg/day, which, as noted above, applies as well to the U.S. population. Accordingly, current Canadian regulations do not call for remedial action at this general level of exposure to protect human health. However, since air transport of PCDD/F from U.S. sources may be responsible for a large part of the exposure of the Canadian population (based on the analysis of Cohen *et al*., 1995), remedial action in the U.S., taken in response to U.S. standards, would probably reduce exposure in Canada as well as the United States.

The foregoing considerations have important implications for environmental policy. When, for example — as it is now — the decision to operate a waste-burning incinerator is based on the direct health-hazard impact experienced only in the nearby area, the public policy issues come down to a straightforward question: Should the community build the incinerator that would expose the people of the community, themselves, to this hazard? Since, in this case, the risk would be self-imposed, the community at risk, through its elected officials or a referendum, could decide whether or not to accept it.

The situation regarding the general population's exposure to PCDD/F is not consistent with the above decision-making methodology. Because of the dominance of air transport in exposure to PCDD/F's and their ability to be transported long distances in the atmosphere, exposure as a result of pollutant transport over regional and continental distances is strongly suspected to be a significant problem. That is, the PCDD/F emitted by any one incinerator (or other source) is combined with that from many other sources, and their collective impact is visited upon people everywhere — whether or not they chose to build an incinerator — through food produced on distant farms. Given the prevailing weather patterns, for example, it is likely that PCDD/F emitted to the air by U.S. sources makes a significant contribution to the PCDD/F content of milk produced on Canadian dairy farms. The same may be true of the impact of Mexican sources on dairy farms in the southwest region of the United States.[19]

Thus, the policy debate must be greatly broadened. While the people in any given community would of course benefit by persuading officials to shut down the local incinerator in favor of alternatives such as recycling, the same remedial action would have to be taken on a much wider scale to significantly reduce the given communities exposure to PCDD/F. Since the impact of any one incinerator is part of the collective impact of many of them, action to reduce the hazard must be collective as well. In sum, policy decisions about PCDD/F sources should not be considered to be strictly local; regional, national, and international inputs into the decisions are necessary.

---

[19]. As an aid to national and international policy formulation, these and other potential source-receptor-exposure relationships need to be accurately characterized. A model-based analysis of the atmospheric transport and deposition of PCDD/F's emitted by sources in North America, with particular attention to the contamination of agricultural food chains, would be of great use in elucidating the relative importance of PCDD/F transport over local, regional and continental scales in leading to human exposure. Such an analysis would be greatly assisted if a sufficient number of environmental measurements of PCDD/F's were made to allow the model's validity to be tested. As noted above, information on emissions sources is relatively limited; the analysis could be greatly improved if accurate, geographically and temporally resolved, congener-specific PCDD/F emissions inventories in North America could be assembled.

7.2 Opportunities for Remedial Action

At present, the regulatory measures that are applicable to sources that generate PCDD/F emissions — and which therefore contribute to long-range continental air transport — are based on the strategy of pollution *control*; to meet regulatory standards, a device capable of sufficiently recapturing or destroying the PCDD/F before it is emitted is attached to the source. Thus, in its latest documentation of PCDD/F emission regulations (for hazardous waste incinerators: U.S. EPA, 1996b), U.S. EPA requires the installation of a control device that represents the "Maximum Achievable Control Technology." This strategy has been practiced, at least in the major source of airborne PCDD/F — municipal waste incinerators — and the industry has sharply improved the efficiency of their control systems in recent years. The most stringent PCDD/F emission standard for municipal waste incinerators, 0.1 ng/m^3, is that recently enacted in Germany. Very extensive and costly controls are required to meet such a limit.

An alternative approach to remedial action is based on the principle of pollution *prevention* rather than control. It has been promulgated most notably by the International Joint Commission, which has called for "zero emission" or "virtual elimination" of PCDD/F's and many other persistent toxic compounds in the Great Lakes. Unlike the control strategy, the goal of the prevention approach is to change the technology of the production process in which the pollutant originates, so that the source no longer produces it. This strategy is designed to break the link, inherent in the technology of the facility, between the production of a "good" and the production of PCDD/F. Thus, a municipal waste incinerator produces a good in the form of trash disposal (and in some cases, electric power) and, at the same time, generates PCDD/F's. The aim of pollution prevention is to find a way to produce the good that does <u>not</u> produce PCDD/F's.

For fundamental, thermodynamic reasons, it is extremely difficult to achieve zero emission — or even to approach it — by means of a control device. The effort needed to accomplish each incremental level of efficiency (i.e., the percent of the pollutant recaptured or destroyed) rises dramatically as the device's required efficiency is increased. In practice, this effect is expressed economically; the cost of a control system rises disproportionately relative to increases in its efficiency. For example, it has been found that the cost of a power plant's sulfur dioxide control system rises from $50 per kW of capacity at 70% efficiency to $2,000 per kW at 90% efficiency. To reach 99.9% efficiency, the system would cost on the order of $4,000 per kW— about 10 times the cost of the power plant itself. Similarly, it has been reported that to achieve the 0.1 ng/m^3 PCDD/F emission rate now required of German municipal waste incinerators, the cost of the control system amounts to about two-thirds of the total cost of the facility. Thus, to approach the goal of zero emission by means of improved PCDD/F control systems, the cost of building and operating the sources would need to rise dramatically.

The task of achieving zero emission through the strategy of pollution prevention is not encumbered by the serious economic constraint that is built into the control strategy. Here, too, there is an economic issue: How will the cost of achieving the economic good produced by a

PCDD/F-generating facility change if a PCDD/F-free technology is used instead to produce the same good? This approach has been used to evaluate the technological and economic feasibility of substituting technologies with dramatically reduced PCDD/F emissions (in some cases, essentially zero emissions) for the production technologies responsible for the major loadings of PCDD/F to the Great Lakes (Commoner *et al*. 1996a). The analysis of replacing municipal waste incinerators in the Great Lakes states and the province of Ontario with an essentially PCDD/F-free technology that produces the same good — i.e., the disposal of an equal amount of waste — may be used as an example.

In 1993 the communities in the Great Lakes region burned 11.8 million tons of residential waste in 54 incinerators, at an average cost to the communities of $57.58 per ton (the tipping fee). A technologically feasible alternative is to use intensive recycling programs to dispose of this same amount of waste instead of continuing to operate the incinerators. The cost of this substitution is the cost of operating the recycling system (additional costs for collection, recyclables processing, and public education are necessary), plus the cost of retiring the bonded debt carried by the incinerators. But this substitution involves two economic gains: the incinerator tip fees are eliminated, and (based on their current market value) the materials recovered by the recycling system would yield an income. When these factors are taken into account, the Great Lakes communities would save about $500 million per year in trash disposal costs if the switch were made to the recycling alternative. There would be a net gain of nearly 3600 waste processing jobs in the region, and 21,000 additional jobs could be created if enterprises were set up to make use of the recovered materials. Finally, since the power produced by the incinerator is now sold to utilities well above the utilities' own cost of power production, closing the incinerators would reduce these costs and hence consumers' electricity rates.

The cost of replacing the Great Lakes region's 609 hospital waste incinerators (as of 1995) — another major source of airborne PCDD/F — with autoclaves (and subsequent landfilling of the sterilized material), a well-established alternative that does not produce PCDD/F, was also analyzed. If, as mandated by impending U.S. EPA regulations, the existing incinerators (many with minimal or no emission controls) were upgraded with Maximum Achievable Control Technology, total regional operating costs would be more than twice the cost of installing autoclaves instead. It is not surprising, therefore, that in the last few years hospital incinerators are being rapidly replaced by autoclaves.

Similar analyses have been made of the technical and economic feasibility of removing PCDD/F-generating inputs from iron sintering plants, of replacing hazardous waste (which generates PCDD/F when burned) with conventional fuels in cement kilns, and of converting the region's pulp and paper mills to totally chlorine-free operations. Together, these changes would increase production costs in the region by about $370 million annually -- less than the savings gained by replacing incinerators. In any case, the total changes in the region's economy brought about by all of the pollution prevention measures amounts to less than 0.01% of the annual regional gross product.

In sum, these preventive changes, which are estimated to virtually eliminate more than three-fourths of the source-specific PCDD/F loading to the Great Lakes, could be achieved with little or no effect on the region's economy -- but with a major gain in environmental quality. Given the general similarity of PCDD/F sources in the rest of North America with those in the Great Lakes region, it is likely that prevention efforts applied in other regions would also be technically and economically feasible.

8. References

Addink, R. and K. Olie (1995). "Mechanisms of Formation and Destruction of Polychlorinated Dibenzo-p-dioxins and Dibenzofurans in Heterogeneous Systems." Environmental Science and Technology **29**(6): 1425-1435.

Ahlborg, U.G., G.C. Becking, L.S. Birnbaum, A. Brouwer, H.J.G.M. Derks, M. Feeley, G. Golor, A. Hanberg, J.C. Larsen, A.K.D. Liem, S.H. Safe, C. Schlatter, F. Waern, M. Younes, and E. Yrjanheikki (1994). "Toxic Equivalency Factors for Dioxin-Like PCB's: Report on a WHO-ECEH and IPCS Consultation, December 1993." Chemosphere **28**(6):1049-1067.

Amendola, G., D. Barna, B. Losser, L. LaFleur, A. McBride, F. Thomas, T. Tiernay, and R. Whittemore (1989). "The Occurrence and Fate of PCDDs and PCDFs in Five Bleached Kraft Pulp and Paper Mills." Chemosphere **18**:181-188.

Arnold, S.F., D.M. Klotz, B.M. Collins, P.M. Vonier, L.J. Guillette Jr., and J.A. McLachlan (1996). "Synergistic Activation of Estrogen Receptor with Combinations of Environmental Chemicals." Science **272**:1489-1492.

Aspelin, A. (1994). Pesticide Industry Sales and Usage: 1992 and 1993 Market Estimates. Washington D.C.: U.S. EPA (Biological and Economic Analysis Division, Office of Pesticide Programs, Office of Prevention, Pesticides and Toxic Substances).

Atkinson, R. (1987). "Estimation Of OH Radical Reaction-Rate Constants and Atmospheric Lifetimes For Polychlorobiphenyls, Dibenzo-Para-Dioxins, and Dibenzofurans." Environmental Science and Technology **21**(3): 305-307.

Atkinson, R. (1991). "Atmospheric Lifetimes Of Dibenzo-*p*-Dioxins and Dibenzofurans." Science Of the Total Environment **104**(1-2): 17-33.

Ayotte, P., E. Dewailly, J.J. Ryan, S. Bruneau, G. Lebel and L. Ferron (1995). "PCBs and dioxin-like compounds in pooled plasma samples from Inuit adults living in Nunavik (Arctic Quebec)." Organohalogen Compounds **26**: 181-186 (Proceedings of 15th Symposium on Dioxins and Related Compounds).

Barrie, L. A., D. Gregor, *et al*. (1992). "Arctic Contaminants - Sources, Occurrence and Pathways." Science Of the Total Environment **122**(1-2): 1-74.

Beck, H., *et al*. (1990). "Influence of Different Regional Emissions and Cardboard Containers on Levels of PCDD, PCDF and Related Compounds in Cow Milk." Chemosphere **21**(6): 789-798.

Beck, H., K. Eckart, W. Mathar, R. Wittkowski (1989). "PCDD and PCDF Body Burden from Food Intake in the Federal Republic of Germany." Chemosphere **18**(1-6):417-424.

Beck, G.G., Smith, T.G. & Addison, R.F. (1994). Organochlorine residues in harp seals *Phoca groenlandica* from the Gulf of St. Lawrence and Hudson Strait: an evaluation of contaminant concentrations and burdens. Can. J. Zool. **72**, 174-182.

Bidleman, T. F. (1988). "Atmospheric Processes - Wet and Dry Deposition Of Organic-Compounds Are Controlled By Their Vapor Particle Partitioning." Environmental Science and Technology **22**(4): 361-367.

Bingham, A.G., C.J. Edmunds, B.W.L. Graham, M.T Jones (1989). "Determination of PCDDs and PCDFs in Car Exhaust." Chemosphere **19**(1-6):669-673.

Birla, P. and R. M. Kamens (1994). "Effect Of Combustion Temperature On the Atmospheric Stability Of Polybrominated Dibenzo-P-Dioxins and Dibenzofurans." Environmental Science and Technology **28**(8): 1437-1443.

Birmingham, B., B. Thorpe, R. Frank, R. Clement, H. Tosine, G. Fleming, J. Ashman, J. Wheeler, B.D. Ripley, and J. Ryan (1989). "Dietary Intake of PCDD and PCDF from food in Ontario, Canada." Chemosphere **19**(6): 507-512.

Bourbonniere, R A. (1996). Depositional History of Sediments from Lake Athabasca, Legend Lake and Weekes Lake: Chlorinated Contaminants. National Hydrology Research Institute Contribution 96-152.

Brochu, C., G. Hamelin, S. Moore, D. Laliberte, and Y. de Lafontaine (1995). "Contribution of PCDD/F's, Planar and Ortho Substituted PCB Congeners to the Total TCDD-Equivalent Concentration in Fish Fillet from the St. Lawrence River." Organohalogen Compounds **26**:293-298.

Broman, D., C. Naf, C. Rolff, and Y. Zebuhr (1991). "Occurrence and Dynamics of Polychlorinated Dibenzo-p-dioxins and Dibenzofurans and Polycyclic Aromatic Hydrocarbons in the Mixed Surface Layer of Remote Coastal and Offshore Waters of the Baltic." Environmental Science and Technology **25**(11):1850-1864.

Brown, J.E. and J.B. Silkworth (1995). "The Natural History of Dioxin Equivalents." Organohalogen Compounds 26:273-276.

Brzuzy, L.P., Hites, R.A. (1995). "Estimating the Atmospheric Deposition of Polychlorinated Dibenzo-p-dioxins and Dibenzofurans from Soils." Environmental Science and Technology **29**(8):2090-2098.

Calamari, D., E. Bacci, *et al*. (1991). "Role Of Plant Biomass In the Global Environmental Partitioning Of Chlorinated Hydrocarbons."Environmental Science and Technology **25**(8): 1489-1495.

CEPA (1993). Polychlorinated Dibenzodioxins and Polychlorinated Dibenzofurans. Canadian Environmental Protection Act. Priority Substances List Assessment Report No. 1. Government of Canada, Environment Canada, Health and Welfare Canada. Ottawa, Ontario, Canada.

Charles, M. Judith, Ronald A. Hites (1987). "Sediments as Archives of Environmental Pollution Trends" in Sources and Fates of Aquatic Pollutants, developed from a symposium sponsored by the Div. of Environmental Chemistry at the 190th Meeting of the American Chemical Society, Chicago, IL, Sept. 8-15, 1985 (Washington, DC: American Chemical Society).

Clarke, A. N., D. J. Wilson, *et al*. (1996). "A Review Of 2,3,7,8-Tetra Chlorinated Dibenzodioxins and Toxicity Equivalencies In Fish In the United-States and International Waterways." Hazardous Waste & Hazardous Materials **13**(4): 419-443.

Clement, R.E., C. Tashiro, G. Suter, E. Reiner, and D. Hollinger (1989). "Chlorinated dibenzo-*p*-dioxins (CDDs) and dibenzofurns (CDFs) in effluents and sludges from pulp and paper mills." Chemosphere **18**:1189-1197.

Cleverly, D. (1996). Personal communication. U.S. EPA, Office of Research and Development, Office of Health and Environmental Assessment, Washington D.C.

Cohen, M., B. Commoner, H. Eisl, P. Bartlett, A. Dickar, C. Hill, J. Quigley, J. Rosenthal (1995). Quantitative Estimation of the Entry of Dioxins, Furans and Hexachlorobenzene into the Great Lakes from Airborne and Waterborne Sources. Center for the Biology of Natural Systems (CBNS), Queens College, Flushing NY.

Commoner, B., K. Shapiro and T. Webster (1987). "The Origin and Health Risks of PCDD and PCDF." Waste Management and Research **5**:327-346

Commoner, B., M. Cohen, P. Bartlett, A. Dickar, H. Eisl, C. Hill, J. Rosenthal (1996a). Zeroing Out Dioxin in the Great Lakes: Within Our Reach. Center for the Biology of Natural Systems (CBNS), Queens College, Flushing NY.

Commoner, B., M. Cohen, P. Bartlett, A. Dickar, H. Eisl, C. Hill, J. Rosenthal (1996b). Dioxin Fallout in the Great Lakes: Where it Comes From; How to Prevent It; At What Cost (Summary). Center for the Biology of Natural Systems (CBNS), Queens College, Flushing NY.

Connett, P. and E. Connett (1994). Waste Not #302, September, Work on Waste USA, 82 Judson Street, Canton, New York 13617, 315-379-9200.

Cook, P.M., D.W. Kuehl, M.K. Walker, R.E. Peterson (1991). "Bioaccumulation and Toxicity of TCDD and Related Compounds in Aquatic Ecosystems." in Banbury Report 35: Biological Basis for Risk Assessment of Dioxins and Related Compounds. Cold Spring Harbor Laboratory Press.

Corbet, R.L., D.C.G. Muir, and G.R.B. Webster (1983). "Fate of 1,3,6,8-T_4CDD in an Outdoor Aquatic System." Chemosphere **12**(4-5):523-527.

Cordero J. (1994). "Finding the Causes of Birth Defects." New England Journal of Medicine **331**(1): 48-49.

Correa, A., W.F. Stewart, H.C. Yeh, and C. Santos-Burgoa (1994). "Exposure Measurement in Case-Control Studies: Reported Methods and Recommendations." Epidemiologic Reviews **16**(1): 18-32.

Costner, P. (1997). The Burning Question: Chlorine and Dioxin. Greenpeace USA, Washington D.C.

Couture, L.A., *et al.* (1990). "A Critical Review of the Developmental Toxicity and Teratogenicity of 2,3,7,8-Tetrachlorodibenzo-p-dioxin." Teratology **42**: 619-627

Craig, G.R., P.L. Orr, J.L. Robertson, and W.M. Vrooman (1990). "Toxicity and Bioaccumulation of AOX and EOX." Pulp & Paper Canada **91**:39-45.

Czuczwa, J., B. McVeety, and R. Hites (1984). "Polychlorinated Dibenzo-p-dioxins and Dibenzofurans in Sediments from Siskiwit Lake, Isle Royale." Science **226**:568-569.

Czuczwa, J., and R. Hites (1986). "Airborne Dioxins and Dibenzofurans: Sources and Fates." Environmental Science and Technology **20**(2):195-200.

De Jong, A.P.J.M., Liem, A.K.D., and Hoogerbrugge, R. (1993). "Study of Polychlorinated Dibenzodioxins and Furans from Municipal Waste Incinerator Emissions in the Netherlands: Analytical Methods and Levels in the Environment and Human Food Chain." Journal of Chromatography **643**:91-106.

DeVito, M. J. and L. S. Birnbaum (1994). "Toxicology of Dioxins and Related Compounds." in Dioxins and Health. A. Schecter, editor. New York, Plenum Press**:** 139-162.

DeVito, M.J., *et al*. (1995). "Comparisons of Estimated Human Body Burdens of Dioxinlike Chemicals and TCDD Body Burdens in Experimentally Exposed Animals." Environmental Health Perspectives **103**(9):820-831.

Dewailly, E., J. J. Ryan, C. Laliberte, S. Bruneau, J. P. Weber, S. Gingras and G. Carrier (1994). "Exposure Of Remote Maritime Populations to Coplanar PCBs." Environmental Health Perspectives **102**(S1): 205-209.

Dewailly, E., P. Ayotte, S. Bruneau, C. Laliberté, D.C.G. Muir and R.J. Norstrom (1993). "Inuit Exposure to Organochlorines Through the Aquatic Food Chain in Arctic Québec." Environmental Health Perspectives **101**: 618-620.

Draxler, R. (1991). "The Accuracy of Trajectories during ANATEX Calculated Using Dynamic Model Analyses Versus Rawinsonde Observations." J. App. Meteor. 30(10): 1446-1467.

Draxler, R. (1992). "Hybrid Single-Particle Lagrangian Integrated Trajectories (HY-SPLIT): Version 3.0 -- User's Guide and Model Description." NOAA Technical Memo. ERL ARL-195 (Silver Spring, MD: NOAA Air Resources Lab.).

Draxler, R. (1994). "HY-SPLIT Deposition Module." (Silver Spring, MD: NOAA Air Resources Lab.), Aug. 29.

Duarte-Davidson, R., S. J. Harrad, S. C. Allen and K. C. Jones (1992). "The Relative Contribution Of Individual PCBs, PCDDs and PCDFs to Toxic Equivalent Values Derived For Bulked Human Breast-Milk Samples From the UK." Chemosphere **25**(11): 1653-1663.

Eitzer, B. D. and R. A. Hites (1989a). "Polychlorinated Dibenzo-Para-Dioxins and Dibenzofurans In the Ambient Atmosphere Of Bloomington, Indiana."Environmental Science and Technology **23**(11): 1389-1395.

Eitzer, B. and R. Hites (1989c). "Dioxins and Furans in the Ambient Atmosphere : A Baseline Study." Chemosphere **18**(1-6): 593-598.

Eitzer, B. (1995). "Polychlorinated Dibenzo-p-dioxins and Dibenzofurans in Raw Milk Samples from Farms Located Near a New Resource Recovery Incinerator." Chemosphere **30**(7):1237-1248.

ENSR Consulting and Engineering (1989). Final Report for the Environmental Baseline Assessment for Dioxins and Dibenzofurans in the Vicinity of the Elk River Generating Station. Volume II - Soils, Crops, Cow's Milk, Sediment, Water, and Fish, prepared for United Power Association, Elk River, Minnesota, by ENSR, Fort Collins, Colorado.

Evans, M.S., R A. Bourbonniere, D.C.G. Muir, W.L. Lockhart, P. Wilkinson, and B.N. Billeck. Depositional History of Sediment in Great Slave Lake: Spatial and Temporal Patterns in Geochronology, Bulk Parameters, PAHs and Chlorinated Contaminants. Report No. 99, Northern River Basin Study, Edmonton Alberta, 173 pp.

Fairchild, W.L., D.C.G. Muir, R.S. Currie and A.L. Yarechewski (1992). "Emerging Insects as a Biotic Pathway for Movement of 2,3,7,8-Tetrachlorodibenzofuran from Lake Sediments." Environ. Toxicol. Chem. **11**, 867-872.

Fanelli *et al*. (1980). "2,3,7,8-Tetrachlorodibenzo-p-dioxin Levels in Cow's Milk from the Contaminated Area of Seveso, Italy." Bulletin of Environmental Contamination and Toxicology **24**:634-639.

Fangmark, I., *et al*. (1993). "Infuence of Combustion Parameters on the Formation of Polychlorinated Dibenzo-p-dioxins, Dibenzofurans, Benzenes, and Biphenyls and Polyaromatic Hydrocarbons in a Pilot Incinerator." Environmental Science and Technology **27**(8):1602-1610.

Feldman, J., and T. Shistar (1997). Poison Poles: A Report About Their Toxic Trail and the Safer Alternatives. National Coalition Against the Misuse of Pesticides. Washington, D.C.

Fiedler, H., O. Hutzinger, and C. Timms (1990). "Dioxins: Sources of Environmental Load and Human Exposure." Tox. & Env. Chem. **29**:157- 234.

Fiedler, H., O. Hutzinger (1992). "Sources and Sinks of Dioxins: Germany." Chemosphere **25**(7-10):1487-1491.

Fisher, B. E. A. (1983). "A Review Of the Processes and Models Of Long-Range Transport Of Air-Pollutants." Atmospheric Environment **17**(10): 1865-1880.

Fletcher, C.L., and W.A. McKay (1993). "Polychlorinated Dibenzo-p-dioxins (PCDDs) and Dibenzofurans (PCDFs) in the Aquatic Environment - A Literature Review." Chemosphere **26**(6):1041-1069.

Freeman, R.A., and J.M. Schroy (1989). "Comparison of the Rate of TCDD Transport at Times Beach and at Elgin AFB." Chemosphere **18**(1-6):1305-1312.

Fries, G. (1995a). "A Review of the Significance of Animal Food Products as Potential Pathways of Human Exposure to Dioxins." Journal of Animal Science **73**:1639-1650

Fries, G. (1995b). "Transport of Organic Environmental Contaminants to Animal Products." Reviews of Environmental Contamination and Toxicology **141**:71-109.

Friesen, K.J., M.D. Segstro, D.C.G. Muir, and G.R.B. Webster (1995). "Effect of the Input Pathway on the Distribution of 1,2,3,4,7-Pentachlorodibenzo-*p*-dioxin in an Aquatic Ecosystem." Environmental Toxicology and Chemistry **14**(11): 1921-1929.

Furst, P., C. Furst, W. Groebel (1990). "Levels of PCDDs and PCDFs in Food-Stuffs from the Federal Republic of Germany." Chemosphere **20**(7-9):787-792.

Furst, P., G. Krause, D. Hein, T. Delschen, K. Wilmers (1993). "PCDD/PCDF in Cow's Milk in Relation to their Levels in Grass and Soil." Chemosphere **27**(8):1349-1357.

Furst, P., Furst, C., and Wilmers, K. (1992). "Survey of Dairy Products for PCDDs, PCDFs, PCBs and HCB." Chemosphere **25**(7-10): 1039-1048.

Grandjean P. (1990). Skin penetration. Taylor & Francis Ltd, London, pp 81-95.

Grundy, S. L., D. A. Bright, *et al*. (1995). "Sources and Signatures of PCDDs and PCDFs in Soils from the Canadian North." Organohalogen Compounds **24**: 63-67.

Hagen, M., A.G. Colodey, W.D. Knapp, and S.C. Samis (1995). "Environmental Response to Decreased Dioxin and Furan Loadings from British Columbia Coastal Pulp Mills." Organohalogen Compounds **24**:251-256.

Hagenmaier, H. *et al*. (1990). "Emission of Polyhalogenated Dibenzodioxins and Dibenzofurans from Combustion Engines." Organohalogen Compounds **2**:329-334.

Hagenmaier, H., J. She, and C. Lindig (1992). "Persistence of Polychlorinated Dibenzo-p-Dioxins and Polychlorinated Dibenzofurans in Contaminated Soil at Maulach and Rastatt in Southwest Germany." Chemosphere **25**(7-10): 1449-1456.

Halonen, I., *et al*. (1994). "The Effect of Inorganic and Organic Chlorine on Formation of Highly Chlorinated Organic Compounds During Incineration: Laboratory Pilot Study." Chemosphere **28**(12):2129-2138.

Hashimoto, S., M. Matsuda, *et al*. (1995a). "Simple Sampling and Analysis Of PCDDs and PCDFs In Japanese Coastal Seawater." Chemosphere **30**(10): 1979-1986.

Hashimoto, S., T. Wakimoto, *et al*. (1995b). "Possible Natural Formation Of Polychlorinated Dibenzo-P-Dioxins As Evidenced By Sediment Analysis From the Yellow Sea, the East-China-Sea and the Pacific-Ocean." Marine Pollution Bulletin **30**(5): 341-346.

Hardell, L., M. Eriksson, *et al*. (1994). "Cancer Epidemiology." in Dioxins and Health. A. Schecter, editor. New York, Plenum Press**:** 525-547.

Harrison, N., *et al*. (1996). "PCDDs and PCDFs in Milk from Farms in Derbyshire, U.K." Chemosphere **32**(3):453-460.

Hattemer-Frey, H., and C.C. Travis (1991). "An Overview of Food Chain Impacts from Municipal Waste Combustion." Municipal Waste Incineration Risk Assessment, edited by C.C. Travis, New York: Plenum Press, pp 87-124.

Hayes, L.C., and S.E. Rodenbeck (1992). "Developing a Public Health Assessment — Impact of a Mercury-Contaminated Discharge to Surface-water." J. Environmental Health **55**(2):16-18.

Hebert, C. E., M. Gamberg, B.T. Elkin, M. Simon, and R.J. Norstrom (1996). "Polychlorinated Dibenzodioxins, Dibenzofurans and Non-Ortho Substituted Polychlorinated-Biphenyls In Caribou (Rangifer-Tarandus) From the Canadian Arctic." Science Of the Total Environment **183**(3): 195-204.

Hirakawa, H., T. Iada, T. Matsueda, R. Nakagawa, T. Hori, and J. Nagayama (1995). "Comparison of Concentrations of PCDDs, PCDFs, PCBs and other Organohalogen Compounds in Human Milk of Primaparas and Multiparas." Organohalogen Compounds **26**:197-200

Hodson, P.V., Castonguay, M., Couillard, C.M., Desjardins, C., Pelletier, É., & McLeod, R. (1994). "Spatial and Temporal Variations in Chemical Contamination of American Eels (*Anguilla Rostrata*) Captured in the Estuary of the St. Lawrence River." Canadian Journal of Fisheries and Aquatic Science **51**: 464-478.

Hoff, R. M., W. M. J. Strachan, *et al*. (1996). "Atmospheric Deposition Of Toxic Chemicals to the Great Lakes: a Review Of Data Through 1994." Atmospheric Environment **30**(20): 3505-3527.

Hsu, S.T. *et al.* (1985). "Discovery and Epidemiology of PCB Poisoning in Taiwan." Environmental Health Perspectives **59**: 5-10.

Huff, J. (1994). "Dioxins and Mammalian Carcinogenesis." in Dioxins and Health. A. Schecter, editor. New York, Plenum Press**:** 389-407.

Iwata, H., S. Tanabe, N. Sakai, R. Tatsukawa (1993). "Distribution of Persistent Organochlorines in the Oceanic Air and Surface Seawater and the Role of [the] Ocean on Their Global Transport and Fate." Environmental Science and Technology **27**(6):1080-1098.

Jarman, W. M., R. J. Norstrom, *et al*. (1996). "Levels Of Organochlorine Compounds, Including PCDDs and PCDFs, In the Blubber Of Cetaceans From the West-Coast Of North-America." Marine Pollution Bulletin **32**(5): 426-436.

Johnson, B. (1992). "A Precis on Exposure Assessment." Journal of Environmental Health **55**(1): 6-9.

Jones, P. D., D. J. Hannah, *et al*. (1996). "Persistent Synthetic Chlorinated Hydrocarbons In Albatross Tissue Samples From Midway Atoll." Environmental Toxicology and Chemistry **15**(10): 1793-1800.

Junge, C. E. (1977). Basic Considerations About Trace Constituents in the Atmosphere as Related to the Fate of Global Pollutants. Fate of Pollutants in the Air and Water Environments. I. H. Suffet. New York, Wiley**:** 7-22.

Kapila, S., A.F. Yanders, C.E. Orazio, J.E. Meadows, S. Cerlesi, T.E. Clevenger (1989). "Field and Laboratory Studies on the Movement and Fate of Tetrachlorodibenzo-p-Dioxin in Soil." Chemosphere **18**(1-6): 1297-1304.

Karasek, F. (1995). "An Overview of Dioxin Formation in Combustion Systems." Organohalogen Compounds **23**:315-317.

Kao, A. S. and C. Venkataraman (1995). "Estimating the Contribution of Reentrainment of the Atmospheric Deposition of Dioxin." Chemosphere **31**(10): 4317-4331.

Kaupp, H., J. Towara, M. McLachlan (1994). "Distribution of Polychlorinated Dibenzo-*p*-Dioxins and Dibenzofurans in Atmospheric Particulate Matter with Respect to Particle Size." Atmospheric Environment **28**(4): 585-593.

Kervliet, N. I. (1994). "Immunotoxicology of Dioxins and Related Compounds." in Dioxins and Health. A. Schecter, editor. New York, Plenum Press**:** 199-225.

Koester, C. J. and R. A. Hites (1992a). "Photodegradation Of Polychlorinated Dioxins and Dibenzofurans Adsorbed to Fly-Ash." Environmental Science and Technology **26**(3): 502-507.

Koester, C., R. Hites (1992b). "Wet and Dry Deposition of Chlorinated Dioxins and Furans." Environmental Science and Technology **26**(7):1375-1382.

Krokos, F., C. S. Creaser, C. Wright and J. R. Startin (1996). "Levels Of Selected Ortho and Non-Ortho Polychlorinated-Biphenyls In UK Retail Milk." Chemosphere **32**(4): 667-673.

Keuhl, D.W., P.M. Cook, A.R. Batterman, D.B. Lothenbach, and B.C. Butterworth (1987). "Bioavailability of Polychlorinated Dibenzo-p-dioxin and Dibenzofurans from Contaminated Wisconsin River Sediment to Carp." Chemosphere **16**:667-679.

Kurtz, D. A., Ed. (1990). Long Range Transport of Pesticides. Chelsea, MI, Lewis Publishers.

Kwok, E.S.C., J. Arey and R. Atkinson (1995). "Gas-Phase Atmospheric Chemistry of Dibenzo-p-dioxin and Dibenzofuran." Environmental Science and Technology **28**(3):528-533.

Kwok, E. S. C. and R. Atkinson (1995). "Estimation Of Hydroxyl Radical Reaction-Rate Constants For Gas-Phase Organic-Compounds Using a Structure-Reactivity Relationship - an Update." Atmospheric Environment **29**(14): 1685-1695.

Kwok, E.S.C., R. Atkinson, and J. Arey (1995). "Rate Constants for the Gas-Phase Reactions of the OH Radical with Dichlorobiphenyls, 1-Chlorodibenzo-p-dioxin, 1,2-Dimethoxybenzene, and Diphenyl Ether: Estimation of OH Radical Reaction Rate Constants for PCBs, PCDDs, and PCDFs." Environmental Science and Technology **29**(6):1591-1598.

LaFleur, L., *et al*. (1990). "Analysis of TCDD and TCDF on the ppq-Level in Milk and Food Sources." Chemosphere, **20**(10-12):1657-1662.

Lassek, E., Jahr, D., and Mayer, R. (1993). "Polychlorinated Dibenzo-p-dioxins and Dibenzofurans in Cows Milk from Bavaria, FRG." Chemosphere **27**(4): 519-534.

Liem, A.K.D., J.M.C. Albers, R.A. Baumann, A.C. van Beuzekom, R.S. den Hartog, R. Hoogerbrugge, A.P.J.M. de Jong, and J.A. Marsman (1995). "PCBs, PCDDs, PCDFs and Organochlorine Pesticides in Human Milk in the Netherlands. Levels and Trends." Organohalogen Compounds **26**:69-74

Lorber, M., D. Cleverly, J. Schaum, L. Phillips, G. Schweer, T. Leighton (1994). "Development and Validation of an Air-to-Beef Food Chain Model for Dioxin-Like Compounds." Science of the Total Environment **156**:39-65.

Macdonald, R. W., W. J. Cretney, N. Crewe, and D. Paton (1992). "A History Of Octachlorodibenzo-*p*-Dioxin, 2,3,7,8-Tetrachlorodibenzofuran, and 3,3',4,4'-Tetrachlorobiphenyl Contamination In Howe Sound, British-Columbia." Environmental Science & Technology **26**(8): 1544-1550.

Marklund, S., C. Rappe, M. Tysklind, K.E. Egeback (1987). "Identification of Polychlorinated Dibenzofurans and Dioxins in Exhausts from Cars Run on Leaded Gasoline." Chemosphere **16**(1):29-36.

Marklund, S., *et al*. (1990). "Emissions of PCDDs and PCDFs in Gasoline and Diesel Fueled Cars." Chemosphere **20**(5):553-561.

Matsumura, T., *et al*. (1995). "PCDDs Concentration in Open-Ocean Water." Organohalogen Compounds 24:353-356.

McCrady, J., *et al*. (1990). Chemosphere **21**(3):359-376.

McLachlan, M., H. Thoma, M. Reissinger, O. Hutzinger (1990). "PCDD/F in an Agricultural Food Chain. Part I: PCDD/F Mass Balance of a Lactating Cow." Chemosphere, **20**(7-9):1013-1020.

McLachlan, M. (1996). "Bioaccumulation of Hydrophobic Chemicals in Agricultural Food Chains." Environmental Science and Technology **30**(1): 252-259.

Merriman, J.C., D.H.J. Anthony, J.A. Kraft and R.J. Wilkinson (1991). "Rainy River Water Quality in the Vicinity of Bleached Kraft Mills." Chemosphere **23**: 1605-1615.

Meylan, W. M. and P. H. Howard (1993). "Computer Estimation Of the Atmospheric Gas-Phase Reaction-Rate Of Organic-Compounds With Hydroxyl Radicals and Ozone." Chemosphere **26**(12): 2293-2299.

Meylan, W.M. and P.H. Howard (1996a). User's Guide for AOPWIN: Atmospheric Oxidation Program. Syracuse, New York: Syracuse Research Corporation.

Meylan, W.M. and P.H. Howard (1996b). AOPWIN: Atmospheric Oxidation Program, v1.82 for Microsoft Windows 3.1. Syracuse, New York: Syracuse Research Corporation (phone: 315-426-3200).

Miller, G., V. Hebert, M. Miille, R. Mitzel, R. Zepp (1989). "Photolysis of Octachlorodibenzo-p-dioxin on Soils: Production of 2,3,7,8-TCDD." Chemosphere **18**(1-6):1265-1274.

Montague, P. (1989), editor. "Dangers of Dioxin Exposures: Absorption Through the Skin." Rachel's Environment & Health Weekly **120** (March 14, 1989). Environmental Research Foundation, Annapolis, MD.

Morcos, R. (1997). Personal Communication with Mr. Raouf Morcos, Co-Chair of the Federal/Provincial Task Force on Dioxins and Furans (phone: 819-953-1137).

Muir, D., B. Braune, B. DeMarch, R. Norstrom, R. Wagemann, M. Gamberg, K. Poole, R. Addison, D. Bright, M. Dodd, W. Duschenko, J. Eamer, M. Evans, B. Elkin, S. Grundy, B. Hargrave, C. Hebert, R. Johnstone, K. Kidd, B. Koenig, L. Lockhart, J. Payne, J. Peddle, and K. Reimer (1996). "Chapter 3. Ecosystem Uptake and Effects." in Shearer,

R. (editor), Canadian Arctic Contaminants Assessment Report, Indian and Northern Affairs Canada, Ottawa 1996.

Muir, D.C.G, S. Lawrence, M. Holoka, W.L. Fairchild, M.D. Segstro, G.R.B. Webster, and M.R. Servos (1992a). "Partitioning of Polychlorinated Dioxins and Furans Between Water, Sediments and Biota in Lake Mesocosms." Chemosphere **25**, 119-124.

Muir, D.C.G., R. Wagemann, B.T. Hargrave, D.J. Thomas, D.B. Peakall, and R.J. Norstrom (1992b). "Arctic Marine Ecosystem Contamination." The Science of the Total Environment **122**:75-134.

Muir, D.C.G, W.L. Fairchild, and D.M. Whittle (1992c). "Predicting Bioaccumulation of Chlorinated Dioxins and Furans in Fish near Canadian Bleached Kraft Mills." Water Poll. Res. J. Can. **27**:487-507.

Norstrom, R. J. and D. C. G. Muir (1994). "Chlorinated-Hydrocarbon Contaminants In Arctic Marine Mammals." Science Of the Total Environment **154**(2-3): 107-128.

Norstrom, R.J., M. Simon and D.C.G. Muir (1990). "Chlorinated Dioxins and Furans in Marine Mammals from the Canadian Arctic." Environmental Pollution **66**, 1-20.

Orazio, C.E., S. Kapila, R.K. Puri, and A.F. Yanders (1992). "Persistence of Chlorinated Dioxins and Furans in the Soil Environment." Chemosphere **25**(7-10):1469-1474.

Ott, W.R. (1985). "Total Human Exposure." Environmental Science and Technology **19**(10): 880-886.

Pacyna, J. M. (1995). "Sources, Particle Size Distribution and Transport of Aerosols." in Airborne Particulate Matter. T. Kouimtzis and C. Samara, editors. Vol. 4-D of The Handbook of Environmental Chemistry. New York, Springer -Verlag. pp 70-97.

Pankow, J. F. (1987). "Review and Comparative Analysis of the Theories on Partitioning Between the Gas and Aerosol Particulate Phases in the Atmosphere." Atmospheric Environment **21**(11): 2275-2283.

Pankow, J. F. (1988). "The Calculated Effects Of Non-Exchangeable Material On the Gas Particle Distributions Of Organic-Compounds." Atmospheric Environment **22**(7): 1405-1409.

Pankow, J. F. and T. F. Bidleman (1991). "Effects Of Temperature, TSP and Per Cent Nonexchangeable Material In Determining the Gas Particle Partitioning Of Organic-Compounds." Atmospheric Environment Part a-General Topics **25**(10): 2241-2249.

Pastershank, G.M. and D.C.G. Muir (1995). Contaminants in Environmental Samples: PCDD's and PCDF's Downstream of Bleached Kraft Mills - Peace and Athabasca Rivers, 1992. Northern River Basins Study Project Report No. 44. Northern River Basin Study, Edmonton, Alberta.

Pennise, D. M. and R. M. Kamens (1996). "Atmospheric Behavior Of Polychlorinated Dibenzo-P-Dioxins and Dibenzofurans and the Effect Of Combustion Temperature." Environmental Science and Technology **30**(9): 2832-2842.

Podoll, R. T., H. M. Jaber, *et al*. (1986). "Tetrachlorodibenzodioxin - Rates Of Volatilization and Photolysis In the Environment." Environmental Science and Technology **20**(5): 490-492.

Puri, R.K., T.E. Clevenger, S. Kapila, A.F. Yanders and R.K. Malhotra (1989). "Studies of Parameters Affecting Translocation of Tetrachlorodibenzo-p-dioxin in Soil." Chemosphere **18**(1-6): 1291-1296.

Prospero, J. M., R. J. Charlson, *et al*. (1983). "The Atmospheric Aerosol System: An Overview." Reviews of Geophysics and Space Physics **21**(7): 1607-1629.

Rappe, C., S. Marklund, *et al*. (1989). "Long Range Transport of PCDD's and PCDF's on Airborne Particles." Chemosphere **18**(1-6): 1283-1290.

Rappe, C., *et al*. (1990a). "Levels of PCDDs and PCDFs in Milk Cartons and in Commercial Milk." Chemosphere **20**(10-12): 1649-1656.

Rappe, C., L.O. Kjeller, and S.E. Kulp (1990b). "Sampling and Analysis of PCDDs and PCDFs in Surface Water and Drinking Water at 0.001 ppq Levels." Organohalogen Compounds **2**:207-210.

Rappe, C., *et al*. (1987). "Polychlorinated Dibenzofurans and Dibenzo-p-Dioxins and Other Chlorinated Contaminants in Cow Milk from Various Locations in Switzerland." Environmental Science and Technology **21**(10):964-970.

Rigo, H.G., A.J. Chandler, and W.S. Lanier (1995). The Relationship Between Chlorine in Waste Streams and Dioxin Emissions from Waste Combustor Stacks. New York, American Society of Mechanical Engineers.

Risebrough, R. W. (1990). Beyond Long-Range Transport: A Model of a Global Gas Chromatographic System. Long Range Transport of Pesticides. D. A. Kurtz. Chelsea, MI, Lewis Publishers**:** 417-426.

Riss A., *et al*. (1990). "Comparison of PCDD/PCDF Levels in Soil, Grass, Cow's Milk, Human Blood and Spruce Needles in an Area of PCDD/PCDF Contamination Through Emissions from a Metal Reclamation Plant." Chemosphere 21(12): 1451-1456.

Rogan, W.J. (1989). "Yu-Cheng." in: Halogenated Biphenyls, Terphenyls, Naphthalenes, Dibenzodioxins and Related Products. edited by R.D. Kimbrough and A.A. Jensen. 2nd edition. Amsterdan: Elsevier Science Publishers. pp 401-415.

Ryan J., *et al*, 1990: "PCDDs/PCDFs in 22 Categories of Food Collected from Six Canadian Cities Between 1985 and 1988." Organohalogen Compounds 1:497-500.

Ryan J.J., L.G. Panopio, D.A. Lewis, and D.F. Weber (1991): "Polychlorinated Dibenzo-p-Dioxins and Polychlorinated Dibenzofurans in Cows' Milk Packaged in Plastic-Coated Bleached Paperboard Containers." J. Agric. Food Chem. **39**:218-223.

Santos-Burgoa, C., *et al*. (1996). "Health Indicators for Environmental Surveillance in Rural, Semi-Rural and Highly Urbanized Settings in Mexico." Epidemiology pp. S58.

Schecter, A. (editor) (1994a). Dioxins and Health. New York, Plenum Press.

Schecter, A. (1994b). "Exposure Assessment: Measurement of Dioxins and Related Chemicals in Human Tissues." Dioxins and Health. A. Schecter, editor. New York, Plenum Press**:** 449-485.

Schecter, A., *et al*. (1994c). "Congener-specific Levels of Dioxins and Dibenzofurans in U.S. Food and Estimated Dioxin Toxic Equivalent Intake." Environmental Health Perspectives 102(11):962-966.

Schecter, A., *et al*. (1989). "Levels of Polychlorinated Dibenzodioxins and Dibenzofurans in Cow's Milk and in Soy Bean Derived Infant Formulas Sold in the United States and Other Countries." Chemosphere **19**(1-6): 913-918.

Schmid, P., and Schlatter, C. (1992). "Polychlorinated Dibenzo-p-Dioxins (PCDDs) and Polychlorinated Dibenzofurans (PCDFs) in Cow's Milk from Switzerland." Chemosphere **24**(8):1013-1030.

Seegal, R. F. and S. L. Schantz (1994). "Neurochemical and Behavioral Sequelae of Exposure to Dioxins and PCBs." in Dioxins and Health. A. Schecter, editor. New York, Plenum Press**:** 409-447.

Segstro, M.D, D.C.G. Muir, M. R. Servos, and G.R.B. Webster (1995). "Long-term Fate and Bioavailability of Sediment-associated Polychlorinated Dibenzo-p-dioxins in Aquatic Mesocosms." Environ. Toxicol. Chem. **14**: 1799-1807.

Servos, M.R., D.C.G. Muir, and G.R.B. Webster (1992a). "Environmental Fate of Polychlorinated Dibenzo-p-dioxins in Lake Enclosures." Canadian Journal of Fisheries and Aquatic Sciences **49**(4): 722-734.

Servos, M.R., D.C.G. Muir, and G.R.B. Webster (1992b). Bioavailability of Polychlorinated Dibenzo-p-dioxins in Lake Enclosures. Canadian Journal of Fisheries and Aquatic Sciences **49**(4): 735-742.

Sewart, A. and K. C. Jones (1996). "A Survey Of PCB Congeners In UK Cows' Milk." Chemosphere **32**(12): 2481-2492.

Silbergeld, E. K. and P.L. deFur (1994). "Risk Assessments of Dioxinlike Compounds." in Dioxins and Health. A. Schecter, editor. New York, Plenum Press**:** 51-78.

Sivils, L.D., S. Kapila, Q. Yan, A.A. Elseewi (1994). "Application of a Two-Dimensional Chromatography System for Gas Phase Photodegradation Studies of Polychlorinated Dibenzo-p-Dioxins." Journal of Chromatography A **688**(1-2):221-230.

Slob, W., M. Olling, H. Derks and A. Dejong (1995). "Congener-Specific Bioavailability Of Pcdd/Fs and Coplanar Pcbs In Cows - Laboratory and Field-Measurements." Chemosphere **31**(8): 3827-3838.

Smith, L.M., T.R. Schwartz, K. Feltz, and T.J. Kubiak (1990). "Determination and Occurrence of AHH-Active Polychlorinated Biphenyls, 2,3,7,8-Tetrachloro-p-Dioxin and 2,3,7,8-Tetrachlorodibenzofuran in Lake Michigan Sediment and Biota. The Question of Their Relative Toxicological Significance." Chemosphere **21**(9):1063-1085.

Smith, K. (1988). "Assessing Total Exposure in the United States." Environment **30**(8):10-15.

Stanley, J.S., and Bauer, K.M., 1989: Chlorinated Dibenzo-p-dioxin and Dibenzofuran Residue Levels in Food, State of California Air Resources Board, ARB Contract #A6-197-32, Sacramento, California.

Stevens, J. and E. Gerbec (1988). "Dioxin in the Agricultural Food Chain." Risk Analysis **8**(3):329-335

Swackhamer, D. L. and R. A. Hites (1988). "Occurrence and bioaccumulation of organochlorine compounds in fishes from Siskiwit Lake, Isle Royale, Lake Superior." Environmental Science and Technology **22**(5): 543-8.

Swanson, S., RC. Rappe, J. Malmstrom, and K. Kringstad (1988). "Emissions of PCDDs and PCDFs from the Pulp Industry." Chemosphere **13**:681-691.

Sweeney, A. (1994). "Reproductive Epidemiology of Dioxins." in Dioxins and Health. A. Schecter, editor. New York, Plenum Press**:** 549-585.

Tatsukawa, R. (1992). "Contamination of Chlorinated Organic Substances in the Ocean Ecosystem." Water Science and Technology **25**(11):1-8

Theelen, R.M.C., A.K.D. Liem, W. Slob, J.H. Van Wijnen (1993). "Intake of 2,3,7,8 Chlorine Substituted Dioxins, Furans, and Planar PCBs From Food in the Netherlands: Median and Distribution." Chemosphere **27**(9):1625-1635.

Theobald, H. M. (1994). "Developmental and Reproductive Toxicity of Dioxins and Other Ah Receptor Agonists." Dioxins and Health. A. Schecter, editor. New York, Plenum Press**:** 309-346.

Thomas, V.M. and T.G. Spiro (1995). "An Estimation of Dioxin Emissions in the United States." Toxicological and Environmental Chemistry **50**:1-37

Thomas, V.M. and T.G. Spiro (1996). "The U.S. Dioxin Inventory: Are There Missing Sources?" Environmental Science and Technology **30**(2): A82-85.

Thornton, J. (1994). Achieving Zero Dioxin: An Emergency Strategy for Dioxin Elimination. Washington D.C., Greenpeace.

Thornton, J. and J. Weinberg (1995). "Dioxin Prevention: Focusing on Chlorine Chemistry." New Solutions **5**(3) 27-38

Tritscher, A., G. S. Clark, *et al*. (1994). "Dose Response Effects of Dioxins: Species Comparison and Implication for Risk Assessment." in Dioxins and Health. A. Schecter, editor. New York, Plenum Press**:** 227-247.

Trudel, L. (1991). Dioxins and Furans in Bottom Sediments Near the 47 Canadian Pulp and Paper Mills Using Chlorine Bleaching. Water Quality Branch, Inland Water Directorate, Environment Canada, Ottawa, Ontario.

Tuinstra, L., M. Huisman and E. R. Boersma (1994). "The Dutch PCB/Dioxin Study: Contents Of Dioxins, Planar and Other PCBs In Human Milk From the Rotterdam and Groningen Area." Chemosphere **29**(9-11): 2267-2277.

Tuinstra, L., M. Huisman, E. R. Boersma, C. Koopman-Esseboom, and P.J. Sauer (1995). "Contents of Dioxins, Planar and Other PCBs In 168 Human Milk Samples." Organohalogen Compounds **26**: 255-256.

Tysklind, M., I. Fangmark, *et al*. (1993). "Atmospheric Transport and Transformation Of Polychlorinated Dibenzo-P-Dioxins and Dibenzofurans." Environmental Science and Technology **27**(10): 2190-2197.

U.S. EPA (1990). Lake Ontario TCDD Bioaccumulation Study. Final Report. May 1990.

U.S. EPA (1991). Feasibility of Environmental Monitoring and Exposure Assessment for a Municipal Waste Combustor: Rutland, Vermont Pilot Study, EPA/600/8-91/007 & EPA/600/8-91/007a (appendices)

U.S. EPA (1994a). Estimating Exposure to Dioxin-Like Compounds. Vol. I: Executive Summary. Vol. II: Properties, Sources, Occurrence and Background Exposures. Vol. III: Site-Specific Assessment Procedures. External Review Draft. "Not for Citation or Quotation." EPA/600/6-88-005Ca,b & c, June (Washington, DC: Office of Research & Development).

U.S. EPA (1994b). Health Assessment Document for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds. Volumes I, II, III. External Review Draft. "Not for Citation of Quotation." EPA/600/BP-92-001a,b & c, June (Washington, DC: Office of Research & Development).

U.S. EPA (1995). Locating and Estimating Air Emissions from Sources of Dioxins and Furans, April 1995 Draft Report prepared by Radian Corporation. Research Triangle Park, NC, U.S. EPA Office of Air Quality Planning and Standards. EPA Contract Number 68-D2-0160.

U.S.EPA (1996a). Emissions Inventory of Section 112(c)(6) Pollutants: Polycyclic Organic Matter (POM), 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD)/2,3,7,8-Tetrachlorodibenzofuran (TCDF), Polychlorinated Biphenyl Compounds (PCBs), Hexachlorobenzene, Mercury, and Alkylated Lead. Draft Report. Research Triangle Park, NC, Emission Factor & Inventory Group and Visibility & Ecosystem Protection Group.

U.S. EPA (1996b). "Revised Standards for Hazardous Waste Combustors." Federal Register, Vol. 61, No. 77, April 19, pp. 17358-17536.

van der Velde, E.G., J.A. Marsman, A.P.J.M. de Jong, R. Hoogerbrugge and A.K.D. Liem (1994). "Analysis and Occurrence Of Toxic Planar PCBs, PCDDs and PCDFs In Milk By Use Of Carbosphere Activated Carbon." Chemosphere **28**(4): 693-702.

van Jaarsveld, J. A. and M. A. A. Schutter (1993). "Modeling the Long-Range Transport and Deposition Of Dioxins - 1st Results For NW Europe." Chemosphere **27**(1-3): 131-139.

van Scheppingen, W. B., A. Verhoeven, P. Mulder, M. J. Addink and C. Smeenk (1996). "Polychlorinated-Biphenyls, Dibenzo-P-Dioxins, and Dibenzofurans In Harbor Porpoises (Phocoena-Phocoena) Stranded On the Dutch Coast Between 1990 and 1993." Archives Of Environmental Contamination and Toxicology **30**(4): 492-502.

Wagenaar, W.J., *et al*. (1995). "A Comparative Study of the Photolytic Degradation of Octachlorodibenzofuran (OCDF) and Octachlorodibenzo-p-Dioxin (OCDD)." Chemosphere **31**(4):2983-2992.

Wagner, J.C. and A.E.S. Green (1993). "Correlation of Chlorinated Organic Compound Emissions from Incineration with Chlorinated Organic Input." Chemosphere **26**(11):2039-2054.

Wania, F. and D. Mackay (1996). "Tracking the Distribution of Persistent Organic Pollutants." Environmental Science and Technology **30**(9): 390A-396A.

Walker, M. K. and R. E. Peterson (1994). "Aquatic Toxicity of Dioxins and Related Compounds." in Dioxins and Health. A. Schecter, editor. New York, Plenum Press**:** 347-387.

Webster, T. and B. Commoner (1994). "Overview: The Dioxin Debate." in Dioxins and Health. A. Schecter, editor. New York, Plenum Press**:** 1-49.

Webster, T. and P. Connett (1997). "Dioxin Emissions Inventories: The Importance of Large Sources." [. . . Submitted for publication (Accepted? Published?). . .]

Welsch-Pausch, K., M. McLachlan, G. Umlauf (1995). "Determination of the Principal Pathways of Polychlorinated Dibenzo-*p*-Dioxins and Dibenzofurans to *Lolium multiflorum* (Welsh Ray Grass)." Environmental Science and Technology **29**(4): 1090-1098.

Wenning, R.J. and T.J. Iannuzzi (1995). "Perspectives on the Toxicity Equivalency Approach to Quantify the Effects of Coplanar Polychlorinated Biphenyls in Ecological Risk Assessment." Organohalogen Compounds **26**:283-285.

Wikstrom, E., G. Lofvenius, C. Rappe, and S. Marklund (1996). "Influence of Level and Form of Chlorine on the Formation of Chlorinated Dioxins, Dibenzofurans, and Benzenes during Combustion of an Artificial Fuel in a Laboratory Reactor." Environmental Science and Technology **30**(5): 1637-1644.

Yamashita, F., *et al.* (1985). "Fetal PCB Syndrome: Clinical Features, Intrauterine Growth Retardation and Possible Alteration in Calcium Metabolism." Environmental Health Perspectives **59**:41-45.

Yunker, M.B. and W.J. Cretney (1996). "Dioxins and Furans in Crab Hepatopancreas: Use of Principal Components Analysis to Classify Congener Patterns and Determine Linkages to Contamination Sources." In: Environmental Fate and Effects of Pulp and Paper Mill Effluents, M.R. Servos, K.R. Munkittrick, J.H. Carey and G.J. Van der Kraak (eds.). St. Lucie Press, Delray, Florida. pp 315-325.

Zook, D. R. and C. Rappe (1994). "Environmental Sources, Distribution, and Fate of Polychlorinated Dibenzodioxins, Dibenzofurans, and Related Compounds." in Dioxins and Health. A. Schecter, editor. New York, Plenum Press**:** 79-113.