

# Canadian Soil QualityPOLYCHLORINATEDGuidelines for the ProtectionDIBENZO-p-DIOXINS ANDof Environmental and Human HealthPOLYCHLORINATEDDIBENZOFURANS (PCDD/Fs)

This fact sheet provides Canadian soil quality guidelines for polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDD/Fs), commonly known as dioxins and furans (PCDD/Fs), for the protection of environmental and human health. These guidelines are based on a modified approach from the CCME Protocol (CCME 1996) in which ambient background concentrations are used in the guideline derivation (Sanexen 2000).

Dioxins and furans have been declared 'toxic' under the *Canadian Environmental Protection Act (CEPA)*. As such, they are slated for virtual elimination under the federal *Toxic Substances Management Policy (TSMP)* (Government of Canada 1995) and the *CCME Policy for* 

*the Management of Toxic Substances* because they are persistent and bioaccumulative and are present in the environment primarily due to human activity.

Canada-wide standards (CWSs) for dioxins and furans are under development and are intended to make a significant contribution to the goal of virtual elimination of these compounds. CWSs focus on anthropogenic releases to both air and soil. The first suite of sectors addressed in the CWS process includes coastal pulp and paper boilers, waste incinerators, conical waste combustors, iron sintering, steel electric arc furnace manufacturing and residential wood stoves. National environmental quality guidelines for dioxins and furans in soil (this document), sediment and animal tissues (Environment Canada 2000)

	Land use			
	Agricultural	Residential/ Parkland	Commercial	Industrial
Guideline	4 <sup>a</sup>	<b>4</b> <sup>a</sup>	<b>4</b> <sup>b</sup>	4 <sup>c</sup>
SQG <sub>HH</sub>	NC <sup>d</sup>	NC <sup>d</sup>	NC <sup>d</sup>	4
Limiting pathway for SQG <sub>HH</sub>	ND	ND	ND	Off-site migration
Provisional SQG <sub>HH</sub>	4 <sup>e</sup>	4 <sup>e</sup>	4 <sup>e</sup>	NC <sup>f</sup>
Limiting pathway for provisional SQG <sub>HH</sub>	Soil ingestion	Soil ingestion	Soil ingestion	ND
SQG <sub>E</sub>	NC <sup>g</sup>	NC <sup>g</sup>	NC <sup>g</sup>	NC <sup>g</sup>
Limiting pathway for SQG <sub>E</sub>	ND	ND	ND	ND
Provisional SQG <sub>E</sub>	NC <sup>h</sup>	NC <sup>h</sup>	NC <sup>h</sup>	NC <sup>h</sup>
Limiting pathway for provisional SQG <sub>E</sub>	ND	ND	ND	ND
Interim soil quality criterion (CCME 1991)	10	1000	No value	No value

#### Table 1. Soil quality guidelines for dioxins and furans (ng TEQ·kg<sup>-1</sup>).

**Notes:** NC = not calculated; ND = not determined;  $SQG_E$  = soil quality guideline for environmental health;  $SQG_{HH}$  = soil quality guideline for human health.

<sup>a</sup>Data are sufficient and adequate to calculate only a provisional SQG<sub>HH</sub>, which is less than the existing interim soil quality criterion (CCME 1991). Thus the provisional SQG<sub>HH</sub> becomes the soil quality guideline, which supersedes the interim soil quality criterion for this land use.

<sup>b</sup>Data are sufficient and adequate to calculate only a provisional  $SQG_{HH}$ . An interim soil quality criterion (CCME 1991) was not established for this land use, therefore, the provisional  $SQG_{HH}$  becomes the soil quality guideline.

<sup>c</sup>Data are sufficient and adequate to calculate only an SQG<sub>HH</sub>. An interim soil quality criterion (CCME 1991) was not established for this land use, therefore, the SQG<sub>HH</sub> becomes the soil quality guideline.

<sup>d</sup>Data are insufficient/inadequate to calculate an SQG<sub>HH</sub> for this land use. However, data are sufficient and adequate to calculate a provisional SQG<sub>HH</sub>.

<sup>e</sup>This value is based on the mean ambient background concentration for Canadian soils.

<sup>f</sup>Because data are sufficient and adequate to calculate an SQG<sub>HH</sub> for this land use, a provisional SQG<sub>HH</sub> is not calculated.

 $g_{\text{Data are insufficient/inadequate to calculate an SQG_E}$  for this land use.

<sup>h</sup>Data are insufficient/inadequate to calculate a provisional SQG<sub>E</sub> for this land use.

serve as "alert levels" that may be used by jurisdictions as benchmarks for the management and monitoring of PCDD/Fs already present in the environment.

## **Background Information**

Dioxins and furans are chlorinated, planar tricyclic aromatic hydrocarbons representing a class of compounds that includes 210 possible congeners. PCDD/F congeners are named according to the position of the chlorine atoms on the molecule. Each possible position on the aromatic cycle is designated by a number from one to nine. The basic structure and numbering of each chemical class are shown in Figure 1.

Dioxin and furan congeners with chlorine substitution in the 2, 3, 7, and 8 positions are believed to elicit their toxicity, at least in vertebrate species, via binding to a cellular protein called the aryl hydrocarbon (*Ah*) receptor. This mode of action is commonly referred to as "dioxinlike" toxicity. Of these congeners, only 7 out of the possible 75 PCDD congeners and 10 out of the 135 PCDF congeners are thought to have "dioxin-like" toxicity. However, the degree of response, or potency, differs depending on the congener.

The congeners 2, 3, 7, 8-tetrachloro-dibenzo-*p*-dioxin  $(2,3,7,8\text{-TCDD}; C_{12}H_4Cl_4O_2)$  and 1, 2, 3, 7, 8 -PCDD  $(C_{12}H_3Cl_5O_2)$  are considered the most toxic of all the dioxin and furan congeners, although the former is the most studied congener of all the dioxins and furans.

Each congener has unique physico-chemical properties. Although these properties vary, PCDD/Fs usually exhibit

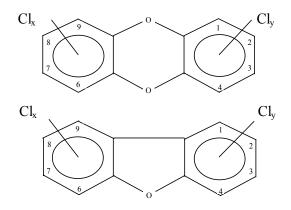


Figure 1. Chemical structure of PCDDs and PCDFs. 2,3,7,8-Substituted congeners have chlorine atoms in at least the four lateral positions, numbered 2,3,7, and 8.

low solubility in water, very low vapour pressure, and high octanol/water partition coefficients (IARC 1997; ATSDR 1998). These properties determine their fate and behaviour in the environment. In particular, they tend to bind strongly to soils.

A wide variety of methods are available to determine concentrations of dioxins and furans in environmental samples. In general, chemical analysis of PCDD/Fs is complex, costly and time and labour intensive. The detection limits of the analytical methods will differ according to the sample background interference, the matrix, and the congeners present, as well as the type of analytical equipment used. The detection limits for PCDD/Fs in soils are usually within the range of  $0.5 - 4.0 \text{ ng}\cdot\text{kg}^{-1}$ .

## *Toxic Equivalency Factors*

Dioxin and furan congeners are rarely encountered individually in the environment, and are generally present as mixtures of several congeners. In order to compare the toxicity of samples with different congener profiles, toxic equivalency factors (TEFs) have been developed that standardize "dioxin-like" substances to a toxicologically equivalent amount of 2,3,7,8-TCDD, the most toxic congener. Toxic equivalents of 2,3,7,8-TCDD are usually expressed as TEQ (Toxic Equivalent). Mathematically, a TEQ is calculated by the following equation:

Total TEQ = 
$$\sum_{i=1}^{II} (C_i \times TEF_i)$$

where:

n

- TEQ : concentration of the mixture of congeners, expressed as toxic equivalent of 2,3,7,8-TCDD
  - : number of congeners (with available TEF value)
- $C_i$  : concentration of congener *i*
- $TEF_i$ : toxic equivalency factor for the congener *i* (unitless).

An internationally-adopted TEF scheme (I-TEFs) was first developed in 1988 by NATO/CCMS (1988a, 1988b). The World Health Organization (WHO) reviewed and revised the TEF values (Table 2) in 1998. The modified TEFs were recommended for use with mammals, in addition to humans, and separate TEFs were developed for birds and fish (van den Berg 1998). Health Canada participated in, and has adopted, the revised WHO TEF scheme for assessing and managing human health risks posed by dioxins and furans in food, water, air and consumer products in which they accidentally arise. Also, the WHO TEF schemes for wildlife and fish have been adopted for

Compounds	I-TEFs (NATO/CCMS)			
		Humans/Mammals	Fish	Birds
Chlorinated dibenzo-p-dioxins				
2,3,7,8-TCDD	1.0	1.0	1.0	1.0
1,2,3,7,8-PeCDD	0.5	1.0	1.0	1.0
1,2,3,4,7,8-HxCDD	0.1	0.1	0.5	0.05
1,2,3,6,7,8-HxCDD	0.1	0.1	0.01	0.01
1,2,3,7,8,9-HxCDD	0.1	0.1	0.01	0.1
1,2,3,4,6,7,8-HpCDD	0.01	0.01	0.001	< 0.001
OCDD	0.001	0.0001	< 0.0001	0.0001
Chlorinated dibenzofurans				
2,3,7,8-TCDF	0.1	0.1	0.05	1.0
1,2,3,7,8-PeCDF	0.05	0.05	0.05	0.1
2,3,4,7,8-PeCDF	0.5	0.5	0.5	1.0
1,2,3,4,7,8-HxCDF	0.1	0.1	0.1	0.1
1,2,3,6,7,8-HxCDF	0.1	0.1	0.1	0.1
1,2,3,7,8,9-HxCDF	0.1	0.1	0.1	0.1
2,3,4,6,7,8-HxCDF	0.1	0.1	0.1	0.1
1,2,3,4,6,7,8-HpCDF	0.01	0.01	0.01	0.01
1,2,3,4,7,8,9-HpCDF	0.01	0.01	0.01	0.01
OCDF	0.001	0.0001	< 0.0001	0.0001

Table 2: Toxicity equivalency factors (TEF) for PCDD/Fs as proposed by NATO/CCMS (1988a) and WHO (van den Berg 1998)

prescribing national environmental quality guidelines, including guidelines for soil quality (Environment Canada 2000). Guidelines presented here are based on the WHO TEF scheme.

# Sources of Dioxins and Furans in Canada

Dioxins and furans are mainly produced as by-products of controlled or accidental combustion or as impurities in the manufacturing of various products. Principal sourceactivities which result in the release of PCDD/Fs to the environment include: waste incineration, chemical manufacturing, petroleum refining, wood burning, metallurgical processes, fuel combustion, and electric power generation. Dioxins and furans are not produced intentionally and they have no known use. They are also naturally present in the environment, for example as a result of forest fires and volcanic activity.

An inventory of anthropogenic releases of PCDD/Fs in Canada has been conducted by Environment Canada and the Federal/Provincial Task Force on Dioxins and Furans (Environment Canada 2001). The major results of this inventory are presented in Table 3. From 1990 to 1997, a substantial reduction in the release of PCDD/Fs was observed in Canada, mainly as a result of the adoption and implementation of more stringent regulations for pulp and paper effluents. The inventory includes details on the potential release of PCDD/Fs directly to soil; for example by land application of municipal or industrial sludge, or by the use of pesticides that could contain trace amounts of PCDD/Fs. The potential releases of PCDD/Fs to the environment from municipal or pulp and paper sludge are expected to be minimal because sludges contain low concentrations of these compounds and are dispersed on land at low application rates. With regard to pesticides, the Pest Management Regulatory Agency has estimated that the annual release of PCDD/Fs to soil is 1 g TEQ/year (Environment Canada 2001). However, for both sludge and pesticide application, repeated applications to the same area could result in an accumulation of PCDD/Fs in the soil, since these compounds are persistent.

# Levels in the Canadian Environment

Dioxins and furans are commonly detected in soil at low levels, and may be present as a result of point source releases, *in situ* contamination or redistribution and deposition via atmospheric transport. Levels of dioxins and furans vary considerably in the Canadian environment, depending on the source and site location. Due to their wide-spread production from a variety of human activities and their persistence, these substances are ubiquitous in the environment and have been found in measurable concentrations in remote areas.

# POLYCHLORINATED DIOXINS AND FURANS (PCDD/Fs)

Few studies have reported ambient background concentrations of PCDD/Fs in Canadian soils. For the purposes of guidelines derivation, ambient background concentrations are defined as concentrations in soil that reflect levels of PCDD/Fs that result mainly from aerial deposition. The levels detected can not be attributed to point source releases or in situ contamination directly to soil. Table 4 illustrates concentrations in Canadian soils from sites that meet this definition of ambient background concentrations. For example, in Ontario, soils classified as rural parkland are associated with land uses that are defined as everything that is not urban, residential, commercial/industrial, transportation rights-of-way, or active agricultural land. This would include parks, cemeteries, schools, forests or woodlots and most large undeveloped areas (OMOEE 1993). In British Columbia, background soil samples were collected from areas thought not to be impacted by immediate source of TCDD/Fs and were believed to reflect ambient levels of TCDD/Fs in the BC environment (Van Oostdam and Ward 1995). In Quebec, soil samples were collected following the fire of a PCB waste warehouse in Saint-Basile-Le-Grand. Samples identified as not impacted by the fire were presumed to be representative of the background for a semi-rural area (Trépanier 1992). Soil samples collected for these assessments are deemed representative of the definition for ambient background concentration for guideline derivation. Higher ambient background concentrations are expected to occur at urban compared to rural sites, due to the influence of local anthropogenic activities on aerial deposition.

In summary, 4 ng TEQ·kg<sup>-1</sup> (using WHO TEFs) is considered representative of the mean background concentration of PCDD/Fs in Canadian soils. This value is based on the highest mean background concentration for Canadian soils of 5.0 ng TEQ·kg<sup>-1</sup> (reported in I-TEFs); the data from Quebec may result in an overestimate of actual background (Table 4). This concentration is consistent with the 98<sup>th</sup> percentile of Ontario's typical range (Table 4), and the USEPA value of 3.5 ng  $\text{TEQ}\cdot\text{kg}^{-1}$  for rural background (USEPA 2000).

The background value of 4 ng TEQ·kg<sup>-1</sup> was determined from a limited number of studies and may not accurately reflect the ambient background concentration of PCDD/Fs in soils elsewhere in Canada. For example, ambient concentrations of PCDD/Fs in soils from a limited number of remote northern sites that were at least 20 km from human activity, ranged from non-detectable to 0.000 009 ng TEQ·kg<sup>-1</sup> (or 9 fg TEQ·kg<sup>-1</sup>). Therefore, the recommended ambient background concentration may not be applicable for northern regions.

Concentrations of PCDD/Fs in soils are higher in areas that receive known inputs as a result of human activities. For example, the concentration of PCDD/Fs in British Columbia soils impacted by pulp and paper mills was up to 255 ng TEQ·kg<sup>-1</sup> (dry weight) (Van Oostdam and Ward 1995). A concentration of 1124 ng TEQ·kg<sup>-1</sup> (dry weight) was reported in soils from Saint-Basile-Le-Grand, Quebec, following a PCB warehouse fire (Trépanier 1992). The highest soil concentrations of PCDD/Fs in soils near a northern abandoned military site, with known anthropogenic impacts, were 2.6 ng TEQ·kg<sup>-1</sup> (Grundy et al. 1997). Although these values are considerably lower than soil concentrations reported for more southern areas of the country, they are still more than 5 orders of magnitude higher than concentrations in other nonimpacted northern soils.

Dioxins and furans are also present in the air (Dann 1998), surface water (Van Oostdam and Ward 1995; Trépanier 1992), sediment (Trudel 1991), and biota (Langlois and Dubuc 1999; Ryan et al. 1986; Phaneuf et al. 1995; Elliot et al. 1996; Hebert et al. 1996; Champoux 1996). Human tissues and fluids also contain detectable amounts of dioxins and furans (Ryan 1985; Ryan et al. 1986; Cole et al. 1997; Ayotte et al. 1997; Dewailly et al. 1991, 1992; Craan et al. 1998). The concentration of PCDD/Fs in these media have been reviewed elsewhere (Environment Canada 2000; Sanexen 2000).

Table 3: Summary of PCDDs/PCDFs releases to all media in Canada (g TEQ\*/yr) (Environment Canada 2001)

Media	1990	1997	1999
Air	427	274	164
Effluent	454	3	3
Soil	19	19	19
TOTAL RELEASES	900	297	186
In Solid Waste	2633	173 <sup>†</sup>	1097

\* TEFs were not standardised for this analysis.

<sup>†</sup> Does not include amounts in pentachlorophenol (PCP).

Location	Mean (ng TEQ·kg <sup>-1</sup> ± SD)	Range (ng TEQ·kg <sup>-1</sup> )	Sample Size	Site Description	Reference
Ontario	$1.7^{a}$		74	rural parkland	OMOEE 1993
British Columbia	5.0 <sup>b</sup>	0.0 - 57.0	53	background	Van Oostdam and Ward 1995
Quebec	$10^{\circ} \pm 16.5$	0.0 - 99 <sup>d</sup>	57	background for semi-rural	Trépanier 1992

Table 4. Dioxin/furan ambient background soil concentrations in Canada (I-TEQ equivalents, dry weight)

<sup>a</sup> The OTR98 (98<sup>th</sup> percentile of the Ontario typical range) is equal to 4.8 ng TEQ·kg<sup>-1</sup>.

<sup>b</sup> Dwernychuk et al. (1991) reported a background mean value of 11.1 ng TEQ·kg<sup>-1</sup>, n=14; results were included in Van Oostdam and Ward (1995).

<sup>c</sup> Geometric mean = 4.4 ng TEQ·kg<sup>-1</sup>

<sup>d</sup> Detection limits were often high, non-detected values of each congener was set equal to half the detection limit.

This could result in an overestimate of actual background concentrations.

## Background Exposure of the Canadian Population

Canadians are exposed to low levels of PCDD/Fs predominantly through their diet, in particular through dietary intake of fish, meat and dairy products. Other exposure pathways include inhalation (PCDD/Fs are found in air in trace amounts in both particulates and vapours), inadvertent ingestion of soil, and absorption through the skin contacting air, soil, or water.

The average intake of PCDD/Fs by Canadians is estimated to range from 2.0 to 6.4 pg TEQ·kg<sup>-1</sup>·day<sup>-1</sup> over a lifetime, depending on location and lifestyle (Sanexen 2000). For example, some Canadians may have a higher rate of exposure compared to the general population as a result of workplace exposures or consumption of unusually high amounts of fish, meat, or dairy products containing elevated levels of PCDD/Fs. These estimates do not take into account the potential intake from smoking, which can significantly increase the total intake of dioxins and furans (Gilman et al. 1991).

#### **Environmental Fate and Behaviour**

Dioxins and furans are persistent in the environment. Photodegradation appears to be the only environmentally significant transformation, occurring mainly on nonsorbed species in the gaseous phase or at the soil-air and water-air interfaces. Hydrolysis, however, does not seem to occur (USEPA 1994).

Once released into the atmosphere, PCDD/Fs tend to adsorb onto particulates, and are removed from the atmosphere by photodegradation and dry and wet deposition. Half-lives of PCDD/Fs in the atmosphere vary from hours to days, depending on the congener, with lower chlorinated dioxins and furans being the main product. In general, the rate of photolysis increases as the degree of chlorination increases (USEPA 1994).

Once deposited on soil, there can be an initial loss of PCDD/Fs by photodegradation and/or volatilisation, with the extent of these processes dependent on several factors, including the exposure to sunlight, climatic conditions, and the form under which the PCDD/Fs were deposited (i.e., airborne particulates versus sludge). Photodegradation is limited to the soil surface, and is not a significant process below the first few millimetres (USEPA 1994). However, studies with octachlorinated dibenzo-p-dioxin have demonstrated that photodegradation acts through reductive dechlorination of the molecule, with chlorine preferentially removed from the 1, 4, 6, and 9 positions. This results in an increase in the concentration of 2,3,7,8-substituted congeners in relation to other congeners in the soil (Miller et al. 1989 and Tysklind et al. 1992, as cited in McLachlan et al. 1996), which may result in increased toxicity. In soil. PCDD/Fs exhibit a low potential for leaching (McLachlan et al. 1996) or volatilisation (USEPA 1994). As a result, in-place burial and erosion of soil to water bodies appears to be the predominant fate of PCDD/Fs sorbed to soil (USEPA 1994). Concentrations in soils may remain significant for long periods when surface runoff to water bodies and wind erosion are limited. PCDD/Fs tend to be resistant to degradation in the soil, with half-lives reported of greater than 10 years (McLachlan et al. 1996).

#### **Bioaccumulation and Biomagnification**

PCDD/Fs tend to bioaccumulate and/or bioconcentrate in living organisms. Bioconcentration refers to the direct uptake of compounds from water whereas bioaccumulation involves biological uptake of the contaminant from all compartments, including water, food and sediment (Branson et al. 1985; Muir et al. 1992). Bioconcentration is measured by calculating a bioconcentration factor (BCF), which is the ratio of the concentration of a substance in the organisms on a lipid basis to the concentration in water. Substances with BCFs in fish greater than 5000 are considered bioaccumulative according to the Federal Toxic Substance Management Policy (Environment Canada 1997).

BCFs for PCDD/Fs have been reported for a number of aquatic organisms. For example, BCFs ranged from 2 710 for 1,2,3,4,6,7,8-HpCDD in fathead minnow to 5 100 000 for 2,3,7,8-TCDD in medaka *(Oryzias latipes)* (Muir et al. 1985; Schmieder et al. 1995; BCFs recalculated on a lipid basis, Environment Canada 2000). BCFs for furans ranged from 21 400 to 240 000 (Loonen et al. 1994; Mehrle et al. 1988, recalculated on a lipid basis by Environment Canada 2000). Biota-sediment accumulation factors for PCDD/Fs (i.e., the ratio of the concentration of a substance in an organism on a lipid basis to the concentration in sediment organic carbon) are generally less than 1, indicating that the concentrations of PCDD/Fs in benthic biota is generally below that in the sediment (Environment Canada 2000).

A few studies have demonstrated that dioxins and furans can also accumulate in terrestrial organisms. BCFs for octachlorinated dibenzo-p-dioxin in carrots ranged from 0.07-0.99 (Schroll and Scheunert 1993). Studies have reported BAFs of 65-80 for TCDD in earthworms (Nash et al. 1980 as cited in Heida et al. 1986), and BAFs of less than 0.1 to 12 for 2,3,7,8-TCDD and a variety of furans in earthworms (Heida et al. 1986). For mammals, BAFs range from 0.05-40 for rats, cattle and monkeys administered 2,3,7,8-TCDD (Kobica et al. 1978; Kenaga 1980; Jensen et al. 1981; Bowman et al. 1985 as cited in Geyer et al. 1986; Parker et al. 1980; Firestone et al. 1979 as cited in Fries 1996). These BAFs indicate that generally the concentration of PCDD/Fs in terrestrial organisms is below, or only slightly elevated above, the concentration in the soil. Heida et al. (1986) suggested, however, that the ability of PCDD/Fs to bioaccumulate in the terrestrial ecosystem may be dependent on the soil matrix.

In aquatic systems, PCDD/Fs appear anomalous compared to other halogenated aromatic hydrocarbons in that they do not biomagnify up the food chain to an appreciable degree (Environment Canada 2000).

Limited data exist to determine if PCDD/Fs biomagnify in terrestrial systems, however it is expected that these compounds would behave in a similar manner as is observed for aquatic systems. Some evidence for potential biomagnification of PCDD/Fs in terrestrial systems has been reported. For example, a fugacity model of an agricultural food chain (air/soil $\rightarrow$ plant $\rightarrow$ cow's milk $\rightarrow$ human milk) predicted no net biomagnification through the system, but biomagnification was observed in the last link of the food chain (cow $\rightarrow$ human) (McLachlan 1996). Similarly, PCDFs biomagnified in voles at a contaminated site, especially 2,3,7,8-tetrachlorinated dibenzofuran and 2,3,4,7,8-pentachlorinated dibenzofuran (Heida et al. 1986).

# **Toxicity of PCDDs and PCDFs**

A multitude of toxic responses to PCDD/F exposure are described in the scientific literature. 2, 3, 7, 8-substituted PCDD/Fs are thought to elicit most of their toxicity via a mechanism involving the binding of dioxin-like compounds to the aryl hydrocarbon (Ah) receptor, a protein conserved across mammals, birds, and fish (Environment Canada 2000). The majority of the available toxicity data has been reported for 2,3,7,8-TCDD, considered the most toxic of the PCDD/Fs. However, PCDD/Fs are found in the environment in mixtures, therefore organisms respond to the cumulative exposure of Ah receptor-mediated chemicals, rather than exposure to any single dioxin-like compound.

There is a lack of data reporting the toxicity of dioxins and furans to plants, microbial processes of the soil, and invertebrates (Sanexen 2000). The toxicity of 2,3,7,8-TCDD to earthworms was evaluated in a single study, and the lethal level ranged from 5 to  $10 \text{ mg TCDD} \cdot \text{kg}^{-1}$  soil (Reinecke and Nash 1984).

A large range in toxic responses was observed in birds (Sanexen 2000). Studies with Bobwhite quail, mallard ducks, ringed turtle doves and chickens gave LD<sub>50</sub>s ranging from 15 000 to greater than 810 000 ng TEQ·kg<sup>-1</sup> for 2,3,7,8-TCDD or a mixture of dioxins (Hudson et al. 1984; Eisler et al. 1986; Greig et al. 1973). However, it should be noted that the age of the test species, the duration of exposure and the method of exposure varied among studies. Studies have also been conducted in which 2,3,7,8-TCDD was injected into eggs. LD<sub>50</sub>s of 122-240 ng TEQ·kg<sup>-1</sup> egg with chickens (Henshel et al. 1997: Allred Strange and 1977) and 1400-2100 ng TEQ·kg<sup>-1</sup> for ring-necked pheasants (Nosek et al. 1992) were obtained. Lowest observable adverse effect levels (LOAELs) for non-lethal endpoints ranged from 135-1000 ng 2,3,7,8-TCDD·kg<sup>-1</sup> for several species (Nosek et al. 1992; Hart et al. 1992; McKinney et al. 1976; Schewtz et al. 1973), however, Eastern bluebirds had a no observable adverse effect level (NOAEL) of

1000 ng TEQ·kg<sup>-1</sup> (Martin et al. 1989 cited in Nosek et al. 1992) which is at the top end of the range for the LOAELs.

A large data set is available on studies on the effect of 2,3,7,8-TCDD to mammalian species (Sanexen 2000). Boening (1998) noted that there is a large inter- and intra-species variability with mammals, with studies showing an 8 400 times difference in the oral  $LD_{50}$  between guinea pigs and hamsters, and a factor of 14 difference in the sensitivity of three strains of mice. Kenaga and Norris (1981) state that the toxicity of 2,3,7,8-TCDD is highly influenced by the carrier (i.e., solvent vs. soil) and the route of exposure.

In mammalian species, NOAELs for sublethal effects ranged from 0.000 7  $\mu$ g TCDD·kg<sup>-1</sup>·day<sup>-1</sup> for guinea pigs (DeCaprio et al. 1986) to 600  $\mu$ g TCDD·kg<sup>-1</sup>·day<sup>-1</sup> for hamsters (Henck et al. 1981). LOAELs for sublethal effects ranged from 0.000 12  $\mu$ g TCDD·kg<sup>-1</sup>·day<sup>-1</sup> for Rhesus monkeys (Schantz et al. 1992) to 1 000  $\mu$ g TCDD·kg<sup>-1</sup>·day<sup>-1</sup> for hamsters (Henck et al. 1981). The highest LOAEL was for lethality in beagle dogs at 3 000  $\mu$ g TCDD·kg<sup>-1</sup>·day<sup>-1</sup> (Schwetz et al. 1973).

Acute, high level human exposure to PCDD/F can cause chloracne, fluctuations in liver enzyme levels in the blood, pulmonary deficiency, possible hepatotoxicity, as well as effects on the central and peripheral nervous system. Although the evidence is somewhat less conclusive, some metabolic, cardiovascular, endocrine, reproductive or developmental responses may also be associated with elevated levels of exposure to PCDD/Fs. Any respiratory responses appear to be due to upper respiratory tract irritation, and not direct toxicity to the respiratory system (ATSDR 1998).

At low levels of exposure, as might be associated with the levels and concentrations of dioxins and furans seen in Canadian air, water, soil and food, concern centres on the potential for exposure to cause cancer. In the United States, 2,3,7,8-TCDD has recently been listed as a "Known to be Human Carcinogen" (NTP 2001). Other agencies have stated that a conclusive link between cancer in humans and PCDD/F exposure has not been established (IARC 1997; WHO 1998). The potential to cause cancer has been demonstrated in some animal species, through a mechanism that does not involve interaction with genetic material. The current position of several agencies is that there is some level of exposure (a threshold) below which cancer induction does not occur (IARC 1997; WHO 1998). Risk management is directed at keeping exposure below this apparent threshold for cancer induction.

# **Guidelines Derivation**

Canadian soil quality guidelines are derived for different land uses following the process outlined in CCME (1996) using different receptors and exposure scenarios for each land use (Table 1).

# Soil Quality Guidelines for Environmental Health

There were insufficient toxicological data available for vascular plants and soil invertebrates to derive soil quality guidelines for soil contact (SQG<sub>SC</sub>). A nutrient and energy cycling check could not be calculated due to a lack of toxicological data on microbial processes. Minimum data requirements also could not be met for the derivation of a soil and food ingestion guideline (SQG<sub>I</sub>). However, a provisional soil and food ingestion guideline for agricultural land use was calculated (Table 5). The provisional SQG<sub>I</sub> of 250 ng TEQ·kg<sup>-1</sup> was based on toxicity data from guinea pigs (Vos et al. 1973). Nonetheless, because an SQG<sub>SC</sub> could not be calculated, environmental soil quality guidelines (SQG<sub>E</sub>) could not be calculated for any of the four land uses.

# Soil Quality Guidelines for Human Health

As discussed above, the scientific community has not currently reached a consensus as to whether PCDD/Fs elicit threshold or non-threshold effects. In 1998, the WHO and ASTDR concluded that although possibly carcinogenic, PCDD/Fs are thought to elicit potential health effects by a non-genotoxic mechanism, and therefore a threshold effects model would be most appropriate. The USEPA (2000) rejected this decision, recommending a non-threshold effects model instead. The current assessments by Health Canada have assumed the existence of a threshold, therefore, in the derivation of the quality guidelines for human health (SQG<sub>HH</sub>) the threshold model was used.

A recent assessment by the World Health Organization has recommended a tolerable daily intake (TDI) for PCDD/Fs of 1 to 4 pg TEQ·kg<sup>-1</sup>·day<sup>-1</sup> (WHO 1998). Implications of this evaluation, as well as other ongoing assessments by the U.S. Environmental Protection Agency, the Joint FAO/WHO Expert Committee on Food Additives, and the Codex Alimentarius Commission, will be considered by Health Canada (R. Newhook 2001, Exposure Assessment / Existing Substances, Health Canada, pers. com.). The tolerable daily intake (TDI) value currently used by Health Canada for PCDD/Fs is 10 pg TEQ·kg<sup>-1</sup>·day<sup>-1</sup> (Health and Welfare Canada 1990). For threshold substances, the TDI is compared with the estimated daily intake (EDI).

For PCDD/Fs, infants and toddlers have EDIs at or above the TDI. Infants up to 6 months of age and infants/toddlers ranging in age from 6 months to 4 years have EDIs of 165 and 3.1 to 11 pg·kg<sup>-1</sup>·d<sup>-1</sup>, respectively (Gilman et al. 1991; Sanexen 2000). The elevated EDIs for infants are a result of the PCDD/F contamination in human breast milk leading to high short-term exposures for breast-fed infants (Gilman et al. 1991). The EDI for children between 5 and 19 can range from 1.3 to 5.0 pg·kg<sup>-1</sup>·d<sup>-1</sup>. Adults of 20 years and older have an estimated EDI of 0.56 to 2.1  $pg \cdot kg^{-1} \cdot d^{-1}$  (Gilman et al. 1991; Sanexen 2000). These EDIs may overestimate current exposure rates. The values from Gilman et al. (1991) are based on data from the early 1980's. Considerable exposure reduction has occurred since that time, and improved analytical methods have resulted in more accurate (and lower) estimates of exposure (G.M. Richardson 2001, Risklogic Scientific Services Inc., pers. com.). However, the estimates in Gilman et al. (1991) might also be higher if updated to include dioxin-like PCBs.

For agricultural, residential/parkland, and commercial lands, a toddler was chosen as the most sensitive receptor (as opposed to infants, who are unlikely to come in direct contact with contaminated soil). Since the EDI is greater than the TDI for toddlers, according to the CCME protocol, it is desirable to prevent or disallow any additional soil contamination above current background levels. Therefore, the Preliminary Soil Quality Guidelines for Human Health (PSQG<sub>HH</sub>) for PCDD/Fs are set at 4 ng TEQ·kg<sup>-1</sup> for agricultural, residential/parkland, and commercial land uses.

Industrial lands typically have limited or restricted access to the public, so occupational exposure of adults will predominate. A  $PSQG_{HH}$  was determined for an adult receptor under the exposure scenarios given by the CCME (1996) for industrial sites. The  $PSQG_{HH}$  in this case is 175 ng TEQ·kg<sup>-1</sup>.

An off-site migration check was also calculated for industrial land use. This check is set at a background concentration of 4 ng TEQ·kg<sup>-1</sup> because any increase above this concentration in industrial soils could result in unacceptable degradation of adjacent residential/parkland or commercial soils through soil erosion.

A groundwater check value is normally calculated, according to the protocol (CCME 1996), to determine concentrations of PCDD/Fs in soil which would not cause

an exceedance of groundwater concentrations above the Canadian Drinking Water Quality Guideline. For PCDD/Fs, there is no Canadian Drinking Water Quality Guideline, and an acceptable drinking water concentration cannot be estimated because the EDI for toddlers exceeds the TDI. Therefore, this check could not be calculated.

# Soil Quality Guidelines for Dioxins and Furans

The final SQG for dioxins and furans for all four land uses is 4 ng TEQ·kg<sup>-1</sup>. Because the EDI is greater than the TDI for sensitive human receptors, the mean Canadian background soil concentration is recommended to minimise any further exposure to these compounds on agricultural, residential/parkland, and commercial lands. On industrial land, the mean background soil concentration is recommended to prevent degradation of adjacent lands due to off-site migration.

The SQGs for PCDD/Fs are considered to be management levels, rather than levels that are protective of human or environmental health, because they are not effects-based. However, due to the conservative nature of the TDI and EDI values and of the guideline derivation protocol, risks associated with ambient levels are considered to be minimal.

# Implementation of the Soil Quality Guidelines for Dioxins and Furans

Dioxins and furans are present in the environment primarily as a result of human activities. These compounds are persistent and at elevated levels are known to be toxic to humans and other organisms. They are subject to long-range atmospheric transport and have been measured in soils remote from human activities. In general, humans are exposed to low levels of dioxins and furans, primarily through food.

The analysis presented in this document indicates that estimated daily exposures to PCDD/Fs from all exposure pathways is at or above the tolerable daily intake for sensitive human receptors (toddlers). In addition, there is some evidence to suggest that in terrestrial food chains, selective biomagnification of PCDD/Fs may occur. This may result in an increase in the risk of effects to environmental receptors at sites with ambient background concentrations of PCDD/Fs.

Consequently, the management of PCDD/Fs at contaminated sites should consider minimizing exposure to PCDD/Fs through all exposure pathways, including soil.

	Land use				
	Agricultural	Residential/ parkland	Commercial	Industrial	
Guideline	4 <sup>a</sup>	<b>4</b> <sup>a</sup>	4 <sup>b</sup>	4 <sup>c</sup>	
Human health guidelines/check values					
SQG <sub>HH</sub>	$NC^d$	$NC^d$	$NC^d$	4 <sup>e</sup>	
Soil ingestion guideline	NC	NC	NC	175 <sup>f</sup>	
Inhalation of indoor air check	$ND^{g}$	$ND^{g}$	$ND^{g}$	ND <sup>g</sup>	
Off-site migration check		—		4	
Groundwater check (drinking water)	$NC^{h}$	$NC^{h}$	$NC^{h}$	$NC^h$	
Produce, meat and milk check	$NC^{i}$	NC <sup>i</sup>	NC <sup>i</sup>	NC <sup>i</sup>	
SQG <sub>HH</sub> —provisional guidelines	$4^j$	4 <sup>j</sup>	4 <sup>j</sup>	$NC^k$	
Limiting pathway for SQG <sub>HH</sub> —provisional	Soil ingestion	Soil ingestion	Soil ingestion	ND	
Environmental health guidelines/check values					
SQG <sub>E</sub>	$NC^{l}$	$NC^{l}$	$NC^{1}$	$NC^{1}$	
Soil contact guideline	$NC^{m}$	NC <sup>m</sup>	$NC^m$	NC <sup>m</sup>	
Soil and food ingestion guideline	250	—		—	
Nutrient and energy cycling check	$NC^m$	$NC^m$	$NC^m$	NC <sup>m</sup>	
Off-site migration check			—	4	
Groundwater check (aquatic life)	NC <sup>n</sup>	NC <sup>n</sup>	NC <sup>n</sup>	NC <sup>n</sup>	
SQG <sub>E</sub> —provisional guidelines	NC	NC	NC	NC	
Limiting pathway for SQG <sub>E</sub> —provisional	ND	ND	ND	ND	
Interim Soil Quality Criteria (CCME 1991)	10	1000	no value	no value	

#### Table 5. Soil quality guidelines and check values for dioxins and furans (ng TEQ·kg<sup>-1</sup>)

**Notes:** NC = not calculated; ND = not determined;  $SQG_E$  = soil quality guideline for environmental health;  $SQG_{HH}$  = soil quality guideline for human health. The dash indicates a guideline/check value that is not part of the exposure scenario for this land use and therefore is not calculated.

<sup>a</sup>Data are sufficient and adequate to calculate only a provisional SQG<sub>HH</sub>. The provisional SQG<sub>HH</sub> is less than the interim soil quality criterion (CCME 1991), and thus becomes the soil quality guideline, which supersedes the interim soil quality guideline for this land use.

<sup>b</sup>Data are sufficient and adequate to calculate only a provisional SQG<sub>HH</sub>. An interim soil quality criterion (CCME 1991) was not established for this land use, therefore, the provisional SQG<sub>HH</sub> becomes the soil quality guideline.

<sup>c</sup>Data are sufficient and adequate to calculate only an SQG<sub>HH</sub>. An interim soil quality criterion (CCME 1991) was not established for this land use, therefore, the SQG<sub>HH</sub> becomes the soil quality guideline.

<sup>d</sup>Data are insufficient/inadequate to calculate an SQG<sub>HH</sub> for this land use. However, data are sufficient and adequate to calculate a provisional SQG<sub>HH</sub>. <sup>e</sup>The SQG<sub>HH</sub> is the lowest of the human health guidelines and check values.

<sup>f</sup>Calculated using the equations and parameters given by CCME (1996) for industrial sites based on an adult receptor.

<sup>g</sup>Exposure estimates determined for all 2,3,7,8-substituted congeners for PCDD/Fs indicate that ambient background concentrations of 4 ng·kg<sup>-1</sup> cannot produce indoor air concentrations that pose a significant health risk due to the low volatility of PCDD/Fs.

<sup>h</sup>Check value was not calculated as there is no drinking water quality guideline for PCDD/Fs.

<sup>i</sup>Not calculated because the PSQG<sub>HH</sub> is set to the ambient background concentration and therefore this pathway would only reflect ambient background conditions.

<sup>j</sup>Values default to ambient background concentrations since the EDI for the most sensitive receptor (toddlers) is or can be greater than the TDI. Therefore, no extra exposure above ambient background should be allowed.

<sup>k</sup>Because data are sufficient and adequate to calculate an SQG<sub>HH</sub>, a provisional SQG<sub>HH</sub> is not calculated.

<sup>1</sup>Insufficient/inadequate data to calculate a SQG<sub>E</sub> at this time.

<sup>m</sup>Data are insufficient/inadequate to calculate this pathway/check value.

<sup>n</sup>Check value was not calculated as there is no aquatic life water quality guideline for PCDD/Fs. Due to their low water solubilities, PCDD/Fs are not expected to be found in the water column to any significant degree.

#### Guidelines Based on Background Concentrations

For the reasons given in previous sections which indicate that exposure should be minimised, the final SQG for dioxins and furans recommended is the mean ambient background concentration for Canadian soils. Where background concentrations of PCDD/Fs are known to be significantly different from the mean national background level, such as in northern areas, local background concentrations may be used as the site-specific SQG.

## Other Dioxin-like Compounds

Biological systems respond to the cumulative exposure to Ah receptor-mediated chemicals, rather than the exposure to any single dioxin-like compound. Deriving soil quality guidelines for dioxins and furans aimed at the protection of human and environmental health should therefore take into account other organic compounds present in the environment that elicit their toxic effect through the same mechanism, namely the Ah receptor. Non-ortho and mono-ortho coplanar polychlorinated biphenyls (PCBs) are such compounds with dioxin-like, Ah receptor mediated effects, and thus should be identified and quantified concomitantly with PCDD/Fs in soil, and included in the TEQ concentration of dioxin-like compounds which is then compared to the guideline. TEFs have been developed for these PCB congeners (van den Berg et al. 1998), allowing for a total TEQ (including dioxins, furans and PCBs) to be calculated. Other polychlorinated organic compounds (e.g., naphthalenes, diphenyl ethers, xanthenes, anthracenes, fluorenes) as well as the brominated or chloro/bromosubstituted analogues of PCDD/Fs may also elicit dioxinlike responses; however, there is insufficient data at this time to determine appropriate TEF values for these compounds (van den Berg et al. 1998).

This adds additional considerations to sites that are contaminated with both PCDD/Fs and PCBs, or with PCBs exclusively, as there is currently a total PCB guideline (which is not based on dioxin-like effects) as well as a PCDD/F guideline which should include coplanar PCBs, when applicable. Both guidelines need to be satisfied when evaluating or remediating a site. Therefore, at a site, a total TEQ consisting of PCDD/Fs and coplanar PCBs should satisfy the PCDD/F guideline and the total PCB concentration (including all PCB congeners) should satisfy the total PCB guideline.

## Exposure Pathways

Calculated values for all possible operational pathways for the four land use types are given in Table 5. However, as described in CCME (1996), certain pathways may not be applicable to certain land uses (i.e., the produce, meat and milk check would not be applicable to an industrial site). While removal of any pathway would not change any of the final SQG values, it is yet important to remain aware of which pathways are operational at a site.

# *PCDD/F Contamination in Sewage and Industrial Sludge*

One method of disposal for sewage and industrial sludge is the direct application of the sludge to soils. While the high nutritive value of the sludge is the focus for its use as a soil amendment, sludge also contains low amounts of contaminants, including dioxins and furans. The application of sludge to soils thus results in the addition of PCDD/Fs to the soil. Considering the persistence of PCDD/Fs in soils, repeated application could lead to an increase in the concentration of these compounds in the soil.

The soil quality guidelines are not "pollute up to" levels for the environment, and were not developed for the purpose of determining acceptable levels of PCDD/Fs in soil resulting from the application of sludge. The development of acceptable levels for sludge application is beyond the scope of the environmental quality guidelines, and guidance must be obtained from the appropriate jurisdictional offices with regard to this.

#### References

- Allred, P.M., and J.R. Strange. 1977. The effects of 2,4,5-trichlorophenoxyacetic acid and 2,3,7,8-tetrachlorodibenzo-*p*-dioxin on developing chicken embryos. Arch. Environ. Contam. Toxicol. 6:483-489.
- ATSDR (Agency for Toxic Substances and Disease Registry). 1998. Toxicological profile for chlorinated dibenzo-*p*-dioxins (update). U.S. Department of Health and Human Services. 678 pp. + appendices.
- Ayotte, P., E. Dewailly, J.J. Ryan, S. Bruneau and G. Lebel. 1997. PCBs and dioxin-like compounds in plasma of adult Inuit living in Nunavik (Arctic Quebec). Chemosphere 34:1459-1468.
- Boening, D.W. 1998. Toxicity of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin to several ecological receptor groups: a short review. Ecotoxicol. Environ. Safety 39:155-163.
- Bowman, R.E. et al. Unpublished results as cited in: N.C. Weerasinghe and M.L. Gross. 1985. Origins of polychlorodibenzo-*p*-dioxins (PCDD) and polychlorodibenzofurans (PCDF) in the environment. <u>In</u>: Dioxins in the Environment. M.A. Kamrin and P.W. Rodgers (Eds). Hemisphere Publishing Corp., Washington, DC. pp 133-151.
- Branson, D.R., I.T. Takahashi, W.M. Parker and G.E. Blau. 1985. Bioconcentration kinetics of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin in

rainbow trout. Environ. Toxicol. Chem. 4:779-788.

- CCME. (Canadian Council of Ministers of the Environment). 1991. Interim Canadian environmental quality criteria for contaminated sites. CCME, Winnipeg.
- 1996. A protocol for the derivation of environmental and human health soil quality guidelines. CCME, Winnipeg. [A summary of the protocol appears in Canadian environmental quality guidelines, Chapter 7, Canadian Council of Ministers of the Environment, 1999, Winnipeg.]
- CEPA (Canadian Environmental Protection Act). 1999. Statutes of Canada, Chapter 33.
- Champoux, L. 1996. PCBs, dioxins and furans in Hooded Merganser (Lophodytes cucullatus), Common Merganser (Mergus merganser) and mink (Mustela vison) collected along the St. Maurice River near La Tuque, Quebec. Environ. Pollut. 92:147-153.
- Cole, D., J. Kearney, J.J. Ryan and A.P. Gilman. 1997. Plasma levels and profiles of dioxin and dioxin-like compounds in Ontario Great Lakes anglers. Chemosphere 34:1401-1409.
- Craan, A.G., and D.A. Haines. 1998. Twenty-five years of surveillance for contaminants in human breast milk. Arch. Environ. Contam. Toxicol. 3:702-710.
- Dann, T. 1998. Ambient air measurements of polycyclic aromatic hydrocarbons (PAH), polychlorinated dibenzo-*p*-dioxins (PCDD) and polychlorinated dibenzofurans in Canada (1987-1997). Report Series Number AAQD 98-3. Analysis and Air Quality Division, Environmental Technology Centre, Environment Canada, Ottawa.
- DeCaprio, A.P., D.N. McMartin, P.W. O'Keefe, R. Rej, J.B. Silkworth, and L.S. Kaminsky. 1986. Subchronic oral toxicity of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin in guinea pig: comparisons with a polychlorinated biphenyl-containing transformer fluid pyrolysate. Fund. Appl. Toxicol. 6:454-463.
- Dewailly, E., A. Nantel, S. Bruneau, C. Laliberté, L. Ferron and S. Gingras. 1992. Breast milk contamination by PCDDs, PCDFs and PCBs in Arctic Quebec: a preliminary assessment. Chemosphere 25:1245-1249.
- Dewailly, E., H. Tremblay-Rousseau, G. Carrier, S. Groulx, S. Gingras, K. Boggess, J. Stanley and J.P. Weber. 1991. PCDDs, PCDFs and PCBs in human milk of women exposed to a PCB fire and of women from the general population of the province of Quebec - Canada. Chemosphere. 23:1831-1835.
- Dwernychuk, L.W., G. Bruce and B. Gordon. 1991. Organochlorine contamination in various environmental compartments related to chemical / combustion sources: Lower Mainland / Vancouver Island / Skeena / Interior Regions. Prepared for BC Environment. ISBN 0-7726-1558-6.
- Eisler, R. 1986. Dioxin hazards to fish, wildlife, and invertebrates: a synoptic review. U.S. Department of Interior - Fish and Wildlife Service, Biological Report 85 (1.8); Contaminant Hazard Reviews Report No. 8, 37 pp.
- Elliot, J.E., L.K. Wilson, K.W. Langelier and R.J. Norstrom. 1996. Bald Eagle mortality and chlorinated hydrocarbon contaminants in livers from British Columbia, Canada, 1989-1994. Environ. Pollut. 94:9-18.
- Environment Canada. 1997. Toxic Substances Management Policy: Scientific justification, polychlorinated dibenzo-*p*-dioxins and polychlorinated dibenzofurans. Candidate substances for management under Track 1 of the Toxic Substances Management Policy. Environment Canada, Ottawa. 36 pp.
  - —. 2000. Canadian sediment quality guidelines for the protection of aquatic life and Canadian tissue residue guidelines for the protection of wildlife consumers of aquatic biota: Polychlorinated dibenzo-pdioxins and polychlorinated dibenzofurans (PCDD/Fs). Technical supporting document. 2 vols. National Guidelines and Standards Office, Environmental Quality Branch, Ottawa.
  - 2001. Inventory of releases of PCDDs/PCDFs. Up-dated edition. Environment Canada and the Federal/Provincial Task Force on

Dioxins and Furans. February 2001. National Office of Pollution Prevention, Hull, QC.

- Firestone, D., M. Clower, A.P. Borsetti, R.H. Teske and P.E. Long. 1979. Polychlorodibenzo-*p*-dioxin and pentachlorophenol residues in milk and blood of cows fed technical pentachlorophenol. J. Agric. Food Chem. 27:1171-1177.
- Fries, G.F. 1996. Ingestion of sludge applied organic chemicals by animals. Sci. Total Environ. 185:93-108.
- Geyer, H.J., I. Scheunert, J.G. Filser and F. Korte. 1986. Bioconcentration potential (BCP) of 2,3,7,8-tetrachlorodibenzo-pdioxin (2,3,7,8-TCDD) in terrestrial organisms including humans. Chemosphere 15:1495-1502.
- Gilman, A., R. Newhook, and B. Birmingham. 1991. An updated assessment of the exposure of Canadians to dioxins and furans. Chemosphere 23:1661-1667.
- Government of Canada. 1995. Toxic Substances Management Policy: Persistence and Bioaccumulation Criteria. Ottawa. 21 pp.
- Greig, J.B., G. Jones, W.H. Butler, and J.M. Barnes. 1973. Toxic effects of 2,3,7,8-tetrachlorodibenzo-p-dioxin. Food Cosmet. Toxicol. 11:585-595.
- Grundy, S.L., D.A. Bright, W.T. Dushenko, M. Dodd, S. Englander, K. Johnston, D. Pier, and K.J. Reimer. 1997. Dioxin and furan signatures in northern Canadian soils: correlation to source signatures using multivariate unmixing techniques. Chemosphere 34:1203-1219.
- Hart, L.E., K.M. Cheng, P.E. Whitehead, R.M. Shah, R.J. Lewis, S.R. Ruschkowski, R.W. Blair, D.C. Bennett, S.M. Bandiera, R.J. Norstrom, and G.D. Bellward. 1991. Dioxin contamination and growth and development in great blue heron embryos. J. Toxicol. Environ. Health 32:331-334.
- Health and Welfare Canada. 1990. Priority Substances List Assessment Report No. 1: Polychlorinated dibenzodioxins and polychlorinated dibenzofurans. ISBN 0-662-96003-3. Ottawa. 64 pp.
- 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. Sci. Total Environ. 185:195-204.
- Heida, H., K. Olie and E. Prins. 1986. Selective accumulation of chlorobenzenes, polychlorinated dibenzofurans and 2,3,7,8-TCDD in wildlife of the Volgermeerpolder, Amsterdam, Holland. Chemosphere 15:1995-2000.
- Henck, J.M., M.A. New, R.J. Kociba, and K.S. Rao. 1981. 2,3,7,8-tetrachlorodibenzo-p-dioxin: acute oral toxicity in hamsters. Toxicol. Appl. Pharmacol. 59:405-407.
- Henshel, D., B. Hehn, W. Ravenska, V. Mylinh, and J.D. Steeves. 1997. The relative sensitivity of chicken embryos to yolk- or air-cellinjected 2,3,7,8-tetrachlorodibenzo-p-dioxin. Environ. Toxicol. Chem. 16:725-732.
- Hudson, R.H., R.K. Tucker, and M.A. Haegele. 1984. Handbook of toxicity of pesticides to wildlife, 2<sup>nd</sup> edition. United States Department of the Interior, Fish and Wildlife Service. Resource Publication 153. Washington D.C. 91 pp.
- IARC (International Agency for Research on Cancer). 1997. IARC Monographs on the evaluation of carcinogenic risk to humans: polychlorinated dibenzo-para-dioxins and polychlorinated dibenzofurans, volume 69. ISBN 92 832 1269 X. Lyon, France. 666 pp.
- Jensen, D.J., R.A. Hummel, N.H. Mahle, C.W. Kocher and H.S. Higgins. 1981. A residue study of beef cattle consuming 2,3,7,8-tetrachlorodibenzo-p-dioxin. J. Agric. Food Chem. 29:265-268.
- Kenaga, E.E. 1980. Correlation of bioconcentration factors of chemicals in aquatic and terrestrial organisms with their physical and chemical properties. Environ. Sci. Technol. 14:553-556.
- Kenaga, E.E., and L.A. Norris. 1983. Environmental toxicity of TCDD. In: Human and environmental risks of chlorinated dioxins and

- related compounds. R.E. Tucker, A.L. Young and A.P. Gray (eds). Plenum Press, New York. pp. 277-299.
- Kobica, R.J., D.G. Keyes, J.E. Beyer, R.M. Carreon, C.E. Wade, A.D. Dittenber, R.P. Kalnins, L.E. Frauson, C.N. Park, S.D. Barnard, R.A. Hummel and C.G. Humiston. 1978. Results of a two-year chronic toxicity and oncogenicity study of 2,3,7,8-tetrachlorodibenzo-*p*dioxin in rats. Toxicol. Appl. Pharmacol. 46:279-303.
- Langlois, C. and N. Dubuc. 1999. Études de suivi des effets sur l'environnement (ESEE) des fabriques de pâtes et papiers: synthèse des résultats des 47 études réalisées au Québec dans le cadre du cycle 1. Environnement Canada. 66 pp. + appendices.
- Loonen, H., M. Tonkes, J.R. Parsons and H.A.J. Govers. 1994. Bioconcentration of polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans in guppies after aqueous exposure to a complex PCDD/PCDF mixture: relationship with molecular structure. Aquat. Toxicol. 30:153-169.
- Martin, S., J. Duncan, S. Thiel, R. Peterson, and M. Lemke. 1989. Evaluation of the effects of dioxin-contaminated sludges on Eastern bluebirds and tree swallows. Report prepared by Nekoosa Papers Inc., Part Edwards, Wisconsin.
- McKinney, J.D., K. Chae, N. Gupta, J.A. Moore, and J.A. Goldstein. 1976. Toxicological assessment of hexachlorobiphenyl isomers and 2,3,7,8-tetrachlorodibenzofuran in chicks. Toxicol. Appl. Pharmacol. 36:65-80.
- McLachlan, M.S. 1996. Bioaccumulation of hydrophobic chemicals in agricultural food chains. Environ. Sci. Technol. 30:252-259.
- McLachlan, M.S., M. Horstmann, and M. Hinkel. 1996. Polychlorinated dibenzo-p-dioxins and dibenzofurans in sewage sludge: sources and fate following sludge application to land. Sci. Total Environ. 185:109-123.
- Mehrle, P.M., D.R. Buckler, E.E. Little, L.M. Smith, J.D. Petty, P.H. Peterman, D.L. Stalling, G.M. De Graeve, J.J. Coyle and W.J. Adams. 1988. Toxicity and bioconcentration of TCDD and TCDF in rainbow trout. Environ. Toxicol. Chem. 7:47-62.
- Miller, G.C., V.R. Hebert, M.J. Miille, R. Mitzel and R.G. Zepp. 1989. Photolysis of octachlorodibenzo-p-dioxin on soils: production of 2,3,7,8-TCDD. Chemosphere. 18:1265-1274.
- Muir, D.C., A.L. Yarechewski and G.R. Webster. 1985. Bioconcentration of four chlorinated dioxins by rainbow trout and fathead minnows. Aquatic Toxicology and Hazard Assessment: Eighth Symposium, ASTM STP 891. American Society for Testing and Materials. pp. 440-454.
- Muir, D.C., W.L. Fairchild and D.M. Whittle. 1992. Predicting bioaccumulation of chlorinated dioxins and furans in fish near Canadian bleached kraft mills. Water Pollut. Res. J. Can. 37:487-507.
- Nash, R.G., and M.L. Beall. 1980. Distribution of silver, 2,4-D and TCDD applied to turf in chambers and field plots. J. Agric. Food Chem. 28:614-623.
- NATO/CCMS (North Atlantic Treaty Organization, Committee on the Challenges of Modern Society). 1988a. Pilot study on international information exchange on dioxins and related compounds. International Toxicity Equivalency Factor (I-TEF) method of risk assessment for complex mixtures of dioxins and related compounds. Report No. 176. 26 pp.
  - ——. 1988b. Scientific basis for the development of International Toxicity Equivalency Factor (I-TEF) method of risk assessment for complex mixtures of dioxins and related compounds. Report No. 178.
- Nosek, J.A., S.R. Craven, J.R. Sullivan, S.S. Hurley, and R.E. Peterson. 1992. Toxicity and reproductive effects of 2,3,7,-tetrachlorodibenzo-*p*-dioxin in ring-necked pheasant hens. J. Toxicol. Environ. Health 35:187-198.
- NTP (National Toxicology Program). 2001. 9th Report on Carcinogens, Revised January 2001. U.S. National Institute of Environmental

Health Sciences, National Toxicology Program. Research Triangle Park, N.C.

- OMOEE (Ontario Ministry of Environment and Energy). 1993. Ontario Typical Range of chemical parameters in soil, vegetation, moss bags and snow. Ontario Ministry of Environment and Energy. PIBS 2792, ISBN 0-7778-1979-1. Queen's Printer for Ontario. 212 pp. + appendices.
- Parker, C.E., W.A. Jones, H.B. Matthews, E.E. McConnell and J.R. Hass. 1980. The chronic toxicity of technical and analytical pentachlorophenol in cattle. II. Chemical analysis of tissues. Toxicol. Appl. Pharmacol. 55:359-369.
- Phaneuf, D., L. DesGranges, N. Plante and J. Rodrigue. 1995. Contamination of local wildlife following a fire at a polychlorinated biphenyls warehouse in St-Basile-Le-Grand, Quebec, Canada. Arch. Environ. Contam. Toxicol. 28:145-153.
- Reinecke, A.J., and R.G. Nash. 1984. Toxicity of 2,3,7,8-TCDD and short-term bioaccumulation by earthworms (Oligochaeta). Soil Biol. Biochem. 16:45-49.
- Ryan, J.J. 1985. Chlorinated dibenzo-p-dioxins and chlorinated dibenzofurans in Canadian human adipose tissue. Chemosphere 14:697-706.
- Ryan, J.J., B.P.Y. Lau and J.A. Hardy. 1986. 2,3,7,8-Tetrachlorodibenzo-p-dioxin and related dioxins and furans in snapping turtle (*Chelydra serpentina*) tissues from the upper St. Lawrence River. Chemosphere 15:537-548.
- Sanexen. 2000. Canadian Soil Quality Guidelines for Dioxins and Furans: Environment and Human Health - Supporting Document (Draft, August 2000). Sanexen Environmental Service Inc., Longueuil, Quebec. 128 pp.
- Schantz, S.L., S.A. Ferguson, and R.E. Bowman. 1992. Effects of 2,3,7,8-tetrachlorodibenzo-p-dioxin on behavior of monkey in peer groups. Neurotoxicol. Teratol. 14:433-446.
- Schmieder, P., D. Lothenbach, J. Tietge, R. Erickson and R. Johnson. 1995. [<sup>3</sup>H]-2,3,7,8-TCDD uptake and elimination kinetics of medaka (Oryzias latipes). Environ. Toxicol. Chem. 14:1735-1743.
- Schroll, R., and I. Scheunert. 1993. Uptake pathways of octachlorodibenzo-*p*-dioxin from soil by carrots. Chemosphere 26:1631-1640.
- Schwetz. B.A., J.M. Norris, G.L. Sparschu, V.K. Rowe, P.J. Gehring, J.L. Emerson, and C.G. Gerbig. Toxicology of chlorinated dibenzo-p-dioxins. Environ. Health Perspect. 5:87-99.
- Trépanier, J.P. 1992. Incendie de l'entrepôt de BPC de Saint-Basile-Le-Grand: Rapport d'interprétation des résultats d'analyses physicochimiques. Ministère de l'Environnement du Québec. 40 pp. + appendices.
- Trudel, L. 1991. Dioxins and furans in bottom sediments near the 47 pulp and paper mills using chlorine bleaching. Water Quality Branch, Inland Waters Directorate, Environment Canada. Ottawa. 88 pp. + appendices.
- Tysklind, M., A.E. Carey, C. Rappe and G.C. Miller. 1992. Photolysis of OCDF and OCDD on soil. Organohalogen Compounds 8:293-296.
- USEPA (U.S. Environmental Protection Agency). 1994. Estimating exposure to dioxin-like compounds - <u>Review Draft.</u> U.S. Environmental Protection Agency, Washington, DC. 3 volumes.
- 2000. Exposure and human health reassessment of 2,3,7,8tetrachlorodibenzo-p-dioxin (TCDD) and related compounds. Draft final. Exposure Assessment and Risk Characterization Group. National Center for Environmental Assessment - Washington Office. Office of Research and Development. EPA/600/P-00/00/Ac.
- van den Berg, M., L. Birnbaum, B.T.C. Bosveld, B. Brunström, P. Cook, M. Feeley, J.P. Giesy, A. Hanberg, R. Hasegawa, S.W. Kennedy, T. Kubiak, J.C. Larsen, F.X. Rolaf van Leeuwen, A.K.D. Liem, C. Nolt, R.E. Peterson, L. Poellinger, S. Safe, D. Schrenk, D. Tillitt, M. Tysklind, M. Younes, F. Waern, and T. Zacharewski. 1998. Toxic

equivalency factors (TEFs) for PCBs, PCDDs, PCDFs for humans and wildlife. Environ. Health Perspect. 106:775-792.

- van Oostdam, J.C., and J.E.H. Ward. 1995. Dioxins and furans in the British Columbia environment. BC Environment, Environmental Protection Department, Victoria, BC. 28 pp. + appendices.
- Vos, J.G., J.A. Moore and J.G. Zinkl. 1973. Effect of 2,3,7,8tetrachlorodibenzo-p-dioxin on the immune system of laboratory animals. Environ. Health Perspect. 5:149-162.
- World Health Organization (WHO). 1998. Assessment of the health risk of dioxins: re-evaluation of the Tolerable Daily Intake (TDI). WHO Consultation, May 25-29 1998, Geneva, Switzerland.

Reference listing:

Canadian Council of Ministers of the Environment. 2002. Canadian soil quality guidelines for the protection of environmental and human health: Dioxins and Furans. In: Canadian environmental quality guidelines, 1999, Canadian Council of Ministers of the Environment, Winnipeg.

For further scientific information, contact:

Environment Canada National Guidelines and Standards Office 351 St. Joseph Blvd. Hull, QC K1A 0H3 Phone: (819) 953-1550 Facsimile: (819) 953-0461 E-mail: ceqg-rcqe@ec.gc.ca Internet: http://www.ec.gc.ca

© Canadian Council of Ministers of the Environment 1999 Excerpt from Publication No. 1299; ISBN 1-896997-34-1 For additional copies, contact:

CCME Documents Toll Free: 1-800-805-3025 www.ccme.ca

Aussi disponible en français.