Ecosystem Health

Science-Based Solutions

National Science Assessment on Dioxins and Furans in the Canadian Aquatic Environment

Report No. 1-5

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Science-Based Solutions
Solutions fondées sur la science

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NOTE TO READERS

The *Ecosystem Health: Science-based Solutions* series is dedicated to the dissemination of scientific knowledge, information and tools for monitoring, assessing, and reporting on ecosystem health to support Canadians in making sound decisions. Documents published in this series include the scientific basis, methods, approaches and frameworks for environmental guidelines and their implementation; monitoring, assessing, and rehabilitating environmental quality in Canada; and, indicator development, environmental reporting and data management. Issues in this series are published *ad libitum*.

This particular issue provides a general overview of the current understanding of dioxins and furans in the Canadian aquatic environment and is based largely on the scientific supporting documents to the Canadian Environment Quality Guidelines (sediment, tissue, soil) for polychlorinated dibenzo-*p*-dioxins and polychlorinated dibenzo furans. For additional information regarding this document, please contact:

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ABSTRACT

Polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) are commonly known as dioxins and furans, respectively. This assessment documents the current status of PCDD/Fs in Canada's aquatic environment, including their fate and behaviour, environmental levels, environmental health hazards, and control measures. PCDD/Fs are planar tricyclic aromatic hydrocarbons, having one to eight chlorine atoms. There are a total of 75 PCDD and 135 PCDF congeners; this assessment focuses on the 7 PCDD and the 10 PCDF congeners that have chlorine atoms attached in at the 2,3,7, and 8 lateral positions. These congeners are the most toxic and best studied. Dioxins and furans typically exhibit low solubility in water, very low vapour pressure, and high octanol/water partition coefficient.

Canadian surface waters may be contaminated with dioxins and furans generated as by-products of controlled or accidental combustion, or as impurities in the manufacturing of various chemical products, metal processing, and pulp and paper production. They can be formed naturally through combustion (i.e., forest fires, volcanoes), although these sources release relatively some amounts of PCDD/Fs compared to anthropogenic sources. Releases of PCDD/Fs to the Canadian environment from anthropogenic sources are tracked by the National Pollutant Release inventory, as mandated under the *Canadian Environmental Protection Act*. From 1990 to 1999, the total amount of PCDD/Fs released into air, water, soil decreased 79%, from 900 to 186 $g \cdot a^{-1}$, expressed as I-TEQs. Most PCDD/Fs (88%) are released into the air. Fifty-two percent or 85 $g \cdot a^{-1}$ of the emissions to air come from waste incineration, and over half of that amount comes from conical burners used in Newfoundland.

Once released into the atmosphere, PCDD/Fs tend to adsorb to particulates, and are subject to photodegradation, and wet and dry deposition. PCDD/Fs are much less mobile (up to 810 km) than other persistent organic pollutants (up to 110 000 km), and deposits decline rapidly with distance from the source. An estimated 12 tonnes of PCDD/Fs are deposited to the Earth's land surfaces, and an estimated 1 tonne to the world's oceans through atmospheric deposition. The most important fate process for PCDD/Fs released directly (e.g., effluent) or indirectly (e.g., atmospheric deposition) into surface water is adsorption to organic matter. In relation to toxicity, this fate process is important because it decreases the bioavailability of PCDD/Fs in the water column. PCDD/Fs bound to organic matter are then deposited onto the bed sediments, making sediments a major sink for PCDD/Fs that enter the water column. PCDD/Fs may persist in natural freshwater and marine sediments for long periods, especially in sediments below the biologically active layer; however, many burrowing benthic organisms (e.g., clams, tubificid worms, polychaetes) may mix surficial sediments with deeper materials.

Dioxins and furans elicit toxic responses in mammals, fish and birds by binding to a specific cellular protein called the arylhydrocarbon receptor (AhR). Although the mode of action is not completely understood, dioxins and furans may interfere with the expression and/or repression of multiple genes, and ultimately affect steroid synthesis and detoxifying mechanisms. Aquatic and semi-aquatic organisms may be exposed to PCDD/Fs through direct contact with water and sediment, and through the consumption of contaminated food. In general, exposure from water may be most important for lower trophic level species (e.g., guppies), whereas consumption of contaminated prey species is most important for large, piscivorous aquatic (*e.g.*, lake trout), mammalian (*e.g.*, mink), and avian species. For species in direct contact with bed sediments (*e.g.*, benthic invertebrates, and carp), this medium represents the primary route of exposure. All 2,3,7,8-substituted PCDD/Fs readily concentrate in lipid-rich tissues of aquatic and semi-aquatic animals, with the lower chlorinated PCDD/Fs bioaccumulating, in general, to a greater degree than the higher chlorinated congeners. Bioconcentration factors (BCFs) recorded for

2,3,7,8-TCDD are the highest of all congeners, ranging from 51 300 to 1 700 000 for Canadian species. Biota-sediment accumulation factors (BSAFs) for 2,3,7,8-TCDD range from 0.03 to 0.93. PCDD/Fs seem unusual in that they do not appear to biomagnify like other halogenated aromatics with comparable hydrophobicities (*e.g.*, PCBs). The greatest biomagnification factors (BMFs) reported were 32 and 76 for herring gulls and mink, respectively.

Adverse biological effects associated with concentrations of 2,3,7,8-substituted PCDD/Fs in sediment include changes in benthic invertebrate abundance, and weight and length are the most common indicators of adverse biological effects. Less frequently observed endpoints include deformities and proportion of sexually mature individuals. In fish, the most commonly observed effects are early life stage mortality, associated lesions (*e.g.*, hemorrhaging and oedematous symptoms), and fin necrosis. Toxic effects that have been commonly documented in mammals and birds include mortality (often delayed), decreased body weight gain, decreased feed consumption, thymic atrophy, histopathologic effects, immunotoxicity, developmental and reproductive effects (including endocrine disruption), biochemical effects, neurotoxicity, and carcinogenesis. Effects observed vary with a number of factors including the dose of the toxic substance, the congener tested, and life stage, strain, species, and gender of the organisms tested. Moreover, the sensitivity of an organism to toxicants depends in part on its capability to metabolise and eliminate these substances. In sediments, adverse effects have been reported in benthic invertebrates at concentrations as low as 1.9 $ng \cdot kg^{-1}$ dw on a TEQ basis, although adverse effects are observed most commonly at concentrations above 231 $ng \cdot kg^{-1}$ dw on a TEQ basis. Rainbow trout appear to be the most sensitive species to PCDD/F exposure through water. On a tissue residue basis, lake trout are nearly as sensitive as rainbow trout to PCDD/F exposure when mortality is compared as the endpoint. Nonetheless, the lowest-observable (adverse) effect level (LOAEL) of 0.038 $ng \cdot L^{-1}$ of TEQ_{fish} for growth (after 28 days of exposure) and for mortality (for 28 days of exposure and 28 days of depuration) in juvenile rainbow trout is the most sensitive endpoint found in the literature as a result of water-borne exposure to PCDD/Fs. Similarly, the most sensitive LOAEL based on a nominal tissue dose/measured tissue residue is 0.027 $\mu g \cdot kg^{-1}$ ww for 2,3,7,8-TCDF. The majority of mammalian toxicity studies have been performed on mice, rats, and guinea pigs. Few data exist on the acute toxicity of 2,3,7,8-TCDD to wildlife species that consume aquatic biota; however, mink may be among the most sensitive species with a LD_{50} of 4200 $ng \cdot kg^{-1}$ bw. Non-lethal effects include decreased body weight gain and food consumption. Impaired reproduction occurred in mink consuming diets of contaminated fish; PCDD/Fs levels in the diets were up to 10.2 $ng \cdot kg^{-1}$ $bw \cdot d^{-1}$ on a TEQ_{mam} basis. At this high dose, females gave birth to fewer young and all young were either stillborn or dead within one day. At lower concentrations, young weighed less, and were more likely to die within six weeks of age. Population declines and physiological abnormalities among other effects are indicators of potential organochlorine exposure in birds, particularly among piscivorous birds. These effects have been extensively studied in the Great Lakes region and on the western Canadian coast. The relatively recent appearance of symptoms of PCDD/F toxicity may be due to declines in other organochlorine compounds, most notably DDT and its metabolite DDE. Moreover, embryonic effects typically associated in dioxin toxicity were not routinely monitored until the 1980s. Nevertheless, effects associated with dioxin toxicity have clearly lessened in recent years, correlating well to reductions in PCDD/F levels. For example, chick oedema disease, characterised by jelly-like subcutaneous oedema on the breast was diagnosed in great blue heron chicks collected from British Columbia in 1988 but not 1991. This observation corresponds to a significant decrease in PCDD/F levels in heron eggs at the site between the two years (from ~530 to 100 $ng \cdot kg^{-1}$ ww on a TEQ basis). A similar trend was noted for herons collected in Vancouver, BC from 1988 to 1992.

There are numerous federal, national, provincial and territorial control measures, policies, and agreements, directly or indirectly addressing dioxins and furans in the Canadian aquatic environment. Significant improvements have been observed in many locations after the implementation of these control measures, however, toxic effects related to PCDD/F concentrations are still noticeable in some areas. More data collection, research and work could be put forward to allow for a more stringent and consistent control of PCDD/F levels in the country.

In recent years, a number of specific issues related to PCDD/F levels and their environmental health consequences have arisen. These range from concentrations of PCDD/Fs in waste sediments at pulp and paper mills to new waste incineration technologies and bioassay advances. This document also explores the scientific underpinnings and management considerations for these emerging issues.

RÉSUMÉ

Les polychlorodibenzo-*p*-dioxines (PCDD) et les polychlorodibenzofuranes (PCDF) sont généralement appelés respectivement dioxines et furanes. La présente évaluation donne des renseignements sur la situation actuelle concernant les PCDD/F dans l'environnement aquatique canadien, y compris leur devenir et comportement, leurs concentrations, les risques environnementaux pour la santé et les mesures de décontamination. Les PCDD/F sont des hydrocarbures aromatiques tricycliques plans, renfermant un à huit atomes de chlore. Il existe en tout 75 congénères de PCDD et 135 congénères de PCDF; la présente évaluation porte sur les 7 congénères de PCDD et les 10 de PCDF qui ont des atomes de chlore en positions latérales 2,3,7 et 8. Ces congénères sont les plus toxiques et les mieux étudiés. Les dioxines et les furanes possèdent généralement une faible hydrosolubilité, une très faible pression de vapeur, et un coefficient de partage octanol-eau élevé.

Les eaux de surface canadiennes peuvent être contaminées par des dioxines et des furanes qui se forment comme produits secondaires lors de certaines combustions contrôlées ou accidentelles, ou encore comme impuretés lors de la fabrication de divers produits chimiques, de la transformation de métaux ou encore de la production de pâtes et papier. Ils peuvent également être formés par combustion naturelle (p. ex. feux de forêt, volcans), sources qui rejettent des quantités relativement faibles de PCDD/F, comparativement aux sources anthropiques. Les rejets de PCDD/F dans l'environnement canadien par les sources anthropiques sont répertoriés dans l'Inventaire national des rejets de polluants, en vertu de la *Loi canadienne sur la protection de l'environnement*. De 1990 à 1999, la quantité totale de PCDD/F rejetée dans l'air, l'eau et le sol a baissé de 79 %, soit de 900 à 186 $g \cdot an^{-1}$, en TEQ-I. La plupart des PCDD/F (88 %) sont rejetés dans l'air. Cinquante-deux pour cent ou 85 $g \cdot an^{-1}$ des émissions dans l'air proviennent de l'incinération de déchets, et plus de la moitié de ce montant est produit par les fours coniques employés à Terre-Neuve.

Une fois rejetés dans l'atmosphère, les PCDD/F ont tendance à être adsorbés à des particules et sont soumis à un processus de photodégradation ainsi que de dépôt sec et humide. Les PCDD/F sont beaucoup moins mobiles (jusqu'à 810 km) que les autres polluants organiques persistants (jusqu'à 110 000 km), et les dépôts diminuent rapidement avec la distance à la source. On estime que le dépôt atmosphérique de PCDD/F est de 12 tonnes sur la surface terrestre de la Terre, et de 1 tonne dans les océans. Le processus le plus important du devenir des PCDD/F rejetés directement (p. ex. effluents) ou indirectement (p. ex. dépôt atmosphérique) à la surface de l'eau est l'adsorption à la matière organique. Ce processus est important pour la toxicité, car il réduit la biodisponibilité des PCDD/F dans la colonne d'eau. Les PCDD/F fixés à la matière organique se déposent ensuite sur les sédiments du lit, ce qui fait de ces derniers un important puits pour les PCDD/F qui entrent dans la colonne d'eau. Les PCDD/F peuvent persister pendant de longues périodes dans les eaux douces naturelles et les sédiments marins, particulièrement dans ceux situés en-dessous de la couche biologiquement active; cependant, beaucoup d'organismes benthiques fouisseurs (p. ex. myes, tubificidés, polychètes) peuvent mélanger les sédiments superficiels avec des matériaux plus profonds.

Les dioxines et les furanes produisent des effets toxiques chez les mammifères, les poissons et les oiseaux en se liant à une protéine cellulaire spécifique, un récepteur d'hydrocarbures aryliques (RHA). Bien que le mode d'action ne soit pas pleinement élucidé, il est possible que les dioxines et les furanes interfèrent avec l'expression et (ou) la répression de gènes multiples et finissent par affecter la synthèse des stéroïdes et les mécanismes de détoxification. Les organisme aquatiques et semi-aquatiques peuvent être exposés aux PCDD/F par contact direct avec l'eau et les sédiments et par consommation d'aliments contaminés. De façon générale,

Fl'eau est probablement la voie d'exposition la plus importante pour les espèces du niveau trophique inférieur (p. ex. le guppy), alors que c'est la consommation d'espèces proies pour les grandes espèces piscivores aquatiques(p. ex. touladi), mammaliennes (p. ex. vison) et aviennes. Dans le cas des espèces en contact direct avec les sédiments du lit (p. ex. invertébrés benthiques et carpes), ces derniers constituent la principale voie d'exposition. Tous les PCDD/F avec substitution en 2,3,7,8 s'accumulent facilement dans les tissus riches en lipides des animaux aquatiques et semi-aquatiques, les PCDD/F les moins chlorés s'accumulant généralement davantage que les congénères plus chlorés. Les facteurs de bioconcentration (FBC) déterminés pour la 2,3,7,8-TCDD sont les plus élevés de tous les congénères; ils se situent dans une plage de 51 300 à 1 700 000 pour les espèces canadiennes. Les facteurs d'accumulation biote-sédiments (FABS) pour la 2,3,7,8-TCDD varient de 0,03 à 0,93. Les PCDD/F ont cela de particulier qu'ils ne semblent pas faire l'objet d'une bioaccumulation comparable à celle d'autres composés aromatiques halogénés possédant des hydrophobicités semblables (p. ex. PCB). Les facteurs de bioamplification (FBA) les plus élevés étaient de 32 et 76 respectivement pour le goéland argenté et le vison.

Parmi les effets biologiques nocifs attribuables à des accumulations de PCDD/F avec substitution en 2,3,7,8 dans les sédiments, il y a les changements dans l'abondance, la masse et la longueur des invertébrés benthiques, qui sont les indicateurs les plus couramment employés pour évaluer ces effets. Comme valeurs de référence toxicologique moins fréquemment utilisées, on peut citer les difformités et la proportion d'individus qui sont des géniteurs mûrs. Chez les poissons, les effets les plus fréquemment observés sont le taux de mortalité à un stade précoce de la vie, les lésions connexes (p. ex. hémorragies et symptômes oedémateux), et la nécrose des nageoires. Parmi les effets toxiques dont font fréquemment état les documents sur les mammifères et les oiseaux , il y a la mortalité (souvent différée), le ralentissement du gain de masse corporelle, la réduction de la consommation alimentaire, l'atrophie du thymus, les effets histopathologiques, l'immunotoxicité, les effets sur le développement et la reproduction (y compris la perturbation du système endocrinien), les effets biochimiques, la neurotoxicité et le pouvoir cancérigène. Les effets observés varient en fonction de divers facteurs, notamment la dose de substance toxique, la nature du congénère testé, le stade de vie, la souche, l'espèce et le sexe de l'organisme testé. De plus, la sensibilité d'un organisme à des substances toxiques dépend en grande partie de sa capacité à métaboliser et à éliminer ces substances. Dans les sédiments, on a signalé des effets nocifs chez des invertébrés benthiques à des concentrations aussi faibles que 1,9 $ng \cdot kg^{-1}$ m. s., en EQT; mais, les effets nocifs sont le plus souvent observés à des concentrations supérieures à 231 $ng \cdot kg^{-1}$ m. s. , en EQT. La truite arc-en-ciel semble être l'espèce la plus sensible à l'exposition aux PCDD/F dans l'eau. Si on se base sur les résidus tissulaires, le touladi est presque aussi sensible que la truite arc-en-ciel à l'exposition aux PCDD/F lorsqu'on utilise la mortalité comme valeur de référence toxicologique. Mais c'est le SENO (seuil avec effet nocif observable), soit 0,038 $ng \cdot L^{-1}$, en $EQT_{poisson}$ pour la croissance (après 28 jours d'exposition) et pour la mortalité (28 jours d'exposition et 28 jours de dépuration) d'alevins de truite arc-en-ciel, qui est la valeur de référence toxicologique la plus sensible retenue dans la documentation traitant des conséquences de l'exposition aux PCDD/F dans l'eau. De la même façon, le SENO le plus sensible, si on se base sur une valeur nominale dose tissulaire/résidus tissulaires mesurés, est de 0,027 $\mu g \cdot kg^{-1}$ m. h. pour le 2,3,7,8-TCDF. La plupart des études de toxicité sur les mammifères ont été effectuées avec des souris, des rats et des cobayes. Il existe peu de données concernant la toxicité aiguë de la 2,3,7,8-TCDD pour les espèces sauvages se nourrissant dans le biote aquatique; cependant, il semble que le vison figure parmi les espèces les plus sensibles, avec une DL_{50} de 4200 $ng \cdot kg^{-1}$ m. c. Parmi les effets non léthaux, il y a le ralentissement du gain de masse corporelle et une baisse de la consommation d'aliments. La reproduction a été affectée chez des visons consommant des aliments à base de poisson

contaminé; les teneurs de PCDD/F des aliments atteignaient 10,2 $ng \cdot kg^{-1}$ m. c. par jour, en $EQT_{mam.}$. À cette valeur élevée de dose, les femelles ont donné naissance à moins de petits, et toute la progéniture était soit mort-née, soit morte en l'espace d'une journée. À des concentrations plus faibles, les petits pesaient moins et couraient davantage le risque de mourir avant d'atteindre l'âge de six semaines. Parmi les divers effets, la diminution de la population et les anomalies physiologiques sont des indicateurs de l'exposition potentielle d'oiseaux aux composés organochlorés, particulièrement chez les oiseaux piscivores. Ces effets ont été largement étudiés dans la région des Grands Lacs et sur la côte ouest du Canada. L'apparition relativement récente de symptômes dus à la toxicité des PCDD/F pourrait être attribuable aux baisses d'autres composés organochlorés, particulièrement du DDT et de son métabolite, le DDE. De plus, les effets embryonnaires, généralement associés à la toxicité des dioxines, n'ont fait l'objet d'un suivi régulier que depuis les années 1980. Cependant, les effets attribuables à la toxicité des dioxines ont nettement diminué ces dernières années, ce qui concorde bien avec les réductions des concentrations de PCDD/F. Par exemple, la maladie de l'oedème du poulet, caractérisée par un oedème sous-cutané gélatineux sur la poitrine, a été diagnostiquée chez les oisillons du grand héron (*Ardea herodias*), recueillis en Colombie-Britannique en 1988, mais non en 1991. Cette observation correspond à une diminution significative des concentrations de PCDD/F dans les œufs de hérons au même site entre les deux années spécifiées (soit de ~ 530 à 100 $ng \cdot kg^{-1}$ m. h. , en EQT). Une tendance similaire a été notée chez les hérons recueillis à Vancouver (C.-B.) de 1988 à 1992.

Il existe de nombreuses mesures, politiques et ententes pour la lutte antipollution, aux niveaux fédéral, national, provincial et territorial, visant directement ou indirectement les dioxines et les furanes présents dans l'environnement aquatique canadien. Des progrès significatifs ont été constatés dans beaucoup d'endroits après la mise en œuvre de ces mesures antipollution; cependant, dans certaines régions, on peut encore déceler des effets toxiques attribuables aux concentrations de PCDD/F. L'obtention de données supplémentaires ainsi que des travaux complémentaires de recherche ou autres pourraient permettre de réduire de façon plus drastique et systématique les concentrations de PCDD/F au pays.

Ces dernières années, un certain nombre de questions spécifiques, liées aux concentrations de PCDD/F et à leurs conséquences pour la santé de l'environnement, ont été soulevées. Ces préoccupations vont des concentrations de PCDD/F dans les sédiments résiduaires des usines de pâtes et papier jusqu'aux nouvelles technologies d'incinération de déchets et au secteur de pointe des bioessais. Le présent document explore également les fondements scientifiques et les considérations relatives à la gestion pour ces nouveaux enjeux.

1. INTRODUCTION

Polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) are commonly known as dioxins and furans. As a class of compounds, PCDDs and PCDFs (henceforth abbreviated as PCDD/Fs) were declared "toxic" substances as defined in Section 11 of the *Canadian Environmental Protection Act*. Since that time, a plethora of management programs and policies have resulted collectively in the decline of emissions and environmental levels, and subsequently improved the health of much of Canada's aquatic environment. The purpose of this national science assessment is to document the current status of dioxins and furans in Canada's aquatic environment, in recognition of Canada's successful management of dioxins and furans in Canada. Notwithstanding, residual levels of dioxins and furans in the environment continue to affect the health aquatic organisms and wildlife that eat aquatic life. And, for this reason, continued investment in research and development of new investigative techniques and remediation technologies, for example, is required.

This national assessment was written with the intent to serve as an overview of PCDD/Fs in the environment for those unfamiliar with these substances and as a reference tool for those who know these substances well. It is based largely on the scientific supporting documents to the Canadian Environmental Quality Guidelines for PCDD/Fs (Environment Canada 2000b, c), with updated information and data wherever available. Production, sources, and pathways for entering the environment are detailed; levels of PCDD/Fs in the tissues of biota in Canada are summarised in tables to facilitate comparisons among regions and species. Temporal declines in levels of PCDD/Fs are highlighted. In addition, available data on the environmental fate and persistence of these substances are evaluated and summarized. Both the historical and current environmental health hazards posed by these chemicals are assessed. Lastly, a comprehensive review of the control measures in Canada is provided.

2. PHYSICO-CHEMICAL PROPERTIES OF PCDD/FS

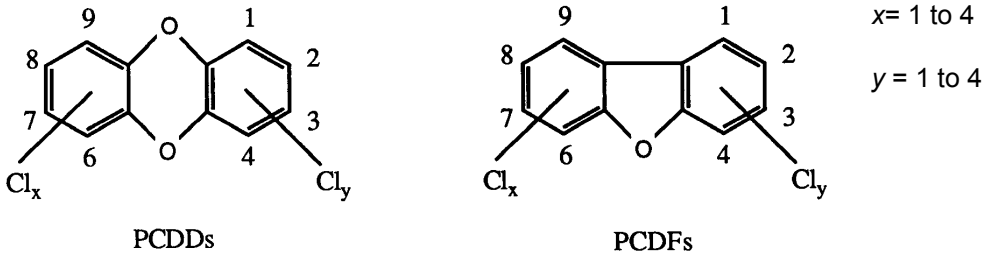
Polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) are planar tricyclic aromatic compounds that are highly persistent, have a strong tendency to accumulate in biological tissues and have a strong affinity for organic carbon in sediments (Government of Canada 1990). There are a total of 75 PCDD and 135 PCDF congeners (Table 2.1); the most studied of which is 2,3,7,8-tetrachloro-*p*-dibenzodioxin (2,3,7,8-TCDD) (Government of Canada 1990).

Table 2.1. Possible number of positional PCDDs and PCDFs congeners.

		Number of Congeners	
		PCDDs	**PCDFs**
Mono	(1 chlorine)	2	4
Di	(2 chlorines)	10	16
Tri	(3 chlorines)	14	28
Tetra	(4 chlorines)	22	38
Penta	(5 chlorines)	14	28
Hexa	(6 chlorines)	10	16
Hepta	(7 chlorines)	2	4
Octa	(8 chlorines)	1	1
	Total	**75**	**135**

(adapted from Sanexen Environmental Services Inc. 2000)

PCDD/F congeners are named according to the position of chlorine atoms on the molecule (*e.g.*, 2,3,7,8-TCDD, 1,2,3,7,8,9-HCDF). Each position on the aromatic cycle is designated by a number. The basic chemical structure of PCDD/Fs is shown in Figure 2.1.



(from Sanexen Environmental Services Inc. 2000)

Figure 2.1. Chemical structure of dioxins and furans

This assessment focuses on the 7 PCDD and 10 PCDF congeners that have chlorine atoms attached in at least the 2, 3, 7, and 8 lateral positions because these congeners are the most

toxic and therefore the best studied. The chemical names of these congeners are abbreviated according to those outlined in Table 2.2. Sometimes, congener abbreviations may include a subscript to distinguish between hexa-substituted (H_6CDD) and hepta-substituted (H_7CDD) congeners, for example, when the chlorine substitution pattern was not provided in the original reference, or to refer to all congeners within a homologue group. The Chemical Abstract Service (CAS) registry numbers of the seventeen congeners are presented in Table 2.2.

As a large number of congeners exist, and as these compounds are only formed in trace amounts (by natural processes or anthropogenic activities, see Chapter 3.0), many congeners have not been studied extensively, and their physico-chemical properties are not always well documented. Physico-chemical properties may vary greatly among the seventeen 2,3,7,8-substituted PCDD/Fs, thus influencing their respective environmental fate and transport behaviour (see Chapter 4.0). Multiple estimates of the physico-chemical properties for a given congener can range widely as well (Table 2.3).

Although the physico-chemical properties vary for each congener (depending on the degree of chlorination), dioxins and furans usually exhibit low solubility in water, very low vapour pressure, and high octanol/water partition coefficient (Table 2.3). Melting and boiling points, as well as octanol/water and octanol/carbon partition coefficients tend to increase with the degree of chlorination; while water solubility, vapour pressure and Henry's Constant tend to decrease from T_4CDD/F to OCDD/F. These properties are determining factors of their fate and behaviour in the environment.

A further observation from Table 2.3 is the large uncertainty associated with several of the values. For instance, reported values for the octanol/water partition coefficient of OCDD range from 7.53 (Sarna et al. 1984) to 13.37 (HSDB 1995) on a logarithmic scale. This range corresponds to a factor of approximately 692 000 between minimal and maximal values. Such differences result in large uncertainties when modelling the environmental fate and behaviour of these substances.

Table 2.2. List of 2,3,7,8-substituted PCDD/F congeners with their respective abbreviations and CAS registry numbers.

Congener	Congener Abbreviation	CAS Registry Number[a]
PCDDs		
2,3,7,8-tetrachlorodibenzo-*p*-dioxin	2,3,7,8-TCDD	1746-01-6
1,2,3,7,8-pentachlorodibenzo-*p*-dioxin	1,2,3,7,8-PCDD	40321-76-4
1,2,3,4,7,8-hexachlorodibenzo-*p*-dioxin	1,2,3,4,7,8-HCDD	39227-28-6
1,2,3,6,7,8-hexachlorodibenzo-*p*-dioxin	1,2,3,6,7,8-HCDD	57653-85-7
1,2,3,7,8,9-hexachlorodibenzo-*p*-dioxin	1,2,3,7,8,9-HCDD	19408-74-3
1,2,3,4,6,7,8-heptachlorodibenzo-*p*-dioxin	1,2,3,4,6,7,8-HCDD	352822-46-9
1,2,3,4,5,6,7,8-octachlorodibenzo-*p*-dioxin	OCDD	3268-87-9
PCDFs		
2,3,7,8-tetrachlorodibenzofuran	2,3,7,8-TCDF	51207-31-9
1,2,3,7,8-pentachlorodibenzofuran	1,2,3,7,8-PCDF	57117-41-6
2,3,4,7,8-pentachlorodibenzofuran	2,3,4,7,8-PCDF	57117-31-4
1,2,3,4,7,8-hexachlorodibenzofuran	1,2,3,4,7,8-HCDF	70648-26-9
1,2,3,6,7,8-hexachlorodibenzofuran	1,2,3,6,7,8-HCDF	577117-44-9
1,2,3,7,8,9-hexachlorodibenzofuran	1,2,3,7,8,9-HCDF	72918-21-9
2,3,4,6,7,8-hexachlorodibenzofuran	2,3,4,6,7,8-HCDF	60851-34-5
1,2,3,4,6,7,8-heptachlorodibenzofuran	1,2,3,4,6,7,8-HCDF	67462-34-4
1,2,3,4,7,8,9-heptachlorodibenzofuran	1,2,3,4,7,8,9-HCDF	58200-70-7
1,2,3,4,5,6,7,8-octachlorodibenzofuran	OCDF	39001-01-2

[a] CAS = Chemical Abstracts Service

(Shiu et al. 1988; Lupp and McCarty 1989; Budavari et al. 1989)

Table 2.3. Principal physico-chemical properties of 2,3,7,8-substituted PCDD/Fs.

Chemical	Melting Point		Boiling Point		Water Solubility			Vapour Pressure			Henry's Constant		log K_{ow}		log K_{oc}	
(M.W. in g mol^{-1}) [a]	Value (°C)	Ref[b]	Value (°C)	Ref[b]	Value ($ng \cdot L^{-1}$)	Temp (°C)	Ref[b]	Value (μPa)	Temp (°C)	Ref[b]	Value ($Pa \cdot m^3 \cdot mol^{-1}$)	Ref[b]	Value	Ref[b]	Value	Ref[b]
2,3,7,8-TCDD (322)	295-325	1	421.2-447	8, 9	7.91-483	20-22, 17.3	10, 11	0.098-0.62	25	15, 5	0.0021-10.34	18, 5	5.38-8.93	22, 23	6.74-7.59	31, 32
1,2,3,7,8-PCDD (356)	240-241	2	465	3				0.058-17.5	25	3, 16			7.00-7.80	24, 25	6.8	24
1,2,3,4,7,8-HCDD (391)	273-275	3, 4	488	3	2.22-7.58	7, 26	12	0.0051-3.96	25	3, 16	4.52	5	7.30-10.40	24, 26	7.1	24
1,2,3,6,7,8-HCDD (391)	285-286	2	488	3				0.0048	25	3			7.60-7.80	25, 24	7.6	24
1,2,3,7,8,9-HCDD (391)	243-244	2	488	3				0.0065	25	3			6.90	24	6.6	24
1,2,3,4,6,7,8-HCDD (425)	264-265	3, 5	507	3	0.90-2.47	7, 26	12	0.00075-1.02	25	3, 16	0.133	5	8.00-11.50	24, 23	7.8	24
OCDD (460)	330-332	3, 5	510	3	0.074-0.97	25, NR	5, 13	0.00011-8.7	25, 20	5, 17	0.683	5	7.53-13.37	27, 28	7.9	24
2,3,7,8-TCDF (306)	219-227	6, 3	438	3	419.2	22.7	14	2-123	25	3, 16	1.50	19	5.80-7.70	25, 24	5.61-7.5	33, 24
1,2,3,7,8-PCDF (340)	225-227	6	465	3				0.23-36.4	25	3, 16	2.66	19	6.79-7.80	29, 24		
2,3,4,7,8-PCDF (340)	196	3	465	3	235.3	22.7	14	0.35-21.7	25	3, 16	2.66	19	6.92-7.60	29, 24	7.4	
1,2,3,4,7,8-HCDF (375)	225.5-226.5	6	488	3	8.25	22.7	14	0.032-8.09	25	3, 16	1.454-2.79	20, 19				
1,2,3,6,7,8-HCDF (375)	232-234	6	488	3	17.7	22.7	14	0.029-8.09	25	3, 16	0.741-2.79	20, 19	7.60	24	7.4	24
1,2,3,7,8,9-HCDF (375)								4.99	25	16	2.79	19	7.00	24	6.8	24
2,3,4,6,7,8-HCDF (375)	239-240	6	488	3				0.026-4.99	25	3, 16	2.79	19	7.00	24	6.8	24

Chemical	Melting Point		Boiling Point		Water Solubility			Vapour Pressure			Henry's Constant		log K_{ow}		log K_{oc}	
(M.W. in g mol^{-1}) [a]	Value (°C)	Ref[b]	Value (°C)	Ref[b]	Value ($ng \cdot L^{-1}$)	Temp (°C)	Ref[b]	Value (μPa)	Temp (°C)	Ref[b]	Value ($Pa \cdot m^3 \cdot mol^{-1}$)	Ref[b]	Value	Ref[b]	Value	Ref[b]
1,2,3,4,6,7,8-HCDF (409)	236	6	507	3	1.35	22.7	14	0.0047-2.24	25	3, 16	0.41-1.425	19, 20	8.10	24	7.9	24
1,2,3,4,7,8,9-HCDF (409)	212-223	6	507	3				0.062-1.3	25	3, 16	0.41	19	6.90	24	6.7	24
OCDF (444)	259-260	7, 3	537	3	1.16	25	14				0.10-0.191	21, 20	7.60-13.37	24, 30	8.57-7.4	33, 24

[a] M.W. = molecular weight

[b] References refer to these: 1. Budavari et al. 1989; 2. ATSDR 1998; 3. Rordorf 1989; 4. Pohland and Yang 1972; 5. Shiu et al. 1988; 6. Kuroki et al. 1984; 7. Gray et al. 1976; 8. Schroy et al. 1985; 9. Pohjanvirta 1991; 10. Adams and Blaine 1986; 11. Lodge 1989; 12. Friesen and Webster 1990; 13. Fiedler et al. 1990; 14. Friesen et al. 1990; 15. Podoll et al. 1986; 16. Eitzer and Hites 1988; 17. Webster et al. 1985; 18. Mabey et al. 1982; 19. Eitzer and Hites 1988; 20. Mackay et al. 1992; 21. Clark and Mackay 1991; 22. Crummett and Stehl 1973; 23. Sarna et al. 1984; 24. Broman et al. 1991; 25. Lupp and McCarty 1989; 26. Burkhard and Kuehl 1986; 27. Doucette 1985; 28. HSDB 1995; 29. Sijm et al. 1989; 30. Sarna et al. 1984; 31. Corbet et al. 1988; 32. Lodge and Cook 1989; 33. US EPA1986

2.1 Mode of Toxic Action

While there is a broad range of observed effects of PCDD/Fs exposure, the proposed mechanism of action is believed to be common across fish, birds and mammals and their developmental stages. Poland et al. (1976) were first to propose that 2,3,7,8-TCDD and related toxic isomers elicit their responses via the initial noncovalent interaction with a specific cytosolic receptor protein, known as the aromatic hydrocarbon receptor (AhR), in target tissues. Under this hypothesis, binding of PCDD/Fs to the receptor is the first and essential step to initiate a toxic response. While the most potent member of this family, 2,3,7,8-TCDD, has the greatest affinity for the AhR (Grassman et al. 1998), the sixteen other 2,3,7,8-substituted PCDD/F congeners are believed to elicit toxic effects through the AhR as well. Congeners with fewer than 4 chlorines and/or additional nonlateral substituents exhibit lower AhR binding affinities and therefore lower toxic potencies (Safe 1991). Recent evidence, however, suggests that 1,2,3,7,8-PCDD is equally a potent agonist to the AhR (van den Berg et al. 1998).

The structure and function of the AhR is reviewed in detail elsewhere (*e.g.*, Hahn et al. 1998; Hu and Bunce 1999); therefore, they are only outlined here. The AhR is a member of the basic helix-loop-helix family of proteins. The AhR functions as ligand-activated transcription factor that controls the expression of several genes. It normally exists as a multiprotein cytosolic complex consisting of the AhR ligand-binding subunit, heat shock proteins, and possibly other proteins (Enan and Matsumura 1996; Perdew and Bradfield 1996).

The ligand (for example, 2,3,7,8-TCDD) enters the cell via passive diffusion through the cell membrane, and binds to the AhR (Figure 2.2). Binding affinity depends on both the characteristics of the host's receptor and the properties of the ligand (Hu and Bunce 1999). Upon ligand binding in the cytosol, the AhR complex dissociates, moves to the nucleus, and interacts with the AhR nuclear translocator (ARNT). The ARNT heterodimer binds to specific response elements on DNA and functions as a ligand-activated transcription factor (Schmidt and Bradfield 1996; Rowlands and Gustafsson 1997). Because the Ah locus controls the expression of not only the aryl hydrocarbon hydroxylase gene, but several other genes as well, the toxicity of dioxin-like compounds may result from the expression or repression of multiple genes (Bryan et al. 1987). Binding of dioxin-like compounds to the AhR correlates well to the induction of mixed-function oxidase (MFO) enzyme systems such as cytochrome P-450-1A1, as measured by ethoxyresorufin *O*-deethylase (EROD), and cytochrome P-450-1A2, as measured by acetanilide-4-hydroxylation (Safe 1990; Brouwer 1991;De Vito et al. 1993). These enzymes belong to a family of 12 cytochrome P-450 isozymes (i.e., a group of enzymes that are chemically distinct but functionally alike) that are found primarily in liver endoplasmic reticulum. Cytochrome P-450 enzymes are key to steroid synthesis, and function in the metabolism of other endogenous compounds. Some P-450s are involved in the biotransformation, conjugation and removal, or bioactivation of xenobiotics (Olsson et al. 1998). Enzyme induction is a biochemical response to PCDD/F exposure, but enzyme activity at or above the threshold (induction, relative to control levels) does not guarantee that a toxic response is imminent nor does it necessarily have any connection to a toxic response, should one be observed.

To date, the majority of research on the AhR has been conducted in mice, humans, and other mammals. Evidence of the existence of the AhR protein and/or P-450-1A1 inducibility is found in all vertebrates (mammalian, avian, and fish species) with a few exceptions [*e.g.*, Atlantic hagfish (*Myxine glutinosa*) and sea lamprey (*Petromyzon marinus*)] (Hahn et al. 1992; Vanden Heuvel and Lucier 1993). There are tremendous gaps in our knowledge of the AhR in certain taxonomic groups. This is particularly true for marine mammals, reptiles, amphibians, and non-teleost fish, for which characteristics of the AhR and its function are virtually nonexistent. It is

also true for invertebrates, in which definitive evidence for a functional homolog of the vertebrate AhR has been difficult to obtain (Hahn 1998). Although an endogenous ligand for the AhR has not been identified, the high degree of conservation of functional domains across taxonomic lines suggests that the AhR is necessary for important cellular processes, particularly during development (Dolwick et al. 1996). Furthermore, the conservation of the AhR across species lends support to the common mode of action theory.

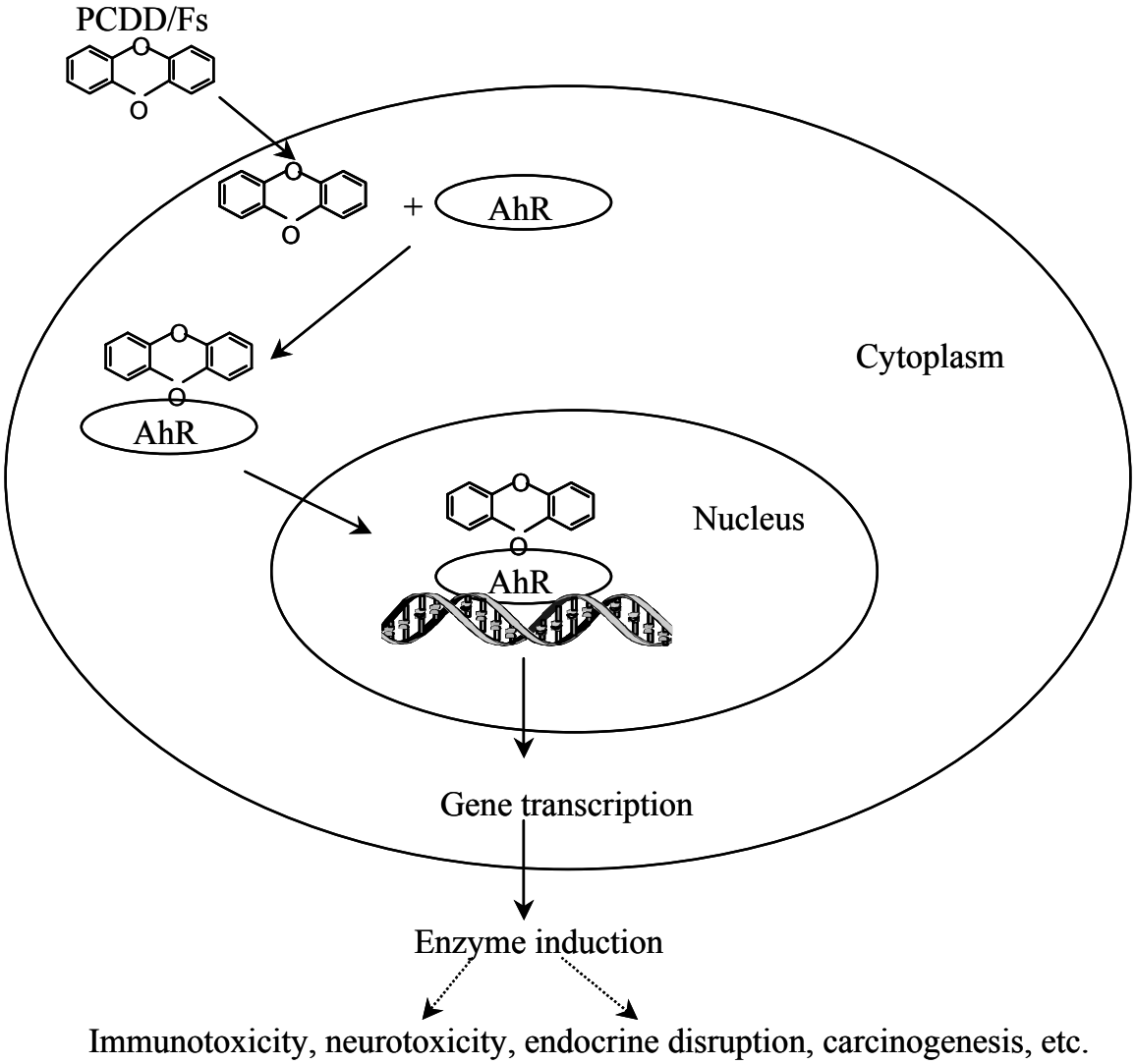


Figure 2.2. Proposed aromatic hydrocarbon receptor (AhR) mediated mechanism of PCDD/F action.

If the consequences of exposure to PCDD and PCDF compounds are dictated by interaction with the AhR, the development of toxic effects in some individuals and not in others may indicate susceptibility differences (McGregor et al. 1998). Current evidence suggests that different AhR alleles, at least in animal models, influence sensitivity of response without affecting the spectrum of biochemical changes produced by dioxins (Birnbaum 1994).

Mechanisms for toxicity that are independent of the AhR likely exist. For example, the AhR protein has not yet been identified in some taxa (*e.g.*, plants). Furthermore, the study of the AhR in invertebrates and early vertebrates could reveal novel features of AhR function, including features that may be ligand-independent (or perhaps independent of halogenated aromatic hydrocarbon ligands) (see Hahn et al. 1997, 1998). Such studies could bring us closer to understanding the ancestral functions of the AhR. Moreover, the mode of action for non-2,3,7,8-substituted PCDD/Fs congeners needs to be identified.

Expressing PCDD/F Concentrations as 2,3,7,8-TCDD Toxic Equivalents (TEQs) - What are TEQs and Why are they Used?

Dioxins and furans exist typically in the environment as complex mixtures. As such, it is often preferable to express their concentrations collectively, rather than as a separate concentration for each individual congener. It is because 2,3,7,8-substituted PCDD/F congeners act commonly on the AhR that the additive toxic potency of a mixture of PCDD/Fs may be expressed as a toxic equivalent (TEQ) relative to 2,3,7,8-TCDD (the most potent inducer of the AhR) (Safe 1990; 1992). In other words, a 2,3,7,8-TCDD TEQ is the sum of concentrations of individual 2,3,7,8-substituted PCDD/F congeners that have been adjusted for their toxic potency relative to 2,3,7,8-TCDD. Concentrations of 2,3,7,8-substituted PCDD/Fs are adjusted using toxic equivalency factors (TEFs) that account for differences in AhR activity and the potential for toxic effects (Safe 1990; 1994). To define, a TEF is an order of magnitude estimate of the toxicity of a compound relative to 2,3,7,8-TCDD (van den Berg et al. 1998). To adjust, mammalian-based TEFs developed by Safe (1990 or 1994) or international TEFs (I-TEFs) developed by NATO/CCMS (1988a, 1988b) are most frequently used to calculate a TEQ, although some TEF values are available for fish and birds. The most recent TEFs for fish, mammals, and birds were developed in 1998 under the Environmental Health and Safety program of the World Health Organization (WHO) and were chosen for use in this document (see Table 2.4; van den Berg et al. 1998). These WHO 1998 TEFs apply to only those congeners with AhR-mediated responses and are based on all available biological and toxicological data including *in vitro*, *in ovo*, and *in vivo* studies measuring various endpoints (*e.g.*, mortality, tumour promotion, and enzyme induction).

The TEF/TEQ approach relies on the availability of appropriate TEFs and assumes that the individual compounds act via a common mechanism and that their toxic effects are additive. Therefore, TEQs derived from analytical analyses necessarily ignore potential synergistic or antagonistic effects amongst the individual compounds in a mixture. In general, it appears that toxic effects are additive especially at ecologically relevant concentrations (Hornung et al. 1996a, 1996b; Walker et al. 1996; Weber et al. 1985; Whyte et al. 1998); however, some evidence suggests that modulation of toxic effects may occur and that some compounds in mixtures do not produce strictly additive effects (Bannister and Safe 1987; Stahl et al. 1992; Rozman et al. 1995).

To apply and compare TEQs, it is imperative that values are calculated using the same set of TEFs. Over the course of development, TEF values and, in some cases, relative rank orders have changed significantly. TEQs calculated using different sets of TEF values, even for the same data, may differ significantly depending on congener composition. Therefore, TEQ-based comparisons should only be made when the same set of TEF values have been applied. For this reason, the raw concentrations for individual congeners, the TEF values, and the TEQ concentrations should be reported. By reporting the full PCDD/F data set, the TEQ may be modified if TEF values are updated in the future. It is important to be consistent and to recognise that calculated TEQs may over- or under-estimate the true TEQ concentration.

Table 2.4. World Health Organization (WHO) toxic equivalency factors (TEFs) for PCDD/Fs).

Congener	Fish TEF	Mammalian TEF	Avian TEF
2,3,7,8-TCDD	1	1	1
1,2,3,7,8-PCDD	1	1	1
1,2,3,4,7,8-HCDD	0.5	0.1	0.05
1,2,3,6,7,8-HCDD	0.01	0.1	0.01
1,2,3,7,8,9-HCDD	0.01	0.1	0.1
1,2,3,4,6,7,8-HCDD	0.001	0.01	<0.001
OCDD	<0.0001	0.0001	0.0001
2,3,7,8-TCDF	0.05	0.1	1
1,2,3,7,8-PCDF	0.05	0.05	0.1
2,3,4,7,8-PCDF	0.5	0.5	1
1,2,3,4,7,8-HCDF	0.1	0.1	0.1
1,2,3,6,7,8-HCDF	0.1	0.1	0.1
1,2,3,7,8,9-HCDF	0.1	0.1	0.1
2,3,4,6,7,8-HCDF	0.1	0.1	0.1
1,2,3,4,6,7,8-HCDF	0.01	0.01	0.01
1,2,3,4,7,8,9-HCDF	0.01	0.01	0.01
OCDD	<0.0001	0.0001	0.0001

(adapted from van den Berg et al. 1998)

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3. SOURCES AND RELEASES OF PCDD/FS TO THE CANADIAN ENVIRONMENT

Polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzo furans (PCDFs) are generated as by-products of controlled or accidental combustion, or as impurities in the manufacturing of various chemical products, metal processing, and pulp and paper production. PCDD/Fs can also be formed through natural combustion processes. The relative contributions from each of these source categories to the Canadian environment will be examined.

3.1 Natural Sources

3.1.1 Forest Fires

Because PCDD/Fs have been detected both in soot and in the flue gases from residential wood burning, it is reasonable to presume that forest fires may also generate these compounds. However, very few studies are available to quantify this hypothesis. Van Oostdam and Ward (1995) reported finding no detectable levels of 2,3,7,8-TCDD/TCDF in the soil and ash samples collected after a forest fire in British Columbia. The soil contained PCDD and PCDF levels (0.05 ng TEQ·kg^{-1}) similar to the local background level (0.02 ng TEQ·kg^{-1}).

To simplify estimates of PCDD/F releases from forest fires, the US EPA (2000) has applied a residential wood burning emission factor to the estimated acres of biomass that were lost to wildfires or prescribed burns. Although the Canadian National Pollutant Release Inventory reports a residential wood combustion factor of 0.5 ng TEQ·kg^{-1} (Environment Canada 2001a), the US EPA has assigned a "low" confidence rating to this approach given the uncertainties surrounding the formation kinetics of PCDDs/Fs during combustion (US EPA 2000).

3.1.2 Volcanoes

It is unlikely that volcanoes represent a significant source of dioxin and furan release to the atmosphere (US EPA 2000). Analysis of groundfall ash after the eruption of Mount St. Helens in 1980 revealed that volcanic particulate emissions were nearly free of detectable PCDDs (with the exception of 0.8 ng·kg^{-1} H_7CDD) upon exiting the volcano, and near the blast zone (~3 to 5 miles), but levels of PCDDs increased (up to 250 ng·kg^{-1}) as the ash passed from rural to urban environments (Lamparski et al. 1990). It was thought that atmospheric PCDDs were becoming associated with the ash through gas-phase sorption or particulate agglomeration (Lamparski et al. 1990).

In an analysis of dust fall and volcanic ash from two active Japanese volcanoes, Takizawa et al. (1994) did not detect any 2,3,7,8-substituted tetra- through hexa-CDD/F congeners (i.e., < 0.1 ng·kg^{-1}). Although the higher chlorinated congeners hepta- and octa-CDD (1.7 to 14 ng·kg^{-1}) and hepta- and octa-CDF (< 0.5 to 4.2 ng·kg^{-1}) were found, the authors were not able to determine whether the observed PCDD/Fs were formed during combustion, or scavenged from the atmosphere.

3.1.3 Biological/Photochemical Processes

There are two suspected biochemical formation mechanisms for PCDD/Fs, including enzyme-mediated formation from chlorophenol precursors (Svenson et al. 1989; Öberg et al. 1990; Öberg and Rappe 1992) and the biodegradation of more chlorinated PCDD/F congeners to less chlorinated (and potentially more toxic) congeners (Barkovskii and Adriaens 1996).

Enzyme-mediated oxidation of chlorophenol compounds can generate PCDD/F compounds in the presence of hydrogen peroxide and a peroxidase enzyme, such as lactoperoxidase in mammals, or horseradish peroxidase (HRP) in plants (Öberg et al. 1990). This peroxidase-mediated enzymatic conversion of chlorophenols is suspected to contribute to the observed *de novo* (new) formation of PCDDs in sewage sludge (Öberg et al. 1992). Morimoto and Tatsumi (1997) found that 42% of pentachlorophenol (PCP) was enzymatically transformed in the presence of HRP, of which 4% was transformed into OCDD. The presence of humic precursors, however, greatly enhanced the enzymatic transformation of PCP (up to 95% of the original), while reducing the amount of OCDD produced; no OCDD was produced in the presence of *p*-coumaric acid (Morimoto and Tatsumi 1997).

In addition to the formation of higher chlorinated congeners (*e.g.*, H_7CDD, OCDD), *in vitro* studies with tri- and pentachlorophenols have produced highly toxic 2,3,7,8- tetra-substituted congeners via this enzymatic oxidation pathway (Svenson et al. 1989; Öberg et al. 1990; Wagner et al. 1990; Öberg and Rappe 1992). In the treatment of 2,4,5- trichlorophenol with a plant peroxidase and hydrogen peroxide, levels of PCDD/Fs were at least 400 times greater than in the control solutions which did not contain either the enzyme, or the hydrogen peroxide (Svenson et al. 1989). Isomer patterns for reaction products vary greatly within PCDD/F groups, and are primarily due to strong differences in reaction kinetics depending on the chlorine-substitution pattern in the chlorophenols (Öberg and Rappe 1992).

Although it is not currently feasible to quantify biodegradation rates, under certain conditions higher chlorinated PCDD/Fs can be biotransformed to more toxic, less chlorinated congeners in sediments, sewage sludge and compost, (US EPA 2000). Barkovskii and Adriaens (1996) demonstrated that up to 10% of OCDD spiked in river sediment was converted to hepta-, hexa-, penta-, tetra-, tri-, di- and monochlorinated isomers, via two distinct dechlorination pathways: the *peri*-dechlorination pathway of 2,3,7,8-substituted hepta- to penta-CDDs which results in the production of 2,3,7,8-TCDD, and the *peri*-lateral pathway which dechlorinates non-2,3,7,8-substituted congeners to produce non-2,3,7,8-TCDD (note that chlorines are located in either the peri [1,4,6, and 9] or lateral [2,3,7 and 8] positions on the benzene rings). The microbially-mediated *peri*-dechlorination of 2,3,7,8-substituted PCDD isomers under anaerobic conditions may be an important pathway previously unaccounted for in the attenuation of chlorinated dioxins in contaminated sediments (Barkovskii and Adriaens 1996).

Biological transformations of congener patterns however, are not universal. Krauss et al. (1994) found that although the composting of household wastes over an 11 week period resulted in an increase in PCDD/F toxicity, from 0.54 to 1.7 $ng \cdot kg^{-1}$ $WHO\text{-}TEQ_{mammals}$. The increase in overall toxicity was due primarily to the formation of H_7CDD and OCDD (Krauss et al. 1994). The observed increases in levels of T_4CDDs and P_5CDDs (from 0.8 to 1.8 $ng \cdot kg^{-1}$, and 0.7 to 1.9 $ng \cdot kg^{-1}$, respectively) were due to non-2,3,7,8-substituted homologues; 2,3,7,8-TCDD and 2,3,7,8-substitued P_5CDDs remained below detection limits (i.e., < 0.1 $ng \cdot kg^{-1}$) (Krauss et al. 1994).

Laboratory studies subjecting PCPs in water to natural or ultraviolet (UV) irradiation have shown the production of higher chlorinated dioxins and furans. The exposure of wood veneers treated with purified, or technical grade PCP to light for 70 days resulted in the formation of OCDD from the photolytic condensation of PCP (Lamparski 1980). The subsequent photodegradation of the OCDD resulted in the production of H_7CDD and H_6CDD, with dechlorination occurring in the *peri*-position (i.e., 1,2,3,4,6,7,8-HCDD dominated H_7CDD congeners) (Lamparski 1980). The UV irradiation of seepage water from a landfill containing PCDD/Fs reduced the overall TEQ despite an increase in several 2,3,7,8-subsitituted PCDD and PCDF congeners (Vollmuth et al.

1994). Difficulties in controlling for volatility and lipophilicity in PCDD/F compounds have prevented the quantification of PCDD/F formation rates from photolysis in natural waters (US EPA 2000).

3.2 Anthropogenic Sources of PCDD/Fs

There are three principle sources of PCDD/Fs to the Canadian environment: point source discharges (to the atmosphere, water, soil), *in situ* contamination, and loadings from long range transport of air pollutants (LRTAP) (CCME 2001a).

An inventory of industrial and municipal sources of PCDD/Fs in Canada has been prepared by the National Office of Pollution Prevention, Environment Canada and forms the basis of the review in this section (Environment Canada 1999, 2001a). Data comprising this inventory comes from a variety of sources across several sectors with a focus on atmospheric emissions. Toxic Equivalency (TEQ) concentrations were calculated using International Toxic Equivalency Factors (I-TEFs). As concentrations of individual congeners were not available, TEQ values could not be re-calculated using WHO TEFs for the purposes of this report. The inventory recommends that all future inventories report PCDD/F values as both I-TEQ and WHO 1998 TEQs (Environment Canada 2001a).

The 1999 Environment Canada dioxin and furan data inventory quantifies emissions from all voluntary reporting sources, and as such there are acknowledged data limitations on PCDD/F releases from some sectors (including Municipal Wastewater Treatment Plants and mining) (Environment Canada 1999, 2001a). As PCDD/F compounds are a Track 1 substance under the *Canadian Environmental Protection Act*, and are destined for virtual elimination, they have been added to the National Pollutants Release Inventory (NPRI) for the 2000 reporting year, and as such, any facility releasing a measurable amount of the substance will be required to report total emissions (Environment Canada 2001a).

Table 3.1. Summary of all PCDD/F releases to Canadian media (g TEQ·a^{-1}) (based on I-TEFs).

Media	**1990**	**1997**	**1999**
Air	427	274	164
Water	454	3	3
Soil	19	19	19
Total Releases	**900**	**297**	**186**
PCDD/Fs in solid waste	2633	173	1097

(from Environment Canada 2001a)

Since 1990, PCDD/F releases to the Canadian environment have decreased by 79% (Table 3.1). The majority of this improvement (63%) is due to the decline in dioxin and furan releases to surface waters from the pulp and paper sector (see Section 3.2.2.1). The remainder of reductions in PCDD/F releases by 1999 were a result of a 62% decrease in emissions to the atmosphere, led by improvements in waste incineration (medical and municipal) and iron sintering facilities (see Sections 3.2.1.1 and 3.2.1.2, respectively). The order of magnitude decrease in PCDD/F releases in solid waste in 1997 are due to a lack of available estimates for PCDD/Fs in pentachlorophenol products for this reporting year (Environment Canada 2001a).

3.2.1 *Releases to the Atmosphere*

Releases to the atmosphere from combustion sources represent the largest contribution of PCDD/Fs to the Canadian environment, and account for approximately 88% of the 186 g TEQ·a^{-1} released (Table 3.1). Airborne PCDD/Fs, in both the vapour and particulate phases may be transported long distances and deposited through wet or dry deposition (Bobet et al. 1990; Steer et al. 1990). As such, aquatic and terrestrial ecosystems, near or far from anthropogenic sources, may become contaminated with PCDD/Fs via this pathway. Combustion sources include municipal waste incinerators, biomedical waste incinerators, coal-fired utility boilers, oil and gas burning, fuel wood burning, forest fires, and cigarette smoking, among others (Sheffield 1985). Combustion of many carbon-based materials with minute quantities of chlorine, organic chlorine compounds or inorganic chlorides leads to the production of PCDD/Fs in limited amounts (Rappe et al. 1987; Hicks and McColl 1995). PCDDs released from combustion sources consist generally of the less toxic congeners (*e.g.*, H_7CDD and OCDD) (Sheffield 1985; Fiedler et al. 1990). This congener profile, however, is often reversed for PCDFs, such that the smaller, more toxic T_4CDF and P_5CDF congeners are the predominant forms (Fiedler et al. 1990).

PCDD/Fs can be found in combustion emissions via three principle mechanisms which are not mutually exclusive (US EPA 2000):

1. PCDD/Fs may be present as contaminants in the combusted organic material, and therefore pass through the furnace and be emitted unaltered;

2. PCDD/Fs can be formed from the thermal breakdown and molecular rearrangement of precursor ring compounds (chlorinated aromatic hydrocarbons which structurally resemble PCDD and PCDF molecules); these ringed precursors which emanate from the combustion zone are a result of incomplete oxidation (combustion) of the source material; and

3. PCDD/Fs can be synthesized from heterogeneous reactions on fly ash involving carbon, oxygen, hydrogen, chlorine and a transition metal catalyst

The rate of formation of these compounds are dependent on the overall combustion efficiency, post-combustion flue gas temperatures and residence times, and the availability of surface catalytic sites to support PCDD/F synthesis (US EPA 2000). PCDD/F compounds are formed outside the combustion zone in lower temperature ranges (*e.g.*, 200 to 450°C) in the presence of a metal precursor, such as copper chloride. There is some discrepancy between the scale of combustion sources as to whether chlorine levels in fuel sources is directly related to PCDD/F output in emissions; the link between chlorine levels in feed stock used in larger commercial scale facilities is less direct (US EPA 2000).

The CCME (2001a), has identified six priority release sectors which account for about 80% of all national emissions:

1. waste incineration (municipal solid waste, hazardous waste, sewage sludge and medical waste)
2. burning of salt-laden wood in British Columbia coastal pulp and paper boilers
3. residential wood combustion
4. iron sintering
5. electric arc furnace steel manufacturing
6. conical municipal waste combustion in Newfoundland

3.2.1.1 Waste Incinerators

The elevated temperature and high concentrations of carbon, oxygen, and chlorine species created during incineration may provide conditions conducive to the formation of dioxins and furans (Hicks and McColl 1995). If incineration conditions are optimal, PCDD/Fs may themselves be destroyed but generally the incineration process is not 100% effective, leaving behind small fractions of the compounds (Hutzinger and Blumich 1985; Hicks and McColl 1995).

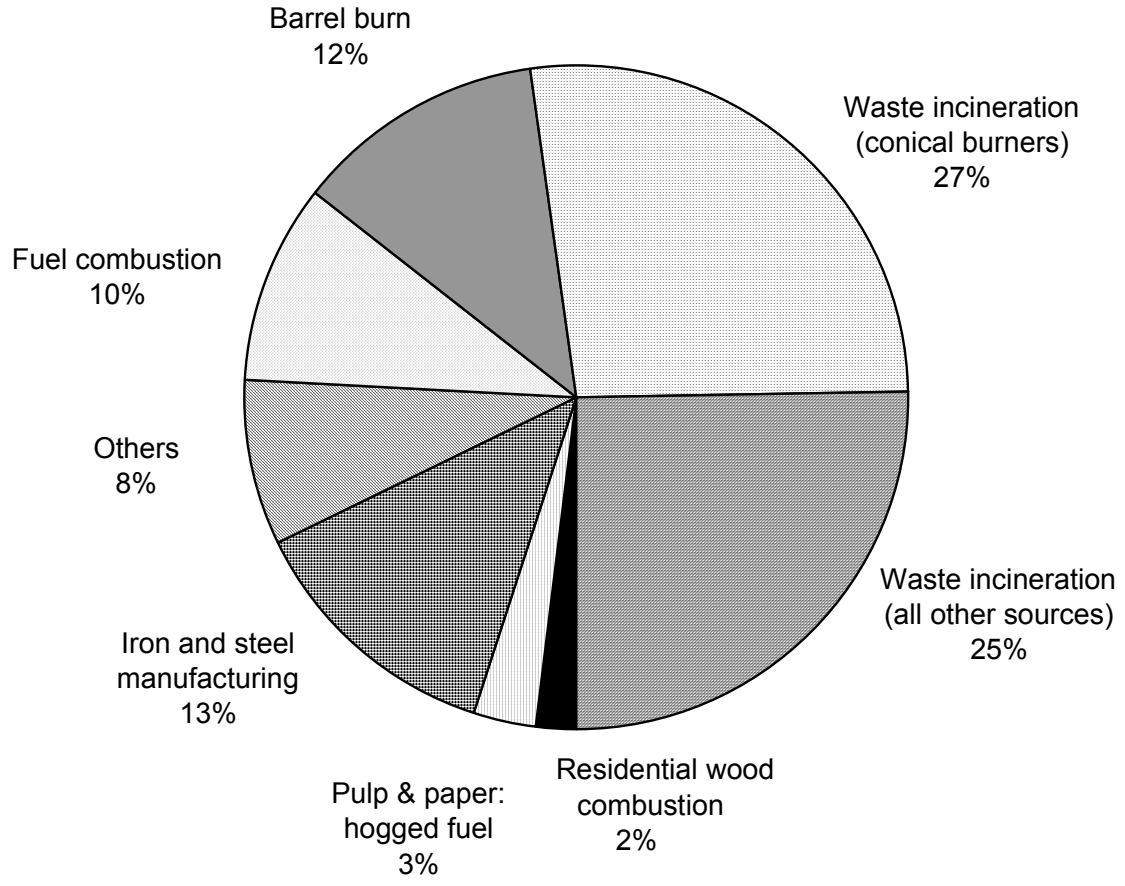
There are several types of incinerators operating in Canada. Municipal waste incinerators, hazardous waste incinerators, and biomedical waste incinerators all contribute in varying degrees to the release of PCDD/Fs to the Canadian environment.

Waste incinerator facilities are the most significant source of emissions of PCDD/Fs in Canada, releasing 85 g TEQ·a^{-1}, or 52% of total emissions to all media in 1999 (Figure 3.1). Rappe et al. (1987) reported that the major source of chlorine in municipal solid waste incinerators is plastic material (*e.g.*, polyvinyl chloride [PVC]), and there is direct evidence for the conversion of PVC to PCDDs and PCDFs (Marklund et al. 1986). There are currently 13 municipal waste incineration facilities operated by the provinces and 241 federally operated facilities (as of 1990). Atmospheric releases of PCDD/Fs from these facilities across Canada have declined dramatically from 143 g TEQ·a^{-1} in 1990 to 9 g TEQ·a^{-1} in 1999 (Environment Canada 2001a). A major contributor to this reduction came from $6 million in renovations to the municipal waste incinerator in Levis, QC which reduced the annual output of 62 g TEQ·a^{-1} from this facility by over 99% (CCME 2001a). Additional reductions were realized by the closure of several facilities between the 1997 and 1999 reporting periods.

Small, municipally operated conical burners are used exclusively in Newfoundland. Approximately 45 units are used to incinerate municipal wastes, producing 43 g TEQ·a^{-1}, or 27% of the total emissions to the atmosphere (Figure 3.1). Currently, the Newfoundland government is looking into plans to reduce PCDD/F emissions through enhanced separation technologies (i.e., recycling) prior to combustion, with the anticipated goal of phasing out all non-essential conical burners for the handling of municipal wastes (CCME 2001a).

Hazardous waste incinerators are of public concern, especially those where PCBs are burned. Typical hazardous waste streams consist of contaminated process wastewater, residues from chemical process industries, paint residues, chemical spill cleanups, solids, soils, oils, and others. The congener profile for PCDD/Fs from hazardous waste incinerators is similar to those reported from municipal waste incinerators (Rappe et al. 1984; Marklund et al. 1986). In 1999, there were seven hazardous waste incineration facilities operating in Canada. Four facilities in Ontario accounted for nearly all of the 7 g TEQ·a^{-1} emitted by this sector in that year (Environment Canada 2001a).

Biomedical waste incineration is a common operation performed at hospitals and has also been identified as a source of dioxins and furans. Biomedical waste incinerators destroy medical waste that includes all hospital waste except for corpses and body parts. Several hospital incinerators have been shut down since 1990, thus reducing PCDD/F releases from 130 g TEQ·a^{-1} in 1990 to 25 g TEQ·a^{-1} in 1999 (Environment Canada 2001a).



(total emissions = 164 g TEQ·a^{-1}; percentages based on loadings rounded to the nearest gram)

(data source: Environment Canada 2001a)

Figure 3.1. Estimates of PCDD/F releases to the atmosphere in 1999 by Canadian municipal, industrial and agricultural sources.

The recent endorsement of the Canada Wide Standards (CWS) for PCDD/Fs by the CCME in 2001 has placed new emission limits for municipal, medical, hazardous and sewage sludge waste incinerators in Canada. PCDD/F emission levels have been found as high as to 126 000 and 177 000 pg TEQ·m^{-3} at municipal and hazardous waste incinerators, respectively (Environment Canada 2001a). Under the CWS, both new and existing facilities are now required to conform to limits of 80 pg TEQ·m^{-3}, while existing sewage sludge incinerators are now limited to emissions of 100 pg TEQ·m^{-3} (CCME 2001a). It is anticipated that adherence to

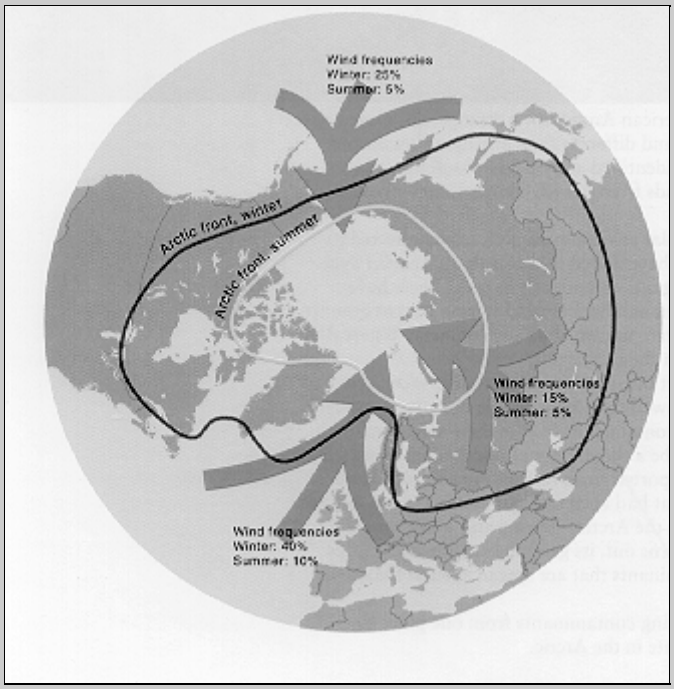
these new CWS will reduce total emissions to the atmosphere by 86% over current levels by 2006 (CCME 2001a).

POPs on the Move: Dioxins and Furans in the Canadian Arctic

"Persistent Organic Pollutants" or POPs include PCDD/Fs and other chemicals such as polychlorinated biphenyls (PCBs) and certain pesticides. POPs are long-lived organic compounds that become more concentrated as they move through the food chain. Concern over the adverse effects of POPs on wildlife and humans prompted the United Nations Environment Program to call for a complete global ban on their production and use through the Stockholm Convention, signed by 91 countries, including Canada, in May 2001.

Organochlorine POPs, including dioxins and furans, are of particular concern in the Canadian Arctic, where they are ubiquitous in the food chain (de March et al. 1998). Concentrations of the most toxic 2,3,7,8-TCDD/F congeners can be found in the nanogram per kilogram wet weight range in freshwater fish (Muir et al. 1997). Although PCDD/Fs tend to be less mobile than other chlorinated POPs, such as hexachlorobenzene (HCB) and hexachlorocyclohexane (HCH), their prevalence in remote Arctic regions is due to their ability to transported over long distances. Characteristic travel distances in air for 2,3,7,8-TCDD (810 km) and OCDD (460 km) are much less than other highly volatile POPs (*e.g.*, ~2000 to 110 000 km) (Beyer et al. 2000). Important contributors to the background levels of PCDD/F contamination found in the Arctic are thought to include municipal waste incinerators in Arctic Canada, Finland, Greenland and Alaska, and metal smelting operations in Sweden, Norway and Russia (de March et al. 1998).

Cold Arctic airmasses play a key role in transporting POPs throughout the Arctic Circle. In the winter, the Arctic front extends well into the southern industrial regions where the majority of PCDD/F releases occur (Figure 3.2). The combination of prevailing winter winds carrying PCDD/Fs northwards (Figure 3.2), and the ability for particles to remain in the winter Arctic airmass for as long as 20 to 30 days (compared with 2 to 5 days in the summer), creates conditions for the long-range transport and accumulation of these contaminants in the polar regions (AMAP 1997).



(source: AMAP 1997)

Figure 3.2. Mean position for the Arctic air mass during January and July, with winter and summer frequencies of prevailing winds.

On a global scale, an estimated 1 tonne of PCDD/Fs are deposited to the world's oceans, and 12 tonnes to the Earth's land surfaces each year from anthropogenic sources (Baker and Hites 1999). Commoner et al. (2000), traced the atmospheric sources of dioxins and furans for remote receptor sites in Nunavut using air transport modelling (Commoner et al. 2000). The estimated amounts of PCDD/Fs being deposited in the Canadian Arctic varied significantly with latitude. Because the amount of dioxins and furans that are deposited decline rapidly with distance from the source, dioxin levels in southern Nunavut at Sanikiluaq were estimated to be 10 times higher than those in Arctic Bay in the far north (Commoner et al. 2000). Of the estimated 37 g TEQ (based on I-TEQs), deposited each year to an area of over two million km^2 in Nunavut, Canadian sources accounted for between 11 and 25% of the total, while the majority of PCDD/Fs (70 to 82%) originated from sources in the United States. Very little of the dioxin load to the Arctic was thought to originate from European sources (i.e., only about 2% of the amount deposited from North American sources) (Commoner et al. 2000). Commoner et al. (2000) suggest that because less than 2% of all individual sources accounted for over 75% of the typical dioxin load modelled for Nunavut, concentrated emission reduction efforts would be required for relatively few North American industrial sectors (Commoner et al. 2000).

Despite this potential for long-range transport and deposition of PCDD/Fs to the Arctic, recent temporal studies on concentrations in people and biota in the Canadian Arctic suggest that PCDD/F contamination is on the decline. In an analysis of PCDD/F levels in Inuit breast milk collected in Nunavik, TEQs (based on WHO TEFs) for all PCDD congeners showed a 3.8-fold decrease between studies conducted in 1990 and 2000 (to 4.7 ng $TEQ_{mam} \cdot kg^{-1}$ lipid), and those for all PCDB congeners decreased by a factor of 2 (to 1.9 ng $TEQ_{mam} \cdot kg^{-1}$ lipid) over the same period (Dewailly 2001). Breast milk samples of residents of the Keewatin district on the west coast of Hudson's Bay in 1997, showed that TEQs (TEFs not provided) for total PCDD (3.6 ng $TEQ \cdot kg^{-1}$ lipid) and total PCDF (1.3 ng $TEQ \cdot kg^{-1}$ lipid) were well below the 1992 national average of pooled samples from southern Canada (10.1 and 4.4 ng $TEQ \cdot kg^{-1}$ lipid, respectively) (Newsome and Ryan 1999). Levels of PCDD/Fs in two of three Arctic seabirds monitored from Prince Leopold Island in Lancaster Sound, Nunavut, have similarly been on the decline over the last 20 years. Between 1975 and 1993 total PCDD concentrations in the livers of black-legged kittiwakes, and fulmars have decreased by a factor of 7 and 2.5, respectively; and total PCDFs have decreased by just over half, for both species (Braune 2001). This decline would suggest that the estimated high PCDD/F deposition rates modelled for the Arctic by Commoner et al. (2000), are not necessarily being reflected in the body burdens of people, or biota in the region (c.f. Dewailly 2001).

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3.2.1.2 Steel Manufacturing, Iron Sintering and Base Metal Smelting

In Canada, metal processing contributed approximately 21 g $TEQ \cdot a^{-1}$ in 1999, or 13% of total atmospheric releases (Figure 3.1). This amount reflects the combined estimates for the iron manufacturing (Canada Gazette 2002a), steel manufacturing (electric arc furnaces and foundries), and base metal smelting sectors. Similar to other sectors, these levels have decreased relative to 1990 estimates of 37 g $TEQ \cdot a^{-1}$ (Environment Canada 2001a).

The 1998 closure of an iron sintering plant in Wawa, Ontario has reduced overall atmospheric PCDD/F emissions from this sector from 25 g $TEQ \cdot a^{-1}$ in 1990 to 6 g $TEQ \cdot a^{-1}$ at the remaining facility in Hamilton, Ontario (Environment Canada 2001a). In a draft technical background paper provided to the Canada Wide Standards Development Committee, the Canadian steel manufacturing sector proposed that best available control technologies could reduce PCDD/F concentration to <200 pg $TEQ \cdot m^{-3}$for new sinter plants and to 500 pg $TEQ \cdot m^{-3}$ for existing sinter plants (CCME 2001a). This change would represent a 93% reduction over current concentrations of 2700 pg $TEQ \cdot m^{-3}$ for the iron sintering plant in Hamilton (Environment Canada 2001a).

There are 13 steel manufacturing plants with electric arc furnaces in Canada, with the majority being in Ontario and Québec (Environment Canada 2001a). With emissions totalling 11 g TEQ·a^{-1} for this sector, electric arc furnaces are the seventh largest source of atmospheric release of PCDD/Fs. Recent stack testing data show that between 44 and 254 pg TEQ·m^{-3} are released in electric arc furnaces in Canada (Environment Canada 1999). Based on performance data from a European facility, best available emission control technology may reduce concentration in Canadian plants to below 100 and 150 pg TEQ·m^{-3} for new and existing electric arc furnaces, respectively (CCME 2001a).

Base metals smelting plants in Ontario and Québec contributed 2.9 and 0.1 g TEQ·a^{-1}, respectively, to the atmosphere in 1999 (Environment Canada 2001a).

3.2.1.3 Burning of salt-laden wood

Coastal pulp and paper mills in British Columbia use hogged wood fuel (chipped bark and other woody debris from stripped logs) to feed pulp boilers. Logs used in this process are transported by sea in flotillas along the coast, which results in substantial uptake of NaCl from the water. When the chipped bark is dried and burned as a fuel source, chlorine is released during the combustion process, resulting in the formation of dioxin and furan compounds. An Alberta pilot scale study comparing PCDD/F levels in emissions from burning salt laden hogged fuel (0.76% wt. chlorine) found contaminated bark produced 3.2 ($\pm$ 0.6) ng TEQ·m^{-3}, compared to 0.2 ng TEQ·m^{-3} in emissions from control fuels (Pandompatam et al. 1997). Atmospheric PCDD/F emission estimates for coastal British Columbia mills in 1999 ranged from 2.7 to 7.6 g TEQ·a^{-1}, with an average of 5 g TEQ·a^{-1} (Environment Canada 2001a).

The industry is currently pursuing options to reduce PCDD/F emissions, including: improving combustion conditions (shutting down old boilers and replacing with new technologies); optimizing secondary collection of the particulates; adding sulphur to the combustion process; injecting ammonia or urea to combustion to inhibit dioxin formation; fluid bed conversion for more efficient combustion; and hog fuel washing/pressing to remove chlorine (CCME 2001a). Industry-mediated improvements have resulted in an overall reduction in emissions levels of 10 g TEQ·a^{-1} in 1990 and 1997, to 5 g TEQ·a^{-1} in 1999 (Environment Canada 2001a). The recently developed CWS for this sector established emission standards of 100 and 500 pg TEQ·m^{-3} for all new and existing boilers, respectively. It is anticipated that this will reduce current emissions a further 45% by 2006 (CCME 2001a).

3.2.1.4 Residential wood burning

Residential burning of chemically-untreated wood is a significant source of PCDD/Fs to the atmosphere, contributing 3 g TEQ·a^{-1} in 1999 (Environment Canada 2001a). According to a 1995 Environment Canada survey, 37% of the wood burning appliances in Canada are residential wood stoves which employ modest, or no emission reduction technology, with the remainder consisting of open fireplaces (54%) and other furnaces (9%) (Environment Canada 2000a).

Based on recent emissions testing of conventional, and U.S. EPA-certified wood stoves with maple and spruce as fuel sources, an emissions factor of 0.5 ng TEQ·kg^{-1} wood burned is used in deriving Canadian atmospheric loading estimates (Environment Canada 2000a, 2001a). The majority of dioxins and furans released were associated with the gaseous phase, and as such,

levels of PCDD/F TEQs from wood stove combustion are unrelated to the release of particulate matter (Environment Canada 2000a). The lower chlorinated furans, especially 2,3,7,8-T_4CDF and 2,3,4,7,8-P_5CDF accounted for the majority of TEQ levels (57 to 81%), with the majority of the remainder being contributed from hexa- to octa-CDDs (Environment Canada 2000a).

The release of dioxins and furans from residential wood combustion is also a national priority issue for the CWS Development Committees for particulate matter, ozone and benzene, and a coordinated effort with the three development committees and non-governmental organizations is currently underway to study emission reduction strategies for this sector (CCME 2001a).

3.2.1.5 Oil, coal, and gas burning and refining

There are conflicting and limited data regarding dioxin formation by the burning of coal. Studies that examine the detection relationships between emission concentrations and other variables such as the chlorine content of coal and combustion conditions are needed (Hutzinger and Blumich 1985). There is some indication that by burning sulphur-rich fuel, such as coal, along with waste in municipal incinerators, the production of dioxins and furans can be significantly reduced. This reduction occurs because sulphur compounds react with metal catalysts present in the combustion process which would otherwise precipitate the Deacon reaction ($HCl + O_2$ to yield Cl_2) and the formation of free chlorine (a precursor for PCDD/F formation) (US EPA 2000).

Reported concentrations of PCDD/Fs in flue stacks from Canadian electric power generating operations using coal fuel sources were 5 and 30 pg TEQ·m^{-3} (Environment Canada 1999). As measured flue concentrations at several facilities were at, or below the detection limits for PCDD/Fs, those facilities have adopted standard emission factors from the US EPA or the Electric Power Research Institute (EPRI) to determine total TEQ loads to the atmosphere (Environment Canada 1999). PCDD/F loading from the electric power generating sector has increased from 3 g TEQ·a^{-1} in 1990 to 5 g TEQ·a^{-1} in 1999 (Environment Canada 2001a).

A Canadian inventory of PCDD/F releases by the petroleum industry is not available (Environment Canada 2001a), however results of a study conducted in Ontario indicate that total PCDD levels in stack emissions from various petroleum refineries were as high as 9 ng·m^{-3}, and total PCDF levels were reached 210 ng·m^{-3} (Thompson et al. 1990).

3.2.1.6 Cement kilns

PCDD/F emission estimates for the 25 cement kilns in Canada totalled approximately 2 g TEQ·a^{-1} in 1999, which represented a 27% reduction over 1990 levels (Environment Canada 2001a). Cement kilns typically burn alternative fuels, such as tires, waste oil, coal, coke, solvents and bunker oil.

3.2.1.7 Miscellaneous atmospheric point sources

Kraft liquor boilers are used by the pulp and paper industry to separate the insoluble pulp from the soluble phase consisting primarily of lignin, which contain phenolic compounds. Following recovery of other chemicals from this “black liquor”, it is then sprayed into a recovery furnace and therefore becomes a potential source of PCDD/Fs (US EPA 2000). Two kraft liquor boilers were tested; one by the industry in British Columbia and one by Environment Canada in

Québec. The concentration of PCDD/Fs exiting the stacks ranged from 4 and 8 $pg \cdot m^{-3}$ (Environment Canada 1999). Using an average emission factor of 6 pg $TEQ \cdot m^{-3}$, Environment Canada (2001) estimated national emissions of 0.6 g $TEQ \cdot a^{-1}$ from this sector.

Volatilization from wood waste at saw mills and pulp and paper mills contributed a further 1 g $TEQ \cdot a^{-1}$.

3.2.1.8 Non-point source emissions to the atmosphere

Diffuse emissions of PCDD/Fs account for approximately 23% of all releases to the atmosphere (12% from barrel burning of household wastes; 6% from diesel fuel combustion; 4% from residential and agricultural fuel combustion; and 1% from in-service utility poles).

Automobiles are known contributors to PCDD/F emissions in Canada. Ballschmiter et al. (1986) identified a series of PCDD/Fs in used motor oil. They suggested chlorinated additives in motor oil or in gasoline as possible sources for the PCDD/Fs, but no quantitative data were reported. TEQ emission factors (based on I-TEFs) for vehicles vary greatly ranging from 0.36 to 520 $pg \cdot km^{-1}$ for gasoline passenger cars and light duty vehicles, from 2.4 to 241 $pg \cdot km^{-1}$ for diesel passenger cars and light duty vehicles, and from 380 to 9500 $pg \cdot km^{-1}$ for diesel buses and trucks (heavy duty vehicles) (Oehme et al. 1991; Geueke et al. 1999). Fuel combustion emissions in Canada were estimated from the number of kilometres travelled by different types of vehicles and by using US EPA emission factors. Diesel traffic contributes an estimated 9 g $TEQ \cdot a^{-1}$, while estimates for residential and agricultural fuel combustion are 7 g $TEQ \cdot a^{-1}$ (Environment Canada 2001a).

The burning of yard wastes, especially those treated with herbicides or wood products with anti-fungal preservatives, under low combustion temperatures and oxygen-starved conditions can result in dioxin and furan synthesis. For example, in a bench-scale furnace operating at 500 - 600°C, Jansson and Sundström (1978) found significant PCDD production in flue gases during the combustion of wood chips treated with tri-, tetra-, and pentachlorophenols (anti-fungal agents), especially under oxygen-starved conditions (*e.g.*, up to 18 µg P_5CDD per g formulation). Similarly, Stehl and Lamparski (1977) generated 2,3,7,8-TCDD in ppm levels by combusting grasses and paper treated with the herbicide 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) in a bench-scale furnace at 600 - 800°C.

Spatial distribution estimates of barrel burning practices are poorly characterized for Canada. As emission estimates are based on limited surveys performed by the US EPA, this may result in a large degree of error surrounding the Canadian estimate of 20 g $TEQ \cdot a^{-1}$ (CCME 2001a; Environment Canada 2001a).

In-service utility poles treated with chlorophenol wood preservatives also provide diffuse sources of PCDD/Fs to the atmosphere due to evaporation of the volatile organics from the above-ground treated sections. Although the amount for evaporative release is quite small per pole, their use is ubiquitous across the country and must therefore be treated as a distinct sector for non-point source release (CCME 2001a). It is estimated that this source provides 2 g $TEQ \cdot a^{-1}$ to the atmosphere (Environment Canada 2001a). Releases of PCDD/Fs to soil from in-service utility poles is discussed below.

3.2.2 Releases to Water

As a result of aggressive federal and provincial regulation, as well as industry-driven initiatives, point source discharges of PCDD/Fs to Canadian surface waters have decreased by 99% relative to 1990 (CCME 2001a, Environment Canada 2001a). Current releases of 3 g TEQ·a^{-1} represent only 2% of releases to all media (Environment Canada 2001a).

3.2.2.1 Pulp and paper mills

PCDD/Fs are formed during the bleaching process in the manufacture of chemical pulp, which accounts for 54% of the wood pulp produced in Canada[1] (Environment Canada 1997a). Until recently, elemental chlorine was the primary agent used to bleach and brighten chemical pulps. During chlorine bleaching, natural phenolic constituents of wood pulp (*e.g.*, lignin) are chlorinated to yield chlorinated phenolic compounds which are precursors of PCDD/Fs (Safe 1991). British Columbia has a unique problem in that pulp wood is typically transported down the Pacific coast to pulp and paper mills. This practise results in large quantities of salt (NaCl) being adsorbed by the wood. The chlorine in the salt then facilitates the production of PCDD/Fs during processing. Sediment near British Columbia coastal mills contain relatively high levels of PCDD/Fs compared to inland mills (Trudel 1991). Historically, significant quantities of the most toxic PCDD/F congeners were discharged directly to freshwater, estuarine, and marine systems (Government of Canada 1990). In 1989, PCDD/F discharges in the final effluent from 45 mills using chlorine bleaching in Canada totalled 145 g·a^{-1} on a WHO-TEQ_{fish} basis (Halliburton and Simpson 1999). PCDD/F signatures in sediments surrounding coastal British Columbia mills are dominated by the higher chlorinated congeners (hexa- through octa-CDDs), with H_6CDD being the dominant homologue group (Bright et al. 1999).

The Canadian pulp and paper industry has made significant reductions in PCDD/F effluent emissions since the implementation of *Pulp and Paper Effluent Regulations* which prohibit the release of any measurable amounts of 2,3,7,8-TCDD or 2,3,7,8-TCDF (Government of Canada 1991). Effluents from all mills and plants were found to be 100% compliant with these limits during 1997 (Environment Canada 1999). Total dioxin and furan releases to receiving waters from pulp and paper mills totalled 3.3 g TEQ·a^{-1} in 1997 and 1999 (Environment Canada 2001a). This constitutes a 99% reduction over 1990 emission levels of 450 g TEQ·a^{-1} from this sector.

The most significant process change contributing to these reductions has been the substitution of chlorine dioxide for elemental chlorine; pulps made with chlorine dioxide are termed elemental chlorine free (ECF). In 1988, elemental chlorine accounted for 61% of bleaching requirement and chlorine dioxide accounted for 15%. By 1993, chlorine dioxide was predominantly used by the sector (73%). The total amount of elemental chlorine used was reduced from 560 kilotonnes to 175 kilotonnes during this time; the projection for 2000 is 10 kilotonnes. Total chlorine free (TCF) pulps are not yet widely produced in Canada, contributing to only 1% of the total chemical pulp production in 1993. Technologies and bleaching agents for TCF pulp include oxygen delignification, ozone delignification, extended cooking, and hydrogen peroxide bleaching. Of these, only oxygen delignification is anticipated to be in significant use in Canada by 2000 (Environment Canada 1997a).

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[1] mechanical pulp, which is chlorine free, accounted for 46% of the wood pulp produced in 1993 (Environment Canada 1997b).

3.2.2.2 Chemical production

PCDD/Fs are impurities in a wide variety of commercial chemicals used in Canada. Trace quantities of PCDD/Fs occur in many chemicals, mainly as a result of high temperatures and chlorinated solvents that are used in manufacturing processes. Substances known to be contaminated with PCDDs and/or PCDFs include chlorophenols (*e.g.,* pentachlorophenol), various pesticides (*e.g.*, 2,4-D and 2,4,5-T), 1,2,4-trichlorobenzene, hexachlorobenzene, tetrachlorobenzoquinones, askarels (PCB mixtures used in electric transformers), and perchloroethylene (Fiedler et al. 1990). PCDD/F profiles in herring gull eggs from Lake Ontario, St. Lawrence River, Niagara River, and Saginaw Bay were consistent with effluents from the historical manufacture of 2,3,5-trichlorophenol or 2,4,5-T, and waste dumps associated with this manufacture (Hebert et al. 1994). Accidental spills and runoff of pentachlorophenol products from lumber treatment and storage sites can also result in the contamination of nearby surface waters (Garrett and Shrimpton 1988).

In Canada, PCDD/Fs released in liquid effluents by the chemical production sector amounted to 3.7 g TEQ·a^{-1} in 1990 (Environment Canada 2001a). By 1997, the majority of these plants had modified their processes resulting in a virtual elimination of releases (0.01 g TEQ·a^{-1}) to surface waters (Environment Canada 1999; 2001a).

3.2.2.3 Municipal wastewater treatment plants

Information on PCDD/F emissions from MWWTPs was not available for the Environment Canada (2001) Inventory of Releases. A subsequent poll of MWWTPs for some of the larger Canadian cities revealed that PCDD/F emissions are not routinely analyzed, and therefore a comprehensive analysis for this sector is not currently available.

Levels of 2,3,7,8-substituted PCDD/F congeners and their overall TEQs were available for four WWTPs from the Greater Toronto Area for a single collection period during June, 2001 (City of Toronto 2001). PCDD/F levels for influent waters at the treatment plants ranged from 0.59 to 1.5 pg TEQ·kg^{-1}, while PCDD/F levels for effluent ranged from <detection to 0.064 pg·kg^{-1} (City of Toronto 2001). Although small amounts of 2,3,7,8-TCDF (0.2 - 0.3 pg TEQ·kg^{-1}) were detected in influent waters for two of Toronto's WWTPs, higher chlorinated hepta- and octa-chlorinated 2,3,7,8-substituted congeners were predominant (up to 44 pg TEQ·kg^{-1}) (City of Toronto 2001). Centrifuge cake consisting of digested sewage sludge collected from the four treatment plants contained much higher PCDD/F concentrations than the influent water, ranging from 7 800 to 26 600 pg TEQ·kg^{-1}TEQ (City of Toronto 2001).

The NPRI survey has included dioxins and furans in the 2000 reporting year period for all sectors, including MWWTPs; results of the survey are anticipated to be made publicly available by 2002 (Morcos 2001).

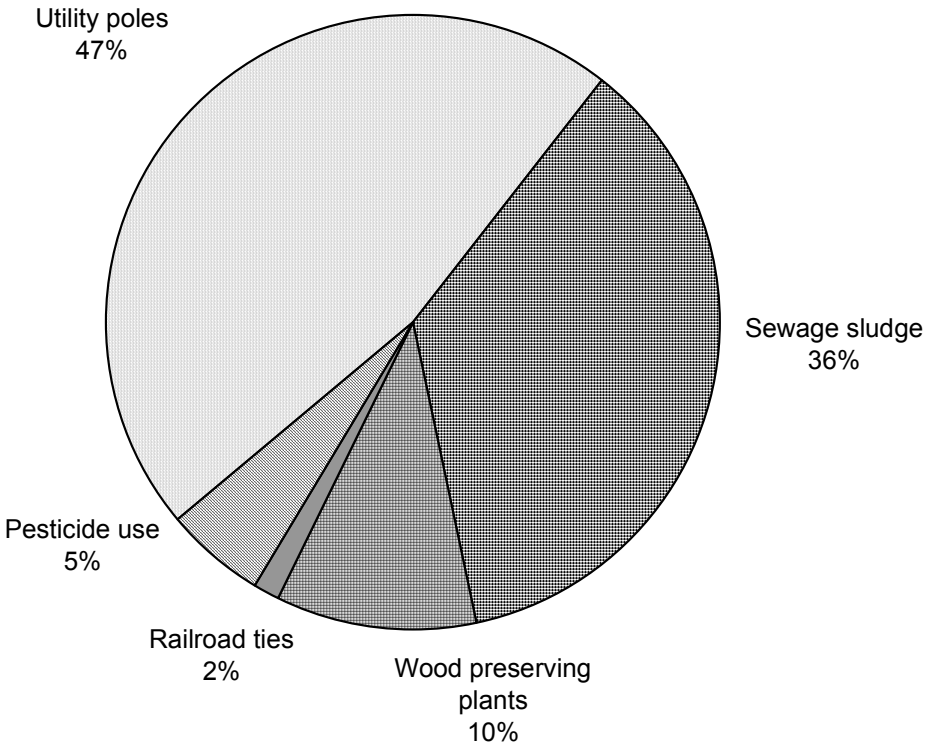
3.2.3 Releases to Soil

Dioxin and furan releases to soils totalled 19 g TEQ·a^{-1} for 1999, representing the second largest source to the Canadian environment (Table 3.1). The widespread use of utility poles and railway ties treated with chlorophenol-based wood preservatives provides a diffuse, but nationally important source of PCDD/Fs to soils (CCME 2001a). Leaching of wood

preservatives from railway ties and in-service utility poles are estimated to contribute 0.3 and 9 g TEQ·a^{-1}, respectively (Environment Canada 2001a). Both sectors account for 49% of the total PCDD/F load released to soils annually (Figure 3.3).

Sewage sludge application to land surfaces for use as fertilizers is the second largest source of PCDD/Fs to soils in Canada (36%). Typical background concentrations of PCDD/Fs in agricultural soils in Ontario are 7 000 pg TEQ·kg^{-1} dw; however, contributions from sewage sludge are estimated at 25 000 pg TEQ·kg^{-1} dw (Environment Canada 2001a). The application of sewage sludge to Canadian soils results in a PCDD/F loading of 7 g TEQ·a^{-1}, with 37% of the total load being released to Ontario soils (Environment Canada 2001a).

The application of pesticide products that contain PCDD and PCDF compounds as impurities in the active ingredients is another potential source of release. Based on sales of technical grade active ingredients in pesticides, and the assumption that all contaminants present in the TGAI are eventually used and enter the environment, the Pest Management Regulatory Agency (PMRA) of Health Canada estimates approximately 1.1 g TEQ are released to soils annually (Health Canada 2000). Nearly all dioxins and furans (99%) released from pest control products are from fungicidal compounds (Health Canada 2000). Due to the assumptions of sales and use of technical grade active ingredients, this value may overestimate actual concentrations released to soils (Health Canada 2000).



(total emissions = 19 g TEQ·a^{-1}; percentages based on loadings rounded to the nearest gram)

(data source: Environment Canada 2001a)

Figure 3.3. Estimates of PCDD/F releases to soils by Canadian municipal and industrial sources for 1999

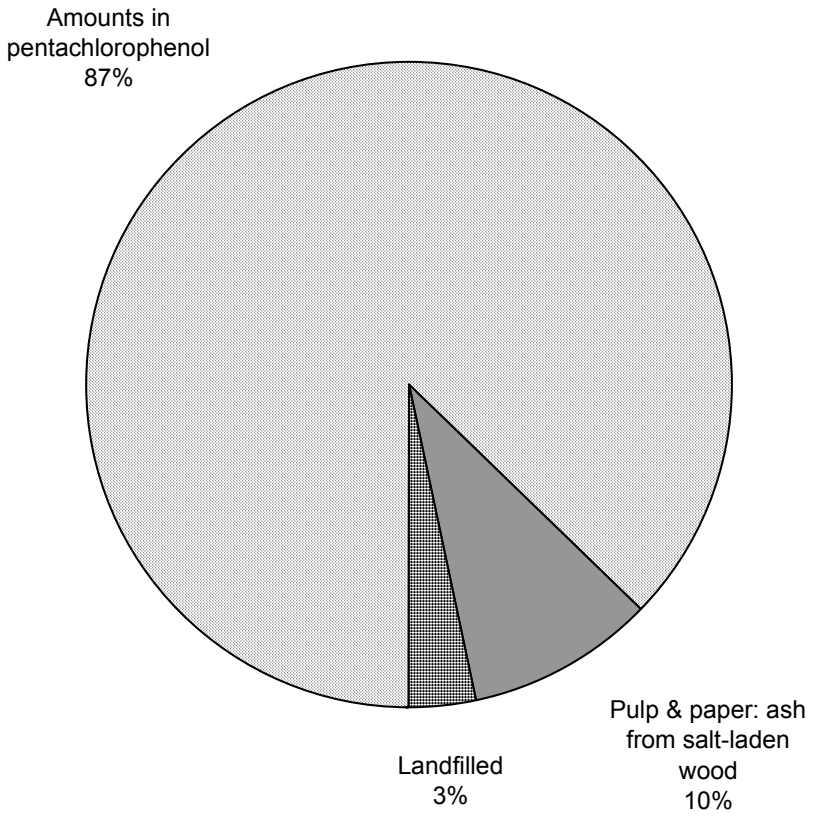
3.2.4 PCDD/Fs in Solid Waste

Solid waste products represent the greatest reservoir for dioxin and furan compounds in the Canadian environment (1097 g TEQ·a^{-1}). This estimate has declined by 58% from the 1990 estimate of 2633 g TEQ·a^{-1}, due almost entirely to reductions from the pentachlorophenol production sector (Environment Canada 2001a). Pentachlorophenols contain the largest amount of PCDD/F compounds (87%) among the solid wastes (Figure 3.4).

Pentachlorophenol (PCP) is a wood preservative known to contain trace amounts of PCDD/F impurities (CCME 1997). Many of the historical uses of PCP in Canada have been restricted in recent years through the *Pest Control Products Act*. Nonetheless, CPI Product Profiles (1991) reported that in 1990, 1000 tonnes of PCP were used at wood preservation facilities. In 1999, an estimated 956 g TEQ·a^{-1} were introduced into the solid waste stream based on the annual sales volumes (and assumed use) of PCDD/F compounds in active ingredients of PCP products (Health Canada 2000; Environment Canada 2001a).

PCDD/F levels in residual ash from hogged fuel at British Columbia coastal pulp and paper mills contribute 105 g TEQ·a^{-1} (10% of total) to the solid waste stream (Environment Canada 2001a). The majority (54%), are contained in ash deposits from one facility in Elk Falls. Ash deposits at eight of the ten facilities across the province (including the Elk Falls plant), are sealed and managed to collect any leachate, with the remaining facilities most likely disposing of ash in landfills. Additional information on ash residues from this sector is pending (Environment Canada 2001a).

Approximately 36 g TEQ·a^{-1} (3% of total) of PCDD/Fs in solid waste are deposited in landfills nationally (Environment Canada 2001a). A wide variety of pulp and paper products are contaminated with PCDD/Fs (Berry et al. 1989) and their ultimate disposal (i.e., burning or landfilling) could result in additional releases to the environment (Sheffield 1985). Finished bleached paper products that contain PCDD/Fs include newsprint, laboratory filter paper, coffee filters, cosmetic tissue, recycled scrap paper, milk cartons and other bleached paperboard containers (Beck et al. 1988; Kitunen and Salkinoja-Salonen 1989; Beck et al. 1990; Ryan et al. 1991; Safe 1991).



(total emissions = 1097 g TEQ·a^{-1}; percentages based on loadings rounded to the nearest gram)

(data source: Environment Canada 2001a)

Figure 3.4. Estimates of PCDD/Fs in solid wastes from Canadian municipal and industrial sources in 1999

4. FATE AND BEHAVIOUR OF PCDD/Fs IN AQUATIC SYSTEMS

The major pathways for 2,3,7,8-substituted PCDD/Fs in the environment are illustrated in Figure 4.1. As by-products of human industrial processes, dioxins and furans will primarily enter the aquatic environment through direct effluent discharge from chlorinated wastewaters, through wet and dry deposition of volatilized compounds present in the atmosphere, or from surface runoff.

4.1 Water

Each of the seventeen 2,3,7,8-substituted PCDD/F congeners has extremely low solubility in water (i.e., <0.5 $\mu g \cdot L^{-1}$) (Table 2.3). Other physico-chemical properties of these substances, such as log K_{ow} and log K_{oc} suggest that PCDD/Fs are likely to form associations with both dissolved and particulate organic matter upon entry into aquatic ecosystems (Table 2.3; Webster et al. 1986). Because they are hydrophobic, the majority of PCDD/Fs released into aquatic systems become associated with the organic carbon in suspended or bed sediments, or the tissues of aquatic organisms (Corbet et al. 1988).

Photolysis may be a significant degradation process for aqueous PCDD/Fs under certain circumstances. Half-lives of PCDD/Fs generally increase with the degree of chlorination, and decrease with solar intensity. Using phototransformation data, molecular extinction coefficients, and solar intensity data for water bodies at 40°N latitude, Choudhry and Webster (1986), estimated the photolytic half-lives for six PCDD congeners (1,2,7,8-TCDD, 1,3,6,8-TCDD, 1,2,3,4,7-PCDD, 1,2,3,4,7,8-HCDD, 1,2,3,4,6,7,8-HCDD, and OCDD). Although direct relationship between the number of chlorine atoms and photolysis rates was not detected, half lives in summer were lowest for 1,2,7,8-TCDD (0.3 d) and highest for 1,2,3,4,6,7,8-HCDD (47.3 d). During seasons of lower solar intensity, half-lives increased, with ranges in spring of 0.35 to 56 days, in fall from 0.53 to 88 d, and in winter from 0.84 to 156 d (Choudhry and Webster 1986). Other studies cited in Environment Canada (2000b) measured half-lives of congeners in pond water (Corbet et al. 1988) and in distilled water (SRC 1989a; Howard 1991). In a study conducted by Friesen et al. (1990), the half-lives of 1,2,3,4,7-PCDD and 1,2,3,4,6,7,8-HCDD in pond water exposed to midsummer sunlight at latitude 50ºN were 0.94 and 2.5 days, respectively. Furthermore, Friesen et al. (1990) determined that the half-lives for the photolytic degradation of 2,3,7,8-TCDF and 2,3,4,7,8-PCDF in lake water were 1.2 and 0.19 days, respectively. Together, these data indicate that aqueous photolysis is likely an important fate process in shallow water, especially for the lower chlorinated PCDDs, during periods of high incident solar radiation. The production of lower chlorinated, more toxic, T_4CDD and P_5CDD congeners could result from the aqueous photolysis of higher chlorinated, less toxic, H_7CDD and OCDD congeners (Miller et al. 1989).

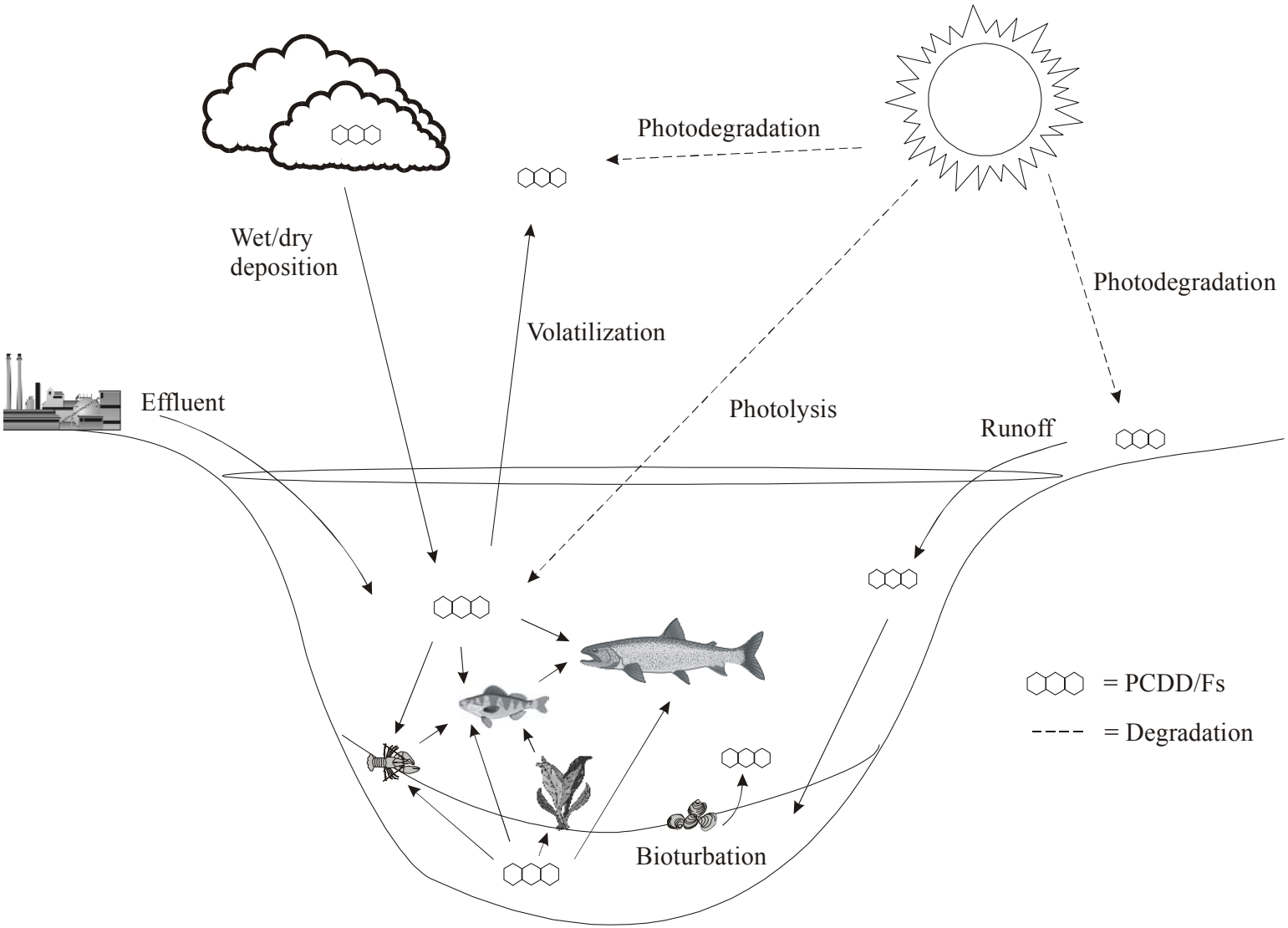


Figure 4.1. General pathways for the movement of polychlorinated dibenzo-*p*-dioxins and polychlorinated dibenzofurans in the aquatic environment.

Studies show that biodegradation is a relatively minor environmental fate process in water (NRCC 1981). In a test of 100 microbial strains, only five of these demonstrate any ability to degrade 2,3,7,8-TCDD (Matsumura and Benezet 1973). An unacclimated aqueous aerobic biodegradation half-life is between 1.15 and 1.62 years for 2,3,7,8-TCDD whereas the anaerobic biodegradation half-life is between 4.58 and 6.45 years (Howard 1991).

The physico-chemical properties of PCDD/Fs suggest that volatilisation is likely an insignificant fate process under most circumstances. Using available data on vapour pressure, the estimated half-life for 2,3,7,8-TCDD volatilisation from a pond is 5.5 years, whereas, that from a lake is 12 years; the difference is likely due to the former having a greater surface area - volume ratio than the latter (OMOE 1985). As the vapour pressures of PCDD/Fs decrease with increasing chlorination, longer half-lives are predicted for the higher molecular weight PCDD/Fs (Hutzinger and Blumich 1985). In accordance with these predictions, the volatilisation of 1,3,6,8-TCDD is minimal during the first 34 days after adding it to outdoor pools (Corbet et al. 1988). Somewhat surprising, however, was the detection of significant quantities of OCDD in air samples collected above the surfaces of outdoor ponds treated with 340 and 680 $ng \cdot L^{-1}$ of ^{14}C-OCDD (Marcheterre et al. 1985).

No data were located on the fate of PCDD/Fs in groundwater. The half-life of 2,3,7,8-TCDD in groundwater is between 2.3 and 3.2 years (SRC 1989a; Howard 1991). This estimate is based on aerobic biodegradation rates observed for this substance in a soil column study in which aerobic groundwater was continuously percolated through quartz sand (Kappeler and Wuhrmann 1978).

The most important fate process for PCDD/Fs released into surface waters is adsorption to organic matter. Concentrations of PCDDs in the water phase (dissolved and particulate form) have been shown to decline rapidly in mesocosm studies, partitioning to suspended particulate matter and dissolved organic matter (Servos et al. 1992). Two studies reporting aqueous half lives of PCDD/Fs have been described in Environment Canada (2000b). Half-lives of congeners were in the order of 14 to 28.5 hours for of 1,3,6,8-TCDD in outdoor pools (Corbet et al. 1988), and 2.6 and 4 days for 2,3,7,8-TCDD and OCDD, respectively, in large lake enclosures of northern Ontario (Servos et al. 1992). Therefore, adsorption to organic matter results in the rapid removal of PCDDs from the aqueous phase of surface waters. In relation to toxicity, this fate process is important because it decreases the bioavailability of PCDD/Fs in the water column, or the fraction of chemical that is available for uptake by aquatic organisms (Suffet et al. 1994). This is not to suggest, however, that all sorbed PCDD/Fs are rendered unavailable for uptake and assimilation by organisms but because this exposure route is likely minor and extremely difficult to quantify, it has not been studied directly.

Another reason that adsorption to organic matter is an important aquatic fate process is that the PCDD/Fs bound to organic matter are then deposited onto the bed sediments, making sediments a major sink for PCDD/Fs that enter the water column. For example, after 34 days, 34 to 80% of the 1,3,6,8-TCDD added to test systems are associated with sediments (Muir et al. 1985a; Corbet et al. 1988). Similarly, sharp increases in the concentration of 2,3,7,8-TCDD in sediments are observed within days of adding this substance to water in a model aquatic ecosystem (Tsushimoto et al. 1982).

4.2 Sediment

PCDD/Fs have high affinities for aquatic sediment and, as such, these substances accumulate to significant levels in this medium (Czuczwa and Hites 1986). Little information was found on

photolysis, hydrolysis, or microbial degradation of PCDD/Fs in aquatic sediments. The results of several laboratory incubation studies suggest that these fate processes are minor. For example, after 675 days under stable aerobic conditions, 80% of the radio-labelled 1,3,6,8-TCDD added to water/sediment systems was still present in pond and lake sediments as the parent compound (Muir et al. 1985a). Similarly, only 1 to 4% of the 2,3,7,8-TCDD added to laboratory sediment/water systems was degraded over a 588 day period (Ward and Matsumura 1978).

The fate of sediment-associated PCDDs is more complex in test systems that are designed to simulate aquatic ecosystems. In a model aquatic ecosystem (roughly 185 m^3) that consisted of water, sediment (4.1% organic matter), two aquatic macrophytes (*Elodea nuttali* and *Ceratophyllum demersum*), and fathead minnows (*Pimephales promelas*), the addition of 3.4 mCi of ^{14}C-2,3,7,8-TCDD (initial measured concentration of 53.7 ng·L^{-1}) resulted in a rapid increase in its concentration in the sediments (up to 2700 ng·kg^{-1} ww) within the first four days of the study (Tsushimoto et al. 1982). Within 50 days, the concentration of 2,3,7,8-TCDD in sediment dropped to 500 ng·kg^{-1}ww, and to 97 ng·kg^{-1}ww within 365 days. During this period, the concentrations of 2,3,7,8-TCDD in the macrophytes and fish increased to over 2000 ng·kg^{-1} ww, indicating significant transfer of this substance into biological tissues, primarily from the sediments. The amount of 2,3,7,8-TCDD associated with macrophytes accounted for more than 85% of the 2,3,7,8-TCDD-radioactivity remaining after 365 days in the mesocosm. After 750 days, sediment-associated 2,3,7,8-TCDD accounted for virtually all of the remaining 2,3,7,8-TCDD-radioactivity measured in the system. It is unclear if this radioactivity was associated with increased levels of 2,3,7,8-TCDD bound to fish faeces; however, the results demonstrate that 2,3,7,8-TCDD may undergo complex cycling between the abiotic and biotic components of the ecosystem.

In a similar study, Servos et al. (1992) investigated the fate of 1,3,6,8-TCDD and OCDD in large (40 m^3) enclosures in Lake 304 of the Experimental Lakes Area of north-western Ontario. These mesocosms consisted of water (2 m deep), sediment (25.4% organic carbon), and the benthic and planktonic organisms that typically occur in the lake. The results of this study indicate that these substances are very stable in bed sediments. After 720 days, sediment-associated 1,3,6,8-TCDD accounted for 57% of the ^{14}C initially added to the mesocosm. Likewise, OCDD in sediment accounted for 55% of the radio-labelled-OCDD originally added to the test system. In shallow outdoor pools, however, only a small proportion (7.9 to 17.7 %) of the 1,3,6,8-TCDD originally added to the test system (which includes water, sediment, rooted plants, and duckweed) associated with sediments after 426 days (Corbet et al. 1988). These investigators suggested that photolysis, uptake and biotransformation by plants, and degradation in sediments are responsible for significant losses of 1,3,6,8-TCDD from sediments.

PCDDs may persist in natural freshwater and marine sediments for long periods (OMOE 1985). For example, low levels of the higher chlorinated PCDD congeners were found in lake sediments that were 300 to 1000 years old (Jansson et al. 1987). Likewise, significant quantities of 1,2,3,4,6,7,9-HCDD (52 ng·kg^{-1}) and OCDD (320 ng·kg^{-1}) were detected in deep sediments from an inland sea in Japan, estimated to be approximately 8120 years old (Hashimoto et al. 1990). These data indicate that PCDDs may be very stable in sediments below the biologically-active layer (i.e., top 5-15 cm), particularly in areas with high sedimentation rates.

Several biological processes redistribute PCDD/Fs within bed sediments and reintroduce these substances into the water column. Many benthic organisms (*e.g.*, tubificid worms, clams, polychaetes) burrow to significant depths in bed sediments, resulting in the mixing of surficial

sediments with deeper materials. For example, tubificid worms mix lake sediments to a depth of 10 cm and release contaminants directly into the water column (Fisher et al. 1980; Karickhoff and Morris 1985). Similarly, clams burrow up to 20-30 cm into bed sediments (Lee, II 1991). Polychaetes are even more effective, burrowing to depths of 50 cm, accounting for more than 90% of the movement of hydrophobic organic contaminants in certain locations (Karickhoff and Morris 1985; Eadie et al. 1988). Thus, historical deposits of PCDD/Fs now found in deep sediments, may be redistributed to less contaminated surficial sediments through bioturbation. Uptake by, and death of, aquatic organisms represents an important cycling process for sediment-associated PCDD/Fs (Tsushimoto et al. 1982).

Once released into the atmosphere, PCDD/Fs tend to adsorb on 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. The major products of photolysis are lower chlorinated dioxins and furans. As a result, the half-life of a PCDD/F mixture, expressed as TEQ (see text box on page 9), would be longer than suggested by each congener's half-life. In general, the rate of photolysis increases as the degree of chlorination increases (USEPA 1994a).

In summary, PCDDs are very stable in sediments and, therefore, tend to persist for extended periods in this environmental matrix; fate processes such as photolysis, hydrolysis, or microbial degradation of PCDD/Fs are believed to be insignificant. As such, PCDD/Fs that are associated with bed sediments may represent long term sources to the aquatic food web (Kuehl et al. 1987b; Muir 1988).

4.3 Air

PCDD/Fs are very stable compounds in the environment. Photodegradation appears to be the only environmentally significant transformation process for these compounds, and is believed to be efficient mainly on nonsorbed species in the gaseous phase, or at the soil-air and water-air interface (USEPA 1994b).

4.4 Soil

Once deposited on soil, there can be an initial loss of PCDD/Fs by photodegradation and/or volatilisation. The extent of these removal processes depends on several factors, including the exposure to sunlight, climatic conditions, and the form under which the PCDD/Fs were deposited on the soil (airborne particulates, sludge applied to land, etc.). Photodegradation is limited to the soil surface, and is not a significant process below the first few millimetres (USEPA 1994a).

In soil, PCDD/Fs exhibit very little potential for significant leaching or volatilisation. This is especially true in soil with a high organic carbon content. As a result, burial in-place and erosion of soil to water bodies appears to be the predominant fate of PCDD/Fs sorbed to soil. The ultimate environmental sink of PCDD/Fs is thus believed to be aquatic sediments (USEPA 1994a). Notwithstanding, concentrations in soils may remain significant for long periods when surface runoff to water bodies and wind erosion are limited.

Many authors suggested different values for the half-life of dioxins in soil, which could be up to ten years if the PCDD/Fs are shielded from sunlight, *e.g.*, at depth in the soil profile or under structures (Jones et al. 1989). These estimates, however, often focused on the single 2,3,7,8-TCDD congener. For this reason, they do not represent the actual behaviour of a mixture of

PCDD/Fs in soil. Half-lives of various congeners of PCDD/Fs in soil were estimated by Mackay et al. (1992). As a general rule, the duration of half-life increases with chlorination level. Because the half-lives of various congeners and the proportions of these congeners in a given mixture may vary from case to case, it is not always appropriate to assess the fate of PCDD/Fs in soil on the sole basis of the 2,3,7,8-TCDD congener half-life. Using the values given by Mackay et al. (1992) for various congeners, Trépanier (1993) estimated that the average half-life of an environmental mixture of PCDD/Fs could reasonably be estimated, in terms of toxic equivalent of 2,3,7,8-TCDD (TEQ, based on I-TEFs) by using an average value of 2 years. The proportions of each congener in the mixture used to obtain this estimate were based on a single soil sample that was contaminated after a fire at a plastic products plant. In fact, establishing adequate or "typical" proportions of the various congeners in soil is quite complex since background levels of most congeners in soil are often below detection limits.

4.5 Aquatic Biota

Aquatic organisms are exposed to PCDD/Fs through direct contact with water and sediment, and through the consumption of contaminated food. The relative importance of each of these exposure routes differs significantly between species and even between various life stages. Batterman et al. (1989) evaluated the importance of each of these exposure routes in lake trout (*Salvelinus namaycush*) and concluded that bioaccumulation occurred primarily through the consumption of contaminated prey species. For carp (*Cyprinus carpio*), however, PCDD/F contaminated bed sediments represented the primary exposure route (van der Weiden et al. 1989b). Direct exposure to PCDD-contaminated water resulted also in bioconcentration of these substances in the tissues of rainbow trout (*Oncorhynchus mykiss*) and fathead minnows (Muir et al. 1985b, 1985c). One study on guppies (*Poecilia reticulata*) under controlled laboratory conditions suggested that for this species water exposure was a more important uptake route for PCDD/Fs than ingestion of contaminated food (Loonen et al. 1993).

PCDD/Fs are highly hydrophobic substances and, as such, are readily accumulated in the tissues of aquatic biota. Yet, PCDD/Fs are atypical of chlorinated aromatic hydrocarbons with comparable hydrophobicity (i.e., polychlorinated benzenes and biphenyls) because their uptake rates are significantly reduced relative to these other compounds (Grimwood and Dobbs 1995). As such, the bioaccumulation of these substances cannot be accurately predicted from their physico-chemical properties (such as log K_{ow}; Table 2.3). Rather, the rate and extent of PCDD/F accumulation in aquatic organisms is dependent on the size of the molecule, position of the chlorine substitution, and the balance between assimilation and depuration rates (Adams et al. 1986; Kuehl et al. 1986; Opperhuizen and Sijm 1990; Sijm et al. 1990).

In rainbow trout and fathead minnows, uptake rates for 1,2,3,7-TCDD and 1,2,3,4,7-PCDD are fast whereas those for larger, more highly chlorinated congeners are slower (Muir et al. 1985b, 1985c). Assimilation efficiencies in both rainbow trout and fathead minnows are greatest for 1,2,3,4,7,8-HCDD (37 and 17%, respectively), whereas efficiencies are less for lower and higher chlorinated congeners (Muir and Yarechewski 1988). In rainbow trout, laterally substituted furans 2,3,7,8-TCDF (59%) and 2,3,4,7,8-PCDF (41%) are more efficiently assimilated than six non-laterally substituted furans (10-27%) and OCDF (18%) (Muir 1991). In comparison, the assimilation efficiency of 2,3,4,7,8-PCDF in guppies is high (34%), whereas that for 2,3,7,8-TCDF is relatively low (3.6%) (Loonen et al. 1991).

Data suggest that depuration rates are species, life stage, and congener specific. A depuration half-life for 2,3,7,8-TCDD was 18 weeks in yellow perch (*Perca flavescens*) administered a single oral dose (Kleeman et al. 1986b). Similarly, depuration half-lives for 2,3,7,8-TCDF and

2,3,4,7,8-PCDF in rainbow trout were approximately 10 weeks (Muir 1991). In contrast, depuration half-lives for non-laterally substituted furans were <1 to 3 weeks. 1,2,3,7-TCDD and 1,2,3,4,7-PCDD were eliminated rapidly with half-lives of 2.7 and 2.5 days, respectively, in rainbow trout fry and 2.8 and 3.2 days, respectively, in fathead minnows (Muir et al. 1985b). The half-lives of the higher chlorinated 1,2,3,4,7,8-HCDD and 1,2,3,4,6,7,8-HCDD were longer in both rainbow trout (~16 days) and fathead minnows (~20 days) than the lower chlorinated congeners.

Depuration rates depend not only on the elimination of the parent compound, but also on the extent to which the parent compound may be converted into readily excretable metabolites (Opperhuizen and Sijm 1990). Biotransformation refers to the process in which one or more biochemical reactions transform a parent compound into a derivative (i.e., metabolite) that 1) may be used to satisfy metabolic requirements, or 2) is detoxified and/or excretable or non-absorbable (Norstrom and Letcher 1997). Data suggest that PCDD/F metabolites, in addition to being excretable, are much less toxic than their parent hydrocarbons (Weber et al. 1982; Ahlborg et al. 1992). In some cases, however, biotransformation of xenobiotics by cytochrome P-450 (mixed function oxydases) is not beneficial because metabolites are more toxic or biologically active than the parent compound (*e.g.*, PAH compounds such as benzo[a]pyrene; McFarland and Clarke 1989). This scenario does not seem to apply to PCDD/Fs.

Studies indicate that fish are able to biotransform several congeners though in some fish this ability may be somewhat limited beyond trichloro-substitution. Gobas and Schrap (1990) evaluated the ability of guppies to metabolise dioxins finding mono-, di- and tri-hydroxy transformation metabolites of 2-MCDD, 2,7-DCDD, and 1,2,3-TCDD but no metabolites for 1,2,3,4-TCDD, OCDD or OCDF in the fish or water. Kleeman et al. (1986a) found that of the 2,3,7,8-TCDD administered in the diet of yellow perch, only 1 to 3% was transformed, with the gallbladder containing almost all of the 2,3,7,8-TCDD metabolites. One of these metabolites was a glucuronide conjugate. In contrast, rainbow trout and fathead minnows are able to readily biotransform some of the more highly chlorinated congeners. For example, 68 to 77% of radio-labelled 1,2,6,8-TCDD was recovered as a metabolite in the bile of rainbow trout following enzyme hydrolysis; the metabolite was a conjugate of a hydroxylated tetrachloro-derivative. Similarly, less than 30% of radio-labelled 1,2,3,7-TCDD and 1,2,3,4,7-PCDD were toluene-extractable from rainbow trout and fathead minnows (whole fish) indicating extensive transformation to polar metabolites (Muir and Yarechewski 1988). Extraction efficiencies indicated that rainbow trout but not fathead minnows transformed 1,2,3,4,7,8-HCDD; neither species transformed 1,2,3,4,6,7,8-HCDD (Muir and Yarechewski 1988). Rainbow trout transformed 2,3,7,8-TCDF to a glucuronide conjugate (Muir et al. 1992a). The whole body elimination rate of 2,3,7,8-TCDF was faster at higher concentrations presumably due to enhanced biotransformation to polar metabolites (Muir et al. 1992a).

PCDD/Fs are distributed throughout the tissues of aquatic organisms with preferential accumulation in tissues with high lipid content (i.e., >4%; Sijm et al. 1990). In rainbow trout, the muscle, skin, liver and intestine are major storage sites for 1,2,3,7-TCDD, 1,2,3,4,7-PCDD, and 2,3,4,7,8-PCDF (Sijm et al. 1990). For example, the visceral fat, carcass, skin, pyloric caeca, and all fatty tissues of rainbow trout retained greater than 90% of the 2,3,7,8-TCDD dose during a 13 week exposure period (Kleeman et al. 1986a). Similarly, in yellow perch, the carcass (including head, fins, bones, and cartilaginous material) and visceral fat contained nearly 80% of the total body burden of 2,3,7,8-TCDD (Kleeman et al. 1986b). Whittle et al. (1993) found that based on lipid content, the crab hepatopancreas contains disproportionately high levels of several PCDD/F congeners owing to the detoxifying function of this organ.

4.6 Mammals and Birds

Mammals and birds take up PCDD/Fs primarily through direct consumption of food, and to a lesser extent through consumption of water and incidental consumption of contaminated sediment. Ankley et al. (1993) studied the uptake of PCDD/Fs by Forster's terns (*Sterna forsteri*), common terns (*Sterna hirundo*), tree swallows (*Tachycineta bicolor*), and red-winged blackbirds (*Agelaius phoeniceus*) at upper trophic levels of two aquatic food chains of the Lower Fox River and Green Bay, Wisconsin. Young birds accumulated small concentrations of 2,3,7,8-TCDD and other PCDD/Fs. Forster's tern chicks took up 1,2,3,6,7,8-HCDF at a rate of 0.001 $ng \cdot d^{-1}$, with greater concentrations of 1,2,3,6,7,8-HCDF, 1,2,3,7,8-PCDD, 1,2,3,6,7,8-HCDD, and 1,2,3,4,6,7,8-HCDD occurring in the chicks than in the eggs from the same nest (Ankley et al. 1993).

Mammals and birds do not possess an efficient elimination route compared to aquatic organisms which can eliminate PCDD/Fs efficiently across their gills (Loonen et al. 1996). In mammals and birds, depuration is possible only through metabolism and subsequent excretion. In mammals, lactating females eliminate PCDD/Fs via milk to their young, whereas in birds, females eliminate PCDD/Fs via eggs (Elliott et al. 1989; Ahlborg and Hanberg 1992). In herring gulls (*Larus argentatus*), mothers transferred 2,3,7,8-TCDD, 1,2,3,6,7,8-HCDD, 1,2,3,7,8-PCDD, 2,3,4,7,8-PCDF, and 2,3,7,8-TCDF to their eggs where the highest concentration was 83 $ng \cdot kg^{-1}$ ww for 2,3,7,8-TCDD (Braune and Norstrom 1989). As chlorination increases, maternal transfer of PCDD/Fs to the eggs decreases despite increased PCDD/F retention in the mothers' livers (Braune and Norstrom 1989).

The rate at which dioxins are eliminated from the body varies from species to species (Ahlborg and Hanberg 1992). For birds, the half-life for whole body elimination of PCDD/Fs was 378 days in nonlaying adult hen pheasants (Nosek et al. 1992). Based on comparisons to other compounds (*e.g.*, hexachlorobenzene) with known half-lives, Braune and Norstrom (1989) speculated that in herring gulls the whole body half-life for 2,3,7,8-TCDD was 100 days.

As described above for fish, the metabolism of 2,3,7,8-TCDD and related compounds (with the exception of OCDD) is necessary prior to urinary and biliary elimination; therefore, the rate of depuration of these compounds is dependent, in part, on the rate of biotransformation (van den Berg et al. 1994b). PCDD/Fs with fewer chlorine atoms are usually biotransformed and eliminated faster than higher chlorinated congeners. In addition, PCDFs are generally metabolised and excreted faster than their corresponding PCDD analogues. In birds, (*e.g.*, herring gulls), 2,3,7,8-TCDF typically contributes a small proportion to the total PCDD/F concentration relative to 2,3,7,8-TCDD, suggesting faster metabolism and shorter half-life for 2,3,7,8-TCDF (Braune and Norstrom 1989). Some animals retain preferentially 2,3,7,8-substituted congeners over other congeners.

When PCDD/Fs accumulate in the body, they are stored in fat and fatty tissues (*e.g.*, liver) (Government of Canada 1990). Small amounts of PCDD/Fs occur in skin, muscle, and other organs, generally in proportion to the fat content of the tissue. TEQ levels (based on I-TEFs) in caribou (*Rangi tarandus*) from the Canadian Arctic were greater in fat tissue than in liver or muscle (0.70, 0.43, < 0.01 $ng \cdot kg^{-1}$ ww, respectively) in 3 year old caribou from the Finalyson herd (Hebert et al. 1996). The livers of herring gulls contained 5 to 55% of the total PCDD/F body burden (Braune and Norstrom 1989).

4.7 Bioconcentration, Bioaccumulation, and Biomagnification

Bioaccumulation and bioconcentration are sometimes used interchangeably to describe the accumulation of organic contaminants in biota; however, the two terms have different meanings and are dependent on the routes by which a contaminant is accumulated. Bioconcentration refers to the direct uptake of compounds from water and retention in the tissues of aquatic organisms, whereas bioaccumulation involves the biological uptake of substances from all environmental compartments, including water, food, and sediment (Branson et al. 1985; Muir et al. 1992b). Biomagnification refers to the increase in tissue concentrations of accumulated chemicals from one trophic level to the next (i.e., organisms contain higher concentrations of the substance than their food sources). The degree to which these three factors occur depends on environmental degradation, sorption, uptake, elimination and biotransformation.

4.7.1 Bioconcentration/Bioaccumulation Factors

Bioconcentration data are generally reported as bioconcentration factors (BCFs) which are defined as the contaminant concentration measured in the biota divided by the contaminant concentration in the water. Ideally, the BCF should reflect a steady-state condition, where the BCF remains constant over time and is described by Oliver and Niimi (1985) in the following equation:

$BCF = C_b/C_w = k_1/k_2$

where:

BCF = bioconcentration factor;

C_b = chemical concentration in the organism ($mg \cdot kg^{-1}$ ww);

C_w = chemical concentration in the water ($mg \cdot L^{-1}$);

k_1 = uptake rate constant; and

k_2 = elimination rate constant.

According to the Toxic Substances Management Policy, a substance is considered bioaccumulative if its BCF in fish is greater than 5000 (Government of Canada 1995).

The distinction between bioaccumulation and bioconcentration is sometimes lost in the literature. Many research efforts have been devoted to measuring PCDD/F accumulation in fish from both processes but frequently values reported as bioconcentration factors are in fact, bioaccumulation factors (BAFs), because they include contributions to total body burdens from food and sediment as well as water exposure pathways. For example, 'BCFs' derived from field data are not considered true BCFs because presumably organisms in the field are ingesting food or other organic matter containing PCDD/Fs in conjunction with uptake across the gills. It is also possible that these organisms are taking up PCDD/Fs through contact or ingestion of contaminated sediment.

The duration of exposure, levels of dissolved organic carbon, concentration of the congener in the water, and species and life stage of the test organism affects the BCF. A detailed summary of published, laboratory derived, BCFs can be found in Environment Canada (2000b). The BCFs were converted to lipid-based values wherever possible to remove some of the variability

associated with different species of aquatic organisms and different life stages. BCFs for 2,3,7,8-substituted congeners are included as these are the most toxic forms and are generally the only congeners reported in tissue samples of higher organisms (van den Berg et al. 1994b). Information pertaining to invertebrate species is not included because studies containing data on the uptake of PCDD/Fs were invariably confounded by the contribution to total exposure by contaminated foodstuffs.

BCFs at steady-state or estimated at steady-state are highest for 2,3,7,8-TCDD with $BCFs_{lipid}$ ranging from 50 900 for rainbow trout to 5 100 000 for medaka, *Oryzias latipes* (Servos et al. 1989; Schmieder et al. 1995). Penta-chlorinated dioxins and furans bioconcentrate to relatively high levels; the $BCFs_{lipid}$ for 1,2,3,7,8-PCDF/1,2,3,4,8-PCDF, 2,3,4,7,8-PCDF, and 1,2,3,7,8-PCDD are 21 400, 240 000, and 331 000, respectively, in guppies (Loonen et al. 1994). The $BCFs_{lipid}$ at steady-state (or estimates thereof) for hexachlorinated congeners are slightly lower, indicating that they do not accumulate to as great a degree as do pentachlorinated dioxins and furans. For the hexachlorinated dioxins and furans, the $BCFs_{lipid}$ range from 11 360 for 1,2,3,4,7,8-HCDD in rainbow trout to 174 000 for 1,2,3,6,7,8-HCDD and 1,2,3,6,7,8-HCDF in guppies (Servos et al. 1989; Loonen et al. 1994). 2,3,7,8-TCDF accumulates to an even lesser degree with $BCFs_{lipid}$ ranging from 21 400 in guppies to 120 980 in rainbow trout (Mehrle et al. 1988; Loonen et al. 1994). Higher-chlorinated congeners (i.e., hepta- and octa-chlorinated) show the least accumulation with a BCF_{lipid} at a minimum of 2710 in fathead minnow for 1,2,3,4,6,7,8-HCDD (Muir et al. 1985b). High $BCFs_{lipid}$ exist for the latter congener (560 540 and 635 780 in rainbow trout; Servos et al. 1989) in relation to the maximum recorded for the heptachlorinated furans and octachlorinated congeners (BCF_{lipid} of 42 700 for 1,2,3,4,6,7,8-HCDF in guppies; Loonen et al. 1994).

The molecular size and/or the solubility characteristics of the molecule may explain the decrease in bioaccumulation observed with the higher chlorinated congeners (i.e., the hexa-, hepta- and octachlorinated dioxins and furans). Membrane permeation by hydrophobic compounds that have an effective cross section larger than 0.95 nm is minimised, thus preventing uptake via the gills (Opperhuizen and Sijm 1990). This idea has been substantiated by other authors who found that the low BCFs of hexa- and octachlorinated congeners were primarily due to steric and solubility factors affecting membrane permeation, rather than to low bioavailability caused by binding with organic carbon (Muir et al. 1985b, 1985c; Gobas and Schrap 1990). Strong binding with organic carbon is associated with compounds that have low water solubility, and has been put forth as another explanation for the low accumulation of the higher chlorinated congeners observed in aquatic organisms. Low water solubility partially accounts for low bioaccumulation because compounds with low water solubility require a longer time to attain steady state conditions than those with high water solubility. Molecular size, water solubility, and sorption to organic matter are related issues that may affect the uptake of the higher chlorinated dioxins and furans.

To summarise, all 2,3,7,8-substituted PCDD/Fs readily concentrate in the tissues of aquatic organisms, with the lower chlorinated PCDD/Fs bioaccumulating, in general, to a greater degree than the higher chlorinated congeners. $BCFs_{lipid}$ recorded for 2,3,7,8-TCDD are the highest of all congeners, ranging from 51 300 to 1 700 000 for resident species. In general, those for PCDFs are lower than their PCDD analogues. These $BCFs_{lipid}$, however, reflect only freshwater conditions as data pertaining to marine/estuarine environments were not available.

4.7.2 Biota-Sediment Accumulation Factors

Bioaccumulation via the food chain, originating in the organic fraction of the sediments, is an important path of uptake of PCDD/Fs by aquatic organisms. In a mesocosm study, emerging aquatic insects alone were capable of removing a small, but biologically significant, portion of 2,3,7,8-TCDF from sediments each year (Fairchild et al. 1992). Aquatic insects are a significant food source for many aquatic and terrestrial predators. As such, they are likely an important link between sediment-associated contaminants and food chains. Hence, the sediments, and in particular the organic fraction, serve as a reservoir for uptake of PCDD/Fs by benthic organisms at the base of the food chain.

While BCFs describe the relationship between the concentration of a compound in an organism and the level of the compound in the surrounding aqueous environment, concentrations of a compound in invertebrates and fish are linked also to concentrations in sediment (Carey et al. 1990; Cook et al. 1991; Muir et al. 1992b, 1992c). Due to the affinity of PCDD/Fs for the fatty tissues in organisms and for the organic fraction of sediments, a biota-sediment accumulation factor (BSAF), sometimes called a bioavailability index (BI), is used commonly to characterise the tissue/sediment relationship. This BSAF is an accumulation factor (concentration in organism on a lipid basis divided by the concentration in sediment organic carbon) that assumes equilibrium of the contaminant between the two compartments. Adjusting or normalising for lipid content of the organism and for organic carbon content of the sediment generally reduces data variability. For example, no statistically significant relationships are found between concentrations in suckers and whitefish and concentrations in sediment unless the data are normalised (Muir et al. 1992b). Similarly, Lake et al. (1990) showed that normalising BSAFs for PCBs in molluscs and polychaetes reduces variability, as did Carey et al. (1990) for 2,3,7,8-TCDD in several Lake Ontario fish species.

Field and laboratory BSAFs for PCDD/Fs for freshwater and marine/estuarine organisms are summarised in detail in Environment Canada (2000b). Due to the limited available data, it is difficult to assess differences in BSAFs for 2,3,7,8-TCDD between freshwater and marine/estuarine environments. In general, fish consuming detritus at the sediment-water interface (i.e., suckers and carp; BSAFs of 0.14 to 0.96; Mah et al. 1989; Muir et al. 1992b), fish preying on filter-feeding insects (i.e., whitefish; BSAFs of 0.28 to 1.88; Mah et al. 1989; Muir et al. 1992b), and invertebrates residing in or on the sediments (i.e., worms, clams and crab; BSAFs of 0.14 to 2.4; Schrock et al. 1997; Yunker and Cretney 2000) have the highest accumulation factors. Pelagic species such as lake trout (*Salvelinus namaycush*), brown trout (*Salvelinus trutta*), and smallmouth bass (*Micropterus dolomieui*), that have relatively much less contact with the sediments, have lower BSAFs for 2,3,7,8-TCDD (0.03 to 0.11; Carey et al. 1990). BSAFs were higher for PCDDs than PCDFs. Winter flounder (*Pseudopleuronectes americanus*) exposed to sediments spiked with raw sewage from St. John's harbour (Newfoundland) has BSAFs varying from 1 to 1.5 for 2,3,7,8-TCDF, the only congener present in fish tissue (Hellou et al. 1999). Field-derived BSAFs for four 2,3,7,8 substituted PCDD/Fs in lake trout were 1 to 3 whereas BSAFs for other congeners were well below 1 (Niimi 1996a). Similarly, non-2,3,7,8-substituted congeners had lower BSAFs than other congeners in a field study by Yunker and Cretney (2000). BSAFs for PCDD/Fs in Dungeness crab (*Cancer magister*) from pulp mill sites in British Columbia decreased with the degree of chlorination of congeners and with log K_{ow} (2.4 for 2,3,7,8-TCDD to 0.07 for OCDD; BSAF of 4 for 2,3,7,8-TCDF to 0.03 for OCDF) (Yunker and Cretney 2000). In contrast, other studies reviewed by Niimi (1996b) suggest little difference between BSAFs for lateral (2,3,7,8 substituted) and non-lateral substituted PCDD/Fs.

The variability in field-derived BSAFs can be caused by several factors. Site-specific conditions, such as the concentration of the compound in the sediment and percent organic matter, as well as the type of tissue (*e.g.*, liver, muscle, etc.) in which the compound is measured influence the magnitude of field-derived BSAFs. Moreover, violations of the assumption of steady-state conditions for field-derived data, a condition which is difficult to ascertain, could be a source of the variability in the data. Also, sediment concentrations of PCDD/Fs reported in the literature were often below or near analytical detection limits. Although there is a certain amount of variability inherent to BSAFs, these measures provide valuable estimates of uptake of PCDD/Fs by aquatic organisms in relation to exposure to contaminated sediments.

4.7.3 Biomagnification

Although it is generally accepted that the food chain is the major source of lipophilic halogenated aromatic hydrocarbons for wildlife, the trophodynamic behaviour of PCDD/Fs is poorly understood. PCDD/Fs appear anomalous compared to other halogenated aromatic hydrocarbons in that they do not biomagnify to an appreciable degree (Grimwood and Dobbs 1995; Niimi 1996). For example, biomagnification factors for PCDD/Fs, although variable, are typically less than one over several trophic levels in a Lake Ontario food chain. In contrast, BMFs for PCBs from the same study often exceed 100 (Niimi 1996). This author suggested that dietary adsorption efficiencies for PCDD/Fs, which are consistently lower and more variable than those for PCBs, and high elimination efficiencies could largely account for the lower BMFs observed for PCDD/Fs. In addition, PCDD/Fs accumulate differently from other organochlorine compounds in that they attain higher residue levels in filter feeding molluscs and bottom feeding fish than top predator fish (Whittle et al. 1993).

Lipid-normalized BMFs for lake trout exposed to environmentally relevant dietary concentrations of ^{3}H-2,3,7,8-TCDD for a period of 50 to 200 days were generally between 0.5 and 1.0 and ranged from 0.17 to 1.5 (Jones et al. 2001). Low BMFs have also been reported for mammalian and avian species that consume aquatic biota. For instance, BMFs for mink (*Mustela vison*), normalised to an average consumption of 0.22 g of food (g of mink)$^{-1}\cdot$d^{-1} and based on a 40% carp diet, are 6.4 to 74.2 for PCDDs and <1 to 75.8 for PCDFs (Tillitt et al. 1996). In general, the BMFs increase with the degree of chlorination (Tillitt et al. 1996). Estimated BMFs for herring gulls (*Larus argentatus*) consuming alewife (*Alosa pseudoharengus*) are 32, 20, 14, 6.6, and 1.3 for 2,3,7,8-TCDD, 1,2,3,6,7,8-HCDD, 1,2,3,7,8-PCDD, 2,3,4,7,8-PCDF, and 2,3,7,8-TCDF, respectively (Braune and Norstrom 1989). BMFs estimated from total TEQs (based on H4IIE bioassay) in double crested cormorants (*Phalacrocorax auritus*) eggs and forage fish from sites in the Great Lake region; values range from 11.7 at Thunder Bay to 56.8 in the Beaver Islands with a mean of 31.3 (Jones et al. 1994).

In harbour porpoises (*Phocoena phocoena*) on the Dutch coast, PCDD and PCDF concentrations are relatively low at 4.6 and 2.1 pg$\cdot$kg^{-1} fat, respectively (van Scheppingen et al. 1996). The authors reported that PCDD/Fs concentrations in porpoises are lower than those found in herring from the same area. Moreover, PCDD/Fs in porpoises contribute $\leq$ 0.5% of the total TEQ (PCDD/Fs and PCBs). The authors concluded that PCDD/Fs do not biomagnify in this food chain. Similarly, harbour porpoises off the coast of California do not have any detectable concentrations of PCCD/Fs, although, in the same study, harbour porpoises off the coast of British Columbia have concentrations of 1,2,3,6,7,8-HCDD of up to 128 ng$\cdot$kg^{-1} ww blubber and 2,3,7,8-TCDF of up to 43 ng$\cdot$kg^{-1} ww blubber (Jarman et al. 1996). The authors attributed this difference to the greater number of pulp mills in British Columbia than in California. The relative contribution of bioconcentrated and bioaccumulated PCDD/Fs were not addressed in this study.

In a study of trophic transfer from Canadian ringed seals (*Phoca hispida*) to polar bears (*Thalarctos maritimus*), all seal samples (n=12) and all but one polar bear sample (n=8) had detectable 2,3,7,8-TCDD at concentrations ranging from 2 to 37 $ng \cdot kg^{-1}$ blubber and from 2 to 21 $ng \cdot kg^{-1}$ fat, respectively (Norstrom et al. 1990). All ringed seal samples contained 2,3,7,8-TCDF at levels of 2 to 7 $ng \cdot kg^{-1}$ blubber, but 2,3,7,8-TCDF was not found in any bear sample. No other PCDF congeners were found in seals or bears. 1,2,3,6,7,8-HCDD was found in only two of the ringed seal samples (8 and 9 $ng \cdot kg^{-1}$ blubber) and none of the polar bear samples. OCDD concentrations were found in four of the seals and all but two of the polar bear samples up to 43 $ng \cdot kg^{-1}$ blubber and up to 12 $ng \cdot kg^{-1}$ fat, respectively. The authors concluded that 2,3,7,8-TCDD, OCDD, and 2,3,7,8-TCDF do not biomagnify from seal to bear (Norstrom et al. 1990).

From the limited data presented here and below in the section on environmental levels in biota, it appears that PCDD/Fs can accumulate to appreciable levels in animals but that biomagnification does not occur; the greatest BMFs reported are 32 and 76 for herring gulls and mink, respectively (Braune and Norstrom 1989; Tillitt et al. 1996). Accumulation of PCCD/Fs may still pose a threat to higher trophic level organisms. For example, in eider ducks (*Somateria mollissima*), 57% of the most toxic PCDD/F congeners are retained in duck tissues compared to 10% of the total PCDD/Fs. Thus, the most toxic congeners tend to accumulate even though the total PCDD/F concentration decreases with increasing trophic level (Broman et al. 1992). In a hazard assessment study of the effects of 2,3,7,8-TCDD through the food chain, Loonen et al. (1996) concluded that fish-eating birds and mammals are at a greater risk of detrimental effects from the accumulation of 2,3,7,8-TCDD. The authors found, through back calculation to water concentrations, that the “no effect” concentrations in water are lower for birds and mammals than for fish and invertebrates (Loonen et al. 1996).

5. CANADIAN ENVIRONMENTAL LEVELS OF PCDD/FS

The majority of these sections originate from Environment Canada (2000b). The supporting document has been updated with studies published after 1998. The names of specific pulp and paper mills mentioned in this assessment may have changed since the publication of the reports cited and this assessment.

5.1 Air

As part of an initiative by the Canadian Council of Resource and Environment Ministers (now the CCME), methodology development for ambient air monitoring for PCDD/Fs began in 1987 (Steer et al. 1990). Environment Canada currently maintains a National Air Pollution Surveillance (NAPS) network that samples ambient air concentrations of PCDD/Fs, among other substances, in Canada (Dann 1998). The sampling equipment includes a high-volume sampler with a dry-gas meter or rotary vane meter and a filter-sorbent sampling system to collect particulate plus vapour phase PCDD/Fs. Daily samples were taken from 40 monitoring sites, but the historic record for monitoring data at most of the sites is limited. For the year 2000, mean PCDD/F TEQs (based on I-TEFs and substituting one half on the detection limit for non-detects) across Canada ranged from 0.127 pg TEQ·m^{-3} in Hamilton, ON to 0.009 pg TEQ·m^{-3} in St. Andrews, NB (Dann 2001). Between 1996 and 1999, dioxins were dominated by octa- and hepta-PCDD congeners, while furans were dominated by tetra- followed by penta-PCDF congeners (Dann et al. 2000).

Polychlorinated dibenzo-*p*-dioxins and polychlorinated dibenzofurans are ubiquitous in rural and urban locations across Canada (Dann et al. 2000). In a summary of a ten year (1989-1999) national PCDD/F monitoring program representing 690 sampling days, Dann et al. (2000) reported that for the more hazardous, less chlorinated PCDD/F congeners, 2,3,7,8-TCDD was present in only 13% of all samples, with a mean and maximum concentration of 0.003 and 0.041 pg·m^{-3}, respectively. 2,3,7,8-TCDF, however occurred in 92% of the samples, with a mean and maximum concentration of 0.058 and 1.06 pg·m^{-3}, respectively (Dann et al. 2000). Pooling all national monitoring data for this ten year period, Dann et al. (2000), reported a national average atmospheric PCDD/F concentration (± 1 S.D.) of 0.045 (± 0.065) pg TEQ·m^{-3} (based on I-TEFs). Recent data for the years 1999 to 2001 suggest that overall PCDD/F concentrations are declining relative to levels in the early 1990s in several urban locations. In cities such as Windsor, Hamilton, and Toronto, PCDD/F levels in the early 1990s were often above 0.1 to 0.2 pg TEQ·m^{-3}, but in 2001 very few samples exceeded 0.050 pg TEQ·m^{-3} (based on I-TEFs) (Dann 2001).

Steer et al. (1990) monitored PCDD/F concentrations at three Ontario locations (Toronto Island, Dorset and Windsor) from 1988-1989. Concentrations of the highly toxic 2,3,7,8-TCDD and 2,3,7,8-TCDF congeners ranged from 0.05 to 1 pg·m^{-3} and from 0.02 to 1 pg·m^{-3}, respectively. The authors reported that based on PCDD/F congener patterns detected, dioxin sources (*e.g.*, waste incineration vs. chemical production sources) were substantially different in the industrial Windsor area than both Toronto Island and the rural Dorset location.

Bobet et al. (1990) reported on PCDD/F levels detected in the first year of operation of an ambient air monitoring network in South-western Ontario. Sampling sites included Windsor and Walpole Island, ON. Levels of 2,3,7,8-TCDD and TCDF were below detection limits at both sites. Other PCDD/F congeners were also detected and reported. The total mean PCDD and PCDF concentrations reported for Windsor were 2.12 and 0.46 pg·m^{-3}, respectively. For

Walpole Island the total mean PCDD level was 0.51 pg·m^{-3}. PCDF levels were not reported for Walpole Island.

Atmospheric levels of PCDD/Fs differ between urban and rural sites in Canada. A comparison of sampling sites in southern Ontario over a period of 8 sampling days during 1998 and 1999, when all stations were operating simultaneously, showed that urban site mean TEQs (based on I-TEFs), were between 2 and 5 times higher than nearby rural sites, with a maximum difference occurring between Hamilton (0.071 pg TEQ·m^{-3}) and Pt. Petre (0.014 pg TEQ·m^{-3}) (Dann et al. 2000). Congener profiles show similar distributions between rural and urban sites, with four isomers, 2,3,4,7,8-PCDF, 2,3,7,8-TCDF, 1,2,3,7,8-PCDD and 1,2,3,4,7,8-HCDF accounting for 66% of the calculated TEQ (Dann et al. 2000). Although congener class distributions at rural sites were enhanced in H_7CDD and OCDD groups relative to urban sites, due to their lower toxicity, congeners in these homologue groups did not make up a significant proportion of the total TEQ (Dann et al. 2000).

Seasonal differences in PCDD/F concentrations also occur in relation to urban and rural locations. Total PCDD/F concentrations in the winter (December, January, February) are approximately double those of summer (June, July, August) at urban sites (Montréal QC, and Toronto and Windsor, ON), and triple at rural sites (Pt. Petre and Simcoe, ON) (Dann et al. 2000). During the winter months, the largest enhancement occurs in the tetra-, penta-, and hexa-PCDD congener classes (homologues), at both urban and rural sites (Dann et al. 2000).

5.2 Water

Canadian surface waters may be contaminated with dioxins and furans from an assortment of sources including atmospheric deposition, effluents from pulp and paper mills using chlorine bleaching, chemical manufacturing, waste incineration, petroleum refining, and sewage sludge, among others (Sheffield 1985; Kuehl et al. 1987a; Hicks and McColl 1995). There are difficulties associated with analysing ambient water for these substances at ultra-trace levels because they are adsorbed by particulate matter or are rapidly taken up by biota. Significant amounts of PCDD/Fs in water may therefore indicate recent inputs.

5.2.1 British Columbia

In November, 1988, a directive to close the crab fishery near a pulp mill in Prince Rupert, British Columbia as well as the crab, prawn, and shrimp fisheries in the Howe Sound areas near the Woodfibre and Port Mellon pulp mills was issued by the Federal Department of Fisheries and Oceans (BCMOE 1989). As a precautionary measure for nearby communities, drinking water samples were collected from twelve sites, where intake sources are downstream of effluent discharges from pulp mills (with the exception of one site upstream from the influence of the two Quesnel mills) (BCMOE 1989). The majority of the measured PCDD/F levels were less than the analytical detection limits (5 to 10 pg·L^{-1} depending upon the congener) (BCMOE 1989). In 1989, groundwater samples in British Columbia were collected and analysed for PCDD/F. PCDD/Fs were not detected (detection limits of 3 to 10 pg·L^{-1}) (BCMOE 1989).

5.2.2 Alberta

Public perception surrounding the presence of PCDD/Fs in bleached kraft mill effluent resulted in concerns with respect to the potential impact of mill effluents on drinking water supplies in Alberta municipalities (Milos 1990). In 1989, a dioxin/furan sampling program was initiated for a few chosen communities downstream of existing pulp mills (Milos 1990). Raw and treated

water samples were collected from seven municipalities. No dioxins or furans were measured within the limits of the method detection level (detection limits from 6 to 70 $pg \cdot L^{-1}$ depending upon the congener) at any of the sites. The authors concluded that there was no evidence of a problem with dioxins or furans in raw or treated drinking waters downstream from existing bleached kraft discharges in Alberta (Milos 1990).

Also in 1989, Alberta Environment collected raw and treated drinking water samples from another seven municipalities in upstream and downstream locations of existing kraft mills and analysed these samples for PCDD/Fs (Alberta Environment 1991). Most of the samples had PCDD/F concentrations below their detection limits (from 2.1 to 12.5 $pg \cdot L^{-1}$ depending upon the congener; Alberta Environment 1991).

In the spring of 1992, Environment Canada initiated water quality investigations in a 200 km range of the Athabasca River (from Hinton to Whitecourt, AB) under the Northern River Basin Study (NRBS). Sites influenced by pulp mill effluent and/or sewage treatment plants were sampled regularly. PCDD/Fs were detected in the Athabasca River at low levels up to 230 km downstream of the town of Hinton. In combined effluent from Hinton, 2,3,7,8-TCDD was the only T_4CDD detected (0.35 $pg \cdot L^{-1}$) whereas five T_4CDFs were detected, with 2,4,6,8-TCDF (75 $pg \cdot L^{-1}$) being the major component (Crosley 1996a).

5.2.3 Ontario

In 1980-1981, water samples were collected from 13 water treatment plants scattered throughout Ontario to examine levels of PCDD. PCDDs were not detected in any of the samples (detection limit of 1000 $pg \cdot L^{-1}$; OMOE 1985). In 1981-1982, none of the water samples from nine water works in western Lake Ontario exceeded the improved detection limit for 2,3,7,8-TCDD of 250 $pg \cdot L^{-1}$ (OMOE 1985). Between the spring and the summer of 1995 and 1996, levels of dioxins and furans were below detection limits in all water samples taken from nine stations in Lakes Erie (dissolved fraction: 880 $pg \cdot L^{-1}$; particulate fraction based on a 10 g sample: 0.01 $pg \cdot L^{-1}$) and thirteen stations in Lake Superior (detection limit not reported) (L'Italien 1996, 1998).

Levels of dioxins and furans were also found to be low in several cities in Ontario. Dioxins and furans have not been detected in any sample analysed to date in the city of Toronto's drinking water supply (City of Toronto 2001). In 2001, levels of dioxins and furans in 2 samples of raw drinking water from the City of Owen Sound were below the Ontario drinking water standard of 15 $pg \cdot L^{-1}$ (OMOE 2000). Levels measured were, respectively, 0.1 $pg \cdot L^{-1}$ in the first quarter (January - March) and 2.9 $pg \cdot L^{-1}$ in the second quarter (April - June) (City of Owen Sound 2001a, 2001b). Similarly, levels of dioxins and furans in two samples taken from the City of Ottawa at Britannia and Lemieux Island water purification plants during the year 2000 were below the Ontario drinking water standard (detected level not reported; detection limit: 1.18 $pg \cdot L^{-1}$) (City of Ottawa 2002).

Precipitation was measured in two sites in Ontario, one rural location (Dorset) and one urban location (Toronto Islands), from 1986-1988 (Tashiro and Clement 1989; Reid et al. 1990). The congener most often measured at high levels was OCDD (17 to 1200 $pg \cdot L^{-1}$) (Tashiro and Clement 1989; Reid et al. 1990). In Dorset, samples taken in the winter months had the highest PCDD/F concentrations. The authors suggested that the high concentrations in the winter months were due to residential wood burning, which is expected to be more prevalent in rural areas (Reid et al. 1990). Overall, PCDD and PCDF levels in precipitation samples throughout Ontario are very low.

The Arctic Monitoring and Assessment Programme

The Arctic Monitoring and Assessment Programme (AMAP) was established in 1991, following the adoption of the Arctic Environmental Protection Strategy (AEPS) by the Ministers of the eight Arctic countries (de March et al. 1998). The objective of this program is to compile current knowledge about the Arctic region, by monitoring levels and assessing effects of selected anthropogenic pollutants in all compartments of the Arctic: air, sediment, freshwater, marine water, soil, biota and humans. Further investigation concerning the sources of high contaminant concentrations and comparison between levels found in Arctic villages and towns are based on these monitoring results.

The Arctic communities are not using the technologies associated with the production and use of most chemical substances found in this remote region. The dependency of indigenous people on a diet extremely rich in fat, representing a direct exposure to various contaminants, environmentally trigger the development of a detailed study on persistent and biomagnifying substances in the Arctic.

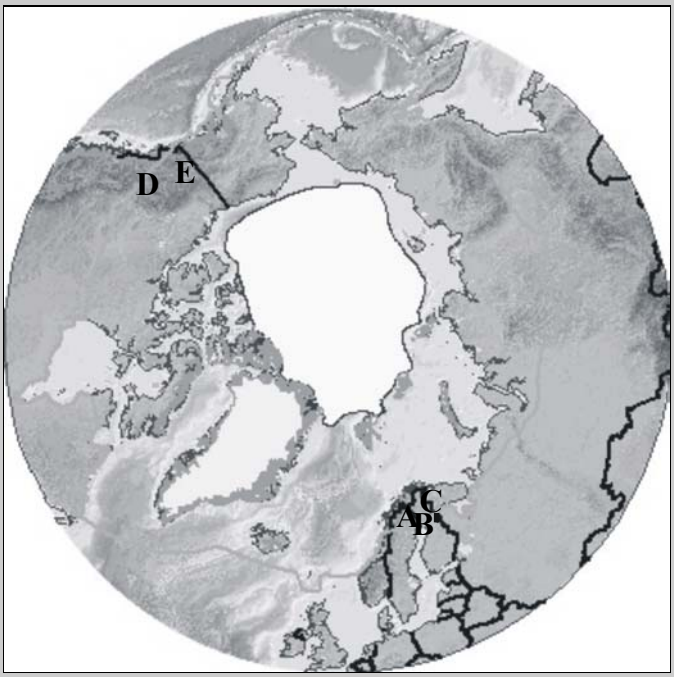
Dioxins and furans were categorised as toxic, persistent and bioaccumulative substances, and were subsequently monitored and studied in various locations across the Arctic. Activities such as combustion, wood burning and metallurgical industries are directly associated with low levels of PCDD/Fs found in the environment, like in the western basin of Great Slave Lake (Evans et al. 1996). Other suspected sources in Canada are waste incineration and pulp and paper mills using chlorine-bleached kraft. A few pulp mills are located within the Arctic Ocean drainage basin, in northern Alberta, on the Wapiti, Peace and Athabasca Rivers and in north-western Ontario and Manitoba (de March et al. 1998).

Freshwater environment

Little information is available on contaminant loading from northward-flowing rivers (de March et al. 1998). PCDD/Fs have been monitored in selected lake sediment cores, originating from Canada, Finland and Norway. PCDD/F concentrations ranged from 0.07 to 0.1 ng TEQ·kg^{-1} dw[2] in Great Slave Lake (Evans et al. 1996), from 1.4 to 4.2 ng TEQ·kg^{-1} dw in various Finland lakes (Vartiainen et al. 1997), and from 2.6 to 68.4 ng TEQ·kg^{-1} dw in four Norwegian lakes (Schlabach and Skotvold 1996a, 1996b). Surface grab samples were generally less contaminated, with concentrations varying from <1 ng TEQ·kg^{-1} dw (Canada) to 4.12 ng TEQ·kg^{-1} dw (Sweden). OCDD was the predominant congener, suggesting the presence of a combustion source in the surroundings. Generally the lower concentrations found in Canada, compared to other Arctic countries, were attributed to higher sedimentation rates in the sampled lakes (de March et al. 1998). TCDD TEQs in sediment samples generally exceeded the Interim Canadian Environmental Quality Guideline for Protection of Aquatic Life of 0.85 ng TEQ_{fish}·kg^{-1} dw.

Char samples collected from the Canadian Arctic had higher TEQ levels (0.14 to 7.4 ng TEQ·kg^{-1} ww) than others sampled in Finland, Norway and Sweden (0.02 to 0.14 ng TEQ·kg^{-1} ww) (de March et al. 1998). Of the three countries sampled, total TEQs in lake whitefish muscle were equal in Canada and Sweden with a value of 0.47 ng TEQ·kg^{-1} ww, and were more contaminated in the Norwegian samples, with a PCDD/F concentration of 8.3 ng TEQ·kg^{-1} ww (de March et al. 1998). As illustrated in Figure 5.1, burbot liver PCDD/F concentrations were higher than levels found in other fish. Lake Laberge, Canada, had the highest level with 166 ng TEQ·kg^{-1} ww (de March et al. 1998). Otherwise, TEQ concentrations were less than 10 ng TEQ·kg^{-1} ww.

[2] PCDD/F TEQs are based on I-TEFs for this entire text box



Sweden:
A) Pajala (3.75 ng TEQ·kg^{-1} ww)

Finland:
B) Pahtajärvi (0.7 ng TEQ·kg^{-1} ww) and
C) Nitsijärvi (2.76 ng TEQ·kg^{-1} ww)

Canada:
D) Lake Laberge (166 ng TEQ·kg^{-1} ww) and
E) Fox Lake (0.8 ng TEQ·kg^{-1} ww).

(data source: de Wit, unpubl. data, Mannio, unpubl. data and Muir and Lockhart 1994; in de March et al. 1998)

Figure 5.1. Total PCDD/F levels found in burbot liver samples.

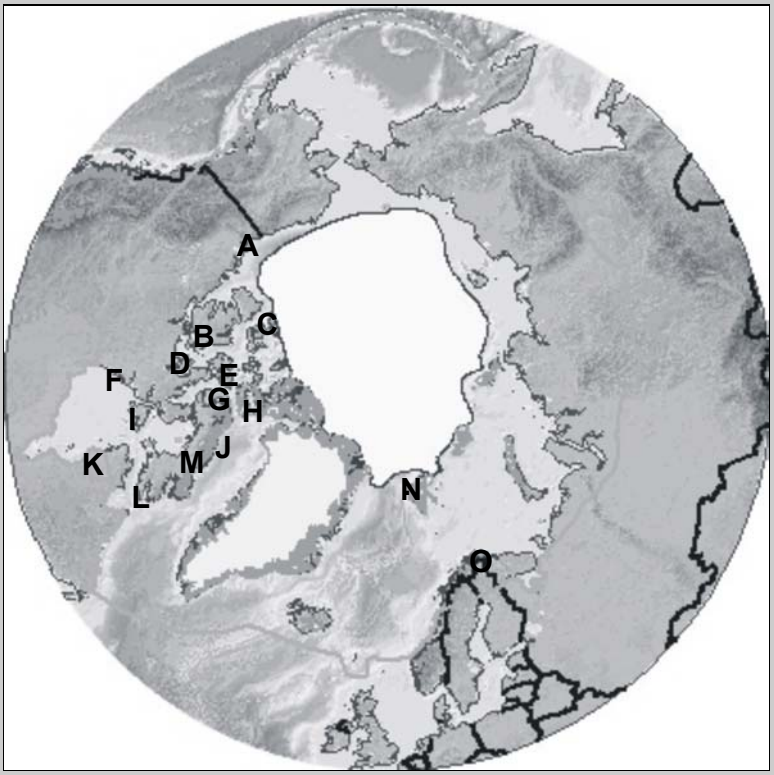
In the Canadian Arctic, fish from the Great Slave Lake had high concentrations of PCDD/Fs compared to smaller surrounding lakes such as Trout, Gordon and Alexie. Moreover, in walleye whole body samples and burbot liver, 2,3,7,8-TCDD/F isomers made up 100% of the total TEQs detected. A bleached kraft mill was identified as the major source of contamination (de March et al. 1998).

Marine environment

PCDD/F levels were determined in marine sediments from northern Norway, Canada and in the Barents Sea. Mean concentrations of PCDD/Fs in core sediment samples from Norway varied between 0.1 and 27 ng TEQ·kg^{-1} dw, with OCDD and H_6CDF as the predominant congeners (Schlabach and Skotvold 1996a, 1996b). In surface grab samples taken from various locations in Canada, mean concentrations varied between 0.2 to 43 ng TEQ·kg^{-1} dw, with TCDD, P_5CDD and H_6CDD as the predominant congeners from the Eskimo Lakes in Mackenzie Delta (MacDonald, unpubl. data 1996 in de March et al. 1998). This pattern is different from the one found in the Mackenzie River Basin in the Great Slave Lake and in European countries, with OCDD as the solely important congener. This can be related to the use of different combustion source between locations with a greater contribution from chlorophenol use in North America compared to Europe (de March et al. 1998).

PCDD/F concentrations in pinnipeds and cetaceans were generally higher in tissue from Barents Sea animals (de March et al. 1998). Profiles in marine mammals blubber differed from the Canadian Arctic and Barents/Greenland animals: ringed seals, polar bears and walrus in the Canadian Arctic had relatively high concentration of 2,3,7,8-TCDD and low concentration of PeCDD/Fs, while in Greenland/Barents Sea, greater levels of 2,3,7,8-TCDF and P_5CDF were found. Moreover, 2,3,7,8-TCDF concentrations in Canadian ringed seals were lower than values reported for Norwegian ringed seals, with levels of 2 to 7 ng TEQ·kg^{-1} ww and 10 to 13 ng TEQ·kg^{-1} ww, respectively. The opposite trend can be observed for 2,3,7,8-TCDD levels: ringed seal blubber from Norway ranged from 3.4 to 12 ng TEQ·kg^{-1} ww (Oehme et al. 1988; Bignert et al. 1989) compared to 2 to 37 ng TEQ·kg^{-1} ww in various locations across the Canadian Arctic (Norstrom et al. 1990). Although these levels were relatively low, they were still higher than those found in Antarctic seals (Oehme et al. 1995a).

PCDD/F concentrations found in subcutaneous fat from polar bears in the Canadian Arctic, in 1983-1984, ranged from 2 to 23 ng TEQ·kg^{-1} ww (Norstrom et al. 1990; Figure 5.2) and a mean of 27 ng TEQ·kg^{-1} ww in liver in the period 1992-1994 was also reported (Letcher et al. 1996). PCDD/F TEQs ranged from 1 to 3.5 ng TEQ·kg^{-1} ww in polar bear milk from Svalbard (Oehme et al. 1995b), with OCDD/F being the predominant congeners.



Canada
A) Tuktoyaktuk (ringed seal: 4.2)
B) Cambridge Bay (ringed seal: 12.4)
C) McClure Strait (polar bear: 18)
D) Spence Bay (ringed seal: 15; polar bear: 23)
E) Barrow Strait (ringed seal: 35.4; polar bear: 20)
F) Rankin Inlet (ringed seal: 2.4; polar bear: 2)
G) Admiralty Inlet (ringed seal: 37.5)
H) Pond Inlet (polar bear: 4)
I) Coral Harbour (ringed seal: 3.7; polar bear 2)
J) Clyde River (polar bear: 5)
K) Inukjuak (ringed seal: 2.4)
L) Lake Harbour (reindeer/caribou: 2.5)
M) Broughton (ringed seal: 11.4; polar bear: 3)

Norwary:
N) Svalbard (ringed seal: 12.9)
O) Jarfjord Homegra (reindeer/caribou: 9.45)

(data source: ringed seal (1986) (Oehhme et al. 1988; Ford et al. 1993; Muir et al. 1995; Norstrom et al. 1990); reindeer/caribou (Muir, unpubl. data; in de March et al. 1998); polar bear (1983/1984) (Norstrom et al. 1990)).

Figure 5.2. Total PCDD/F levels (ng TEQ·kg^{-1} ww) in ringed seals, reindeer/caribou, and polar bears.

Terrestrial and freshwater Arctic animals

Data are limited regarding terrestrial Arctic animals and included a relatively small number of species: reindeer/caribou, mink, otter, peregrine falcon, white-tailed eagle and osprey. In Canada, PCDD/F levels were extremely low in animals from all herds, with mean TEQs ranging from <0.01 to 9.45 ng TEQ·kg^{-1} ww (de March et al. 1998). A west to east trend of increasing PCDD/F and TEQ levels were observed in caribou within the Canadian Arctic. Levels found in other Arctic countries were similar to those found in Canada (Figure 5.2).

Historical profiles and trends of PCDD/Fs in the Arctic

Lake sediment sampling from various Arctic locations gave information concerning deposition history of contaminants in this remote region. Recent declines in PCDD/F deposition were observed following a major increase in the 1940s, with industrialisation. In fact, elevated concentrations in sediment cores coincide with the operation of chlorinated bleached kraft pulp and paper mills within the drainage basin and the use of pentachlorophenol as a wood preservative and as a pesticide. Decreases in concentrations can be attributed to more stringent laws controlling the discharge of contaminants, replacement of "old' technology by safer and cleaner device and public awareness towards the misuse of numerous contaminants.

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5.3 Sediment

Environmental monitoring of PCDD/F levels in sediments has focused on contaminated sites, particularly in the vicinity of pulp and paper mills; in a few cases reference sites were sampled for comparison purposes. Although some recent studies are included, most data discussed were collected prior to the *Pulp and Paper Effluent Regulations* of 1992, which resulted in significant reductions in PCDD/F effluent emissions (see Chapter 3.0). The majority of data come from British Columbia with lesser amounts from Alberta, Manitoba, Ontario, Québec, Saskatchewan, New Brunswick, and Nova Scotia. No data are available from Prince Edward Island, Newfoundland, Yukon, Northwest Territories or Nunavut. As such, levels presented herein are not necessarily representative of the nation. Recent data are needed to adequately assess the condition of sediments across Canada. TEQs are based on WHO 1998 TEF values for fish (TEQ_{fish}) in units of $ng \cdot kg^{-1}$ dw unless otherwise stated.

5.3.1 Freshwater Sediment

5.3.1.1 British Columbia

In British Columbia, Mah et al. (1989) and Trudel (1991) reported concentrations of PCDD/Fs in sediments collected in 1987-88 at locations upstream and downstream of pulp mills. At the upstream sites, 2,3,7,8-substituted PCDD/F congeners in bed sediments were below the detection limits (<1 to 200 $ng \cdot kg^{-1}$ dw depending on congener and laboratory), with the exception of one site located on the Fraser River, upstream of a pulp mill at Quesnel (Mah et al. 1989; Trudel 1991). The presence of these 2,3,7,8-substituted congeners at Quesnel is peculiar not only because it applies to an upstream site but also because this was a control mill which did not use a chlorine bleaching process[3]. In contrast, most of the sediment samples collected downstream of the pulp mills contained PCDD/Fs, with the exception of those from the Kitimat River (Trudel 1991). 2,3,7,8-TCDF was detected most commonly, though H_6CDD/Fs, H_7CDD/Fs, OCDD/F, and others were found at some sites. The sediments with the highest PCDD/F concentrations (158 $ng \cdot kg^{-1}$ dw, on a TEQ_{fish} basis) were collected downstream from Weyerhauser Canada Limited, in the Thompson River (Mah et al. 1989; Trudel 1991). The

[3] Non-chlorine bleaching mills may also generate PCDD/Fs through the use of wood chips contaminated with chlorophenols and recycled paper (Elliott et al. 1989; Beck et al. 1988).

highest concentration of 2,3,7,8-TCDF measured at that site was 3168 ng·kg^{-1} dw (1.2% OC) (Mah et al. 1989; Trudel 1991). The lowest TEQ_{fish} value downstream of the mill was 3.45 ng·kg^{-1} dw. The corresponding 2,3,7,8-TCDF concentration was 69 ng·kg^{-1} dw (0.28% OC) (Mah et al. 1989; Trudel 1991).

Historical Inputs of PCDD/Fs in Kamloops Lake, British Columbia

The condition of sediments in the vicinity of pulp and paper mills with respect to PCDD/Fs has dramatically improved compared to previous conditions more than a decade ago. Levels of PCDD/Fs have been decreasing since the 1970s and 1980s, as supported by a study conducted in Kamloops Lake, British Columbia by MacDonald et al. (1998).

Historical inputs of PCDD/Fs into Kamloops Lake, British Columbia, were estimated by analyzing and dating layers of a sediment core sampled in 1994 (Macdonald et al. 1998). Kamloops Lake is situated in the Fraser River Basin and only one bleached kraft pulp mill (Weyerhaeuser) was constructed upstream of the lake. PCDD/Fs were statistically separated in three groups (Group I: lower chlorinated PCDD/Fs [including 2,3,7,8-TCDD/F and P_5CDFs]; Group II: intermediately chlorinated PCDDs [including P_5CDD, H_6CDDs and H_7CDDs]; and Group III: higher chlorinated PCDD/F congeners [including H_7CDDs, OCDD as well as H_6DFs to OCDF]). Group I congeners seemed to be related to pulp mill effluent, while Group II seemed to originate from polychlorinated phenol (PCP) contaminated wood chips used in pulping from the 1960s and 1970s, which were subsequently phased out in the late 1970s and early 1980s. Group III congeners appeared to originate from sources other than pulp mill effluent, such as combustion processes, PCP treated power poles and long range transport.

In general, temporal patterns of PCDD/Fs in sediment were similar between the three congener groups (Macdonald et al. 1998). Concentrations of Group I congeners started to increase in the 1960s, when the pulp mill opened, substantially increased until the late 1980s to concentrations up to 1784 ng·kg^{-1} dw (for 2,3,7,8-TCDF) and then dramatically declined after that, due to changes in the bleaching process. Group II congeners also increased in the 1960s but reached a peak earlier, in the 1970s, and have subsequently declined. Finally, Group III congeners showed roughly the same increase as the other groups in the mid 1960s and 1970s, with a subsequent decline in concentrations. However, a spike in the 1940s and 1950s, before the opening of the mill, could be due to contamination from PCP treated power poles.

The more recent layer of the core (interval from late 1993 to mid-1994) showed that levels of PCDD/Fs in the sediments are low (Macdonald et al. 1998). The TEQ_{fish} value for the site was 2.7 ng·kg^{-1} dw. Concentrations of PCDDs generally increased with the degree of chlorination, ranging from 0.2 ng·kg^{-1} dw for 1,2,3,7,8-PCDD to 85 ng·kg^{-1} dw for OCDD. This trend was not observed for PCDFs, the most prevalent congener in the sediment being 2,3,7,8-TCDF with a concentration of 24.6 ng·kg^{-1} dw. OCDF was the congener measured in highest concentration after 2,3,7,8-TCDF, with a concentration of 4.5 ng·kg^{-1} dw. The PCDF congener found in lowest concentrations was 1,2,3,7,8-PCDF (0.4 ng·kg^{-1} dw). The congener 2,3,7,8-TCDF was detected at concentrations of 0.9 ng·kg^{-1} dw.

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TEQ_{fish} values and levels of 2,3,7,8-TCDF similar to those reported downstream from Weyerhauser Canada Limited in 1987-1988 were detected downstream of Crestbrook Forest Industries Limited on the Kootenay River. Concentrations of PCDD/Fs, on a TEQ_{fish} basis, ranged from and 1.35 and 111 ng·kg^{-1} dw, where levels of 2,3,7,8-TCDF ranged from 27 to 2217 ng·kg^{-1} dw, at 13% OC (Mah et al. 1989; Trudel 1991). PCDD/F concentrations, based on TEQ_{fish}, in sediments at Williston Lake near Fletcher Challenge Canada Limited ranged from 49

to 104 ng·kg^{-1} dw. The range in concentrations of 2,3,7,8-TCDF at that site was 982 to 2077 ng·kg^{-1} dw, at 10.6% OC (Mah et al. 1989; Trudel 1991).

Sediment samples were collected in 1989 from many locations upstream and downstream of four non-chlorine pulp and paper mills, and one petroleum refinery along the Fraser, Pine and Peace Rivers in British Columbia (Tuominen and Sekela 1992). Levels of PCDD/Fs in the vicinity of these industries were lower than those downstream of chlorine-based pulp and paper mills (Mah et al. 1989; Trudel 1991). The only two congeners detected were 2,3,7,8-TCDF and OCDD, at only 5 of 27 sites. The highest PCDD/F concentrations, measured on a TEQ_{fish} basis, was 1.3 ng·kg^{-1} dw (at 1.88% OC), downstream of Scott Paper Limited, New Westminster, on the Fraser River. Concentrations of 2,3,7,8-TCDF at that site were 24 ng·kg^{-1} dw. Similarly, TEQ_{fish} values downstream of Paperboard Industries Corporation, Burnaby, also on the Fraser River, were 1.1 ng·kg^{-1} dw, while levels of 2,3,7,8-TCDF were 22 ng·kg^{-1} dw (at 0.61% OC) (Tuominen and Sekela 1992).

Dwernychuk et al. (1991a, b) conducted sediment quality surveys on the Fraser, Thompson, and Kootenay River systems in 1990. Although sampling sites were not the same as those used by Mah et al. (1989) and Trudel (1991), PCDD/F concentrations and their corresponding TEQ_{fish} levels in 1990 generally appear lower than those in 1987-88. In these large British Columbia rivers, 2,3,7,8-TCDF was detected most commonly though H_6CDDs, H_7CDDs, OCDD/F were found at some sites (Dwernychuk et al. 1991a, b). Results from those studies showed that TEQ_{fish} levels in the Fraser River were relatively low at sites upstream of Quesnel (<0.02 ng·kg^{-1} dw). The south arm of the river had the highest TEQ_{fish} value (0.3 ng·kg^{-1} dw) and 2,3,7,8-TCDF concentrations (7.8 ng·kg^{-1} dw; 4.6% OC) (Dwernychuk et al. 1991b). In the Thompson River, TEQ_{fish} values and associated levels of 2,3,7,8-TCDF in sediments were highest at Wallachin Station (2.1 ng·kg^{-1} dw and 42 ng·kg^{-1} dw, respectively, at 3.5% OC) (Dwernychuk et al. 1991b). For the Kootenay River, TEQ_{fish} values for sites along a 167 km stretch of the river in 1990 ranged from 0.012 to 2.85 ng·kg^{-1} dw, where concentrations of 2,3,7,8-TCDF ranged from <1 to 23 ng·kg^{-1} dw (Dwernychuk et al. 1991a). No consistent spatial trend was evident.

Background sediment samples are thought to be characteristic of ambient levels of dioxins and furans in the environment (van Oostdam and Ward 1995). Throughout British Columbia, the TEQ_{fish} level in background sediment samples calculated from mean PCDD/F concentrations in 1991-1993 was 1.4 ng·kg^{-1} dw; the TEQ_{fish} levels for secondary sediment samples (i.e., collected from impacted sites) ranged from 6.3 to 20.7 ng·kg^{-1} dw in 1991-1993 (van Oostdam and Ward 1995). Background sediment samples were collected in sites not thought to be impacted by an immediate source of PCDD/Fs, while secondary sediment samples were collected in the receiving environment adjacent to a PCDD/F source (van Oostdam and Ward 1995).

5.3.1.2 Alberta

In Alberta, PCDD/F levels in sediment are typically lower than those in British Columbia although spatial and temporal trends in both provinces are similar. For example, sediment samples collected upstream of two mills on the Athabasca River contained less PCDD/Fs (below detection limits of 1 to 4 ng·kg^{-1} dw) than those collected downstream (up to 1.8 ng·kg^{-1} dw based on TEQ_{fish}; Trudel 1991). PCDD/F levels in depositional sediments collected along the Athabasca River were generally lower in 1993 than in 1992 but the reverse

trend occurred for suspended sediments (Crosley 1996a). The highest TEQ_{fish} levels, up to 3.7 ng·kg^{-1} dw (at 6.2% OC), were reported for suspended sediments collected in 1993.

Typical levels of total PCDD/Fs in sediments from three streams in the greater area of Virginia Hill, Northern Alberta, following forest fires in 1998 ranged from 1.82 ng·kg^{-1} dw and 4.98 ng·kg^{-1} dw (at 0.55 and 2.2% OC, respectively), with OCDD representing more than 70% of total PCDD/F concentration at all sites (Ikonomou et al. 1999). The low PCDD/F concentrations seemed to indicate that the sediments were not impacted by forest fires and that concentrations detected represented background levels of contamination.

5.3.1.3 Saskatchewan and Manitoba

In 1988, 2,3,7,8-TCDD/Fs, at one mill in Saskatchewan and at a control mill not using chlorine bleaching in Manitoba, were below detection limits, which varied from 2 to 15 ng·kg^{-1} dw depending upon the congener (Trudel 1991).

5.3.1.4 Ontario

PCDD levels in sediments collected upstream and downstream of Ontario pulp mills were comparable to those in British Columbia (Trudel 1991). In 1986, concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF reached as high as 66 ng·kg^{-1} dw and 1200 ng·kg^{-1} dw (4.8% OC), respectively, downstream from a pulp mill on the Wabigoon River, near Dryden. The TEQ_{fish} value for this site was 126 ng·kg^{-1} dw. OCDD and OCDF were the two congeners most commonly found at high levels. Samples collected upstream of pulp mills had levels of 2,3,7,8-TCDD/Fs below detection limits (from 3 to 10 ng·kg^{-1} dw). Three years later, in 1989, only the Spanish River contained detectable levels of 2,3,7,8-substituted PCDD/Fs; up to 2.9 ng·kg^{-1} dw on a TEQ_{fish} basis (Trudel 1991).

Sediments collected from central Lake Ontario, from 1992 to 1994, had levels of 2,3,7,8-TCDD and 2,3,7,8-TCDF of 5 and 12 ng·kg^{-1} dw, respectively, with a TEQ_{fish} of 17.3 ng·kg^{-1} dw (Morrison et al. 1999). In the western part of the lake in 1995, levels of PCDD/Fs in sediments varied greatly, ranging from <1 ng·kg^{-1} dw to 1700 ng·kg^{-1} dw for 1,2,3,7,8,9-HCDF and OCDD, respectively (Marvin et al. 2000). Colonisation of the sediments by the mussel *Dreissena* influenced PCDD/F levels. Levels of 2,3,7,8-TCDD were 2.6 and 2.5 ng·kg^{-1} dw in sediments with and without mussels, respectively. For 2,3,7,8-TCDF, sediments with *Dreissena* had levels of 7.4 ng·kg^{-1} dw, while those without the mussels had levels of 5.2 ng·kg^{-1} dw (Marvin et al. 2000). Therefore, TEQ_{fish} values in sediments where the mussel was absent were lower than those where *Dreissena* was present. The TEQ_{fish} values were 10.5 and 8.6 ng·kg^{-1} dw for sediments with and without mussels, at 2.8 and 1.3% OC, respectively (Marvin et al. 2000).

In 1998, levels of 2,3,7,8-substituted PCDD/Fs in sediments from several sites on the American side of the Detroit River and on the Rouge River in southeastern Michigan ranged from 69 to 1420 ng·kg^{-1} dw (Kannan et al. 2001). Concentrations of PCDD/Fs ranged from 3.2 to 61.6 ng·kg^{-1} dw for these sites, on a TEQ_{fish} basis. The sites are at close proximity to Canada, as the Detroit River separates the two countries, while linking Lake St. Clair and Lake Erie. Despite the short distance separating the two shores, differences in PCDD/F levels in sediments might exist between the nations due to water currents and depositional zones, or differences in inputs. Unfortunately, no recent studies were located where PCDD/Fs were measured in Canadian sediments of the Detroit River or elsewhere in the nearby region.

5.3.1.5 Québec

All data available for Québec were collected in 1988, downstream of pulp mills with the exception of one upstream site. Maximum TEQ_{fish} levels at these sites were somewhat lower than those for British Columbia and Ontario but higher than those for Alberta. The highest 2,3,7,8-TCDD and 2,3,7,8-TCDF concentrations, 31 $ng \cdot kg^{-1}$ dw and 57 $ng \cdot kg^{-1}$ dw (4.8% OC), respectively, were measured downstream from a pulp and paper mill in Quévillon River, near Lebel-sur-Quévillon. The TEQ_{fish} for this site was 47 $ng \cdot kg^{-1}$ dw. OCDD and OCDF were the most abundant and commonly detected congeners (Trudel 1991).

5.3.1.6 New Brunswick and Nova Scotia

Limited data from New Brunswick and Nova Scotia suggest that PCDD/F levels in sediment are relatively low in these provinces. The highest TEQ_{fish} level reported, 15 $ng \cdot kg^{-1}$ dw, was measured downstream of a mill on the Saint John River (Trudel 1991). In contrast to other upstream sites studied by Trudel (1991), sediment from both upstream sites in New Brunswick and Nova Scotia contained detectable amounts of 2,3,7,8-substituted PCDD/Fs.

5.3.2 Marine Sediment

Fewer data are available for marine and estuarine sediments compared to freshwater sediments, although PCDD/Fs have been measured in both the Pacific and Atlantic regions of Canada. All data are from the vicinity of pulp mills and therefore may not be indicative of background levels. Unlike river systems, coastal environments do not permit distinct upstream and downstream sampling. In these cases, sediment samples are collected near the discharge outfall or the effluent diffuser, at some distance away from the mill. The most contaminated coastal marine areas of British Columbia appear to be Howe Sound at Squamish and Hecate Strait, with TEQ_{fish} levels up to 127 and 101 $ng \cdot kg^{-1}$ dw, respectively (Trudel 1991).

5.3.2.1 British Columbia

Sediment samples in fjords, channels and straits near nine bleach-kraft marine pulp mill sites in British Columbia were collected between 1990 and 1995 (Yunker and Cretney 2000). Mean 2,3,7,8-TCDD and 2,3,7,8-TCDF concentrations were 5.7 and 190 $ng \cdot kg^{-1}$ dw, respectively. The TEQ_{fish} level for those sites was 120 $ng \cdot kg^{-1}$ dw, calculated from the average concentrations 2,3,7,8-PCDD/F congeners (Yunker and Cretney 2000). Mean concentrations of dioxins in the sediments generally increased with the degree of chlorination (5.7 $ng \cdot kg^{-1}$ dw for 2,3,7,8-TCDD to 447 $ng \cdot kg^{-1}$ dw for OCDD). This trend was not observed for furans. The furan congener most present in the sediment was 2,3,7,8-TCDF (190 $ng \cdot kg^{-1}$ dw), followed by OCDF and 1,2,3,4,6,7,8-HCDF (65 and 54 $ng \cdot kg^{-1}$ dw, respectively; Yunker and Cretney 2000).

5.3.2.2 Québec

In 1994, levels of the seventeen 2,3,7,8-substituted PCDD/Fs were measured in and near Baie des Anglais on the St. Lawrence Estuary near the town of Baie Comeau, Québec (Lee et al. 1999). Three sites were selected with increasing distance from shore, where a pulp and paper mill, an aluminium refinery, and a grain storage facility were located. Levels of PCDD/Fs decreased with distance from shore, the nearest site had a TEQ_{fish} value of 27 $ng \cdot kg^{-1}$ dw, while

the value at the farthest site was more than 50-fold lower (0.5 ng·kg^{-1} dw) (Lee et al. 1999). Levels of 2,3,7,8-TCDD and 2,3,7,8-TCDF ranged from <0.03 to 0.06 ng·kg^{-1} dw and from 1.6 to 200 ng·kg^{-1} dw, respectively, from the nearest to the farthest site from shore (Lee et al. 1999).

5.3.2.3 Atlantic Coast

Sediment samples collected from the Atlantic coast were much less contaminated than those from the Pacific coast. The most contaminated coastal site in Nova Scotia was Port Hawkesbury with a TEQ_{fish} level of 10.5 ng·kg^{-1} dw (8.5% OC; Trudel 1991). Sediment from the Miramichi River estuary in New Brunswick contained higher TEQ_{fish} levels (6.5 ng·kg^{-1} dw; 8.3% OC) upstream than downstream (1 to 3.9 ng·kg^{-1} dw; 2% OC) of a pulp mill, though the difference in organic carbon content between the two sites might have been an influencing factor (Trudel 1991). TEQ_{fish} levels in the St. Lawrence estuary were similar with values up to 4.9 ng·kg^{-1} dw (Brochu et al. 1995).

Fraser River Basin - Dioxins and Furans on the Decline

The Fraser River Basin is a critically important region of British Columbia, draining approximately one quarter of the province, with an area of 234 000 km^2 (Dorcey and Griggs 1991) (Figure 5.3). The basin covers an area from the Strait of Georgia to the Rocky Mountains and includes many tributaries (Dorcey and Griggs 1991). The Fraser River is the fifth longest river in Canada, running 1375 km, and has a mean annual flow of 3972 $m^3 \cdot s^{-1}$ (Dorcey and Griggs 1991). It is a crucial habitat for hundreds of thousands of migratory birds and waterfowl and is the most important system for the production of salmon in British Columbia and possibly the world (Dorcey and Griggs 1991). Two and a half million people, more than two-thirds of the population of British Columbia, live in the Fraser River basin (Environment Canada 1998a). The basin supports large portions of British Columbia's commercial forests (48%), metal mining operations (60%) and farmland (45%) and its lower portion is one of Canada's most productive agricultural areas (Paquet 1994).

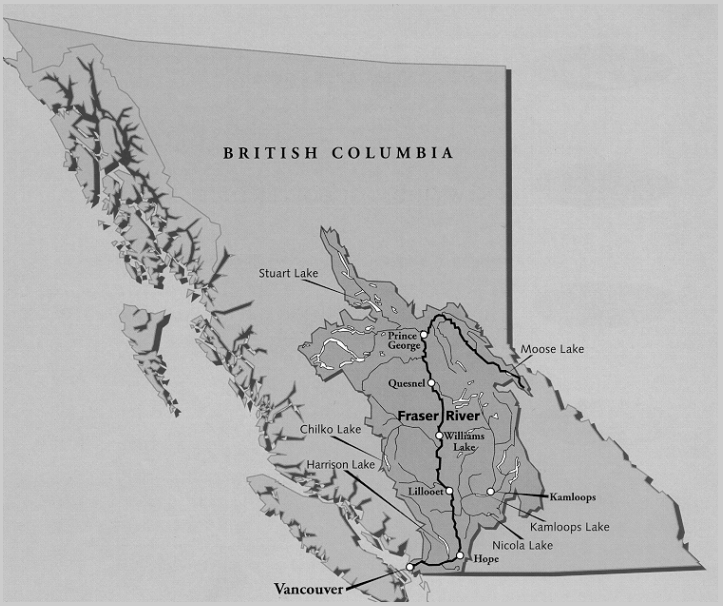

Figure 5.3. Map of the Fraser River Basin, British Columbia.

With the expanding industrial activities and urbanization during the 1980s, the protection of the Fraser River ecosystem became a concern for public and scientific communities. Large quantities of effluents containing organochlorines were being emitted daily to the Fraser River. The Fraser Basin Management Program was created jointly in the 1980s by federal, provincial, and municipal governments. A contribution of the federal government was the Fraser River Action Plan (FRAP). Started in June 1991, the FRAP was a seven year program sponsored by Environment Canada and the Department of Fisheries and Oceans. In addition to the FRAP, more stringent pulp and paper effluent regulations were put in place by the federal government in 1992.

Government programs such as the FRAP, federal pulp and paper regulations, and subsequent changes in the bleaching process of pulp and paper mills have resulted in significant reductions in levels of dioxins and furans released into the Fraser River Basin. Releases of 2,3,7,8-TCDD from all bleached kraft mills in the Fraser River decreased by more than 98% between 1990 and 1993 (BC MELP 1994), while a 92% reduction in releases of 2,3,7,8-TCDF were observed between 1990 and 1994 (BC MELP 1995). By 1996, new technology permitted a 99% reduction the amount of dioxins and furans in effluents from pulp and paper mills in the Fraser River basin (Environment Canada 1998b).

Since the early 1990s, levels of dioxins and furans in sediments, bird tissues and fish have declined by more than 90% in the Fraser River Basin (Environment Canada 1998b). Levels of 2,3,7,8-TCDD and 2,3,7,8-TCDF in suspended sediments between 1992 and 1996 were 95-99% lower than those measured in 1990 (Brewer et al. 1999). In 1992, levels of 2,3,7,8-TCDD and 2,3,7,8-TCDF in tissues of whitefish (*Prosopium williamsoni*) from the Fraser River were 26 to 95% and 74 to 98% lower, respectively, than those sampled in 1990-1991 (BC MELP 1994). Other studies showed that levels of 2,3,7,8-TCDD and 2,3,7,8-TCDF in whitefish muscle have decreased from 61 to 1.1 $ng \cdot kg^{-1}$ and from 390 to 10.6 $ng \cdot kg^{-1}$, respectively, near the Kamloops, BC pulp mill (Hatfield Consultants Ltd. 1995; Raymond et al. 1999). Adverse effects from dioxins and furans on indicator species in the Basin appear to be on the decline.

Reduced breeding success of Great Blue Herons (*Ardea herodias*) in the Fraser River Basin in the 1980s, associated with elevated dioxins and furans tissue levels, has since improved (Environment Canada 1998c). From 1992 to 1997, the difference between higher osprey (*Pandion haliaetus*) fledging success upstream from pulp mills and lower success downstream has narrowed to the point where they can no longer be distinguished (Environment Canada 1998d). Levels of total dioxins and furans in cormorant (*Phalacrocorax auritus*) eggs were 290 $ng \cdot kg^{-1}$ in 1985 and decreased to less than 52 $ng \cdot kg^{-1}$ in 1992 (a reduction of more than 82%) (Wilson et al. 1996). A subsequent study showed that TEQ levels for 2,3,4,7,8-PCDF, 1,2,3,7,8-PCDD and 2,3,7,8-TCDD in double-crested cormorant eggs from the Fraser River Estuary declined from more than 100 to less than 25 $ng \cdot kg^{-1}$ ww between 1985 and 1994 (Wilson et al. 1999).

The goals of the FRAP were to restore the natural productive capacity of the Fraser River Basin, to reduce and clean up pollution, and to develop a management program for the basin based on sustainable development principles (Environment Canada 1998a). As a result of a cooperative effort from federal, provincial, and municipal governments as well as from industry, organizations, commissions and private firms, the goals of the FRAP have been met.

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5.3.3 Summary

In summary, although a handful of recent studies reporting levels of PCDD/Fs in sediments have been located from British Columbia, Alberta, Ontario, and Québec, most data were gathered more than a decade ago. They are sporadic, not allowing for easy spatial and temporal comparisons. As the majority of sampling for PCDD/Fs in sediments occurred in the vicinity of pulp mills, these levels are not likely to be representative for the majority of Canada. As demonstrated in this section (and in the text box on the Fraser River), levels of PCDD/Fs in sediments have been drastically decreasing for more than a decade as a result of regulations and changes in the bleaching processes of pulp and paper mills. Up-to-date data are needed to adequately assess the current state of PCDD/Fs in Canadian sediments.

5.4 Aquatic Organisms

There are a large number of studies that have determined the levels of PCDD/Fs in Canadian biota. Tissue residue concentrations of PCDD/Fs in freshwater and marine biota, and in birds and mammals are expressed as TEQ_{fish}, TEQ_{birds} and $TEQ_{mammals}$ and were calculated using the WHO 1998 TEFs derivation scheme for 2,3,7,8-substituted congeners only (van den Berg et al. 1998) unless stated otherwise. No data on PCDD/F levels in aquatic plants were found in the literature.

Levels of PCDD/F are distributed in the text according to different aquatic organisms, provinces and water type. A summary table, showing the information obtained by various Environment Canada sources, is displayed in each section.

5.4.1 Freshwater and Marine Invertebrates

Studies on PCDD/Fs in freshwater and marine invertebrates are limited in Canada (Tables 5.1. and 5.2).

Table 5.1. Canadian PCDD/F data available for freshwater invertebrates (1986 to 1995). Environment Canada compilation.

Tissues	Species	2,3,7,8-TCDD ($ng \cdot kg^{-1}$ ww)	2,3,7,8-TCDF ($ng \cdot kg^{-1}$ ww)	TEQ ($ng \cdot kg^{-1}$ ww)
hepatopancreas; muscle; tissue; whole body	amphipod; benthic macroinvertebrate; crayfish; dreissena; mussel; mysis; zooplankton	<0.7 to 17	<0.69 to 1000	TEQ_{mam}: 0.13 to 125.47 TEQ_{fish}: 0.07 to 76.47 TEQ_{bird}: 0.11 to 1035.94

Table 5.2. Canadian PCDD/F data available for marine invertebrates (1983 to 1998). Environment Canada compilation.

Tissues	Species	2,3,7,8-TCDD ($ng \cdot kg^{-1}$ ww)	2,3,7,8-TCDF ($ng \cdot kg^{-1}$ ww)	TEQ ($ng \cdot kg^{-1}$ ww)
digestive gland; hepatopancreas; muscle; tissue; whole body	bentnose clam; blue mussel; butter clam; crab; dungeness crab; geoduck clam; horse clam; lobster; mussel; oyster; red crab; rock crab; shrimp; snow crab; whelk	0 to 662	0 to 24 968	TEQ_{mam}: 0 to 3440.34 TEQ_{fish}: 0 to 2126.25 TEQ_{bird}: 0 to 25 939.97

5.4.1.1 British Columbia

Studies by Dwernychuk et al. (1991a, 1993) along the Fraser and Kootenay Rivers in British Columbia, both impacted by pulp and paper mill effluent, examined the PCDD/F concentrations in various benthic macroinvertebrates. The results showed that the PCDD/F concentrations were higher downstream of a pulp paper mill (10.5 ng $TEQ_{fish} \cdot kg^{-1}$) than upstream (0.07 ng $TEQ_{fish} \cdot kg^{-1}$). Along the Fraser River, a composite sample of crayfish (*Pacificastus* sp.) muscle had non-detectable concentrations of PCDD/Fs whereas a composite sample of crayfish hepatopancreas had 15.4 ng I-$TEQ \cdot kg^{-1}$ lipid (Dwernychuk et al. 1993).

A large study by the Canadian Pulp and Paper Association (CPPA 1989) measured levels of PCDD/Fs in marine invertebrates at certain distances from numerous pulp and paper mill outfalls along the Pacific coast of British Columbia. The TEQ_{fish} ranged from 0.2 $ng \cdot kg^{-1}$ to 72 $ng \cdot kg^{-1}$ in muscle tissues of shrimp (*Pandalus borealis*), prawn and dungeness crab (*Cancer magister*). The highest concentrations were measured in the hepatopancreas (up to 403.8 ng $TEQ_{fish} \cdot kg^{-1}$) of crabs and in the soft tissues of clams (*Photothaca staminea*) and oysters. Norstrom et al. (1988) sampled a variety of bivalve species in 1987 with TEQ_{fish} levels up to 89 $ng \cdot kg^{-1}$ ww in the soft tissue of oysters (*Crassostrea* spp.); higher TEQ_{fish} (up to 2126 $ng \cdot kg^{-1}$ ww) levels were observed in hepatopancreas tissue of dungeness crabs (*Cancer magister*). Dungeness crab hepatopancreas, sampled near pulp mills in British Columbia, showed 2,3,7,8-TCDD/F concentrations ranging from 0.40 to 100 $ng \cdot kg^{-1}$ ww, and from 19 to 4500 $ng \cdot kg^{-1}$ dw, respectively, for a TEQ_{fish} value of 66.6 $ng \cdot kg^{-1}$ ww (Yunker and Cretney 2000).

5.4.1.2 Ontario

In Ontario, mussels (*Elliptio complenata*) sampled in 1986 showed concentrations of PCDD/Fs below the detection limits for all dioxins and furans congeners (Richman 1995). In a sample from Stanjikoming Bay, Ontario, 2,3,7,8-TCDD and 2,3,7,8-TCDF were found (2.3 and

2.7 ng·kg^{-1} ww, respectively) downstream of a waste disposal site (Hayton et al. 1990). Niimi (1996) measured PCDD/Fs and PCBs in zooplankton (*Diacyclops thomasi* and *Bosmina* sp.), amphipods (*Diporeia hoyi*), and mysis (*Mysis relicta*) collected from Lake Ontario; TEQ_{fish} levels were 0.11, 22.41, and 3.4 ng·kg^{-1} ww, respectively. Low levels of 2,3,7,8- substituted PCDD/F were found in *Dreissena* samples collected in 1995 near Port Dalhousie, western Lake Ontario (Marvin et al. 2000). Concentrations of 7.9 and 1.5 ng·kg^{-1} dw were reported for 2,3,7,8-TCDD/F respectively, with a 3.24 ng TEQ_{fish}·kg^{-1} ww level.

5.4.1.3 Québec

In Québec, TEQ_{fish} levels ranged from 0.17 in whelk (*Buccinum undatum*) to 2.74 ng·kg^{-1} ww in snow crab (*Chionoecetes opilio*) tissue (Brochu et al. 1995). Clement et al. (1987) measured PCDD/F levels in the digestive glands of lobsters (*Homarus americanus*) captured in New Brunswick and Nova Scotia; total TCDF concentrations up to 590 ng·kg^{-1} ww were found.

5.4.2 Freshwater Fish

Several surveys of contaminants conducted in British Columbia, Québec, Ontario (data for the Great Lakes Basin includes some for fish captured within the American border) and New Brunswick to characterise PCDD/Fs levels in freshwater fish (Table 5.3).

Table 5.3. Canadian PCDD/F data available for freshwater fish (1984 to 1999). Environment Canada compilation.

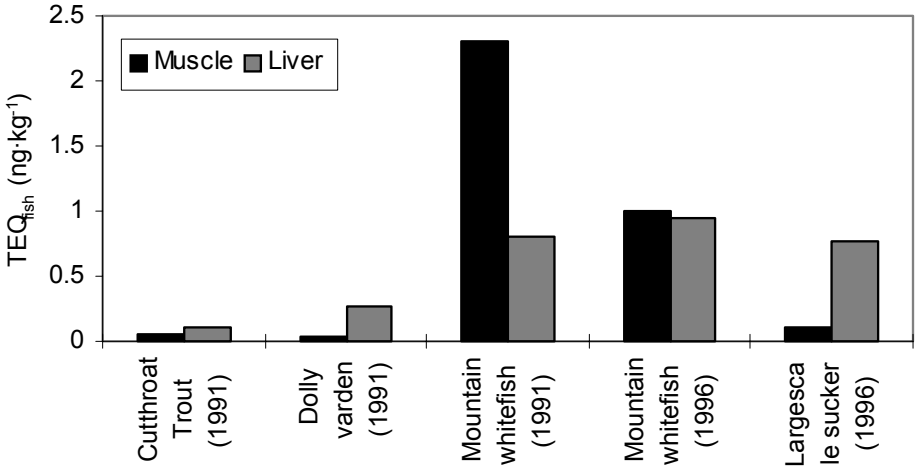
Tissues	Species	Total PCDD/Fs (ng·kg^{-1} ww)	TEQ (ng·kg^{-1} ww)
carcass; composite; dorsal, red and white muscle; egg; liver; regurgitated prey; roe; skinless fillet; tissue; viscera; whole fish	alewife; American eel; atlantic salmon; atlantic tomcod; brook trout; brown bullhead; brown trout; bullhead; burbot; channel catfish; chinook salmon; coast range sculpin; coho salmon; cresent gunnel; cutthroat trout; dolly varden; goldfish; kokanee; lake sturgeon; lake trout; lake whitefish; largescale sucker; longnose sucker; mountain whitefish; Moxastoma macrolepidotum; northern sucker; northern pike; northern squawfish; plainfin midshipman; prickly sculpin; rainbow trout; rock sole; sablefish; sculpin; shiner perch; shorthead redhorse sucker; slender sole; smallmouth bass; smelt; walleye; white sturgeon; white sucker; yellow perch	0 to 674.70	TEQ_{mam}: 0 to 111.70 TEQ_{fish}: ND to 115.99 TEQ_{bird}: ND to 656.47

ND = Not detectable

5.4.2.1 British Columbia

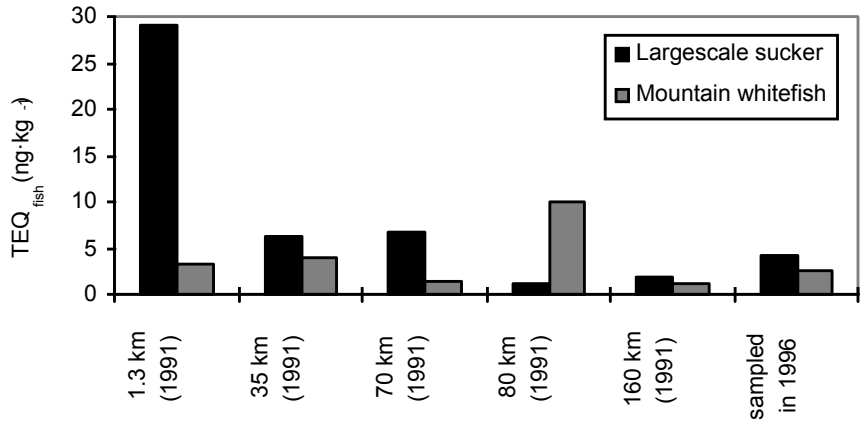
Crestbrook Forest Industries Ltd. (CFI) (Dwernychuk et al. 1991a, b; 1993; and 1996a, b) commissioned multiple studies along the Kootenay, Fraser, and Thompson Rivers in British Columbia. Effluents from pulp and paper mills and other sources such as sewage treatment plants impact these systems. Mountain whitefish (*Prosopium williamsoni*) and large scale sucker (*Catostomus macrocheilus*) were generally more contaminated than dolly varden (*Salvelinus malma*), rainbow trout (*Oncorhynchus mykiss*) and kokanee (*Oncorhynchus nerka*)

(Figure 5.4). Liver tissue of all fish species commonly contained higher levels of PCDD/Fs than muscle tissue (Figures 5.5 and 5.6). TEQ_{fish} levels in fish from the Kootenay River declined between 1990 (29 $ng \cdot kg^{-1}$ ww) and 1996 (4.3 $ng \cdot kg^{-1}$ ww) (Dwernychuk et al. 1991a, 1996a). Moreover, the results indicate that PCDD/Fs concentrations decreased with increasing distance from the pulp and paper mills (Figures 5.5 and 5.6).



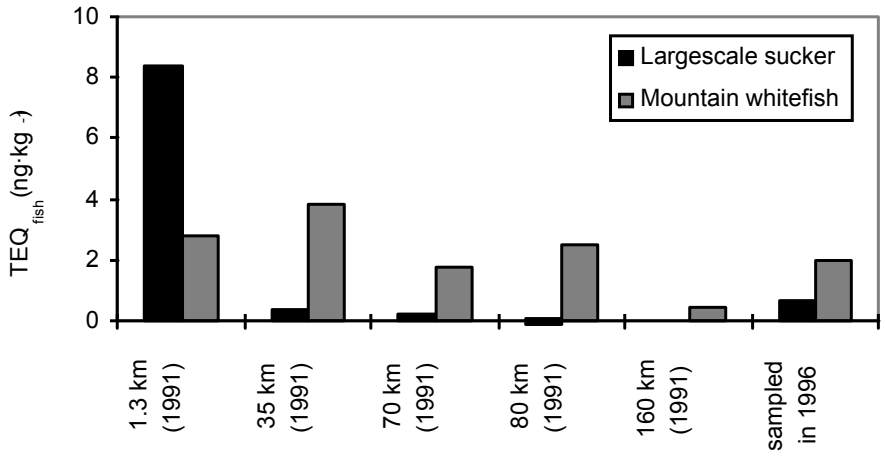
(data source: Dwernychuk et al. 1991a and Dwernychuk et al. 1996a)

Figure 5.4. TEQ_{fish} levels in liver and muscle of fish collected from the Kootenay River, upstream Crestbrook Industries, BC.



(data source: Dwernychuk et al. 1991a and Dwernychuk et al. 1996a)

Figure 5.5. TEQ_{fish} levels in liver of fish collected from the Kootenay River, downstream Crestbrook Industries, BC.



(data source: Dwernychuk et al. 1991a and Dwernychuk et al. 1996a)

Figure 5.6. TEQ_{fish} levels in muscle of fish collected from the Kootenay River, downstream Crestbrook Industries, BC

The Fraser River survey was largely limited to mountain whitefish and demonstrated a similar pattern of decreasing PCDD/F levels with time in fish tissue. For example, TEQ_{fish} levels in liver tissue of mountain whitefish collected from the Fraser River, near Stoner, BC, were 69.72, 18.65, and 0.05 $ng \cdot kg^{-1}$ ww in 1990, 1992 and 1995, respectively (Dwernychuk et al. 1991a, 1993, 1996b). TEQ_{fish} levels in muscle tissue of these fish were 45.82, 4.80 and 0.06 $ng \cdot kg^{-1}$ ww, respectively. In comparison to fish from the Kootenay River, fish from the Fraser River were up to 10 times more contaminated in 1990 but by 1995, TEQ_{fish} levels in fish from the two rivers were similar. White sturgeon (*Acipenser transmontanus*) were collected along the length of the Fraser River in 1991 by MacDonald et al. (1997). Red muscle contained the highest concentrations of PCDD/F followed by liver tissue and then by white muscle. TEQ_{fish} levels in red muscle, liver, and 7white muscle ranged from 4.58 to 67.26, from 8.00 to 23.29 and from 0.23 to 4.8 $ng \cdot kg^{-1}$ ww, respectively. For these fish, PCDD/Fs account for >99% of TEQ_{fish} when both PCDD/Fs and PCBs are incorporated into the estimate.

PCDD/Fs were measured in the carcasses of juvenile chinook salmon (*Oncorhynchus tshawytscha*) from six sites on the upper Fraser, Nechako and Thompson rivers. PCDD/F TEQ_{fish} concentrations ranged from 0.16 to 0.41 $ng \cdot kg^{-1}$ and from 0.09 to 0.26 $ng \cdot kg^{-1}$, respectively (Wilson et al. 2000). In general, the PCDD/F concentrations were low in fish from all sites, with some congeners near the detection limit. PCDD/Fs and PCBs found in the carcasses of the fish contributed to total contaminant burdens of less than 1 $ng \cdot kg^{-1}$.

5.4.2.2 Alberta

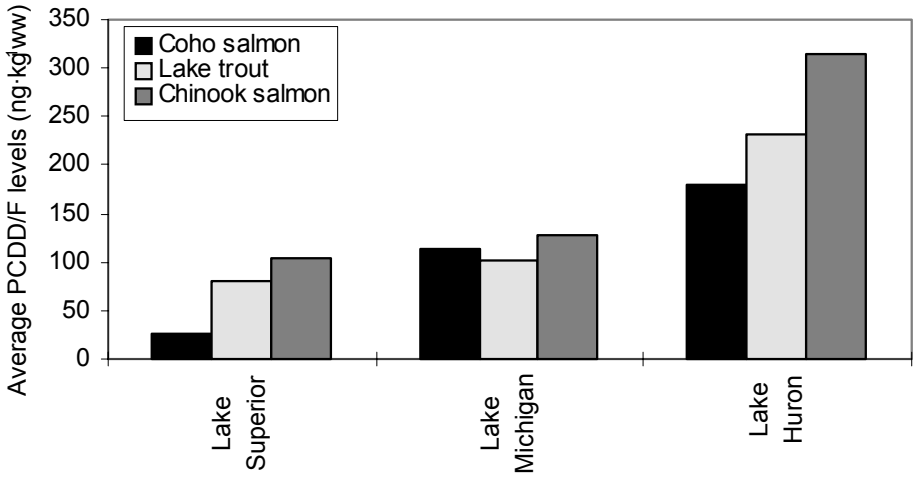
Arctic grayling (*Thymallus arcticus*) and mountain whitefish were sampled in 1998 after forest fires in Alberta. At a site partially impacted by the fires, the average total levels of PCDD/Fs were 0.78 $pg \cdot g^{-1}$ ww compared to the reference site concentrations of 0.48 $pg \cdot g^{-1}$ ww (Ikonomou et al. 1999).

5.4.2.3 Ontario

Most data from Ontario represent the Great Lakes. TEQ_{fish} levels in fish sampled throughout the Great Lakes in 1984 ranged from 6.93 to 17.00 ng·kg^{-1} ww in walleye (*Stizostedion vitreum*) and from 5.66 to 69.98 ng·kg^{-1} ww in lake trout (*Salvelinus namaycush*) (de Vault et al. 1989). TEQs (based on TEFs from Clemons et al. 1994; and Safe et al. 1987) in white sucker (*Catastomus commersoni*) collected from Jackfish Bay, in Lake Superior, declined following process and treatment improvements at a nearby kraft mill (van den Heuvel et al. 1996). Total TEQs (based on H4IIE bioassay) were however, similar throughout the same time period (1990-1993), suggesting that PCDD/Fs may not be solely responsible for the MFO induction observed in fish exposed to pulp mill effluent.

Niimi (1996) analysed four fish species collected from Lake Ontario in 1992 for PCDD/Fs and PCBs. Total TEQ_{fish} levels calculated from PCDD/F and PCB concentrations increased with trophic level, ranging from 3.83 ng·kg^{-1}ww for alewife (*Alosa pseudoharengus*) to 93.74 ng·kg^{-1}ww for lake trout (*Salvelinus namaycush)*; PCDD/Fs accounted for 74 to 94% of the total TEQ_{fish}.

PCDD/F homolog levels were analysed in dorsal muscle tissue and eggs of chinook salmon (*Oncorhynchus tshawytscha*), coho salmon (*Oncorhynchus kisutch)* and lake trout (*Salvelinus namaycush*) collected from Lakes Superior, Michigan and Huron in 1987 and 1988. In almost the entire fish composite, at least one of the 210 congeners was detected in the muscle tissue (Giesy et al. 1999). PCDF concentrations were always higher than PCDDs. The chinook salmon accumulated the greatest concentration of contaminant (4 to 550.5 ng·kg^{-1}) than the lake trout (28.6 to 447.4 ng·kg^{-1}) and the coho salmon (5 to 336.9 ng·kg^{-1}; Figure 5.4). OCDD/F and TCDD/F were the predominant congeners found in the samples. The mean total concentration of PCDD/Fs were greater in fishes collected from Lake Huron, especially in Saginaw Bay, where the chinook salmon contained PCDD/F concentrations of 215 and 380 ng·kg^{-1}ww, respectively (Figure 5.7).



(data source: Giesy et al. 1999)

Figure 5.7. Average total PCDD/F levels in dorsal muscle tissue of fish species collected in 1987 and 1988.

The Province of Ontario has monitored contaminant levels in freshwater fish through its Sport Fish Contaminant Program (http://www.ene.gov.on.ca/envision/guide/index.htm). Unpublished results, obtained from the Great Lakes, their connecting channels, and inland locations, indicate fish from the inland lakes and rivers are less impacted than those from the Great Lakes (Cox 2000; personal communication).

5.4.2.4 Québec

Of several fish species monitored in Québec, white sucker was the most commonly collected species between 1989 and 1996. Maximum TEQ_{fish} levels reported in 1989 through 1996, for white sucker, were generally associated with the Saint-Maurice region. Still, TEQ_{fish} levels in this region declined with time. White sucker had TEQ_{fish} levels up to 43.36, 16.59, and 4.44 $ng \cdot kg^{-1}$ ww in 1989, 1993, and 1996, respectively (Laliberte 1999).

In a study carried out as part of the first phase of the Environmental Effects Monitoring (EEM) Program conducted under the Canadian *Pulp and Paper Regulations*, Langlois and Dubuc (1999) reported PCDD/Fs concentrations in fish samples collected from eight locations in Québec. The results indicated that there was not a significant difference between concentrations measured in sportfish muscles from the "exposure zones" (downstream from pulp and paper mill effluents) and those measured from "reference zones". Mean PCDD/F concentrations for the eight locations were 0.126 ng I-TEQ$\cdot kg^{-1}$ for the "reference zones" (range: n.d.-0.4 ng I-TEQ$\cdot kg^{-1}$) and 0.138 ng I-TEQ$\cdot kg^{-1}$ for the "exposure zones" (range: n.d. - 0.28 ng I-TEQ$\cdot kg^{-1}$).

Environmental Effects Monitoring (EEM) Pulp and Paper Mills Program: A PCDD/F Survey

The Environmental Effects Monitoring pulp and paper mills program falls under the Pulp and Paper Effluent Regulations of the *Fisheries Act*. The objective of this program is to assess and monitor adverse effects potentially associated to the discharge of mill effluents in the receiving waters. This Canadian program, conducted by 126 mills, is structured in three to four years sequences, known as "cycles". At the end of the cycle, individual mills are asked to submit their interpretative reports to Environment Canada for further analyses and comparisons.

In Cycle 1, mills that were using chlorine bleaching or had used chlorine bleaching in the past were required to conduct a tissue analysis of edible portions of fish for chlorinated dioxin and furan congeners (Environment Canada 2001b). If results were below fish consumption guidelines for human health, emitted by Health Canada, the mill was exempted of conducting such analysis in subsequent cycles. As a result, only ten mills conducted fish tissue residue analysis in Cycle 2 (Environment Canada 2001b).

PCDD/F levels, reported by Skookumchuk Pulp Operations, located in Cranbrook BC, are herein used to compare PCDD/F results obtained in 1994 (Cycle 1) and 1996 (Cycle 2). TEQs were calculated using WHO 1998 TEF values for fish. Overall, a decrease in PCDD/F levels can be observed for largescale sucker *(Catostomus macrocheilus)* and mountain whitefish *(Prosopium williamsoni)* liver and muscle samples (Figure 5.8 and 5.9). Some locations, however, tended to show higher muscle tissue concentrations in Cycle 2, while liver concentrations were always low except in the case of *Catostomus macrocheilus* at the upstream site. PCDD/F levels, although detectable, are not as high as those reported elsewhere in the country (see Table 5.3).

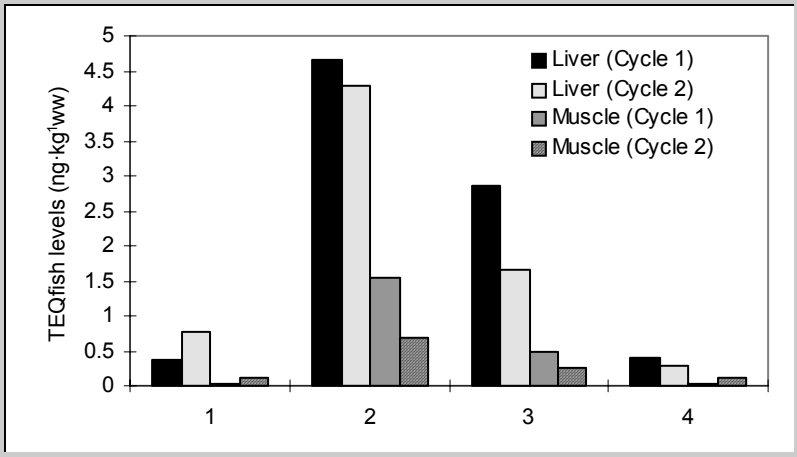


Figure 5.8. EEM pulp and paper mills program: PCDD/Fs in largescale suckers sampled along the Kootenay River: (1) upstream ca. 20 km; (2) near-field; (3) downstream ca. 15-40 km; and (4) downstream ca. 50-80 km of Skookumchuk Pulp Operations, Cranbrook

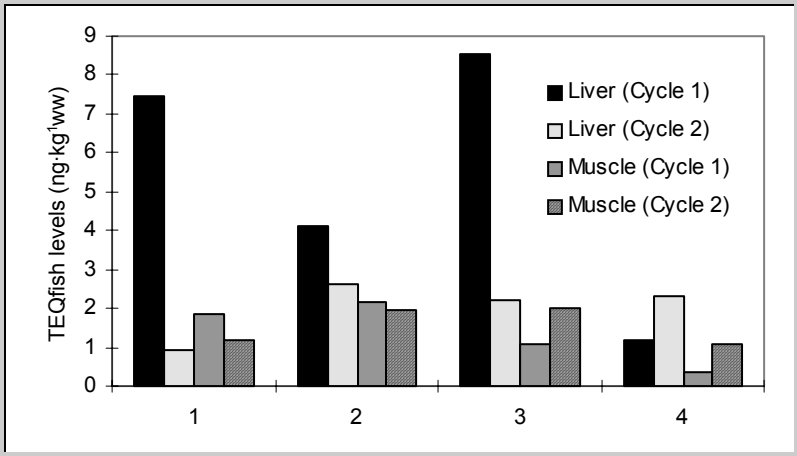


Figure 5.9. EEM pulp and paper mills program: PCDD/Fs in mountain whitefish sampled along the Kootenay River: (1) upstream ca. 20 km; (2) near-field; (3) downstream ca. 15-40 km; and (4) downstream ca. 50-80 km of Skookumchuk Pulp Operations, Cranbrook

(Data supplied by National EEM Office, Environment Canada)

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Environment Canada

5.4.3 Marine Fish

5.4.3.1 British Colombia

Very few studies examined the PCDD/F levels in marine fish (Table 5.4). Concentrations of 2,3,7,8-TCDD/F in several fish species from the Fraser River estuary were measured in 1991 by Harfenist et al. (1995). All species contained 2,3,7,8-TCDF at levels ranging from 2.0 to 22 $ng \cdot kg^{-1}$ ww. None but two had detected levels of 2,3,7,8-TCDD. Studies summarised in Environment Canada (2000b) indicate a maximum level of 10.4 ng $TEQ_{fish} \cdot kg^{-1}$, with most of the data ranging from non-detectable to 10 ng $TEQ_{fish} \cdot kg^{-1}$.

5.4.3.2 New Brunswick

Tissue concentrations of mummichog (*Fundulus heteroclitus*), collected from the receiving waters of bleached-kraft mill (BKM) effluent in Miramichi and Boutouche Estuaries, New Brunswick, were analysed for PCDD/F levels (Couillard and Nellis 1999). Up to fifty times more PCDD/Fs were found in fish from Miramichi (up to 206 ng $TEQ_{fish} \cdot kg^{-1}$) than from Boutouche (12 ng $TEQ_{fish} \cdot kg^{-1}$). At Miramichi, concentrations found in males were higher than in female for seven of the PCDD/Fs congeners by an approximate factor of 1.4.

Table 5.4. Canadian PCDD/F data available for marine fish (1986 to 1995): Environment Canada compilation.

Tissues	Species	Total PCDD/Fs ($ng \cdot kg^{-1}$ ww)	TEQ ($ng \cdot kg^{-1}$ ww)
homogenate; lever; muscle; regurgitated prey; tissue; whole	black cod; brown rockfish; cod; dogfish; dusky rockfish; English sole; eulachon; Greenland halibut; mummichog; pacific staghorn sculpin; peamouth chub; quillback rockfish; ratfish; redside shiner; rock sole; shiner perch; sole; staghorn sculpin; starry flounder; three spine stickleback, tomcod	ND to 91 100	TEQ_{mam}: 0 to 20 505 TEQ_{fish}: 0.05 to 20 643 TEQ_{bird}: 1 to 27 348

ND = Not Detectable

5.4.4 Reptiles and amphibians

The vast majority of the information available on PCDD/F concentrations in reptiles in Canada is limited to snapping turtles (*Chelydra serpentina*). For some studies, TEQ levels were not calculated because TEFs have not yet been developed nor has use of the WHO 1998 TEFs for fish, mammals, or birds been validated for reptiles (Table 5.5).

Table 5.5. Canadian PCDD/F data available for reptiles and amphibians (1984 to 1998). Environment Canada compilation.

Tissues	Species	2,3,7,8-TCDD ($ng \cdot kg^{-1}$ ww)	2,3,7,8-TCDF ($ng \cdot kg^{-1}$ ww)	Total PCDD/Fs ($ng \cdot kg^{-1}$ ww)
egg; fat; liver; tissue	snapping turtle	ND to 1030	ND to 330	0.9 to 5506

ND = Not Detectable

Northern leopard frogs, collected in 1994 and 1995 from the Green Bay ecosystem, Wisconsin, were analysed for whole carcasses concentrations of PCDD/Fs. Only 2,3,7,8-TCDF, 1,2,3,4,6,7,8-HCDF and 1,2,3,4,6,7,8-HCDD were detected at levels of 26.21, 28.88 $ng \cdot kg^{-1}$ ww and 31.12 $ng \cdot kg^{-1}$ ww, respectively (Huang et al. 1999).

5.4.4.1 Ontario

Concentrations of 2,3,7,8-TCDD in snapping turtles, collected from the St. Lawrence River in 1984-1985, were higher in fat (232 to 474 $ng \cdot kg^{-1}$ ww) than in liver (32 to 107 $ng \cdot kg^{-1}$ ww) (Ryan et al. 1986). In general, concentrations of 2,3,7,8-TCDD were higher than levels of 2,3,7,8-TCDF in fat (up to 330 $ng \cdot kg^{-1}$ ww) and liver (up to 74 $ng \cdot kg^{-1}$ ww) (Ryan et al. 1986). Snapping turtle eggs collected from Lake Ontario contained 2,3,7,8-TCDD concentrations up to 1030 $ng \cdot kg^{-1}$ ww (Bishop et al. 1996). Eggs taken from Algonquin Provincial Park in 1989 had the lowest levels of PCDD/F reported; concentrations of all congeners were below the detection limit of 2 $ng \cdot kg^{-1}$ with the exception of OCDD (16.8 $ng \cdot kg^{-1}$ lipid) (Bishop et al. 1996). Snapping turtle eggs collected on the shore of the St. Lawrence River, two to thirteen km downstream from PCB-contaminated landfill sites, showed a total PCDD/F concentration of 85.8 $ng \cdot kg^{-1}$ ww (de Solla et al. 2001). The 2,3,4,7,8-PCDF congener concentration was the highest with 60.48 $ng \cdot kg^{-1}$ ww and the concentration of the other 2,3,7,8-PCDD/F substituted congeners ranged from 0.89 to 7.09 $ng \cdot kg^{-1}$ ww. Bishop et al. (1994) determined those ecological or physiological parameters such as individual variation in feeding locations and/or food preferences and metabolism may be more important in determining contaminant levels than age, clutch size, or mass.

Bishop et al. (1998) measured PCDD/Fs levels in embryos and hatchlings from eggs of common snapping turtle (Chelydra serpentina serpentina) in 1989 to 1991. A total of eight PCDDs and fourteen PCDFs were analysed in the Great Lakes basin, St. Lawrence River and Algonquin Park. Eggs from Lake Ontario contained elevated PCDD/F concentrations, while fewer contaminant levels were detected in Lake Erie; the lowest levels were found in eggs from Algonquin Provincial Park. Concentration of 2,3,7,8-TCDD ranged from 3.6 to 27.4 $ng \cdot kg^{-1}$ ww, when detected. Levels of 2,3,7,8-TCDF were undetectable in all the samples collected. OCDD and 1,2,3,4,6,7,8-HCDD represented the two congeners found the most frequently in the sampled area. Mean total 2,3,7,8-substituted congener concentrations ranged from 0.9 to 139.7 $ng \cdot kg^{-1}$ ww, in Lake Sasajewun (Algonquin Provincial Park) and Lynde Creek (Lake Ontario).

5.4.4.2 Québec

The only PCDD/Fs Canadian concentrations found for amphibian tissues were reported by Phaneuf et al. (1995). Following the 1988 PCB warehouse fire at Saint-Basile-Le-Grand, in the province of Québec, five samples of two species of frogs were collected in the plume zone and three samples in remote zones (control). The samples were collected one to ten months after the fire and were analysed for PCDD/Fs. Mean PCDD/F concentrations in the plume zone were 108.20 and 95.40 $ng \cdot kg^{-1}$, respectively, whereas, in the control zone, only OCDD was detected with a 57 $ng \cdot kg^{-1}$ level (Phaneuf et al. 1995).

5.4.5 Birds

There exists numerous studies concerning dioxins and furans levels in birds' eggs and tissues in Canada; however, they are geographically limited to the west coast and to the Great Lakes region (Table 5.6). The use of eggs for monitoring the concentrations of organochlorine contaminants in the environment is partly responsible for this important body of data (Ewins et al. 1994). Trends in PCDD/F levels are more difficult to ascertain in bird tissues likely due to variability in the feeding behaviour, metabolism, prey items consumed and exposure to PCDD/F sources and emissions.

Herring gull eggs collected in 1989 from Saginaw Bay contained 557.2 $ng \cdot kg^{-1}$ ww of TEQs (based on H4IIE bioassay) whereas chicks aged 4, 10 and 21 days had 226.5, 96.85, and 398.92 $ng \cdot kg^{-1}$ ww of TEQs, respectively (Jones et al. 1994). The decrease in TEQs at 10 days of age can be attributed principally to growth dilution. A similar pattern was observed in double-crested cormorant eggs collected from several locations; TEQs (based on H4IIE bioassay) ranged from 26.86 to 149.62 $ng \cdot kg^{-1}$ ww, from 20.85 to 91.08 $ng \cdot kg^{-1}$ ww, from 17.9 to 85.22 $ng \cdot kg^{-1}$ ww, and from 17.8 to 168.34 $ng \cdot kg^{-1}$ ww for 4, 10, 21, and 32 days old chicks, respectively. These values are similar in magnitude to TEQ_{bird} levels for eggs of the same species collected in 1988-90 in British Columbia (Whitehead et al. 1992; Jones et al. 1994; Sanderson et al. 1994b).

Eggs of double-crested cormorants and herring gulls were collected from the Michigan waters of the Great Lakes in 1998 and showed no significant difference between their sum of 2,3,7,8-substituted PCDD/Fs (Kannan et al. 2001). Ratios of the PCDD/F levels were greater in herring gulls than in double-crested cormorants, meaning that the latter possess better abilities to metabolise PCDF congeners rapidly. Total concentrations of PCDD/Fs ranged from 7.4 to 94 $pg \cdot g^{-1}$ ww and from 3.4 to 76 $pg \cdot g^{-1}$ ww, respectively. OCDD represented the most important congener in cormorants while TCDD predominated in herring gulls.

Not surprisingly, higher concentrations have been measured in piscivorous than in insectivorous bird species. For example, concentrations of PCDD/Fs were greatest in eggs and chicks of Forster's tern (*Sterna forsteri*) and common terns (*Sterna hirundo*), less in those of tree swallows (*Tachycineta bicolor*), and least in those of red-winged blackbird (*Agelaius phoeniceus*) (Ankley et al. 1993) from the Lower Fox River and Green Bay, Wisconsin.

Table 5.6. Canadian PCDD/F data available for birds (1971 to 1998). Environment Canada compilation.

Tissues	Species	2,3,7,8-TCDD ($ng \cdot kg^{-1}$ ww)	2,3,7,8-TCDF ($ng \cdot kg^{-1}$ ww)	TEQ_{bird} ($ng \cdot kg^{-1}$ ww)
breast/ pectoral muscle; carcass pools; egg; liver; nestling; plasma; tissue; whole body; yolk sacs	bald eagle; barrow's goldeneye; bufflehead; canvasback; Caspian tern; common goldeneye; common loon; common merganser; double-crested cormorant; goldeneye; great blue heron; greater scaup; harlequin duck; herring gull; hooded merganser; lesser scaup; mallard; night heron; oldsquaw duck; osprey; pied-billed grebe; red-necked grebe; surf scoter; tree swallow; western grebe; white-winged scoter	ND to 3560	ND to 24 100	0 to 32 907.63

ND = Not Detectable

5.4.5.1 British Columbia

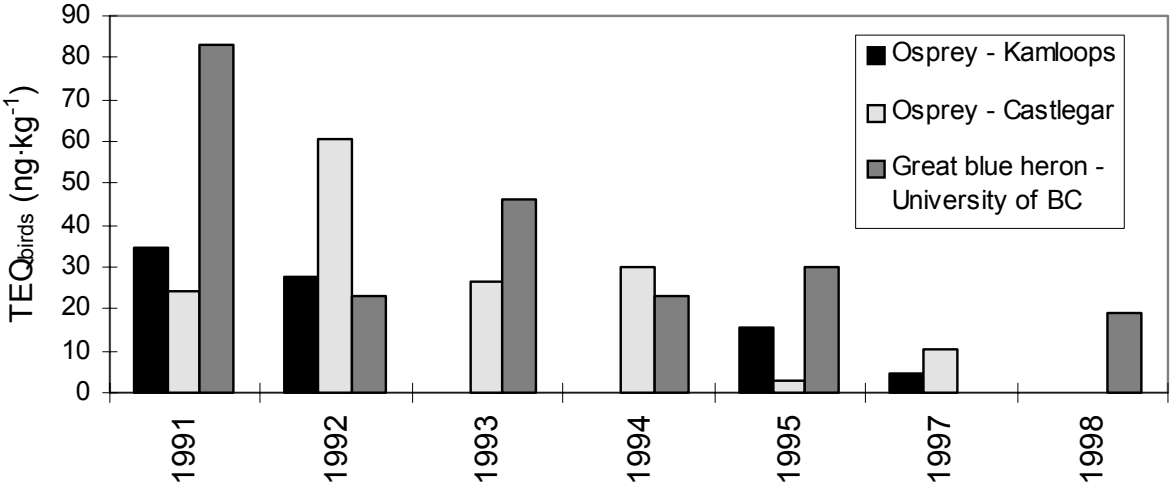
By far, bald eagle (*Haliaeetus leucocephalus*) eggs and yolk sacs collected from the Powell River in British Columbia were the most heavily contaminated, particularly with 2,3,7,8-TCDF. TEQ_{bird} levels as high as 32,907 $ng \cdot kg^{-1}$ ww were calculated for yolk sacs collected in 1992 at Evenden Point (Elliott et al. 1996a). At other sites along Powell River, including Limekiln Bay and Scuttle Bay, eggs and yolk sacs of bald eagles contained high levels of 2,3,7,8-TCDF contributing to TEQ_{bird} levels up to 24,405 $ng \cdot kg^{-1}$ ww (Elliott et al. 1996a). As birds are believed to readily metabolise and eliminate 2,3,7,8-TCDF, the authors suggested that the high levels of 2,3,7,8-TCDF were indicative of recent exposure (Elliott et al. 1996a).

From 1991 to 1995, bald eagles populations, breeding near three pulp and paper mills in the Fraser River estuary, showed higher 2,3,7,8-substituted PCDD/F levels (ranged from 0.02 to 4.5 $ng \cdot kg^{-1}$ ww) compared to control sites (from 0.01 to 0.97 $ng \cdot kg^{-1}$ ww), but no significant relationship between productivity and TEQ_{bird} was established (Elliott and Norstrom 1998). TEQ_{bird} levels ranged from 0.37 to 6.08 $ng \cdot kg^{-1}$ ww. TEQ_{bird} levels in the eggs of double-crested cormorants (*Phalacrocorax auritus*) and great blue herons (*Ardea herodias*), collected in 1986-1992 from various locations, were lower relative to eggs of bald eagles, with TEQ_{bird} levels ranging from 16.1 to 519.3 $ng \cdot kg^{-1}$ ww, (Elliott et al. 1989; Bellward et al. 1990; Whitehead et al. 1992; Sanderson et al. 1994a, 1994b).

In contrast to bald eagle eggs, those of great blue herons contained greater amounts of 2,3,7,8-TCDD than 2,3,7,8-TCDF, though both declined in several areas where PCDD/F levels in pulp and paper mill effluents were reduced (Sanderson et al. 1994a). Accordingly, during the 1983 to 1998 period, levels of dominant PCDD/F congeners in eggs of great blue herons on the coast of British Columbia decreased after pulp mills restrictions in chlorine technologies. The highest

contaminant levels were detected in the early to mid-1980s and the lowest values were detected in the 1994 samples (Elliott et al. 2001a). TEQ_{bird} levels ranged from 2 $ng \cdot kg^{-1}$ ww in Quamichan Lake, in 1989, to 522 $ng \cdot kg^{-1}$ ww in Crofton, in 1987; the later site being close to a pulp and paper mill. Mean concentrations of 2,3,7,8-TCDD/F ranged from <2 $ng \cdot kg^{-1}$ ww in Quamichan Lake, in 1989, to 209 $ng \cdot kg^{-1}$ ww in Crofton, in 1987, and from <0.3 $ng \cdot kg^{-1}$ ww in Holden Lake, in 1993, and Little River, in 1992, to 29 $ng \cdot kg^{-1}$ ww in Powell River, in 1988, respectively.

Elliott et al. (1998a) sampled nests of osprey (*Pandion haliaetus*) upstream and downstream of bleached kraft pulp mills in the Fraser and Columbia River drainage systems between 1991 and 1997 (Figure 5.10). They found no significant temporal trends in 2,3,7,8-TCDD/F levels in ospreys between 1991 and 1994, despite changes in bleaching technology, but observed an overall decrease in levels in great blue heron (Elliott et al. 2001a; Figure 5.10). Only by 1997, 2,3,7,8-TCDD/F levels in ospreys were significantly lower than previous years. Higher chlorinated PCDD/Fs were found in many osprey eggs at a sampling site on the Fraser River; concentrations of 1,2,3,4,6,7,8-HCDD and OCDD ranged from <1 to 1100 $ng \cdot kg^{-1}$ ww and from <1 to 7000 $ng \cdot kg^{-1}$ ww respectively. In plasma samples, only 1,2,3,4,6,7,8-HCDD and OCDD were detected. In all cases, osprey eggs collected downstream of bleach kraft mill contained higher mean concentrations of 2,3,7,8-TCDD/F compared to eggs collected from upstream nests. In 1995-1996, Elliott et al. (2001b) found greater mean 2,3,7,8-TCDD concentrations (2930 $ng \cdot kg^{-1}$ lipid) in osprey eggs downstream of a large pulp mill in Castlegar compared to a reference site (33.7 $ng \cdot kg^{-1}$ lipid).



(data source: Elliott et al. 1998a, 2001a).

Figure 5.10. TEQ_{bird} trends in eggs of osprey and great blue heron sampled in BC from 1991-1998

Importance of aquatic insects as a dietary source of organochlorine contaminants was investigated by Harris and Elliott (2000) in 1994 with the insectivorous tree swallow (*Tachycineta bicolor*) at locations upstream and downstream of pulp mills in British Columbia. Relatively elevated levels of chlorinated contaminants were observed in tree swallows at downstream sites, with a predominance of higher chlorinated congeners such as 1,2,3,4,6,7,8-HCDD/F and OCDD. Similar congener patterns have been reported for other wildlife species including bald eagle chicks from the Fraser River delta (Elliott and Norstrom 1998) and mink and river otter from the Columbia River (Elliott et al. 1998a; Harding et al. 1999). The major

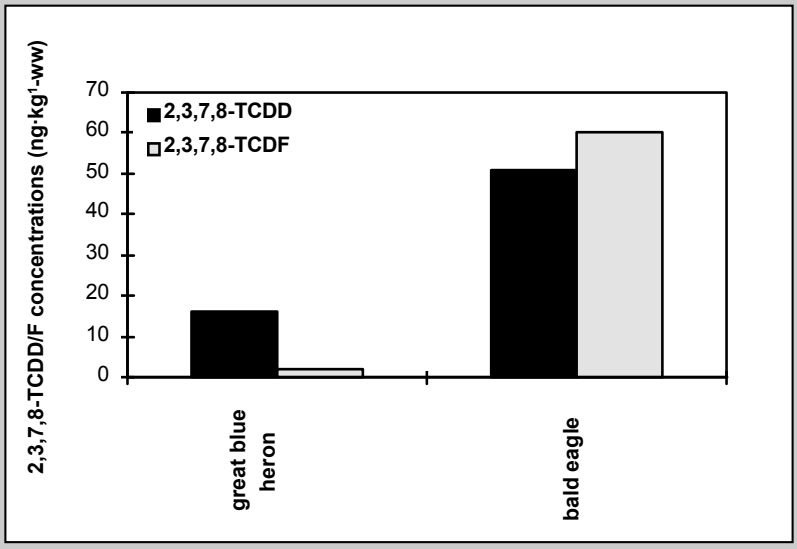
contributor to total TEQ_{bird} was 2,3,7,8-TCDF. Total TEQ_{bird} levels ranged from 0.58 to 1.29 $ng \cdot kg^{-1}$ ww and from 3.1 to 3.17 $ng \cdot kg^{-1}$ ww for sites upstream and downstream of Thompson and Fraser rivers, respectively (Harris and Elliott 2000).

Typically in birds, PCDD/Fs accumulate to higher levels in liver tissue than muscle tissue. For example, liver tissues of surf scoter (*Melanitta perspicillata*), common goldeneye (*Bucephala clangula*), and common mergansers (*Mergus merganser*) from Howe Sound contained 42.74, 73.1 and 220.19 ng $TEQ_{bird} \cdot kg^{-1}$ ww, respectively, whereas breast muscle tissue from the same birds contained 33.2, 30.0 and 144.88 ng $TEQ_{bird} \cdot kg^{-1}$ ww, respectively (Elliott and Martin 1998). Some contradictory data exists. For example, while liver tissue of buffleheads (*Bucephala albeola*) collected in 1991 from Prince Rupert, BC contained greater levels (124.2 $ng \cdot kg^{-1}$ ww) than breast muscle tissue (70 ng $TEQ_{bird} \cdot kg^{-1}$ ww), the reverse occurred in buffleheads collected in 1990 from Crofton, BC (37.7 $ng \cdot kg^{-1}$ ww in breast muscle and 13.0 $ng \cdot kg^{-1}$ ww in liver tissue) (Elliott and Martin 1998).

Bioaccumulation of 2,3,7,8-TCDD/F in Bird Tissues

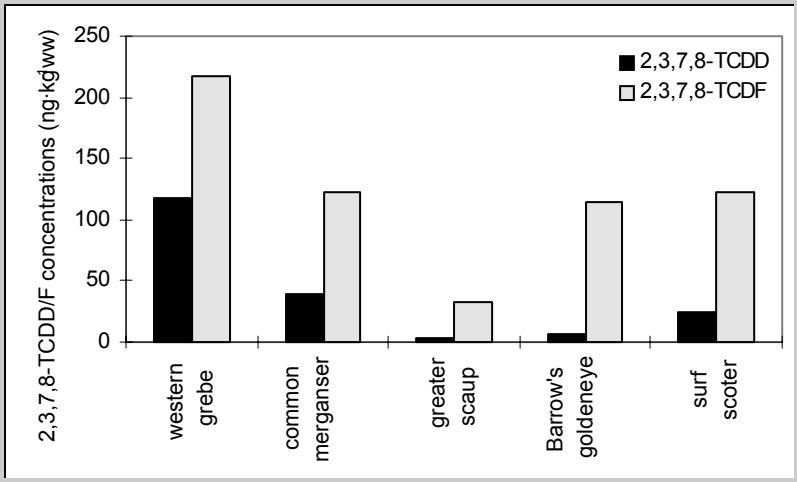
Several field studies were conducted on birds in British Colombia at locations upstream and downstream stream of pulp and paper mills before the implementation of the sector regulations. Although concentrations PCDD/Fs have since decreased, these results demonstrate the relative tissue distribution of these chemicals. Based on the chemical analyses of tissue samples (egg, liver and breast muscle) the following observations were made.

Bald eagles tended to accumulate more 2,3,7,8-TCDD/F in their eggs than the great blue heron, with levels ranging from 51 to 60 ng·kg^{-1} ww and 2 to 16 ng·kg^{-1} ww, respectively (Figure 5.11; Elliott et al. 1996b, 2001a). PCDD/F concentrations in the liver were higher than those reported in the eggs and breast muscle of various birds (Figure 5.12). Levels of 2,3,7,8-TCDF were higher (32 to 217 ng·kg^{-1} ww) than 2,3,7,8-TCDD (3 to 117 ng·kg^{-1} ww) in all the liver tissues analysed (Vermeer et al. 1993; Elliott and Martin 1998). Levels of 2,3,7,8-TCDF were higher than 2,3,7,8-TCDD in the breast muscle, with concentrations ranging from 1 to 9 ng·kg^{-1} ww and 36 to 75 ng·kg^{-1} ww, respectively (Figure 5.13; Elliott and Martin 1998).



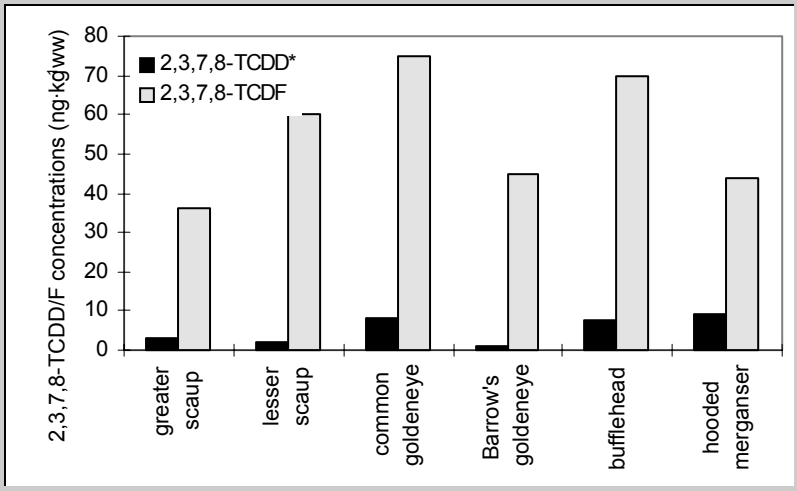
(data source: Elliott et al. 1996b; 2001a)

Figure 5.11. Concentrations of 2,3,7,8-TCDD/F found in eggs collected in Crofton, BC (1991).



(data source: Vermeer et al. 1993, Elliott and Martin 1998)

Figure 5.12. Concentrations of 2,3,7,8-TCDD/F found in livers of birds collected in Port Alberni, BC (1989).



(data source: Elliott and Martin 1998)

* Concentrations of 2,3,7,8-TCDD in greater scaup, lesser scaup, Barrow's goldeneye, and bufflehead were set at one-half the detection limit.

Figure 5.13. Concentrations of 2,3,7,8-TCDD/F found in breast muscles of birds collected in Prince Rupert, BC (1991).

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Environment Canada

5.4.5.2 Alberta

In a study of the Peace and Athabasca Rivers in northern Alberta, four of six canvasbacks (*Aythya valisineria*) livers contained relatively low PCDD/F levels, ranging from

<0.2 to 1.4 ng·kg^{-1} of 1,2,3,7,8-PCDD, 2,3,7,8-TCDF, and 2,3,4,7,8-PCDF; 2,3,7,8-TCDF being the most commonly detected (Wayland 1996).

5.4.5.3 Ontario

The Great Lakes were considered heavily contaminated freshwater ecosystems in the 1960s and 1970s. For example, 2,3,7,8-TCDD levels as high as 2350 ng·kg^{-1} were found in herring gull (*Larus argentatus*) eggs collected from 1971 to 1982 (Bishop and Weseloh 1990). The introduction of regulatory controls has helped to reduce contaminant levels in Great Lakes fish and subsequently contaminants in aquatic birds (Ewins et al. 1994). Levels of PCDD/Fs were determined in the eggs of double-crested cormorants (*Phalacrocorax auritus*) from the Canadian Great Lakes between 1989 and 1991 (Ryckman et al. 1998). In 1989, similar levels of seven PCDD and five PCDF congeners were observed in all Great Lakes samples, with concentrations of less than 30 ng·kg^{-1} ww for PCDD and between 1 and 5 ng·kg^{-1} ww for PCDF (8 to 21 ng·kg^{-1} ww for 2,3,4,7,8-PCDF). TEQ_{birds} levels ranged from 35.19 to 100.3 ng·kg^{-1} ww for the period studied. Lorenzen et al. (1999) determined that PCDD/F fraction in herring gull embryo yolk sacs accounted for less than 1% of the total contaminant residues. These authors reported that in all Great Lakes samples taken in 1997, mean total PCDD/F concentrations were below 500 ng·kg^{-1} ww.

5.4.5.4 Québec

Eggs of common mergansers collected from La Tuque, Québec near the St. Maurice River had higher TEQ_{bird} levels (165.48 to 382.34 ng·kg^{-1} ww) than fledglings (0.39 to 23.91 ng·kg^{-1} ww; Champoux 1996). Twelve double-crested cormorant eggs collected from Last Mountain Lake in Saskatchewan had a relatively low average TEQ_{bird} level of 16.12 ng·kg^{-1} ww (Sanderson et al. 1994b).

National Chemical Residues Survey in Birds: PCDD/F Analysis and Conclusions

In 1988, a national survey was initiated in order to obtain recent data on contaminants in gamebirds - to assess the human risk associated with eating those birds - and to identify species and locations were contaminants residue levels may impair avian health (Braune et al. 1999). Chemical residues in Canadian waterfowl and gamebirds, representative of the species most commonly shot by hunters, were collected between 1987 and 1995 at diverse locations across the country. A total of 3 957 birds, pooled into 834 composite samples, representing 44 species from 126 Canadian sites were included in the final report (Braune et al. 1999). Sub-composite samples of muscle and eggs were analysed for PCDD/Fs.

Although PCDD/F were not commonly detected, Braune et al. (1999) reported concentrations of PCDD/F in pectoral muscle of loons (*Gavia* sp.), mergansers (*Merganser* sp.), scoter (*Melanitta* sp.), scaup (*Aythya affinis*), oldsquaw (*Clangula hyemalis*), eider (*Somateria mollissima*), mallards (*Anas platyrhynchos*) and ringed-necked ducks (*Aythya collaris*). The highest levels of 2,3,7,8-TCDD/F (9.7 ng·kg^{-1}) were found in pectoral muscles of lesser scaup sampled at Lake St. Pierre, Québec and in surf scoter (*Melanitta perspicillata*; 13 ng·kg^{-1}) from Westham Island, British Colombia. Eggs collected solely from the Great Slave Lake, Northwest Territories, showed detectable PCDD concentrations in red-breasted mergansers (*Mergus serrator*) and herring gulls. The highest level of most contaminants was found in birds feeding at the higher trophic level, such as mergansers, loons and gulls (Braune et al. 1999).

5.4.6 Semi-Aquatic and Terrestrial Mammals

There is a paucity of data available on PCDD/F levels in terrestrial and semi-aquatic mammals in Canada (Table 5.7).

Table 5.7. Canadian PCDD/F data available for terrestrial and semi-aquatic mammals (1988 to 1996). Environment Canada compilation.

Tissues	Species	2,3,7,8-TCDD ($ng \cdot kg^{-1}$ ww)	2,3,7,8-TCDF ($ng \cdot kg^{-1}$ ww)	TEQ_{mam} ($ng \cdot kg^{-1}$ ww)
fat; liver; muscle; whole body	mink; muskrat; otter; mice; caribou	ND to 11	ND to 2	ND to 32.47

ND = not detectable

5.4.6.1 Alberta

In mink (*Mustela vison*) from the Peace and Athabasca Rivers in northern Alberta, only 2,3,7,8-TCDD was detected in two of the three pooled liver samples at 0.2 and 0.6 $ng \cdot kg^{-1}$ ww (Wayland 1995b). Elliott et al. (1998b) found that concentrations of 2,3,7,8-TCDD/F in river otters and minks were below detection limits, except in one otter collected near a pulp and paper mill on the Upper Columbia with 11 $ng \cdot kg^{-1}$ ww. The mammals were obtained from the Fraser and Columbia Rivers in the winters of 1990/1991 and 1991/1992. Generally, levels of PCDD/Fs were low (<10 $ng \cdot kg^{-1}$ ww) but exceptions were found for both species. As an example, one otter from the Columbia River had an OCDD level of 2200 $ng \cdot kg^{-1}$ ww, and one poolled sample of mink from the Fraser River had high 1,2,3,4,6,7,8-HCDD (122.7 $ng \cdot kg^{-1}$ ww) and OCDD (186.2 $ng \cdot kg^{-1}$ ww) concentrations. The authors found that I-TEQ levels (Safe 1990) in the Columbia River otters were generally more elevated than in the Fraser River. Furthermore, Harding et al. (1999) found low concentrations of 2,3,7,8-TCDD/F (<5 $ng \cdot kg^{-1}$ ww) in all liver samples of minks and river otters obtained in the winters of 1994/1995 and 1995/1996 from the same area. The Lower Fraser Valley otter pools had relatively elevated OCDD and other higher chlorinated PCDD/Fs compared to the Columbia River samples.

5.4.6.2 Québec

Mink from La Tuque, Québec had levels of 2.3 and up to 32.5 ng $TEQ_{mam} \cdot kg^{-1}$ ww, upstream and downstream of a pulp mill on St. Maurice River, respectively (Champoux 1996). PCDD/Fs were found in muskrat livers and two species of mice (whole animal), in Québec, where animals were collected after the PCB warehouse fire of Saint-Basile-Le-Grand (Phaneuf et al. 1995). One muskrat collected from the plume zone showed low concentrations of PCDD/Fs congeners, 0.62 ng kg^{-1} on a TEQ_{mam} basis. PCDD/F concentrations in mice (whole animal) were 0.06 ng $TEQ_{mam} \cdot kg^{-1}$ for remote areas and 1.47 ng $TEQ_{mam} \cdot kg^{-1}$ for the plume zone.

5.4.6.3 Northwest Territories and Yukon

In a study conducted in the Northwest Territories, essentially no PCDD/Fs were found in livers of mink (<1 $ng \cdot kg^{-1}$ lipid; Muir et al. 1997). Hebert et al. (1996) found very low or non-detectable PCDD/F levels in fat and muscle of caribou (*Rangifer tarandus*) from the Northwest Territories and Yukon.

5.4.7 Marine Mammals

Marine mammals tend to be long lived and are at the top of the food chain. Like other biota, they tend to accumulate hydrophobic substances like PCDD/Fs in tissues with high lipid content (Muir et al. 1996; Table 5.8).

Table 5.8. Canadian PCDD/F data available for marine mammals (1982 to 1996). Environment Canada compilation.

Tissues	Species	2,3,7,8-TCDD ($ng \cdot kg^{-1}$ ww)	2,3,7,8-TCDF ($ng \cdot kg^{-1}$ ww)	TEQ_{mam} ($ng \cdot kg^{-1}$ ww)
blubber; fat; liver; maternal blood; maternal blubber; oil; pup blubber; tissue	beluga; dall's porpoise; false killer whale; grey whale; harbour porpoise; harbour seal; killer whale; narwhal; pilot whale; polar beau; ringed seal; risso's dolphin; Sable Island grey seal; walrus; white-beaked dolphin	ND to 37	ND to 109	ND to 92.48

ND = Not detectable

5.4.7.1 Pacific coast

Off the Pacific coast of Canada, relatively low TEQ_{mam} levels were reported for harbour porpoise, *Phocoena phocoena* (up to 41.2 $ng \cdot kg^{-1}$ ww) and several whale species (up to 29.2 $ng \cdot kg^{-1}$ ww; Burlinson 1991; Jarman et al. 1996). In harbour seal (*Phoca vitulina*), TEQ_{mam} levels ranged from 1.89 to 92.48 $ng \cdot kg^{-1}$ ww (Addison et al. 1996). Simms et al. (2000) reported sums of PCDD/F concentrations of 343.9 and 47.9 $ng \cdot kg^{-1}$ lipid, respectively, in fasted harbour seals collected in British Columbia between 1993 and 1996. Concentrations of 187.4 and 20.3 $ng \cdot kg^{-1}$ lipid PCDD/F, respectively, were also found in nursing free-ranging harbour seal pups. Total TEQ_{mam} found in the seals ranged between 44.1 and 52.7 $ng \cdot kg^{-1}$ lipid.

Generally, PCDD/F concentrations in killer whale (*Orcinus orca),* sampled between 1993 and 1996 from the coastal waters of British Columbia, were low, with many congeners being undetectable (Ross et al. 2000). Furthermore, PCDD/F congeners contributed approximately 3% of the total TEQ_{mam} concentrations; the TEQ_{mam} levels for PCDDs and PCDFs ranged from 6.45 to 9.50 $ng \cdot kg^{-1}$ lipid and 4.31 to 5.16 $ng \cdot kg^{-1}$ lipid, respectively, depending on the individual age and sex of the seal. Age, sex and inter-community differences in the concentrations of PCDD/Fs were not significant and probably reflect low dietary levels and metabolic removal of dioxin-like compounds in killer whales. The authors suggested that total 2,3,7,8-TCDD TEQs, although low in this study, potentially surpassed adverse effect levels for harbour seals.

5.4.7.2 Arctic coast

All but one of seventeen polar bears (*Thalarctos maritimus*) had detectable levels of 2,3,7,8-TCDD ranging from 2 to 23 $ng \cdot kg^{-1}$ ww, whereas none had detectable levels (<2 $ng \cdot kg^{-1}$ ww) of 2,3,7,8-TCDF (Muir and Norstrom 1990; Norstrom et al. 1990). Polar bears, beluga whales (*Delphinapterus leucas*), narwhal (*Monodon monoceros*) and ringed seals (*Phoca hispida*) were reported by Muir and Norstrom (1990) and Norstrom et al. (1990) to have 2,3,7,8-TCDD concentrations ranging from <2 to 37 $ng \cdot kg^{-1}$ ww, 2,3,7,8-TCDF concentrations ranging from <2

to 60.5 ng·kg^{-1} ww, 1,2,3,6,7,8-HCDD ranging from <4 to 9 ng·kg^{-1} ww, and OCDD ranging from <8 to 44 ng·kg^{-1} ww.

In beluga whales from the St. Lawrence estuary, no PCDD/Fs were detected in liver tissue (Muir et al. 1996). Low levels of PCDFs were detected in the beluga blubber samples, whereas PCDDs were undetectable; the highest concentrations of PCDFs (8 to 31 ng·kg^{-1} ww) were detected in blubber of a male (Muir et al. 1996). In contrast, Gauthier et al. (1998) found no PCDFs and only two PCDD congeners in tissues of a neonate St. Lawrence beluga whale. They found OCDD in all tissues; ranging from 12 ng·kg^{-1} lipid in brain to 1138 ng·g^{-1} lipid in liver. In kidney and liver, only 1,2,3,4,6,7,8-HCDD was detected at concentrations of 29 and 31 ng·kg^{-1} lipid, respectively. PCDDs contributed approximately 0.08 to 1.1 % of the total TEQs (based on I-TEFs, 1988) in all tissue samples.

Are Dioxins and Furans in Arctic Wildlife a Problem?

Chlorinated dibenzo-*p*-dioxins and dibenzofurans are colloquially referred to as 'dioxins' and 'furans'. One member of the family, 2,3,7,8-tetrachlorodibenzodioxin, or TCDD, has received the most attention because it is the most toxic. However, dioxins with five (PCDD), six (HCDD), and eight (OCDD) chlorines are also frequently found in the environment. Usually, only 2,3,7,8-tetrachlorodibenzofuran (TCDF) and pentachlorodibenzofurans (PCDF) are present in vertebrates. In the southern Canadian environment, the most important historical source of TCDD and TCDF to the environment was bleached kraft pulpmill effluent. The problem was most acute in the coastal areas of British Columbia in the mid-1980s, but was largely solved by changes in practices and improved effluent treatment (Elliott et al. 2001a). TCDD was also a problem historically in Lake Ontario because of industrial effluents from the chlorine chemical industry in the Niagara Falls area, but this contamination decreased rapidly in the 1970s and 1980s (Hebert et al. 1994). Pentachlorophenol fungicides contributed primarily OCDD, which is not readily available for accumulation in biota. Commercial PCB mixtures contained traces of PCDF and likely contributed to the environment during active use in the past. Combustion, primarily municipal and hospital waste incineration, is presently the major contributor of both dioxins and furans to the Canadian environment.

Based on air trajectory analysis, a report in 2000 indicated that combustion sources in the northeast United States would be the major contributor of dioxins and furans to parts of the eastern Arctic, but did not deal with whether this was occurring or not (Commoner et al. 2000). The Commoner report implied that herbivores, such as caribou, may accumulate dioxins and furans deposited from the atmosphere on lichens and other plants. However, a study in 1996 had already shown that caribou throughout the Canadian Arctic had levels of dioxins and furans that were among the lowest ever reported in wildlife (Hebert et al. 1996). Total TCDD equivalent concentrations were less than 1 $ng \cdot kg^{-1}$ (part per trillion) in caribou fat, and less than 0.01 $ng \cdot kg^{-1}$ in muscle using the international TEF (I-TEF) indices ({NATO (North Atlantic Treaty Organization) 1988 NATO1988A /id}). This study clearly indicated that dioxins and furans are not transported in significant amounts to the Arctic to constitute a problem in Arctic herbivores or species that consume them, such as wolves and humans. TCDD equivalents due to PCBs were higher than those from dioxins and furans, but still very low (0.4 - 2.5 $ng \cdot g^{-1}$).

Dioxins and furans, like other POPs, tend to bioaccumulate to higher levels in aquatic/marine than in terrestrial food webs because transfer from the medium (water) to plankton and organic particulate matter at the bottom of the food web is efficient, and food chains tend to be longer. Marine mammals and animals that eat them, including polar bears (which eat primarily ringed seal) and humans, accumulate the highest concentrations of POPs such as PCBs and chlordanes in the Arctic environment (Norstrom and Muir 1994). A study in 1990 (Norstrom et al. 1990) showed that concentrations of TCDD were 2 to 40 $ng \cdot kg^{-1}$ in ringed seal blubber, less than 2 to18 $ng \cdot kg^{-1}$ in polar bear fat, and less than 2 $ng \cdot kg^{-1}$ in beluga blubber. Similar concentrations of OCDD were found, along with traces of TCDF. Compared to marine mammals on the Pacific coast, these concentrations were very low (Jarman et al. 1996). Thus, significant bioaccumulation of dioxins and furans does not appear to be occurring in the Arctic marine mammal food web, although concentrations in animals at the apex of the food web are higher than in terrestrial herbivores.

Temporal trends, 1984-1990, of TCDD were determined in polar bear fat samples from three areas in the Canadian Arctic (Norstrom 1997). As can be seen from Figure 5.14, concentrations were lower in two of the areas, and more evenly distributed in 1990, at around 3 $ng \cdot kg^{-1}$. These concentrations are too low to be of concern to either the polar bear or animals that consume them.

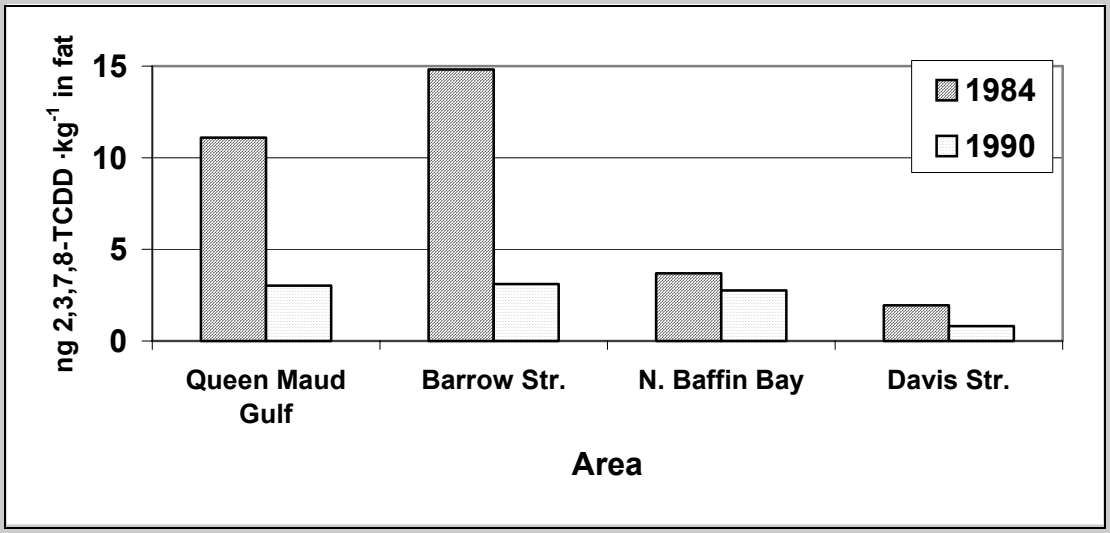

Figure 5.14. Temporal trends in TCDD levels in polar bear lipids from the Canadian Arctic.

Dioxin and furan concentrations were determined in eggs of three species of Seabirds, black-legged kitiwake, northern fulmar and thick-billed murre, from Prince Leopold Island, near Barrow Strait in 1993 (Braune and Simon 2002 in prep). TCDD concentrations were in the order of 1 $ng \cdot kg^{-1}$. Concentrations of PCDD and HCDD were higher than TCDD in the same species, and ranged from 1.3 - 9.6 $ng \cdot kg^{-1}$. Unlike marine mammals, furans were higher than dioxins, particularly 2,3,4,7,8-PCDF, which ranged from 7 to 38 $ng \cdot kg^{-1}$. The pattern of PCDD/F contamination in Prince Leopold Island seabirds is much more similar to the pattern of dioxin/furan contamination in European and European Arctic biota (Oehme et al. 1988) than that observed in resident species in the Canadian Arctic. These seabirds, which migrate to various parts of the North Atlantic Ocean, are probably picking up the PCDD/F contamination during their southern migration rather than in the Arctic.

Dioxin, furans and TCDD equivalents have been determined in breast milk from Inuit women in villages from the west coast of Hudson Bay (Dewailly 2001) in 1990 and 2000. The mean TCDD concentration was 2.2 $ng \cdot kg^{-1}$ in 1996-2000. The mean total TCDD equivalent concentration was 17 $ng \cdot kg^{-1}$, which is comparable to that found in the general population of Québec. There is therefore no evidence that consumption of Arctic animals results in a significant input of dioxins and furans to the Inuit diet. Most of the TCDD equivalents were from dioxin-like PCBs. Dioxin, furan and TCDD equivalent concentrations declined 3.8 fold between 1990 and 2000.

In summary, there is no evidence that dioxins and furans in Arctic fish and wildlife constitute a problem in either the terrestrial or marine environment. Limited data suggests that the already low concentrations are decreasing slowly over time. In most species, TCDD equivalent concentrations are dominated by dioxin-like PCBs rather than dioxins and furans.

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5.4.7.3 Atlantic Coast

There is a lack of data from marine mammals on the Atlantic coast. Muir and Norstrom (1990) reported very low levels (below detection) of PCDD/Fs in white-beaked dolphins (*Lagenorhynchus albirostris*), beluga whales, and pilot whales (*Globicephala melaena*). PCDD/F concentrations were below 20 $ng \cdot g^{-1}$ lipid in blubber samples of mother-pup pairs of grey seals (*Halichoerus grypus*) from Sable Island (Addison et al. 1999). This level represented one of the lowest observed in seals, being lower or similar to concentrations from the Canadian Arctic. In addition, PCDD/Fs were detectable sporadically in blood samples and were measurable in milk samples, however, their contribution to the total TEQ (WHO 1998) was about 5%. Zitko et al. (1998) reported levels of PCDD/F in harp seals (*Phoca groenlandica*) found in the waters of Southern Labrador, exposed to two major sources of contaminants: nursing milk and diet. Only 2,3,7,8-TCDF was detected in all liver and blubber samples; with higher concentrations in female tissues. A value of 2.4 $ng \cdot kg^{-1}$ lipid was found in male blubber compared to 3.6 $ng \cdot kg^{-1}$ lipid in female. In addition, female blubber contained trace of 2,3,7,8-TCDD, 1.4 $ng \cdot kg^{-1}$ lipid of 1,2,3,7,8-PCDD and 1.2 $pg \cdot g^{-1}$ lipid of 2,3,4,7,8-PCDF, while these compounds were not detected in males. PCDD/Fs were not detected in pools of muscle and kidney obtained from seals of either sex. The variability among marine mammal species may reflect differences in exposure related to trophic status and proximity to PCDD/F sources, sex of animals sampled and life cycle duration.

The Northern River Basins Study: A Thorough Assessment of Environmental Quality

The Northern River Basins Study (NRBS) was initiated in 1991, soon after a decision by the Alberta government permitting the Alberta-Pacific kraft pulp mill industries to develop a project near Athabasca, Alberta (Irwin Huberman Consulting 1997). As a result of this decision, public concerns were raised towards the potential industrial, municipal and agricultural impacts on the Peace, Athabasca and Slave Basins (Figure 5.15), if such industries were to be permitted or expanded in the surroundings. The NRBS was a five year study, engaging the governments of Canada, Alberta and the Northwest Territories for the primary purpose of collecting baseline scientific information concerning the impacts and effects of human activities on the different aquatic ecosystems in order to ensure their protection, restoration and wise use. Approximately 150 scientific reports were produced during this study (Irwin Huberman Consulting 1997).

Due to the scale of the project, the study was subdivided in eight sections, each focusing on complementary areas of research: contaminants (including dioxins and furans), food chain, nutrients, hydrology and hydraulics, synthesis and models, traditional knowledge, drinking water and other uses.

In light of the broad geographical area and the complexity of the basins, a 200 km reach of the Athabasca River, between Hinton and Whitecourt, AB, was selected in 1992 for monitoring and to conduct different analyses (Crosley 1996a). This region received pulp and paper mills effluent mixed with municipal effluents, commonly referred to Hinton combined effluents, from the town of Hinton, AB. Dioxins and furans were monitored in water, various types of sediments, benthic invertebrates, biofilms, fish, wildlife and birds.

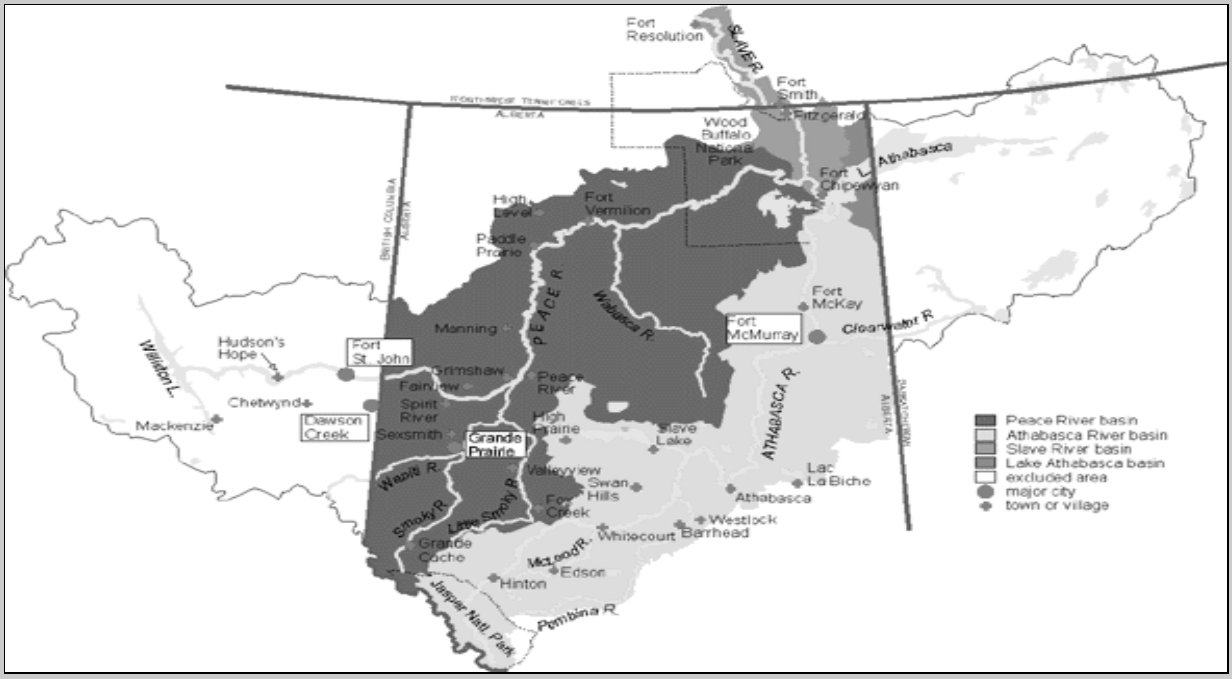

Figure 5.15. Map of watersheds included in the Northern River Basin Study.

Water and sediments

Analyses performed in the spring of 1992 showed very low PCDD/F concentrations in water samples and higher concentrations in suspended sediments; three of the most toxic PCDD/Fs (2,3,7,8-TCDD, 1,2,3,7,8-PCDD and 2,3,4,7,8-PCDF) were measured at levels ranging from 1.4 to 11 $g \cdot kg^{-1}$ dw in Hinton combined effluent (Crosley 1996a). In 1993, after the expansion of the pulp and paper industries in the study area, lower chlorinated congeners were detected in the waters of the Athabasca River, some of them as far as 230 km downstream of Hinton (Crosley 1996b). Notwithstanding that the levels detected in 1993 were slightly higher than in 1992, temporal evolution was similar in both cases. Total PCDD/Fs were detected in suspended and deposited sediments of the majority of the effluents, ranging from 0.11 to 3600 $ng \cdot kg^{-1}$dw (Crosley 1996b). Samples of bottom sediments, originating from the Peace and Athabasca Basins in 1994 and 1995, showed low PCDD/F concentrations (Crosley 1996c). The TEQ levels tended to be similar at all sites and in both size fractions, clustered from 0.3-0.4 ng $TEQ \cdot kg^{-1}$ (Crosley 1996c).

Sediment cores, from Great Slave Lake, showed a decline in PCDD/F concentrations from the 1950s to the mid 1990s (Evans et al. 1996). Total PCDD/Fs found in the cores of 1994 had an average concentration of 36.7 ± 21.4 $ng \cdot kg^{-1}$ and 3.8 ± 3.6 $ng \cdot kg^{-1}$, respectively, and were mostly comprised of the higher chlorinated congeners (Evans et al. 1996). In 1992, Pastershank and Muir also observed low TEQ levels in water, sediments, invertebrates and fish downstream of Weldwood pulp mill, Hinton AB (Pastershank and Muir 1995). In biofilms, a matrix of bacteria, extracellular substances and abiotic materials firmly attached to surfaces in a moist environment, only three congeners (2,3,7,8-TCDF, 1,2,3,4,6,7,8-HpCDD and OCDD) were detected, ranging from 0.38 to 10 $ng \cdot kg^{-1}$. Concentrations of 2,3,7,8-TCDD/F found in invertebrates ranged from 0.5 to 13 ng $TEQ \cdot kg^{-1}$ww (Pastershank and Muir 1995).

Fish

Compared to levels previously measured in 1987 and 1989 in the mountain whitefish (*Prosopium williamsoni*), concentrations on TCDD/F decreased 2.5 fold in 1992, downstream of the Weldwood of Canada Ltd. BKM in Hinton, AB (Pastershank and Muir 1995). Furthermore, 2,3,7,8-TCDD/F concentrations in fish tissues decreased between 1992 and 1994 (Muir and Pastershank 1997). Of all the fish species, burbot (*Lota lota*) tended to bioaccumulate more PCDD/Fs; however, levels decreased significantly over time: from 33.1 to 68.5 pg $TEQ \cdot g^{-1}$ ww in 1991-1992 to 3.8 pg $TEQ \cdot g^{-1}$ ww in 1994, in the Peace River Basin (Wrona et al. 1996). These declines were linked to a substitution in the bleaching process used by the pulp and paper mill industry.

Wildlife

In the winter of 1991-1992, very low levels of 2,3,7,8-TCDD were detected in mink (*Mustela vison*) from the Peace and Athabasca Rivers region (Wayland 1995a). Dioxin and furan residues in canvasbacks (*Aythya valisineria*) and muskrats (*Ondatra zibethicus*) from the Chipewan reserve in 1992, were also low at an average of 2.6 pg $TEQ \cdot g^{-1}$ and below the detection limits, respectively (Wayland 1996). Common mergansers analysed in 1992 showed low PCDD/F concentrations, birds with the highest 2,3,7,8-TCDF levels were detected downstream of a pulp and paper mill (Wayland 1995b).

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6. TOXIC EFFECTS OF PCDD/Fs OBSERVED IN AQUATIC AND SEMI-AQUATIC ORGANISMS

A multitude of studies on exposure to and subsequent effects of PCDD/Fs exist in the scientific literature. The aim of the present chapter is not to conduct an exhaustive review, but rather to present an overview of the types of toxicity data that exist. Several recent reviews on the effects of PCDD/Fs on aquatic organisms (Fitzsimons 1995; Grimwood and Dobbs 1995; Boening 1998, Environment Canada 2000b), birds (Bosveld and van den Berg 1994; Henshel 1998), mammals (Kerkvliet 1995; Birnbaum and Tuomisto 2000; Yonemoto 2000), and biota in general (Peterson et al. 1993; Crisp et al. 1998) are available and the reader is directed to these reviews for more detailed information on topics that are summarised in this chapter.

Toxic effects that have been commonly documented include mortality (often delayed), decreased body weight gain, decreased feed consumption, thymic atrophy, histopathologic effects, immunotoxicity, developmental and reproductive effects (including endocrine disruption), biochemical effects, neurotoxicity, and carcinogenesis. In fish, the most commonly observed effects are early life stage mortality, associated lesions (*e.g.*, hemorrhaging and oedematous symptoms), and fin necrosis (Grimwood and Dobbs 1995). Clearly, it is unlikely that the complete spectrum of effects would be observed in any single species but the data indicate that PCDD/Fs and related compounds elicit the same qualitative pattern of responses within each species (Safe 1986). Effects observed vary with a number of factors including the dose of the toxic substance, the congener tested, and life stage, strain, species, and gender of the organisms tested. Moreover, the sensitivity of an organism to toxicants depends in part on its capability to metabolise and eliminate these substances.

For reasons discussed in Chapter 2, enzyme induction is not considered a suitable endpoint to evaluate toxic effects of PCDD/Fs and is not discussed here; however, biochemical effects in fish, mammals, and birds are reviewed elsewhere (Goksøyr and Husøy 1998; Wu et al. 2001).

Concentrations of PCDD/Fs in this chapter are expressed as toxic equivalents (TEQs) using the WHO 1998 TEF values for fish, mammals, and birds where appropriate (van den Berg et al. 1998; see TEQ text box on page 9)

6.1 Water-Based Toxicity

There exists a large amount of data from water-based toxicity studies on aquatic organisms. Although uptake from water is an important exposure pathway for only a limited number of species (*e.g.,* guppies, *Poecilia reticulata*), data from water-based toxicity studies can provide information on species sensitivities and relative toxicities of individual congeners. Acute and chronic PCDD/F toxicity data, including lethal, growth, and reproductive endpoints, for fish, amphibians, invertebrates, and plants are summarised below. More limited data exist for aquatic species other than fish, but evidence suggests that amphibians, aquatic invertebrates and aquatic plants are relatively less sensitive to the toxic effects of 2,3,7,8-TCDD.

6.1.1 Fish

Acute and chronic PCDD/F toxicity data for 9 families and 19 species of fish, of which 7 families and 15 species are resident in Canadian waters are discussed in Environment Canada (2000b). The following is a brief discussion on some of the toxic effects of PCDD/Fs on freshwater fish.

Freshwater fish exhibit a wide range of sensitivities to PCDD/Fs. Exposure to 2,3,7,8-TCDD and related compounds may result in a variety of adverse effects in fish, including: reduced survival and growth rates (Mehrle et al. 1988; Hornung et al. 1999); fin necrosis (Kleeman et al. 1988); oedema (Helder 1981); reproductive failure (Walker et al. 1991, 1992); and teratogenic effects (Helder 1981). Rainbow trout (*Oncorhynchus mykiss*) yield the most sensitive responses in terms of lowest observable effect levels (LOELs) when exposed to PCDD/F in water. A LOEL of 0.038 ng·L^{-1} of TEQ_{fish} for growth (after 28 days of exposure) and for mortality (for 28 days of exposure and 28 days of depuration) in juveniles exposed to 2,3,7,8-TCDF is reported by Mehrle et al. (1988). Similarly, Miller et al. (1973) exposed juvenile coho salmon *(O. kisutch)* to nominal concentrations of 2,3,7,8-TCDD ranging from 0 to 5.6 ng·L^{-1} concentrations as low as 0.056 ng·L^{-1} are coincident with increased mortality measured within 60 days following exposure. In a subsequent publication based on the same data, *O. kisutch* fry exposed to 5.6 ng·L^{-1} or more for 48 and 96 hours experienced significant growth rate reductions and mortality (Miller et al. 1979). Lake trout (*Salvelinus namaycush*) and brook trout (*Salvelinus fontinalis*) exhibit the least sensitivity of the salmonids, when exposed as eggs to waterborne 2,3,7,8-TCDD, with LOELs of 10 ng·L^{-1} for mortality to swim-up (Spitsbergen et al. 1991) and 8 ng·L^{-1} for sac fry mortality (Walker and Peterson 1994). While embryotoxicity assays for sensitive endpoints using fish from non-salmonid taxonomic groups yield relatively low LOELs for lesions in medaka (*Oryzias latipes*), fathead minnow (*Pimephales promelas*), northern pike (*Esox lucius*), and guppy (0.4, 0.37, 0.1, 0.1 ng·L^{-1}, respectively) (Miller et al. 1979; Wisk and Cooper 1990a, 1990b; Olivieri and Cooper 1997, respectively), these parameters are still an order of magnitude higher than the LOEL for mortality and growth response of 0.038 ng·L^{-1} for rainbow trout.

On a tissue residue basis, lake trout are nearly as sensitive as rainbow trout to PCDD/F exposure when mortality is compared as the endpoint (Environment Canada 2000b). In addition to evaluating the effects of 2,3,7,8-TCDD on juvenile rainbow trout, Mehrle et al. (1988) examined their response to concentrations of 2,3,7,8-TCDF (<0.001 to 0.44 ng TEQ·L^{-1}); the LOEL for mortality was 27 ng TEQ·kg^{-1} ww. Similar LOELs, ranging from 36 to 61 ng TEQ·kg^{-1} ww (in eggs), for lake trout sac fry mortality are published (Guiney et al. 1996). While salmonids appear to be the most sensitive taxonomic group, the LOEL for lesions (40 ng TEQ·kg^{-1} ww) in fathead minnows exposed as eggs from 0 to 10.2 ng·L^{-1} of 2,3,7,8-TCDD (Olivieri and Cooper 1997) falls within the range of LOELs determined for rainbow and lake trout. Walker and Peterson (1994) found that the tissue-based LOEL for mortality in brook trout sac fry exposed as eggs to 0 to 30 ng·L^{-1} of 2,3,7,8-TCDD was 185 ng·kg^{-1} ww egg. Elonen et al. (1998) exposed eggs from five species of fish resident in Canada to 2,3,7,8-TCDD. Tissue-based LOELs for survival ranged from 270 ng·kg^{-1} ww egg for lake herring to 1 800 ng·kg^{-1} ww egg for northern pike.

6.1.2 Amphibians and Reptiles

Although few researchers have addressed the responses of amphibians to PCDD/Fs, limited data indicate that this group of organisms is relatively insensitive to the toxic effects of 2,3,7,8-TCDD. Eggs of American toad (*Bufo americanus*) and green frog (*Rana clamitans*) exposed for 24 hours to 3 to 30 000 and 300 to 100 000 ng·L^{-1} of 2,3,7,8-TCDD, respectively, showed no significant increase in mortality relative to controls (Jung and Walker 1997). These authors also reported that mean concentrations of 2,3,7,8-TCDD as high as 19 331 and 73 717 ng·kg^{-1} ww have been measured in the American toad and green frog eggs, respectively. Leopard frogs (*Rana pipiens*) are more sensitive to the toxic effects of 2,3,7,8-TCDD as mortality is significantly increased (>10%) in eggs that were exposed to 3 000 ng·L^{-1} and display a mean

concentration of 17 486 ng·kg^{-1} (Jung and Walker 1997). No information was found on the toxic effects of PCDD/Fs to reptiles.

6.1.3 Invertebrates

In general, invertebrates appear to be less sensitive to the toxic effects of water-borne 2,3,7,8-TCDD than fish. For example, the survival rate of the water flea, *Daphnia magna*, exposed to concentrations ranging from 0.2 to 1 030 ng·L^{-1} for 48 hours was similar to that of the control group (Adams et al. 1986). Likewise, *D. magna* exposed for up to 32 days to an average 2,3,7,8-TCDD concentration of 3.1 ng·L^{-1} experienced no adverse effects on growth or reproduction (Yockim et al. 1978). Pupation of mosquito (*Aedes aegypti*) larvae was unaffected by a 17 day exposure to 200 ng·L^{-1} of 2,3,7,8-TCDD (Miller et al. 1973).

Oligochaetes and snails appear to be somewhat more sensitive to 2,3,7,8-TCDD than crustaceans or insects. Miller et al. (1973) reported that a 55 day exposure to 200 ng·L^{-1} of 2,3,7,8-TCDD resulted in a 19% reduction in the reproductive success of the oligochaete, *Paranais* sp. This response was manifested by slower population growth and lower overall biomass in the test group relative to the control group. Similarly, snails (*Physa* spp) exposed to 200 ng·L^{-1} for a period of 36 days experienced a 30% reduction in reproductive success (Miller et al. 1973). Snails (*Helosoma* spp.) following a 32 day exposure to an average concentration of 3.1 ng·L^{-1} experienced no adverse effects on growth or reproduction (Yockim et al. 1978).

6.1.4 Plants

Aquatic plants appear to be relatively insensitive to the toxic effects of 2,3,7,8-TCDD. Yockim et al. (1978) reported that long-term (32 days) exposure to 2,3,7,8-TCDD levels of 3.1 ng·L^{-1} had no effect on the growth or reproduction in the green algae, *Oedogonium cardiacum*. Similarly, Isensee and Jones (1975) reported no adverse effects when they exposed algae (*O. cardiacum*) or duckweed *(Lemna minor)* for 33 days to 2,3,7,8-TCDD concentrations of 1330 ng·L^{-1}. Furthermore, aquatic macrophytes, slender waterweed (*Elodea nuttalli*) and coontail (*Certophyllum emersum*) following exposure to 53.7 ng·L^{-1} for several months experienced no adverse effects (Tsushimoto et al. 1982).

6.2 Sediment-Based Toxicity

Sediments are a complex matrix and are generally heterogeneous in their physical, chemical, and biological characteristics. Sediment-associated organisms may be exposed to PCDD/Fs bound to particulate matter as well as those dissolved in the interstitial and/or overlying waters. Consequently, specialised tests are required to determine the effects of sediment PCDD/Fs on benthic organisms. In spiked-sediment toxicity tests, known quantities of a chemical or specific mixture are added to test sediments and toxicological endpoints are measured. In addition to cause-effect data that is generated from spiked-sediment toxicity tests, numerous other studies have evaluated the toxicity of field-collected sediments that contain a mixture of sediment-associated chemicals (*e.g.*, Call et al. 1991; Ingersoll et al. 1996; Jaagumagi and Bedard 1997). These studies provide evidence for associations between exposure to chemicals in sediments and responses in biota.

Toxicity data from a number of field studies (4 families and 6 species of benthic organisms) have been compiled and evaluated and are described in full in Environment Canada (2000b). The following paragraphs provide examples that illustrate the use of field data in assessing associations between sediment-associated PCDD/F concentrations and toxicological endpoints.

Concentrations of PCDD/Fs are expressed as TEQ_{fish} (ng·kg^{-1} dw) that were calculated using the WHO 1998 TEF values for fish (van den Berg et al. 1998; see TEQ text box on page 9).

Mortality tests, based on field data, for a variety of benthic organisms indicate that concentrations as low as 0.68 ng·kg^{-1} dw can significantly reduce survival (36.2% mortality) in the amphipod *Hyalella azteca* (Ingersoll et al. 1996). These same authors, summarised information collected from benthic surveys of several areas in the Great Lakes basin. They found that TEQ_{fish} levels of PCDD/Fs in sediments from 0.12 ng·kg^{-1} dw (Chironomidae) to 2 100 ng·kg^{-1} dw [*Hexagenia limbata* (mayfly)] affected the relative densities of species present there.

In other studies, the toxicity of field-collected sediments has been assessed using sediment bioassays. For example, sediments collected from Canagagigue Creek, ON, were assessed for their potential toxicity (survival and growth) to an ephemeroptern *(Hexagenia limbata)*, midge larvae *(Chironomus tentans)*, and fathead minnow (Jaagumagi and Bedard 1997). On a TEQ_{fish}, basis, the PCDD/F concentration ranged from 10.86 to 184.79 ng·kg^{-1} dw. Field-collected sediments that significantly reduced survival and growth were deemed 'toxic'. For example, sediments containing a TEQ_{fish} level of 184.79 ng·kg^{-1} dw were significantly toxic (i.e., 50% mortality) to *H. limbata* after 21 days of exposure (Jaagumagi and Bedard 1997). In comparison, sediments with a TEQ_{fish}·level of 10.86 ng·kg^{-1} dw were not significantly toxic (i.e., 0% mortality) to *H. limbata* in the same bioassay (Jaagumagi and Bedard 1997).

Sediments containing a TEQ_{fish} level of 983.49 ng·kg^{-1} dw were significantly toxic (i.e., 50% mortality) to *H. azteca* after 10 days of exposure (Call et al. 1991). Meanwhile, sediments containing a TEQ_{fish} level of 234 ng·kg^{-1} dw were significantly toxic (i.e., 100% mortality) to *Chironomus tentans* (midge) after 10 days of exposure (Ingersoll et al. 1996).

Non-lethal effects of sediment-associated PCDD/Fs include growth deformities, and reductions in weight and length in benthic organisms. At the same time, species abundance and sexual maturity in benthic organisms can also be compromised (Ingersoll et al. 1996; Table 6.1).

Table 6.1. Summary of non-mortality endpoints for sediment-associated PCDD/Fs.

Species	Endpoint	PCDD/F Concentration (ng TEQ_{fish}·kg^{-1} dw)
Gastropoda	Low abundance	0.19
C. tentans	Growth deformities	1.07
C. tentans	Reductions in weight	1.09
H. azteca	Reduction in length	3.02
H. azteca	Sexual maturity	3.61

Note: Only the studies with the lowest significant PCDD/F concentrations for each endpoint are discussed (Ingersoll et al. 1996). Please consult Environment Canada (2000) for a thorough examination of all the studies.

6.3 Dietary-Based Toxicity

6.3.1 Amphibians and Reptiles

No toxicity studies were located on the effects of dietary consumption of PCDD/Fs to amphibians and reptiles. Single doses of 2,3,7,8-TCDD, administered by interperitoneal injection, of up to 1 000 000 ng·kg^{-1} bw to bull frog (*Rana catesbeiana*) tadpoles and 500 000 ng·kg^{-1} bw to adults, for a period of 50 and 35 days, respectively, failed to produce any dose related effects on survival (Beatty et al. 1976). This study along with water-based

exposure studies discussed above suggest that amphibians are relatively insensitive to 2,3,7,8-TCDD.

6.3.2 Fish

Ingestion of contaminated food can be just as important as uptake of PCDD/Fs from water for some fish (Loonen et al. 1993). Dietary exposure to fish is only briefly outlined here, please consult Hawkes and Norris (1977) and Tietge et al. (1998) for further discussions. Tietge et al. (1998) fed adult brook trout food contaminated with TCDD concentrations to achieve whole body loading doses of 75, 150, 300, 600, and 1200 $ng \cdot kg^{-1}$ bw. These authors estimated net dietary assimilation of TCDD to be 89% of the applied dose, and the distribution of TCDD generally followed the distribution of lipids in live, gonads, fat, blood, and muscle. Survival, growth, gonad development, and egg production were not affected in any treatment. The onset of spawning was delayed by 13 days in the 1200 $ng \cdot kg^{-1}$ group, suggesting that TCDD might have affected ovulation.

6.3.3 Mammals

The majority of the dietary-based mammalian toxicity studies have been performed on mice, rats, and guinea pigs. Acute lethal doses of 2,3,7,8-TCDD span four orders of magnitude in mammalian receptors (Environment Canada 2000b). For many species, mortality due to single doses of 2,3,7,8-TCDD is delayed (i.e., 5 to 45 days; U.S. Environmental Protection Agency 1987). In addition to mortality, decreased body weight gain and/or food consumption are observed in guinea pigs, rats and mink. Other symptoms of acute toxicity may include depletion of adipose tissue, ulcerations of the stomach, mottled and discoloured livers and kidneys, and changes in relative organ weights (Hochstein et al. 1988, 2001; Environment Canada 2000b). As this report focuses on the aquatic environment. For a review of studies using other test species, please consult De Caprio et al. 1986; Suter-Hofmann and Schlatter 1989; Nagao et al. 1993; and Environment Canada 2000b).

Few data exist on the acute toxicity of 2,3,7,8-TCDD to wildlife species that consume aquatic biota; however, mink may be among the most sensitive species (LD_{50} = 4200 $ng \cdot kg^{-1}$ bw; Hochstein et al. 1988). Non-lethal effects include decreased body weight gain and food consumption (Hochstein et al. 1988, 2001). For example, Hochstein et al. (1988) reported significant decreases (11%) in the body weights of mink (28 days post-exposure) following a single administration of 2500 $ng \cdot kg^{-1} \cdot$bw of 2,3,7,8-TCDD.

Numerous studies demonstrate that 2,3,7,8-TCDD is foetotoxic and teratogenic in rodents, with effects commonly observed at doses that are not overtly toxic to the mother (Nagao et al. 1993). A study assessing the cumulative effects of dioxin-like compounds (PCDD/Fs and PCBs) on reproduction and development in mink is available. Mink were fed diets containing 0, 10, 20 or 40% contaminated carp from Lake Michigan prior to and throughout the reproductive period (26 weeks total). Mink consumed an average of 0.011, 0.013, 0.012, and 150 $ng \cdot kg^{-1}$ $bw \cdot d^{-1}$ of PCDD/Fs, or 0.23, 3.89, 7.34, and 10.2 $ng \cdot kg^{-1}$ $bw \cdot d^{-1}$ of TEQ_{mam}, respectively, with PCDD/Fs contributing 6-32% of the total TEQ_{mam} concentration (Heaton et al. 1995; TEQ_{mam} re-calculated with WHO 1998 TEFs for mammals). Females fed the 40% carp diet whelped significantly fewer kits, and all kits were either stillborn or dead within one day. There is a significant inverse dose-dependent response between weights of kits and proportion of carp in the maternal diet, with 20 and 40% carp diet groups significantly different from the control. Reduced body weights of kits under the 10% carp diet occurred at three weeks of age whereas reduced survival of kits occurred at three and six weeks of age. Percent survival to six weeks of age (weaning) are 85,

28, 11.5, and 0% for the 0, 10, 20, and 40% carp diets, respectively. Relative organ weights of kits whelped and nursed by treated females were generally less than those of the control group. TEQ concentrations in the liver of the adult females ranged from <10 to 656 $ng \cdot kg^{-1}$ ww (as estimated by the H4IIE bioassay; Tillitt et al. 1996).

Research into 2,3,7,8-TCDD effects on the immune system has been almost entirely conducted on mice, despite immunosuppression being reported as a common symptom associated with 2,3,7,8-TCDD exposure (Environment Canada 2000b). In mice, 2,3,7,8-TCDD affects both the specific and non-specific defence mechanisms of the immune system that in turn adversely affects host defence capabilities (Holladay et al. 1991). Cellular and humoral immunity are the two main components of the specific immune response whereas the complement system is the main component of the non-specific immune response; all are susceptible to 2,3,7,8-TCDD. For further information of the immunotoxic effects of 2,3,7,8-TCDD, the reader is directed to a review by Vos et al. (1998).

Several studies indicate that chronic dietary exposure to low levels of 2,3,7,8-TCDD may result in an increased incidence of tumours in rodents (see Environment Canada 2000b). Rats appear more sensitive than mice, and males more sensitive than females, for both species. Currently, there are no known studies addressing this in wildlife that consume aquatic biota.

6.3.4 Birds

Although the available data, particularly for piscivorous birds, are limited, it appears that birds are slightly less sensitive than mammals to the effects of PCDD/Fs. Nonetheless, the effects associated with acute and chronic exposures to PCDD/Fs are similar, including lethality, reduced growth rates, liver enlargement, and reproductive impairment.

Limited data on the acute oral toxicity of 2,3,7,8-TCDD to avian species indicate that birds exhibit a broad range of sensitivities to this substance (Environment Canada 2000b). Mallard ducks (*Anas platyrhynchos*) appear to be relatively resistant to 2,3,7,8-TCDD, with a LD_{50} of > 108 000 $ng \cdot kg^{-1}$ bw (Hudson et al. 1984). Only one study examining the effects of maternally administered dioxin on reproduction in birds is available and its test species, the ring-necked pheasant (*Phasianus colchicus*) is non-aquatic (Nosek et al. 1992). Birds treated with 1000 $ng \cdot kg^{-1}$ bw for 7 weeks experienced delayed onset of mortality, significant reduction in egg production, and embryos from those eggs had a significantly higher cumulative percent mortality.

Population declines and physiological abnormalities among other effects are indicators of potential organochlorine exposure in birds, particularly among piscivorous birds (Ewins et al. 1994). These effects have been extensively studied in the Great Lakes region and on the western Canadian coast. Results from studies employing field collected eggs demonstrate a strong association between exposure of dioxin-like compounds and impaired reproduction, although, the relative contributions to the toxic effects by individual compounds may vary significantly between species and locations (Bosveld and van den Berg 1994). The relatively recent appearance of symptoms of PCDD/F toxicity may be due to declines in other organochlorine compounds, most notably DDT and its metabolite DDE. Owing to pesticide contamination, eggs were previously unable to survive long enough for PCDD/F toxicity to manifest (Giesy et al. 1994). Moreover, embryonic effects typically associated in dioxin toxicity were not routinely monitored until the 1980s. Nevertheless, effects associated with dioxin toxicity have clearly lessened in recent years, correlating well to reductions in PCDD/F levels.

Great blue heron (*Ardea herodias*) chicks hatched from eggs collected from five sites in and around the Strait of Georgia, British Columbia have asymmetric brains (Henshel et al. 1995). Forebrain asymmetry correlates best with 2,3,7,8-TCDD concentration in the egg, although forebrain depth is more strongly associated with TEQs (based on Safe 1990 TEFs), indicating that other PCDD/Fs may also influence brain development. Brain asymmetry is commonly associated with 2,3,7,8-TCDD levels in eggs above 60 ng·kg^{-1} ww, though effects occur at concentrations as low as 13 ng·kg^{-1} ww (Henshel 1998). EC_{50}s for asymmetry in brain angle, depth, height, and width are 53, 44, 40, and 32 ng·kg^{-1} ww of 2,3,7,8-TCDD and 99, 79, 83, and 65 ng·kg^{-1} ww of TEQs (based on Safe 1990 TEFs), respectively (Henshel 1998).

Chick oedema disease, characterised by jelly-like subcutaneous oedema on the breast, occurred in 15-33.3% of great blue heron chicks hatched from eggs collected from British Columbia (Hart et al. 1991). Chicks also measured significantly smaller in yolk-free weight, kidney and stomach weight, tibia length and weight, beak length, and down follicle density than those from the reference/control site. Levels of 2,3,7,8-TCDD in the eggs correlate significantly to: yolk-free weight; tibia length weight; beak length; and, kidney and stomach weights. Mean 2,3,7,8-TCDD levels in eggs are 173 ng·kg^{-1} ww of TEQ for the BC colonies (Hart et al. 1991). In a similar study, subcutaneous oedema was diagnosed in great blue heron chicks collected from British Columbia in 1988 but not 1991. This observation corresponds to a significant decrease in PCDD/F levels in heron eggs at the site between the two years (from ~530 to 100 ng·kg^{-1} ww of TEQs) (Sanderson et al. 1994a; based on Safe 1990 TEFs and include PCDD/Fs and PCBs). A similar trend was noted for herons collected in Vancouver, BC from 1988 to 1992 (Sanderson et al. 1994a). Morphological measurements including, body weight, yolk weight, wing length, and brain asymmetry, are negatively correlated to TEQ levels (81 to 500 ng·kg^{-1} ww; based on Safe 1990 TEFs and include PCDD/Fs and PCBs) in eggs of double-crested cormorants (*Phalacrocorax auritus*) collected from five colonies across Canada (Sanderson et al. 1994a; Henshel et al. 1997).

Bald eagles (*Haliaeetus leucocephalus*) appear to be relatively tolerant of dioxin toxicity. No significant concentration-related morphological, physiological, or histological effects were found in bald eagle chicks collected as eggs from pulp mill and references sites along the southern coast of British Columbia (Elliott et al. 1996c). Total TEQ_{bird} concentrations (WHO 1998 TEFs for birds) ranged from 7600 ng·kg^{-1} lipid in the egg yolks from West Vancouver Island to 25 630 ng·kg^{-1} lipid in those from the Powell River (% lipid ~8.8-23%); PCDD/Fs account for 22 to 60% of the total TEQ_{bird}.

Reproductive impairment occurred in wood ducks (*Aix sponsa*) collected downstream from a point source in Arkansas. Nests closest to the point source (9 km and 17 km downstream) experienced a significant reduction in eggs hatched and ducklings that left the nest compared to nests located 58 km and 111 km downstream (White and Seginak 1994; White and Hoffman 1995). Of eggs that fail to hatch, 20% are cracked and desiccated; 45% are addled. All eggs are contaminated with a variety of PCDD/F congeners with average concentrations of individual congeners as high as 60 ng·kg^{-1} ww. Researchers estimated a threshold TEQ level of 20-50 ng·kg^{-1} ww (based on I-TEFs) for nest success, hatching success, and duckling production (White and Seginak 1994; White and Hoffman 1995).

Only a single study examining the effects of PCDD/Fs on the avian immune system (white leghorn chicks, *Gallus domesticus)* is available (McKinney et al. 1976). Reduced spleen weight and depletion of lymphocytes in the spleen were observed when day old chicks were exposed to 2,3,7,8-TCDF at a TEQ_{bird} dose of 1000 and 5000 ng·kg^{-1} bw·d^{-1} for 21 days

No studies were located on the carcinogenic effects of PCDD/Fs in avian species.

7. MANAGEMENT OF PCDD/Fs IN THE CANADIAN AQUATIC ENVIRONMENT

Polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) have been highly regulated in several countries for the last decade or so, after the discovery of their potential toxic effects on human and ecosystem health. The toxicological dangers associated with these substances were observed for the first time during the Vietnam War, as a result of their presence in the herbicide Agent Orange (Sharpe 2000). Their presence was also identified in the stack gas of municipal waste incinerators in 1977 (Kilgroe 1996) and in a hog-fuel furnace in 1987 (Luthe and Prahacs 1993).

Dioxin contamination was recognised in Canada after the outbreak of reproductive and developmental effects in fish eating birds in the Great Lakes in the mid 1960's (Ruchel and Luscombe 1998). The association of dioxins with pulp mill effluents and paper products was discovered in the United States in 1987 (Harrison and Hoberg 1991). In the early 1990s, formation of dioxins during the chlorine bleaching of paper products became an issue, increasing people's awareness towards this problem (Glenn 1999). The Canadian federal government officially recognised dioxins and furans as toxic substances in 1990, under the *Canadian Environmental Protection Act* (*CEPA*), based on their deleterious effects and impacts on the environment (Environment Canada and Health and Welfare Canada 1990). Since that time, the elaboration and application of control measures have been taking place against the anthropogenic inputs of dioxins and furans into the Canadian environment. Most chemical sources of dioxins and furans are already subject to stringent controls in Canada.

This chapter highlights the principal regulations, legislation, objectives and other control measures and programs at the federal, provincial and territorial levels, concerning dioxin and furan releases to the Canadian aquatic environment (Table 7.1). Incorporation of other compartments, such as soil and air, was performed when possible, as numerous sectors and activities have an impact on the aquatic environment. Many of the reported legislation, regulations, objectives and control measures are generic and may not specifically address dioxins and furans; however, they indirectly allow for their control. Numerical details on selected regulations and an evaluation of their efficiency are provided, when available. Certain international approaches are also addressed when Canada is directly concerned. Finally, a number of best management practices addressing dioxins and furans are reviewed.

Table 7.1 Legislation, regulations, objectives, guidelines and associated control measures relevant to dioxins and furans, as of March 2002 (listed in chronological order).

Jurisdiction	Relevant Legislation, Regulations, Objectives, Guidelines and Control Measures
Federal	1979 - *Pest Control Products Regulations* - limit 2,3,7,8-TCDD to a maximum of 100 $\mu g \cdot L^{-1}$ in 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and 100 $\mu g \cdot L^{-1}$ for 2-(2,4,5-trichlorophenoxy) propanoic acid (fenoprop)
	1981 - *Pest Control Products Act* - limits dioxin content in 2,4-dichlorophenoxyacetic acid (2,4-D) to 10 $\mu g \cdot L^{-1}$ of any specific dioxin isomer 1985 - limits total hexachlorodioxin in technical pentachlorophenol or tetrachlorophenol to a maximum of 5 $mg \cdot L^{-1}$
	1985 - *Arctic Waters Pollution Prevention Act* - prevents the pollution of areas of the Arctic waters adjacent to the mainland and islands
	1985 - *Canada Water Act* - now part of the *Canadian Environmental Protection Act*, restricts the pollution of waters
	1985 - *Department of the Environment Act* - preserves, controls and enhances the quality of the natural environment
	1985 - *Fisheries Act* 1992 - *Pulp and Paper Effluent Regulations* Environmental Effects Monitoring (EEM) Program - monitoring of dioxins and furans in fish tissues in pulp and paper receiving waters
	1985 - *Food and Drugs Act* - Health and Welfare Canada developed a PCDD/F human consumption guideline of 20 ng L^{-1} TEQ for edible portions of fish
	1987 - Federal Water Policy
	1988, 1999 - *Canadian Environmental Protection Act* (*CEPA*) 1990 - Priority Substances List (PSL) - PCDD/F, declared toxic as defined in section 11 of *CEPA*, were added to PSL for future regulation under *CEPA* 1992 - *National Pulp and Paper Regulatory Package for the Canadian Pulp and Paper Industry* - monitoring of dioxins and furans in mills effluent, no surveillance in receiving waters 1992 - *Pulp and Paper Mill Effluent Chlorinated Dioxins & Furans Regulations* 1992 - *Pulp and Paper Mill Defoamer and Wood Chip Regulations* 1992 - National Pollutant Release Inventory (NPRI) 1995 - Federal-Provincial Advisory Committee for *CEPA* (CEPA-FRAC) - established the Federal/Provincial Task Force on Dioxins and Furans 1999 - release of the Dioxins & Furans & Hexachlorobenzene Inventory of Releases
	1990 - *Federal Mobile PCB Treatment and Destruction Regulations* - impose a restrictive operation on systems disposed on federal land identified as potential sources of PCDD/Fs
	1991 to 1998 - Fraser River Action Plan (FRAP) - the goals of this plan were to restore the natural productive capacity, to reduce and clean up pollution, and to develop a management program for the Fraser River Basin based on sustainable development principles
	1992 - *Canadian Environmental Assessment Act* - encourages responsible authorities to promote a healthy environment
	1992 - *Export and Import of Hazardous Wastes Regulations* - Environment Canada is controlling the shipments of hazardous wastes and the action plan in case of an accidental spill
	1992 - *Transportation of Dangerous Goods Act (TDGA)* - promotes public safety in the transportation of poisonous (toxic) and miscellaneous products

Jurisdiction	Relevant Legislation, Regulations, Objectives, Guidelines and Control Measures
	1994 - ARET Initiative (Accelerated Reduction / Elimination of Toxic Substances)
	1994 - Canada-Ontario Agreement (COA) 1994, 2002 - Canada-Ontario Agreement (COA) Respecting the Great Lakes Basin Ecosystem 1998 - Canada-Ontario Agreement (COA) Toxic Substances Emission Inventory
	1994 - Chlorinated Substances Action Plan
	1995 - Memorandum of Understanding (MOU) between Environment Canada and importers of chloranil - prevents the release of chloranil which contains small concentration of dioxins and furans
	1995 -Toxic Substances Management Policy (TSMP) - program introduced in 1995 to target the virtual elimination from the environment of toxic, bioaccumulative and persistent substances resulting from human activities 1997 - TSMP: PCDD/Fs - PCDD/Fs satisfy criteria for track 1 substances under TSMP, and therefore, are stated for virtual elimination from the environment 1999 - TSMP integrated into *CEPA*
	1998 – Northern River Ecosystem Initiative (NREI) - program introduced in 1998 to address the various recommendations established under the Northern River Basins Study
	1999 - Strategic Option Process (SOP) for the Management of *CEPA*-toxic substances - voluntary approach, developed by Environment Canada and Health Canada with the participation of various organisations, managing toxic substances in order to reduce their potential of exposure in various industrial sectors (Electric Power Generation, Steel Manufacturing and Wood Preservation) and determine if regulations are needed under *CEPA*
	2001 - Bill 20 - *Drinking Water Protection Act* - prohibits the contamination of drinking water via the reporting of threats
National	1969 - National Air Pollution Surveillance (NAPS) Network - joint program between the federal and provincial governments monitoring ambient Canadian air quality and measuring potentially toxic air contaminants
	1998 - Canada-Wide Accord on Environmental Harmonisation - represents a common vision to govern the partnerships between the federal, provincial and territorial governments by agreeing to the “polluter pays” principle and pollution prevention strategies 2001 - Canada-Wide Standards for Dioxins and Furans
	Canadian Council of the Ministers of the Environment (CCME) Codes of Practice and Guidelines 1989 - CCME Operating and Emission Guidelines for Municipal Solid Waste Incinerators 1989 - CCME Code of Practice for Used Oil Management in Canada 1990 - CCME Guidelines for Mobile Polychlorinated Biphenyl Destruction Systems 1990 - CCME Guidelines for Mobile Polychlorinated Biphenyl Treatment Systems 1991 - CCME National Guidelines for the Landfilling of Hazardous Waste 1991 - Interim Canadian Environmental Quality Criteria for Contaminated Sites - environmental PCDD/Fs cleanup criteria, expressed in I-TEQs - Interim assessment criteria for soil: 0.00001 $\mu g \cdot g^{-1}$ - Interim remediation criteria for soil: - agricultural: 0.00001 $\mu g \cdot g^{-1}$ - residential/parkland: 0.001 $\mu g \cdot g^{-1}$ 1992 - CCME Guidelines for the Management of Biomedical Waste in Canada 1992 - CCME National Guidelines for Hazardous Waste Incineration Facilities 1996 - CCME National Guidelines for the Use of Hazardous and Non-Hazardous Wastes as Supplementary Fuels in Cement Kilns

Jurisdiction	Relevant Legislation, Regulations, Objectives, Guidelines and Control Measures
	1998 - CCME Policy for the Management of Toxic Substances (PMTS) - sets out a comprehensive and concerted approach for the management of toxic substances; dioxins and furans are identified as toxic substances
	2001 - Canadian Tissue Residue Guideline for the Protection of Consumers of Aquatic Life and Sediment Quality Guidelines for the Protection of Aquatic Life
	2002 - Canadian Soil Quality Guidelines for the Protection of Environmental Health (including groundwater)
Alberta	1992 - *Environmental Protection and Enhancement Act* 1996 - *Waste Control Regulation* - regulates tetra-, penta-, hexa - chlorodibenzo-p-dioxins and chloro-dibenzofurans in concentrations greater than 1 $\mu g \cdot L^{-1}$
	1996 - *Water Act* - supports and promotes the management and conservation of waters
	1998 - Water Quality Guidelines for the Protection of Freshwater Aquatic Life - 2,3,7,8-TCDD: acute <10 and chronic: <0.01 $ng \cdot L^{-1}$
British Columbia	1990 - *Pulp Mill and Pulp and Paper Mill Liquid Effluent Control Regulation* 1992 - *Port Alberni Pulp & Paper Effluent Regulations*
	1991 - Emission Criteria for Biomedical Waste Incinerators
	1996 - *Environment Management Act* - management, protection and enhancement of the environment
	1996 - *Ministry of Environment Act* - administers matters related to the environment
	1996 - *Waste Management Act* *Special Waste Regulation* 1996 - *Contaminated Sites Regulation*
Manitoba	1987 - *Environment Act* - sustains the quality of the environment
	1988 - *Atmospheric Pollution Regulation* - prohibits the discharge of any air contaminant into the atmosphere
	1990 - Manitoba Water Policies - set of guidelines designed to protect and enhance water quality and conservation; standard for dioxins and furans is currently under development
New Brunswick	1973 - *Clean Environment Act* - controls the release of contaminants into the environment 1982 - *Water Quality Regulation* - limits the discharge of contaminants in water
Newfoundland	1990 - *Environmental Assessment Act* - protects the environment and life quality in the province
	1995 - *Environment Act* - restricts the discharge or deposit of any kind of material that may cause pollution or impair the water quality 1989 - *Mobile PCB Destruction Facility Regulations* - limit the concentrations of dioxins and furans emitted in the environment 1996 - *Air Pollution Control Regulations* - annual average air quality standard of 5 pg $TEQ \cdot m^{-3}$ for PCDD/Fs - a PCDD/F standard of 20 pg $TEQ \cdot m^{-3}$ for an average 72 hours period
	1998 - *Waste Management Act* - supports and promotes the protection, enhancement and wise use of the environment through waste management programs
Northwest Territories	1988 - *Environmental Protection Act* - consolidation of spill contingency planning and reporting regulations

Jurisdiction	Relevant Legislation, Regulations, Objectives, Guidelines and Control Measures
	- Environmental Guideline for Industrial Waste Discharges in the NWT - land filling is prohibited if a leachate contains PCDD/Fs (hexa-, penta-, tetra) in a concentration greater than 1 $\mu g \cdot L^{-1}$ Guideline for the General Management of Hazardous Waste in the NWT storage and management of hazardous waste
Northwest Territories and Nunavut	1988 - *Environment Rights Act* - citizens of the Northwest Territories and Nunavut have the right to protect and obtain information concerning the quality, quantity or concentration of any chemical released in the environment
	1988 - *Natural Resources Conservation Trust Act* - promotes through education, research and demonstration the awareness, enhancement and protection of the environment
	1988 - *Water Resources Agreements Act* - regulates and controls the quality and quantity of water resources
Nova Scotia	1994-1995 - *Environment Act* - supports and promotes the protection of the environment
Nunavut	1999 - *Environmental Protection Act* - preserves, protects and enhances the quality of the environment
Ontario	1990 - *Environmental Protection Act* 1992 - *Waste Management Act* - provides an environmental evaluation for the safe disposal of hazardous liquid industrial waste 1993 - Effluent Monitoring and Effluent Limits - Pulp and Paper Sector, Effluent monitoring and Effluent Limits - Petroleum Sector 1995 - Effluent Monitoring and Effluent Limits - Inorganic Chemical Sector, Effluent Monitoring and Effluent Limits - Organic Chemical Manufacturing Sector 1996 - Combustion and Air Pollution Control Requirements for New Municipal Waste Incinerators (Guideline A-7) - dioxin and furan emissions must not exceed 0.14 $ng \cdot m^{-3}$ (I-TEQ) 1997 - Guidelines for Use at Contaminated Sites in Ontario
	1990 - *General - Air Pollution Regulations* - provides two kinds of standards to protect air quality: Ambient Air Quality Criteria (AAQC) for the general quality of air and Point of Impingement (POI) for the control of air emissions from industrial sources of pollution
	1990 - *Mobile PCB Destruction Facilities Regulations* - restrictive use in accordance with standards concerning dioxins and furans
	1990 - *Ontario Water Resources Act* - prohibition of the discharge of polluting material
	1992 - Ontario's Ministry of the Environment and Energy List of Candidate Substances for Bans, Phase-Outs or Reductions - identifies the inherent toxic substances (PCDD/Fs included in the list) and opportunity to ban or phase-out these substances
	1993 - *Clean Water Regulation for the Pulp and Paper Sector* - contains effluent limits for all direct discharge mills
	1994 - Ambient Air Quality Criteria - 30 pg $TEQ \cdot m^{-3}$ (dioxins) - 5 pg $TEQ \cdot m^{-3}$ (dioxins - 24 hours)
	1994 - Ontario Drinking Water Objectives - interim maximum acceptable concentration (IMAC) for dioxins and furans set to 15 pg $TEQ \cdot L^{-1}$ (I-TEQs)
	Residential Soil Remediation Criterion - 1000 pg $TEQ \cdot g^{-1}$ Agricultural Soil Remediation Criterion - 10 pg $TEQ \cdot g^{-1}$

Jurisdiction	Relevant Legislation, Regulations, Objectives, Guidelines and Control Measures
	Provincial Water Quality Guideline - 0.02 pg·L^{-1}
	1999 - The Great Eastern Ontario Wood Stove Change-Out Program to Reduce Particulate and Toxics from Residential Wood burning - promotes cleaner, safer and more efficient residential wood burning by offering rebates on new cleaner-burning approved appliances
Prince Edward Island	1988 - *Environmental Protection Act* - management, protection and enhancement of the environment
Québec	1977 - *Environment Quality Act* 1992 - *Pulp and Paper Mill Regulation* - limits total 2,3,7,8-TCDD (TEQ) to a concentration less than 15 pg·L^{-1} in any effluent 1997 - *Hazardous Materials Regulation* - restriction on discharge of contaminants into the environment
Saskatchewan	1980 - *Environmental Assessment Act* - provides a mechanism for requiring and undertaking environmental impact assessment
	1984 - *Environmental Management and Protection Act* - enhancement and protection of the environment 1981 - *Environmental Spill Control Regulations* - provides requirements to report a spill and prohibit its disposal
	1986-1987-1988 - *Clean Air Act* - controls, prohibits and regulates air pollution by addressing the emission, transport and deposition of air contaminants
	1989 - *Clean Air Regulations* - regulate the concentration of air contaminants
Yukon Territory	1991 - *Environment Act* - ensures the wise management of the Yukon environment by regulating waste management and release of contaminants and hazardous substances in the environment 1995 - *Special Waste Regulations* - restrict the release, transport and dilution of special waste in the environment 1996 - *Contaminated Sites Regulations*
International (programs in which Canada is a participating country)	1909 - International Joint Commission 1972 - Water Quality Objective - a 10 pg TEQ·L^{-1} limit for 2,3,7,8-TCDD was established for transboundary waters (water, sediment or tissue of aquatic organisms) between Canada and the United States 1972 / 1978 - Canada-U.S. Great Lakes Water Quality Agreement (GLWQA) 1987 - Remedial Action Plans (RAPs) for Great Lakes Areas of Concern (AOCs) 1987 - Lake Erie, Ontario and Superior Lakewide Management Plans 1990 - Integrated Atmospheric Deposition Network (IADN) - recommendations that Lake Superior be designated as a demonstration area where discharges and emissions of toxic substances would not be permitted 1990 - Binational Program to Restore and Protect the Lake Superior Basin 1992 - International Joint Commission on Great Lakes Water Quality 1997 - Great Lakes Binational Toxics Strategy (GLBTS) - Canada / United States Strategy for the Virtual Elimination of Persistent Toxic Substances in the Great Lakes
	1986 - Bilateral Agreement between Canada and the United States - controls the transport of dangerous wastes between countries
	1986 - Great Lakes Toxic Substances Control Agreement 1990 - Great Lakes Commission Regional Air Pollutants Inventory Development

Jurisdiction	Relevant Legislation, Regulations, Objectives, Guidelines and Control Measures
	System (RAPIDS) - collection of air pollutant emissions, including dioxins and furans
	1988 - North Atlantic Treaty Organisation on the Challenges of Modern Society (NATO-CCMS) - Pilot Study on International Information Exchange on Dioxins and Related Compounds
	1991 - Arctic Monitoring and Assessment Programme (AMAP) - provides reliable information as well as scientific advice on action to be taken in order to reduce and eliminate Arctic contaminant threats
	1991 - Northern Contaminants Program (NCP) - the objective of this program is to reduce or eliminate contaminants in northern traditionally harvested food and to assist the community in making decision regarding their food use
	1994 - Commission for Environmental Co-operation Tri-lateral North American Regional Action Plan - co-operation established between Canada, the United States and Mexico to deal with transboundary environmental concerns 1999 - North American Agreement on Environmental Co-operation (NAAEC) Resolution on Sound Management of Chemicals (SMOC) - North American Regional Action Plans (NARAPs) - reduce or phase out dioxins and furans from the environment
	1994 - North American Free Trade Agreement (NAFTA) - builds environmental safeguards between Canada and the United States - evaluation of activities known as micro-sources of dioxins and furans
	1998 - United Nations Economic Commission for Europe (UNECE) Long-Range Transboundary Air Pollution (LRTAP) Initiative - agreement of 43 countries on a regional Persistent Organic Pollutants (POPs) - Protocol controlling, reducing and eliminating discharges, emissions and losses of persistent organic pollutants, including dioxins, below a level selected for each country (levels of 1990 for Canada)
	2000 - Office of the Great Lakes - Lake Huron Initiative Action Plan - targets the immediate efforts to reduce or/and eliminate critical or priority (dioxins) pollutants in order to protect or maintain fish and wildlife habitat as well as the biodiversity of Lake Huron
	2001 - Stockholm Convention on Persistent Organic Pollutants

7.1 Selected Federal Initiatives

The following initiatives were selected from the list above based on the large amount of information and description available and their relevance towards the dioxins and furans issue.

7.1.1 The Northern River Ecosystem Initiative (1998-2003)

On the basis of the recommendations, findings and results obtained following the completion of the Northern River Basin Study (NRBS), the governments of Alberta, Northwest Territories and Canada took the initiative of pursuing the environmental management of the Athabasca, Peace and Slave Rivers and their respective basins. Several programs and initiatives are presently being developed to ensure the examination and establishment of the recommendations found under the NRBS.

Consequently, actions were initiated under the *Northern Rivers Ecosystem Initiative* (NREI), launched in March 1998, to respect the various engagements concluded at the time of the NRBS. This five year program incorporates many social priorities, such as: pollution prevention; hydrology; endocrine disruptions; contaminants (including dioxins and furans); drinking water; nutrients and an enhanced surveillance of environmental effects (NREI 1999).

Many NRBS recommendations have been addressed by the NREI. The elimination of the use, production and discharge of contaminants (including PCDD/Fs), within a period of 10 years, has already started. Significant reduction in the amount of dioxins and furans found in the effluents, fish tissues and other wildlife are associated with the improvement of pulp and paper technologies, especially at the Weldwood mill, Hinton and at the Weyerhaeuser mill, Grande Prairie; the numerous PCDD/F measures developed and the implementation of federal and provincial regulations and laws (*Environmental Protection Act*, *Industrial Effluent Limits Policy* of Alberta, *CEPA*, *Fisheries Act* and *TSMP*). Moreover, oxygen delignification and elemental chlorine-free technologies are now accessible and considerably reduce PCDD/F levels found in the aquatic environment. Furthermore, Alberta and Canada governments have initiated a study focusing on persistent contaminants found in fish and are examining fish living downstream of Hinton and Grande Prairie mills. The results will be further scrutinised and compared to the NRBS reported concentrations.

Contaminant inputs in Wapiti and Smoky Rivers were reduced to satisfy the NRBS recommendations. Industries were approached to develop action plans aiming for the reduction in the loading of their contaminants. One of them, Weyerhaeuser Canada, in co-operation with Alberta Health and Wellness and Alberta Environment, is conducting additional fish contaminant sampling to facilitate the review of the existing fish consumption advisories for dioxin and furans on the Smoky and Wapiti rivers (NREI 1999).

In 1997, a sediment and contaminant transport study was undertaken in the Great Slave Lake Delta to assess the contaminant distribution and sediment deposition; as requested in the NRBS. Until now, the results show that concentrations of PCDD/Fs are higher in the delta distribution channels of the Slave River than in the mainstem of the river or lakebed. In the future, more evaluations need to be conducted in the delta habitats.

Since 1996, numerous progress were made based on different actions, laws, regulations, international agreements and studies. With initiatives such as a national program on pollution prevention (*CEPA*), more stringent discharge limits for pulp and paper mills and signature of the *Mackenzie River Basin Transboundary Waters Master Agreement* in 1998 and the creation of the Mackenzie River Basin Board, environmental progress were observed and many more are probably on the verge of being fulfilled.

7.1.2 The ARET (Accelerated Reduction/Elimination of Toxics) Initiative (1994)

This multi-stakeholder pollution prevention program, launched in March 1994, involved and promoted co-operation between industry, health and professional organisations, and governmental departments. Its purpose was to diminish adverse effects of toxic substances on human health and the environment by accelerating the reduction or elimination of toxic substance emissions (ARET Secretariat 2000). The ARET substances list includes 117 toxic substances, distributed in five distinct categories for future action:

- A-1: Substances that are considered persistent, bioaccumulative and toxic (PBT) (*e.g.*, 2,3,7,8-tetrachlorodibenzo-p-dioxin and 2,3,7,8-tetrachlorodibenzofuran);

- A-2: No consensus was reached for these substances regarding their addition in category A-1 (*e.g.*, cadmium);

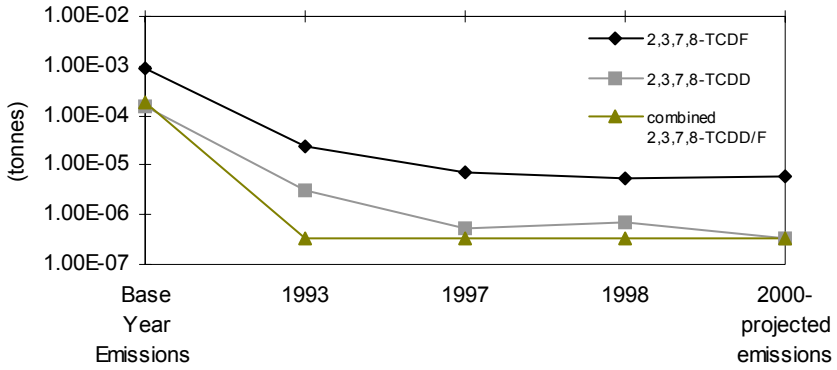
- B-1: Substances that are considered bioaccumulative and toxic (*e.g.*, anthracene);

- B-2: Substances that are considered persistent and toxic (*e.g.*, zinc);

- B-3: Substances that are considered toxic only (*e.g.*, benzene).

The ARET program aimed for the virtual elimination (90% as of 2000) of category A-1 emissions, while a reduction of other toxic categories to levels insufficient to cause harm to human health and the environment was considered (Environment Canada 2000c). To achieve this objective, selected Canadian companies, institutions and government departments and agencies volunteered to reduce or eliminate their toxic emissions by the year 2000. As of 1998, 316 facilities from 169 companies and governmental organisations submitted action plans, while an additional 142 organisations filed a Declaration of Support for ARET (ARET Secretariat 2000).

Overall, emissions of ARET substances in 1998 totalled 13,026 tonnes, a 67% decrease from base-year emission levels (ARET Secretariat 2000). Furthermore, as of 1998, substances included in the category A-1 have been reduced by 54%. In 1998, 2,3,7,8-TCDD/F already met the short-term emission reduction targets for the year 2000, showing a reduction of 100% and 99%, respectively (Figure 7.1). Short-term goals for the ARET program were established until the year 2000; a renewal process is currently being initiated (ARET Secretariat 2000).



(data source: ARET Secretariat 2000)

Figure 7.1. ARET Initiative: Short-term reduction in 2,3,7,8-TCDD/F emissions.

7.1.3 The Chlorinated Substances Action Plan (CSAP) (1994)

This plan was developed in order to manage chlorinated toxic substances that may impair the quality of the environment and human health. This program targets toxic chlorinated substances for virtual elimination or significant reduction from the environment. The action plan is divided in a five-part approach (Environment Canada and Health Canada 2000):

1. Numerous federal and national initiatives (*TSMP, CWS, ARET and the Federal/Provincial Task Force on Dioxins and Furans)* are now used to target and reduce the emissions of PCDD/Fs in the environment;

2. Scientific and technical research is being performed to improve PCDD/Fs knowledge on human health and the environmental effects;

3. Tolerance levels for specific compounds are being developed by Health Canada;

4. With the help of tools, such as the *NPRI*, Canadian citizens have access to up-to-date environmental information;

5. By participating in various conventions and programs around the world, Canada gathers new ideas and limits the duplication of scientific efforts.

7.1.4 Canadian Environmental Protection Act (1988, 1999)

CEPA is an act respecting pollution prevention and the protection of the environment and human health in order to contribute to sustainable development. It represents Canada's primary piece of environmental legislation, providing new tools to control the emissions of toxic substances towards their virtual elimination.

7.1.4.1 National Pulp and Paper Regulatory Package for the Canadian Pulp and Paper Industry

A National Pulp and Paper Regulatory Package for the Canadian Pulp and Paper Industry was put in place in 1992, addressing the problem of water contamination by dioxins and furans. Contrary to the Environment Effects Monitoring (EEM) program, under the *Fisheries Act*, biotic analyses and monitoring of dioxins and furans in the receiving environment are not required. PCDD/F analyses in the final effluents are the only requirement. According to the regulations, the effluent can not contain any measurable concentration of 2,3,7,8-TCDD/F, as defined in the *Reference Method for the Determination of Polychlorinated Dibenzo-para-dioxins (PCDDs) and Polychlorinated Dibenzofurans (PCDFs) in Pulp Mill Effluents* (Environment Canada 1992a). *The Defoamer and Wood Chip Regulations* stipulate that the use of defoamer is restricted to those containing less than 40 $\mu g \cdot L^{-1}$ or less by weight of dibenzofuran and 10 $\mu g \cdot L^{-1}$ or less by weight of dibenzo-*p*-dioxin. For mills using a chlorine bleaching process, wood chips made from wood treated with polychlorinated phenols are not permitted in the treatment process. Furthermore, the *Chlorinated Dioxins and Furans Regulations* require that all mills provide Environment Canada with the results of their analyses of dioxins and furans in their final effluent.

In addition, the *Federal Mobile PCB Treatment and Destruction Regulations* restrict the emission of a gas, liquid and solid that contains a concentration exceeding 12 $\mu g \cdot L^{-1}$, 0.6 $\mu g \cdot L^{-1}$ and 1 $\mu g \cdot kg^{-1}$, respectively, of 2,3,7,8-PCDD/Fs.

7.1.4.2 The National Pollutant Release Inventory (NPRI) (1992)

This initiative was established in 1992 to have an easier access to information regarding the releases of contaminants to air, water and land. As of 2000, 268 substances comprise the NPRI (Environment Canada 2002). Any person in Canada who owns or operates a facility is required, under certain conditions, to submit a report to *NPRI*, by June 1st of the following year, as mandated under *CEPA*. This database is the only legislated, nation-wide publicly accessible inventory of pollution for emissions and transfers in Canada.

7.1.4.3 Federal/Provincial Task Force on Dioxins and Furans (1995)

In January 1999, the Federal/Provincial Task Force on Dioxins and Furans, established by the Federal-Provincial Advisory Committee for *CEPA* (CEPA-FRAC), released the Dioxins & Furans & Hexachlorobenzene Inventory of Releases. The intent of this document is to give an understanding of the current anthropogenic sources spread across the country. Numerical information associated with dioxin and furan emissions are limited, and as a consequence, this survey needs frequent updates through the *NPRI*. An action plan, consistent with the objective of virtual elimination as per the *Toxic Substances Management Policy* (TSMP), is currently being developed.

7.1.5 Federal Water Policy (1987)

The goals of this Environment Canada policy are to anticipate and prevent the contamination of all Canadian waters by harmful substances and to encourage the restoration of the contaminated waters. By doing so, the “polluter pays” principle is promoted for an effective management of the water resource using different regulations, guidelines and codes of practice (Environment Canada 1987).

7.1.6 Fisheries Act (1985)

The 1985 *Fisheries Act* provides broad information for the protection of all fish habitat and is exclusively concerned with the point sources emissions of deleterious substances in the aquatic environment. When this Act was introduced, it was designed to address the basic concerns for coal ash and ballast (oil); dioxins and furans are not mentioned specifically in the text. Notwithstanding, Section 36 prohibits the release of any substance that could have a deleterious impact on fish and their habitat.

In May 1992, the federal government passed amendments to the *Pulp and Paper Effluent Regulations*. These included more stringent effluent limits and required every pulp and paper mill using chlorine bleaching in Canada to conduct an Environmental Effects Monitoring (EEM) program to analyse PCDD/Fs tissue concentrations in commercial and recreational fish in the receiving environment (Couillard and Nellis 1999), as opposed to the final effluent sampling under *CEPA*.

National Environmental Effects Monitoring Program: Dioxins and Furans Analysis

Background

As part of the 1992 *Pulp and Paper Effluent Regulations* (PPER) under the *Fisheries Act*, pulp and paper mills in Canada subject to the PPER are required to conduct Environmental Effects Monitoring (EEM) to determine if the mill effluent is having an effect on fish[4], fish habitat and the usability of fisheries resources (Environment Canada 1992b). EEM is an iterative monitoring program that is conducted as a three or four year sequence of monitoring phases called "Cycles". Regional Environment Canada officials review each site-specific study designs and interpretative reports that are submitted by the mills. The National EEM Office co-ordinates and manages the EEM program to ensure national consistency.

Cycle 1 was completed in April 1996. This was followed by a one year review period of the EEM program, which led to improvements and more site specific flexibility, including the use of "decision trees" to determine which monitoring would be required at each mill, dependent upon the previous results. Cycle 2 was completed in April 2000.

In order to determine the usability of fisheries resources in accordance with the *Fisheries Act*, a dioxin and furan analysis of fish tissue may be required as part of the EEM program (Environment Canada 1997b). By monitoring dioxins and furans in fish tissue, the information can be used to assess the effectiveness of the *Pulp and Paper Chlorinated Dioxins and Furans Regulations* implemented under *the Canadian Environmental Protection Act* (*CEPA*) (Environment Canada 1992b). Under *CEPA*, pulp and paper mill effluents are required to have "non-measurable" levels of dioxins[5], as defined by the Regulations.

Cycle 1

All mills in Canada that were using or had used chlorine bleaching in the past were required to conduct an analysis in fish tissue for PCDD/Fs in Cycle 1. Thirty-seven freshwater mills and thirteen marine mills conducted a fish tissue analysis in Cycle 1. Based on the Cycle 1 EEM data, some general conclusions with regards to the dioxins and furans monitoring could be made. Nationally, the mills discharging effluent to the freshwater environment generally showed low (<1 $pg \cdot g^{-1}$) to non-detectable levels of dioxins in fish tissue. In Atlantic Canada, low to non-detectable levels of dioxins were found, but no shellfish (lobster or crab) hepatopancreas were sampled. In British Columbia, marine bleached kraft mills found high (<1 to 2949 $pg \cdot g^{-1}$) dioxin levels in crab hepatopancreas and fish liver (Environment Canada 1997c).

Cycle 2

For Cycle 2, a decision tree was developed to allow a site-specific determination of whether a dioxins analysis should be conducted by a mill (Environment Canada 1997b; 1998e). The decision tree questions if levels of dioxins in the previous Cycle were a) below fish consumption guidelines, b) there was no consumption related fisheries advisory or closure based on dioxins contamination in the receiving environment of the mill and the mill has been in compliance with the *CEPA* regulations for dioxins in the preceding twelve months. In this situation, based on the outcome of the decision tree, a mill could be exempted from conducting a dioxins analysis in fish tissue for EEM.

Cycle 1 results found that in general, levels of dioxins were low, which resulted in the majority of mills in Canada being exempt from a dioxins analysis for Cycle 2. Only nine studies (with a total of ten mills) conducted a fish tissue analysis of dioxins in Cycle 2. All of the BC marine mills had levels of dioxins

[4] Fish: includes finfish and shellfish, as defined by the *Fisheries Act*.

[5] Dioxins: refers to all 2,3,7,8-substituted polychlorinated dibenzo-*p*-dioxins and dibenzofurans.

above the Health Canada guidelines for consumption[6] (range: non-detectable to 188.5 $pg \cdot g^{-1}$). The BC freshwater mills and the Ontario mill had levels of dioxins below the Health Canada consumption guidelines (range: 0.28 –1.32 $pg \cdot g^{-1}$).

Conclusions

The results of the dioxins fish tissue analysis as part of the EEM program showed improvement in levels of dioxins in Cycle 2, with only seven of 126 mills in Canada which are subject to the PPER having levels of dioxins above the Health Canada consumption guidelines. The levels of dioxins in the marine mills dropped from a maximum of 2949 $pg \cdot g^{-1}$ in Cycle 1 to 188.5 $pg \cdot g^{-1}$ in Cycle 2.

Currently, all of the marine mills in British Columbia, which had levels of dioxins exceeding the guidelines in Cycle 2, are also subject to the Fisheries and Oceans Canada and Environment Canada organochlorine trend-monitoring program. Data collected from this program can be used to fulfil the dioxins requirement for EEM. In many of these areas in BC, there are commercial closures and recreational and native fisheries advisories that continue to be in effect.

For Cycle 3, which will be completed in April 2004, the same decision tree that was used in Cycle 2 will be used to determine site specifically if a mill will conduct a dioxins analysis in fish tissue.

Guidance for Determining Follow-up Actions when Effects Have Been Identified in Environmental Effects Monitoring (EEM) (Environment Canada 2001c) was developed by Environment Canada, in consultation with Fisheries and Oceans Canada. It provides guidance to regulatory agencies, regulated facilities, and EEM practitioners for determining follow-up actions when effects have been identified in EEM. It discusses the factors that need to be considered for determining follow-up actions, and explains the roles and responsibilities of government, industry, and stakeholders in this process.

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7.2 National Program and Initiatives

7.2.1 Canadian Environmental Quality Guidelines

Canadian Environmental Quality Guidelines are nationally endorsed science-based goals for the quality of atmospheric, aquatic, and terrestrial ecosystems. CEQGs are mandated under the *Canadian Environmental Protection Act*, 1999, Part 3, Section 54(1)(b) which states that "the Minister shall issue environmental quality guidelines specifying recommendations in quantitative or qualitative terms to support and maintain particular uses of the environment". They are developed under the auspices of the Canadian Council of Ministers of the Environment (CCME). CEQGs are defined functionally as numerical concentrations or narrative statements that are recommended as levels that should result in negligible risk to biota, their functions, or any interactions that are integral to sustaining the health of ecosystems and the designated resource they support.

Canadian environmental quality guidelines for PCDD/Fs include Canadian sediment quality guidelines for the protection of aquatic life and Canadian tissue residue guidelines for the protection of wildlife consumers of aquatic biota, and Canadian soil quality guidelines for the protection of environmental health (Table 7.2). They were derived according to formal scientific

[6] 30 $pg \cdot g^{-1}$ in crab and lobster hepatopancreas, 15 $pg \cdot g^{-1}$ in fish liver.

protocols (CCME 1995; 1996; 1998) on a toxic equivalency basis (TEQ). Although a protocol exists for the development of Canadian water quality guidelines, such guidelines were not derived for PCDD/Fs. The Canadian Council of Ministers of the Environment recognises that for highly persistent, bioaccumulative substances such as PCDD/Fs, water quality guidelines have a high level of scientific uncertainty and are of limited practical management value, and are therefore, no longer recommended (CCME 1999). For a detailed explanation of the process used to derive the Canadian Environmental Quality Guidelines for dioxins and furans, please consult Environment Canada (2000b).

Table 7.2. Sediment, Tissue Residue, and Soil Quality Guidelines for PCDD/Fs.

	Interim Sediment Quality Guidelines (ng TEQ·kg^{-1}) dw	Tissue Residue Guidelines (ng TEQ·kg^{-1} diet ww)	Soil Quality Guidelines (ng TEQ·kg^{-1})
Freshwater	0.85		
Marine	0.85		
Mammalian		0.71	
Avian		4.75	
Agricultural			4
Residential / Parkland			4
Commercial			4
Industrial			4

(Sources: CCME 2000a, 2000b and 2001b)

7.2.2 Canada-Wide Standards for Dioxins and Furans (2001)

The *Canada-Wide Standards for Dioxins and Furans* are the only standards that address substances that are designated for virtual elimination under *CEPA* (CCME 2001a).

These standards focus on anthropogenic sources that release dioxins and furans to the atmosphere and soil on a continual basis. Priorities for early action include: waste incineration (municipal solid waste, hazardous waste, sewage sludge and medical waste); burning salt-laden wood in coastal pulp and paper boilers (British Columbia); residential wood combustion; iron sintering; electric arc furnace steel manufacturing; and, conical municipal waste combustion (Newfoundland) (CCME 2001a; Canada Gazette 2002a, b). The ultimate goal of this strategy is to achieve a combined emission reduction for municipal waste incineration and coastal pulp and paper boilers of at least 80% by the year 2006 through the application of pollution prevention strategies and the setting of emission limits for all facilities (CCME 2001a). Existing and new facilities located in Canada agreed to meet the following standards (in I-TEQ units; Canada Gazette 2000):

Municipal Waste Incineration, Medical Waste Incineration and Hazardous Waste Incineration: < 80 pg·m^{-3};

Sewage Sludge Incineration

- < 80 pg·m^{-3} (new facilities, by the year 2005)
- < 100 pg·m^{-3} (old facilities, by the year 2005);

Pulp and Paper Boilers burning Salt Laden Wood

- < 100 pg·m^{-3} for new boilers

- < 500 pg·m^{-3} for all existing boilers by 2006

Furthermore, federal authorities must support international actions with respect to PCDD/Fs, as well as maintain the National Pollutants Release Inventory (NPRI) up to date in order to monitor the progress status or modify the pollution prevention strategy if the results are insufficient. A final report due in 2008 will evaluate whether the targets have been met (CCME 2001a).

Canada-Wide Standards for PCDD/Fs have recently been adopted for iron-ore sintering plants, and steel manufacturing electric arc furnaces (Canada Gazette 2002a, b). The sintering of iron-bearing steel mill secondary materials and ore is unique to Ontario. Currently, there is only one remaining sintering plant in Canada (Canada Gazette 2002a). The Iron Sintering Plant CWS ultimate objective is to reduce the PCDD/F concentrations in the exhaust to below the level of quantification (LOQ) set at 32 pg TEQ·Rm^{-3}, without any correction for oxygen content. Furthermore, it proposed the reduction of stack emissions to a concentration less than 200 pg·m^{-3} for the new iron sintering facilities. For existing iron sintering plants, a three-phase plan is proposed:

- Phase 1: stack emissions shall be less than 1350 pg TEQ·m^{-3} by 2002 (representing a 50% reduction from 1998 levels);

- Phase 2: stack emissions shall be less than 500 pg·m^{-3} for all existing iron sintering plants by the year 2005 (representing a 80% reduction from 1998 levels);

- Phase 3: stack emissions shall be less than 200 pg·m^{-3} for all existing iron sintering plants by 2010 (representing a 90% reduction from 1998 levels)

(Canada Gazette 2002a).

The Canada-Wide Standard for Dioxins and Furans from Steel Manufacturing EAF proposes that emissions shall be less than 100 pg I-TEQ·m^{-3} for new or modified steel manufacturing EAF (Canada Gazette 2002b). For existing furnaces, a two-phase model is proposed:

- Phase 1: emissions shall be less than 150 pg I-TEQ·m^{-3} at all plants by 2006;

- Phase 2: emissions shall be less than 100 pg I-TEQ·m^{-3} at all plants by 2010.

The Canada-Wide Standards for other sectors, such as small sources and releases to soil, should be completed by December 31st 2001 (Canada Gazette 2002b).

7.2.3 CCME Guidelines and Codes of Practice

Numerous programs and initiatives were undertaken in collaboration with provincial and federal governments in order to achieve virtual elimination of toxic substances impairing the Canadian environment. Accordingly, dioxins and furans are addressed by the CCME, in a series of guidelines, to limit PCDD/F emissions from various sectors.

7.2.3.1 Code of Practice for Used Oil Management in Canada

The Canadian Council of Resource and Environment Ministers (CCREM) recommends a source emission limit for municipal solid waste incinerators of 0.5 ng·m^{-3} for total PCDD/Fs, based on a capability of best available emission control technology of 0 to 24 ng·m^{-3} (CCME 1989a).

7.2.3.2 Operating and Emission Guidelines for Municipal Solid Waste Incinerators

The objective if this guideline was to consider PCDD/Fs as toxic contaminants and limit their emissions to the atmosphere. The tetra- penta- hexa- hepta and octa PCDD/Fs were considered in the stack discharge limit of 0.5 ng I-TEQ·m^{-3} (CCME 1989b).

7.2.3.3 Guidelines for Mobile Polychlorinated Biphenyl Destruction Systems and Guidelines for the Mobile Polychlorinated Biphenyl Treatment systems

The technical purpose of these guidelines is to limit the release of PCB and other compounds from the destruction process. It is recommended that solid process residues have a PCDD/F TEQ concentration of less than 1 μg·kg^{-1} of waste to be environmentally friendly. It is also recommended that the maximum concentration of TEQ in aqueous waste should not exceed 0.6 ng·L^{-1}. In addition, PCDD/Fs should be determined in all waste streams to prevent their introduction into the environment (CCME 1990a; 1990b).

7.2.3.4 National Guidelines for the Landfilling of Hazardous Waste

These guidelines addressed the various strategies put forward in dealing with hazardous wastes for the protection of the environment. Limitations regarding landfilling, dilution, treatment (*e.g.*, detoxification) and appropriate management methods can be indirectly applied to PCDD/Fs (CCME 1991).

7.2.3.5 Guidelines for the Management of Biomedical Waste in Canada

These guidelines recommend a stack discharge limit for new incinerators, with a charging capacity exceeding 200 kg·h^{-1}, of 0.5 ng TEQ·m^{-3} for total PCDD/Fs at 11% O_2 (CCME 1992a).

7.2.3.6 National Guidelines for Hazardous Waste Incineration Facilities

These guidelines provide guidance on the design and operation of facilities burning hazardous wastes to minimise the emission of toxic contaminants into the environment. The incinerators must not emit more than 0.5 ng TEQ·m^{-3} of dioxins and furans in the atmosphere (CCME 1992b and 1992c).

7.2.3.7 National Guidelines for the Use of Hazardous and Non-Hazardous Wastes as Supplementary Fuels in Cement Kilns

The use of wastes as fuels in cement kilns has the potential to significantly reduce the cost, conserve fossil fuel resources and provide disposal capacity for wastes. The proposed

emission limits for dioxins and furans at existing plants (prior to 1995) is 0.5 ng TEQ·m^{-3}, and for new plants; 0.1 ng TEQ·m^{-3} (CCME 1996a, b).

7.3 Selected Provincial and Territorial Initiatives

Provincial jurisdictions regulate the concentration of dioxins and furans through general legislation such as the *Environmental Protection and Enhancement Act* in Alberta; the *Environmental Management Act* in British Columbia, the *Environment Act* in Manitoba, Nova Scotia, Yukon Territory and Newfoundland; the *Clean Environment Act* in New-Brunswick; the *Environmental Protection Act* in Nunavut, Ontario and Prince Edward Island; the *Environmental Quality Act* in Québec and finally the *Environmental Management and Protection Act* in Saskatchewan. Dioxins and furans, although not addressed directly in any of these Acts, are covered indirectly through general statements referring to the alteration of the quality of the environment by toxic substances (dioxins and furans are toxic under *CEPA*) are given. Control measures, addressing PCDD/Fs specifically, can be found in Table 7.1.

7.3.1 *British Columbia*

The proximity of the natural resources (wood and water) is associated with the flourishing development of the pulp and paper sector in the province of British Columbia. In 1990, the *Pulp Mill and Paper Mill Liquid Effluent Control* was established in order to prohibit the discharge of Halogenated Organic Compounds (AOX) in the aquatic environment, indicators of PCDD/F contamination. According to the geographical location of certain mills, specific effluent discharge limits are put in place to minimise the impacts of toxic substances on sensitive ecosystems. The *Port Alberni Pulp and Paper Effluent Regulations* prescribed certain deleterious substances in relation to the MacMillan Bloedel pulp and paper mill until 1993. Furthermore, the *Emission Criteria for Biomedical Waste Incinerators*, prepared by the Municipal Solid and Biomedical Waste Branch of BC government, were developed in June 1991. These guidelines set the limits for various contaminant emissions into the atmosphere. Total emissions of PCDD/F are set at 0.5 ng TEQ·m^{-3} (Glenn 1999).

British Columbia treats dioxins as special wastes under the *Special Waste Regulation.* Wastes containing dioxins at a concentration greater than 100 μg TEQ·g^{-1} by weight are classified as special waste and subject to regulatory provisions regarding their storage, transport, treatment and disposal.

Considering the variety of sources of dioxins and furans in British Columbia, standards for soil protection were developed according to the *Contaminated Sites Regulations* under the *Waste Management Act*. Five different land uses were considered for setting the standards for human health and environmental protection. Intake of contaminated soil was set at 0.35 ng TEQ·g^{-1} for agricultural, urban park and residential land uses and at 1 ng TEQ·g^{-1} for commercial land use for the protection of the human health. There was no standard developed for industrial land use for the protection of the human health. Toxicity to soil invertebrates and plants was set to 0.01 ng TEQ·g^{-1} for agricultural land use, 1 ng TEQ·g^{-1} for urban park and residential uses and 2.5 ng TEQ·g^{-1} for commercial and industrial land uses. These numbers were set equal to the interim Canadian soil quality guideline because of a lack of acceptable environmental data. Furthermore, soil relocation to a different site is not required if the PCDD/F concentration in the soil is less than 350 ng TEQ·g^{-1} for non-agricultural land. For agricultural land use, soil relocation is not necessary if the PCDD/F concentration in soil is less than 0.01 ng·g^{-1}. The waste disposal without authorisation is prohibited if the PCDD/F concentrations are greater than 2.5 ng TEQ·g^{-1}.

The protection of the aquatic environment is indirectly covered by these regulations, considering that surface water and groundwater should not contain concentrations of PCDD/F greater or equal to the standards for human health and environmental protection, stated above.

7.3.2 *Ontario*

The Ontario government revealed, in December 2001, a comprehensive hazardous waste plan regulating the hospital medical waste incinerators sector and the destruction of stored PCB wastes. The plan contains three draft regulations. The first part would phase out existing hospital incinerators, sources of dioxins and furans emissions, within one year after the proposed regulation takes effect. The second regulation would set the requirements for the transportation and treatment of medical wastes. The third regulation concerns the destruction of about 100,000 tons of PCB wastes stored at 1,000 sites across the province (Waste News 2001).

This province has elaborated a series of regulations to control the emissions of dioxins and furans from a variety of sources. The *Effluent Monitoring and Effluent Limits - Pulp and Paper Sector, - Petroleum Sector, - Organic Chemical Manufacturing Sector* and- *Inorganic Chemical Sector* were established under the *Environmental Protection Act* and came into force in 1998. The discharge of contaminants into the environment is addressed under Part IV of the regulation "Parameter and Lethality Limits". The quality of each processed effluent monitoring stream must meet non-measurable concentrations of 2,3,7,8-TCDD/F set at 20 $pg \cdot L^{-1}$ and 50 $pg \cdot L^{-1}$, respectively. Moreover, each discharger must ensure that the concentration of 2,3,7,8- substituted dioxin and furan congeners in any sample is less than 60 pg $TEQ \cdot L^{-1}$ at any effluent sampling point. Another goal of the Ontario Ministry of the Environment is to eliminate the generation of AOX (indicators of dioxin contamination) at discharger plants by the year 2002.

Moreover, the Ministry of the Environment and Energy developed the *Guidelines for Use at Contaminated Sites in Ontario* in 1997 (OMEE 1997), in replacement of the *Guidelines for the Decommissioning and Clean-up of Sites in Ontario* (1989) and the *Interim Guidelines for the Assessment and Management of Petroleum Contaminated Sites in Ontario* (1993). These guidelines are to be used when property owners are cleaning or redeveloping contaminated sites as they give guidance and information regarding environmental conditions of a site and if restoration is required. Site restoration can be evaluated using three different approaches:

A) Use the soil quality criteria to restore the site at background concentration for a specific contaminant;

B) Use the soil and groundwater quality criteria to provide protection against the potential for adverse effects to human and ecological health and the natural environment (Table 7.3.). Criteria may be derived for three different land uses (agricultural, residential/parkland and industrial/commercial), for potable groundwater use to protect the quality of the drinking water, and for nonpotable groundwater use to protect aquatic life in receiving surface waters and against groundwater vapours.

C) Site specific risk assessment (SSRA): Use to estimate the contaminant exposure risk posed to humans, plants, wildlife and the natural environment, from two different depths (above and below 1.5m) and two different soil textures.

Table 7.3. PCDD/F Guidelines for Use at Contaminated Sites in Ontario

	Agricultural land use	Residential / parkland land use	Industrial / commercial land use
Soil remediation criteria[a] in a potable groundwater situation (ng TEQ·g^{-1})	0.01	1.0	1.0
Soil remediation criteria[a] in a nonpotable groundwater situation (ng TEQ·g^{-1})		1.0	1.0
Potable groundwater remediation criteria (μg·L^{-1})	0.000015	0.000015	0.000015
Nonpotable groundwater remediation criteria (μg·L^{-1})	0.000015	0.000015	0.000015
Subsurface soil remediation criteria[b] in a potable groundwater situation (ng TEQ·g^{-1})		1.0	No value
Subsurface soil remediation criteria[b] in a nonpotable groundwater situation (ng TEQ·g^{-1})		1.0	No value
Soil background concentration (ng TEQ·g^{-1})	0.007	0.007	0.007

[a] Soil criteria for inorganics apply only where surface soil pH is 5.0 to 9.0.
[b] Soil criteria for inorganics apply only where soil pH is 5.0 to 11.0.

(modified from OMEE 1997)

7.3.3 *Yukon*

Under the *Contaminated Sites Regulations* issued for the Yukon Territory, PCDD/F soil standards (Table 7.4) were derived in order to prevent toxicity to soil invertebrates and plants. Different limits were derived to offer an environmental protection towards site-specific land uses:

Table 7.4. Soils standards in the Yukon

Land use	Agricultural	Park	Residential	Commercial	Industrial
standard (ng·g^{-1})	0.01	1	1	2.5	2.5

7.4 International control measures for dioxins and furans - Canadian implications

Some control measures and programs are elaborated with the participation of a large number of countries in order to concentrate the efforts to eliminate possible duplication of scientific research.

7.4.1 Stockholm Convention (2001)

Recently, Canada was the first country to sign and ratify the Stockholm Convention on persistent organic pollutants for the protection of human and environmental health. The objective of this protocol was to promote the exchange of information between parties in order to reduce or eliminate releases from unintentional production of twelve toxic POPs, including dioxins and furans (UNEP 2001). Different options addressed by the convention were: an evaluation of current and projected releases; efficacy of the laws and policies; promotion of education and training and the promotion of the available practical measures for source elimination. Canada became the first country to make a specific funding commitment of $20M

to help countries find alternatives to the use of POPs. The federal government is also considering the development of regional action plans for the reduction of dioxins and furans in the environment (Envirozine 2001).

7.4.2 North Atlantic Treaty Organisation on the Challenges of Modern Society (NATO-CCMS)

In 1988, the first international scientific meeting introducing an inventory of regulations and statutes on dioxins and furans took place. Canada, Denmark, the Federal Republic of Germany, Italy, the Netherlands, Norway, the United Kingdom and the United States provided their current legislation concerning these substances in various sectors such as air quality emissions, food, water, hazardous waste & disposal, pesticides and the manufacture of chemicals (Bottimore et al. 1988). At that time, Canada had only ten regulations addressing dioxins and furans, mainly on the issues of pesticides. Of these, five were provincial (Ontario) and mostly related to water issues (Bottimore et al. 1988). This information exchange project was the precursor to the adoption of the international toxicity equivalency factor (I-TEF), a mode of conversion for mixtures of dioxins relative to the most toxic congener for comparison among congeners.

Great Lakes Water Quality Agreement: Achievements and Future Goals

Approximately 1/5 of the world's supply of freshwater is found in the Great Lakes Basin, home to millions of Canadians and Americans (BEC 1997). During the early 1970s, pollution in the Great Lakes became obvious; the Great Lakes were seriously damaged by the presence of chemicals and other pollutants, resulting in a high concentration of algae and a decrease in fish and wildlife populations (BEC 1997). In order to manage and protect waters from pollution along this Canada - United States border, these two countries created, the International Joint Commission (IJC) in 1909, a co-operative program assisting the governmental organisations in finding solutions to problems in these waters (Battelle 2000a). The *Great Lakes Water Quality Agreement* was created in 1972 to restore the health of the ecosystem. In 1978, the end of discharges of toxic substances, that persist in the environment, including dioxins and furans, were targeted by this expanded agreement. Furthermore, in 1987, *Remedial Action Plans (RAPs) for Great Lakes Areas of Concern (AOCs)* were put into place under the *GLWQA*. *RAP*s required the cleanup (runoff, airborne, groundwater, etc.) of 43 areas of concern in the Basin (12 in the Canadian Great Lakes and 5 shared with the US), in order to restore the beneficial uses and ecosystem integrity (Battelle 2000a).

Many federal programs were developed by the two governments under the terms of *GLWQA* to meet the objectives of restoring and protecting the Great Lakes. One of them was the *Integrated Atmospheric Deposition Network* (IADN), focusing on the identification of airborne toxic substances and their sources, their movements and the different trends in atmospheric deposition in the Great Lakes and was developed under the Canada-U.S. Great Lakes Water Quality Agreement. In 1994, the *Canada-Ontario Agreement Respecting the Great Lakes Basin Ecosystem* (COA) was established to ensure the implementation of the RAPs, recommended in the 1987 GLWQA by the year 2000 (Battelle 2000a). Over the past decade, significant reductions of persistent bioaccumulative substances were achieved; PCDD/Fs were reduced by more than 75% in the Great Lakes region (Governments of Canada and Ontario 2001). Furthermore, a 90% reduction in the release of PCDD/Fs is anticipated by 2005, compared to releases of 1998. Several policies and programs, in co-operation with the COA, such as CWS and performance review of waste incinerators in the Ontario province, are making progress towards virtual elimination of PCDD/Fs. The *Canada-Ontario Agreement Toxic Substances Emission Inventory* (1998) provides information gathered in the *Air Pollutants Emissions Inventory for Criteria Air Contaminants* (CAC - 1997) and in the *1997 Priority Substances Inventory* regarding the potential sources of PCDD/F emissions such as iron steel, thermal power generation and wood preservation industries.

In 1989, the IJC recommended that Lake Superior be designated as a demonstration area where discharges and emissions of toxic substances would not be permitted. In response, Canada and the United States developed a *Binational Program to Restore and Protect the Lake Superior Basin* (1991) through activities such as pollution prevention, enhanced regulatory measures and the development of a Lake Superior Binational Forum with the participation of citizens and stakeholders. Furthermore, under the GLWQA, both governments took the initiative to develop *Lakewide Management Plans* (LaMP) for each of the five Great Lakes (Battelle 2000a). These plans target the protection, preservation and the restoration of water quality and beneficial uses of the lakes, with an 80% reduction of 2,3,7,8-TCDD by 2005 in Lake Superior. Lake Huron, however, does not possess a lakewide management planning process. Instead, a *Lake Huron Initiative Action Plan* was developed, aiming for the identification of immediate and future efforts to reduce or eliminate the concentration of critical pollutants in the lake and the protection of the biodiversity.

Finally, in 1997, the *Great Lakes Binational Toxics Strategy* (GLBTS) - *Canada / United States Strategy for the Virtual Elimination of Persistent Toxic Substances in the Great Lakes* was created. This strategy was intended to encourage ongoing programs or emerging initiatives to better address toxic releases, taking into account the entire life-cycle of the chemicals, and to provide assistance to specific activities such as LaMPs, RAPs and domestic program processes. The Canadian challenge was to achieve a 90% reduction (Ontario) in 2,3,7,8-TCDD/F levels by 2000, keeping in mind that virtual elimination was based on 1988 substance levels (BEC 1997).

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7.5 Efficacy of the Canadian legislation to control source emissions of PCDD/Fs

Canadian industries responded to PCDD/F concerns by reducing or eliminating their emissions in the Canadian environment. Many studies have been performed in order to determine the efficacy of the regulations described previously.

7.5.1 Pulp and Paper Sector

Bleached chemical pulp production was first implicated as a potential source of polychlorinated dioxins and furans in the fall of 1985 (Luthe 1998). Knowing that small concentrations of these highly toxic substances may impair the health of aquatic organisms (*e.g.*, deformities or death), the federal government prescribed the collection of data regarding the releases associated with pulp and paper mills from 1987 to 1989. A federal trend-monitoring program between the year 1990 and 1995 was performed following data collection (Hagen et al. 1997) to look at average percent decline of PCDD/Fs in a variety of aquatic species. The results were encouraging: a decrease of 61% in sediments, 85% in crab muscle, 80% in crab hepatopancreas, 93% in oyster, 92% in prawn and 87% in shrimp were observed (Hagen et al. 1997). At other locations, however, contamination of the BC aquatic environment by dioxins and furans resulted in shellfish harvesting restrictions covering up to 1200 km^2 between 1988 and 1995 (Weber et al. 1997).

A series of surveys carried out annually prior to and after the enforcement of the *National Pulp and Paper Regulatory Package for the Canadian Pulp and Paper Industry* under *CEPA* in 1992, indicated that effluent discharges of dioxins from the bleached chemical pulping sector have been reduced to approximately 2 $g \cdot a^{-1}$. This concentration represents a near 99% reduction of PCDD/F between 1988 and 1994 (Luthe et al. 1994; Luthe 1998; Hiester et al. 1997). Emission problems appeared to be closely linked to several power boilers from coastal mills burning salt-laden wood waste. The ash associated with these practices is a likely source of the site-specific PCDD/Fs contamination in the final effluent (Luthe et al. 1994).

In 1993, the mills in Miramichi Estuary, New Brunswick, partially substituted chlorine dioxide in order to reduce the 2,3,7,8-TCDD concentrations in the final effluents from 0.1 $ng \cdot L^{-1}$ in 1988, to 0.005 $ng \cdot L^{-1}$ in 1995. Also, a reduction from 1 to 0.013 $ng \cdot L^{-1}$ for 2,3,7,8-TCDF was achieved for the same time period (Couillard and Nellis 1999). Furthermore, Kamloops Lake, situated in the Fraser River system, BC, showed minor sediment contamination in the 1930s-1950s, followed by large quantities of H_6CDD and TCDD/Fs in 1965, year of the mill construction. Levels of PCDD/Fs were drastically reduced in the 1990s, as a result of the implementation of federal regulations (Macdonald et al. 1998; see chapter 5.0.).

Overall, releases from the pulp and paper sector have been reduced to below the level prescribed in the legislation (Glenn 1999). Moreover, discharges of dioxins and furans to the aquatic environment reached non-measurable levels in 1995 (Canada Gazette 2000) and have been virtually eliminated from the industry's discharges where chlorine gas has been removed (Owens 1996). In 2001, all chemical pulp mills in Canada have successfully implemented the process to reduce or prevent the formation of dioxins. Furthermore, the chlorine substitution (Cl) for chlorine dioxide (ClO_2) as a bleaching agent seemed to have eliminated the production of TCDD/Fs (Macdonald et al. 1998).

7.5.2 Impacts on the environment

The Great Lakes Water Quality Board has identified eleven critical pollutants that present risks to human health and the aquatic ecosystem; the halogenated aromatic hydrocarbons TCDDs and TCDFs are included in that list (IJC 2001). In the 1970s and early 1980s, the identification of pollution point sources helped to reduce inputs of TCDDs in Lake Ontario and into Saginaw Bay, Michigan (Grasman et al. 1998). Biomonitoring during the 1980s and 1990s indicated the potential of PCDD/Fs to persist in the environment at highly contaminated sites (Ludwig et al. 1996; Grasman et al. 1998). Accordingly, there were no significant temporal trends in birds for 2,3,7,8-TCDD/F between 1991 and 1994 downstream of the Castlegar (BC) pulp mill, despite changes in bleaching technology in 1993 (Elliott et al. 1998a; see chapter 5.0.). By 1997, concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF in birds were already significantly lower than previous years in nests sampled downstream of Castlegar and Kamloops (BC) (Elliott et al. 1998a). Bird populations have recovered on the regional scale since the implementation of the legislation concerning many organochlorine compounds and the use of alternative chemicals; however, the presence of these contaminants in the environment still continues to cause reproductive and physiological effects to some species.

7.5.3 Atmospheric releases

According to the Federal/Provincial Task Force on Dioxins and Furans, changes in combustion technology and filtration of the particulate phase were responsible for an 18% reduction, from all sources combined, in atmospheric releases between 1990 and 1997 (Glenn 1999). A 90%

reduction was achieved through adjustments, such as temperature increases (Luthe et al. 1996). An additional 25% reduction in releases of 200 g TEQ·a^{-1} was expected in 1999.

7.5.4 Soil releases

Few control measures directly apply to soil releases of dioxins and furans. This may be related to the ban of certain pesticides in recent years. The application of sewage sludge to agricultural land, use of pesticides, and the use of treated wood have not encountered drastic changes in the past years. No changes in soil releases were therefore observed between 1990 and 1999 (Glenn 1999).

7.6 Best Management Practices - International

Knowledge of the aquatic toxicity of dioxins and furans was the starting point for the development of best management practices to reduce the formation of these substances. Many strategies exist to minimise the formation of dioxins and furans at the source, especially in the pulp and paper mill and incineration industries. The use of a hog fuel dryer, capable of recycling flue gas dioxins back to the lower furnace for destruction represents one option (Luthe et al. 1996). A polymer-induced settling of ash in clarifiers, prior to discharge, could decrease the amount of dioxins released to the atmosphere (Luthe et al. 1994). The reduction of the chlorine: sulphur ratio is an encouraging method because the chlorine concentration is directly related to the amount of dioxin emissions (Luthe et al. 1997). The installation of an electrostatic precipitator and of a high stack to push furnace gases above inversion layers could reduce the amount of PCDD/Fs found in the aquatic environment (Macdonald et al. 1998).

In Canada, wood stoves are a high priority sector, with previous and ongoing activities including workshops, educational campaigns, a pilot change out program in eastern Ontario in early 1999 (Table 7.1) and a National point-of-purchase campaign (Battelle 2000b). However, there is currently no method to continuously monitor the PCDD/F emissions to the atmosphere. USEPA's strategy is to place limits on CO concentrations, steam load, particulate matter control device operating temperature and opacity (Kilgroe 1996). The first three parameters concern the partitioning of PCDD/Fs. Opacity ensures the proper operation of the particulate matter control device in collecting solid phase PCDD/Fs (Kilgroe 1996). Regulatory agencies encourage voluntary efforts such as upgrading systems, but these efforts should be considered minimum because there are no incentives presently in place.

More efficient combustion processes could greatly reduce the formation of dioxins and furans. Combustion technology should ensure adequate waste-burn-out and therefore, produce minimal products of incomplete combustion (Kilgroe 1996; Luthe and Prahacs 1993). PCDD/F formation is temperature dependent: minimising the retention time at 200-450^{o}C may be effective since their formation occurs predominantly at low temperatures (170-450^{o}C) (Luthe et al. 1996). Thus, by increasing the temperature of combustion and by pulverising the solid fuels in suspension burners, virtually complete combustion will lead to non-detectable dioxins emissions (Luthe and Prahacs 1993, 1996). Also, a secondary treatment (physical or chemical) of the effluent before discharge should help to reduce the concentrations of dioxins and furans entering the aquatic environment.

Dioxins tend to bind strongly to soil and particulate. If contaminated water were passed through soil, dioxins in water would be reduced to non-detectable levels (Luthe et al. 1997). Furthermore, land filling of the ash represents another management practice because dioxins will be associated with particulate and show lesser toxicity (Luthe et al. 1996).

8. EMERGING ISSUES

In recent years, a number of specific issues related to PCDD/F levels and their environmental health consequences have arisen. These range from concentrations of PCDD/Fs in waste sediments at pulp and paper mills to new waste incineration technologies and bioassay advances. This chapter explores the scientific underpinnings and management considerations for these emerging issues.

8.1 Recent Advancements in Waste Incineration Technologies

Waste incineration is a popular technology used to dispose of large quantities of municipal, industrial and medical wastes. It offers a relatively inexpensive treatment method, with the opportunity for further energy recovery, and in densely populated areas with little available landfill space, it is often the primary waste treatment option. For example, over 70% of waste (by mass) in Japan is incinerated (Kobylecki et al. 2001). As incomplete combustion can lead to the accumulation of dioxin-forming compounds in flue gases (see Section 3.2.1), PCDD/F compounds are readily released from waste incinerators, representing the largest source of emissions to the Canadian environment (Environment Canada 2001a). The complex pathways for dioxin-forming precursor compounds such as chlorophenols and chlorobenzenes during incineration at municipal waste incinerators severely limits the ability to predict dioxin congener patterns from these facilities (Iino et al. 2001).

Fluidized bed combustion (FBC) incinerators offer the advantage of operating at lower temperatures (*e.g.*, 800-950°C), and are being increasingly used for the disposal of municipal wastes (Anthony et al. 2001). Gullett et al. (2000) has shown that despite the lower operating temperatures in these incinerators and the presence of chlorine precursors and metal catalysts (which are required to initiate PCDD/F formation), the co-firing of refuse derived fuel (RDF) with a sulphur source (i.e., coal) can reduce the amount of PCDD/Fs formed during incineration. Sulphur dioxide is thought to interfere with the metal catalysts for PCDD/F formation in the fly ash, and/or it may reduce Cl_2 to HCl via the Deacon reaction, thereby limiting the reactivity with chlorine atoms (Gullett et al. 2000). In a study conducted by Natural Resources Canada and Environment Canada, the addition of low levels of SO_2 (i.e., Cl/S ratio of ~1) to pelletized wood and PVC feed stock in a FBC incinerator produced total dioxin and furan levels of 31.0 pg TEQ·m^{-3} (based in I-TEFs), which is less than half the amount seen in control stock without SO_2 (66.3 and 77.2 pg TEQ·m^{-3}); however, adding high levels of SO_2 (i.e., Cl/S ratio of ~0.5) doubled the amount of PCDD/Fs released, to 114.7 pg TEQ·m^{-3} (Anthony et al. 2001). Anthony et al. (2001) also cautions that PAH emissions increased by almost 100%, even under low SO_2 additions, which may offset realized environmental benefits from reduced PCDD/F levels.

Recent combustion tests with pellets formed from municipal waste incinerator fly ash have demonstrated that re-burning the pelletized ash under high temperatures (700ºC) for 1800 s can reduce residual dioxin concentrations of 862 ng TEQ·kg^{-1} (based on I-TEFs) in the ash by more than 99.7% (Kobylecki et al. 2001). In these tests, less than 3% of the dioxins originally present in the pellets were present in the escaping flue gases, resulting in ~97% of the PCDD/Fs being completely pyrolized (Kobylecki et al. 2001). Although increasing the reaction temperature to 900ºC allows a shorter burn time of 180 s (to provide over 99% PCDD/F decomposition), reactions at this temperature are not recommended, as heavy metal and chlorine retention efficiency would be compromised (Kobylecki et al. 2001).

Dioxin emission reduction policies for municipal waste incinerators have recently been implemented in Japan in an effort to meet 1999 World Health Organization tolerable daily intake (TDI) levels of 4 pg TEQ·kg body wt^{-1} (based on WHO-TEFs) (Kishimoto et al. 2001). Emission standards range between 0.1 and 5 ng TEQ·m^{-3}, depending on the age, and type of incinerator (Kishimoto et al. 2001). At a projected reduction of 1910 g TEQ·a^{-1} (representing a 44% reduction over 1996 baseline levels), the estimated average cost to reach these targets for the existing 1655 Japanese plants is ¥18.6 million per g TEQ reduced (i.e., approximately $220 000 CAN; based on March, 2002 exchange rates) (Kishimoto et al. 2001).

8.2 PCDD/F treatment strategies for waste sediments in the pulp and paper industry

Wastewaters from the pulp and paper industry are commonly treated in aerated stabilization basins (ASBs) and/or sludge lagoons prior to their release to receiving waters (NCASI 1999). Although sediments recently deposited in these basins (i.e., those deposited after the mid-1980s), generally contain very low levels of PCDD/Fs because of improved bleaching technologies, historical sludges can contain high levels of organochlorine (*e.g.*, > 50 parts per trillion), and therefore require remediation. As the usable lifetimes of ASBs are limited by sediment accumulation, pulp mills face the challenge of removing, dewatering, storing and ultimately disposing of sludges that may be contaminated with PCDD/F compounds (NCASI 1999). Some of the more feasible options for ASB sludges include solidification/stabilization, supercritical water oxidation, dehalogenation, controlled solid phase biological treatment, and photolysis/photodegradation (NCASI 1999):

Solidification and stabilization (S/S) technologies have been used for a number of years, commonly on an *in situ* basis, and are most appropriate for sludges already landfilled. In this process, the PCDD/Fs are either physically bound or enclosed within the stabilized sludge matrix (generally using portland cement as the solidification material). This technique contains the PCDD/Fs on site without first destroying them. In the supercritical water oxidation process, the contaminated sludge, water and an oxidant are combined in a reactor, where PCDD/Fs are made soluble, and subsequently oxidized to less harmful constituents. Depending on the composition of residuals, further treatment may be required prior to disposal. Although pilot studies on a similar process by Kimberly-Clark resulted in TCDD destruction efficiencies in the range of 95%, these were done using approximately 20 L of material only. Dehalogenation is a base-catalyzed decomposition process, in which sodium bicarbonate is added to crushed soils and heated above 330°C to remove chlorine groups and partially volatilize the contaminants. These processes have been successfully used to remediate PCB contaminated soils, and have been shown to achieve PCDD/F destruction efficiencies in excess of 99% for PCDD/F levels in the 2000 ppb range. Controlled solid phase biological treatment uses microbial inocula (i.e., white rot fungi), to biodegrade chlorinated organic compounds. This is a commercialized process using prescribed nutrient and microbe formulations under environmentally optimized conditions. As volatile organic compounds (VOCs) can result from the breakdown process, air escaping the composting sludge piles (which can reach ~7 m in height) may be collected for VOC destruction prior to atmospheric release. The use of white rot fungi to degrade TCDD is in the early stages of development, but solid phase biological treatment systems have been used to reduce selected chlorinated organic compounds in the 1000 ppm range by up to 97%. Photolytic processes use ultraviolet radiation either from the sun, or artificial sources to rapidly degrade the TCDD molecule. A major advantage to this technique, is that ASB sludges containing PCDD/Fs in the parts per trillion range could be applied directly to farmers fields, in addition to an appropriate microbial inoculum, such as a UV tolerant strain of white rot fungi to enhance degradation rates. A bench-scale technology using photothermal detoxification units

(PDUs), combines UV light and heat (at 200° to 600°C) to initiate degradation, and could be used to treat de-watered ASB sludge materials.

8.3 Modern pulp bleaching processes

The production of pulp involves the removal of lignin, a binding agent in wood fibres which inhibits the brightening of pulp fibres, usually through the use of caustic chemicals, followed by a bleaching process (see text box on p. 113). Pulp mill effluents consist of a complex mixture of organic compounds, including wood-derived carbohydrates and lignin derivatives, phenolic compounds, and organochlorine compounds (including PCDD/Fs , among others) (Gifford 1996).

Particular concern over the production of hydrophobic, lipophilic organochlorine contaminants in wastewater streams from bleached kraft mill effluents (BKME) has led to the substitution of elemental chlorine with chlorine dioxide (ClO_2) during the first stage of the bleaching process. There are fundamental differences in the chemistry of elemental chlorine and chlorine dioxide which result in very different by-products. Not only does chlorine destroy the lignin by oxidizing it to form quinone, or ring- opened structures, it also reacts through an electrophilic substitution mechanism to chlorinate aromatic rings (*e.g.*, form PCDD/Fs) and some aliphatic side chain groups (LaFleur 1996). In contrast, ClO_2 only oxidizes lignin, primarily resulting in the formation of ring- opened muconic acid (ester) structures (LaFleur 1996). Small quantities of chlorinated organics can however be formed during chlorine dioxide bleaching. This is most likely due to the formation of hypochlorous acid, which remains in equilibrium with chlorine depending on the pH of the solution. Because the hypochlorous acid/chlorine will react extremely quickly with lignins after they are formed, chlorine levels are kept to a minimum. As a result, there is far less chlorination of organics with ClO_2 bleaching, than in full chlorine bleaching where the initial load of elemental chlorine is very high, allowing the polychlorination of organic compounds to occur (LaFleur 1996).

According to Solomon et al. (1994), the formation of highly hydrophobic compounds such as dioxins is virtually eliminated by increasing ClO_2 substitution to 100%. For a mill in Grande Prairie, Alberta, the switch to using 100% ClO_2 for bleaching, in addition to implementing condensate stripping technology and upgrading the ASBs have not only reduced dioxin concentrations below detection limits (i.e., 0.8 - 4 ppq), but have resulted in low levels of adsorbable organic halide (AOX) discharge (0.5 kg air dried metric tonnes), and a concomitant decrease of organochlorine compounds in the receiving waters and suspended sediments (Gifford 1996). For Canadian pulp and paper producers, the switch to ClO_2 use following the 1992 implementation of the *Pulp and Paper Effluent Regulations* has resulted in the virtual elimination of PCDD/F compounds from their effluents (see Section 3.2.2.1).

In addition to the widespread shift to elemental chlorine free (ECF) bleaching technologies using chlorine dioxide, several international mills have experimented with the installation of total chlorine free (TCF) bleaching processes. These TCF systems replace ClO_2 use with hydrogen peroxide, ozone, or peroxyacetic acids as oxidizers to remove the lignin from the pulp (Folke et al. 1996). Although there are virtually no PCDD/F compounds produced from either processes, TCF systems can offer the added benefit of lower AOX levels and chemical oxygen demand (COD) in plant effluent; however depending on the TCF process, elevated levels of H_2O_2 and/or O_3 may also be found in the effluent (Folke et al. 1996). There also appears to be very little difference in the acute toxicity of either ECF or TCF effluents to aquatic organisms. By exposing bacteria, algae, water fleas and fish eggs/larvae to both untreated and secondary treated ECF, and TCF effluents, Ahtiainen et al. (1996), found no remarkable differences in

toxicity between the two processes, and suggested that the observed effects were most likely due to the natural constituents in the wood (*e.g.*, resin acids). Folke et al. (1996) suggest that the decision of whether to use a TCF or an ECF system is less critical than the need to modernize the pulping operation and minimize potential discharge errors through operator awareness.

Pulp Bleaching Technology in Canada Today

Wood pulps are bleached to modify and improve the properties of the fibres, to make them suitable for specific products and applications. Wood is made of three main components, excluding water: cellulose (fibre), hemicelluloses (sugars), and lignin. The traditional means of creating pulp was to separate fibres by grinding whole logs against a rotating stone. These mechanical pulps (or high yield pulps) incorporate a high percentage of the biomass into the final product; they are still used in the production of newsprint, for example. The pulp may be bleached, with hydrosulphite or hydrogen peroxide, to increase the brightness for "value-added" paper grades. Wood pulp is also produced by using chemical means to separate the fibre from the lignin. Early chemical pulp processes used sulphur dioxide (SO_2) as the reactive agent in "cooking", or delignifying, the pulp. In Canada, ammonium and magnesium-based sulphite pulps are produced for use in paper and other speciality products. By far the predominant type of chemical pulp is sulphate (or kraft) pulp, which uses sodium sulphide (Na_2S) and sodium hydroxide (NaOH) in the cooking liquor. In kraft pulping, much of the organic material that is removed from the fibre is used to fuel boilers that drive a chemical recovery process. A complex chemical recovery cycle regenerates the pulping chemicals.

The kraft pulping process removes approximately 95% of the wood's lignin. The alkaline pulping process results in a highly coloured residual lignin. This lignin is removed and the fibre is brightened in a subsequent bleaching process. Canada is a major global exporter of bleached kraft pulp. Although certain pulp applications require specialized pulp properties, pulp is differentiated primarily by brightness and strength (often characterised by viscosity, or fibrelength). Brightness is measured as the reflectance of a certain wavelength from a sheet of pulp. Many paper producers specify a brightness of 90%ISO in order to produce the "white" paper products that the marketplace seemingly demands, so the major product from Canadian pulp mills is full market brightness (90%ISO) kraft pulp.

While the organic products of the kraft cooking process are directed back to a recovery process, the organic material removed in the bleaching process is directed to an effluent treatment process prior to the release of process water to the environment. The nature of the organic material, and its effect on the environment, has been a subject of intense debate and study, especially over the past fifteen years, since dioxins were first found in pulp mill effluent.

Early chemical pulp bleaching processes used chlorine to delignify the pulp. Lignin is a complex aromatic macromolecule. Chlorine reacts by addition and substitution onto the aromatic rings, causing the lignin macromolecule to break into fragments. These lignin fragments are solubilized in a subsequent alkaline treatment (extraction stage). The pulp was brightened in subsequent sodium hypochlorite treatments. The use of chlorine dioxide as a pulp bleaching chemical was developed in the 1920s, but it was not implemented widely on a commercial basis in Canada until the mid-1940s. The development of chlorine dioxide applications and an effective method for producing chlorine dioxide at a pulp mill site by Prof. Howard Rapson, at University of Toronto, were critical to the development of modern bleaching processes. By the mid-1980s, a small amount of chlorine dioxide was added to the initial chlorination stage, to preserve pulp viscosity. A series of chlorine dioxide brightening stages followed the first extraction stage. The use of chlorine in the initial delignification stage resulted in a significant amount of chlorinated organic material in the bleach plant effluent. The chlorinated organic material is characterized by the adsorbable organic halogen (AOX) measurement. The discovery of 2,3,7,8-tetrachlorodibenzo-ρ-dioxin ("dioxin") in the untreated effluent from Swedish kraft pulp mills precipitated a tremendous change in bleaching technology. Canadian research was instrumental for understanding the nature of dioxin formation in the bleaching process, and methods were developed for eliminating the formation of dioxin. Further, bleaching process changes have resulted in a tremendous decrease in the formation of AOX.

In the early 1990s, most bleached kraft pulp mills in Canada eliminated the use of chlorine in pulp bleaching and replaced it with chlorine dioxide. Chlorine dioxide delignifies primarily by oxidation and aromatic substitution. The result is a drastically decreased formation of halogenated organic material and the virtual elimination of multi-chlorinated products, such as dioxin.

Several pulping and bleaching technologies have been introduced to decrease the dependence on chlorine dioxide as the primary bleaching chemical. A number of pulp mills in Sweden and Finland are capable of producing bleached kraft pulps that are totally chlorine free (TCF). A small volume of TCF bleached pulp has been produced in Canadian sulphite pulp mills, and on a limited development basis in some kraft pulp mills. The following technologies are available to Canadian pulp mills, or are under development, for decreasing the amount of chlorine-containing compounds used in pulp bleaching.

Pulping

The goal of the pulping process is to removal lignin without affecting the quality of the remaining fibre or losing fibre yield. The lignin removal process can be driven by high temperature, high alkali content, and other harsh process conditions at the expense of pulp yield. A variety of modified cooking processes have been introduced to minimize extreme chemical conditions required for lignin removal throughout the cooking cycle. Chemical additives such as polysulphide and anthraquinone are being used to enhance lignin removal without sacrificing pulp yield.

Oxygen delignification uses oxygen gas in an alkaline environment to oxidise and remove lignin. The technology has been available since the early 1970s. Its use has increased steadily in Canada in the past decade. Many mills have expressed concern over the potential impact on fibre properties, especially the impact on the strength of Canada's premium long fibre softwood pulps. Oxygen delignification systems require large technological investments, are very expensive (>$25M), and the mill must have sufficient recovery system capacity to handle the higher organic load. The benefits are a 25-60% decrease in lignin entering the bleach plant, and the consequent decrease in bleaching chemical requirement and COD in bleach plant effluent.

Bleaching

Chlorine dioxide is used in the initial delignification stage. The amount of chlorine dioxide required is directly proportional to the incoming pulp lignin content. Chlorine dioxide also reacts with organic and inorganic materials that carry over with the pulp. Improved brownstock pulp washing technologies significantly decrease chemical consumption in the bleach plant.

Hexenuronic acids (HexA) are by-products of the kraft cook, especially prevalent in hardwood pulps. Chlorine dioxide reacts with HexA to form oxalic acid, therefore the presence of HexA contributes to increased chlorine dioxide consumption and to mill scaling issues (due to high oxalate content in bleach plant filtrates). Several mills have implemented the use of acid pre-treatment stages to eliminate HexA and transition metals ahead of the bleach plant. A new technology, which has been implemented commercially in Finland, eliminates HexA in a molybdate-catalysed acid peroxide pretreatment. This technology decreases the requirement for chlorine dioxide by eliminating HexA and removing lignin.

The use of xylanase, an enzyme that reacts at the fibre-lignin interface and enhances lignin removal in subsequent bleaching stages, has been evaluated in many Canadian pulp mills, and is being adopted in many U.S. pulp mills in response to the EPA Cluster Rules requirements. Mills claim a decreased requirement for chlorine dioxide of 10-20%. Xylanase and the molybdate-catalysed peroxide pretreatment can both be applied in the brownstock storage chest, prior to the bleach plant, with only minor adjustments to typical process conditions.

Hydrogen peroxide is being used to reinforce the alkaline extraction stages in most Canadian kraft pulp mills. Hydrogen peroxide oxidises the end groups of residual lignin fragments to improve lignin solubility and removal. Hydrogen peroxide also brightens the fibre and decreases the amount of chlorine dioxide

required in the later brightening stages. Hydrogen peroxide also reacts with dissolved organics, and can be decomposed in reactions catalysed by transition metals. It is not as efficient as chlorine dioxide, at delignification, but it is a cost-effective adjunct to chlorine dioxide that can be used to decrease chlorine dioxide use in the bleach plant.

The kinetics of the hydrogen peroxide reaction limit its effective consumption under the process conditions of typical conventional bleach plants. Typically, 3-6 kg per tonne of pulp are added, resulting in a savings of 5-10 kg of chlorine dioxide. In TCF bleaching sequences, hydrogen peroxide is the primary brightening chemical, considerably higher application amounts are required. In order to achieve high final pulp brightness, hydrogen peroxide consumption must be driven by higher reaction time and temperature. Specialized high pressure hydrogen peroxide reaction towers have been installed in several European pulp mills. These bleaching stages require transition metal removal pretreatments to ensure effective bleaching performance. These hydrogen peroxide stages (often called, PO-stages) have an associated high capital cost. There are no PO-stages in use in Canada. A similar alternative is the installation of a high pressure/temperature reaction tube in association with a standard Eop-stage. Several high temperature Eop stages operating in Canadian pulp mills have enabled drastic reductions in the chlorine dioxide application to the initial delignification stage.

Ozone is a strong oxidiser that can be used for pulp delignification as a direct replacement for chlorine dioxide. Early TCF bleaching sequences used chelation pretreatments followed by repeated high doses of hydrogen peroxide. Aggressive pulping processes, both cooking and oxygen delignification, removed as much lignin as possible prior to the bleach plant. Final brightness was limited to approximately 80%ISO and pulp strength suffered. Ozone was used at many Scandinavian mills to further the delignification and produce higher brightness TCF kraft pulp. Ozone stages are operated under acid conditions. The pH adjustments, required to get from the ozone stage conditions to the hydrogen peroxide bleaching conditions, introduce a large amount of Na and S to the kraft liquor cycle. This situation has been avoided in several TCF mills through the use of distilled peracetic acid for delignification and hydrogen peroxide activation.

Ozone and distilled peracetic have be implemented in ECF bleaching sequences to good effect. Ozone is added, in conjunction with chlorine dioxide, in the initial delignification stage at one Canadian mill. It provides a significant reduction in the chlorine dioxide requirement. Several other DZ-stages have been implemented worldwide. In a similar fashion, distilled peracetic acid has been added to chlorine dioxide brightening stages to replace a portion of the chlorine dioxide. Distilled peracetic acid is also added to the bleached pulp storage chest in several European mills to increase brightness, and decrease the requirement for chlorine dioxide in the bleach plant. This application has the added benefit of providing cost savings through decreased paper chemical requirements in integrated pulp and paper complexes.

Technologies such as laccase/mediator systems, dimethyldioxirane, manganese porphyrin mimetics, and polyoxometalate complexes are at an early research stage. Most of these new technologies have to overcome extremely high capital costs, severe hygiene/occupational health issues, hazardous handling issues, or high chemical costs.

Canadian pulp mills were quick to replace chlorine by chlorine dioxide as the bleaching technology developed. In the process, most mills have introduced hydrogen peroxide to the bleach plant to decrease the requirement for chlorine dioxide. Most mills have invested heavily in secondary waste treatment facilities, and made large investments in their chlorine dioxide generating facilities in order to eliminate chlorine from the bleach plant. Chlorine dioxide is a very effective bleaching chemical. Market demands have not forced Canadian pulp mills to make TCF kraft pulps, so the above mentioned technologies have been adopted as mill production becomes limited by chlorine dioxide generating capacity, or other issues such as effluent colour, AOX level, BOD/COD, or water availability.

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8.4 Use of Biological Screening Methods for Determining Dioxin-like Toxicity to Aquatic Animals

2,3,7,8-TCDD toxic equivalencies are commonly derived analytically, whereby concentrations of individual congeners known to act on the aryl hydrocarbon receptor (AhR)-binding mechanism are quantified, multiplied by their respective toxic equivalency factors (*e.g.*, WHO 1998 TEFs), and tallied to produce a total TEQ value. This technique relies on the assumption that the observed toxic equivalencies act in an additive fashion (Clemons et al. 1998). More recently, however, cell line bioassays have become popular for monitoring the combined toxic potencies of all compounds in complex environmental mixtures that act on AhRs (Clemons et al. 1998; Courtenay et al. 1999; Whyte et al. 2002). As biomarkers, these bioassays quantify the amount of AhR-binding compounds that are already active in the tissues, thereby sidestepping difficulties in assessing the various bioavailabilities of the different congeners (Schmitt and Dethloff 2000). Bioassays also offer the advantage of allowing a higher sample throughput, resulting in faster sample processing at a much lower cost than congener specific chemical analyses for the derivation of PCDD/F TEQs (van Overmeire et al. 2001). As such, they can provide a powerful tool for monitoring the level of exposure to a suite of chemicals with the common AhR-binding toxic mode of action (see Section 6.1), particularly in the case where an *a priori* knowledge of contaminant congeners do not exist.

The principle indicator used in bioassays to detect the presence of AhR-binding compounds in cells is the induction of the cytochrome P4501A (CYP1A) enzyme. The cytochrome P450 family of monooxygenase (or, primary oxidative) enzymes catalyze a number of metabolic reactions, including the oxidation of xenobiotic chemicals (Di Giulio et al. 1995). The amount of CYP1A activity in the cells is directly related to total exposure, thereby providing an indication of the effort spent on detoxifying these exogenous contaminants (Schmitt and Dethloff 2000; Whyte et al. 2002). Although there are several bioassays which will measure the induction of CYP1A, biochemical analysis for the presence of known AhR-ligands, such as planar chlorinated hydrocarbons (PCHs; including PCDD/Fs), is most often done by measuring either aryl hydrocarbon hydroxylase (AHH) or, more commonly, 7-ethoxyresorufin-*O*-deethylase (EROD) activity (Clemons et al. 1998).

In bioassay derived-TEQs, tissue samples from fish exposed to potential AhR-ligands are homogenized, and exposed to active cell cultures which posses CYP1A enzymes. Common cell lines used for this bioassay are the rainbow trout liver RTL-W1 and the rainbow trout gonadal RTG-2 cell lines for piscine receptors, and the rat hepatoma H4IIE cell line for mammalian receptors (Whyte et al. 1998). Any PCDD/Fs present from the original tissue sample will bind to aryl hydrocarbon receptors within the cell cultures, stimulating the induction of CYP1A1. The amount of CYP1A1 induced in the cells (as measured by either AHH, or EROD activity) is then expressed relative to an equivalent amount of activity induced by pure 2,3,7,8-TCDD exposure to obtain a TEQ value (Whyte et al. 1998).

There are several factors to consider when comparing analytically-derived TEQs to those from bioassays (van Overmeire et al. 2001). Bioassays can detect all compounds acting as AhR agonists, while chemical analysis is restricted to a limited number of congeners. The TEF-approach used in analytical techniques assumes additive responses from AhR-binding congeners. While this is generally upheld for PCDD/Fs, non-additive interactions between certain PCBs and 2,3,7,8-TCDD have been observed (van den Berg et al. 1994a; Clemons et al. 1998; Safe 1998). In addition, bioassay-specific relative potency factors are sometimes used to derive TEFs (*e.g.*, Whyte et al. 1998), rather than WHO-TEFs, which can influence final TEQ values. Finally, detection limits in the analysis of congeners may limit analytically-derived TEQ estimates, whereas bioassays do not suffer from this bias (van Overmeire et al. 2001). Despite

these differences between techniques, an analysis of livers from lake trout obtained from selected sites in Lakes Ontario and Superior, found no significant differences between analytically-derived TEQs, and TEQs derived from either mammalian (H4IIE), or piscine (RTL-W1 or RTG-2) cell lines (Whyte et al. 1998). This suggested that all of the AhR-active compounds in the tissues were accounted for by the chemical analyses, and that the toxic potentials of these congeners were acting in an additive fashion (Whyte et al. 1998). Bioassays may therefore be used to compliment traditional analytical techniques. Establishing threshold values for bioassay derived TEQs would provide an initial screening mechanism for potential toxic effects. In samples with unacceptably high contaminant levels, congener-specific analysis could then quantify exposure levels, and aid in identifying specific sources.

Various supporting evidence suggests that exposure to 2,3,7,8-TCDD, and related chemicals, initiates an AhR-mediated toxic responses in animals; including alterations in gene expression, quantitative structure and activity relationship studies, and the apparent lack of toxicity of 2,3,7,8-TCDD in genetically modified AhR knockout mice (van Overmeire et al. 2001, and references therein). An important distinction with EROD activity is in its use as an indicator of contaminant exposure, rather than an indicator of effect (Whyte et al. 2002). Because of the close association between observed AhR-mediated toxic effects, and CYP1A induction, it is most likely that EROD activity in fish precedes the observed toxic effects of certain AhR ligands (Whyte et al. 2002). However, no direct mechanistic links between EROD activity and any observed physiological effects have yet been established (Whyte et al. 1998). In the study of Great Lakes lake trout livers, Whyte et al. (1998) found no correlation between chemically- or bioassay-derived TEQs and EROD activity in fish at each site, suggesting that the two indicators are measuring related, but different processes. As PCHs have half-lives ranging from weeks, to years, TEQs would appear to measure the store of EROD-inducing compounds accumulated over an extended period, whereas the short induction and decay timescale of EROD activity assays (*e.g.*, days), suggests EROD activity likely reflects a more recent exposure to AhR-binding compounds (Whyte et al. 1998). The poor correlation between TEQs and EROD activity may also be a result of the loss of EROD-inducing compounds in samples *in vivo* through metabolization, or from the loss of compounds during the extraction procedure; both of which would affect the amount of compound in an extract, and therefore in the derived TEQ value (Whyte et al. 1998).

The role of CYP1A induction as a biomarker for exposure to planar halogenated/ aromatic hydrocarbon compounds has been realized for nearly 3 decades (Whyte et al. 2002). Recent advancements in the understanding of enzyme induction are expanding the applicability of this technique for analysing exposure of AhR-ligand contaminants to aquatic animals. The response of CYP1A genes to TCDD toxicity in fish is well known, especially during sensitive early life stages. Although the two forms of rainbow trout cytochrome P450A1 that are induced by exposure to dioxin-like compounds are known (i.e., CYP1A1 and CYP1A3), due to their high degree of similarity in enzyme function (i.e., 96% of their amino acids are the same), their relative roles in CYP1A induction have remained unclear (Cao et al. 2000). Using reverse transcription-polymerase chain reaction (RT-PCR) and a ribonuclease protection analysis (RPA), Cao et al. (2000) found that gene expression response to 2,3,7,8-TCDD exposure differed according to cell type in rainbow trout. Although CYP1A3 activity was greater than CYP1A1 in intestinal tissues, CYP1A1 activity was preferentially induced in trout liver, heart, kidney, and trout sac fry, while no CYP1A3 activity was found in the commonly used rainbow trout hepatoma (RTH)-149, or rainbow trout gonad (RTG)-2 cell lines used for bioassays (Cao et al. 2000).

EROD activity is a widely used indicator for CYP1A activity. However, it has been shown that CYP1A protein levels and enzyme activities in fishes can be (1) inhibited by high substrate concentrations; (2) degraded by other contaminants; and (3) modulated by intrinsic biological factors such as sex and reproductive state (Courtenay et al. 1999, and references therein). Alternatively, measuring induction at the transcriptional level as CYP1A mRNA may minimize these interferences. Courtenay et al. (1999) demonstrated that CYP1A mRNA induction in Atlantic tomcod was linearly dose-dependent on 2,3,7,8-TCDD exposure, and that CYP1A mRNA made an excellent biomarker for comparing exposure to polycyclic- and halogenated aromatic hydrocarbons (including PCDD/Fs) within the bottom-dwelling tomcod in a number of rivers along the North American Atlantic coast that ranged from unindustrialized to heavily industrialized.

Recently, AhRs have been partially characterised for lesser known aquatic non-mammalian vertebrates such as the common tern (*Sterna hirundo*), and an amphibian, the mudpuppy (*Necturus maculosus*) (Karchner et al. 2000). These initial steps towards a more complete mechanistic understanding of the AhR signalling pathway may provide additional insight into species-specific differences in exposure sensitivity to dioxin-like compounds in aquatic environments (Karchner et al. 2000).

The extensive validation of cell line bioassays using EROD induction as a biomarker for dioxin-like activity has led to its use in global biomonitoring programs, including the North Sea Task Force, Environment/Cellulose, Mediterranean Pollution Network, and the French National Observation Network (Whyte et al. 2002, and references therein). As part of the United States national biomonitoring program on the health of fish in riverine ecosystems (Biomonitoring of Environmental Status and Trends - BEST), the U.S. Geological Survey included the HP4IIE rat hepatoma cell bioassay and the hepatic EROD activity assays as screening tools for polyhalogenated hydrocarbon contamination in fish (Schmitt and Dethloff 2000). Following the analysis of fish carcasses for PCBs, and organochlorine levels by gas chromatography, extracts from composite whole fish samples are screened for AHH-active compounds using the H4IIE bioassay. Total dioxin TEQs are determined by removing all PAHs from the sample matrix, and scaling the cumulative concentrations of remaining AHH-active PCBs, and PCDD/Fs, to a 2,3,7,8-TCDD standard. To determine the cumulative impact from exposure to all AHH-active compounds, including PAHs, EROD activity from the livers are then measured. By comparing EROD results with those from chemical analysis and HP4IIE endpoints, it is possible to determine which classes of chemicals were responsible for the cumulative effect on EROD (Schmitt and Dethloff 2000).

Currently, there are no national programs that use cell line bioassays to monitor the accumulation of AhR-binding compounds in Canadian aquatic biota. However, Environment Canada, in partnership with the University of Waterloo, Ontario Ministry of Environment and the Environment Canada St. Lawrence River Institute, are working towards developing a standardized test method for conducting rainbow trout hepatocyte bioassays. This rainbow trout bioassay has potential to provide an alternative approach to the standard 96hr-LC_{50} toxicity assay currently required for compliance monitoring, and environmental effects monitoring programs. The relatively small number of liver tissue samples required for this assay would therefore reduce the large numbers of test fish sacrificed under the current lethality test (Van Aggelen 2002, pers. comm.).

In conclusion, cell line bioassay techniques offer the advantage of being able to identify and characterize potential AhR agonists in complex environmental mixtures where an *a priori* knowledge of the compound's identity does not exist, while also providing an estimate of the

relative toxic potency of these samples (Denison et al. 2000). Because of this, cell line bioassays have been proven to be a useful tool for monitoring wastewaters from bleached kraft mills and other industrial effluents that may contain PCDD/Fs, and other dioxin-like compounds (Whyte et al. 2002).

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