Swan Hills Treatment Center

Long-Term Follow-up
Human Health Risk Assessment Program

Wild Game and Fish Monitoring

1997 - 2007

November 2009

Government of Alberta

Alberta

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EXECUTIVE SUMMARY

The Swan Hills Treatment Centre is a facility for the safe disposal of special wastes located approximately 12 kilometers northeast of the town of Swan Hills. On October 16, 1996, a malfunction of a transformer furnace was discovered, which caused the release of a portion of process gases containing the contaminants of polychlorinated biphenyls (PCBs), polychlorinated dibenzo-ρ-dioxins and dibenzofurans (PCDD/Fs) into the ambient air. In 1997, Alberta Health and Wellness conducted a human health risk assessment study to estimate human exposure and evaluate the effectiveness of public health interventions. As a result of the study, food consumption advisories were issued for wild game and fish taken within a 30 km radius of the facility. A long-term environmental monitoring and human exposure assessment program has been ongoing since 1998 to monitor concentrations of PCBs and PCDD/Fs in wild game and fish, review current food consumption advisories and protect public health of local residents. As part of this long-term monitoring program, human blood monitoring, wildlife tissue monitoring, fish tissue monitoring, a background contaminants survey and a contaminant exposure assessment have been conducted. This report discusses the results from the following studies:

1. The results of analyses of deer taken within a 30 km radius of the facility and the reference areas in 1999, 2001, 2003 and 2007 for the levels of PCBs, PCDD/Fs and dioxin-like Toxic Equivalency (TEQ);
2. The results of analyses of brook trout taken from Chrystina Lake in 2000, 2003 and 2007 for the levels of PCBs, PCDD/Fs and dioxin-like TEQs; and
3. The estimation of human exposure to dioxin-like TEQs and PCBs through consumption of wild game and brook trout taken from the Swan Hills area.

The results of these studies indicate that

1. As compared to the contaminant levels in deer in1997 in *the area of the facility*, the ΣPCB levels in liver and muscle samples were statistically significantly decreased in1999 – 2007. The levels of ΣPCDD/Fs and Σdioxin-like TEQs were not statistically significantly changed in all types of samples in 1999 – 2007.
2. As compared to the contaminant levels in deer in 1999 in *the reference areas*, the levels of ΣPCBs, ΣPCDD/Fs and Σdioxin-like TEQs were still higher in all types of samples except for ΣPCDD/F levels in muscle samples.
3. As compared to the contaminant levels in brook trout taken from *Chrystina Lake* in 1997 nearby *the facility*, the ΣPCDD/F and Σdioxin-like TEQ levels in liver and muscle samples were statistically significantly decreased in 2000 – 2007. The levels of ΣPCBs were not statistically significantly changed in all types of samples in 2000 – 2007.
4. As compared to the contaminant levels in northern pike taken from *the reference lakes* in 1997, the levels of ΣPCBs and Σdioxin-like TEQs were still higher in all types of brook trout samples except for Σdioxin-like TEQ levels in brook trout liver

samples. The levels of ΣPCDD/Fs were not statistically significantly changed in all types of samples in 2000 – 2007.

Comparison of Results between 1997 and 2007

	Results in 1999-2007 Compared to in 1997 Swan Hills Treatment Center Area				Results in 1999-2007 Compared to in 1999 Reference Area			
	Deer		Brook Trout		Deer		Brook Trout	
	Liver	*Muscle*	*Liver*	*Muscle*	*Liver*	*Muscle*	*Liver*	*Muscle*
∑PCBs	Decline	Decline	Similar	Similar	Higher	Higher	Higher	Higher
∑PCDD/Fs	Similar	Similar	Decline	Decline	Higher	Similar	Similar	Similar
∑TEQs	Similar	Similar	Decline	Decline	Higher	Higher	Similar	Higher

5. The levels of ΣPCBs, ΣPCDD/Fs and Σdioxin-like TEQs in deer decreased with increased distance from the facility. The inverse relationships were particularly observed at a distance of within 15 km radius of the facility fence during the period of 1999 – 2007. This inverse relationship suggests that the contamination is limited to the immediate vicinity of the facility.
6. Distribution of congener patterns for ∑PCBs, ∑PCDD/Fs and ∑dioxin-like TEQs in deer and brook trout in 1999-2007 were consistent with those observed in 1997. Similarity of distribution patterns of the congeners indicates that the sources of contamination could come from the facility.
7. The estimated daily intakes for consuming deer muscles in the high intake group (190 g/d) based on the 90th percentile level of Σdioxin-like TEQs and ΣPCBs in 1999 – 2007 were within the Health Canada guidelines.
8. The estimated daily intakes for consuming brook trout muscles in the high intake group (170 g/d) based on the 90th percentile level of Σdioxin-like TEQs and ΣPCBs in 2000 – 2007 exceeded the Health Canada guidelines.
9. Restriction of consumption of wild game and brook trout taken nearby the Swan Hills area was indicated by the health risk assessment. The existing food consumption advisories are still effective.
10. Food consumption advisories are voluntary measures to reduce potential health risk to local food consumers. The balance between risk and benefits of consumption of contaminant-containing local food needs to be understood and considered by consumers.

The results were reviewed by the Science Advice Committee and the Public Health Management Committee. Long-term wildlife and fish monitoring should continue in order to review these existing food consumption advisories.

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1. Introduction

The Swan Hills Treatment Centre is a facility for the safe disposal of special wastes located approximately 12 kilometers northeast of the town of Swan Hills. On October 16, 1996, a malfunction of a transformer furnace was discovered to have caused the release of a portion of process gases containing polychlorinated biphenyls (PCBs), polychlorinated dibenzo-ρ-dioxins and dibenzofurans (PCDD/Fs) into the ambient air. In 1997, Alberta Health and Wellness conducted a human health risk assessment study to estimate human exposure and to evaluate the effectiveness of public health interventions. As a result of the study, food consumption advisories for wild game and fish taken within a 30 km radius of the facility were issued.

A long-term environmental monitoring and human exposure assessment program has been ongoing since 1998 to monitor concentrations of PCBs and PCDD/Fs in wild game and fish, to review current food consumption advisories and to protect the health of local residents. As part of this long-term monitoring program, the wildlife tissue monitoring, fish tissue monitoring, and exposure assessment were carried out every 2 or 3 years.

In this report, the information from the following studies is presented:

(1). The results of analyses of tissues of deer taken within a 30 km radius of the facility and the reference areas in 1999, 2001, 2003 and 2007 for concentrations of PCBs and PCDD/Fs;
(2). The results analyses of tissues of fish collected from Chrystina Lake in 2000, 2003 and 2007 for concentrations of PCBs and PCDD/Fs; and
(3). The estimation of human exposure to PCBs and PCDD/Fs through consumption of wild game and fish from the Swan Hills area.

2. Wildlife Tissue Monitoring: Deer

2.1 Materials and Methods

2.1.1 Field Collection

1997
Both fresh and frozen tissues samples were collected. Fresh deer samples were taken directly from the area designated for the study. Four whitetail deer were collected at distances of 0 km (fence), 10 km, 20 km, 30 km to the east of the facility in the direction of the prevailing winds. Eleven road-kill adult deer carcasses were collected from other locations in Alberta as a control group. Frozen deer and moose samples were taken from animals preserved in home freezers and donated by local licensed hunters and First Nations people. Approximately 40 people donated sixty frozen deer and moose meat samples collected between October 1996 and February 1997 from within a 30 km kilometer radius of the facility. All specimens consisting of muscle, liver and kidney were kept frozen at -20 °C prior to laboratory analysis.

1998–1999
Field collection was carried out in December 1998 and January 1999. Nine whitetail deer and mule deer were collected at distances of 1 - 25 km to the east and west of the Swan Hills Treatment Center. Ten deer were collected at a distance of 100 km to the west of the facility as a control group. Representative muscle and liver samples were taken from each deer. All samples were kept frozen at – 20 °C prior to laboratory analysis.

2000–2001
Field collection was carried out in December 2000 and January 2001. Six whitetail deer and mule deer were collected at distances of 1 - 30 km to the east and west of the Swan Hills Treatment Center. Representative muscle, liver and fat samples were taken from each deer. All samples were kept frozen at - 20 °C prior to laboratory analysis.

2002–2003
Field collection was carried out in December 2002 and February 2003. Seven whitetail deer and mule deer were collected at distances of 1 - 30 km to the east and west of the Swan Hills Treatment Center. Representative muscle, liver and fat samples were taken from each deer. All samples were kept frozen at - 20 °C prior to analysis.

2006–2007
Field collection was carried out in December 2006 and June 2007. Fourteen whitetail deer and mule deer were collected at distances of 1 - 30 km to the east and west of the Swan Hills Treatment Center. Representative muscle, liver and fat samples were taken from each deer. All samples were kept frozen at - 20 °C prior to analysis.

2.1.2 PCBs and PCDD/Fs Analysis

1997

PCDDs/Fs and PCBs determinations for all samples were performed by the MAXXAM Laboratory, Mississauga, Ontario. Analytical methods and QA/QC assurance were described in Environment Canada EPS 1/RM/23 (1992), Environment Canada AMD 96-05 (1996) and USEPA Method 1613 (1994). Forty four PCBs were analyzed: #18, #28, #33, #37, #44, #49, #52, #70, #74, #77, #81, #87, #99, #101, #114, #118, #119, #123, #126, #128, #137, #138, #151, #153, #156, #157, #158, #167, #168, #169, #170, #177, #180, #183, #187, #189, #191 and #209. Each sample was homogenized and sub-sampled for analysis. Prior to the initial extraction, samples were fortified with fifteen $^{13}C_{12}$-labeled PCDD/Fs and eight $^{13}C_{12}$-labeled PCBs. Samples were digested overnight in concentrated hydrochloric acid and then extracted with 50/50 dichloromethane/hexane for one hour. This extraction was repeated several times. Lipid content was determined gravimetrically from the remaining extract. The extracts were subjected to an acid/base silica cleanup, reconcentrated and split into two equal portions by weight. One portion, for PCDD/F analysis, was cleaned up on alumina following the standard operating procedure for PCDD/Fs. The PCB portion was cleaned up on a modified alumina column. Extracts were analyzed separately for PCBs and PCDD/Fs on an Autospec Ultima High Resolution Mass Spectrometer, interfaced with a Hewlett Packard Gas Chromatograph. PCBs were separated at EI 8,000 mode and PCDD/Fs at EI 10,000 mode. Fused silica capillary columns (60 meter, 0.25 mm ID, 0.25 μm film thickness) were used for determining PCDD/Fs and PCB congeners, respectively. Injector temperature was 265^{o} C. The total time of the GC run was 50 min. Congeners were detected in the selected ion monitoring (SIM) mode.

1999, 2001 and 2003

PCDD/Fs and PCBs determinations for all samples were performed by the Fisheries and Oceans Regional Dioxin Laboratory at the Institute of Ocean Sciences in Sidney, British Columbia. The methodologies used to process the samples, the criteria used for identification and quantification and the quality assurance quality control protocols followed are described in detail elsewhere (Ikonomou *et al.* 2001). From each sample, four aliquots were collected from the carbon-fibre fractionation, the last part of the sample clean-up process. Fraction-I contained the di-ortho PCBs, fraction-II the mono-ortho PCBs, fraction-III the non-ortho PCBs and fraction-IV the PCDDs and PCDFs. In fractions I to III all the possible 209 PCB congeners were measured with minimum isomeric interference. Analyses of all fractions were conducted by high-resolution gas chromatograph/high-resolution mass spectrometry (HRGC/HRMS). For all analyses the MS was operated at 10000 resolution under positive EI conditions and data were acquired in the Ion Monitoring Mode (SIM). The concentrations of identified compounds and their method detection limits (MDLs) were calculated by the internal standard

method using mean relative response factors determined from calibration standard runs, made before and after each batch of samples was run. Detection limits range from 0.01 to 0.12 pg/g for PCDD/Fs, 0.04 to 0.08 pg/g for non-ortho PCBs, 0.1 pg/g for mono-ortho PCBs and 0.1 to 0.2 pg/g for di-ortho PCBs.

2007

PCDD/Fs and PCBs determinations for all deer samples were performed by the AXYS Analytic Services in Sidney, British Columbia. PCDD/F analysis was based on the USEPA Methods 1613 (Tissues) (USEPA 1994). PCB analysis was based on the USEPA Methods 1668A (Tissue) (USEPA 1999). Detection limits were 0.05 pg/g for PCDD/Fs, and 0.1-1.32 pg/g for 209 PCB congeners.

2.1.3 Statistical Analysis

Toxic Equivalency Factors (TEFs) are an approach supported by the World Health Organization (WHO) to provide an internationally consistent method to evaluate mixtures of dioxin and dioxin-like compounds which individually exhibit highly variable toxicity. The rationale for the TEF concept and the criteria for inclusion of any compound was established at two WHO expert meetings (Ahlborg et al. 1994; Van den Berg et al. 1998) and reaffirmed at the most recent WHO expert meeting in 2005 (Van den Berg et al. 2006). The premise for the TEF concept is that the most toxic of the dioxins, 2,3,7,8 – tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) was found to exert its toxic action through the aryl hydrocarbon receptor (AhR) a protein found in most vertebrate species which mediates the toxic process. While 2,3,7,8-TCDD has been consistently found to be the most potent compound to show the AhR-mediated toxic effect, many other dioxin-like compounds (other PCDDs, PCDFs and some PCBs) also exert substantial toxic effects through the AhR receptor. Research has generally suggested that mixtures of different compounds which operate through the AhR receptor will show an overall toxicity that is approximately equivalent to the summation of the toxicity of each compound added in proportion to its individual concentration with each modified in proportion to its relative effect potency (REP), a measure of each individual compound's potency of AhR binding in relation to 2,3,7,8-TCDD. The REP values have been used to develop toxicity equivalency factors (TEFs) for dioxin and dioxin-like compounds. The TEF values for each contributing compound are multiplied by the actual concentration of each contributing compound and the adjusted concentrations are added together to produce a toxicity equivalence estimate (TEQ), which for a group of compounds summed together yields a ΣTEQ for that group of compounds. This approach can be summarized in the following equation:

$$\sum TEQ = \sum_{i=1}^{7} (PCDD_i \times TEF_i) + \sum_{j=1}^{10} (PCDF_j \times TEF_j) + \sum_{k=1}^{12} (PCB_k \times TEF_k)$$

For a compound to be included in the TEF concept, it must (Van den Berg et al. 2006):

- "*Show a structural relationship to the PCDDs and PCDFs*
- *Bind to the AhR*
- *Elicit AhR-mediated biochemical and toxic responses*
- *Be persistent and accumulate in the food chain*."

TEF values represent an expert assessment of the evidence on REP data and they are not precise. The latest WHO expert group (Van den Berg 2006) decided to choose TEF values in increments of 1/10 (i.e. 1.0, 0.1, 0.01, etc) and the logarithmic midpoint of each decade (i.e. 0.3, 0.03, etc).

The validity of the TEQ approach has been confirmed by a considerable body of research evidence (Fattore et al. 2000, Gao et al. 1999, Hamm et al. 2003, Walker et al. 2005), but the variability of input experimental data and the reliance on expert judgment suggests that the quantitative toxicity estimate is likely meaningful within a factor of 10. The approach was found to be predictive within a factor of 2 for experimental studies on developmental reproductive endpoints in rats (Hamm et al. 2003). Likewise, Walker et al. (2005) tested the hypothesis of dose additivity using the TEQ approach for individual and mixtures of dioxin-like compounds and found that the predictive ability of the TEQ approach was sufficient to preclude rejecting the dose-additivity hypothesis for experimental rodent carcinogenesis.

The non-detected levels of PCBs, PCDD/Fs and TEQs were treated as zero for calculation. The concentrations of PCBs, PCDD/Fs and TEQs were not normally distributed among the samples. Because the distributions were skewed, for the purpose of statistical analysis, the concentration values were transformed into natural logarithm prior to analysis. Statistical analysis involving t-test, correlation analysis and linear regression analysis were performed by using SPSS 15.0 software.

2.2 Results and Discussions

2.2.1 PCBs and PCDD/Fs: 1997

Means for $\sum PCB_{homologues}$ and $\sum PCCD/F_{homologues}$, wet weight, were significantly elevated for all liver ($p<0.05$) and fat samples ($p<0.05$, with the exception of PCDD/Fs) in the study area relative to the Alberta control areas in 1997. ∑dioxin-like TEQ levels were significantly elevated in all types of samples ($p<0.05$) in the study area. ∑PCCD/F TEQ levels increased with a decrease of distance from the facility of 10, 20 and 30 km ($R = -0.849$, $p<0.005$). No apparent correlation was observed between contaminant levels and age and sex of deer. The results of $\sum PCB_{congener}$, $\sum PCDD/F_{congener}$ and ∑TEQ levels in fresh samples are presented in Table1.

Table 1 Means of PCB and PCDD/F Levels in Fresh Deer Samples in 1997

	Study Area		Alberta Control	
	Liver (n=4)	Muscle (n=4)	Liver (n= 11)	Muscle (n=11)
Mean of $\sum PCB_{congener}$* (ng/g, lipid-basis) (range)	2408 (215-4490)	987 (nd-3565)	194 (nd-1177)	158 (nd-821)
Mean of $\sum PCDD/F_{congener}$ (pg/g, lipid-basis) (range)	74271 (555-208404)	165 (nd-405)	3063 (nd-22247)	95 (nd-470)
TEQ** (pg/kg, lipid-basis) ∑ Dioxin-like compounds (range)	12467 (72-32037)	788 (nd-3000)	136 (15-819)	13 (0.98-92)

* Sum of 44 individual congener levels. $\sum PCB_{homologues}$ were measured, $\sum PCB_{congener}$ accounted for 43% of $\sum PCB_{homologues}$. ** TEFs: WHO-IPCS for PCBs and NATO_CCMS for PCDD/Fs

A wide range of individual PCB congeners were detected. Hexa- (36%) and hepta-chlorobiphenyls (25%-30%) were the major PCB homologue groups in the samples from the study area while tri-, tetra-, octa- and deca- chlorobiphenyls were minor constituents. The CB congeners 8, 138, 153, and 180 constituted 55% to 64% of $\sum PCB_{congener}$ in the liver and muscle samples from the study area. With the exception of muscle samples from the study area, the majority of ∑dioxin-like TEQ was due to PCDD/Fs, ranging from 65% to 78%. 2,3,4,7,8-penta CDF was prevalent in the liver samples from the study area, accounting for 30% of ∑dioxin-like TEQ.

In muscle samples from the study area, ∑dioxin-like TEQ was largely due to PCBs. *Non-ortho* CB 126 was dominant among these samples, accounting for 97% of ∑dioxin-like TEQ. In contrast, 1,2,3,6,7,8-hexa CDD was the major congener in the liver (37%) and muscle (44%) from the control areas. 2,3,7,8-TCDD was not detected in the samples from the study area (at a detection limit of 0.5 pg/g whole weight). CB126 was not detected in the control samples. The findings are consistent with the results in the

company's monitoring programs in which CB 126, 138, 153 and 180 were found as major contributors in vegetation, soil, spruce needle and snow pack near the facility.

PCBs and PCDD/Fs were detected in 21 out of 50 frozen muscle samples and in 8 out of 10 liver and kidney samples donated by local residents in 1997. Means for $\sum PCB_{homologues}$ and $\sum PCCD/F_{homologues}$ were significantly elevated for all muscle samples from within the 20 km radius relative to outside the 20 km radius of the facility. Hexa-chlorobiphenyls was the dominated among the PCB homologue group (76%) for all muscle samples. The majority of ∑dioxin-like TEQ was due to PCBs, with CB 126 accounting for 86% of ∑dioxin-like TEQ.

Significantly higher levels of PCBs and PCDD/Fs in deer from the study area and similar PCB congener patterns observed in various media near the facility indicate that contamination has occurred in the ecosystems near the facility. Many studies have shown that an increased atmospheric deposition of PCBs contributes to an increased PCB burden in plants and herbivores (Eisler and Belisle 1995, Larsson *et al.* 1990). Lichens, moss and browse (as the primary food items of herbivores for the winter) are abundant in the vicinity of the facility and have been used to monitor airborne pollutants. The mobility of deer and moose is restricted to a relatively small area in harsh winters with heavy snow cover (Lesage et al. 2000). Deer and moose in the study area are likely to consume plants nearby. The inverse relationship between measured contaminant levels in deer and distance from the facility suggested the occurrence of the air-plant-herbivore pathway.

Accumulation of PCB congeners varies within different types of environmental samples and locations. The higher-chlorinated congeners have been more frequently observed in marine food chains and predators while the lower-chlorinated congeners are abundant in herbivores because lower chlorinated congeners are more likely to persist in vegetation (Larsson *et al.* 1990, Muir *et al.* 1988). Some studies reported an increased level of lower-chlorinated congeners in various animals (Georgii *et al.* 1994). The similar distribution of lower and higher chlorinated congeners in Alberta control samples implies that the potential exposure for deer in most of Alberta come from long range air transport and diverse sources.

Non-ortho PCBs (77, 126 and 169) are widely distributed in the environment but at comparatively low levels. Low levels of *non-ortho* PCBs were found in deer from Alberta controls. But a very high level for CB126 was observed in deer from the study area. *Non-ortho* PCBs were also detected in the samples of Labrador tea leaves, live moss, soil and voles near the facility in the company's monitoring program (Brown *et al.* 1995; Kimbrough 1995). Combustion processes could be the source of the increased environmental levels of the coplanar congeners characterized by 3,3',4,4' substitution such as CB 77, 105, 126, , 156, 157, 169, 170 and 189. CB 77 has been found to be more biodegradable than CB126 and CB169 (Kannan *et al.* 1989, Tanabe *et al.* 1987). Elsewhere, levels of CB126 in environmental media and the highly biodegradable nature of CB 77 may have caused CB126 to be the main CB congener found at a high level in deer collected near the facility.

2.2.2 PCBs: 1999–2007

The mean concentrations of ΣPCBs and their homologues in tissue samples between 1999 and 2007 are summarized in Tables 2, 3 and 4. The concentrations reported here are lipid-basis. In the 1999 study, 47 of 209 PCB congeners were not detected in muscle samples, and 38 congeners were not detected in liver samples. In the 2001 study, 49 congeners were not detected in muscle samples, 45 congeners were not detected in liver samples and 35 congeners were not detected in fat samples. In the 2003 study, 55 congeners were not detected in muscle samples, 37 congeners were not detected in liver samples and 35 congeners were not detected in fat samples. In the 2007 study, 42 congeners were not detected in muscle samples, and 46 congeners were not detected in liver samples.

The mean level of PCB homologues in the 1999 study was 21 ng/g in muscle samples and 47 ng/g in liver samples. The mean levels were 5.3 ng/g in muscle samples and 5 ng/g in liver samples in the reference areas. The mean level in the 2001 study was 291 ng/g in muscle samples, 317 ng/g in liver samples, and 506 ng/g in fat samples. The mean level in the 2003 study was 39 ng/g in muscle samples, 92 ng/g in liver samples and 20.5 ng/g in fat samples. The mean level in the 2007 study was 67 ng/g in muscle samples, and 85 ng/g in liver samples.

CB-153, CB-138 and CB-180 were the major contributing congeners. In the 1999 study, the abundant congeners in ∑PCBs were CB-138 (9%), CB-153 (19%), and CB-180 (9%) for samples from the study area. In the 2001 study, CB-153 accounted for 22% to 28% of ΣPCBs in all types of samples, CB-138 accounted for 11% to 15%, and CB180 accounted for 13% to 17%. In the 2003 study, CB-153 accounted for 9% to 20% of ΣCBs in all types of samples, CB-138 accounted for 6% to 8%, and CB180 accounted for 3% to 14%. In the 2007 study, CB-153 accounted for 21% to 25% of ΣCBs in all types of samples, CB-138 accounted for 6% to 7%, and CB180 accounted for 17%.

Non-ortho CBs constituted a very small proportion of ∑PCBs. Major contributors in the non-ortho CBs group were CB-11, CB-15 and CB-37 for all samples from the study and reference areas. CB-126 concentrations were significantly higher in liver samples from the study area (0.8 ng/g in 1999, 17 ng/g in 2001, 0.35 ng/g in 2003 and 1.6 ng/g in 2007) than those in the reference areas (0.009 ng/g in 1999).

For samples from the 1999 reference area, CB-8, CB-28, CB-138, CB-153, and CB-180 accounted for 11%, 6%, 4%, 6% and 2% of ∑PCBs, respectively. The major contributors in deer from the reference site were the lower-chlorinated congeners. Lower chlorinated congeners are likely to persist in vegetation. Thus, they are more frequently detected in herbivores including deer. High proportions of some higher chlorinated congeners observed in deer from the study area suggest that localized exposure sources for deer were influenced by the facility in this area.

Mean migration distance of deer is around 10 km and home range size is around 5 km in the winter (Burris 2005). Thus, deer can be effectively used as an indicator to monitor

the change of the contaminant concentrations nearby the facility. The contaminant levels in moose were not monitored in this long-term follow-up program due to the large home range size of moose. Most moose make seasonal movements for calving, rutting, and wintering areas. Mean home range size was around 200 km during these transitions (Stenhouse et al. 1994). This factor inevitably complicates the interpretation of monitoring of moose.

The ΣPCB concentrations at distances to the facility fence in deer tissue samples collected between 1999 and 2007 are presented in Table 5. A hierarchical multiple regression analysis was conducted to determine the joint effects of distance from the Swan Hills Treatment Centre, the year in which the deer was taken, and the interactions on ΣPCB concentrations in muscle and liver samples. Due to a skewed distribution, ΣPCB has been transformed to its natural logarithm prior to analysis. The distance from the facility is the most important factor in estimating ΣPCB concentrations. ΣPCB concentrations in muscle samples ($r = - 0.63$, $p < 0.001$) and liver samples ($r = - 0.75$, $p < 0.001$) decreased with increased distance from the facility fence (Figure 1). Predicted ΣPCB concentrations in 1999 - 2007 have converged by about 15 km from the facility fence (Figure 2). This finding suggests that contamination from the facility has occurred in the ecosystem in vicinity of the facility.

The statistical summaries of ΣPCB levels in 1997 - 2007 are presented in Table 6. The ΣPCB levels in liver and muscle samples varied significantly from year to year. In particular, ΣPCB levels in 2001 were higher than the levels in other years after controlling distance of the catch from the facility (Figure 2). The mean of ΣPCB concentrations was higher in liver samples (317 ng/g) and muscles samples (291 ng/g) in 2001 than those in 1999, 2003 and 2007.The high mean level resulted from the influence on the mean of the very high level of ΣPCBs in one deer taken 0.5 km from the facility fence (Table 5, bolded value; Figure 1).

Overall, ΣPCB levels collected between 1999 and 2007 were decreased as compared to the levels in liver samples ($p < 0.001$) and muscle samples ($p = 0.15$) in 1997, but the levels were still significantly higher than those in deer collected from the reference area in 1999 ($p < 0.001$) (Figure 3).

Table 2 Means of PCB Homologues in Deer Muscle Samples, 1999–2007

(ng/g, lipid-basis)

	2007 Study Area (N=14)	2003 Study Area (N=7)	2001 Study Area (N=6)	1999 Study Area (N=9)	1999 Control (N=10)
Non-ortho					
mono-CBs	0.23	–	–	–	–
di-CBs	0.15	1.08	0.67	0.40	0.23
tri-CBs	0.02	0.31	1.17	0.24	0.08
tetra-CBs	0.01	0.13	0.51	0.09	0.06
penta-CBs	0.04	0.03	0.13	0.07	0.01
hexa-CBs	<0.05	0.01	0.04	0.004	0.002
Total	**0.21**	**1.6**	**2.5**	**0.80**	**0.38**
Mono-ortho					
mono-CBs	0.03	–	–	–	–
di-CBs	0.10	0.99	0.00	0.32	0.72
tri-CBs	0.31	2.07	3.46	0.92	0.68
tetra-CBs	0.56	3.10	7.74	0.39	0.20
penta-CBs	2.26	2.65	13.75	1.32	0.21
hexa-CBs	3.19	0.52	7.05	0.51	0.06
hepta-CBs	0.45	0.00	0.60	0.06	0.002
Total	**6.9**	**9.3**	**33**	**3.5**	**1.9**
Di-ortho					
di-CBs	0.07	0.04	0.23	0.04	0.06
tri-CBs	0.18	1.23	1.58	0.34	0.31
tetra-CBs	0.26	3.03	5.72	0.75	0.49
penta-CBs	1.33	5.18	22.03	1.25	0.53
hexa-CBs	19.4	10.2	116	7.58	1.00
hepta-CBs	24.1	6.57	79.5	4.74	0.46
octa-CBs	12.3	1.47	28.2	1.83	0.16
nona-CBs	1.53	0.16	2.34	0.24	0.04
deca-CBs	0.29	0.09	0.37	0.08	0.07
Total	**60**	**28**	**256**	**17**	**3.1**
Total CBs	**67**	**39**	**291**	**21**	**5.3**
% *non-ortho*	3.2	4.0	1.0	4.0	7.0
% *mono-ortho*	6.7	24	11	17	35
% *di-ortho*	90	72	88	79	58
% CB 138	6	6	10	9	4
% CB 153	21	9	22	19	6
% CB 180	17	6	14	9	2

Note: N = sample number; " – " = not measured

Table 3 Means of PCB Homologues in Deer Liver Samples, 1999–2007

(ng/g, lipid-basis)

	2007 Study Area (N=14)	2003 Study Area (N=7)	2001 Study Area (N=6)	1999 Study Area (N=9)	1999 Control (N=10)
Non-ortho					
mono-CBs	0.01	–	–	–	–
di-CBs	0.03	0.75	0.20	0.84	0.53
tri-CBs	<0.05	1.08	0.27	0.28	0.10
tetra-CBs	0.01	0.40	0.28	0.18	0.04
penta-CBs	1.56	0.35	16.60	0.96	0.02
hexa-CBs	<0.05	0.01	0.72	0.03	0.002
Total	**1.6**	**2.6**	**18**	**2.3**	**0.69**
Mono-ortho					
mono-CBs	<0.05	–	–	–	–
di-CBs	0.01	0.30	0.18	0.35	0.34
tri-CBs	0.09	2.04	2.25	1.16	0.89
tetra-CBs	0.34	10.4	3.63	0.92	0.15
penta-CBs	2.35	7.43	17.8	1.90	0.12
hexa-CBs	4.82	1.15	15.6	0.93	0.04
hepta-CBs	0.47	0.01	0.98	0.09	0.002
Total	**8.1**	**21**	**40**	**5.4**	**1.5**
Di-ortho					
di-CBs	<0.14	0.04	0.09	0.18	0.04
tri-CBs	0.03	0.49	0.67	1.01	0.21
tetra-CBs	0.07	2.87	1.34	2.19	0.41
penta-CBs	1.4	15.7	6.96	3.26	0.37
hexa-CBs	28.7	32.5	139	8.43	0.40
hepta-CBs	32.2	13.8	79.3	12.1	0.75
octa-CBs	11.5	2.89	29.3	11.7	0.52
nona-CBs	0.69	0.18	1.55	0.41	0.03
deca-CBs	0.21	0.04	0.21	0.18	0.04
Total	**75**	**69**	**258**	**39.4**	**2.8**
Total CBs	**85**	**92**	**317**	**47**	**5**
% *non-ortho*	2	3	5	5	14
% *mono-ortho*	10	23	13	11	31
% *di-ortho*	88	74	82	84	55
% CB 138	7	8	12	8	4
% CB 153	25	10	28	15	7
% CB 180	17	3	12	8	2

Note: N = sample number; " – " = not measured

Table 4 Means of PCB Homologues in Deer Fat Samples in 2001 and 2003

(ng/g, lipid-basis)

2001

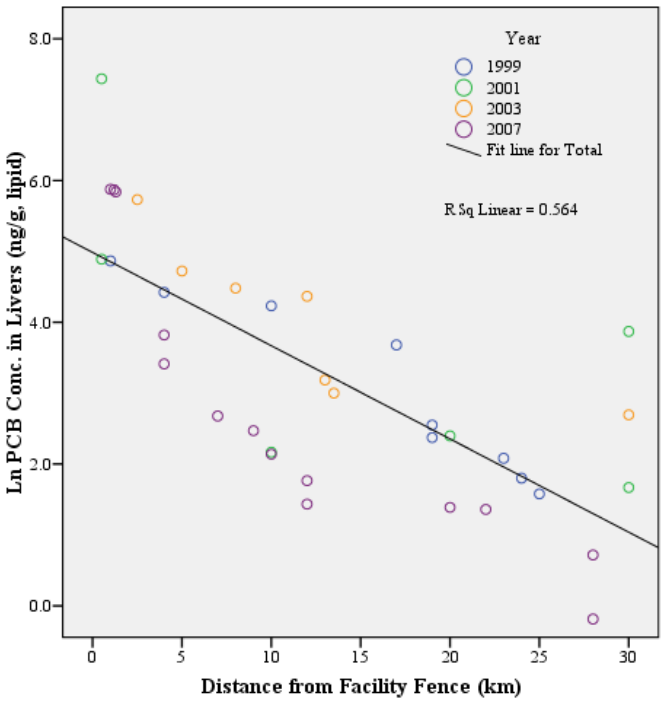
Non-ortho		*Mono-ortho*		*Di-ortho*			
						Total CBs	*506*
di-CBs	0.04	*di-CBs*	0.19	*di-CBs*	0.01	% of *non-ortho*	0.08
tri-CBs	0.02	*tri-PCBs*	0.54	*tri-CBs*	0.08	% of *mono-ortho*	7.62
tetra-CBs	0.05	*tetra-CBs*	1.89	*tetra-CBs*	0.33	% of *di-ortho*	92.3
penta-CBs	0.24	*penta-CBs*	22.53	*penta-CBs*	19.95		
hexa-CBs	0.06	*hexa-CBs*	12.54	*hexa-CBs*	244.0		
Total	**0.41**	*hepta-CBs*	0.85	*hepta-CBs*	157.1		
		Total	**38.5**	*octa-CBs*	41.37		
				nona-CBs	2.58		
				deca-CBs	0.27		
				Total	**466.7**		

2003

Non-ortho		*Mono-ortho*		*Di-ortho*			
						Total CBs	***20.5***
di-CBs	0.08	*di-CBs*	0.05	*di-CBs*	0.00	% of *non-ortho*	1
tri-CBs	0.11	*tri-PCBs*	0.62	*tri-CBs*	0.16	% of *mono-ortho*	26
tetra-CBs	0.02	*tetra-CBs*	1.37	*tetra-CBs*	0.34	% of *di-ortho*	73
penta-CBs	0.02	*penta-CBs*	2.32	*penta-CBs*	0.08		
hexa-CBs	0.01	*hexa-CBs*	0.87	*hexa-CBs*	6.37		
Total	**0.24**	*hepta-CBs*	0.07	*hepta-CBs*	5.31		
		Total	**5.29**	*octa-CBs*	1.74		
				nona-CBs	0.21		
				deca-CBs	0.04		
				Total	**14.97**		

Table 5 ΣPCB Levels in Deer vs. Distance to Facility Fence

Sample #	Distance (Km)	Liver (ng/g, lipid-basis)	Muscle (ng/g, lipid-basis)
2007			
1	1	354	216
2	1.2	343	369
3	1.3	356	200
4	4	46	25
5	4	30	43
6	7	0.83	2.0
7	9	15	15
8	10	12	10
9	12	5.9	12
10	12	3.9	5.7
11	20	4.0	7.9
12	22	4.2	9.8
13	28	8.5	7.3
14	28	2.1	11
2003			
1	2.5	24	47
2	5	78	44
3	8	308	35
4	12	113	50
5	13	20	35
6	13.5	15	52
7	30	88	11
2001			
1	**0.5**	**1695**	**1523**
2	0.5	133	121
3	10	8.7	15
4	20	11	11
5	30	5.3	4.3
6	30	48	70
1999			
1	1.0	130	84
2	4.0	69	52
3	10	83	12
4	17	13	14
5	19	11	5.6
6	19	40	6.3
7	23	4.8	3.2
8	24	6.1	3.9
9	25	8.0	8.1



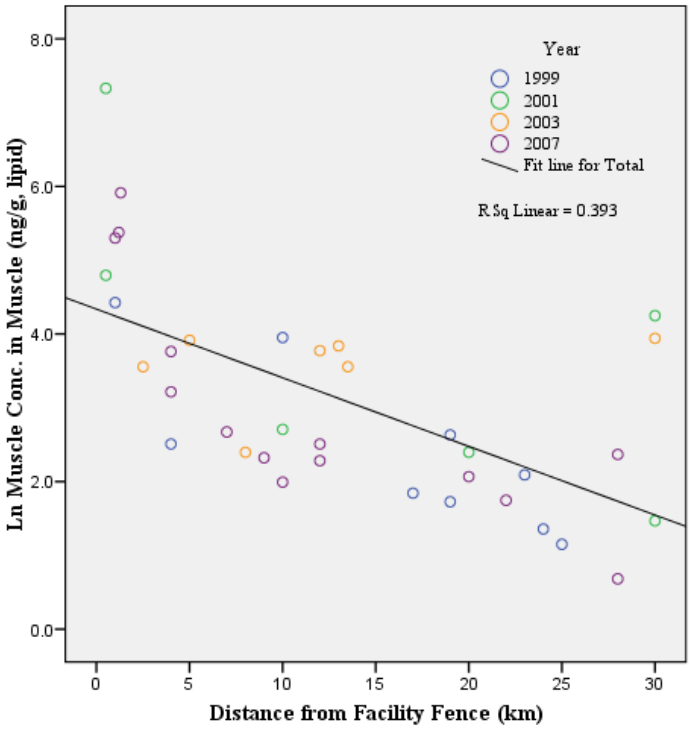
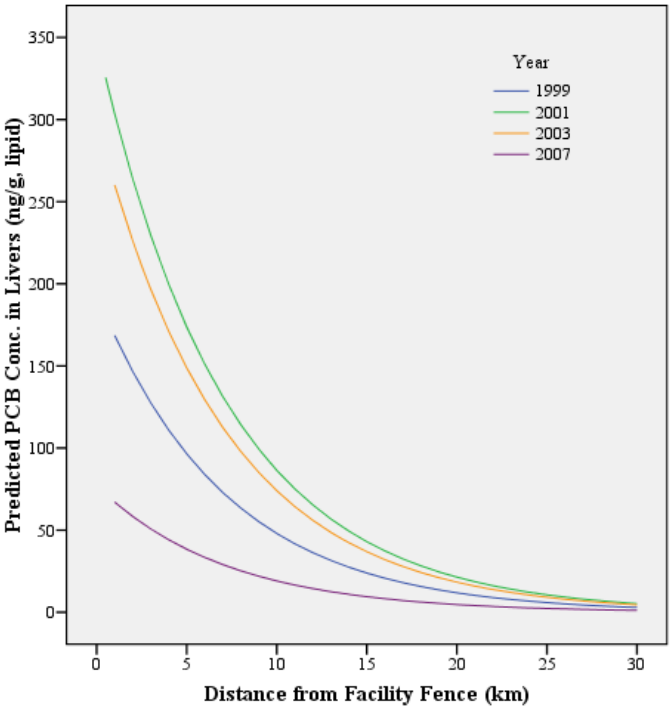


Figure 1 ΣPCB Levels in Deer Liver and Muscle Samples vs. Distance

[t-test: ΣPCB concentrations at distance from the facility fence ≤ 15 km vs >15 km, p <0.001]



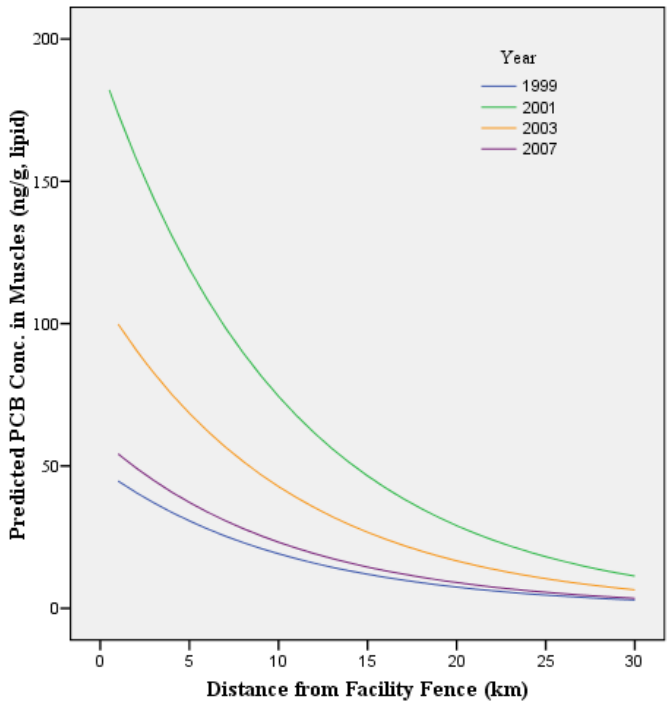


Figure 2 Predicted ΣPCB Levels in Deer Liver and Muscle Samples vs. Distance by Years

Table 6 Statistical Summaries of ΣPCB Levels in Deer (ng/g, lipid-basis)**, 1997 - 2007**

	Sample #	Mean	Median	SD	Min	Max	95% Confidence	
							Lower	Upper
Liver								
1997	4	2408	2464	2214	215	4490	-1116	5932
1999	9	47	13	44	4.9	130	6.3	75
2001	6	317*	30	677	5.3	1694	-393	1027
2003	7	92	79	103	15	308	-2.4	187
2007	14	85	10	145	0.83	356	0.95	168
1999 Ref	10	5.0	3.7	1.7	2.3	7.7	2.9	5.3
Muscle								
1997	4	987	191	1728	non-detected	3565	-1763	3737
1999	9	21	8.1	28	3.2	84	-0.46	42
2001	6	291*	238	605	4.3	1522	-344	926
2003	7	39	44	14	11	52	26	52
2007	14	67	11	113	2.0	370	1.8	131
1999 Ref	10	5.3	4.5	3.1	1.5	10	3.0	7.4

Note: The levels were based on a sum of 44 congeners in 1997 and a sum of 209 congeners in 1999 – 2007
* high value is due to the highest value detected in one deer close to the facility fence.

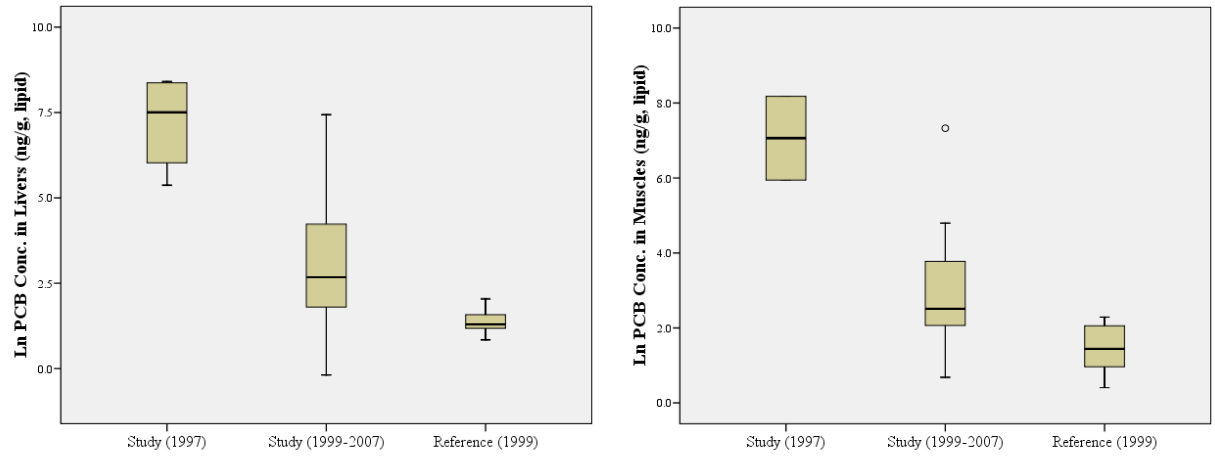


Figure 3 ΣPCB Levels in Study Area and Reference Area

* t – test for ln concentrations in reference area (1999) vs. study area (1999 – 2007): $p < 0.001$ in liver and muscle samples. The levels in 1997 vs. 1999 – 2007: $p < 0.01$ in liver samples and $p = 0.15$ in muscle samples.
Marker o is the outlet of PCB level in one muscle sample collected close to the facility fence.

2.2.3 PCDD/Fs: 1999–2007

The mean values of ∑PCDD/Fs and their homologues in 1999 are summarized in Table 7. The concentrations reported here are lipid-basis. In the 1999 study, all the 2,3,7,8-substituted PCDD/F congeners (17 in total) were detected in liver samples from the study area. Eight PCDD/Fs congeners were detected in muscle samples from the study area.

The mean values of ∑PCDD/Fs and their homologues in 2001 are summarized in Table 8. In the 2001 study, all 17 PCDD/Fs congeners were detected in liver and fat samples. Nine of 17 congeners were detected in muscle samples. The mean values of ∑PCDD/Fs and their homologues in 2003 are summarized in Table 9. In the 2003 study, fifteen of out 17 PCDDs/Fs congeners were detected in liver samples and fats. Five of out 17 congeners were detected in muscle samples. The mean values of ∑PCDD/Fs and their homologues in 2007 are summarized in Table 10. In the 2007 study, all PCDD/Fs congeners were detected in liver samples. Fourteen of out 17 congeners were detected in muscle samples.

OCDD was a major congener in muscle samples in the 1999 and 2003 studies. 2,3,4,7,8-PeCDF dominated in muscle samples in the 2001 and 2007 studies. 2,3,4,7,8-PeCDF was a major congener in liver samples in 1999, 2001 and 2003 studies. 2,3,4,7,8-PeCDF may be a marker congener present in the emissions of the special waste treatment facility as it has been observed to be the major congener in soil, vegetation, sediment, fish and voles collected near the facility since 1996.

The ΣPCDD/F concentrations at distances to the facility fence in deer tissues collected between 1999 and 2007 are presented in Table 11. A hierarchical multiple regression analysis was conducted to determine the joint effects of distance from the Swan Hills Treatment Centre, the year in which the deer was taken, and the interactions on ΣPCDD/F concentrations in muscle and liver samples. Due to a skewed distribution, ΣPCDD/F has been transformed to its natural logarithm prior to analysis. The distance from the facility is the most important factor in estimating ΣPCDD/F concentrations. ΣPCDD/F concentrations in muscle samples ($r = - 0.54$, $p < 0.001$) and liver samples ($r = - 0.75$, $p < 0.001$) decreased with increased distance from the facility fence (Figure 4). Predicted ΣPCDD/F concentrations in 1999 - 2007 have converged by about 15 km from the facility fence (Figure 5). This finding suggests that contamination from the facility has occurred in the ecosystem in vicinity of the facility.

The statistical summaries of ΣPCDD/F levels in 1997 - 2007 are presented in Table 12. The ΣPCDD/F levels in liver and muscle samples varied significantly from year to year. In particular, ΣPCDD/F levels in liver samples in 2007 were higher than the levels in other years after controlling distance of the catch from the facility (Figure 5). In 2007, five deer were taken within 5 km radius of the facility fence. The ΣPCDD/F levels in liver samples were higher in these five deer. If compared mean levels (Table 12), the mean

of ΣPCDD/F concentrations (29,980 pg/g) was higher in the liver samples in 2001 than those in 1999, 2003 and 2007, but the median levels was low (244 pg/g). The higher mean level (168749 pg/g, lipid) resulted from presence of the very high level of ΣPCDD/Fs in one deer taken 0.5 km from the facility fence (Table 11).

Overall, the ΣPCDD/F levels in liver samples in 1999 - 2007 were not statistically changed as compared to the levels in 1997 ($p > 0.05$). The ΣPCDD/F levels in1997 and 1999 – 2007 in liver samples were still significantly higher than those in deer collected from the reference area ($p < 0.001$) (Figure 6). The ΣPCDD/F levels in muscle samples in 1997 and 1999 - 2007 were low and not statistically significantly different in the reference areas. This means that the ΣPCDD/F levels in muscle samples were not changed too much in the study area and reference area over the past ten years.

Table 7 Means of PCDD/Fs in Deer in 1999 (pg/g, lipid-basis)

	Study Area				**Reference Area**			
	Muscle (n=9)		**Liver (n=9)**		**Muscle (n=10)**		**Liver (n=10)**	
	Conc.	**%**	**Conc.**	**%**	**Conc.**	**%**	**Conc.**	**%**
Lipid content		2.3		3.0		3.5		3.7
2,3,7,8-TCDD	<0.08	0.00	2.2	0.07	<0.08	0.00	<0.08	0.00
1,2,3,7,8-PeCDD	<0.08	0.00	28.9	0.93	<0.08	0.00	2.16	1.57
1,2,3,4,7,8-HxCDD	<0.10	0.00	44.2	1.42	<0.10	0.00	4.56	3.31
1,2,3,6,7,8-HxCDD	8.92	7.56	79.5	2.54	6.52	10.63	10.31	7.49
1,2,3,7,8,9-HxCDD	<0.10	0.00	29.4	0.94	<0.10	0.00	2.87	2.09
1,2,3,4,6,7,8-HpCDD	5.82	4.93	258	8.26	7.90	12.89	40.88	29.70
OCDD	81.82	69.34	295	9.43	38.98	63.59	53.87	39.13
2,3,7,8-TCDF	1.00	0.84	17.5	0.56	<0.05	0.00	<0.05	0.00
1,2,3,7,8-PeCDF	<0.06	0.00	4.4	0.14	<0.06	0.00	<0.06	0.00
2,3,4,7,8-PeCDF	6.68	5.66	1842	58.96	<0.06	0.00	6.00	4.36
1,2,3,4,7,8-HxCDF	0.90	0.76	218	6.96	<0.08	0.00	2.95	2.14
1,2,3,6,7,8-HxCDF	<0.08	0.00	120	3.83	<0.08	0.00	2.62	1.91
1,2,3,7,8,9-HxCDF	<0.08	0.00	96.0	3.07	0.27	0.45	1.97	1.43
2,3,4,6,7,8-HxCDF	<0.08	0.00	0.5	0.01	<0.08	0.00	<0.08	0.00
1,2,3,4,6,7,8-HpCDF	4.46	3.78	65.6	2.10	3.80	6.20	5.84	4.24
1,2,3,4,7,8,9-HpCDF	<0.10	0.00	6.5	0.21	<0.10	0.00	0.56	0.41
OCDF	8.42	7.13	17.8	0.57	3.82	6.24	3.07	2.23
$\sum$PCDD/F$_{congener}$	**118**	**100**	**3125**	**100**	**61**	**100**	**138**	**100**
Σ TCDD	5.4	4.13	5.5	0.18	3.3	4.36	3.7	2.57
Σ PeCDD	<0.08	0.00	28.9	0.92	<0.08	0.00	2.2	1.50
Σ HxCDD	11	8.16	154	4.89	9.7	12.29	18	12.82
Σ HpCDD	10	7.78	271	8.59	15	19.36	43	29.57
Σ OCDD	82	62.98	295	9.34	39	50.83	54	37.36
Σ TCDF	1.0	0.77	19	0.59	<0.05	0.00	<0.05	0.00
Σ PeCDF	7.3	5.58	1853	58.71	0.1	0.19	6.0	4.15
Σ HxCDF	0.9	0.69	436	13.82	0.8	0.99	7.6	5.26
Σ HpCDF	4.5	3.43	76	2.41	5.1	6.69	6.7	4.65
Σ OCDF	8.4	6.48	18	0.57	3.8	4.99	3.1	2.13
ΣPCDD/F$_{homologues}$	**131**	**100**	**3156**	**100**	**77**	**100**	**144**	**100**
% of $\sum$PCDD/F$_{congener}$ in ΣPCDD/F$_{homologues}$		91		99		80		96

Table 8 Means of PCDD/Fs in Deer in 2001 (pg/g, lipid-basis)

	Muscle (n=6)		Liver (n=6)		Fat (n=6)	
	Conc.	**%**	**Conc.**	**%**	**Conc.**	**%**
Lipid content		2.53		3.69		65
2,3,7,8-TCDD	<0.06	0.00	5.7	0.02	0.29	0.25
1,2,3,7,8-PeCDD	<0.08	0.00	31.3	0.10	0.92	0.79
1,2,3,4,7,8-HxCDD	<0.10	0.00	46.1	0.15	0.76	0.66
1,2,3,6,7,8-HxCDD	<0.10	0.00	47.9	0.16	1.26	1.09
1,2,3,7,8,9-HxCDD	<0.10	0.00	10.3	0.03	0.31	0.27
1,2,3,4,6,7,8-HpCDD	3.89	3.55	210	0.70	1.81	1.56
OCDD	25.56	23.35	285	0.95	3.30	2.84
2,3,7,8-TCDF	7.99	7.30	147	0.49	10.77	9.27
1,2,3,7,8-PeCDF	1.42	1.30	8.5	0.03	2.92	2.51
2,3,4,7,8-PeCDF	54.49	49.78	24155	80.57	76.95	66.23
1,2,3,4,7,8-HxCDF	8.13	7.43	2576	8.59	9.92	8.54
1,2,3,6,7,8-HxCDF	1.88	1.72	992	3.31	3.05	2.62
1,2,3,7,8,9-HxCDF	<0.08	0.00	1009	3.37	2.23	1.92
2,3,4,6,7,8-HxCDF	<0.08	0.00	1.0	0.00	0.00	0.00
1,2,3,4,6,7,8-HpCDF	2.43	2.22	386	1.29	1.25	1.08
1,2,3,4,7,8,9-HpCDF	<0.10	0.00	38.6	0.13	0.06	0.05
OCDF	3.67	3.35	31.6	0.11	0.37	0.32
$\sum$PCDD/F$_{congener}$	**109**	**100**	**29980**	**100**	**116**	**100**
Σ TCDD	0.2	0.20	5.7	0.02	0.3	0.21
Σ PeCDD	<0.08	0.00	31	0.10	1.0	0.72
Σ HxCDD	3.4	2.81	104	0.35	3.1	2.26
Σ HpCDD	7.7	6.35	210	0.70	2.5	1.80
Σ OCDD	25.6	20.98	285	0.95	3.3	2.41
Σ TCDF	8.0	6.56	161	0.54	14	10.56
Σ PeCDF	60.7	49.79	24206	80.58	94	68.40
Σ HxCDF	10.0	8.24	4578	15.24	17	12.32
Σ HpCDF	2.5	2.06	425	1.42	1.4	1.04
Σ OCDF	3.7	3.01	32	0.11	0.4	0.27
ΣPCDD/F$_{homologues}$	**122**	**100**	**30038**	**100**	**137**	**100**
% of $\sum$PCDD/F$_{congener}$ in ΣPCDD/F$_{homologues}$		90		99		85

Table 9 Means of PCDD/Fs in Deer in 2003 (pg/g, lipid-basis)

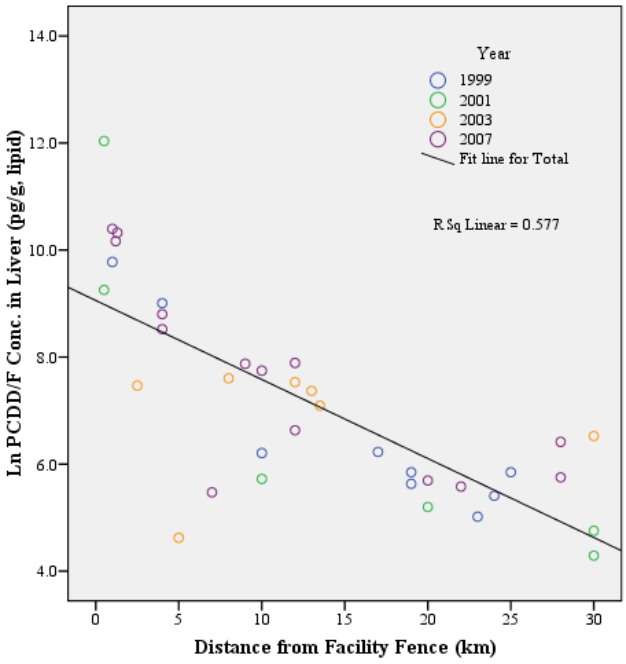
	Muscle (n=7)		Liver (n=7)		Fat (n=7)	
	Conc.	**%**	**Conc.**	**%**	**Conc.**	**%**
Lipid content		0.88		4.07		63
2,3,7,8-TCDD	<0.12	0.00	2.96	0.23	0.18	0.70
1,2,3,7,8-PeCDD	<0.11	0.00	28.18	2.15	1.51	5.73
1,2,3,4,7,8-HxCDD	<0.14	0.00	40.84	3.11	1.74	6.60
1,2,3,6,7,8-HxCDD	<0.14	0.00	55.12	4.20	2.77	10.49
1,2,3,7,8,9-HxCDD	<0.14	0.00	21.58	1.64	0.13	0.48
1,2,3,4,6,7,8-HpCDD	14.69	20.38	257.4	19.60	3.39	12.86
OCDD	52.09	72.26	225.2	17.15	4.50	17.05
2,3,7,8-TCDF	2.73	3.79	6.63	0.51	1.22	4.61
1,2,3,7,8-PeCDF	<0.10	0.00	<0.11	0.00	0.16	0.61
2,3,4,7,8-PeCDF	0.91	1.26	376.2	28.65	7.58	28.73
1,2,3,4,7,8-HxCDF	1.67	2.32	84.00	6.40	1.83	6.93
1,2,3,6,7,8-HxCDF	<0.09	0.00	69.30	5.28	0.65	2.45
1,2,3,7,8,9-HxCDF	<0.09	0.00	52.92	4.03	0.31	1.18
2,3,4,6,7,8-HxCDF	<0.09	0.00	<0.08	0.00	<0.18	0.00
1,2,3,4,6,7,8-HpCDF	<0.13	0.00	79.25	6.04	0.34	1.28
1,2,3,4,7,8,9-HpCDF	<0.13	0.00	3.72	0.28	<0.35	0.00
OCDF	<0.19	0.00	9.82	0.75	0.08	0.31
$\sum$PCDD/F$_{congener}$	**72**	**100**	**1313**	**100**	**26**	**100**
Σ TCDD	<0.11	0.00	3.84	0.29	0.36	1.20
Σ PeCDD	<0.11	0.00	28.18	2.13	1.91	6.45
Σ HxCDD	24.62	30.01	261.1	19.75	3.89	13.11
Σ HpCDD	<0.16	0.00	120.9	9.14	5.49	18.53
Σ OCDD	52.09	63.51	225.2	17.03	4.50	15.18
Σ TCDF	2.73	3.33	6.63	0.50	1.31	4.41
Σ PeCDF	0.91	1.11	376.8	28.50	8.78	29.61
Σ HxCDF	1.67	2.04	206.8	15.64	2.97	10.00
Σ HpCDF	<0.13	0.00	83.00	6.28	0.36	1.23
Σ OCDF	<0.19	0.00	9.82	0.74	0.08	0.28
ΣPCDD/F$_{homologues}$	**82**	**100**	**1322**	**100**	**30**	**100**
% of $\sum$PCDD/F$_{congener}$ in ΣPCDD/F$_{homologues}$		87.9		99.3		89.0

Table 10 Means of PCDD/Fs in Deer in 2007 (pg/g, lipid-basis)

	Muscle (n=14)		Liver (n=14)	
	Conc.	**%**	**Conc.**	**%**
Lipid content		1.82		4.58
2,3,7,8-TCDD	0.68	0.95	5.86	0.07
1,2,3,7,8-PeCDD	2.58	3.60	46	0.58
1,2,3,4,7,8-HxCDD	1.59	2.22	67	0.84
1,2,3,6,7,8-HxCDD	3.37	4.71	72	0.91
1,2,3,7,8,9-HxCDD	0.10	0.15	25	0.32
1,2,3,4,6,7,8-HpCDD	6.04	8.43	362	4.57
OCDD	8.26	11.5	413	5.21
2,3,7,8-TCDF	0.42	0.59	14	0.18
1,2,3,7,8-PeCDF	<0.05	0.00	3.67	0.05
2,3,4,7,8-PeCDF	35	49	4978	63
1,2,3,4,7,8-HxCDF	5.65	7.88	847	11
1,2,3,6,7,8-HxCDF	1.26	1.75	350	4.42
1,2,3,7,8,9-HxCDF	<0.05	0.00	1.17	0.01
2,3,4,6,7,8-HxCDF	3.31	4.61	365	4.61
1,2,3,4,6,7,8-HpCDF	1.86	2.60	246	3.10
1,2,3,4,7,8,9-HpCDF	<0.05	0.00	21	0.26
OCDF	1.33	1.85	104	1.31
$\sum$PCDD/F$_{congener}$	**72**	**100**	**7921**	**100**
Σ TCDD	<0.05	0.00	4.91	0.06
Σ PeCDD	1.34	2.25	46	0.58
Σ HxCDD	2.32	3.90	165	2.06
Σ HpCDD	4.06	6.81	365	4.56
Σ OCDD	7.21	12	413	5.16
Σ TCDF	0.12	0.20	23	0.29
Σ PeCDF	35	58	4992	62
Σ HxCDF	7.72	13	1574	20
Σ HpCDF	0.71	1.19	277	3.46
Σ OCDF	1.33	2.23	142	1.77
ΣPCDD/F$_{homologues}$	**60**	**100**	**8002**	**100**
% of $\sum$PCDD/F$_{congener}$ in ΣPCDD/F$_{homologues}$		120		99

Table 11 ΣPCDD/F Levels in Deer vs. Distance to Facility Fence

Sample #	Distance (km)	Liver (pg/g, lipid-basis)	Muscle (pg/g, lipid-basis)
2007			
1	1	32744	148
2	1.2	25957	222
3	1.3	30434	129
4	4	6640	89
5	4	5021	70
6	7	238	13
7	9	2628	99
8	10	2313	48
9	12	2673	55
10	12	760	20
11	20	297	28
12	22	265	30
13	28	611	10
14	28	315	42
2003			
1	2.5	1748	50
2	5	102	non-detected
3	8	2006	236
4	12	1867	28
5	13	1585	142
6	13.5	1203	27
7	30	681	23
2001			
1	0.5	168749	478
2	0.5	10459	145
3	10	306	10
4	20	181	0.00
5	30	116	5.28
6	30	73	19
1999			
1	1.0	17618	98
2	4.0	8157	102
3	10	496	114
4	17	507	47
5	19	347	21
6	19	279	23
7	23	151	17
8	24	223	65
9	25	347	-



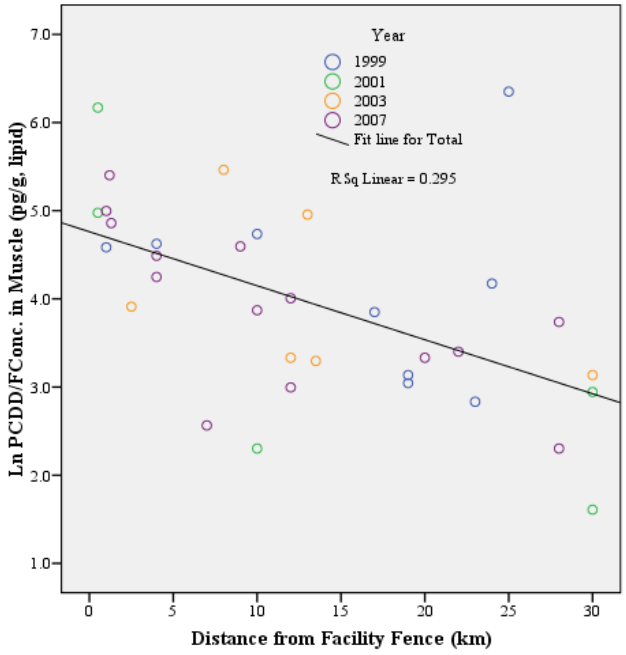
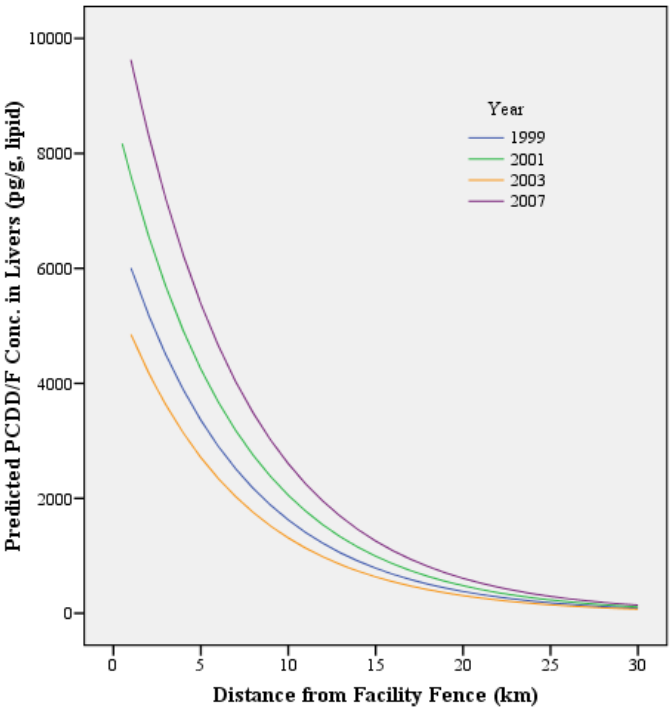


Figure 4 ΣPCDD/F Levels in Deer Liver and Muscle Samples vs. Distance

[t-test: ΣPCDD/F concentrations at distance from the facility fence ≤ 15 vs. >15 km, p <0.05]



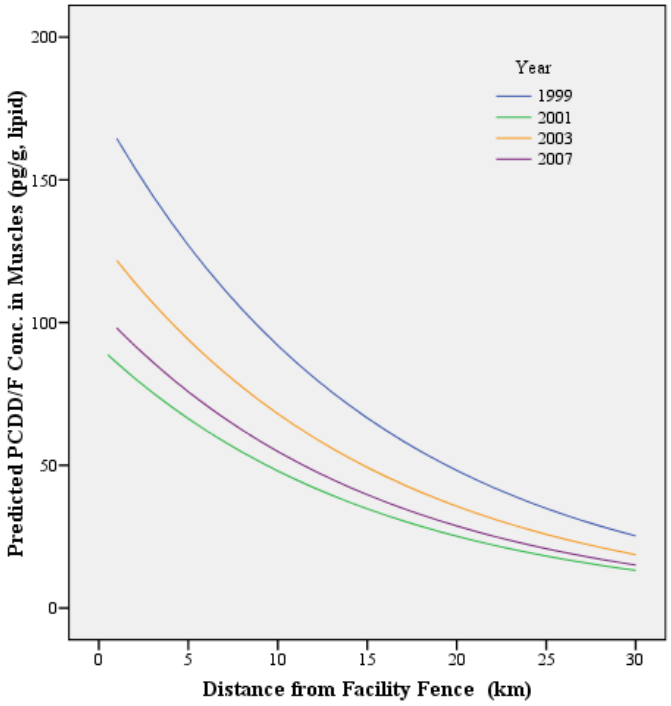


Figure 5 Predicted ΣPCDD/F Levels in Deer Liver and Muscle Samples vs. Distance by Years

Table 12 Statistical Summaries of ΣPCDD/F Levels in Deer 1997 - 2007

(pg/g, lipid-basis)

	Sample #	Mean	Median	SD	Min	Max	95% Confidence	
							Lower	Upper
Liver								
1997	4	74271	44062	94249	555	208404	-75700	224242
1999	9	3125	347	6020	151	17618	1501	7753
2001	6	29980*	244	68107	73	168749	-41493	101454
2003	7	1313	1585	699	102	2006	667	1959
2007	14	7921	2471	12037	238	32744	971	14871
1999 Ref	10	138	37	277	23	921	-61	336
Muscle								
1997	4	165	128	181	non-detected	405	-122	453
1999	9	118	65	174	17	573	-17	252
2001	6	110	15	189	non-detected	478	-89	308
2003	7	72	28	86	non-detected	236	-6.8	151
2007	14	72	52	61	10	222	37	107
1999 Ref	10	61	44	45	13	128	29	94

Note: The levels were based on a sum of 44 congeners in 1997 and a sum of 209 congeners in 1999 – 2007

* high value is due to the highest value detected in one deer close to the facility fence.

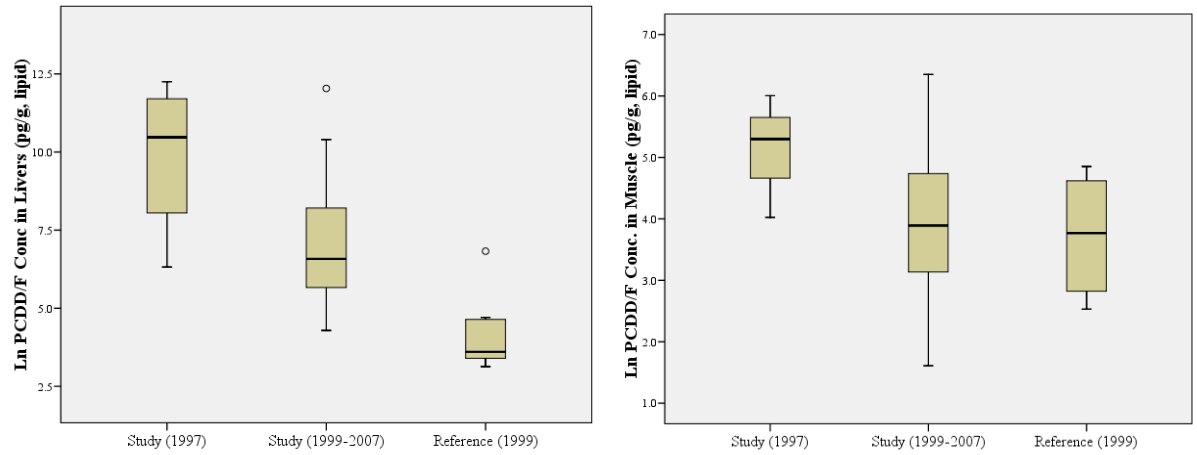


Figure 6 ΣPCDD/F Levels in Study Area and Reference Area

* t – test for ln concentrations in reference area (1999) vs. study area (1999 – 2007): $p < 0.001$ in liver samples and $p = 0.68$ in muscle samples. The levels in study area in 1997 vs. 1999 – 2007: $p = 0.12$ in liver samples and $p = 0.17$ in muscle samples.

2.2.4 Dioxin-like TEQs: 1999–2007

Mean values of ΣPCB-TEQ, ΣPCDD/F-TEQ and ΣTEQ in 1999 in the study area and reference area are presented in Table 13. The concentrations reported here are lipid-basis. In the 1999 study, ΣPCDD/F-TEQ accounted for 68% and 88% of ΣTEQ in muscle samples and liver samples in the study area. The major component of ΣPCDD/F-TEQ was 2,3,4,7,8-PeCDF at the levels of 62 pg/g in muscle samples and 533 pg/g in liver samples. The proportions of 2,3,4,7,8-PeCDF-TEQ in ΣTEQ were 42% in muscle samples and 75% in liver samples. In the reference areas, 1,2,3,6,7,8-HxCDD was a major contributor (81% of ΣTEQ) in muscle samples. 2,3,4,7,8-PeCDF was a major contributor (31% of ΣTEQ) in liver samples, but the level of 2,3,4,7,8-PeCDF-TEQ was very low (non-detected in muscle samples and 1.8 pg/g in liver samples).

ΣPCB-TEQ accounted for 32% to 12% of ΣTEQ in muscle samples and liver samples in the study area. The major component of ΣPCB-TEQ was CB-126-TEQ. The proportions of CB-126-TEQ in ΣTEQ were 28% in muscle samples and 11% in liver samples. The levels of CB-126-TEQ were1.35 pg/g (88% of ΣTEQ) in muscle samples and 84 pg/g (11% of ΣTEQ) in liver samples. In the reference areas, CB-126-TEQ accounted for 11% and 12% of ΣTEQ in muscle samples and liver samples, but the lower levels were measured (0.11 in muscle samples and 0.99 pg/g in liver samples).

Mean values of ΣPCB-TEQ, ΣPCDD/F-TEQ and ΣTEQ in 2001 are presented in Table 14. ΣPCDD/F-TEQ in ΣTEQ accounted for 61% in muscle samples, 82% in liver samples, and 50% in fat samples. The major component of ΣPCDD/F-TEQ was 2,3,4,7,8-PeCDF. The proportions of 2,3,4,7,8-PeCDF-TEQ in ΣTEQ were 55% in muscle samples, 77% in liver samples, and 43% in fat samples. The level of 2,3,4,7,8-PeCDF-TEQ in liver samples significantly increased to 7246 pg/g in 2001. This is due to presence of very high level of 2,3,4,7,8-PeCDF-TEQ (43896 pg/g) in one deer caught at 0.5 km to the facility fence.

ΣPCB-TEQ in ΣTEQ accounted for 39% in muscle samples, 18% in liver samples, and 50% in fat samples. The major component of ΣPCB-TEQ was CB-126-TEQ. The proportions of CB-126-TEQ in ΣTEQ were 32% in muscle samples, 18% in liver samples, and 44% in fat samples. The level of CB-126-TEQ in liver samples significantly increased to 1660 pg/g in 2001. This is due to presence of very high level of CB-126-TEQ (9690 pg/g) in one deer caught at 0.5 km to the facility fence.

Mean values of ΣPCB-TEQ, ΣPCDD/F-TEQ and ΣTEQ in 2003 are presented in Table 15. ΣPCDD/F-TEQ in ΣTEQ accounted for 32% in muscle samples, 83% in liver samples, and 64% in fat samples. The major component of ΣPCDD/F-TEQ was 2,3,4,7,8-PeCDF in liver samples. The proportions of 2,3,4,7,8-PeCDF-TEQ in ΣTEQ were 10% in muscle samples, 52% in liver samples, and 30% in fat samples.

ΣPCB-TEQ accounted for 68% of ΣTEQ in muscle samples, 17% in liver samples, and 36% in fat samples. The major component of ΣPCB-TEQ was CB-126-TEQ. The

proportions of CB-126-TEQ in ΣTEQ were 55% in muscle samples, 16% in liver samples, and 32% in fat samples.

Mean values of ΣPCB-TEQ, ΣPCDD/F-TEQ and ΣTEQ in 2007 are presented in Table 16. ΣPCDD/F-TEQ in ΣTEQ accounted for 81% in muscle samples, and 92 % in liver samples. The major component of ΣPCDD/F-TEQ was 2,3,4,7,8-PeCDF in muscle samples and liver samples. The proportions of 2,3,4,7,8-PeCDF-TEQ in ΣTEQ were 55% in muscle samples, and 79% in liver samples.

ΣPCB-TEQ accounted for 19% of ΣTEQ in muscle samples and 8% in liver samples. The major component of ΣPCB-TEQ was CB-126-TEQ. The proportions of CB-126-TEQ in ΣTEQ were18% in muscle samples, and 8% in liver samples.

Major congeners contributing to ∑TEQ were 2,3,4,7,8-PeCDF and CB-126 in deer collected from the Swan Hills area. These congeners may mark the presence of the emissions from the special waste treatment facility as they have been observed to be a major congener in soil, vegetation, sediment, fish and voles collected near the facility since 1996.

ΣTEQ concentrations at distances to the facility fence in deer tissues collected between 1999 and 2007 are presented in Table 17. A hierarchical multiple regression analysis was conducted to determine the joint effects of distance from the Swan Hills Treatment Centre, the year in which the deer was taken, and the interactions on ΣTEQ concentrations in muscle and liver samples. Due to a skewed distribution, ΣTEQ has been transformed to its natural logarithm prior to analysis. The distance from the facility is the most important factor in estimating ΣTEQ concentrations. ΣTEQ concentrations in muscle samples ($r = - 0.70$, $p < 0.001$) and liver samples ($r = - 0.78$, $p < 0.001$) decreased with increased distance from the facility fence (Figure 7). Predicted ΣTEQ concentrations in liver and muscle samples in 1999 - 2007 have converged by about 15 km from the facility fence (Figure 8). This finding suggests that contamination from the facility has occurred in the ecosystem in vicinity of the facility.

The statistical summaries of ΣTEQ levels between 1997 and 2007 are presented in Table 18. ΣTEQ levels in liver and muscle samples varied significantly from year to year. In particular, ΣTEQ levels in liver and muscle samples in 2007 was higher than the levels in other years after controlling distance of the catch from the facility (Figure 8). In 2007, five deer were taken within 5 km radius of the facility fence. The ΣTEQ levels in liver samples were higher in these five deer. If compared to the mean levels (Table 18), the mean of ΣTEQ concentrations (9455 pg/g) was higher in liver samples in 2001 than those in 1999, 2003 and 2007, but the median levels was low (39 pg/g). The higher mean level resulted from presence of the very high level of ΣTEQ in one deer taken 0.5 km from the facility fence (Figure 7).

Overall, ΣTEQ levels in liver samples and muscle samples in 1999-2007 were not statistically changed as compared to the levels in 1997 ($p > 0.05$), and the levels were

still significantly higher than those in deer collected from the reference area ($p < 0.001$) (Figure 9).

Table 13 Means of ΣTEQ in Deer in 1999 (pg/g, lipid-basis)

	Study Area				Reference Area			
	Muscle		Liver		Muscle		Liver	
	Conc.	%	Conc.	%	Conc.	%	Conc.	%
CB-77	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0
CB-81	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0
CB-126	1.35	88	84	99	0.11	60	0.99	94
CB-169	0.13	8.6	0.96	1.1	0.07	36	0.06	6.0
CB-105	0.01	0.7	0.02	0.0	0.00	0.0	0.00	0.0
CB-114	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0
CB-118	0.02	1.5	0.05	0.1	0.01	2.2	0.00	0.0
CB-123	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0
CB-156	0.01	0.6	0.02	0.0	0.00	0.0	0.00	0.0
CB-157	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0
CB-167	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0
CB-189	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0
2,3,7,8-TCDD	0.00	0.0	2.23	0.3	0.00	0.0	0.00	0.0
1,2,3,7,8-PeCDD	0.00	0.0	29	4.5	0.00	0.0	2.16	31
1,2,3,4,7,8-HxCDD	0.00	0.0	4.42	0.7	0.00	0.0	0.46	6.5
1,2,3,6,7,8-HxCDD	0.89	28	7.95	1.2	0.65	81	1.03	15
1,2,3,7,8,9-HxCDD	0.00	0.0	2.94	0.5	0.00	0.0	0.29	4.1
1,2,3,4,6,7,8-HpCDD	0.06	1.8	2.58	0.4	0.08	9.8	0.41	5.9
OCDD	0.03	0.8	0.09	0.0	0.01	1.4	0.02	0.2
2,3,7,8-TCDF	0.10	3.1	1.75	0.3	0.00	0.0	0.00	0.0
1,2,3,7,8-PeCDF	0.00	0.0	0.13	0.0	0.00	0.0	0.00	0.0
2,3,4,7,8-PeCDF	2.00	62	553	85	0.00	0.0	1.8	26
1,2,3,4,7,8-HxCDF	0.09	2.8	22	3.4	0.00	0.0	0.30	4.2
1,2,3,6,7,8-HxCDF	0.00	0.0	12	1.8	0.00	0.0	0.26	3.8
1,2,3,7,8,9-HxCDF	0.00	0.0	9.6	1.5	0.03	3.4	0.20	2.8
2,3,4,6,7,8-HxCDF	0.00	0.0	0.05	0.0	0.00	0.0	0.00	0.0
1,2,3,4,6,7,8-HpCDF	0.05	1.4	0.66	0.1	0.04	4.7	0.06	0.8
1,2,3,4,7,8,9-HpCDF	0.00	0.0	0.07	0.0	0.00	0.0	0.01	0.1
OCDF	0.00	0.0	0.01	0.0	0.00	0.1	0.00	0.0
ΣPCB-TEQ	1.5		85		0.19		1.05	
ΣPCDD/F-TEQ	3.2		648		0.81		7.00	
Σ TEQ	**4.7**		**733**		**1.00**		**8.05**	
% of ∑PCB-TEQ in ∑TEQ		32		12		20		13
% of ∑PCDD/F-TEQ in ∑TEQ		68		88		80		87
% of 2,3,4,7,8-PeCDF in ∑PCDD/F-TEQ		62		85		0.0		26
% of 2,3,4,7,8-PeCDF in Σ TEQ		42		75		0.0		22
% of PCB-126 in ∑PCBs-TEQ		88		99		60		94
% of PCB-126 in ΣTEQ		28		11		11		12

Values shown for each congener are adjusted by the individual congener-specific TEF factors.

Table 14 Means of ΣTEQ in Deer in 2001 (pg/g, lipid-basis)

	Muscle		Liver		Fat	
	Conc.	%	Conc.	%	Conc.	%
CB-77	0.03	0.2	0.01	0.0	0.00	0.0
CB-81	0.00	0.0	0.04	0.0	0.00	0.0
CB-126	9.7	83	1660	99	24	89
CB-169	1.3	11	22	1.3	1.8	6.7
CB-105	0.11	1.0	0.12	0.0	0.18	0.7
CB-114	0.01	0.1	0.01	0.0	0.02	0.1
CB-118	0.28	2.4	0.39	0.0	0.47	1.7
CB-123	0.00	0.0	0.00	0.0	0.00	0.0
CB-156	0.16	1.4	0.34	0.0	0.30	1.1
CB-157	0.03	0.3	0.08	0.0	0.05	0.2
CB-167	0.01	0.1	0.05	0.0	0.03	0.1
CB-189	0.02	0.2	0.03	0.0	0.03	0.1
2,3,7,8-TCDD	0.00	0.0	5.7	0.1	0.29	1.1
1,2,3,7,8-PeCDD	0.00	0.0	31	0.4	0.92	3.4
1,2,3,4,7,8-HxCDD	0.00	0.0	4.6	0.1	0.08	0.3
1,2,3,6,7,8-HxCDD	0.00	0.0	4.8	0.1	0.13	0.5
1,2,3,7,8,9-HxCDD	0.00	0.0	1.0	0.0	0.03	0.1
1,2,3,4,6,7,8-HpCDD	0.04	0.2	2.1	0.0	0.02	0.1
OCDD	0.01	0.0	0.09	0.0	0.00	0.0
2,3,7,8-TCDF	0.80	4.4	15	0.2	1.1	4.0
1,2,3,7,8-PeCDF	0.04	0.2	0.26	0.0	0.09	0.3
2,3,4,7,8-PeCDF	16	90	7246	93	23	85
1,2,3,4,7,8-HxCDF	0.81	4.5	258	3.3	0.99	3.6
1,2,3,6,7,8-HxCDF	0.19	1.0	99	1.3	0.30	1.1
1,2,3,7,8,9-HxCDF	0.00	0.0	101	1.3	0.22	0.8
2,3,4,6,7,8-HxCDF	0.00	0.0	0.10	0.0	0.00	0.0
1,2,3,4,6,7,8-HpCDF	0.02	0.1	3.86	0.0	0.01	0.0
1,2,3,4,7,8,9-HpCDF	0.00	0.0	0.39	0.0	0.00	0.0
OCDF	0.00	0.0	0.01	0.0	0.00	0.0
ΣPCBs TEQ	12		1683		27	
ΣPCDDs/Fs TEQ	18		7773		27	
Σ TEQ	**30**		**9456**		**54**	
% of ∑PCB-TEQ in ∑TEQ		39		18		50
% of ∑PCDD/F-TEQ in ∑TEQ		61		82		50
% of 2,3,4,7,8-PeCDF in ∑PCDD/F-TEQ		90		93		85
% of 2,3,4,7,8-PeCDF in Σ TEQ		55		77		43
% of PCB-126 in ∑PCB-TEQ		83		99		89
% of PCB-126 in Σ TEQ		32		18		44

Values shown for each congener are adjusted by the individual congener-specific TEF factors.

Table 15 Means of ΣTEQ in Deer in 2003 (pg/g, lipid-basis)

	Muscle		Liver		Fat	
	Conc.	%	Conc.	%	Conc.	%
CB-77	0.01	0.4	0.03	0.1	0.00	0.0
CB-81	0.00	0.1	0.01	0.0	0.00	0.0
CB-126	1.48	81	35	98	2.00	87
CB-169	0.24	13	0.43	1.2	0.22	8.0
CB-105	0.02	1.1	0.06	0.2	0.02	0.6
CB-114	0.00	0.1	0.00	0.0	0.00	0.0
CB-118	0.05	2.9	0.14	0.4	0.05	1.8
CB-123	0.00	0.0	0.00	0.0	0.00	0.0
CB-156	0.01	0.4	0.02	0.1	0.02	0.6
CB-157	0.01	0.4	0.01	0.0	0.00	0.0
CB-167	0.00	0.0	0.00	0.0	0.00	0.0
CB-189	0.00	0.0	0.00	0.0	0.00	0.0
2,3,7,8-TCDD	0.00	0.0	3.0	1.6	0.18	3.8
1,2,3,7,8-PeCDD	0.00	0.0	28	16	1.5	31
1,2,3,4,7,8-HxCDD	0.00	0.0	4.1	2.3	0.17	3.6
1,2,3,6,7,8-HxCDD	0.00	0.0	5.5	3.1	0.28	5.7
1,2,3,7,8,9-HxCDD	0.00	0.0	2.2	1.2	0.01	0.3
1,2,3,4,6,7,8-HpCDD	0.15	17	2.6	1.4	0.03	0.7
OCDD	0.02	1.8	0.07	0.0	0.00	0.0
2,3,7,8-TCDF	0.27	31	0.66	0.4	0.12	2.5
1,2,3,7,8-PeCDF	0.00	0.0	0.00	0.0	0.00	0.1
2,3,4,7,8-PeCDF	0.27	31	113	63	2.0	47
1,2,3,4,7,8-HxCDF	0.17	19	8.0	4.7	0.18	3.7
1,2,3,6,7,8-HxCDF	0.00	0.0	7.0	3.8	0.06	1.3
1,2,3,7,8,9-HxCDF	0.00	0.0	5.0	2.9	0.03	0.6
2,3,4,6,7,8-HxCDF	0.00	0.0	0.00	0.0	0.00	0.0
1,2,3,4,6,7,8-HpCDF	0.00	0.0	0.8	0.4	0.00	0.1
1,2,3,4,7,8,9-HpCDF	0.00	0.0	0.04	0.0	0.00	0.0
OCDF	0.00	0.0	0.00	0.0	0.00	0.0
ΣPCBs TEQ	1.82		36		2.31	
ΣPCDD/F TEQ	0.88		180		4.56	
Σ TEQ	**2.69**		**216**		**6.87**	
% of ∑PCB-TEQ in ∑TEQ		68		17		34
% of ∑PCDD/F-TEQ in ∑TEQ		32		83		66
% of 2,3,4,7,8-PeCDF in ∑PCDD/F-TEQ		31		63		43
% of 2,3,4,7,8-PeCDF in Σ TEQ		10		52		29
% of PCB-126 in ∑PCB-TEQ		81		98		87
% of PCB-126 in Σ TEQ		55		16		29

Values shown for each congener are adjusted by the individual congener-specific TEF factors.

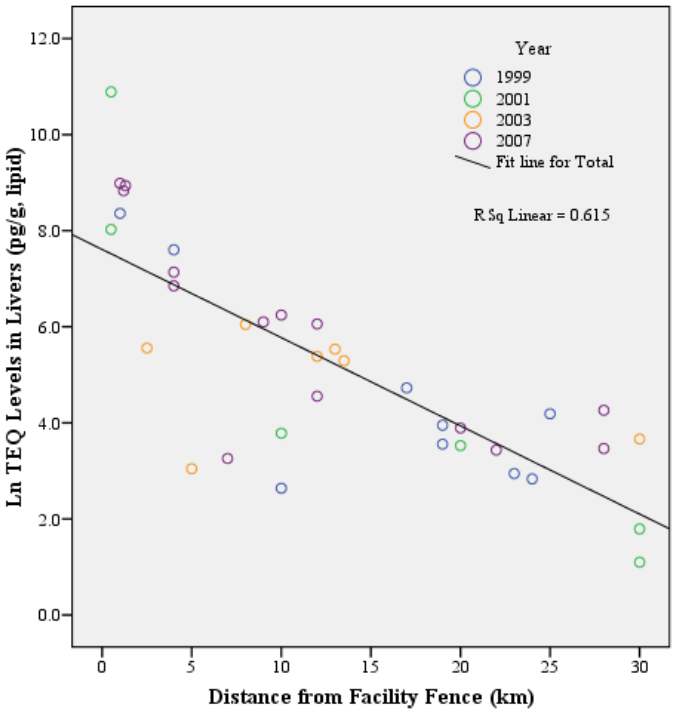
Table 16 Means of ΣTEQ in Deer in 2007 (pg/g, lipid-basis)

	Muscle		Liver	
	Conc.	%	Conc.	%
CB-77	0.00	0.0	0.00	0.0
CB-81	0.00	0.0	0.00	0.0
CB-126	3.48	95	156	99.9
CB-169	0.00	0.0	0.00	0.0
CB-105	0.02	0.4	0.02	0.0
CB-114	0.00	0.1	0.00	0.0
CB-118	0.05	1.3	0.05	0.0
CB-123	0.00	0.0	0.00	0.0
CB-156	0.09	2.5	0.14	0.1
CB-157	0.00	0.0	0.00	0.0
CB-167	0.00	0.1	0.01	0.0
CB-189	0.01	0.4	0.01	0.0
2,3,7,8-TCDD	0.68	4.4	5.9	0.3
1,2,3,7,8-PeCDD	2.58	17	46	2.7
1,2,3,4,7,8-HxCDD	0.16	1.0	6.7	0.4
1,2,3,6,7,8-HxCDD	0.34	2.2	7.2	0.4
1,2,3,7,8,9-HxCDD	0.01	0.1	2.5	0.1
1,2,3,4,6,7,8-HpCDD	0.06	0.4	3.6	0.2
OCDD	0.00	0.0	0.12	0.0
2,3,7,8-TCDF	0.04	0.3	1.41	0.1
1,2,3,7,8-PeCDF	0.00	0.0	0.11	0.0
2,3,4,7,8-PeCDF	11	68	1493	87
1,2,3,4,7,8-HxCDF	0.56	3.6	85	4.9
1,2,3,6,7,8-HxCDF	0.13	0.8	35	2.0
1,2,3,7,8,9-HxCDF	0.00	0.0	0.12	0.0
2,3,4,6,7,8-HxCDF	0.33	2.1	37	2.1
1,2,3,4,6,7,8-HpCDF	0.02	0.1	2.46	0.1
1,2,3,4,7,8,9-HpCDF	0.00	0.0	0.21	0.0
OCDF	0.00	0.0	0.03	0.0
ΣPCBs TEQ	3.7		156	
ΣPCDD/F TEQ	15.9		1726	
Σ TEQ	**19.6**		**1882**	
% of ∑PCB-TEQ in ∑TEQ		19		8
% of ∑PCDD/F-TEQ in ∑TEQ		81		92
% of 2,3,4,7,8-PeCDF in ∑PCDD/F-TEQ		69		87
% of 2,3,4,7,8-PeCDF in Σ TEQ		56		79
% of PCB-126 in ∑PCB-TEQ		95		100
% of PCB-126 in Σ TEQ		18		8

Values shown for each congener are adjusted by the individual congener-specific TEF factors.

Table 17 ΣTEQ Levels in Deer vs. Distance to Facility Fence

Sample #	Distance (km)	Liver (pg/g, lipid-basis)	Muscle (pg/g, lipid-basis)
2007			
1	1	7993	37
2	1.2	6844	71
3	1.3	7617	34
4	4	1258	22
5	4	945	18
6	7	26	1.4
7	9	447	23
8	10	515	16
9	12	428	15
10	12	95	3.0
11	20	49	3.5
12	22	31	9.5
13	28	71	0.9
14	28	32	13
2003			
1	2.5	259	3.9
2	5	21	3.6
3	8	423	3.9
4	12	218	2.2
5	13	253	1.8
6	13.5	199	2.5
7	30	39	0.9
2001			
1	0.5	53586	135
2	0.5	3059	41
3	10	44	1.0
4	20	34	1.4
5	30	6	1.3
6	30	3	0.01
1999			
1	1.0	4274	13
2	4.0	2004	17
3	10	14	3.4
4	17	113	2.4
5	19	52	1.5
6	19	35	1.5
7	23	19	1.0
8	24	17	0.6
9	25	66	2.5



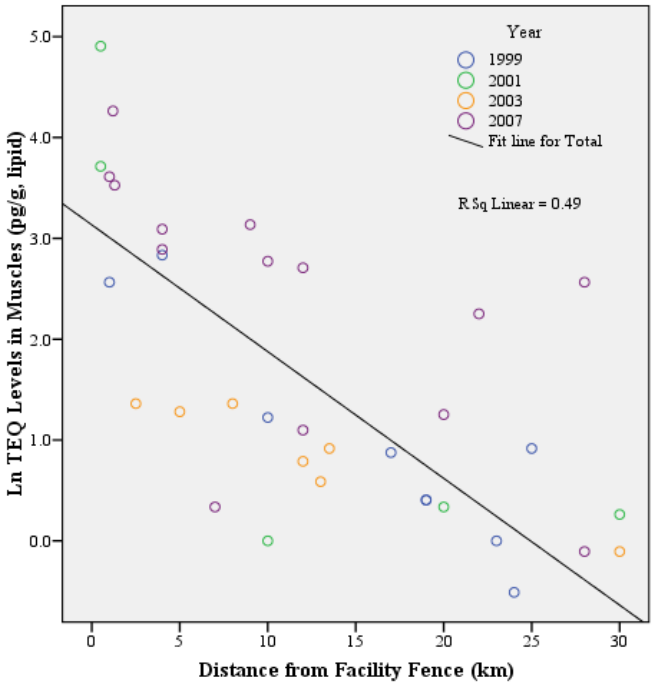
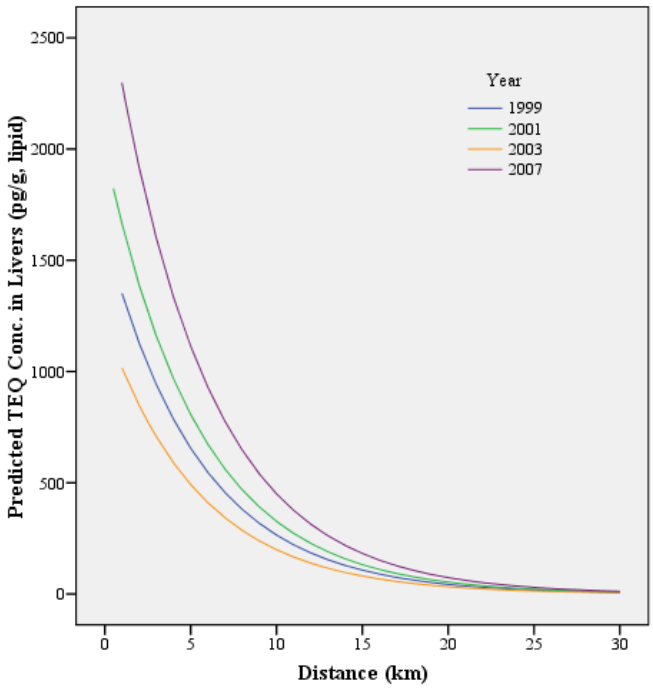


Figure 7 Dioxin-like TEQ Levels in Deer Liver and Muscle Samples vs. Distance

[t-test: ΣPCDD/F concentrations at distance from the facility fence ≤ 15 vs. >15 km, p <0.05]



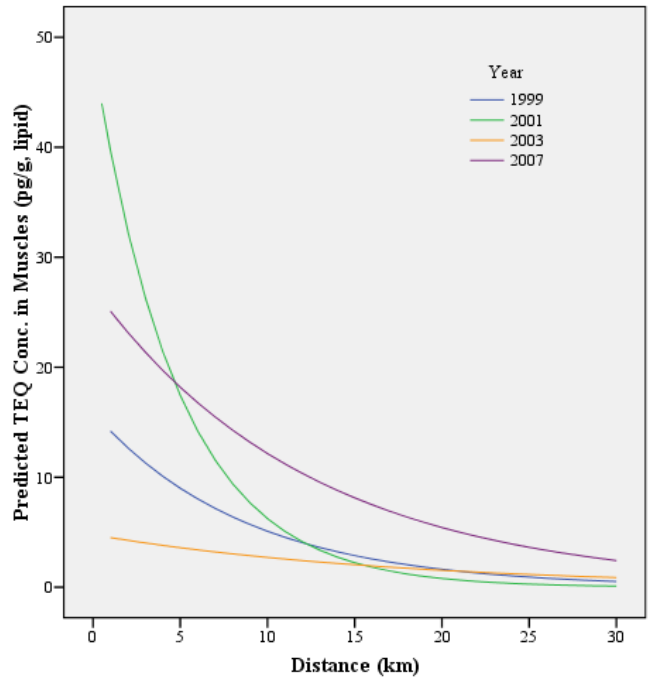


Figure 8 Predicted TEQ Levels in Deer Liver and Muscle Samples vs. Distance by Years

Table 18 Statistical Summaries of ΣTEQ Levels in Deer (pg/g, lipid), 1997 - 2007

	Sample #	Mean	Median	SD	Min	Max	95% Confidence	
							Lower	Upper
Liver								
1997	4	12467	8880	13692	72	32037	-9320	34254
1999	9	733	52	1477	14	4274	-403	1869
2001	6	9455*	39	21653	3.0	53586	13268	32179
2003	7	216	218	150	21	423	78	354
2007	14	1882	438	3067	9.0	7994	111	3653
1999 Ref	10	8.0	1.3	18	0.9	58	-4.5	21
Muscle								
1997	4	788	76	1476	nd	3000	-1561	3137
1999	9	4.8	2.4	6.0	0.6	17	0.2	9.3
2001	6	30	1.3	54	0.01	135	-27	87
2003	7	2.7	2.5	1.2	0.9	3.9	1.6	3.8
2007	14	19	16	19	0.9	71	8.3	30
1999 Ref	10	1.0	1.0	0.5	0.3	2.0	0.6	1.4

Note: The levels were based on a sum of 44 congeners in 1997 and a sum of 209 congeners in 1999 – 2007

* high value is due to the highest value detected in one deer close to the facility fence.

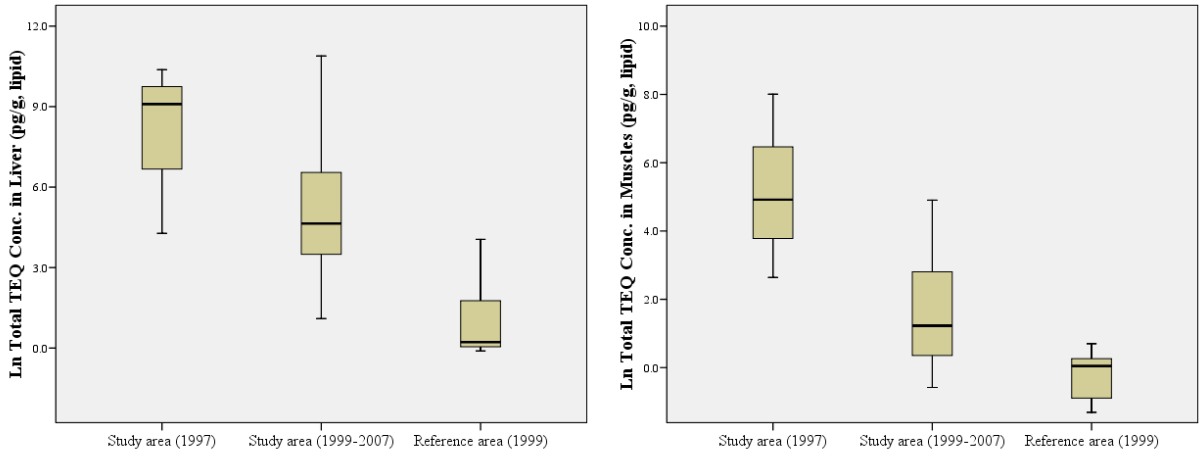


Figure 9 Dioxin-like TEQ Levels in Study Area and Reference Area

* t – test for ln concentrations in reference area (1999) vs. study area (1999 – 2007): $p < 0.001$ in liver samples and muscle samples. The levels in study area in 1997 vs. 1999 – 2007: $p = 0.11$ in liver samples and $p = 0.13$ in muscle samples.

2.3 Summary

As compared to the contaminant levels in deer in1997 in the area of the Swan Hills Treatment Center, the ΣPCB levels in liver and muscle samples were statistically significantly decreased in1999 – 2009. The levels of ΣPCDD/Fs and Σdioxin-TEQs were not statistically significantly changed in all types samples in 1999 – 2007.

As compared to the contaminant levels in deer in 1999 in the reference areas, the levels of ΣPCB, ΣPCDD/F and Σdioxin-TEQs were still higher in all types of samples except for ΣPCDD/F levels in muscle samples.

The inverse relationship between ΣPCB, ΣPCDD/F and Σdioxin-TEQ concentrations vs. distance were observed in deer collected at a distance of within 15 km radius of the facility fence in 1999 - 2007. Similar to the 1997 results, the contaminant concentrations in all the samples decreased with distance from the facility fence. This inverse relationship suggests that the contamination is limited to the immediate vicinity of the facility.

Distribution patterns of ∑PCDD/Fs, ∑PCBs and ∑dioxin-TEQ in the 1999 - 2007 studies were consistent with those observed in the 1997 study and the annual monitoring programs conducted by the company. Similarity of distribution patterns of the contaminants in various media indicates that the sources of contamination could come from the facility.

3. Fish Tissue Monitoring

3.1 Materials and Methods

3.1.1 Field Collection

1997

Field collection was carried out during June and July, 1997. A total of 16 brook trout were collected from Chrystina Lake, about 1.5 km northeast of the facility, with an average age of 2.0 years (a range of 1.0 to 3.0) and average weight of 112 g (a range of 60 to 229). Seventeen northern pike were collected from Roche Lake, about 20 km east of the facility, with an average age of 4.4 years (a range of 3.0 to 6.0) and average weight of 1.2 kg (a range of 0.8 to 2.3). A total of 32 northern pike were collected from Chip Lake (a reference lake) with an average age of 5.4 years (a range of 4 to 7) and average weight of 1.1 kg (a range of 0.6 to 1.5). Both muscle and liver were analyzed. For Chrystina and Roche Lake samples, each composite sample was formed from four (or five) fish from a single species from the same lake with approximately the same length and weight. For Chip Lake samples, each composite sample was formed from 6 or 7 fish. A total of 26 composite samples were formed. All specimens were kept frozen at -20^{o} C prior to laboratory analysis.

2000

Field collection was carried out during August and September, 2000. A total of 12 brook trout were collected netting from Chrystina Lake, about 1.5 km northeast of the facility, with an average total length of 295 mm and average weight of 318 g. All samples were kept frozen at -20^{o} C prior to contaminants analysis.

2003

Field collection was carried out during August, 2003. A total of 12 brook trout were collected via netting from Chrystina Lake, about 1.5 km northeast of the facility, with an average total length of 320 mm and average weight of 390 g. All samples were kept frozen at -20^{o} C prior to contaminants analysis.

2007

Field collection was carried out during October and December, 2007. A total of 15 brook trout were collected via icing fish from Chrystina Lake, about 1.5 km northeast of the facility, with an average total length of 335 mm and average weight of 570 g. The weight in six fish was 800 g. All samples were kept frozen at -20^{o} C prior to contaminants analysis.

3.1.2 PCBs and PCDD/Fs Analysis

1997

PCDD/Fs and PCBs determinations for all samples were performed by the MAXXAM Laboratory, Mississauga, Ontario. Analytical methods and QA/QC assurance were described in Environmental Canada EPS 1/RM/23 (1992), Environmental Canada AMD 96-05 (1996) and USEPA Method 1613 (1994). Each sample was homogenized and subsampled for analysis. Prior to the initial extraction, samples were fortified with fifteen $^{13}C_{12}$-labeled PCDD/Fs with the exception of OCDF, and eight $^{13}C_{12}$-labeled PCBs. Samples were digested overnight in concentrated hydrochloric acid and then extracted with 50/50 dichloromethane/hexane for one hour. This extraction was repeated several times. Lipid content was determined gravimetrically from the remaining extract. The extracts were subjected to an acid/base silica cleanup, reconcentrated and split into two equal portions by weight. One portion, for PCDD/F analysis, was cleaned up on alumina following the standard operating procedure for PCDD/PCDFs. The PCB portion was cleaned up on a modified alumina column. Extracts were analyzed separately for PCBs and PCDD/Fs on an Autospec Ultima High Resolution Mass Spectrometer, interfaced with a Hewlett Packard Gas Chromatograph. PCBs were separated at EI 8,000 mode and PCDD/Fs at EI 10,000 mode. Fused silica capillary columns (60 meter, 0.25 mm ID, 0.25 μm film thickness) were used for determining PCDD/Fs and PCB congeners, respectively. Injector temperature was 265 ^{o}C. The total time of the GC run was 50 min. Congeners were detected in the selected ion monitoring (SIM) mode.

2000, 2003 and 2007

PCDD/Fs and PCBs determinations for all samples were performed by the Fisheries and Oceans Regional Dioxin Laboratory at the Institute of Ocean Sciences in Sidney, British Columbia. The methodologies used to process the samples, the criteria used for identification and quantification and the quality assurance/quality control protocols followed are described in detail elsewhere (Ikonomou et al. 2001). From each sample four aliquots were collected from the carbon-fibre fractionation, the last part of the sample clean-up process. Fraction-I contained the di-ortho PCBs, fraction-II the mono-ortho PCBs, fraction-III the non-ortho PCBs and fraction-IV the PCDDs and PCDFs. In fractions I to III all the possible 209 PCB congeners were measured with minimum isomeric interference. Analyses of all fractions were conducted by high-resolution gas chromatograph/high-resolution mass spectrometry (HRGC/HRMS). For all analyses the MS was operated at 10000 resolution under positive EI conditions and data were acquired in the Single Ion Monitoring Mode (SIM). The concentrations of identified compounds and their method detection limits (MDLs) were calculated by the internal standard method using mean relative response factors determined from calibration standard runs, made before and after each batch of samples was run. Detection limits

range from 0.01 to 0.12 pg/g for PCDDs/Fs, 0.04 to 0.08 pg/g for non-ortho PCBs, 0.1 pg/g for mono-ortho PCBs and 0.1 to 0.2 pg/g for di-ortho PCBs.

3.1.3 Statistical Analysis

Toxic Equivalency Factors (TEFs) are toxicity potency factors proposed by the World Health Organization (WHO) as a consistent method to evaluate the toxicities of highly variable mixtures of dioxin and dioxin-like compounds. TEF values are not precise. WHO reevaluated the TEFs in 2005 (ver den Berg et al. 2006). The TEFs were assigned to individual dioxin family member compounds and dioxin-like compounds (12 PCB compounds). The mass or concentration of each compound is measured and multiplied by the appropriate potency factor (TEF). The summed toxicity-weighted mass quantity is known as dioxin-Toxic Equivalents (dioxin-TEQ). Calculation of TEQ by using TEF estimates is explained in Section 2.1.3.

The concentrations of PCBs, PCDD/Fs and TEQs were not normally distributed among the samples. For the purpose of statistical analysis, the concentration values were transformed into $\log_{10}$. t-test, correlation analysis and linear regression analysis were performed by using SPSS 15.0 software.

3.2 Results and Discussions

3.2.1 PCBs and PCDD/Fs: 1997

Summary of PCB and PCDD/F levels in all species and locations are presented in Table 19. $\sum PCB_{congener}$ and $\sum PCCD/F_{congener}$ concentrations in brook trout from Chrystina Lake were significantly higher ($p<0.01$) than those in northern pike from Roche and Chip lakes, which did not differ from each other. Under normal circumstance, northern pike, a predator (piscivorous), would be expected to have higher contaminant concentrations than brook trout which feed on planktonic invertebrates (Kidd et al. 1995). Pike with larger size and greater age would also contribute an expectation of higher contaminant loading. Consequently, the lower contaminant values in pike from Roche and Chip lakes indicate very low contaminant background. In contrast, brook trout caught for chemical analysis were smaller and younger. Concentrations of these contaminants were generally one order of magnitude higher in brook trout compared to northern pike. These results are consistent with a localized source of contamination.

Table 19 Means of PCBs and PCDD/Fs in Fish, 1997

Tissue	Muscle			Liver		
Lake	Chrystina	Roche	Chip	Chrystina	Roche	Chip
Fish species	*brook trout*	*northern pike*	*northern pike*	*brook trout*	*northern pike*	*northern pike*
$\sum PCB_{congener*}$ (ng/g, ww) (range)	18[a] (9.7-27)	1.0 (0.3-2.8)	0.25 (0.04-0.7)	70[a] (41-117)	7.8 (1.2-14)	6.4 (3-18)
$\sum PCDD/F_{congener}$ (pg/g, ww) (range)	22[a] (12-30)	0.93 (0.7-1.1)	0.68 (ND-1.2)	227[a] (55-351)	1.2 (ND-2)	7.5 (ND-19)
TEQ (pg/g, ww) ∑ Dioxin-like compounds** (range)	12.4[a] (6-19)	0.24 (0.01-1)	0.004 (ND-0.007)	61[a] (24-107)	3.2 (0.7-5.5)	2.4 (1-7)

a: Difference statistically significant at $p<0.01$

* Sum of 44 individual congener levels. **Sum of ∑CB-TEQ and ∑PCDD/F-TEQ

A wide range of individual PCB congeners was detected. Hexachlorobiphenyl (about 50%) was a prevalent homologue group while di-, tri-, tetra-, octa- and deca-chlorobiphenyls were minor constituents across all species and locations. CB 101, 118, 138, 153, and 180 constituted 50% of $\sum PCB_{congener}$ for brook trout, 60% for pike liver and 70% for pike muscle. The findings also are consistent with the results in the

company's monitoring programs and two other relevant studies in which CB 138, 153 and 180 were found as major contributors in vegetation, soil, spruce needle and snow pack near the facility. These findings are also more consistent with a longer range impact, albeit at a low level, of the contaminants emitted from the facility.

The majority of ∑dioxin-like TEQ in all samples was due to PCBs. The most important contributors were *non-ortho* congeners which accounted for 78% of ∑dioxin-like TEQ in brook trout muscle, 73% in brook trout liver, over 90% in pike with the exception of pike muscle from Chip Lake. CB 169 alone contributed 58% to 70% of ∑dioxin-like TEQ in brook trout liver and muscle, respectively. CB 126 was not detected in northern pike. A major contributor in pike muscle from Chip Lake was CB 118, contributing 53% to ∑dioxin-like TEQ. This finding is comparable to the results of the company's monitoring program in which elevated levels of *non-ortho* PCBs were found in Labrador tea leaves, live moss and soils.

Combustion processes could be the source of the increased environmental levels of the coplanar congeners characterized by 3,3',4,4' substitution such as 169, 126, 77, 105, 156, 157, 170 and 189 (Brown et al. 1995, Kimbrough 1995). The air emission from the facility may contribute to higher levels of *non-ortho* PCBs in brook trout.

2,3,7,8-tetra CDF and 2,3,4,7,8-penta CDF were prevalent in brook trout, accounting for 22% (muscle) to 27% (liver) of ∑dioxin-like TEQ. OCDD and 1,2,3,7,8-penta CDF represented 44% of ∑dioxin-like TEQ in pike muscle from Chip Lake. PCDD/Fs were minor constituents in pike liver and muscle from Roche Lake and pike liver from Chip Lake. 2,3,7,8-TCDD was not detected among species and locations at detection limits of 0.5 pg/g whole weight. The patterns of PCDD/Fs for most incineration sources are likely to include almost every congener (Hallikainen et al. 1997).

2,3,7,8-tetra CDF was observed in northern pike in northern Alberta and Northwest Territories (Muir, et al. 1997, Pastershank et al. 1996, Sanderson et al. 1997). The results from the company's environmental monitoring program showed that the high levels of 2,3,7,8-tetra CDF and 2,3,4,7,8-penta CDF were measured in Labrador tea leaves, live moss, wild game and voles near the facility, with TCDF predominating in tea leaves and live moss and 2,3,4,7,8-penta CDF in wild game and voles. The results are consistent with those from the current study in which the most prominent congeners in brook trout were 2,3,7,8-TCDF and 2,3,4,7,8-penta CDF. The comparable PCB and PCDD/F profiles suggested that the elevated levels of PCBs and PCDD/Fs in brook trout from Chrystina Lake could be attributed to contaminant emissions from the facility.

3.2.2 PCBs: 2000–2007

The mean values of ∑PCBs and their homologues in 2000, 2003 and 2007 are summarized in Table 20 and 21. The concentrations in this section are expressed on a lipid-basis. The data on the concentrations of ∑PCBs, lipid-basis in liver samples in 1997 were not available. For comparison, an average of lipid contents of 4.9% was

derived from the lipid contents between 2000 and 2007 and this was used for calculating the lipid-based concentrations for 1997 data. In 2000 the mean concentrations of ∑PCBs were 7455 ng/g in muscle samples and 2134 ng/g in liver samples. In 2003 the mean concentrations of ∑PCBs were 7906 ng/g in muscle samples and 3381 ng/g in liver samples. In 2007, the mean concentrations of ∑PCBs were 21555 ng/g in muscle samples and 3122 ng/g in liver samples.

In the 2000 study, the dominant PCB congeners were CB153 and CB138, accounting for 13% to 16% of ∑PCBs in muscle and liver samples and 10% to 12% of ∑PCBs in muscle and liver samples, respectively. The concentrations of CB 153 were 967 ng/g in muscle and 332 ng/g in liver samples. The concentrations of CB 138 were 765 ng/g in muscle samples and 253 ng/g in liver samples. In the 2003 study, the dominant PCB congeners were also CB153 and CB138, accounting for 11% of ∑PCBs in muscle and liver samples and 10% of ∑PCBs in muscle and liver samples, respectively. The concentrations of CB 153 were 896 ng/g in muscle samples and 379 ng/g in liver samples. The concentrations of CB 138 were 827 ng/g in muscle samples and 356 ng/g in liver samples. In the 2007 study, the dominant PCB congeners were also CB153 and CB138, accounting for 10% of ∑PCBs in muscle and liver samples. The concentrations of CB 153 were 2149 ng/g in muscle samples and 305 ng/g in liver samples. The concentrations of CB 138 were 2219 ng/g in muscle samples and 320 ng/g in liver samples.

The statistical summaries of ΣPCB levels between 1997 and 2007 are presented in Table 22. The ΣPCB levels in liver and muscle samples of brook trout collected from Chrystina Lake between 2000 and 2007 were not statistically significantly changed as compared to the levels in 1997, but the levels were still significantly higher than those in northern pike collected from the reference lakes in 1997 (Figure 10). The ΣPCB levels in muscle samples of brook trout collected from Chrystina Lake in 2007 were significantly higher than those in 2000 and 2003 ($p < 0.01$) (Figure 11).Brook trout taken in 2007 (mean = 557 grams) were larger than those taken in 2000 (mean = 390) and 2003 (mean = 317 grams) from Chrystina lake. After controlling weight in a multiple regression model, ΣPCB levels in muscle samples still remain high. Thus, size of fish is not an important factor that influences the ΣPCB levels in 2007.

Table 20 Means of PCB Homologues in Fish Muscle Samples 2000-2007

(ng/g, lipid-basis)

	2007	2003	2000
Non-ortho			
di-CBs	9.8	1.8	1.9
tri-CBs	3.8	1.7	47
tetra-CBs	38	13	16
penta-CBs	7.0	2.1	1.6
hexa-CBs	0.3	0.07	0.07
Total	**59**	**19**	**67**
Mono-ortho			
di-CBs	13	0.5	2.2
tri-CBs	84	34	66
tetra-CBs	912	310	476
penta-CBs	2429	775	576
hexa-CBs	269	84	82
hepta-CBs	12	1.4	4.2
Total	**3719**	**1205**	**1206**
Di-ortho			
di-CBs	3.3	0.1	0.4
tri-CBs	16	3.4	8.8
tetra-CBs	461	320	315
penta-CBs	4612	1520	1215
hexa-CBs	7746	3137	2923
hepta-CBs	4249	1412	1460
octa-CBs	657	196	251
nona-CBs	28	12	9.2
deca-CBs	3.9	1.7	0.9
Total	**17776**	**6602**	**6183**
Total CBs	**21555**	**7826**	**7456**
% *non-ortho*	0.3	0.2	0.9
% *mono-ortho*	17.2	15.4	16
% *di-ortho*	82.5	84.4	83

Table 21 Means of PCB Homologues in Fish Liver Samples, 2000-2007

(ng/g, lipid-basis)

	2007	2003	2000
Non-ortho			
di-CBs	1.2	0.5	0.7
tri-CBs	0.5	1.0	1.5
tetra-CBs	2.9	6.4	1.8
penta-CBs	0.5	1.1	0.5
hexa-CBs	0.0	0.04	0.03
Total	**5.2**	**9.1**	**4.5**
Mono-ortho			
di-CBs	1.7	0.3	1.5
tri-CBs	16	15	17
tetra-CBs	142	151	46
penta-CBs	338	374	167
hexa-CBs	40	44	24
hepta-CBs	2.2	0.9	1.1
Total	**540**	**585**	**258**
Di-ortho			
di-CBs	0.2	0.02	0.2
tri-CBs	2.6	1.4	2.9
tetra-CBs	68	124	50
penta-CBs	593	646	306
hexa-CBs	1134	1309	1002
hepta-CBs	654	621	432
octa-CBs	121	82	77
nona-CBs	4.3	3.3	2.1
deca-CBs	0.5	0.6	0.1
Total	**2577**	**2787**	**1872**
Total CBs	**3123**	**3382**	**2133**
% *non-ortho*	0.2	0.3	0.2
% *mono-ortho*	17.3	17.3	12.1
% *di-ortho*	82.5	82.5	87.7

Table 22 Statistical Summaries of ΣPCB Levels in Fish 1997 - 2007

(ng/g, lipid-basis)

	Sample	Mean	Median	SD	Min	Max	95% Confidence Lower	95% Confidence Upper
Liver								
1997	4 (pooled)							
2000	12	1429	1247	678	843	2380	350	2509
2003	11	2134	1765	1152	765	4479	1402	2866
2007	15	3381	3085	2281	770	10458	1859	4385
1997 Ref	9 (pooled)	218	93	301	44	1006	-14	449
Muscle								
1997	4 (pooled)	7584	4869	7129	2505	18093	3760	18927
2000	12	7457	6655	3980	2122	16067	4928	9985
2003	11	7654	5336	8301	1799	29765	2077	13232
2007	15	21554	12512	2317	6880	88811	8698	34411
1997 Ref	9 (pooled)	144	123	136	33	423	39	248

Note: (1) The levels were based on a sum of 44 congeners in 1997 and a sum of 209 congeners in 2000 – 2007. (2) The lipid content (4.9%) for liver samples in 1997 was derived from average values of lipid contents in 2000 – 2007. (3) Fish species in 1997 reference lakes were northern pike.

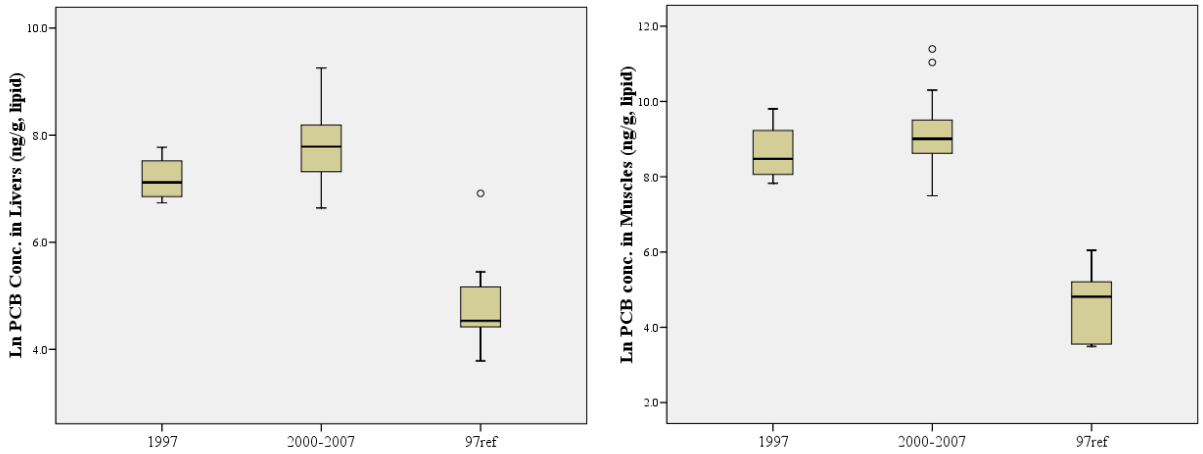


Figure 10 ΣPCB Levels in Brook Trout

t – test for ln concentrations in brook trout in Chrystina Lake 1997 vs. brook trout in 2000 – 2007: $p = 0.07$ in liver samples and $p = 0.41$ in muscle samples. The levels in northern pike in the reference lake in 1997 vs. brook trout in Chrystina Lake in 2000 – 2007: $p < 0.001$ in liver and muscle samples.

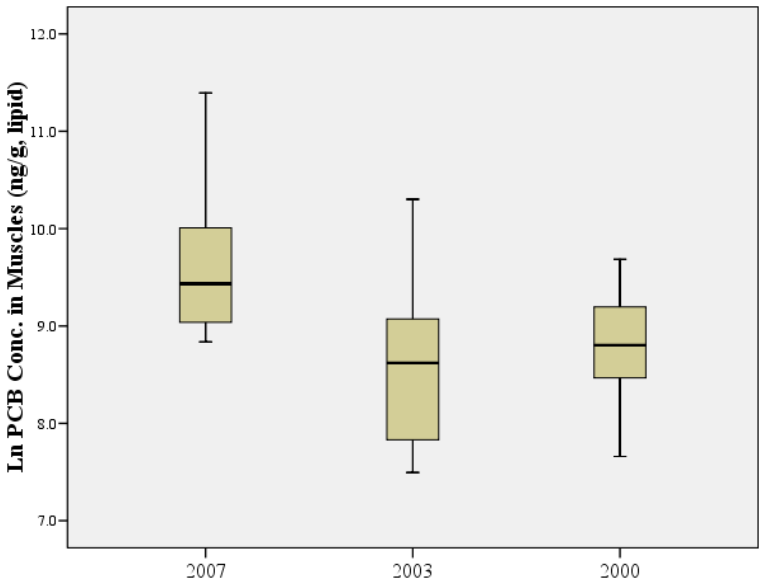


Figure 11 ΣPCB Levels in Brook Trout Muscle Samples in Chrystina Lake, 2000-2007

t – test: the levels in 2007 vs. in 2000 or 2003: $p < 0.01$.

3.2.3 PCDD/Fs: 2000–2007

The mean values of ∑PCDD/Fs are summarized in Table 23 and 24. The concentrations in this section are expressed on a lipid basis. The data on the concentrations of ∑PCBs, lipid basis in liver samples in 1997 were not available. For comparison, average of lipid contents of 4.9% derived from the lipid contents between 2000 and 2007 was used for calculating the lipid-based concentrations for 1997 data. The mean concentrations of ∑PCDD/Fs in muscle were 288 pg/g in 2000, 102 pg/g in 2003, and 581 pg/g in 2007. The mean values of ∑PCDD/Fs in liver samples were 121 pg/g in 2000, 67 pg/g in 2003, and 137 pg/g in 2007.

Table 23 Means of PCDD/Fs in Brook Trout Muscle Samples (pg/g, lipid-basis)

	2007 (n=15)		2003 (n=12)		2000 (n=12)	
	Conc.	**%**	**Conc.**	**%**	**Conc.**	**%**
Lipid content		0.29		0.86		0.42
2,3,7,8-TCDD	<0.06	0.0	< 0.06	0.0	<0.06	0.0
1,2,3,7,8-PeCDD	<0.08	0.0	< 0.08	0.0	<0.08	0.0
1,2,3,4,7,8-HxCDD	<0.10	0.0	< 0.10	0.0	<0.10	0.0
1,2,3,6,7,8-HxCDD	<0.10	0.0	< 0.10	0.0	32	11.12
1,2,3,7,8,9-HxCDD	<0.10	0.0	< 0.10	0.0	<0.10	0.0
1,2,3,4,6,7,8-HpCDD	23	4.0	1.6	1.5	17	5.8
OCDD	119	21	1.6	1.5	129	45
2,3,7,8-TCDF	390	70	94	91	105	36
1,2,3,7,8-PeCDF	<0.06	0.0	< 0.06	0.0	<0.06	0.0
2,3,4,7,8-PeCDF	27	5.0	5.7	5.5	5.7	2.0
1,2,3,4,7,8-HxCDF	<0.08	0.0	< 0.08	0.0	<0.08	0.0
1,2,3,6,7,8-HxCDF	<0.08	0.0	< 0.08	0.0	<0.08	0.0
1,2,3,7,8,9-HxCDF	<0.08	0.0	< 0.08	0.0	<0.08	0.0
2,3,4,6,7,8-HxCDF	<0.08	0.0	< 0.08	0.0	<0.08	0.0
1,2,3,4,6,7,8-HpCDF	<0.10	0.0	< 0.10	0.0	<0.10	0.0
1,2,3,4,7,8,9-HpCDF	<0.10	0.0	< 0.10	0.0	<0.10	0.0
OCDF	<0.12	0.0	< 0.12	0.0	<0.12	0.0
∑PCDD/F$_{congener}$	**559**	**100**	**103**	**100**	**289**	**100**
Σ TCDD	<0.06	0.0	< 0.06	0.0	14	4.0
Σ PeCDD	<0.08	0.0	< 0.08	0.0	<0.08	0.0
Σ HxCDD	4.5	1.0	< 0.10	0.0	39	12
Σ HpCDD	31	5.0	2.6	2.5	28	9.0
Σ OCDD	119	21	1.6	1.5	129	40
Σ TCDF	394	68	95	91	105	33
Σ PeCDF	32	5.0	5.8	5.0	6.0	2.0
Σ HxCDF	<0.10	0.0	< 0.08	0.0	<0.08	0.0
Σ HpCDF	<0.10	0.0	< 0.10	0.0	<0.10	0.0
Σ OCDF	<0.12	0.0	< 0.12	0.0	<0.12	0.0
ΣPCDD/F$_{homologues}$	**581**	**100**	**105**	**100**	**321**	**100**
% of ∑PCDD/F$_{congener}$ in ΣPCDD/F$_{homologues}$		96		98		90

Table 24 Means of PCDD/Fs in Brook Trout Liver Samples (pg/g, lipid-basis)

	2007 (n=15)		2003 (n=12)		2000 (n=12)	
	Conc.	**%**	**Conc.**	**%**	**Conc.**	**%**
Lipid content		3.2		6.2		6.0
2,3,7,8-TCDD	0.2	0.2	0.1	0.2	1.0	0.9
1,2,3,7,8-PeCDD	0.8	0.6	0.1	0.1	1.4	1.2
1,2,3,4,7,8-HxCDD	<0.1	0.0	< 0.11	0.0	1.7	1.4
1,2,3,6,7,8-HxCDD	1.8	1.5	< 0.11	0.0	13	11
1,2,3,7,8,9-HxCDD	<0.1	0.0	< 0.11	0.0	1.6	1.3
1,2,3,4,6,7,8-HpCDD	10	8.0	0.9	1.3	9.5	8.0
OCDD	24	19	0.9	1.3	39	32
2,3,7,8-TCDF	76	60	55	83	35	29
1,2,3,7,8-PeCDF	2.3	2.0	0.3	0.4	1.2	1.0
2,3,4,7,8-PeCDF	11	8.0	8.4	12.5	7.7	6.0
1,2,3,4,7,8-HxCDF	<0.08	0.0	0.6	0.9	<0.12	0.0
1,2,3,6,7,8-HxCDF	<0.08	0.0	< 0.08	0.00	<0.12	0.0
1,2,3,7,8,9-HxCDF	<0.08	0.0	< 0.08	0.00	1.3	1.0
2,3,4,6,7,8-HxCDF	<0.08	0.0	< 0.10	0.00	<0.12	0.0
1,2,3,4,6,7,8-HpCDF	0.3	0.2	0.3	0.3	3.0	2.5
1,2,3,4,7,8,9-HpCDF	<0.1	0.0	< 0.10	0.00	1.8	1.0
OCDF	0.6	0.5	< 0.13	0.00	4.5	3.7
$\sum$PCDD/F$_{congener}$	**127**	**100**	**67**	**100**	**122**	**100**
Σ TCDD	1.0	0.7	17	15	4.6	3.4
Σ PeCDD	0.8	0.5	9.0	7.0	1.4	1.0
Σ HxCDD	3.0	2.2	20	17	19	14
Σ HpCDD	15	11	0.5	0.4	15	11
Σ OCDD	24	17	1.0	0.7	39	29
Σ TCDF	77	57	58	50	36	26
Σ PeCDF	15	11	10	9.0	9.5	7.0
Σ HxCDF	<0.1	0.0	0.6	0.5	1.3	1.0
Σ HpCDF	0.3	0.2	0.3	0.2	4.8	3.6
Σ OCDF	0.6	0.4	< 0.13	0.0	4.5	3.0
ΣPCDD/F$_{homologues}$	**137**	**100**	**116**	**100**	**135**	**100**
% of $\sum$PCDD/F$_{congener}$ ΣPCDD/F$_{homologues}$		62		57		90

In 2000 study, the prevalent congeners were OCDD and 2,3,7,8 TCDF, with OCDD accounting for 45% of $\sum$PCDD/Fs in muscle samples and 31% in liver samples, and with 2,3,7,8 TCDF accounting for 36% of $\sum$PCDD/Fs in muscle samples and 27% in liver samples. In 2003 study, the prevalent congeners were 2,3,7,8 TCDF, accounting for 91% of $\sum$PCDD/Fs in muscle samples and 83% in liver samples. In 2007 study, the prevalent congeners were OCDD and 2,3,7,8 TCDF. OCDD accounted for 21% of $\sum$PCDD/Fs in muscle samples and 19% in liver samples. 2,3,7,8 TCDF accounted for 70% in muscle samples and 60% in liver samples. The pattern of congeners in fish tissue samples was different than in deer tissue samples. The major contributor in deer samples was 2,3,4,7,8-PeCDF.

The statistical summaries of ΣPCDD/F levels between 1997 and 2007 are presented in Table 25. The ΣPCDD/F levels in the liver and muscle samples of brook trout collected from Chrystina Lake between 2000 and 2007 were significantly lower than the levels in 1997 ($p < 0.01$) (Figure 12). The ΣPCDD/F levels in the liver and muscle samples of brook trout collected from Chrystina Lake between 2000 and 2007 were similar to those in fish collected from the reference lakes in 1997 (p: 0.61 – 0.67).

Table 25 Statistical Summaries of ΣPCDD/F Levels in Fish (pg/g, lipid), **1997 - 2007**

	Sample	Mean	Median	SD	Min	Max	95% Confidence Lower	95% Confidence Upper
Liver								
1997	4 (pooled)	9122	6606	7755	2879	20400	3217	21463
2000	12	288	254	233	107	976	140	436
2003	11	99	74	95	39	377	36	164
2007	15	559	408	367	124	1247	356	762
1997 Ref	9 (pooled)	332	167	429	nd	1333	2.0	662
Muscle								
1997	4 (pooled)	3466	3826	2235	835	5375	-91	7021
2000	12	121	74	114	46	436	49	194
2003	11	67	63	33	34	151	45	89
2007	15	294	171	377	55	1495	85	502
1997 Ref	9 (pooled)	188	139	207	nd	583	29	346

Note: (1) The levels were based on a sum of 44 congeners in 1997 and a sum of 209 congeners in 2000 – 2007. (2) The lipid content (4.9%) for liver samples in 1997 was estimated from average values of lipid contents in 2000 – 2007. (3) Fish species in 1997 reference lakes were northern pike.

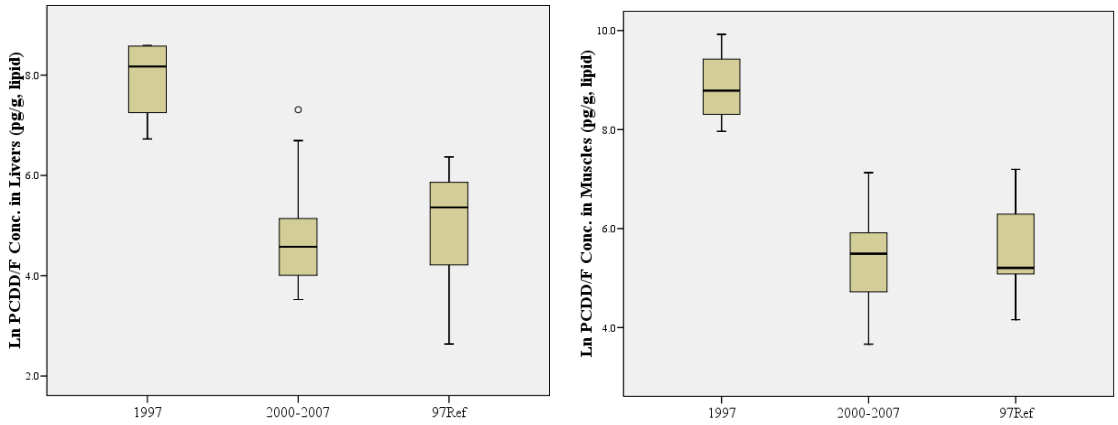


Figure 12 ΣPCDD/F Levels in Brook Trout

t – test for ln concentrations in brook trout in Chrystina Lake 1997 vs. brook trout in 2000 – 2007: $p < 0.01$ in liver samples and muscle samples. The levels in northern pike in the reference lake in 1997 vs. brook trout in Chrystina Lake in 2000 – 2007: $p = 0.67$ in liver samples and $p = 0.61$ in muscle samples.

3.2.4 Dioxin-like TEQs: 2000–2007

The mean values of ∑TEQ in 2000, 2003 and 2007 are summarized in Table 26 and 27. The data on the concentrations of ∑PCBs, lipid-basis in liver samples in 1997 were not available. For comparison, average of lipid contents of 4.9% derived from the lipid contents between 2000 and 2007 was used for calculating the lipid-based concentrations for 1997 data. In the 2000 study, the mean concentrations of ∑TEQ were 161 pg/g in muscle samples and 55 pg/g in liver samples. In the 2003 study, the mean concentrations of ∑TEQ were 224 pg/g in muscle samples and 123 pg/g in liver samples. In the 2007 study, the mean concentrations of ∑TEQ were 835 pg/g, lipid-basis in muscle samples and 76 pg/g in liver samples.

The majority of ∑TEQ in all the samples was due to PCBs (64%–95% in muscle samples and 81%–93% in liver samples). The most important contributor was CB-126, accounting for 83%-89% of ∑PCB-TEQ in muscle and liver samples. CB-126 also accounted for 70%–84% of ∑TEQ in muscle and liver samples. 2,3,7,8 TCDF was prevalent in brook trout, accounting for 67%–85% of ∑PCDD/F-TEQ in muscle samples and 34%–64% in liver samples. However, they were not major contributors of ∑TEQ (4%–6%of ∑TEQ in muscle samples, and 4%–10% in liver samples). The findings are comparable to the results of the annual environmental monitoring program for the Swan Hills Treatment Center in which elevated levels of CB-126 TEQ and 2,3,7,8TCDF TEQ were found in Labrador tea leaves, live moss and soils.

Combustion processes could be the source of the elevated environmental levels of the coplanar congeners characterized by 3,3',4,4' substitution such as CB77, CB126, CB169, CB105, CB156, CB157, CB170 and CB189. The comparable PCBs and PCCD/Fs profiles suggested that the air emission from the facility may contribute to the elevated levels of PCBs and PCDD/Fs in brook trout from Chrystina Lake.

The statistical summaries of ΣTEQ levels between 1997 and 2007 are presented in Table 28. The TEQ levels in the liver and muscle samples of brook trout collected from Chrystina Lake between 2000 and 2007 were significantly lower than the levels in 1997 ($p < 0.01$) (Figure 13). The ΣTEQ levels in the liver samples of brook trout collected between 2000 and 2007 were similar to those in northern pike collected from the reference lakes in 1997 ($p = 0.87$). The TEQ levels in the muscle samples of brook trout collected between 2000 and 2007 were significantly higher than the levels in northern pike collected from the reference lake in 1997 ($p < 0.001$). The ΣTEQ levels in brook trout muscle samples were significantly higher in 2007 than those in 2000 and 2003 ($p < 0.05$) (Figure 14). Book trout taken in 2007 (mean = 557 grams) were larger than those taken in 2000 (mean = 390) and 2003 (mean = 317 grams) from Chrystina Lake. After controlling weight in a multiple regression model, ΣTEQ levels in muscle samples still remain high. Thus, size of fish is not an important factor that influences the ΣTEQ levels in brook trout taken from Chrystina Lake in 2007.

Table 26 Means of ∑TEQs in Brook Trout Muscle Samples (pg/g, lipid-basis)

	2007 (n=15)		2003 (n=12)		2000 (n=12)	
	Conc	**%**	**Conc.**	**%**	**Conc.**	**%**
CB-77	3.67	0.5	1.17	0.6	1.39	1.0
CB-81	0.25	0.0	0.13	0.6	0.22	0.2
CB-126	703	89	186	87	123	84
CB-169	7.5	1.0	2.21	1.0	2.17	1.5
CB-105	13	1.7	5.74	2.7	4.72	3.2
CB-114	1.09	0.1	0.30	0.1	0.29	0.2
CB-118	49	6.2	15	7.1	11	7.7
CB-123	0.78	0.1	0.37	0.2	0.17	0.1
CB-156	4.6	0.6	1.40	0.7	1.49	1.0
CB-157	0.8	0.1	0.28	0.1	0.24	0.2
CB-167	2.4	0.3	0.50	0.2	0.66	0.5
CB-189	0.4	0.1	0.04	0.02	0.13	0.1
2,3,7,8-TCDD	0.00	0.0	0.00	0.0	0.00	0.0
1,2,3,7,8-PeCDD	0.00	0.0	0.00	0.0	0.00	0.0
1,2,3,4,7,8-HxCDD	0.00	0.0	0.00	0.0	0.00	0.0
1,2,3,6,7,8-HxCDD	0.00	0.0	0.00	0.0	3.20	21
1,2,3,7,8,9-HxCDD	0.00	0.0	0.00	0.0	0.00	0.0
1,2,3,4,6,7,8-HpCDD	0.23	0.5	0.2	0.1	0.17	1.1
OCDD	0.04	0.1	0.00	0.0	0.04	0.2
2,3,7,8-TCDF	39	82	6.40	85	11	67
1,2,3,7,8-PeCDF	0.00	0.0	0.00	0.0	0.00	0.0
2,3,4,7,8-PxCDF	8.21	17	1.70	15	1.7	11
1,2,3,4,7,8-HxCDF	0.00	0.0	0.00	0.0	0.00	0.0
1,2,3,6,7,8-HxCDF	0.00	0.0	0.00	0.0	0.00	0.0
1,2,3,7,8,9-HxCDF	0.00	0.0	0.00	0.0	0.00	0.0
2,3,4,6,7,8-HxCDF	0.00	0.0	0.00	0.0	0.00	0.0
1,2,3,4,6,7,8-HpCDF	0.00	0.0	0.00	0.0	0.00	0.0
1,2,3,4,7,8,9-HpCDF	0.00	0.0	0.00	0.0	0.00	0.0
OCDF	0.00	0.0	0.00	0.0	0.00	0.0
ΣPCBs TEQ	788		213		146	
ΣPCDD/Fs TEQ	47		11		16	
Σ TEQ	**835**		**224**		**161**	
% of ∑PCBs-TEQ in ∑TEQ		64		95		90
% of ∑PCDD/Fs-TEQ in ∑TEQ		6		5		10
% of 2,3,7,8-TCDF in ∑PCDD/Fs-TEQ		82		85		67
% of 2,3,7,8-TCDF in ΣTEQ		4.7		4.2		6.5
% of PCB-126 in ∑PCBs-TEQ		89		87		84
% of PCB-126 in Σ TEQ		84		83		76

Values shown for each congener are adjusted by the individual congener-specific TEF factors.

Table 27 Means of ∑TEQs in Brook Trout Liver Samples (pg/g, lipid-basis)

	2007 (n=15)		2003 (n=12)		2000 (n=12)	
	Conc	**%**	**Conc.**	**%**	**Conc.**	**%**
CB-77	0.29	0.5	0.57	0.5	0.16	0.4
CB-81	0.00	0.0	0.07	0.1	0.03	0.1
CB-126	53	83	101	82	38	85
CB-169	0.00	0.0	1.28	1.0	0.87	2.0
CB-105	1.74	2.7	2.81	2.3	1.36	3.0
CB-114	0.14	0.2	0.15	0.1	0.09	0.2
CB-118	7.02	11	7.26	5.9	3.19	7.2
CB-123	0.10	0.2	0.15	0.1	0.05	0.1
CB-156	0.66	1.0	0.71	0.6	0.43	1.0
CB-157	0.13	0.2	0.15	0.1	0.07	0.2
CB-167	0.36	0.6	0.26	0.1	0.21	0.5
CB-189	0.07	0.1	0.03	0.0	0.03	0.1
2,3,7,8-TCDD	0.20	1.6	0.15	1.7	1.02	10
1,2,3,7,8-PeCDD	0.80	6.6	0.13	1.6	1.43	14
1,2,3,4,7,8-HxCDD	0.00	0.0	0.00	0.0	0.17	1.6
1,2,3,6,7,8-HxCDD	0.18	1.5	0.00	0.0	1.28	13
1,2,3,7,8,9-HxCDD	0.00	0.0	0.00	0.0	0.16	1.6
1,2,3,4,6,7,8-HpCDD	0.10	0.8	0.01	0.1	0.10	0.9
OCDD	0.01	0.1	0.00	0.0	0.01	0.1
2,3,7,8-TCDF	7.57	62	5.47	64	3.50	34
1,2,3,7,8-PeCDF	0.07	0.6	0.01	0.1	0.04	0.4
2,3,4,7,8-PxCDF	3.19	26	2.71	32	2.30	23
1,2,3,4,7,8-HxCDF	0.00	0.0	0.04	0.5	0.00	0.0
1,2,3,6,7,8-HxCDF	0.00	0.0	0.00	0.0	0.00	0.0
1,2,3,7,8,9-HxCDF	0.00	0.0	0.00	0.0	0.13	1.3
2,3,4,6,7,8-HxCDF	0.00	0.0	0.00	0.0	0.00	0.0
1,2,3,4,6,7,8-HpCDF	0.00	0.0	0.00	0.0	0.03	0.3
1,2,3,4,7,8,9-HpCDF	0.00	0.0	0.00	0.0	0.02	0.2
OCDF	0.00	0.0	0.00	0.0	0.00	0.0
ΣPCBs TEQ	64		114		45	
ΣPCDD/Fs TEQ	12		9		10	
Σ TEQ	**76**		**123**		**55**	
% of ∑PCBs-TEQ in ∑TEQ		84		93		81
% of ∑PCDD/Fs-TEQ in ∑TEQ		16		7		19
% of 2,3,7,8-TCDF in ∑PCDD/Fs-TEQ		62		64		34
% of 2,3,7,8-TCDF in ΣTEQ		10		4.4		6.4
% of PCB-126 in ∑PCBs-TEQ		83		88		86
% of PCB-126 in Σ TEQ		70		82		70

Values shown for each congener are adjusted by the individual congener-specific TEF factors.

Table 28 Statistical Summaries of ΣTEQ Levels in Fish (pg/g, lipid), 1997 - 2007

	Sample	Mean	Median	SD	Min	Max	95% Confidence Lower	95% Confidence Upper
Liver								
1997	4 (pooled)	1257	1173	730	492	2188	95	2417
2000	12	55	37	39	22	137	30	79
2003	11	123	125	60	49	235	83	164
2007	15	76	63	48	22	193	49	102
1997 Ref	9 (pooled)	172	55	261	15	792	-28	373
Muscle								
1997	4 (pooled)	5340	3601	5135	1288	12867	2832	13511
2000	12	161	145	77	68	338	112	210
2003	11	210	152	204	63	720	73	347
2007	15	835	249	1906	132	7674	-221	1890
1997 Ref	9 (pooled)	17	1.0	48	nd	145	-20	54

Note: (1) The levels were based on a sum of 44 congeners in 1997 and a sum of 209 congeners in 2000 – 2007. (2) The lipid content (4.9%) for liver samples in 1997 was derived from average values of lipid contents in 2000 – 2007. (3) Fish species in 1997 reference lakes were northern pike.

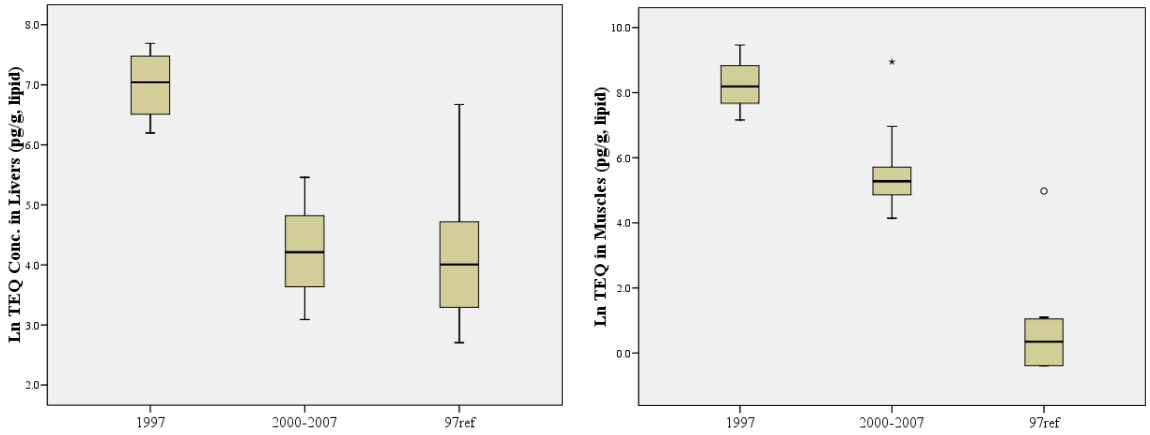


Figure 13 Dioxin-like TEQ Levels in Brook Trout

t – test for ln concentrations in brook trout in Chrystina Lake in 1997 vs. in 2000-2007: $p < 0.01$ in liver samples and muscle samples. The concentrations in northern pike in reference lake in 1997 vs. brook trout in Chrystina Lake in 2000 – 2007: $p = 0.87$ in liver samples and $p < 0.001$ in muscle samples.

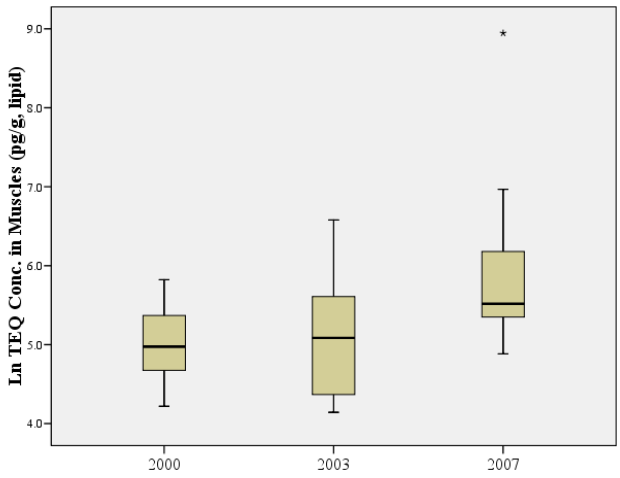


Figure 14 TEQ levels in Brook Trout Muscle Samples in Chrystina Lake, 2000 - 2007

t – test: the levels in 2007 vs. those in 2000 or 2003: $p < 0.05$

3.3 Summary

As compared to the contaminant levels in1997 in brook trout taken from Chrystina Lake nearby the Swan Hills Treatment Center, the ΣPCDD/F and Σdioxin-TEQ levels in liver and muscle samples were statistically significantly decreased in 2000 – 2009. The levels of ΣPCB were not statistically significantly changed in all types of samples in 2000 – 2007.

As compared to the contaminant levels in 1999 in northern pike taken from the reference lakes, the levels of ΣPCB, and Σdioxin-TEQs were still higher in all types of brook trout samples except for Σdioxin-TEQ levels in liver samples taken from Chrystina Lake. The levels of ΣPCDD/F were not statistically significantly changed in all types of samples in 2000 – 2007.

Distribution patterns of ∑PCDD/Fs, ∑PCBs and ∑dioxin-like TEQs in brook trout in the 2000, 2003 and 2007 studies were consistent with those observed in the 1997 study and the annual monitoring programs conducted by the company. Similarity of distribution patterns of the contaminants in various media indicates that the sources of contamination could come from the facility.

4. Estimation of Daily Intake and Exposure Ratios

4.1 Materials and Methods

Diet and Activity Survey - 1997

A diet and activity survey was conducted through telephone interviews during March and April 1997. The survey was divided into two phases. Three hundred and twenty-seven of 370 respondents (88%), including 12 aboriginal people, participated in the first telephone interview. A second telephone interview was then conducted with 100 participants selected from those who had participated in the first telephone interview. Participants were asked to recall their consumption of wild game and fish and their outdoor recreational activities within a 100 km radius of the facility for the previous 12 months. Specifically, the initial survey was used to determine types of outdoor activities within the study area; frequency, duration and amount of wild game and fish consumption; and the respondents' awareness of and adherence to the existing food consumption advisory. The second survey requested demographic characteristics (age, gender, ethnic group, weight and height, occupation, duration of residency, number of persons in the household); proportion of activity time spent indoors and outdoors and detailed information about outdoor activities; detailed information about daily food consumption including consumption of wild game, fish and wild fruit, vegetables and herbs, and cooking and preparation techniques for wild game and fish; and more detailed information about lifestyle (use of alcohol and cigarettes, health conditions and perception of the current health advisory).

Estimates of Daily Intake and Exposure Ratio

The exposure ratio reflects the ratio between the actual level of exposure (external dose) in a particular circumstance and a reference standard derived with precautionary uncertainty factors from observed toxicity in humans or animals. In the current assessment, the estimated daily intake and exposure ratios provide insight into additional exposure that might be expected from consuming local wild game and fish.

Estimated daily intake (EDI) was calculated as follows:

$$EDI = C * IR * BF/BW$$

C is measured concentrations of contaminants, IR is food consumption rate, BF is bioavailability factor (assuming 100%), and BW is average body weight (73 kg for Albertans).
Exposure ratios (ER) were calculated by using the following equation:

$$ER = EDI/TDI$$

TDI is tolerable daily intake.

4.2 Results and Discussions

4.2.1 Tolerable Daily Intakes

A tolerable daily intake (TDI) is the amount of a substance that can be ingested daily over a lifetime without appreciable health risk. It is expressed in relation to the bodyweight (bw) in order to allow for different body size, such as for children of different ages. A daily intake of 10 pg/kg bw/day is 730 pg/day for an average 73 kg person.

Because about 90% of overall exposure to PCDD/Fs comes through diet in the general population, the Joint Expert Committee on Food Additives, an expert group of the World Health Organization and the Food and Agriculture Organization of the United Nations, has set a "tolerable monthly intake" level of 70 pg/kg bw/month (~ 2.3 pg/ kg bw/day) for PCDD/Fs (JECFA 2001).

The studies conducted in two Canadian cities between 1998 and 1999 showed that the average dietary intake of PCDD/Fs was 0.62 pg TEQ/kw bw /day. This intake level is within the TDI level considered by Joint Expert Committee on Food Additives. Although the Health Canada Official TDI for PCDD/Fs is 10 pg/kw bw/day (Health Canada 1996), the Food Directorate, Health Canada, generally endorses and applies the TDIs for food contaminants derived by the JECFA. Therefore, Health Canada recommended that preliminary quantitative risk assessments for PCDD/Fs and dioxin-like compounds in Canada employ this more conservative TDI (Health Canada 2005; Feeley 2009).

PCDD/Fs accumulate gradually in the body over a period of about 30 years. Afterward, the level of intake will be about the same as the level of elimination from the body. The total body burden could be about 2000 times higher than the average daily intake (UKCOT 2001). For example, an intake of 10 times the TDI on a single day would result in a 0.5% increase in the body burden. This small increase would not be sufficient to have any adverse health effect. As a result occasionally ingesting PCDD/F in an amount more than the TDI, it would not be expected to result in harmful effects.

Health Canada proposed a TDI of 0.13 μg/kg bw/d for ∑PCBs (Dewailly et al. 2007; Feeley 2009). The average dietary intake of PCB range from 0.005 to 0.01 μg/kg bw/d in Canadian population.

4.2.2 Diet and Activity Survey: 1997

A total of 123 (38%) respondents had consumed wild game taken from the study area and 127 (39%) had consumed fish (Table 29). Moose, deer and grouse are the most common wild game for consumption. The most commonly consumed fish species were walleye, northern pike, perch, brook trout, lake whitefish and arctic grayling. The average consumption rate was 35 grams/day of wild game and 15 grams/day of fish. A

small proportion of consumers ate a relatively large quantity of local wild game and fish. Aboriginal people may have higher rates of consumption of food from local sources, though specific data for aboriginal people living in the study area are not available.

Table 29 Consumption Rate for Wild Game and Fish Table

Consumption Group*	Wild Game		Fish	
	Mean (g/d)	% consumed (n=123)	Mean (g/d)	% consumed (n=127)
High Intake (>100 g/d)	191	8	167	2
Medium Intake (30-99 g/d)	58	25	47	13
Low Intake (5-29 g/d)	13	31	13	28
Very Low Intake (<4 g/d)	2	36	2	57

* based on consuming muscle portion

4.2.3 Exposure Ratios: 1997

Estimated exposure ratios are presented in Table 30. The exposure ratios for high and medium consumption groups were greater than one as compared to Health Canada Official TDI for TCDD at the 90th percentile concentrations of ∑dioxin-like TEQ in deer and brook trout muscle samples. A value of 10 pg/kg bw/d has been adopted by Health Canada in 1996 (Health Canada 1996). This TDI value was used to calculate the food consumption limits in 1997.

Table 30 Exposure Ratio in High and Medium Intake Groups, 1997

Species	∑TEQ Conc. (pg/g, ww)	Percentile	Exposure Ratio (High Intake)	Exposure Ratio (Medium Intake)
Deer Muscle	67	90th	**17**	**5.5**
	2.4	50th	<1	<1
Deer Liver	1730	90th	**4.3**	-
Brook Trout Muscle	29	90th	**6.6**	**1.8**
	12	50th	**2.8**	<1

Note: High Intake Group - 190 g/d for consuming deer meat, 2 g/d for consuming deer liver tissue, and 170 g/d for consuming brook trout fillet. Medium Intake Group - 60 g/d for consuming deer meat, and 50 g/d for consuming brook trout fillet.

Based on concentrations at the 90th percentile of ∑dioxin-like TEQ, consumption limits were recommended (Table 31). These consumption limits provide precautionary guidance to manage risk associated with exposure to PCBs and PCDD/Fs for individuals who consume deer or moose meat taken within a 30 km radius of the facility and/or brook trout from Chrystina lake near the facility. The estimated values represent

the amount of meat from deer or moose and from edible portions of brook trout expected to generate a risk no greater than the tolerable daily intake proposed by Health Canada, based on a lifetime of daily consumption at that consumption limit. Because the contaminants tend to accumulate in the internal organs in various animals and the measured levels of the contaminants were relatively much higher in the liver samples, people should avoid consumption of viscera from wild game and fish. The current Health Canada TDI for TCDD is based on the potential for exposure to contribute to cancer. The toxicity of TCDD and related congeners also includes reproductive, developmental and immunotoxic effects. Children and pregnant women or women who are breast-feeding are susceptible groups and they should avoid consuming wild game and fish.

Table 31 Species-Specific Consumption Limits

Parameter	Wild Game Meat	Fish
Species	deer and moose	brook trout
Location	within 30 km radius of the facility	Chrystina lake
Type of tissue	muscle	muscle
Health Canada TDI for TCDD(pg/kg bw/d)	10	10
Body weight (kg) based on Alberta average	73	73
Consumption limit (gram/month)	370	740
Consumption limit (oz/month)	13	26

Wild game and fish may supplement the diet of a number of people living in the area surrounding the facility. Concern has been raised by both recreational users and traditional users because these two groups consume more wild game and fish than the general population. The balance between nutritional benefits and health risks arising from the consumption of wild food containing trace levels of contaminants is an important consideration in issuing public health advisories.

Food consumption advisories were issued by the Provincial Chief Medical Officer in 1997 (Appendix A). The current advisories address this issue in three ways. First, the dietary survey attempted to determine the extent of wild game and fish consumption by residents of the study area. Survey results indicate that only a small proportion of people ate wild game and fish caught near the facility at high consumption rates. Second, the advisories provided consumption limits developed from risk estimates rather than an outright ban on consumption. These limits do indicate that wild game and fish meats may still be safely consumed in moderation. Consumption of viscera (liver, kidney etc.) is not recommended. Third, the advisories are restricted to a 30 km radius of the facility in accordance with evidence that substantial contamination with PCBs, PCDDs and PCDFs is restricted to areas near the facility. Measured contaminant levels outside the 30 km zone represents background contamination arising from long range

transport. Therefore, traditional and recreational users can still safely consume wild game and fish meat obtained from outside the affected area. Finally, it should be noted that the consumption limits provided in the advisories were calculated in reference to uncooked food. Many studies have shown that appropriate food preparation and cooking techniques can reduce the concentrations of PCBs, PCDDs and PCDFs in fish and meat. Thus, techniques such as removing the skin prior to cooking, broiling and baking are recommended for individuals who continue to consume wild game and fish taken from the areas immediately surrounding the facility.

4.2.4 Estimated Daily Intake and Exposure Ratios: 1999 – 2007

Dioxin-like TEQ

Estimated daily intake and exposure ratio for dioxin-like TEQ between 1999 and 2007 are summarized in Table 32. Two different levels of dioxin-like TEQ concentrations were used for estimating daily intakes for high intake group which represented 2% of the survey population. The 90th percentile concentrations likely represented the levels from deer taken nearby the facility fence. Thus, the calculation based on the 90th percentile concentrations in high intake group represented the worst case scenario. The estimated daily intakes for consuming deer meat in this worst case scenario ranged from 0.46 to1.76 pg TEQ/kg bw/d. The exposure ratios for muscle tissues in deer for all consumption groups were less than one. In the latest Canadian dietary intake survey, the average adult intake for consuming retail food was 0.62 pg TEQ/kg bw/d (Health Canada 2005). If adding the amount of 1.35 pg TEQ/kg bw/d from the wild game diet in the 2007 study, the daily intake would reach 86% (1.97 pg TEQ/kg bw/d) of the Health Canada interim TDI value for dioxin-like compounds. If individuals occasionally consumed wild game meat (not every day for life-time) taken from the Swan Hills area, that level of exposure would not be expected to result in harmful effects.

Consuming the wild game meat taken close to the facility fence should be avoided. In the 2007 study, the highest level of ∑TEQ was observed in one deer taken 1.2 km far from the facility fence (Max. conc. in Table 32). The exposure ratio was over for that case exceeded one.

The exposure ratios for consuming deer liver tissues in the 2001 and 2007 studies for consumption of 2 grams of liver per day were over one. Consuming internal organs (visera) of the wild game taken from the Swan Hills area should be avoided.

The estimated daily intakes for consuming brook trout in the worst case scenario for the high wild game intake group ranged from 2.24 to 5.68 pg TEQ/kg bw/d between 2000 and 2007. The exposure ratios for consuming brook trout muscle samples from 2003 and 2007 studies were over one. If adding the amount of 4.17 pg TEQ/kg bw/d from brook trout diet in the 2007 study, the daily intake would be about two-fold higher than (4.8 pg TEQ/kg bw/d) of the Health Canada interim TDI value for dioxin-like compounds.

Table 32 Estimated Daily Intake and Exposure Ratios in High Intake Group – ∑TEQ

Species	Year	∑TEQ Conc. (pg/g, ww)	Percen-tile	Estimated Daily Intake (pg TEQ/kg bw/d)	Exposure Ratio
Deer Muscle	1999	0.18	90th	0.46	<1
		0.02	50th	0.09	<1
	2001	0.68	90th	1.76	<1
		0.04	50th	0.11	<1
	2003	0.04	90th	0.10	<1
		0.03	50th	0.07	<1
	2007	0.52	90th	1.35	<1
		0.33	50th	0.83	<1
		1.23	Max	3.2	**1.4**
Deer Liver	1999	87	90th	2.39	~ 1
	2001	969	90th	27	**12**
	2003	14	90th	0.38	<1
	2007	371	90th	10	**4**
Brook Trout Muscle	2000	0.96	90th	2.24	<1
		0.46	50th	1.06	<1
	2003	2.44	90th	5.68	**2.5**
		1.17	50th	2.72	**1.2**
	2007	1.79	90th	4.17	**1.8**
		0.66	50th	1.54	<1

Note: High Intake Group - 190 g/d for consuming deer meat, 2 g/d for consuming deer liver tissue, and 170 g/d for consuming brook trout fillet. The TDI for dioxin-like compounds is 2.3 pg TEQ/kg bw/d.

PCBs

Estimated daily intake and exposure ratio for ∑PCBs between 1999 and 2007 are summarized in Table 33. Note that exposure levels are expressed in μg/kg bw/d not pg/kg bw/d. Thus, the calculation based on the 90th percentile concentrations in high intake group represented the worst case scenario. The estimated daily intakes for consuming deer meat in this worst case scenario ranged from 0.001 to 0.02 μg PCB/kg bw/d. The exposure ratios for muscle tissues in deer for all consumption groups were less than one. In the latest Canadian dietary intake survey, the average adult intake for consuming retail food was 0.005 – 0.01 μg PCB/kg bw/d (Health Canada 2005). If adding the amount of 0.008 μg PCB/kg bw/d from the wild game diet in the 2007 study, the daily intake would reach only 1% (0.018 μg PCB /kg bw/d) of the Health Canada interim TDI value for ∑PCBs. The estimated daily intakes in the worst case scenario for the high fish intake group ranged from 0.10 to 0.17 μg PCB /kg bw/d between 2000 and 2007. The exposure ratios for consuming brook trout muscle samples from 2003 and 2007 studies were over one.

Table 33 Estimated Daily Intake and Exposure Ratios in High Intake Group - ∑PCBs

Species	Year	∑PCB Conc. (μg/kg, ww)	Percen-tile	Estimated Daily Intake (μg PCB/kg bw/d)	Exposure Ratio
Deer Muscle	1999	0.67	90th	0.002	<1
	2001	6.11	90th	0.02	<1
	2003	0.51	90th	0.001	<1
	2007	2.95	90th	0.008	<1
Brook Trout Muscle	2000	44	90th	0.10	<1
	2003	70	90th	0.16	**1.3**
	2007	72	90th	0.17	**1.3**

Note: High Intake Group - 190 g/d for consuming deer meat, and 170 g/d for consuming brook trout fillet. The TDI for PCBs is 0.13 μg PCB/kg bw/d.

4.2.5 Benefits and Risks of Local Food Consumption

Consumption of fish and wild game is an important part of the cultural traditions of many groups in Alberta. Also, the benefits of local food consumption, particularly fish consumption, are a recent focus of public health interest. Fish is an important supplier of nutrition for people, because it contains beneficial nutrients like the long-chain omega-3 fatty acids like eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), vitamin D, selenium and iodine. Fish is considered an excellent source of high quality protein. The benefits of fish consumption include the prevention of cardiovascular diseases, myocardial infarction (heart attack), and arrhythmia, especially reduction of risk for ischemic heart disease and stroke (Zhang et al. 1999; Chan and Egeland 2004; Bouzanc et al. 2005; Koning et al. 2005; Kris-Etherton et al. 2005; Stern 2005). Health Canada reviewed the consistent evidence on an association between reduced risk of sudden cardiac death and fish consumption frequency at least once per week (Health Canada 2007). From a nutritional perspective, regular fish consumption is beneficial to the general population.

Fish consumption is important for neurodevelopment in infant and young children. DHA is an integral structural component of the brain and essential nutrient for pregnant women. DHA can be easily and rapidly absorbed into the developing fetal brain during gestation and in the earlier years of life of young children (Dovydaitis 2008). DHA was found to improve the visual-motor development in healthy term infants (Uauy et al. 2003; Oken et al. 2008). Some studies showed that fish consumption could increase a child's intelligence quotient (Helland et al. 2003; Dunstan et al. 2008). For pregnant and breastfeeding women and women of childbearing age, fish consumption is important because it supplies DHA that is beneficial for the brain development of infants.

From toxicological perspective, food could be associated with environmental contaminants like PCBs and PCDD/Fs, which pose a potential threat to humans. PCBs and PCDD/Fs were detected in retailed meats and dairy products, wildlife, freshwater fish and seafood in Canada. Compared with the health benefits of food intake, the health risks of these chemical levels are very low and should not influence individual decisions about food consumption (Mozaffarian and Rimm, 2006; Mozaffarian 2009). Compared with retailed food and local food in the reference areas, wild game meat and brook trout taken from the surrounding of the Swan Hills Treatment Center have higher PCB and PCDD/F levels, so local advisories should be consulted.

Communication to the public about the competition between benefits and risks is important to include in food consumption advisory. In order to protect all human consumers, issuing food consumption advisory is one risk management option. Food consumption advisories are designed to reduce potential health risks of consumption for local food consumers. Advisories should provide the necessary information to the public, so that local food consumers can voluntarily restrict their food consumption to a level judged to be safe. Food consumption advisories are voluntary activities. Food consumption advisories should enable people to make informed decision about what is a safe amount of local food consumption in order to address risks posed by environmental hazards, and to optimize the nutritional benefits of local food consumption with regard to preventable disease while improving neurodevelopment in infants and young children.

Health Canada and World Health Organization developed a guideline for consuming food containing dioxin-like compounds to reduce the small chance of being affected by potential health risks (JECFA 2001; Health Canada 2005). 2.3 pg/kg bw/d of TDI for dioxin-like chemicals was set to protect against on the developing male reproductive system resulting from the maternal body burden of dioxin-like compounds (JECFA 2001; SCAN 2004). This guideline was considered adequate to protect against possible adverse health effects like cancer and cardiovascular diseases.

PCBs and PCDD/Fs can stay in the human body for many years. Pregnant and breasting women should avoid consuming local food containing the high levels of PCBs and PCDD/Fs to minimize exposure to the fetus. Young children are one of the most sensitive groups, so they should avoid consuming local food containing the high levels of PCBs and PCDD/Fs. PCBs and PCDD/Fs can concentrate in the fat-rich tissues such as livers and kidney. Consumption of internal organs of wild game and fish should be avoided. Meanwhile, in order to minimize exposure to PCBs and PCDD/Fs, preparation of wild game meat and fish by trimming visible fat from food and removing skin, cooking by using methods like baking, broiling, roasting, barbecuing or microwaving instead of frying, and draining off extra fat after cooking are encouraged.

4.3 Summary

The estimated daily intakes for consuming deer muscles in the high intake group (190 g/d) based on the 90th percentile level of Σdioxin-like TEQs and ΣPCBs in 1999 – 2007 were within the Health Canada guidelines. The estimated daily intakes for consuming brook trout muscles in the high intake group (170 g/d) based on the 90th percentile level of Σdioxin-like TEQs and ΣPCBs in 2000 – 2007 exceeded the Health Canada guidelines.

Restriction of consumption of wild game and brook trout taken nearby the Swan Hills area was indicated by the health risk assessment. The existing food consumption advisories are still effective. Food consumption advisories are voluntary measures to reduce potential health risk to local food consumers. The balance between risk and benefits of consumption of contaminant-containing local food needs to be understood and considered by consumers.

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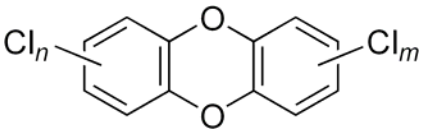
GLOSSARY

CB Chlorobiphenyl – a compound composed of 2 joined aromatic rings with some level of chlorination of the ring, any one of the polychlorinated biphenyls (PCBs).

congener One of a group of related chemical compounds, i.e. the various individual chlorobiphenyl and dioxin/furan compounds are referred to as congeners.

dioxin Dioxin is a generic term for a group of more than 200 complex compounds, all of which contain chlorine (specialist name: polychlorinated dibenzodioxins and furans, PCDDs and PCDFs). Seventeen of the compounds in this group have been found to be toxic to varying extents, of which 2,3,7,8 tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) is the most toxic.

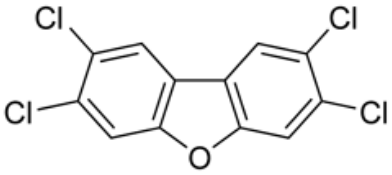


dioxin-like Dioxin-like refers to any compound which exhibits a toxic mechanism similar to 2,3,7,8-TCDD.

di-ortho A PCB that has two chlorine substitutions at any ortho position.

ER The exposure ratio (ER) reflects the ratio between the actual level of exposure (external dose) in a particular circumstance and a reference standard associated with observed toxicity in humans or animals.

furans Chlorinated dibenzofurans, or CDFs, are a family of chemicals that contain one to eight chlorine atoms attached to the carbon atoms of the parent chemical, dibenzofuran, and often associated with dioxins (PCDDs).



homologue For compounds like polychlorinated biphenyls, polychlorinated dibenzodioxins and polychlorinated dibenzofurans, there can be multiple chlorine substitutions and homologue refers to a collection of congeners for one of these class of compounds that all contain the same number of substituted chlorines.

lipid-basis The persistent, bioaccumulative, toxic compounds under study generally much more lipid (fat) soluble than they are water soluble. As a result, these contaminants will concentration in lipids, meaning that the total body or organ-specific burden of these lipophilic compounds will be influenced by the lipid content of the whole body or organ under study. As a result, lipophilic contaminants are commonly expressed as mass of contaminant per mass of lipid, rather than the mass of the entire organ or organism.

lipophilic A characteristic of a substance such they are more fat soluble than water soluble.

MDL Method detection limit – defined as 2.5 to 3 times the signal to noise ratio of the instrument being used to detect a contaminant. The MDL will be specific for each congener.

mono-ortho A PCB that has one chlorine substitutions at any ortho position.

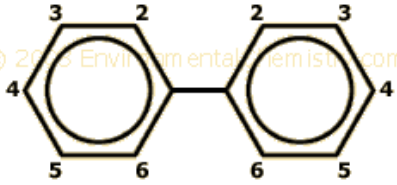
ng A unit of measuring concentration: 10^{-9} gram

non-ortho A PCB that has no chlorine substitutions at any ortho position.

ortho The position on the biphenyl ring of a PCB that is adjacent to bond between the two rings. Chlorines on carbons 2 or 2' and/or 6 or 6' are in the ortho position (the chorine is bonded to a carbon immediately adjacent to Carbon 1 or 1').

Ortho

PCB Polychlorinated biphenyls (PCB) are a family of synthetic chemical compounds consisting of chlorine, carbon and hydrogen. PCBs consist of two benzene rings with a carbon to carbon bond between carbon 1 on one ring and carbon 1' on the second ring with a varying number of chlorines.



∑PCBs A summation of levels of PCB concentration

PCB-TEQ PCB congeners have TEFs associated with dioxin-like toxicity.

∑PCB-TEQ A summation of levels of PCB-TEQ concentration.

PCDD/Fs Polychlorinated dibenzo-ρ-dioxins (dioxin or PCDD) and dibenzofurans (furan or PCDF). There are 135 different types of CDFs with varying harmful health and environmental effects. The compounds that contain chlorine atoms at the 2,3,7,8-positions of the dibenzofuran molecule are known to be especially harmful.

∑PCDD/F A summation of levels of PCDD/F concentration

PCDD/F-TEQ PCDD/PCDF congeners have TEFs associated with dioxin-like toxicity.

∑PCDD/F-TEQ a summation of levels of PCDD/F-TEQ concentration

pg A unit of measuring concentration: 10^{-12} gram

REP Relative effect potency – an experimental measure of the potency of a given dioxin or dioxin-like compound at binding the AhR receptor relative to 2,3,7,8-TCDD

SIM Selected ion monitoring is a technique used in chromatography linked to mass spectrometry whereby monitoring for a set of characteristic fragment ions (according to their mass) for a contaminant that is being analyzed will be used to identify and quantify the presence of a specific contaminant.

TDI A tolerable daily intake (TDI) is the amount of a substance that can be ingested daily over a lifetime without appreciable health risk. It is expressed in relation to the bodyweight (bw) in order to allow for different body size, such as for children of different ages.

TEF Toxic Equivalency Factors (TEFs) are toxicity potency factors that are used by the regulators globally as a consistent method to evaluate the toxicities of highly variable mixtures of dioxin compounds. In the dioxin family, 2,3,7,8-TCDD is the "habanero" of the bunch, the most studied and the most toxic member, and it is assigned a TEF of one. (One other dioxin-like compound, 1,2,3,7,8-PnCDD, also has a TEF of one.) The other family members are less toxic than 2,3,7,8-TCDD, and are also much less studied on an individual basis.

TEQ Toxic equivalency approach using TEF for individual dioxin and/or dioxin-like compounds.

∑TEQ A summation of the toxicity of a mixture of dioxin and/or dioxin-like compounds making use of the TEF for each individual compound.

WHO-TEF World Health Organization (WHO) approaches for the derivation of TEFs based on available literature. These can include quantitative (statistical) methods such as establishing an uncertainty range of available relative potency data and application of a specified cut-off value to derive TEF values; application of weighting factors to existing data, etc.

Appendix A

Food Consumption Advisories in 1997

Alberta
HEALTH

Backgrounder

Edmonton, October 30, 1997

Wild Game and Fish Public Health Advisory

Wild Game

- limit eating wild game taken from within a 30 km radius of Swan Hills Treatment Centre to 13 ounces per month (370 grams);
- avoid eating organ meat (liver, kidney) or using fat from wild game harvested within a 30 km radius of the treatment centre
- pregnant or breast feeding women should avoid eating wild game taken from within a 30 km radius of the treatment centre
- young children should avoid eating wild game taken from within a 30 km radius of the treatment centre

Fish

- limit eating fish taken from within a 20 kilometre radius of the Swan Hills Waste Treatment Centre to 6 oz (170 grams) per week or less
- avoid eating fish organs or eggs taken from lakes within the 20 kilometre radius
- avoid eating fish from lakes within the 20 kilometre radius if pregnant or breast feeding
- young children should avoid eating fish taken from within the 20 kilometre radius

Fish Preparation Instructions

- remove the skin before cooking the fish
- trim the fat from the fish (belly flap, sides, back and under the skin)
- broil or bake the fish on a rack so the fats drips away
- do not use the drippings to prepare any other recipes

The advisory applies to animals and fish taken in the fall of 1996, as well as to those taken in the current year.

Alberta Health
Communications
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Appendix B

Toxic Equivalency Factors

The 2005 World Health Organization Re-evaluation of Human and Mammalian Toxic Equivalency Factors (TEFs) for Dioxins and Dioxin-like Compounds

Compound	PCB#	TEF for Human
2,3,7,8-TCDD		1.0000
1,2,3,7,8-PeCDD		1.0000
1,2,3,4,7,8-HxCDD		0.1000
1,2,3,6,7,8-HxCDD		0.1000
1,2,3,7,8,9-HxCDD		0.1000
1,2,3,4,6,7,8-HpCDD		0.0100
OCDD		0.0003
2,3,7,8-TCDF		0.1000
1,2,3,7,8-PeCDF		0.0300
2,3,4,7,8-PeCDF		0.3000
1,2,3,4,7,8-HxCDF		0.1000
1,2,3,6,7,8-HxCDF		0.1000
2,3,4,6,7,8-HxCDF		0.1000
1,2,3,7,8,9-HxCDF		0.1000
1,2,3,4,6,7,8-HpCDF		0.0100
1,2,3,4,7,8,9-HpCDF		0.0100
OCDF		0.0003
3,3',4,4'-TeCB	77	0.00010
3,4,4',5-TeCB	81	0.00030
3,3',4,4',5-PeCB	126	0.10000
3,3',4,4',5,5'-HxCB	169	0.03000
2,3,3',4,4'-PeCB	105	0.00003
2,3,4,4',5-PeCB	114	0.00003
2,3',4,4',5-PeCB	118	0.00003
2',3,4,4',5-PeCB	123	0.00003
2,3,3',4,4',5-HxCB	156	0.00003
2,3,3',4,4',5'-HxCB	157	0.00003
2,3',4,4',5,5'-HxCB	167	0.00003
2,3,3',4,4',5,5'-HpCB	189	0.00003

Source: van den BM, Birnbaum LS, Denison M, De Vito M, Farland W, Feeley M, Fiedler H, Hakansson H, Hanberg A, Haws L, Rose M, Safe S, Schrenk D, Tohyama C, Tritscher A, Tuomisto J, Tysklind M, Walker N, Peterson RE. The 2005 World Health Organization reevaluation of human and Mammalian toxic equivalency factors for dioxins and dioxin-like compounds. Toxicol Sci 93:223-241 (2006).

Appendix C

PCB Congeners

List of PCB Congeners

Congener	#	Congener	#	Congener	#	Congener	#
2-MoCB	1	2,2',6,6'-TeCB	54	2,3,3',4',5-PeCB	107	2,3,3',4,5,6-HxCB	160
3-MoCB	2	2,3,3',4'-TeCB	55	2,3,3',4,5'-PeCB	108	2,3,3',4,5',6-HxCB	161
4-MoCB	3	2,3,3',4'-TeCB	56	2,3,3',4,6-PeCB	109	2,3,3',4',5,5'-HxCB	162
2,2'-DiCB	4	2,3,3',5-TeCB	57	2,3,3',4',6-PeCB	110	2,3,3',4',5,6-HxCB	163
2,3-DiCB	5	2,3,3',5'-TeCB	58	2,3,3',5,5'-PeCB	111	2,3,3',4',5',6-HxCB	164
2,3'-DiCB	6	2,3,3',6-TeCB	59	2,3,3',5,6-PeCB	112	2,3,3',5,5',6-HxCB	165
2,4-DiCB	7	2,3,4,4'-TeCB	60	2,3,3',5',6-PeCB	113	2,3,4,4',5,6-HxCB	166
2,4'-DiCB	8	2,3,4,5-TeCB	61	2,3,4,4',5-PeCB	114	2,3,4,4',5,5'-HxCB	167
2,5-DiCB	9	2,3,4,6-TeCB	62	2,3,4,4',6-PeCB	115	2,3',4,4',5',6-HxCB	168
2,6-DiCB	10	2,3,4',5-TeCB	63	2,3,4,5,6-PeCB	116	3,3',4,4',5,5'-HxCB	169
3,3'-DiCB	11	2,3,4',6-TeCB	64	2,3,4',5,6-PeCB	117	2,2',3,3',4,4',5-HpCB	170
3,4-DiCB	12	2,3,5,6-TeCB	65	2,3',4,4',5-PeCB	118	2,2',3,3',4,4',6-HpCB	171
3,4'-DiCB	13	2,3',4,4'-TeCB	66	2,3',4,4',6-PeCB	119	2,2',3,3',4,5,5'-HpCB	172
3,5-DiCB	14	2,3',4,5-TeCB	67	2,3',4,5,5'-PeCB	120	2,2',3,3',4,5,6-HpCB	173
4,4'-DiCB	15	2,3',4,5'-TeCB	68	2,3',4,5,6-PeCB	121	2,2',3,3',4,5,6'-HpCB	174
2,2',3-TrCB	16	2,3',4,6-TeCB	69	2',3,3',4,5-PeCB	122	2,2',3,3',4,5',6-HpCB	175
2,2',4-TrCB	17	2,3',4',5-TeCB	70	2',3,4,4',5-PeCB	123	2,2',3,3',4,6,6'-HpCB	176
2,2',5-TrCB	18	2,3',4',6-TeCB	71	2',3,4,5,5'-PeCB	124	2,2',3,3',4',5,6-HpCB	177
2,2',6-TrCB	19	2,3',5,5'-TeCB	72	2',3,4,5,6'-PeCB	125	2,2',3,3',5,5',6-HpCB	178
2,3,3'-TrCB	20	2,3',5',6-TeCB	73	3,3',4,4',5-PeCB	126	2,2',3,3',5,6,6'-HpCB	179
2,3,4-TrCB	21	2,4,4',5-TeCB	74	3,3',4,5,5'-PeCB	127	2,2',3,4,4',5,5'-HpCB	180
2,3,4'-TrCB	22	2,4,4',6-TeCB	75	2,2',3,3',4,4'-HxCB	128	2,2',3,4,4',5,6-HpCB	181
2,3,5-TrCB	23	2',3,4',5-TeCB	76	2,2',3,3',4,5-HxCB	129	2,2',3,4,4',5,6'-HpCB	182
2,3,6-TrCB	24	3,3',4,4'-TeCB	77	2,2',3,3',4,5'-HxCB	130	2,2',3,4,4',5',6-HpCB	183
2,3',4-TrCB	25	3,3',4,5-TeCB	78	2,2',3,3',4,6-HxCB	131	2,2',3,4,4',6,6'-HpCB	184
2,3',5-TrCB	26	3,3',4,5'-TeCB	79	2,2',3,3',4,6'-HxCB	132	2,2',3,4,5,5',6-HpCB	185
2,3',6-TrCB	27	3,3',5,5'-TeCB	80	2,2',3,3',5,5'-HxCB	133	2,2',3,4,5,6,6'-HpCB	186
2,4,4'-TrCB	28	3,4,4',5-TeCB	81	2,2',3,3',5,6-HxCB	134	2,2',3,4,5,5',6-HpCB	187
2,4,5-TrCB	29	2,2',3,3',4-PeCB	82	2,2',3,3',5,6'-HxCB	135	2,2',3,4',5,6,6'-HpCB	188
2,4,6-TrCB	30	2,2',3,3',5-PeCB	83	2,2',3,3',6,6'-HxCB	136	2,3,3',4,4',5,5'-HpCB	189
2,4',5-TrCB	31	2,2',3,3',6-PeCB	84	2,2',3,4,4',5-HxCB	137	2,3,3',4,4',5,6-HpCB	190
2,4',6-TrCB	32	2,2',3,4,4'-PeCB	85	2,2',3,4,4',5'-HxCB	138	2,3,3',4,4',5',6-HpCB	191
2',3,4-TrCB	33	2,2',3,4,5-PeCB	86	2,2',3,4,4',6-HxCB	139	2,3,3',4,5,5',6-HpCB	192
2',3,5-TrCB	34	2,2',3,4,5'-PeCB	87	2,2',3,4,4',6'-HxCB	140	2,3,3',4',5,5',6-HpCB	193
3,3',4-TrCB	35	2,2',3,4,6-PeCB	88	2,2',3,4,5,5'-HxCB	141	2,2',3,3',4,4',5,5'-OcCB	194
3,3',5-TrCB	36	2,2',3,4,6'-	89	2,2',3,4,5,6-HxCB	142	2,2',3,3',4,4',5,6-OcCB	195

		PeCB					
3,4,4'-TrCB	37	2,2',3,4',5-PeCB	90	2,2',3,4,5,6'-HxCB	143	2,2',3,3',4,4',5,6'-OcCB	196
3,4,5-TrCB	38	2,2',3,4',6-PeCB	91	2,2',3,4,5',6-HxCB	144	2,2',3,3',4,4',6,6'-OcCB	197
3,4',5-TrCB	39	2,2',3,5,5'-PeCB	92	2,2',3,4,6,6'-HxCB	145	2,2',3,3',4,5,5',6-OcCB	198
2,2',3,3'-TeCB	40	2,2',3,5,6-PeCB	93	2,2',3,4',5,5'-HxCB	146	2,2',3,3',4,5,5',6'-OcCB	199
2,2',3,4-TeCB	41	2,2',3,5,6'-PeCB	94	2,2',3,4',5,6-HxCB	147	2,2',3,3',4,5,6,6'-OcCB	200
2,2',3,4'-TeCB	42	2,2',3,5',6-PeCB	95	2,2',3,4',5,6'-HxCB	148	2,2',3,3',4,5',6,6'-OcCB	201
2,2',3,5-TeCB	43	2,2',3,6,6'-PeCB	96	2,2',3,4',5',6-HxCB	149	2,2',3,3',5,5',6,6'-OcCB	202
2,2',3,5'-TeCB	44	2,2',3',4,5-PeCB	97	2,2',3,4',6,6'-HxCB	150	2,2',3,4,4',5,5',6-OcCB	203
2,2',3,6-TeCB	45	2,2',3',4,6-PeCB	98	2,2',3,5,5',6-HxCB	151	2,2',3,4,4',5,6,6'-OcCB	204
2,2',3,6'-TeCB	46	2,2',4,4',5-PeCB	99	2,2',3,5,6,6'-HxCB	152	2,3,3',4,4',5,5',6-OcCB	205
2,2',3,4'-TeCB	47	2,2',4,4',6-PeCB	100	2,2',4,4',5,5'-HxCB	153	2,2',3,3',4,4',5,5',6-NoCB	206
2,2',4,5-TeCB	48	2,2',4,5,5'-PeCB	101	2,2',4,4',5',6-HxCB	154	2,2',3,3',4,4',5,6,6'-NoCB	207
2,2',4,5'-TeCB	49	2,2',4,5,6'-PeCB	102	2,2',4,4',6,6'-HxCB	155	2,2',3,3',4,5,5',6,6'-NoCB	208
2,2',4,6-TeCB	50	2,2',4,5,6'-PeCB	103	2,3,3',4,4',5-HxCB	156	DeCB	209
2,2',4,6'-TeCB	51	2,2',4,6,6'-PeCB	104	2,3,3',4,4',5'-HxCB	157		
2,2',5,5'-TeCB	52	2,3,3'4,4'-PeCB	105	2,3,3',4,4',6-HxCB	158		
2,2',5,6'-TeCB	53	2,3,3',4,5-PeCB	106	2,3,3',4,5,5'-HxCB	159		

MoCB = monochlorobiphenyl
DiCB = dichlorobiphenyl
TrCB = trichlorobiphenyl
TeCB = tetrachlorobiphenyl
PeCB = pentachlorobiphenyl
HxCB = hexachlorobiphenyl
HpCB = heptachlorobiphenyl
OcCB = octachlorobiphenyl
NoCB = nonaclorobiphenyl
DeCB = decachlorobiphenyl

Typical Co-Eluters in Laboratory Analysis

PCBs # 12 & 13	PCBs # 88 & 91
PCBs # 18 & 30	PCBs # 90, 101 & 113
PCBs # 26 & 29	PCBs # 86, 87, 97, 108, 119 & 125

PCBs # 21 & 33	PCBs # 107 & 124
PCBs # 50 & 53	PCBs # 135 & 151
PCBs # 45 & 51	PCBs # 147 & 149
PCBs # 43 & 73	PCBs # 139 & 140
PCBs # 49 & 69	PCBs # 153 & 168
PCBs # 44, 47 & 65	PCBs # 129, 138 & 163
PCBs # 59, 62 & 75	PCBs # 128 & 166
PCBs # 41, 71 & 40	PCBs # 156 & 157
PCBs # 70, 61, 74 & 76	PCBs # 171 & 173
PCBs # 93 & 100	PCBs # 180 & 193
PCBs # 98 & 102	PCBs # 198 & 199