TRENDS IN POLYCHLORINATED DIBENZO-*P*-DIOXIN/DIBENZOFURAN (PCDD/F) CONCENTRATIONS IN LAKE ONTARIO SALMONIDS COLLECTED FROM 1978 TO 1999

O'Keefe PW[1], Connor S[1], Hilker D[1], Skinner L[2], Sloan R[2] and Storm R[1]

[1]Wadsworth Center, New York State Department of Health, P.O. Box 509, Empire State Plaza, Albany NY 12201, USA; [2]New York State Department of Environmental Conservation, Division of Fish, Wildlife and Marine Resources, 625 Broadway, Albany NY 12233, USA

Introduction

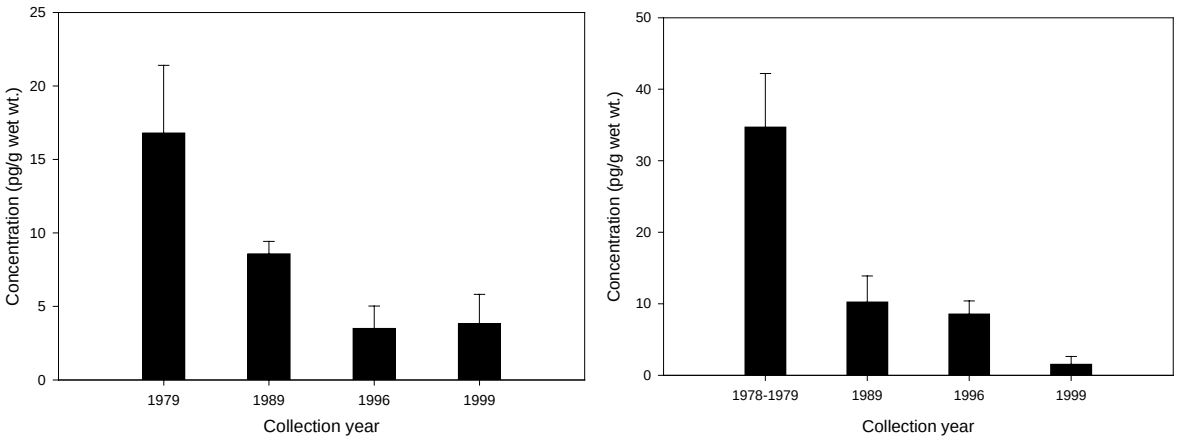
Polychlorinated dibenzo-*p*-dioxins/dibenzofurans (PCDDs/Fs) are present in biota[1] and sediments[1] in all the Great Lakes as a result of atmospheric deposition. However there appear to be additional point source inputs to some of the lakes, in particular inputs of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD) to Lake Ontario[1,2]. Atmospheric inputs have declined since the advent of legislation to control emissions from power plants etc. and point sources also have been controlled and, in many cases, eliminated. As a consequence of these control measures it was found that PCDD/F concentrations in lake trout[3] and herring gull eggs[4] from the Great Lakes declined in specimens collected between 1977 and the mid-to late 1980s. However these studies and a more recent lake trout study[5] have also shown that that since the late 1980s the downward trend is smaller and, to a certain extent, concentrations appear to have stabilized. The three major PCDD/F congeners that have been identified in Lake Ontario biota are: 2,3,7,8-TCDD; 2,3,7,8-TCDF and 2,3,4,7,8-PeCDF. In the study reported here we examine the concentration changes for these three congeners in chinook and coho salmon and rainbow trout (steelhead) collected at the same location in Lake Ontario during 1978/80, 1989, 1996 and 1999.

Materials and Methods

Rainbow trout samples were collected by electro-fishing in the Salmon River estuary, and chinook and coho salmon were collected in the inlet flume of the Salmon River fish hatchery prior to spawning. In 1996, additional coho salmon were collected in April and May by electro-fishing at Sandy Creek. The individual fish of each species were determined to be between two and three years old, and between three and nine fish were available per species per time point. Ground tissue was prepared from skinned fillets. While all the fish were extracted by Soxhlet (1999 collection) or by high speed homogenization with either methylene chloride or methylene chloride/hexane mixtures, there were some variations in the methods used to clean up and analyze the extracts from the fish collected at the different time points. Fish from the 1978/80 collection were cleaned up by a combination of MgO/Celite, alumina and Florisil chromatography. These extracts were then subjected to reverse-phase and normal phase HPLC with final analysis of 2,3,7,8-TCDD by capillary GC/high resolution MS (Kratos MS 50)[2]. Extracts from the fish samples collected in 1989 and 1996 were partitioned with sulfuric acid to remove lipids, and cleaned up by a semi-automated chromatography system consisting of acid alumina, graphitized carbon and neutral or acid alumina columns coupled in series[6]. The fish extracts from the 1999 collection were cleaned up by an FMS Power-Prep system (Fluid Management Systems Inc., Waltham, MA, USA), a commercial version of the semi-automated chromatography system described above. The columns in this system were: (1) multi-layer silica (acidified, basic and neutral), (2) basic alumina and (3) graphitized carbon. Both 2,3,7,8- and non-2,3,7,8 substituted PCDDs/Fs were recovered in the extracts processed by the FMS system while only 2,3,7,8-substituted congeners were recovered from the semi-automated system. Analysis of tetra- to octa-CDD/F compounds was accomplished by capillary GC (60m DB5 or DBXL columns)/low resolution MS (Hewlett Packard 5971 MSD). Lipids were determined gravimetrically after removal of solvent from extracts.

Results

The fish samples collected between 1978 and 1980 were analyzed only for 2,3,7,8-TCDD while fish collected in 1989, 1996 and 1999 were analyzed for tetra- to octa-CDDs/Fs. Consequently, in the case of 2,3,7,8-TCDD, it was possible to examine the concentration trend over a 20-year time period. It is apparent from the data shown in Figure 1 that there was a 2- to 3- fold decrease in 2,3,7,8-TCDD concentrations in chinook salmon and rainbow trout between 1978/79 and 1989. Since 1989 there has been a more gradual decline in 2,3,7,8-TCDD levels in the rainbow trout followed by a stabilization after 1996 (a similar temporal trend was found for 2,3,7,8-TCDD concentrations in coho salmon). In chinook salmon there was another 4-fold decline in 2,3,7,8-TCDD concentrations between 1996 and 1999.

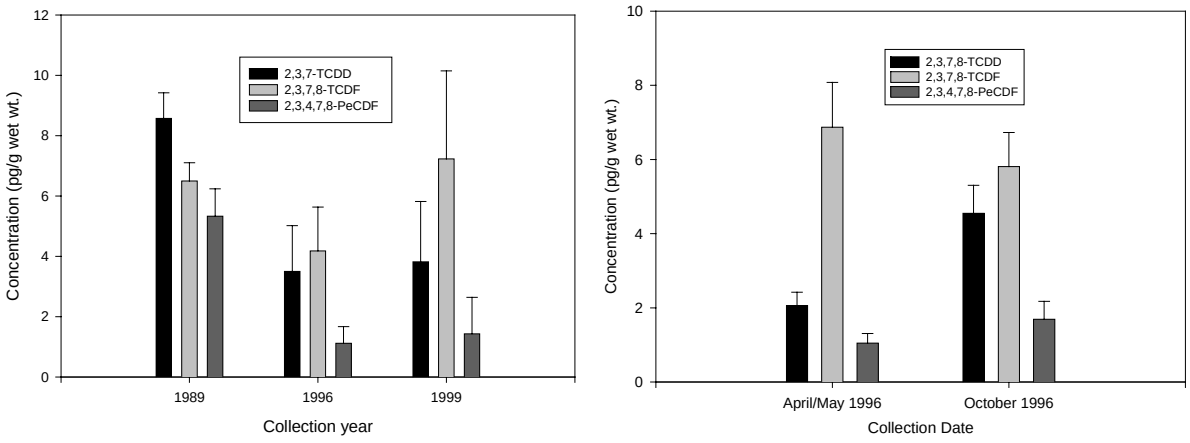


(a) (b)

Figure 1. Temporal trends in 2,3,7,8-TCDD concentrations in rainbow trout (a) and chinook salmon (b) collected from Lake Ontario at the Salmon River estuary (rainbow trout) and the Salmon River fish hatchery (chinook salmon).

While samples were only collected at four time points linear regression showed that the 2,3,7,8-TCDD concentrations in the three salmonid species were highly correlated with sampling year (r^2 values from 0.78 to 0.85 and *p*-values from 1.58E-09 to 1.24E-06). In a study where radionuclide dating was used for sectioning a sediment core from the Rochester basin of Lake Ontario, it was found that there was a 2,3,7,8-TCDD concentration of 68 pg/g dry wt in 1978 which had declined to 28 pg/g dry wt by 1987, the year in which the core was collected[7]. Therefore the 2-fold reduction in salmonid 2,3,7,8-TCDD concentrations between 1978/80 and 1989 was reflected in a similar reduction in the 2,3,7,8-TCDD concentration in sediments over the same time period. This was expected since it has been found that the lipid-based concentrations in fish of hydrophobic contaminants such as PCDDs/Fs can be related to the organic carbon-based concentrations of the same compounds in sediment by biota-sediment accumulation factors (BSAFs)[7]. The sediment data do not extend beyond the year 1987 and therefore we do not know if the stabilization of 2,3,7,8-TCDD concentrations found in the salmonids since 1989 has occurred for 2,3,7,8-TCDD in the sediments.

Samples collected in 1989, 1996 and 1999 were analyzed for tetra- to octa-CDDs/Fs. The following 2,3,7,8-substituted congeners were identified in the sample extracts: 2,3,7,8-TCDD; 1,2,3,7,8-PeCDD; 1,2,3,6,7,8-HxCDD; 2,3,7,8-TCDF; 1,2,3,7,8-PeCDF; 2,3,4,7,8-PeCDF; 1,2,3,4,7,8-HxCDF and 1,2,3,6,7,8-HxCDF. However only 2,3,7,8-TCDD; 2,3,7,8-TCDF and 2,3,4,7,8-PeCDF were found in the majority of the samples. Concentration data for these three congeners in rainbow trout are shown in Figure 2(a). It is apparent, during the time period of 1989 to 1999, that the concentrations of both 2,3,7,8-TCDD and 2,3,4,7,8-PeCDF decreased at faster rates than the concentration of 2,3,7,8-TCDF. As a result of the different rates of decline in congener concentrations the dominant congener in 1999 was 2,3,7,8-TCDF whereas 2,3,7,8-TCDD was the dominant congener in 1989. A similar pattern change was found to occur in chinook salmon although the pattern change was not readily apparent in coho salmon. The temporal change in the pattern for these three congeners was also found to occur in the Lake Ontario sediment core discussed above[7]. At one time there were significant releases of 2,3,7,8-TCDD to the Niagara River from landfills and industrial discharges in the Niagara Falls, NY area[1]. In a study of temporal trends of PCDDs/Fs in lake trout from Lake Ontario the declining proportion of 2,3,7,8-TCDD relative to 2,3,7,8-TCDF was attributed to reduced impacts of these discharges to the Niagara River, the main water source for Lake Ontario[3]. Previous studies conducted in our laboratory demonstrated that air samples collected downwind from industrial sources in Niagara Falls had elevated PCDD/F concentrations, particularly tetra-CDFs, compared to samples collected at an upwind control site[8]. It is possible that these and more distant atmospheric emissions are now the dominant sources of PCDDs/Fs to Lake Ontario rather than the releases of 2,3,7,8-TCDD to the Niagara River.



(a) (b)

Figure 2. Concentrations of, 2,3,7,8-TCDD; 2,3,7,8-TCDF and 2,3,4,7,8-PeCDF in (a) rainbow trout collected in the Salmon River estuary in 1989, 1996 and 1999 and in (b) coho salmon collected at two time points in 1996.

While the temporal trend data for the three PCDD/F congeners were all obtained from samples collected during the Fall (Autumn), in 1996 additional samples of coho salmon were obtained during the months of April and May. When the congener patterns for the two seasonal sampling events were compared we found that 2,3,7, 8-TCDD and, to a lesser extent, 2,3,4,7,8-PeCDF were enriched relative to 2,3,7,8-TCDF in the coho salmon collected in October (Figure 2(b)). Mobilization of lipids may play a role in the pattern change. The October samples were collected from fish at the point of spawning and the lipid concentration was 2.1 ± 1.5 % (mean ± s.d.) while the lipid concentration

of the fish collected in April/May was 8.3 ± 3.1 %. In North Sea flounder the lowest lipid concentrations occur in March, immediately after the spawning period, and the highest lipid concentrations are found in the Fall (Autumn). In a study where the March/December concentration ratios were determined for individual PCB congeners in North Sea flounder it was found that there was a significant positive correlation between the ratios and the log K_{OW} (K_{OW} = N-octanol/water partition coefficient) values for the congeners[9]. Since it has been reported that 2,3,7,8-TCDF and 2,3,7, 8-TCDD have log K_{OW} values of 5.82 and 7.02 respectively[10], our finding that 2,3,7, 8-TCDD is enriched relative to 2,3,7,8-TCDF under lipid-depletion conditions was expected based on the results of the flounder study. The log K_{OW} of a compound is in fact a parameter closely related to the compound's lipophillic potential. When lipid content is reduced as a result of migration, spawning etc. the results of both studies suggest that compounds with high log K_{OW} values are more readily retained in fish lipid than compounds with low K_{OW} values. Metabolism may also influence the relative concentrations of the two congeners. When lipids are mobilized there is the potential for PCDD/F transportation to the liver via the bloodstream. Available data from studies with rainbow trout suggest that 2,3,7,8-TCDF is more readily metabolized by hepatic Cytochrome P450 enzymes than 2,3,7,8-TCDD[11,12]. Additional and more extensive studies will be required to provide a detailed explanation of the influence of seasonal sampling on PCDD/F congener patterns in salmonids.

Acknowledgments
Technical assistance with sample analysis was provided by Carol Meyer, Brigitte Bachner, Robert Donnelly, Kathleen Dillon and Thomas Papura.

References

1. Norstrom RJ. In: *Persistent Organic Pollutants in the Great Lakes,* Hites RA. (ed.), The Handbook of Environmental Chemistry, Vol 5, Water Pollution, Part N, Springer-Verlag, Heidelberg, 2006:71.
2. O'Keefe P, Meyer C, Hilker D, Aldous K, Jelus-Tyror B, Dillon K, Donnelly R, Horn E, Sloan. *Chemosphere* 1983;12:325.
3. Huestis SY, Servos MR, Whittle DM, van den Heuvel M, Dixon DG. *Environ Toxicol Chem* 1997;16:154.
4. Hebert CE, Norstrom RJ, Simon M, Braune BM, Weseloh DV, Macdonald CR. *Environ Sci Technol* 1994;28:1268.
5. Awad E, Fletcher R, Hayton A. *Organohalogen Comp* 2005; CD ID:1643.
6. O'Keefe PW, Smith RM, Hilker DR, Aldous K, Gilday W. In: *Chlorinated Dioxins and Dibenzofurans in the Total Enviroment II,* Keith LH, Rappe C, Choudhary G. (eds.), Butterworth Press, Boston, 1985:111.
7. Cook PM, Robbins JA, Endicott DD, Lodge KB, Guiney PD, Walker MK, Zabel EW, Peterson RF. *Environ Sci Technol* 2003;37:3864, additional core data provided by PM Cook.
8. Smith RM, O'Keefe PW, Aldous K, Connor S, Lavin P, Wade E. *Chemosphere* 1990;20:1447.
9. Eggens ML, Opperhuizen A, Boon JP. *Chemosphere* 1996;33:1579.
10. Burkhard LP, Kuehl DW. *Chemosphere* 1986;15:163.
11. Kleeman JP, Olson JR, Chen SM, Peterson RE. *Toxicol Appl Pharmacol* 1986;83:391.
12. Steward AR, Maslanka R, Pangrekar J, Kumar S, Sikka HC. *Toxicol Appl Pharmacol* 1996;139:418.