Biomagnification of Polychlorinated Naphthalenes and Dioxin-like PCBs in Lake Ontario Biota

Paul Helm[1], D. Michael Whittle[2], Gregg T. Tomy[3], Aaron T. Fisk[4], Chris Marvin[5]

[1]Ontario Ministry Of The Environment
[2]GLLFAS, Fisheries and Oceans Canada
[3]Freshwater Institute, Fisheries and Oceans Canada
[4]University of Georgia
[5]NWRI, Environment Canada

Introduction

Polychlorinated naphthalenes (PCNs) are persistent, bioaccumulative compounds[1,2] which have a planar structure and exhibit dioxin-like toxicity.[3-5] PCNs were used as dielectrics for flame resistance and insulation in capacitors and cables[1,2] and are in PCB mixtures[6] and combustion emissions.[7,8] The accumulation of PCNs in Great Lakes biota has been shown in fish from Lakes Superior, Michigan & Huron, and the Detroit River,[9] and in herring gull and cormorant eggs.[10] Biomagnification in the benthic food web (algae-Dreissenid mussel-round goby) in the St. Clair River area has also been investigated.[11] This study was undertaken to determine levels of PCNs in Lake Ontario biota and sediments, comparing to previous PCN reports and dioxin-like (DL) PCBs. Biomagnification of PCNs is assessed as a function of trophic level using stable nitrogen isotope measurements and compared to DL-PCBs in the Lake Ontario food web.

Materials and Methods

Sample collection / Preparation: Forage fish (alewife, rainbow smelt, slimy sculpin) and invertebrates (plankton, *Mysisrelicta*, *Diporeiahoyi*) were collected from western and northern Lake Ontario in September 2003. Lake trout were in the eastern basin of the lake in June & July of 2002. Walleye and lake whitefish from the Bay of Quinte were also collected in 2002. Fish samples were homogenized whole (trout, walleye, whitefish) or as composites (forage fish, 5-25 organisms). Invertebrate samples were composites of >200 whole organisms. Surface sediments were collected from the western, central, and eastern parts of Lake Ontario in 1998 and archived frozen.

Extraction / Clean-up: Wet tissues were mixed with hydromatrix, ^{13}C-PCN and ^{13}C-PCB recovery internal standards were added, and then extracted by automated solvent extraction with 50:50 dichloromethane (DCM):hexane. An aliquot of the extract was used for lipid determination by gravimetry while the remaining extract was cleaned using gel permeation chromatography and on Florisil. Sediments were mixed with sodium sulfate and extracted with DCM by Soxhlet. All samples were fractioned on silicic acid and mini carbon columns.[12]

Analysis: PCNs and DL-PCBs were analyzed by GC-MSD using a 60 m DB-5 and quantified using a Halowax PCN external standard mixture of known composition[13] and individual PCBs (81, 77, 118, 114, 105, 126, 156, 169).[12] Major coeluting PCN congeners (CNs-52/60, -66/67, and -64/68) were separated using the Rt-bDEXcst column on the GC-MSD.[14]

Results and Discussion

Levels: SPCN (tri-octaCN) concentrations (wet weight; ww) increased from net plankton (mostly zooplankton) to forage fish to predator fish (Figure 1). *Diporeia* & slimy sculpin were elevated relative to other invertebrates and forage fish, respectively. The concentrations in lake trout from Lake Ontario were greater than reported in Lakes Huron (1.0 ng/g ww), Michigan (1.2 ng/g ww), and Superior (0.35 ng/g ww), as well as Siskiwit Lake (0.25 ng/g ww).[9] Surface sediment concentrations of SPCN ranged from 21-38 ng/g dry weight with octaCN, the heptaCNs, and the hexaCNs as the dominant homologs.

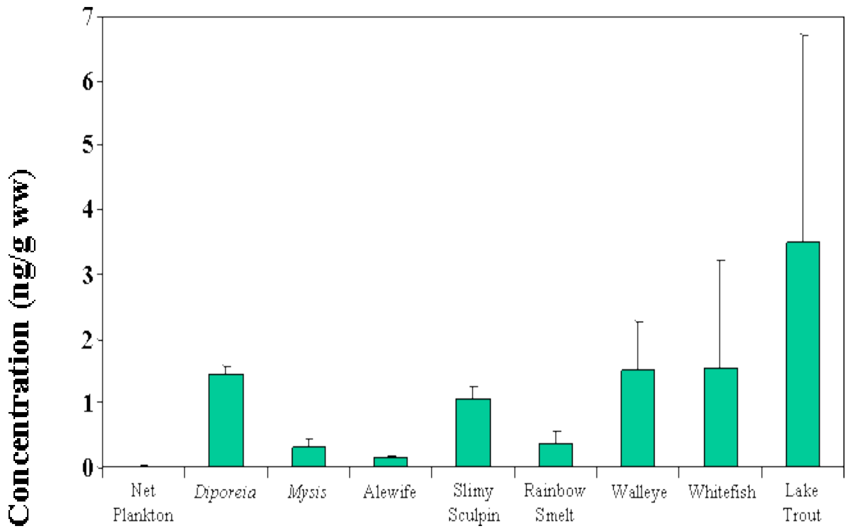

Figure 1: Mean (±1 SD) SPCN concentrations (wet weight) in biota collected from Lake Ontario.

Biomagnification: Trophic magnification was examined for the invertebrate–forage fish–lake trout food web from Lake Ontario using the linear relation between the natural logarithm of concentration (lipid-adjusted; lw) and trophic level (TL) determined from nitrogen isotopes[15,16]

$$TL = 2 + (d^{15}N_{consumer} - d^{15}N_{net\ plankton})/3.8$$

$$\ln [conc] = b{*}TL - a$$

$$\text{Trophic Magnification Factor (TMF)} = e^{b}.$$

Figure 2 shows this relation for SPCN with a TMF of 3.1 which is similar to the 3.0 found for SPCBs calculated in the same manner from the data of Kiriluk et al.[17] TMFs for individual PCN congeners which are more bioaccumulative, as well as the DL-PCBs, are listed in Table 1. *Diporeia* were excluded from TMF regressions for PCNs as they fell well above the regression line, but could not be excluded for the DL-PCBs. This may indicate that the benthic pathway is a more important source of PCNs to the upper trophic levels than for the DL-PCBs. A similar observation was made for PFOS in *Diporeia* and slimy sculpin in Lake Ontario.[16]

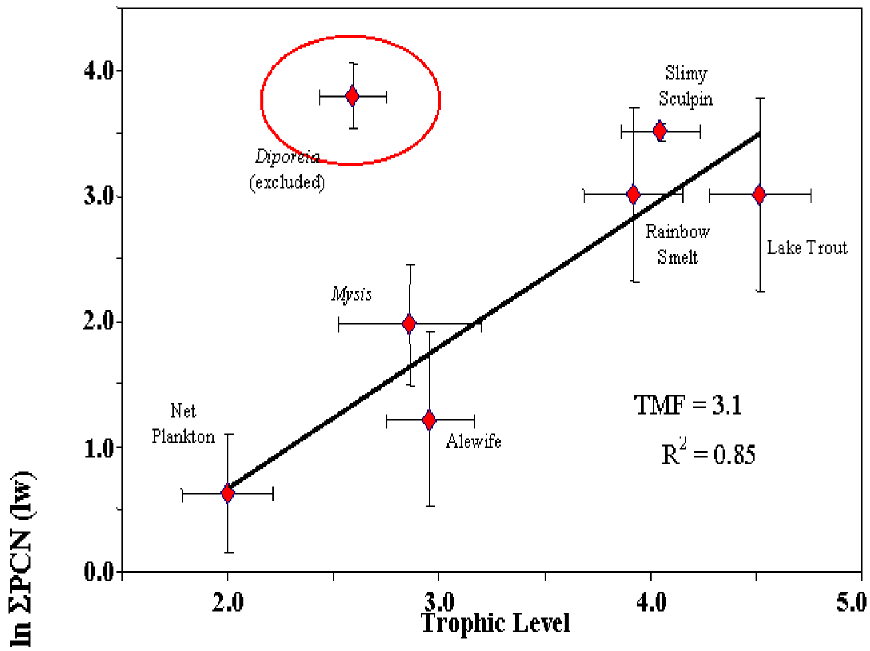

Figure 2: Plot of mean concentration (lw) (± 1 SD) vs. trophic level (± 1 SD) for SPCN (tri-octaCN). *Diporeia* was excluded from the regression.

Table 1: TMFs, representing accumulation through the entire food web, and BMFs (lake trout:weighted diet [90% alewife, 7% rainbow smelt, 2% slimy sculpin][16] predator-prey accumulation) for several PCNs and the DL-PCBs.

PCN #	TMF	BMF	PCB #	TMF	BMF
42	2.4	5.5	81	3.0	5.6
52	4.4	7.5	77	2.1	5.7
60	4.1	7.4	118	4.4	6.9
61	3.5	8.6	114	6.3	19.8
66	5.6	6.1	105	4.7	8.5
67	5.3	6.7	126	3.8	7.8

64	6.8	7.4	156	5.7	12.5
68	6.9	7.7	169	4.1	8.6
69	4.1	6.9			

Biomagnification factors (BMFs; concentrations (lw) in predator / prey) for lake trout:weighted diet also indicate biomagnification. BMFs for selected PCN congeners are compared to those for the DL-PCBs in Table 1. BMFs for this relationship were generally similar between the bioaccumulative PCNs and the DL-PCBs. However, in other predator-prey scenarios (e.g. lake trout:rainbow smelt & lake trout:slimysculpin), PCN BMFs were lower than DL-PCB BMFs, suggesting that uptake behaviour of PCNs and DL-PCBs differ, or they accumulate from different sources. For example, biota-sediment accumulation factors (BSAFs) were approximately 0.1 for the PCNs, but were twice that for the DL-PCBs.

Relative Toxicity: Dioxin toxic equivalents (TEQ) from PCNs and DL-PCBs were compared using assay-determined relative potencies (REPs).[3-5,18] PCNs contributed 12-22% of PCN + DL-PCB TEQ in lake trout. PCN TEQ contributions were greater in the benthic organisms (*Diporeia* and slimy sculpin), indicating sediment is an important contributor of the more toxic PCNs.

Conclusions

PCNs are present in Lake Ontario biota with higher lake trout concentrations than reported elsewhere in the Great Lakes. PCNs biomagnify in the Lake Ontariofoodweb, but the role of the benthic pathway may vary between PCNs and DL-PCBs. PCNs merit a more complete assessment of their source areas and contributions to dioxin toxicity in the lower Great Lakes.

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References

1. Falandysz J. (1998) *Environ. Pollut*. 101:77-90.

2. Jakobsson E. and Asplund L. (1999) in: New Types of Persistent Halogenated Compounds (Paasivirta J., Ed.), Springer-Verlag, 97-126.

3. Hanberg A., Ståhlberg M., Georgellis A., de Wit C. and Ahlborg U. (1991) *Pharma. Toxicol.* 69:442-449.

4. Blankenship A., Kannan K., Villalobos S., Villeneuve D., Falandysz J., Imagawa T., Jakobsson E. and Giesy J. (2000) *Environ. Sci. Technol.*34:3153-3158.

5. Villeneuve D, Kannan K., Khim J., Falandysz J., Nikiforov V., Blankenship A. and Giesy J. (2000) *Arch. Environ. Contam. Toxicol.* 39:273-281.

6. Yamashita N., Kannan K., Imagawa T., Miyazaki A. and Giesy J. (2000) *Environ. Sci. Technol.* 34:4236-4241.

7. Helm P. and Bidleman T. (2003) *Environ. Sci. Technol.* 37:1075-1082.

8. Lee R., Coleman P., Jones J., Jones K. and Lohmann R. (2005) *Environ. Sci. Technol.* 39:1436-1447.

9. Kannan K, Yamashita N, Imagawa T, Decoen W, Khim J., Day R., Summer C. and Giesy J. (2000) *Environ. Sci. Technol.* 34:566-572.

10. Kannan K., Hilscherova K., Imagawa T., Yamashita N., Williams L. and Giesy J. (2001) *Environ. Sci. Technol.* 35:441-447.

11. Hanari N., Kannan K., Horii Y., Taniyasu S., Yamashita N., Jude D. and Berg M. (2004) *Arch. Environ. Contam. Toxicol.* 47:84-93.

12. Helm P., Bidleman T., Stern G. and Koczanski K. (2002) *Environ. Pollut.* 119:69-78.

13. Harner T. and Bidleman T. (1997) *Atmos. Environ.* 31:4009-4016.

14. Helm P., Jantunen L., Bidleman T. and Dorman F. (1999) *J. High Resol. Chromatogr.* 22:639-643.

15. Tomy G., Budakowski W., Halldorson T., Whittle M., Keir M., Marvin C., MacInnis G. and Alaee M. (2004) *Environ. Sci. Technol.* 38:2298-2303.

16. Martin J., Whittle M., Muir D. and Mabury S. (2004) *Environ. Sci. Technol.* 38: 5379-5385.

17. Kiriluk R., Servos M., Whittle M., Cabana G. and Rasmussen J. (1995) *Can. J. Fish. Aquat. Sci.* 52:2660-2674.

18. Giesy J., Jude D., Tillitt D., Gale R., Meadows J., Zajieck J., Peterman P., Verbrugge D., Sanderson J., Schwartz T. and Tuchman M. (1997) *Environ. Toxicol. Chem.* 16:713-724.