Polychlorinated Naphthalenes in the Great Lakes Environment

Paul Helm[1], Kurunthachalam Kannan[2], Terry F. Bidleman[3]

[1]Ontario Ministry Of The Environment
[2]New York State Department of Health
[3]Meteorological Service of Canada, Environment Canada

Introduction

Polychlorinated naphthalenes (PCNs) were found to have desirable insulating and flame resistance properties resulting in their use as dielectrics in capacitors, insulation in cable coverings, but were also used in dye-making and as fungicides in wood.[1,2] They are persistent in the environment, tend to bioaccumulate, and have dioxin-like toxicity.[1,2] This paper summarizes recent and past findings on the sources and environmental occurrence of PCNs in the Great Lakes environment and is based on a chapter in an upcoming book reviewing the contamination of the Great Lakes basin by several persistent organic pollutants (POPs).

Toxicity

Occupational and accidental exposures of humans to PCN mixtures have demonstrated the highly toxic nature of these compounds.[3] Observed effects include skin lesions (chloracne) and liver degeneration (yellow atrophy), and PCNs were the first agents to cause widespread outbreaks of chloracne in the United States. For example, chloracne and yellow atrophy were documented among individuals employed at a cable manufacturing facility using Halowax in West Chester County, New York, in the 1940s.[4]

Chronic exposure to PCNs, particularly penta- and hexaCNs, induce hepatic ethoxyresorufin *O*-deethylase (EROD) and aryl hydrocarbon hydroxylase (AHH) activities and oxidative stress resulting in increased lipid peroxidation, decreased hepatic vitamins A and E, and decreased catalase and superoxidedismutase activities in exposed laboratory animals.[5] The potencies of several PCN congeners have been determined using *in vitro* enzyme assays relative to 2,3,7,8-TCDD to allow for estimates of TEQ due to PCNs.[6,7,8] These relative potencies (REPs), up to 0.001 to 0.004 for several congeners (hexaCNs-66, -67, -68, -69, -70, -63, and heptaCN-73) are similar to or greater than many of the dioxin-like (DL) PCBs as determined in similar assays.[9]

Sources to the Great Lakes

The dominant source of PCNs in the Great Lakes is the past usage of technical Halowax mixtures. Evaporative losses, discharges, and spills/leaks during production, use, and disposal of products containing Halowaxes resulted in emissions into the air and water of the region. Ongoing emissions from old in-use materials and environmental cycling from reservoirs such as soils and sediments are likely factors in current environmental levels. PCNs are also present in Aroclor PCB mixtures (5-67 mg/g)[10], which were used in the Great Lakes region, and are formed and/or emitted during industrial processes like chlor-alkali production[1,2] and by combustion such as in municipal waste incinerators (MWI).[11,12]Flyashes from a MWI, a cement kiln, a hospital waste incinerator, and an iron sintering plant from the Great Lakes area all contained PCNs.[13]

The variation of congener profiles between technical PCN mixtures and combustion flyashes has been reported.[14] Certain congeners are not detected or are trace components of technical PCN and PCB mixtures but are formed in combustion processes, while others are present at significant proportions in technical mixtures as well as in combustion sources. The presence and/or relative enhancement of these combustion marker congeners (e.g. triCNs-13, -26, tetraCNs-44, -29, pentaCN-54, hexaCNs-66/67 and -70) in sample profiles are indicative of combustion source contributions. This has been observed in the Great Lakes as congeners CN-44, -29, and -54 were present in sediments in the upper Detroit River area.[15] The presence/enhancement of many combustion-related congeners in air

samples from north Toronto suggested a strong influence of combustion sources, estimated to be contributing up to 50% of PCNs in the air.[13]

Occurrence in the Great Lakes

Several monitoring studies undertaken in the past decade within the Great Lakes region have included PCNs, allowing spatial trends to emerge which are similar to many other POPs in the basin (Figure 1). PCN concentrations in several media (air, sediment, fish) are greater in the more intensely developed areas of the Great Lakes region.

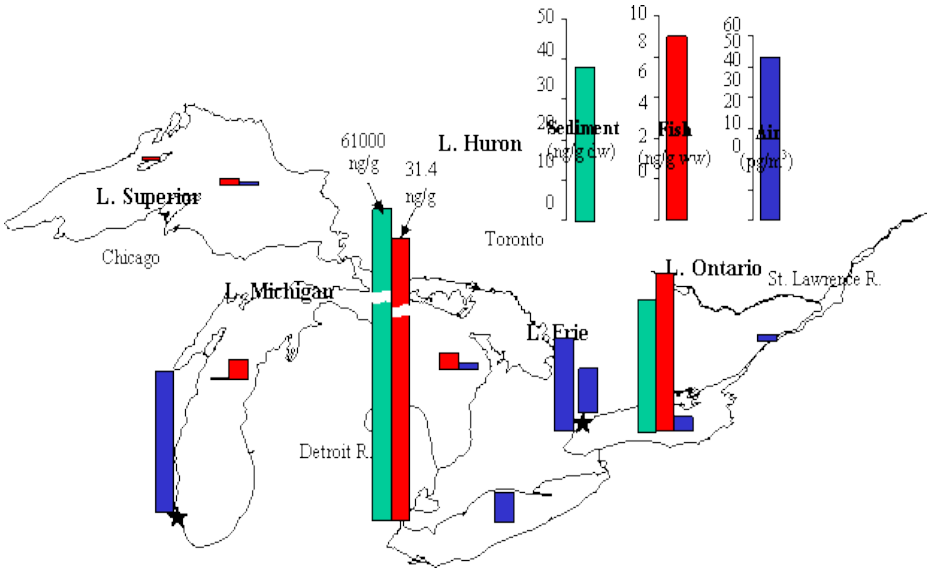

Figure 1: Spatial distribution of PCNs in air,[13,16,17] sediment,[15,18,19,20] and predator fish[20,21] in the Great Lakes region. Bars represent mean air concentrations (tetra-octaCNs; pg/m^3), maximum sediment concentrations (ng/g dw),

and maximum fish concentrations (ng/g ww).

Air: PCNs have been measured in air over the Great Lakes[17] and from urban,[13,16] suburban, semirural and rural sites[22] around the Great Lakes region. Concentrations (tri-octaCNs) were greater in urban areas such as Chicago (23-378 pg/m^3)[16] and Toronto (7-84 pg/m^3)[13,16] and lower over Lake Ontario (9-18 pg/m^3) and a rural area near Cornwall, ON (6.5 pg/m^3).[17] Tetra-octaCN concentrations were lower over Lakes Superior and Huron than Lakes Ontario and Erie which have greater populations and more development in their basins.[17] Passive air sampling within the region has demonstrated a similar spatial trend from urban to rural/remote areas.[22]

PCN homolog distributions in air were dominated by the tri- and tetraCNs. Penta- and hexaCN homolog contributions decreased in more remote locations (upper St. Lawrence R., Lake Superior), indicating long range transport favouring the less chlorinated homologs.[17] Congener profiles for air over the lakes showed the presence of combustion markers (tetraCNs-44, -29, and pentaCN-54), indicating combustion contributions, but that evaporative sources of technical mixtures were dominant.[17] A site in north Toronto was more strongly influenced by combustion sources, particular during fall/winter when cooler temperatures reduce the contributions of evaporative sources.[13]

Water: PCNs have been measured in Great Lakes water in only one study. Concentrations for triCNs and tetraCNs ranging from 12-19 pg/L and 4-8 pg/L, respectively, were reported in surface waters from Lake Ontario in June 2000.[17]

Sediments: The Detroit River area from Lake St. Clair to the mouth at Lake Erie has been well studied for PCNs in sediment[15,18] and suspended sediment.[19] The most highly contaminated area is the Trenton Channel, with up to 61 mg/g dry weight [dw] observed in surface sediments[18] and 8 mg/g dw in suspended sediments,[19] which has been impacted by industrial discharges. Concentrations were much lower in surface sediments from Lake Michigan (0.3-0.8 ng/g dw)[15] and Lake Ontario (21-38 ng/g dw).[20] The penta- and hexaCN homologs were prevalent in Detroit River sediments and suspended sediments, implicating inputs of Halowax materials as the major sources.[15,18,19] The presence of combustion marker congeners were noted in the upper Detroit River sediments indicating some inputs of these sources.[15] The homolog profile in Lake Ontario sediments differed with a prevalence of octaCN followed by the hepta- and hexaCNs.[20]

Biota: Kannan et al. (2000) first reported on PCN levels in fishes from the Great Lakes region, finding SPCN concentrations ranging 240-370 pg/g wet weight (ww), 1200 pg/g ww, and 980-1100 pg/g ww in Lakes Superior, Michigan, and Huron, respectively.[21] Lake trout from Lake Ontario ranged from 1300-9000 pg/g ww.[20] The greatest concentrations were in fish from the Detroit River (1300-31000 pg/g ww) and congener profiles in these fish reflected Halowax contamination.[21] PCN concentrations and congener profiles were also determined in the eggs of fish-eating birds (herring gulls [83-1300 pg/g ww] and double-crested cormorants [380-2400 pg/g ww]) from Lakes Superior and Huron.[23] Profiles were dominated by CN-66/67 in herring gull eggs and CNs-42, -52/60, -61, and -66/67 in cormorant eggs.

Relative Toxicity: Using REPs determined from *in vitro* assays, the toxic potential of PCNs has been compared to that of PCBs and/or PCDD/Fs in several media from the Great Lakes region. PCNs contributed 64-68% to PCN+PCB TEQ in urban Chicago[16] and downtown Toronto[13] air. In sediments[15] and suspended sediments[19] from the Detroit River area, PCNs were the greatest contributors to TEQ amongst PCBs and PCDD/Fs. In Lake Ontario sediments, 85% of the PCN+PCB TEQ was due to PCNs.[20] In fish, PCNs accounted for >50% and 12-22% of the PCN+PCB TEQ from the Detroit River[21] and Lake Ontario lake trout,[20] respectively. In herring gull and cormorant eggs, PCNs contributed ~2% of PCN, PCB, and PCDD/F TEQ.[23]

Biomagnification

The biomagnification of PCNs have been assessed in two recent studies in the Great Lakes region. Hanari et al. found biomagnification factors (BMF, lipid normalized concentration in predator/prey) ranging from 3-10 in for zebra mussels:algae in the St. Clair River but magnification was not observed from the mussels to the round goby.[24] Helm et al. examined trophic magnification in a Lake Ontario food web, relating concentrations to the trophic level as

determined by stable isotopes of nitrogen.[20]Trophic magnification factors (TMFs) ranged from 2.9 to 6.9 for the most bioaccumulative congeners. These were similar to TMFs for the DL-PCBs.[20] As a general trend,biomagnificationfavours the penta- and hexaCNs as trophic level increases (Figure 2). The congener profile also shifts, favouring congeners that are less subject to metabolism having no adjacent unsubstituted carbons, particularly CNs-42, -52/60, and -66/67.[1,20,21,23,24]

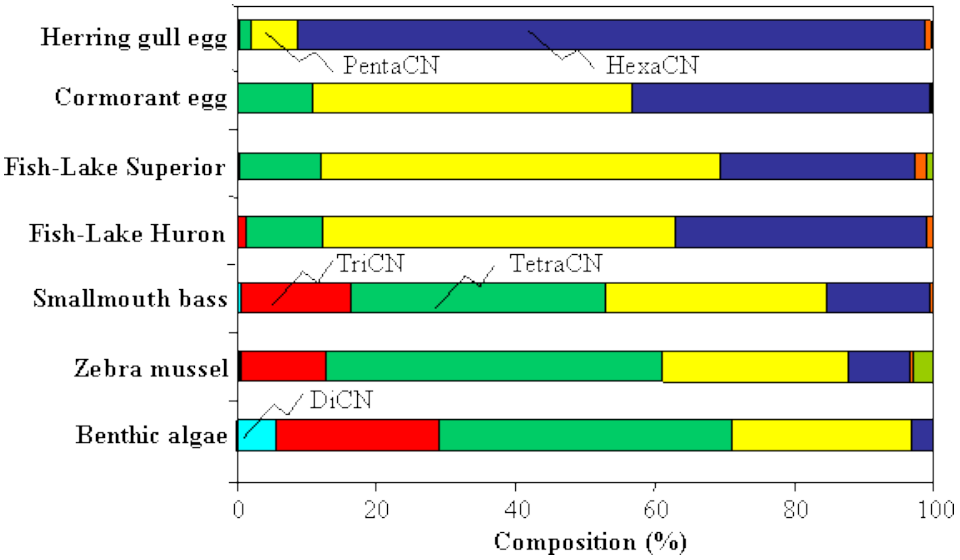

Figure 2: Shift in homolog composition resulting from biomagnification of the penta- and hexaCNs with increase in trophic level.[21,23,24] Note smallmouth bass, zebra mussels, and benthic algae are from the St. Clair River.

Conclusions and Recommendations

PCNs are present in environmental media throughout the Great Lakes basin and biomagnify through its aquatic food webs. Concentrations in air, sediment, and fish are highest in the lower Great Lakes where the influence of sources from urban, industrialized areas is apparent. Relative toxicity comparisons indicate that in many media and locations, PCNs may be as important as the DL-PCBs in TEQ contributions. A more detailed assessment of sediment concentrations throughout the region is merited to identify other potential PCN “hotspots” besides the Detroit River area. The contributions of combustion sources, both currently and in retrospect, should be considered further. Downstream impacts of PCN contamination on biota of the St. Lawrence Estuary is also an area for further study.

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. Hayward D. (1998) *Environ. Res.* 76A : 1-18.

4. Ward E., Ruder A., Suruda A., Smith A., Fessler-Flesch C. and Zahm S. (1996) *Am. J. Indust. Med.* 30: 225-233.

5. Mantyla E. and Ahotupa M. (1993) *Chemosphere* 27: 383-390.

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

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

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

9. 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.

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

11. Oehme M., Manø S. and Mikalsen A. (1987) *Chemosphere* 16: 143-153

12. Abad E., Caixach J. and Rivera J. (1999) *Chemosphere* 38: 109-120.

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

14. Imagawa & Yamashita (1994) *OrganohalogenCmpds.* 19: 215-218.

15. Kannan K., Kober J., Kang Y., Masunaga S., Nakanishi J., Ostaszewski A. and Giesy J. (2001) *Environ. Toxicol. Chem.* 20: 1878-1889.

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

17. Helm P., Jantunen L., Ridal J. and Bidleman, T. (2003) *Environ. Toxicol. Chem.* 22: 1937-1944.

18. Furlong E., Carter D. and Hites R. (1988) *J. Great Lakes Res*. 14: 489-501.

19. Marvin C., Alaee M., Painter S., Charlton M., Kauss P., Kolic T., MacPherson K., Takeuchi D. and Reiner E. (2002) *Chemosphere* 49: 111-120.

20. Helm P., Whittle M., Tomy G., Fisk A. and Marvin C. (2005) *Organohalogen Cmpds.* (this volume).

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

22. Harner T., Shoeib M., Gouin T. and Blanchard P. (2005) *Organohalogen Cmpds.* (this volume).

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

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