Dioxins in the Great Lakes

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Introduction

The story of 'dioxins', polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs), in the Great Lakes began in the late 1970s when 2378-TeCDD was reported to be in fish from the Tittabawassee River in Michigan downstream from a large DOW chemical complex in Midland, and in Lake Ontario fish[1]. The Love Canal waste dumpsite issue was gaining wide coverage in the press about the same time[2], so stories about 'the most toxic chemical known to man' created immediate consternation in the Great Lakes community. In the 25 years since dioxin concerns began in the Great Lakes, an enormous amount of information has been generated on sources, deposition, concentrations in sediments and biota, temporal trends and eco-/human toxicology of PCDD/Fs. In this overview, the history of 'dioxin' contamination and its impacts in the Great Lakes is summarized.

Herring Gulls and Fish

After the initial discovery of 2378-TeCDD in Great Lakes fish, the focus shifted to herring gull eggs. Concentrations of 2378-TeCDD in herring gull eggs from Saginaw Bay (Lake Huron) and Lake Ontario were found to be in the order of 100 ng/kg wet weight, 4-6 times higher than in eggs from Lakes Michigan, Huron and Erie in 1980[3], providing early evidence that Saginaw Bay and Lake Ontario were the areas of most concern. Higher chlorinated congeners were also found[4,5] . In the 1980s, geographical coverage was not comprehensive for surveys of PCDD/Fs in fish. Frequently only 2378-TeCDD was analyzed and sampling was done inconsistently so that comparison among areas was difficult. However, the findings were consistent with those from analysis of herring gull eggs. Stalling et al.[6] found no detectable PCDDs other than 2378-TeCDD in lake trout from from the upper Great Lakes, but all fish had 2378-TeCDF at concentrations of 5-34 ng kg^{-1}. In another study of PCDD/F concentrations in fish around the Great Lakes, Saginaw Bay, Lake Ontario and the Niagara Falls area were again recognized as primary areas of concern[5].

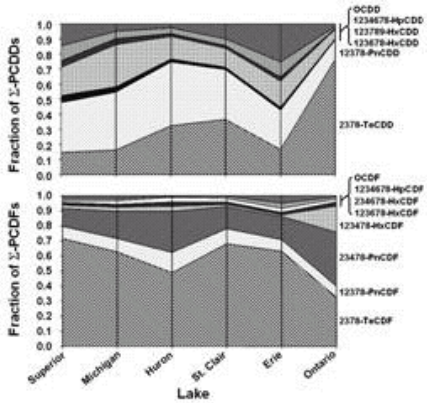

Figure 1. Fractional contribution of congeners to total PCDDs and PCDFs concentrations in lake trout from the Great Lakes in 1984 adapted from De Vault et al.[8]

De Vault et al.[7] carried out the first comprehensive, isomer-specific determination of PCDD/Fs in predator fish (lake trout and walleye, 1984) which had good geographical coverage among the Great Lakes, and compared the same or similar species in each area. Lake Superior and Lake Erie had the lowest total concentrations of both S-PCDDs and S-PCDFs, and lake Ontario had by far the largest concentration of PCDDs, primarily due to 2378-TeCDD. The overall ranking of concentrations in predator fish was PCDDs: Lake Ontario > Lake Huron » Lake Michigan » Lake St. Clair > Lake Erie » Lake Superior; PCDFs: Lake Ontario » Lake Michigan » Lake Huron > Lake St. Clair > Lake Erie > Lake Superior. The fractional contribution of the various congeners to S-PCDDs and S-PCDFs is shown in Figure 1. Lake Erie had a relatively high proportion of OCDD compared to the other lakes. The large contributions of 2378-TeCDD and 123478-HxCDF to S-PCDD/Fs in Lake Ontario predator fish are unique among the lakes. These results are consistent with a primary atmospheric source of PCDD/Fs to the Great Lakes modified by local inputs of particular congeners.

Sediments

Hites and coworkers documented the PCDD/F homolog concentrations in sediment cores from several areas in and around the Great Lakes. Profiles of PCDD/F relative concentrations in Saginaw Bay and southern Lake Huron, 1981, were compared to various sources[8]. The homolog profiles were similar among areas. The isomer makeup was similar to combustion, particularly the dominance of OCDD and OCDF. Depth profiles matched quite closely the USA production of chloro-aromatic compounds. It was concluded that most of the PCDD/Fs in these sediments originated from combustion of chlorinated organic compounds in various wastes.

Pearson et al.[10] determined the accumulation of PCDD/F homologs in sediment cores from Lake Superior, Lake Michigan and Lake Ontario in 1994. Historical accumulation rates, homolog compositions and atmospheric deposition rates all indicated a primarily atmospheric source to Lake Superior. The accumulation rate of PCDDs and PCDFs was 7-14 fold higher in Lake Ontario than Lake Michigan. It was concluded that >65-95% of the loading of both PCDDs and > 95% of PCDFs to Lake Ontario was non-atmospheric, probably via the Niagara River. Homolog profiles in Lake Ontario sediments were between that of pentachlorophenol and electrolytic sludge, which is highly enriched in PCDFs[11]. Sediment inventories to 1994 of S-PCDD/F were estimated to be 870 ± 330 kg in Lake Superior, 1700 ± 710 kg in Lake Michigan, and 5800 ± 800 kg in Lake Ontario[10].

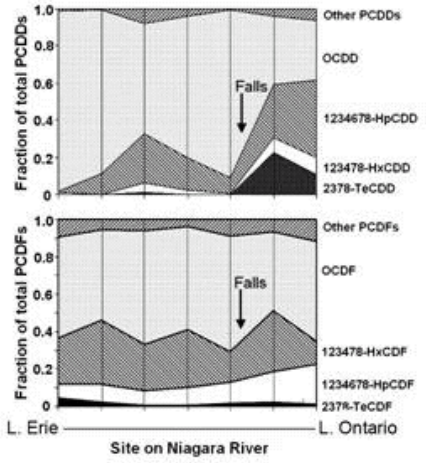

Figure 2. Fractional composition of major congeners in total PCDDs and PCDFs in Niagara River sediments in 2000, adapted from Richman[12]

PCDD/F concentrations in sediments along the Niagara River in 2000 were determined by Richman[12]. The fractional contribution of the major PCDD and PCDF congeners to their respective total concentrations in Niagara River sediments is shown in Figure 2. Despite large variations in concentration among sites, the PCDF pattern is remarkably similar along the river. Downstream from Niagara Falls, the proportion of 2378-TeCDD jumped to 11-23% of S-PCDDs as a result of runoff from the Hyde Park hazardous waste landfill, which is only 600 m from the Niagara River gorge. This congener pattern is also found in Lake Ontario sediments[13].

Besides the Niagara River, the Saginaw River and tributaries are the only other major point source of PCDD/Fs in the Great Lakes, to Saginaw Bay, Lake Huron[14]. Because the river flow is much lower than the Niagara River, and Saginaw Bay is shallow, most of this contamination does not reach Lake Huron proper. Hilscherovaet al.[15] surveyed PCDD/F concentrations in sediments and flood plain soils along the Tittabawasee River. Downstream of the Dow Chemical plant at Midland, MI. PCDD/F concentrations in sediments were 1-2 orders of magnitude higher than upstream, and similar to those in the lower reaches of the Saginaw River[14]. Flood plain soils downstream of Midland also had very high PCDD/F concentrations.

Sources

All evidence from analysis of biota and sediments points to Lake Ontario being by far the most seriously impacted by PCDD/F contamination among the Great Lakes, primarily from chlorine chemical industry along the Niagara River on the US side. Sediment and fish data are consistent with electrolytic sludge and trichlorophenol production wastes in the Niagara River being the primary sources of PCDD/Fs to Lake Ontario. Patterns of PCDD/Fs concentrations in herring gull eggs are also in agreement, taking into account bioavailability and bioaccumulation efficiencies of the various congeners[15]**.** Estimates of sediment inventories showed Lake Ontario to have 3.4 times more S-PCDD/Fs than Lake Michigan, and 6.6 times more than Lake Superior. By far the PCDD/F contaminant of most concern in Lake Ontario was 2378-TeCDD. Atmospheric sources of PCDD/Fs were dominant in all other areas except Saginaw Bay, and possibly Green Bay, Lake Michigan, although good PCDD/F data are not available for this area.

Temporal Trends

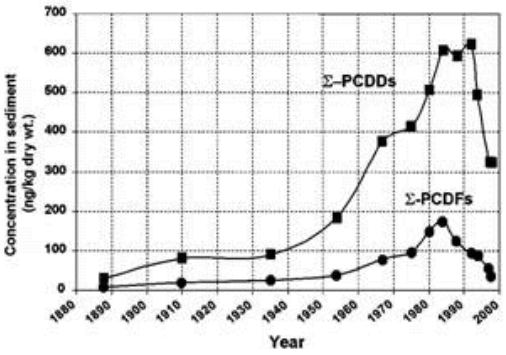

Figure 3. Trends in PCDD and PCDF concentrations in sediments, Siskiwit Lake, Isle Royale, Lake Superior, from Baker and Hites.[16]

Long-term temporal trends of PCDD/F atmospheric loading to the Great Lakes are most easily obtained from sediment cores. The information on historical PCDD/F atmospheric deposition rates in the Great Lakes basin was updated by analysis of two sediment cores obtained from Siskiwit Lake, Isle Royale, Lake Superior in 1998[16]. There were few notable changes in the pattern of homologs over the whole 1888-1998 period (Figure 3). Translated into depositional fluxes, these data indicated a slow increase in PCDD/F deposition between 1888 and 1940. The increases in PCDD/Fs deposition post 1940 tracked the production of chlorocarbons in North America[17]. Total PCDD/F depositional flux in Siskiwit Lake peaked at ca. 9.5 pg/cm^2/y about 1975-1980. Between 1980 and 1998, depositional fluxes of PCDD/Fs decreased ca. 50%, presumably because of abatement of combustion sources. If this trend continues, PCDD/F deposition to the Great Lakes from the atmosphere may be approaching the pre-chloro-organic production background level.

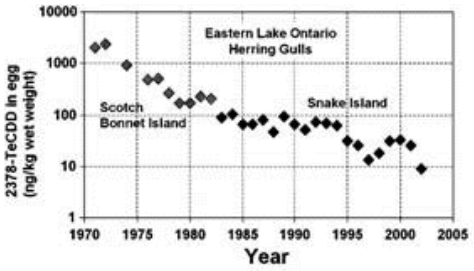

Figure 4. Temporal trend of 2378-TeCDD concentrations in herring gull eggs, Lake Ontario (Environment Canada).

Historical trends of 2378-TeCDD contamination in Lake Ontario, of most interest from a toxicological standpoint, are best demonstrated from analysis of archived herring gull eggs carried out by Environment Canada, summarized in Figure 4. Concentrations of 2378-TeCDD declined exponentially between 1971 and 1985, and remained relatively constant between 1985 and 1995. Trends post 1995 were erratic, but tending downwards, probably in response to various remediation efforts in the Niagara Falls area.

Effects

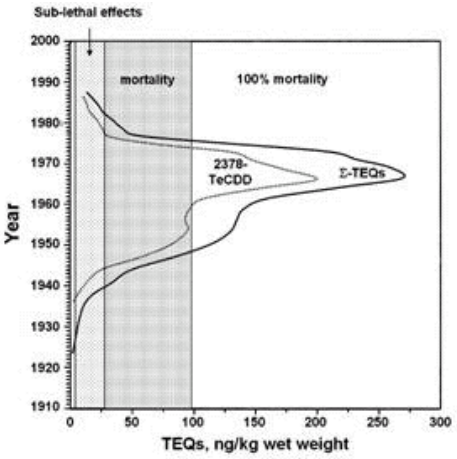

Figure 5. Temporal trends in early life-stage toxicity of 2378-TeCDD and TEQs in Lake Ontario lake trout eggs, adapted from Cook et al.[18]

While dioxin-like toxicity may be implicated in reproductive impairment and live deformities in fish-eating birds in various parts of the Great Lakes, much of this was probably due to exposure to certain PCB congeners, particularly CB126. There is statistical evidence that reproductive failure of herring gulls in Lake Ontario in the early 1970s may have been due in part to 2378-TeCDD exposure. However, the only clear-cut case that can be made for effects of PCDD/Fs in the Great Lakes is reproductive failure of lake trout in Lake Ontario. Figure 5 shows the reconstructed history of 2378-TeCDD and total TeCDD-equivalent concentrations in Lake Ontario lake trout eggs, relative to three levels of early life stage impairment: 100% mortality, some mortality, and sub-lethal effects[18]. Even before accounting for total TEQs, concentrations of 2378-TeCDD were sufficient to cause 100% mortality of lake trout fry between 1960 and 1975. Evidence presented by Cook et al.[18] strongly supports this contention. A thorough analysis of lake trout population trends, which included stocking of fry sporadically from the early 1900s to the 1940s, showed that natural reproduction ceased in 1950s, in congruence with the model. Furthermore, experimental studies showed decreasing mortality of fry hatched from Lake Ontario lake trout eggs in the 1977-91 period from ca. 50% to no incremental mortality, which was very close to the expected improvement based on the TEQ model.

Conclusions

There is no doubt that substantial progress has been made in reducing both atmospheric and land-based input of PCDD/F contamination in the Great Lakes. In many cases concentrations in fish and seabirds are close to 2 orders

of magnitude lower than historical peaks in the early 1970s. Atmospheric fluxes to the lakes seem to be approaching the pre-organochlorine production background level. Lake Ontario and, to a lesser extent, Lakes Michigan and Huron continue to have additional input from local sources above atmospheric input. However, there is some evidence that even in these lakes, the relative importance of direct input is diminishing. Thus, the dioxin issue is a mature one in the Great Lakes. Other organohalogen contaminants, primarily PCBs and PBDEs, are of greater concern at present.

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