ORGANOCHLORINE RESIDUES IN AND THEIR EFFECTS ON FISH AND WILDLIFE OF THE NORTH AMERICAN GREAT LAKES

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Introduction

The contamination of the North American Great Lakes with organochlorine compounds have been much studied and represent one of the areas where these compounds have been shown to cause population-level effects on wildlife. For nearly two centuries the Great Lakes have been the receiving waters for industrial and municipal wastes. The fact that the lakes were so large resulted in the commonly held thought that it was impossible to contaminate the lakes to a sufficient degree to cause adverse effects. As the populations increased and the complexity and magnitude of the industries grew it became apparent that it was indeed possible to contaminate the lakes to the extent that adverse effects would be observed. The experiences in the Great Lakes have resulted in greater understanding of the potential hazards of releasing persistent, bioaccumulative toxic compounds into the environment. Many of the compounds, which have been historical or current problems, are no longer manufactured or their use is restricted. Concentrations of the most problematic compounds such as DDTs and PCBs are currently declining, but the rate of decline seems to be decreasing such that it will be a long time before the concentrations in both abiotic and biotic compartments of the Great Lakes environment.[1]

Fish

Fish from all of the Great Lakes contain measurable concentrations of organochlorines (OCs) (Giesy and Snyder, 1998). In general fish from Lakes Ontario and Michigan tend to have the greatest concentrations of OCs. Concentrations of OCs remain the greatest in Lakes Michigan and Ontario. Fish from Lake Ontario contain the greatest concentrations of the insecticides Mirex, DDT and Dieldrin, while the concentrations of Toxaphene are greatest in fishes from Lake Superior. The least concentrations of most persistent, synthetic, chlorinated hydrocarbons are observed in fishes from Lakes. Concentrations of OCs in fish tissues have decreased by a factor of approximately 25 since maximum concentrations were reached in the lower Great Lakes in the mid 1970s. While there are differences among species and locations, in general the trends for persistent OCs are either decreasing or staying the same.

There is considerable difference of opinion in the literature as to the extent to which contaminants have affected Great Lakes fishes. Populations of several species have changed drastically from historical records. Historically, the reproductive success of salmonid fishes in the Great Lakes was much poorer than that of the same species raised on the west coast of the United States. These effects were often attributed to the effects of toxic substances, but it was difficult to demonstrate the cause-effect linkage. Specifically, historically, several fish species have exhibited reproductive deficits that were thought to be related to exposure to OCs. It is difficult to understand or predict the potential effects of complex environmental mixtures of OCs on biota. Not only are there a

great number of compounds, but the concentrations of the individual components change as a function of space and time. The mixture to which organisms are exposed at one time or at one location may be very different from that to which they are exposed at other times or locations

Of the many OCs measured in fish and their eggs, those that have most often been implicated in adverse effects are DDTs, PCBs and polychlorinated-dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs). The greatest concentrations of xenobiotics observed in fish eggs and thus likely potential causes of egg mortality were PCBs (11 mg/kg) and DDT (7 mg/kg)[1]. It was thought that DDT and PCBs were most likely responsible for the observed toxicity. Although both DDT and PCBs could cause lethality of lake trout eggs and fry in laboratory studies the concentrations required to cause 30-50% mortality were as much as 25 times greater than the concentrations observed in the eggs at that time. Thus, while concentrations of DDT observed historically in Great Lakes salmonids were in the range of the threshold for adverse effects, as determined in laboratory studies, it is unlikely that those concentrations were the major cause of adverse effects seen in the eggs of feral fishes from the great lakes.

Currently, it is felt that PCDD and PCDF and some of the non- and mono-ortho-substituted PCBs are responsible for blue-sac disease and impaired reproductive performance of lake trout in the lower Great Lakes.[1] Current concentrations of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin equivalents (TEQ), a measure of the total toxicity of a mixture relative to 2,3,7,8-TCDD, are near the threshold for mortality in lake trout fry. Furthermore, until recently, lake trout hatched from females collected from the Great Lakes suffered a relatively great incidence of blue-sac disease. Thus, it can be concluded that it is likely that historical concentrations of TEQ could have limited reproductive capacity of some of the more sensitive species of fish, such as lake trout. However, many other factors, such as over fishing, habitat loss, changes in genetic strains and effects of sea lamprey are likely to have contributed to declines in fish stocks.

Conclusions about the potential effects of OCs on fish reproduction are complicated by recent discoveries that some of the effects that have been attributed to OCs might be due to thiamine (vitamin B_1) deficiency. This causes a number of effects on adult fish, but the first effects are seen in the fry. It is known that some fishes, including herring, contain great activities of thiaminase, an enzyme that degrades.[1] It has been concluded, based on several studies that that Cayuga Syndrome, which is similar to the effects observed in Great Lakes fishes was caused by thiaminase in the herring that represented the major portion of the diet of the Cayuga Atlantic salmon. In addition to thiaminase, some commonly found xenobiotics can also cause a reduction in thiamine content of animals. Thus, the observed syndrome may be caused by either thiaminase activity or xenobiotics.

Birds

Current concentrations of PCDD, PCDF and PCBs in both Great Lakes, piscivorous birds and their prey are less than they were in the 1960s and 70s and some bird populations, such as double-crested cormorants and herring gulls, have made dramatic recoveries since that time. Populations of other species, such as common and Forster's tern continue to decline. The concentrations of TEQ in several species appear to be greater than the threshold for discernable, population-level effects at several locations around the Great Lakes. For instance, sub-populations of double-crested cormorants and Caspian terns in Saginaw and Green Bays continue to display abnormally great rates of developmental

deformities and embryo-lethality. In general, all of the populations of fish-eating birds of the Great Lakes are displaying symptoms of exposure to chlorinated chemicals at the biochemical level. These exposures are still causing lethality and deformities in embryos of all of the populations that we have examined. The observed effects are greater than what is observed in less contaminated populations off of the Great Lakes, however, these effects are translated into biologically significant population-level effects only in the more contaminated areas, such as Saginaw and Green Bays. The results of laboratory and field studies indicate that the lethality of and deformities in embryos of fish-eating birds of the Great Lakes are due to the toxic effects of multiple compounds, which express their effects through a common mechanism of action. In addition, the use of TEQ values seems to explain the observed effects better than single instrumental measurements of individual compounds.[2]

Mammals

In addition to the colonial, fish-eating water birds, several other populations have been reported to be affected by contaminants in Great Lakes fishes. Populations of mustelids, including mink (*Mustela vison*) and river otter (*Lutra canadensus*) have declined in the regions along the Great Lakes or along rivers which are not blocked by dams, to which fishes of the Great Lakes have access.[3] It is difficult to conduct risk assessment for mink because accurate information on their diets is limited. A number of researchers have reported that feeding fish from the Great Lakes has resulted in adverse effects on ranch mink.[4] But, in these studies not all of the compounds were quantified. Mink have been simultaneously exposed to a number of synthetic, halogenated compounds, including OC insecticides. Because the concentrations of a number of these compounds have been inter-correlated, it has been difficult to separate the effects and determine which of these compounds were most likely to have caused adverse effects in populations of wild mink or ranch mink which were fed fish from the Great Lakes. Several studies have been conducted to examine the effects of these compounds. Even historically, when the concentrations of OC pesticides in the tissue of fishes from the Great Lakes were greater, it has been concluded that it was unlikely that they were the cause of the effects which were observed when fish from the Great Lakes were fed to ranch mink.

Of all of the pollutants to which mink have been exposed, PCBs seem to have had the greatest impact.[3] Mink are one of the most sensitive organisms to the effects of PCBs.[4,5] In an attempt to determine if current concentrations of PCBs represent a risk to mink, hazard indices were calculated, assuming that mink ate only Great Lakes fish in their diet. Hazard indices for PCBs in fishes from the Great Lakes ranged from a minimum of 6.4 to a maximum of 83. Percent allowable consumption values for fish in the Great Lakes fish were all less than 100% and ranged from as little as 1.2% to as much as 19 %, depending on species and location.[3] Thus, there is no combination of fishes, which would result in a nonhazardous diet. The average allowable fish in the diet for all of the Great fish was 7.5%. It is unknown whether a large dose of PCBs over a short period of time would be as toxic as the same total dose received over a longer period of time. However, since the resident populations of fishes in the upstream locations currently contain some PCBs, the exposure of the mink to PCBs in the spawning salmon for a short period of time would be even more hazardous. The timing of the exposure of mink to the salmon is such that they could be eating large quantities of salmon for several months. The onset of the cold season is such that the carcasses of the salmon can persist along the shore for a long period of time. The late fall and early winter, which is the period when mink are mating and the females are pregmant and, thus, is a critical period of exposure to toxicants. During this period several species of anadromous salmonids migrate into the Michigan rivers to spawn. The coho and chinook salmon die soon after spawning and are deposited on the shores of the river. In this

way they could be a source of contaminants to mink. For this reason, we determined the duration that it would take a mink to receive its annual dose of TEQ or total PCBs. To make this calculation we assumed that mink would eat only the fish species of interest. This is not an unreasonable assumption, since it would be expected that mink would take the readily available fish as their entire diet if it were available. Reciprocity of toxic effects was also assumed. That is, it was assumed that a short-term dose would be equivalent to the same dose delivered over an entire year. This is a conservative assumption, since similar doses are generally more toxic when applied acutely. Thus, our calculations would tend to underestimate the toxicity of the acute doses. The annual allowable doses of total PCBs or TEQ were determined to be 657 ng of TEQ or 5,913 ug of total PCBs. If mink ate chinook salmon from the Great Lakes they would be exposed to 390 ug PCBs and 8.55 ng of TEQ per day, respectively.[3] Thus, it would take 15.1 or 77 days for mink to receive their total annual dose of PCBs or TEQ, respectively. At least for chinook salmon, the critical contaminant for the purposes of hazard assessment would be total concentrations of PCBs. Consuming chinook salmon for as little as two weeks would deliver the annual does to mink that would be expected to affect reproduction.

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