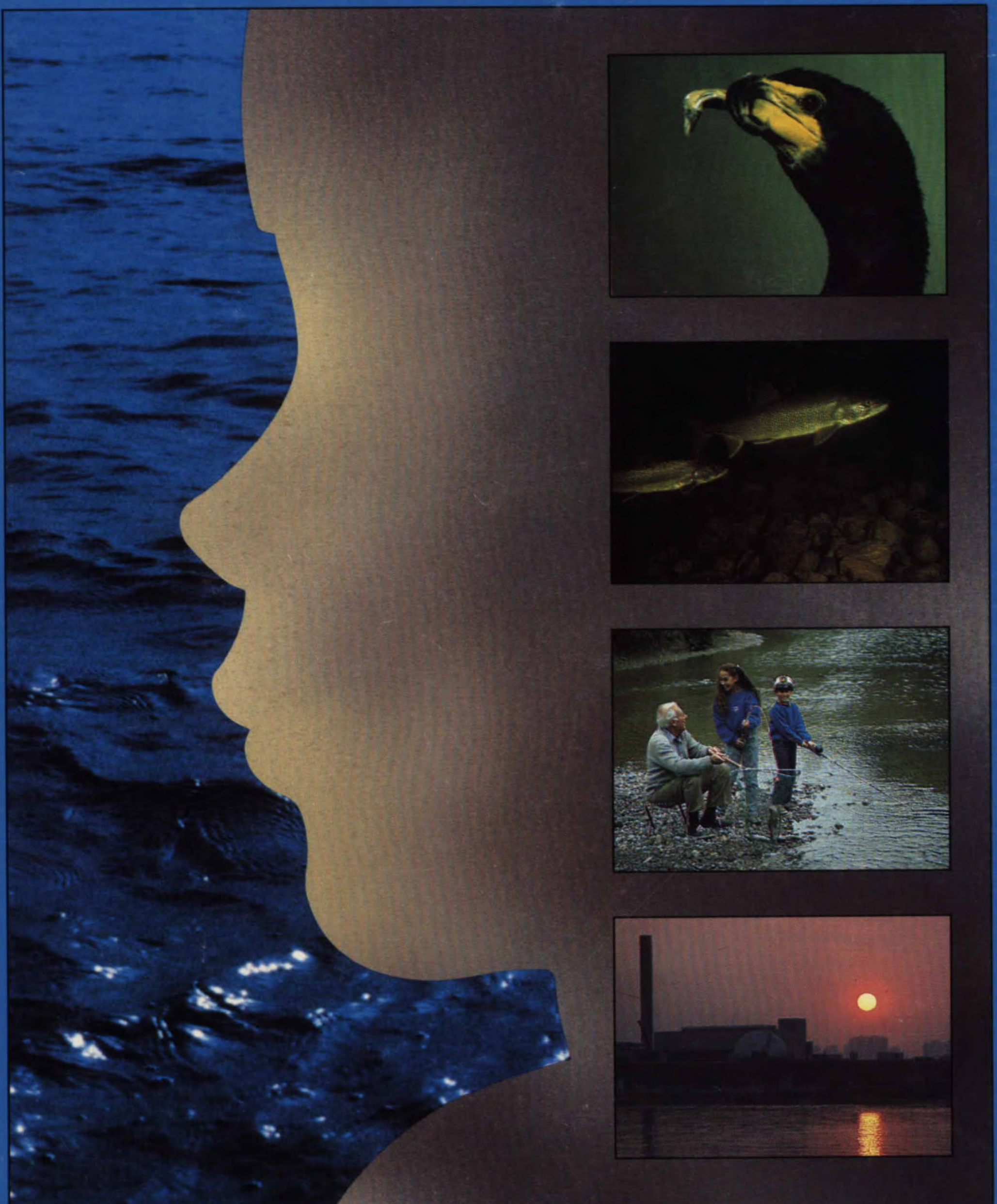
Toxic Chemicals in the Great Lakes and Associated Effects

VOLUME II — Effects



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TOXIC CHEMICALS IN THE GREAT LAKES AND ASSOCIATED EFFECTS

VOLUME II
EFFECTS

March 1991

ENVIRONMENT CANADA
DEPARTMENT OF FISHERIES AND OCEANS
HEALTH AND WELFARE CANADA

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INTRODUCTION

The increased commercial production and widespread use of synthetic toxic chemicals and metals since the 1940s have resulted in the contamination of the environment. While the presence of persistent toxic chemicals in the Great Lakes has been known for some time, concerns have been directed in recent years towards the environmental and human health effects of these chemicals.

During the past two and a half years, Environment Canada, the Department of Fisheries and Oceans, and Health and Welfare Canada have worked together to compile the scientific literature on the levels, trends and effects of persistent toxic chemicals in the Great Lakes basin. This has resulted in a two volume technical document entitled *Toxic Chemicals in the Great Lakes and Associated Effects*. Volume I of this report is divided into four parts. It summarizes data on the concentrations of toxic chemicals in water and sediments, invertebrates and fish, wildlife, and humans. Volume II also contains four parts. It reviews what is known about the effects of persistent toxic chemicals on fish, wildlife and people and presents a concluding synthesis which interprets the significance of the levels and effects information in the two volumes. Each part of the report begins with an executive summary.

Based on our knowledge of chemicals and their toxicology, a pattern is emerging that suggests that persistent chemicals in the environment have a significant effect on fish and wildlife species. We still know very little about the effects of a person's lifetime exposure to toxic organic chemicals and metals. Despite some uncertainties, it is clear that toxic chemicals are a threat to the entire ecosystem. As a result, the principle of "virtual elimination" of persistent toxic substances from the lakes has been adopted in the Canada-US Great Lakes Water Quality Agreement. The national governments of both countries together with the province of Ontario, the eight Great Lake states and the large municipalities are moving towards this goal using the regulatory processes, pollution prevention strategies and public education. In the meantime, government programs continue to monitor the health of the ecosystem, assess the extent to which it is impaired by toxic chemicals and develop means to reduce the impact of exposures.

TOXIC CHEMICALS IN THE GREAT LAKES AND ASSOCIATED EFFECTS

VOLUME II PART 1

EFFECTS OF CONTAMINANTS ON AQUATIC BIOTA

EXECUTIVE SUMMARY

The Great Lakes fishery has changed dramatically since 1900. Commercial catches of historically important species such as lake trout, whitefish, herring, blue pike and ciscoes have declined due to the combined effects of competition, exploitation, eutrophication and habitat destruction. In most of the Great Lakes, the commercial fishery for highly valued species has been replaced by smelt, chub, eels, yellow perch, white bass, bullhead, and suckers. To reverse these trends, fisheries agencies implemented fish stocking programs, sea lamprey control, habitat conservation and quota management. In addition, water quality legislation was introduced to reduce inputs of phosphorus and persistent toxic chemicals that accumulate in fish flesh. The result of these actions has been a dramatic improvement in water quality and indications of a recovering fishery.

Although there is good evidence that some stocks are recovering, all is not well with the fishery. Important commercial species such as the lake trout have not recovered in any of the Great Lakes. Fish community composition and abundance have declined near urban areas due to extensive habitat loss, eutrophication, and the acute and sub-lethal effects of chemical contamination. Some bottom-dwelling species such as the brown bullhead and white sucker develop liver tumours. The specific causes are unknown but there is strong circumstantial evidence that tumour frequency increases at sites contaminated with known carcinogens. Export markets for Lake Ontario eels remain closed due to high concentrations of PCBs and mirex and consumption advisories exist for at least one species on all of the Great Lakes. Not surprisingly, the long-lived fish such as lake trout accumulate the highest body burdens of contaminants. Consumption advisories for lake trout and chinook salmon are in effect at every location on Lake Ontario where these species were tested.

Toxic chemicals affect fish at the molecular, cellular, individual, population, and community levels. Contaminant effects at the community and population levels are difficult to separate from the combined effects of overfishing, habitat destruction, eutrophication and the introduction of exotic species. Community effects are rarely associated with a specific contaminant and are characterized by a reduction in the diversity of species. Sensitive species are eliminated and contaminant tolerant species increase. Most of the

contaminant effects at the community level have been reported for plankton and benthos. There are some cases, such as the recruitment failure of Lake Michigan lake trout, where chemicals are implicated in population failure. However, it has not been possible to separate responsible chemicals from the complex suite of chemical and biological stressors in the Great Lakes.

The evidence for contaminant effects on fish becomes more convincing at the individual and cellular levels. The relationship between contaminants (polynuclear aromatic hydrocarbons) and tumour frequency is reinforced by observations of increased liver enzyme activity needed to break down contaminants and the presence of carcinogenic metabolites in the bile of exposed fish. The chemical link is further strengthened by laboratory studies showing that fish exposed to chemicals extracted from contaminated sediment develop liver tumours. Additional laboratory evidence shows that fish exposed to these chemicals in the field and in the laboratory develop DNA adducts, a step in the process of carcinogenesis. Other pathological indicators of stress that have been monitored in the Great Lakes include fin ray asymmetry, vertebral anomalies, thyroid hyperplasia and benign epithelial papillomas.

Several tests have been used successfully to demonstrate the effects of chemical exposure at the cellular level. These include the induction of mixed function oxidase (MFO) enzymes, a liver enzyme system responsible for breaking down contaminants, and the inhibition of amino levulinic acid dehydratase (ALA-D), an enzyme system that measures exposure to lead.

There are a number of pathological, physiological, and biochemical diagnostic procedures clearly indicating that Great Lakes fish are accumulating and metabolizing environmental contaminants. The combined effects of many chemicals under different physical and chemical conditions in the Great Lakes are not always apparent. We suspect contaminants are involved in reproductive failure, tumour development and other pathological anomalies, and a large number of physiological and biochemical changes whose biological significance is not yet fully understood. It is unlikely, given the large number of variables, that science will be able to unravel the ecological impacts of the combinations of chemicals in the Great Lakes. Decisions to control chemicals in the Great Lakes must be based on strong circumstantial evidence in the field and confirming laboratory toxicology.

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1. A BRIEF HISTORY OF THE GREAT LAKES FISHERY

Historically, the Great Lakes were renowned for their productive fisheries. The cold water fisheries of Lakes Superior, Michigan, Huron, and Ontario were dominated by highly valued species such as lake trout, lake whitefish, lake herring, and deepwater ciscoes. Commercial records indicate that between 1900 and 1933, coldwater species made up 77% of the total annual harvest of 32,000 tonnes (Christie, 1974). Lake Erie, the shallowest and most productive of the Great Lakes, supported important commercial fisheries for blue pike, herring, perch, and walleye. During the 1930s, Lake Erie contributed 46% of the total Great Lakes commercial harvest.

The Great Lakes fishery has changed substantially since 1900. Commercial lake trout harvests in all of the Great Lakes decreased by an average 94% between 1938 and 1960 (Table 1). In Lake Ontario, 83% of the commercial fishery for lake herring and ciscoes disappeared between 1941 and 1952, and commercial catches of whitefish decreased by 92% from 1961 to 1967 (Christie, 1974). In Lake Erie, catches of lake herring collapsed from a peak of 18,000 tonnes in 1917 to only 900 tonnes in 1935, a reduction of 95% (Christie, 1974). Dramatic changes occurred in the fisheries of all the Great Lakes as lake trout, whitefish, herring, ciscoes, blue pike and walleye were replaced by smaller, less desirable species.

Decreasing lake trout and herring stocks had less impact on the total fish harvests from Lakes Erie and Michigan. In fact, total commercial harvest increased by 15 and 56% respectively, during this period as fishermen turned their attention to less valuable species. Similarly, the composition of the commercial catch changed in all of the Great Lakes as highly valued stocks decreased. In Lake Ontario, the commercial fishery shifted from herring, whitefish and lake trout, to yellow perch, American eel, smelt, carp, bullheads and suckers. Yellow perch, smelt, white bass and walleye replaced herring and blue pike in Lake Erie. The Lake Michigan fishery, which was traditionally dominated by lake trout, whitefish and lake herring, shifted to alewife, whitefish, smelt, chub, and yellow perch. In all of the Great Lakes, the lower valued alewife and smelt replaced lake herring as the most abundant pelagic species.

In an effort to reverse this downward trend, fisheries agencies implemented salmonid stocking programs, lamprey control, fish habitat conservation measures and quota management. In addition, water quality legislation was introduced to reduce inputs of phosphorus, toxic chemicals, and persistent, bioaccumulable contaminants (IJC, 1987a). The result of these management strategies has been an improvement in water quality (IJC, 1987a) and a recovering fishery. For example, walleye stocks have increased in western Lake Erie (Hatch *et al.*, 1987) and in eastern Lake Ontario (Hurley, 1986) and there is evidence that whitefish stocks are recovering in the eastern basin (Christie *et al.*, 1987). Successful stocking programs for lake trout, brown trout, rainbow trout, and Pacific salmon have resulted in large salmonid populations in Lake Ontario. Bloater stocks have improved substantially in Lake Michigan (Crowder *et al.*, 1987) and the commercial fishery for lake whitefish in Lakes Huron and Michigan has been restored to its 1930s status.

TABLE 1. DECREASE OF THE COMMERCIAL LAKE TROUT HARVEST IN THE GREAT LAKES

Lake	Year	Catch (tonnes)	Year	Catch (tonnes)	Decrease (%)
Ontario[A]	1940	90	1950	5	98
Huron[B]	1938	2268	1954	76	96
Michigan[B]	1944	2948	1953	181	94
Superior[B]	1950	2041	1960	227	89

Sources: A = Christie, 1974
B = Smith and Tibbles, 1980

Although commercial and recreational fisheries are improving throughout the Great Lakes, there is still cause for concern. Lake trout and herring stocks in Lakes Ontario, Huron, and Superior have not recovered. Total annual harvests for the three lakes during the 1982-86 period were 32, 59 and 62% lower than the period from 1932 to 1936 (Table 2). The decreasing fishery reflects the combined effects of all stresses on the Great Lakes ecosystem, including fishery closures caused by high contaminant levels. Lake trout populations are maintained by intensive stocking programs and there is evidence of poor recruitment in all of the Great Lakes (Mac, 1988). Fish community composition and abundance has decreased in urban areas because of eutrophication, habitat loss, and toxic chemicals (Whillans, 1979, Hurley, 1986). Tumours have been reported in brown bullheads and white suckers living in heavily contaminated environments (Black, 1988) and there is some evidence that contaminants are inducing detoxification enzymes in fish from polluted sites (Luxon *et al.*, 1987).

TABLE 2. CHANGES IN THE MEAN ANNUAL COMMERCIAL CATCH (TONNES) IN THE GREAT LAKES BETWEEN 1932-1936, AND 1982-1986, AND THE CHANGE IN THE PROPORTION OF THE TOTAL CATCH CONTRIBUTED BY PREFERRED SPECIES (LAKE TROUT, WHITEFISH AND LAKE HERRING)

	Total Catch (tonnes)		Percent Change	
Lake	**1932-1936**	**1982-1986**	**Total Catch**	**Preferred Species**
Ontario	1454	988	-32	-54
Erie	19897	22903	+15	-5
Huron	9883	4071	-59	-8
Michigan	11206	17452	+56	-33
Superior	8232	3080	-62	-15

Sources: Baldwin *et al.*, 1979 and the Great Lakes Fisheries Commission Annual Reports

Although levels of PCBs, DDT, mercury, and mirex in fish have decreased significantly since the early 1970s (Baumann and Whittle, 1988; D'Itri, 1988), high contaminant levels still restrict the commercial market for some species, such as the American eel from Lake Ontario. While recreational opportunities for trout and Pacific salmon have improved, consumption advisories for lake trout are in effect at all of the sites tested in Lake Ontario and at 60% of the sites tested in Lake Superior.

2. NON-CHEMICAL FACTORS AFFECTING THE FISHERY

Numerous investigators have reviewed and assessed the effects of historic changes in fish stocks to identify and understand the reasons for their decrease (Hartman, 1988; Egerton, 1985; Regier and Hartman, 1973; Christie, 1974). The most obvious factors include overfishing (Christie, 1974), nearshore habitat destruction (Whillans, 1979; 1982), eutrophication (Hurley, 1986; Evans and Loftus, 1987), environmental contaminants (Mac *et al.*, 1985; Black, 1988) and the introduction of exotic species, particularly sea lamprey (Smith and Tibbles, 1980), American smelt (Christie, 1974), alewife (Crowder *et al.*, 1987) and white perch (Hurley, 1986).

2.1 THE IMPACT OF EXOTIC SPECIES ON THE GREAT LAKES FISHERY

Exotic species have gained access to the Great Lakes in ship ballast water and through bait bucket introductions. Other species present in the St. Lawrence River or Lake Ontario gained access to the remaining lakes through the Erie Barge and Welland Canals.

The competitive and predatory activities of invading species, most notably sea lamprey, alewife and smelt have had a major influence on the composition of the Great Lakes fish community. The first noticeable event in the collapse of the commercial fisheries was the decrease of native lake trout stocks. Although lamprey were reported to be in Lake Ontario as early as 1830, their presence in the upper lakes was assured with the opening of the Welland Canal. By 1936, lamprey had reached Lake Michigan, and by the 1950s, lake trout stocks had collapsed in Lakes Ontario, Huron, Michigan and Superior.

Lake trout are the preferred prey of the sea lamprey. However, as lake trout stocks decreased, the lamprey preyed on other species. Lamprey movement from Lake Ontario to Lake Superior was followed by dramatic reductions in stocks of lake trout, burbot, whitefish and lake herring. Lamprey fed on all but the smallest species. Widespread lamprey predation, overfishing and habitat alteration caused the decrease of lake whitefish and burbot stocks in Lakes Superior, Michigan, Huron and Ontario, and a concurrent recovery of the smaller ciscoes, the preferred prey of lake trout and burbot (Christie, 1974).

With the formation of the Great Lakes Fishery Commission in 1955, a bilateral sea lamprey control program was initiated. By 1980, U.S. and Canadian governments had spent approximately $54 million to control sea lamprey (Fetterolf, 1980). Control measures included electric weirs, low head barrier dams, and 3-trifluoromethyl-4-nitrophenol, a toxicant used to poison the stream dwelling larval stage of the lamprey. Lamprey control was augmented by an intensive stocking program to rehabilitate lake trout populations and to establish desirable recreational species, such as the rainbow trout, brown trout, splake, and Pacific salmon. The results of these control measures were a significant decrease in sea lamprey spawning runs, decreases in the incidence of lamprey wounds on prey species and positive responses of major fish stocks (Smith and Tibbles, 1980).

The number of fish added to the lakes has increased annually since the mid 1960s. In 1984, fisheries agencies introduced approximately 16 million salmonids into Lake Michigan (Eck and Wells, 1987) and 20 million into Lake Ontario (O'Gorman *et al.*, 1987). Intensive stocking has created a highly successful sport fishery, particularly in Lakes Ontario, Huron, and Michigan. The gross economic value of the Great Lakes sport fishery is estimated at $4 billion, considerably higher than the commercial fishery which has been valued at approximately $133 million.

Although stocking has successfully restored large numbers of adult lake trout in the Great Lakes, there is evidence that the introduced fish are not reproducing successfully (Mac, 1988). The cause is unknown. Fisheries agencies have not been able to isolate the factors responsible for poor lake trout recruitment. Many hypotheses have been proposed including inappropriate genetic stock, predation, competition, limited numbers of mature adults, contaminants, eutrophication and poor spawning habitat.

The introduction of large salmonids has substantially changed the fish community and predator-prey relationships in Lakes Michigan and Ontario. Pacific salmon, rainbow trout, brown trout and lake trout are sharing the top predator role, formerly held by lake trout and burbot. The large numbers of predators have contributed to the decrease in alewife stocks in Lake Michigan (Crowder *et al.*, 1987; Eck and Wells, 1987) and may do the same in Lake Ontario (Christie *et al.*, 1987; O'Gorman *et al.*, 1987).

Introductions of exotic species to the Great Lakes are not limited to lamprey and Pacific salmon. Twenty-three species of fish have been deliberately introduced since 1870 (Emery, 1985) and eight of the 23 have successfully colonized the Great Lakes. Five of these are salmonids. Of the remaining three, only the carp is considered to be an undesirable species because of its destructive effects on nearshore habitat. At least 14 fish species have been accidentally introduced to the Great Lakes since 1819. Eight have successfully colonized the Great Lakes. Three of the eight species, alewife, rainbow smelt and white perch, are recognized as major influences on the fish community of the Great Lakes.

Accidental introductions of invertebrates also have significant effects on the Great Lakes fishery. The zebra mussel, (*Dressenia polymorpha*), was first reported in Lake St. Clair in 1986. By 1989, the mussel had successfully colonized all of the nearshore zone of western Lake Erie and was moving at an estimated 240 km/year. *Dressenia* has already been reported in Lakes Ontario, Michigan, Huron and Superior and may eventually colonize most of the rocky shoreline in the lower lakes and connected waterways. The long term implications for the fishery are unknown. Possible effects include reduced spawning habitat for walleye and lake trout, reduced phytoplankton and zooplankton populations in the littoral zone, and altered energy flow through the food web (Mackie *et al.* 1989).

Rainbow smelt were introduced to the Great Lakes system in Crystal Lake, Michigan, in 1912. Populations were established in all of the Great Lakes by 1964. The origin of the alewife in the Great Lakes is uncertain but the species was reported as early as 1890 in Lake Ontario and was present in nuisance numbers in Lakes Erie, Huron, and Michigan by 1957. The proliferation of smelt and alewife throughout the Great Lakes coincided with the collapse of top predator stocks, such as lake trout, burbot, and walleye.

Increasing stocks of smelt and alewife had significant adverse effects on the cool and coldwater fish community of the Great Lakes. Christie (1974) attributed the decrease of herring stocks in Lakes Ontario, Huron, Michigan and Superior to smelt invasions, believing that smelt competed with herring for benthic food organisms and preyed on young herring. Similar observations were made in the inland lakes by Evans and Loftus (1987) who reported that smelt affected other species directly (predation) and indirectly by redirecting energy flows in the aquatic system. As a result, smelt influenced recruitment, growth, and food web restructuring and were implicated in the recruitment failure of Atlantic salmon, lake trout, lake herring, walleye, burbot, emerald shiner, alewife, and bloater (Evans and Loftus, 1987). Similarly, alewife proliferation was associated with decreases in stocks of lake herring, deep-water ciscoes, smelt and yellow perch. Eck and Wells (1987) hypothesized that alewife affected native stocks in Lake Michigan by preying on the early life stages, causing the decrease of bloater, lake herring, yellow perch, emerald shiner and deepwater sculpin stocks. By 1970, smelt and alewife were firmly established in the Great Lakes and had become the principal prey of lake trout and the introduced salmonids.

2.2 THE IMPACT OF OVERFISHING ON THE GREAT LAKES FISHERY

A discussion of fishing pressure in a report on contaminants may appear to be irrelevant. However, over-exploitation from commercial and recreational fishing is an important stressor affecting Great Lakes fish communities. The effects of overfishing and environmental stress on fish communities are very similar and have been the subject of long standing debate on Lake Erie (Egerton, 1985).

Not surprisingly, stocks respond to overfishing and deteriorating environments in similar ways and efforts to separate them are complicated by the similarity of symptoms. Overfished stocks are dominated by young fish, few if any strong year classes, increased growth, early maturity, and an increase in the proportion of mature females in the population (Hatch *et al.*, 1987). At the same time, compensating mechanisms occur within the ecosystem as predator and prey relationships adjust to a new equilibrium. Observers looking for contaminant effects at the population and community levels would recognize a disturbed ecosystem but may not be able to discern the cause.

Christie (1974) suggests that successful stocks are maintained by occasional strong year classes that seem to coincide with optimum climatic conditions. Of course, optimum conditions do not occur every year and stock survival depends upon a 'reserve' of older mature fish in the population that can take advantage of exceptional conditions. Populations that are heavily fished (or preyed upon by lamprey or adversely affected by contaminants) lose the older fish and must depend upon young females to maintain the stock. Young fish may have only one or two spawning years before they enter the fishery and their success depends upon good climatic conditions. The affected populations have lost their capacity to cope with several years of bad weather and poor recruitment. Overfishing rarely affects an entire stock. However, populations that have been severely depleted by overfishing may lose their ecological advantage and be replaced by other stocks (Christie, 1974). An excellent example of lost 'ecological advantage' occurred when white perch stocks collapsed in the Bay of Quinte following a massive winter die off in 1978. This reduced pressure on walleye stocks and resulted in a strong year class that is still sustaining walleye populations in the Bay ten years later (Hurley, 1986).

Although fisheries biologists have difficulty identifying the causes responsible for stock collapses, there is little doubt that overfishing has been a major factor. Christie (1974) lists several stocks including the black fin ciscoe (Lake Michigan), lake sturgeon (all lakes), walleye and blue pike (Lake Erie) that collapsed as a result of overfishing. Loftus *et al.* (1987) suggested that over-exploitation was a major stress impacting fish communities, long before sea lamprey populations caused the collapse of top predators.

Hatch *et al.* (1987) reviewed the recent history of walleye stocks in Lake Erie and provided a case history of interactions between contaminants and overfishing. Prior to 1936, the walleye fishery yielded between 334 and 1356 tonnes per year. Only 4 years yielded less than 600 tonnes. Walleye stocks increased slowly from 1936 to 1940 and then increased dramatically from 1946 to 1955. The Lake Erie walleye harvest peaked in 1956 at 6975 tonnes. Fishing pressure continued and an additional 6000 tonnes was harvested in 1957. The total harvest of almost 13000 tonnes over two years was too much for the fishery. The stock collapsed in 1958 and with minor fluctuations, remained at low levels until 1970 (only 161 tonnes were caught in 1969). Kutkuhn (cited in Hatch *et al.*, 1987) estimated that fishing mortality during the 1960s exceeded 90% of the fishable stock.

By 1970, the environment in Lake Erie was severely degraded. Uncontrolled nutrient addition from sewage treatment plants and agricultural runoff resulted in increased eutrophication accompanied by increased phytoplankton production and deoxygenation of the hypolimnion. Reduced oxygen in the deeper basin of the lake was responsible for the loss of important benthic populations of *Hexagenia* species in western Lake Erie and lost summer habitat for top predators.

The International Joint Commission initiated a program to manage phosphorus inputs and control eutrophication through Pollution from Land Use Reference Group (PLUARG). At the same time, DDT, PCBs and mercury were being recognised as important contaminants and there was concern that poor water quality was responsible for the declining fishery. In 1970, mercury concentrations in walleye flesh exceeded the 0.5 mg/kg guideline. The commercial fishery was closed and anglers were warned not to eat their catch. Fisheries agencies used the closed fishery as an opportunity to establish a quota management program that was implemented when the fishery reopened in 1973.

The effect was an immediate improvement in year class strength that continued from 1970 to the mid 1980s. In 1984, the estimated fishable stock exceeded 32 million walleye (Hatch *et al.*, 1987). While water quality improvements in Lake Erie have ultimately improved the Lake Erie fishery, the increase in walleye stocks immediately following the ban on commercial fishing suggests that overfishing was the major factor responsible for the stock collapse in the late 1950s.

2.3 THE IMPACT OF HABITAT DESTRUCTION ON THE GREAT LAKES FISHERY

The destruction of fish habitat (including water quality) was perhaps the most significant and easily observed stress affecting the nearshore fish community in Lake Ontario. Most of the effects resulted from the discharge of untreated effluents into the lake and from the filling and restructuring of the littoral zone to accommodate industrial and urban development. Not surprisingly, early settlers in the Great Lakes basin located on sheltered embayments and large river mouths such as Hamilton Harbour, the Bay of Quinte, Severn Sound, Toronto Harbour, Saginaw Bay and Lake St. Clair. In many cases, the presence of a thriving fishery was at least partly responsible for the first settlements. All of these shallow, mesotrophic environments supported diverse, productive fisheries that were economically and socially important to the Great Lakes community.

Development flourished in the early and mid 1900s at the expense of fish habitat, producing dramatic and predictable decreases in historically important fish communities. Habitat degradation resulted from several activities, including:

a) Deforestation of the drainage basin with subsequent increases in siltation and water temperature;
b) The construction of dams, which blocked access to spawning areas and increased silt deposition over valuable riffle areas;
c) The infilling and restructuring of shorelines to create agricultural and urban land and to prevent erosion;
d) The draining and filling of coastal marshes, which eliminated spawning and nursery areas for many nearshore and open lake species;
e) The discharge of solid and liquid wastes such as wood fibre, untreated sewage, and many organic and inorganic chemicals into lakes and rivers and;
f) Eutrophication due to phosphorus inputs, which results in increased algal biomass (including *Cladophora*), decreased light penetration in the littoral zone, and reduced dissolved oxygen concentrations.

The effects of lost physical habitat combined with increasing eutrophication have been described for Hamilton and Toronto Harbours (Whillans, 1979) and for the Bay of Quinte (Hurley, 1986). Although industrial development was greater in Hamilton and Toronto than in Quinte, the changes in the fish community were similar, progressing from high valued species dominated by top predators, such as pike, bass, and walleye, to undesirable species dominated by white perch, alewife, bullheads, and carp (Whillans, 1979; Hurley, 1986). The trend towards decreased piscivores and increased numbers of planktivores and benthivores characterizes all nearshore fisheries impacted by eutrophication and physical habitat loss.

The history of coastal marshes (wetlands) in the Great Lakes illustrates the extent and impact of habitat destruction on fish communities. Wetlands play an important role in the energy dynamics of the Great Lakes by converting solar energy into biomass. As a result, marsh communities are considered to be among the most productive on earth and comparable to tropical rain forests (cited by Herdendorf, 1987).

Stephenson (1988) analyzed the fish community in five Lake Ontario wetlands. She found more than 40% of the fish species present in Lake Ontario relied on the marshes during at least one critical life stage and that half of these were important to the commercial or sport fishery. In addition, pelagic species that did not use the wetlands directly relied on them indirectly for food. Herdendorf (1987) reported that 90% of the standing fish crop of Lake Erie marshes is made up of forage species that are the main food source for open-water piscivores.

Coastal marshes in the Great Lakes have disappeared at an alarming rate. They have been drained for agricultural purposes, filled to create development property, diked to control water level fluctuations and dredged to allow boat access. Other factors responsible for the destruction of wetlands include high water levels, filling due to siltation and eutrophication.

Approximately 50% of the wetlands on the Canadian side of Lake St. Clair have disappeared (McCullough, 1985) along with 71% of the wetlands on the U.S. side of the lake (Jaworski and Raphael, 1978). Similar losses have occurred in other lakes. Whillans (1982) calculated that 43% of the historical wetlands on the Canadian shore of Lake Ontario have disappeared. Marsh destruction has been particularly severe in urban centres such as the Dundas Marsh in Hamilton Harbour where more than 80% of the wetland disappeared in the last 40 years (Hamilton Harbour Remedial Action Plan, 1990).

The loss of river habitats, marshes, reefs, beaches and natural shorelines is perhaps the most important stressor affecting the fisheries in the lower lakes. The most productive habitats in sheltered embayments and estuaries are often the most affected. The effects extend beyond the nearshore and influence the entire lake community. Not surprisingly, the effects of habitat loss on fish communities are difficult to distinguish from the adverse effects of toxic chemicals.

3. TOXIC CHEMICALS AND THEIR EFFECTS ON FISH AND THE FISHERY

The effects of toxic chemicals on the Great Lakes ecosystem have been reviewed by Fitchko (1986), Evans (1988) and Schmidtke (1988). Their papers summarize the current state of knowledge of chemical effects on Great Lakes biota. They emphasize the diverse taxa that are affected by contaminants and the variety of effects, ranging from physiological dysfunction in phytoplankton to pathological anomalies in fish and benthos. These authors stress the difficulty of extrapolating physiological, biochemical and pathological effects observed at the individual level to effects at the community level.

The presence of chemical residues in plankton, benthos, forage fish and top predators indicates that all trophic levels are exposed to a suite of organic and inorganic chemicals. Field observations of biochemical and pathological responses, such as induced mixed function oxidase enzymes (MFO) and tumours in fish indicate that animals are not simply acting as fat storage depots for toxic chemicals. They are metabolizing them, and in the process, exposing their complex systems of molecular and cellular interactions to seemingly endless combinations of chemicals. Although dose-response relationships have been developed for many individual chemicals, interactive effects between mixture of chemicals occur. There is no doubt that the toxic effects that have been observed under controlled laboratory conditions are also occurring in the Great Lakes.

It is not difficult to understand that contaminants limit human uses of Great Lakes fish. The effects are clearly demonstrated by consumption advisories, lost market opportunities and changes in the lifestyles of native Canadians.

3.1 THE EFFECTS OF CONTAMINANTS ON THE GREAT LAKES SPORT FISHERY

Fisheries agencies have made major commitments to rehabilitate and enhance Great Lakes sport fisheries. Lamprey control, fish stocking, habitat restoration of important spawning rivers and reductions in nutrient inputs to the Great Lakes, have resulted in a flourishing recreational fishery. Millions of salmonids (lake trout, splake, rainbow trout, brown trout, coho salmon, chinook salmon and Atlantic salmon) are stocked annually to restore declining populations and increase opportunities for sport fishing. As a result, the Great Lakes support the largest recreational fishery in North America. Sport fishing provided fishing opportunities for more than 1 million anglers and 11,000 worker-years in Canada in 1985 (Talhelm, 1987). The gross economic value of the Great Lakes fishery has been estimated to be approximately $4 billion annually (Boulanger and Charbonneau, 1989).

Despite these apparent successes, sport fish consumption advisories are issued by the state and provincial jurisdictions in the Great Lakes basin. Some Great Lakes species accumulate high body burdens of persistent, bioaccumulable contaminants such as PCBs, DDT, mirex, chlordane, toxaphene, furans, dioxins and mercury. Chemical concentrations in fish are affected by the physical properties of the chemical, such as resistance to degradation, lipophilicity, and molecular structure. Biological factors influencing chemical concentrations in fish include the route of chemical uptake (food, water), the duration of exposure, the concentrations of chemicals in water and food, and the depuration rate of chemicals as a result of excretion and metabolic conversion (Niimi, 1990).

Organochlorines such as PCBs, DDT and mirex are stored in the fat cells of fish and accumulate in species that have a high proportion of body fat, such as lake trout, rainbow trout and salmon. Long lived top predators such as the lake trout accumulate high body burdens of organochlorines because of the increased exposure time (6 year old fish are common in Lake Ontario), increased chemical dose because of long food-chains (by eating alewife and smelt that are accumulating chemicals from benthos, plankton and fish), and increased capacity for chemical storage (high body fat). For these reasons, long-lived predators (trout, salmon and walleye) are the most contaminated species in the Great Lakes (see Volume I, Part 2).

Provincial and state agencies maintain sport fish contaminant monitoring programs to assist consumers in selecting fish that have the lowest contaminant levels. In Canada, the Ontario Ministry of the Environment (OMOE) and the Ontario Ministry of Natural Resources (OMNR) jointly publish an annual "Guide to Eating Ontario Sport Fish". Consumption advisories are based on the concentration of contaminants in fish and the frequency of eating fish meals. In effect, these are estimates of the chemical dose to humans consuming Great Lakes fish. Results of the U.S. and

Canadian sport fish consumption advisory programs for 1987 have been summarized by Rathke and McRae (1989). In 1987, consumption advisories were in effect for at least one species in 36 of the 42 Areas of Concern identified by the IJC. Data from the 1989 Guide indicate that fish from Lake Ontario are the most contaminated (Table 3). Consumption advisories affect 22 species of fish in Lake Ontario. PCB and mirex concentrations in 11 fish species from Lake Ontario exceeded the federal consumption guidelines (Rathke and McRae 1989). Consumption advisories for chinook salmon and lake trout occur at every site in Lake Ontario where these species were tested. Advisories occur the least often in Lake Erie, possibly reflecting the low numbers of long lived lake trout and the interaction between eutrophication and contaminants. Surprisingly, consumption advisories are in effect for walleye and siscowet from Lake Superior at all the sites where these species were tested. Most of these advisories are a result of high mercury concentrations which are due to natural mercury contamination from the watersheds. (For a more detailed discussion of sport fish consumption issues and Great Lakes Areas of Concern, see Volume I, Part 2.)

PCBs, mirex and mercury are responsible for most of the fish consumption advisories. In Ontario, 66% of the consumption advisories were a result of mercury concentrations exceeding 0.5 mg/kg, 35% were due to PCBs exceeding the 2.0 mg/kg guideline and 53% of restrictions in Lake Ontario and the lower Niagara and St. Lawrence Rivers were due to mirex concentrations above 0.1 mg/kg (Rathke and McRae, 1989). Consumption advisories remain in effect for a high proportion of the lake trout in Lakes Ontario, Huron and Superior, despite a significant decrease in contaminant levels in top predators. The trend towards lower contaminant concentrations is likely to continue and result in fewer consumption advisories.

It is difficult to determine the social and economic impact of contaminants on the Great Lakes fishery. Pacific salmon and trout contribute 13% of the total catch in Lake Ontario. However, more than 60% of the anglers discard their fish, presumably in response to consumption advisories. The total value of the fishery if it were uncontaminated is unknown. There may be many people who are reluctant to catch fish they cannot eat. There is little doubt that an uncontaminated fishery would increase the social and economic value of the Great Lakes.

3.2 THE EFFECTS OF CONTAMINANTS ON THE COMMERCIAL FISHERY

The Great Lakes support the largest freshwater fishery in the world. While the economic value of commercial fishing is much smaller than the estimated $4 billion value of the sport fishery, it is economically and socially important to residents of the Great Lakes basin. In 1985, the landed value of the commercial fishery was estimated to be $54 million and the final sales value of food fish from the Great Lakes was approximately $133 million. The total economic benefit of the food fishery on the regional economy was about $270 million. The industry also provided approximately 9,000 worker-years in 1985.

The commercial fishing industry has been adversely affected by contaminant levels in fish which have reduced marketing opportunities. In 1970, mercury concentrations exceeded federal guidelines in commercial species from parts of Lake Huron, Lake St. Clair, western Lake Erie, eastern Lake Ontario (east of 76° 50'), the Ottawa River, Lake St. Francis and the St. Lawrence River. During the same period, the federal government imposed a ban on fish with levels of DDT and PCBs greater than 2 mg/kg. This closed commercial salmon fishing in Lakes Huron, Erie, and Ontario. In Lake Michigan, the U.S. government seized 80% of the total commercial catch of coho, chub and whitefish. This resulted in a loss of $2.8 million (cited in Fitchko, 1986). Subsequently, mirex was discovered in Lake Ontario fish. It was the fourth major contaminant to be detected in less than 10 years. In

TABLE 3. THE PROPORTION OF SITES WITH CONSUMPTION ADVISORIES FOR TOP PREDATORS IN THE GREAT LAKES, 1989. CALCULATED AS A PERCENT OF THE NUMBER OF SITES ( ) WHERE THE SPECIES WAS TESTED

Species	Lake Ontario	Lake Erie	Lake Huron	Lake Superior
Lake trout	100(8)	0(1)	43(7)	60(35)
Siscowet	-	-	-	100(7)
Rainbow	55(11)	0(3)	25(12)	0(4)
Coho	80(5)	0(6)	33(3)	-
Chinook	100(5)	-	0(5)	0(3)
Walleye	75(16)	31(13)	87(16)	100(7)

Source: Ontario Ministry of the Environment. 1989 Guide to Eating Ontario Sport Fish

1982, the German government restricted the importation of American eels by stating that it would accept fish with a maximum concentration of 0.1 mg/kg of mirex. This was because of concerns about the potential health effects associated with the ingestion of contaminated fish. In addition, new criteria were established for eight other organic chemicals at concentrations considerably less than the Canadian federal guidelines.

As analytical techniques improved, other highly toxic chemicals, such as polynuclear aromatic hydrocarbons (PAHs), toxaphene, chlorinated benzenes, chlorinated phenols, furans and dioxins, were discovered in Great Lakes fish. This threatened the international reputation of Canadian fish products. In 1983, Japan refused to purchase smelt from Lake Erie because of a mistaken belief that smelt were contaminated with dioxins. Chemical analyses by the Department of Fisheries resolved the issue and the market was reopened in 1985. However, this incident demonstrates the potential impact on the fishery if importing countries lose confidence in the quality of Great Lakes fish. Canada exports almost 95% of the commercial catch. Before leaving Ontario, commercial fish are processed in a certified facility, and federally inspected under the fish inspection program of the Department of Fisheries and Oceans. There would be severe economic and social impacts for the industry if international markets were lost.

4. HOW TOXIC CHEMICALS AFFECT FISH

Toxic chemicals enhance, retard, or in some way alter the normal biochemical and physiological functions of animals and plants. Dixon *et al.* (1985) described six levels of organization that are affected by chemicals. They reported that toxic effects occur at the *molecular* level (e.g., enzymes, vitamins, electrolytes), the *cellular* level (e.g., alterations in cell structure and function), the *physiological* level (e.g., organ functions such as respiration, excretion, reproduction digestion, etc.), the *individual* level (e.g., death, behavioral changes, swimming ability), the *population* level (e.g., production of young, abundance, distribution, etc.) and at the *community* level (e.g., numbers of species and the abundance of each). These six levels do not exist independently. Effects at one level interact with effects at other levels (Figure 1). For example, contaminants that alter the production of sex hormones (molecular level) in lake trout will affect the structure of the reproductive organs and the quality, quantity, and timing of eggs and sperm (cellular level). Severe disruption may cause reproductive failure (individual level). If these effects occur throughout a strong year class, poor recruitment will follow (population level) and ultimately, as the population of trout decreases, other species, such as smelt, alewife and sculpins, will increase in the absence of predation. These changes will subsequently affect benthic, zooplankton, and phytoplankton populations, in effect, changing the lake's ecological structure. This hypothetical example may not be too far from the truth. Mac (1988) presented strong circumstantial evidence that contaminants reduced embryo survival and contributed to reproductive failure in lake trout from Lake Michigan.

The effects of some chemicals on fish are specific and in these cases cause and effect relationships can be established. For example, exposure to inorganic lead inhibits the blood enzyme, alpha-amino levulinic acid dehydratase (ALA-D), in fish. This contaminant specific indicator was used to identify the source and extent of lead contamination in Lake Ontario fish (Hodson *et al.*, 1984b). Other molecular indicators of exposure include the inhibition of blood and brain enzymes, such as acetylcholinesterase (in response to exposure to organophosphate and carbamate insecticides) and increasing levels of liver proteins (metallothioneins) in fish following exposure to cadmium, copper, mercury and zinc (Klaverkamp *et al.*, 1984). Specific responses to chemicals also occur at the cellular level. For example, fish exposed during critical stages to benzo(a)pyrene (B[a]P), a polynuclear aromatic hydrocarbon, will develop morphological anomalies. Similarly, exposure to B[a]P will cause liver tumours in sensitive fish species (individual level).

The specificity of chemical effects decreases as one moves from the molecular level towards the community level. As a result, contaminant effects become more difficult to separate from the effects of non-chemical stressors. As a general rule, it is almost impossible to distinguish specific contaminant effects on fish populations and communities without additional studies of chemical contamination and specific molecular indicators. For this reason, Dixon *et al.* (1985) and Niimi (1990) argued for a commonly-accepted suite of contaminant effects indicators to measure the effects of toxic chemicals at several levels of organization. In the absence of clearly defined chemical effects at the population level, both authors emphasized the need to understand the mechanisms, rather than the effects of individual chemicals. They further emphasized the need for cooperative studies between toxicologists and population biologists.

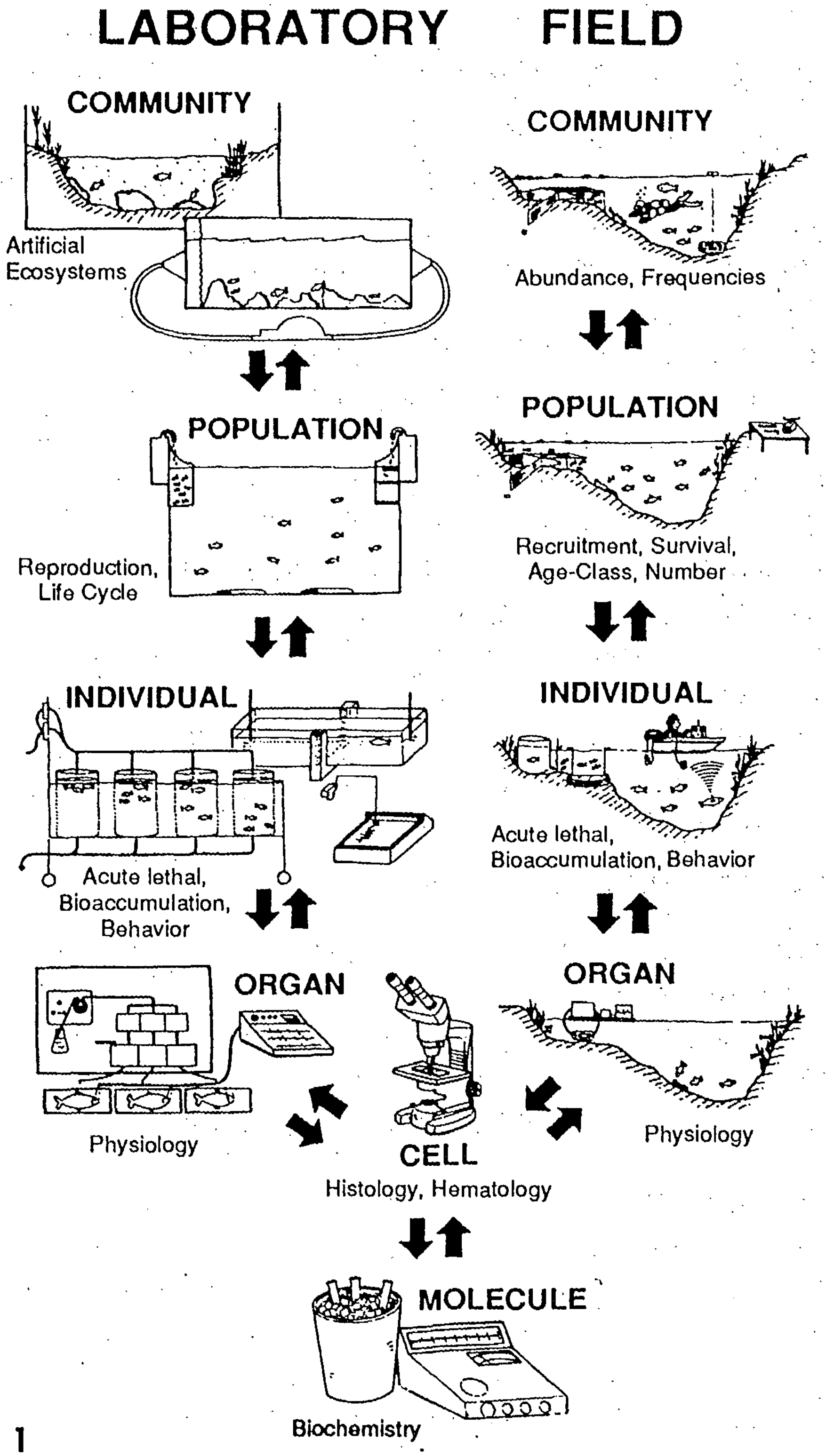

LABORATORY
FIELD
COMMUNITY
Artificial Ecosystems
COMMUNITY
Abundance, Frequencies
POPULATION
Reproduction, Life Cycle
POPULATION
Recruitment, Survival, Age-Class, Number
INDIVIDUAL
Acute lethal, Bioaccumulation, Behavior
INDIVIDUAL
Acute lethal, Bioaccumulation, Behavior
ORGAN
Physiology
ORGAN
Physiology
CELL
Histology, Hematology
MOLECULE
Biochemistry

Figure 1

A framework for linking contaminant effects at several levels of organization in field and laboratory studies. Reproduced from Dixon *et al.* 1985. Figure 1 was kindly provided by Dr. J.F. Klaverkamp, Department of Fisheries and Oceans.

5. EVIDENCE OF CONTAMINANTS EFFECTS ON GREAT LAKES FISH

It is difficult to document the effects of toxic chemicals on wild populations. Laboratory experiments demonstrate that contaminants affect molecular and cellular functions and may eventually cause mortality, reproductive failure, reduced growth, poor condition, and altered behaviour (Niimi, 1990; Dixon *et al.*, 1985). Similar population and community responses are observed when fish are exposed to deteriorating habitat, competition, poor weather and over exploitation (Colby, 1984). The effects of chemicals on fish communities are often masked by non-chemical stressors and affected animals are rarely observed in the wild because they quickly succumb to predation or other stresses. As a result, almost all of the data on contaminant effects on wild populations and communities are based on strong circumstantial evidence (Black, 1988; Mac, 1988; Mix, 1985). Ryder (1988) concluded that "The current state of our knowledge of cause-effect relationships between contaminants loadings and multi-species fish yields is abysmally deficient in most areas. Particularly distressing is our lack of understanding of how contaminants mixtures affect interrelationships within aquatic communities...."

5.1 EFFECTS AT THE MOLECULAR AND CELLULAR LEVELS

Procedures used to diagnose the effects of contaminants at the molecular and cellular levels often focus on biochemical measurements, particularly enzyme systems. The sensitivity, specificity, relevance to population health, and potential use of molecular and cellular indicators have been reviewed by Dixon *et al.* (1985), Fitchko (1986), Giesy *et al.* (1988) and Niimi (1990).

Niimi (1990) identified 13 biochemical tests which were successfully used to measure the effects of contaminants on fish in the laboratory or in the field. Several of these, such as the induction of mixed function oxidase (MFO) enzymes, inhibition of amino levulinic acid dehydratase (ALA-D), inhibition of cholinesterase activity and interference with collagen synthesis, are promising indicators of contaminants. Although several biochemical tests have been used in site-specific studies, only MFO and ALA-D have been used in lake wide monitoring programs to assess the health of Great Lakes fish. Part 2 of this volume discusses equivalents that have been used in wildlife.

5.1.1 Mixed Function Oxidase (MFO) Enzymes

Mixed function oxidase enzymes are found in invertebrates, fish, birds, and mammals. They are important in reproduction because they metabolize steroid hormones; regulate moulting in aquatic invertebrates and metabolize foreign compounds facilitating their excretion (Lee, 1988). Environmental contaminants are metabolized in the liver, kidney and intestine. The highest level of enzyme activity is in the liver. Toxic chemicals are metabolized by oxidation, hydroxylation and dealkylation into polar and water-soluble metabolites which are subsequently excreted (Lee, 1988). The MFO enzyme system is a non-specific indicator of chemical exposure because it is induced when fish are exposed to PCBs, polynuclear aromatic hydrocarbons and dioxins (Luxon *et al.*, 1987).

Many biochemical indicators are difficult to relate to population health. However, the role of MFO enzymes in the regulation of steroid hormones suggests that enzyme induction may serve as an early warning indicator of reproductive failure, although the literature is inconsistent on this point. Preliminary laboratory and field studies of flatfish in Puget Sound support this hypothesis (Johnson *et al.*, 1988). Marine studies in San Francisco Bay correlated MFO induction in starry flounder with reduced fertilization success (cited in Lee, 1988). However, Lee also reported several cases where viable fish populations with high MFO activity were reproducing successfully.

MFO enzymes can also activate chemical carcinogens such as B[a]P and initiate tumour production in fish. B[a]P is not carcinogenic itself but the metabolites of B[a]P, formed after MFO metabolism, are potent mutagens and carcinogens known to react with cellular genetic material and induce liver tumours in fish (Varanasi *et al.*, 1984). Although this relationship has been confirmed in laboratory experiments, Fabacher and Baumann (1985) could not find a relationship between MFO activity and chemical contamination in brown bullheads with tumours. This was probably because of the long lag time between the metabolic activation of carcinogens and the development of the tumours (Kirby *et al.*, 1989).

Luxon *et al.* (1987) conducted the first basin-wide study of MFO activity in Great Lakes fish. They examined MFO activity in lake trout from Lakes Ontario, Huron, Superior, and from Lake Opeongo, an inland lake in

Algonquin Park, Ontario. MFO activity in fish from western Lake Ontario (the most contaminated site) was 6 to 62 times greater than that in fish from other location (Figure 2). Although Luxon *et al.* (1987) did not look for adverse reproductive effects, there are indications from other studies of reproductive impairment in lake trout from Lake Ontario (Ruby and Cairns, 1983; Fitzsimons and Cairns, 1987). More recent work (Hodson *et al.*, 1989) confirms the preliminary work on MFO enzymes by Luxon *et al.* (1987). Lake trout and white suckers from Lakes Ontario and Michigan had higher levels of MFO activity relative to fish from Lakes Superior, Huron and Erie (Figures 2 and 3). MFO activity in white suckers confirmed that fish caught in the nearshore zone had higher MFO activities than fish caught in the open lakes.

Binder and Letch (1984) induced MFO enzymes in Lake Michigan lake trout embryos. They found that embryos from contaminated lake trout had MFO activity 3.5 to 6.5 times higher than the activity from similar stocks of hatchery fish. The results indicated that effects of parental exposure were experienced by the offspring. MFO activity decreased several months after the embryos were transferred to clean water.

The significance of contaminants transfer from parents to developing embryos may be particularly important if eggs are exposed to PAHs or other chemicals that require metabolic activation to become mutagens. Laboratory studies (Hendricks, 1982; Metcalfe *et al.*, 1988) have shown that fish are most sensitive to carcinogens during the egg and fry stages of development. Hendricks (1982) briefly immersed rainbow trout eggs in aflatoxin, a potent carcinogen. Ten months later a high proportion of the exposed fish had developed liver tumours. This showed that aflatoxin penetrated the protective egg membrane and was metabolized by the developing embryo.

The possibility of tumour development is increased by exposure to environmental mutagens and carcinogens during the early life stages. However, there are only a few chemicals like aflatoxin in the Great Lakes environment that are capable of direct tumour induction. The most common mutagens in the Great Lakes are the polynuclear aromatic hydrocarbons, such as benzo(a)pyrene (B[a]P). But, as mentioned above, B[a]P is not itself mutagenic. It becomes mutagenic only after it has been synthesized by the MFO system. The ability of fish to metabolise B[a]p increases as MFO activity increases (Stein *et al.*, 1987). As a result, embryos which are exposed to MFO

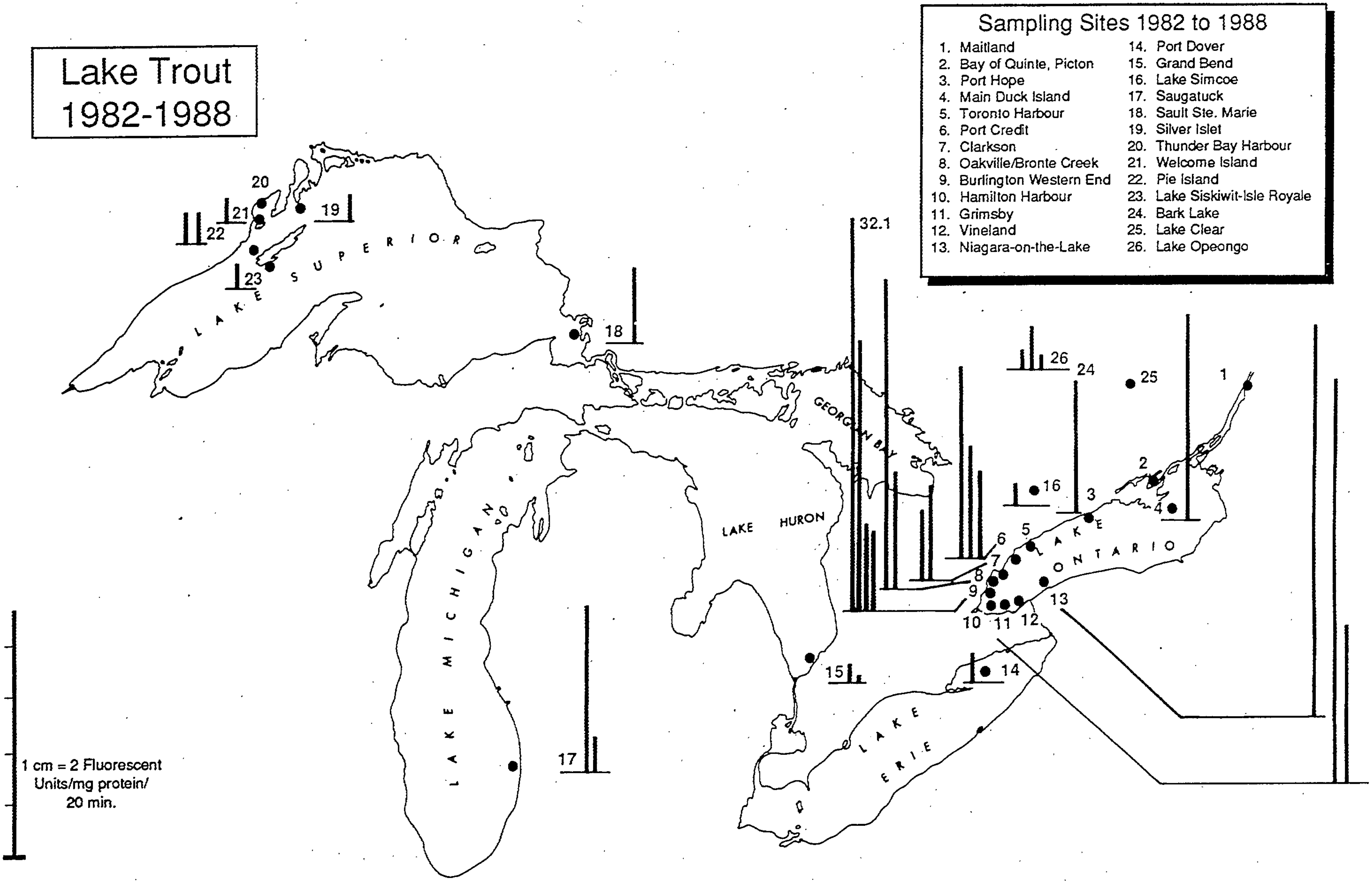

Lake Trout
1982-1988
Sampling Sites 1982 to 1988
1. Maitland
2. Bay of Quinte, Picton
3. Port Hope
4. Main Duck Island
5. Toronto Harbour
6. Port Credit
7. Clarkson
8. Oakville/Bronte Creek
9. Burlington Western End
10. Hamilton Harbour
11. Grimsby
12. Vineland
13. Niagara-on-the-Lake
14. Port Dover
15. Grand Bend
16. Lake Simcoe
17. Saugatuck
18. Sault Ste. Marie
19. Silver Islet
20. Thunder Bay Harbour
21. Welcome Island
22. Pie Island
23. Lake Siskiwit-Isle Royale
24. Bark Lake
25. Lake Clear
26. Lake Opeongo
LAKE SUPERIOR
LAKE MICHIGAN
LAKE HURON
GEORGIAN BAY
LAKE ONTARIO
LAKE ERIE
32.1
1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18
19
20
21
22
23
24
25
26
1 cm = 2 Fluorescent Units/mg protein/ 20 min.

Figure 2

Mixed function oxidase (aryl hydrocarbon hydroxylase) activity in lake trout (*Salvelinus namaycush*) from the Great Lakes captured between 1982 and 1988 (fluorescent units/mg postmitochondrial supernatant protein/ 20 min.). From Hodson *et al.*, 1989.

Note: Lake trout were not found at sites 1, 5, 10, 12, 20, 24, and 25.

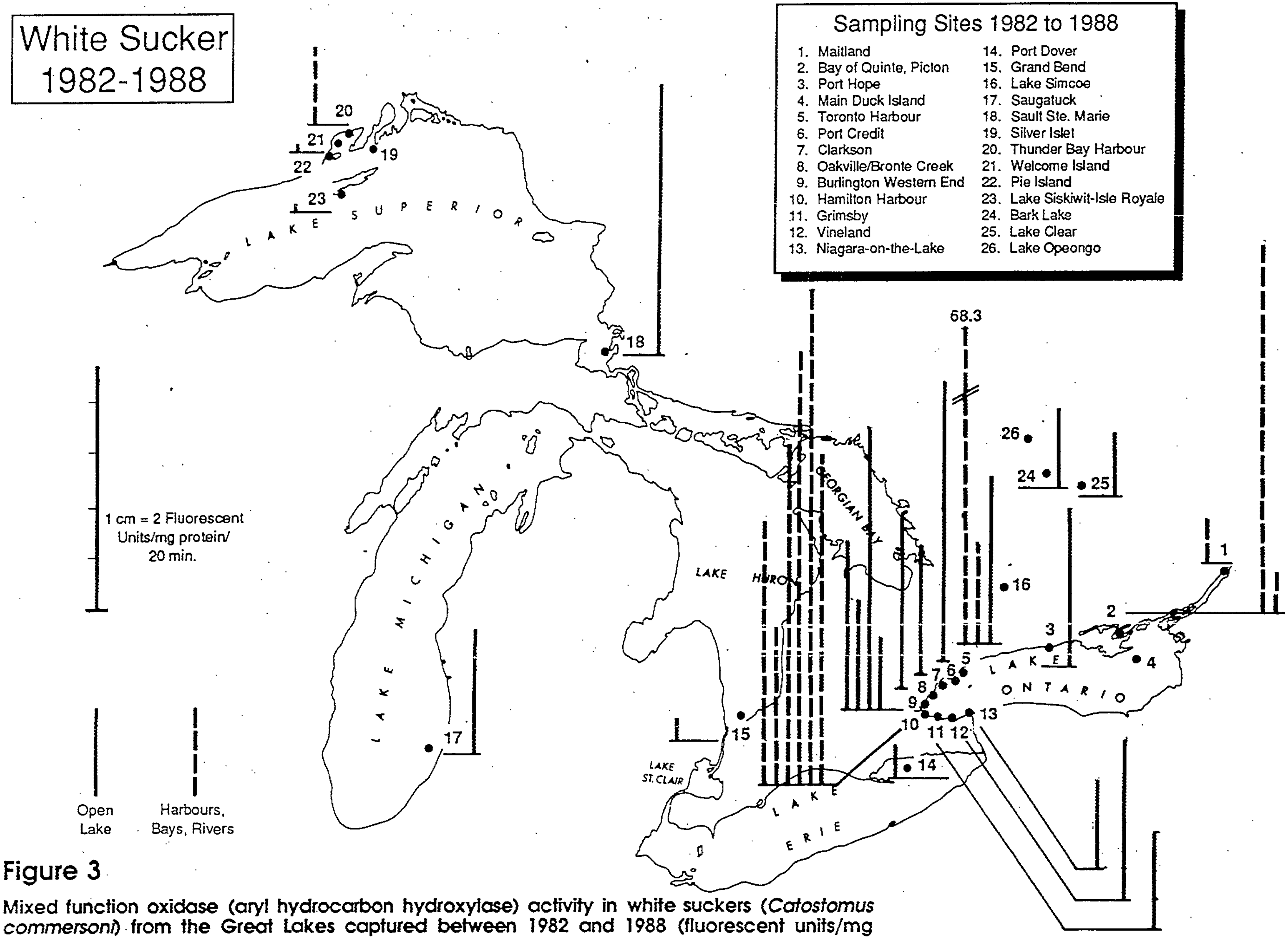

White Sucker
1982-1988
Sampling Sites 1982 to 1988
1. Maitland
2. Bay of Quinte, Picton
3. Port Hope
4. Main Duck Island
5. Toronto Harbour
6. Port Credit
7. Clarkson
8. Oakville/Bronte Creek
9. Burlington Western End
10. Hamilton Harbour
11. Grimsby
12. Vineland
13. Niagara-on-the-Lake
14. Port Dover
15. Grand Bend
16. Lake Simcoe
17. Saugatuck
18. Sault Ste. Marie
19. Silver Islet
20. Thunder Bay Harbour
21. Welcome Island
22. Pie Island
23. Lake Siskiwit-Isle Royale
24. Bark Lake
25. Lake Clear
26. Lake Opeongo
LAKE SUPERIOR
LAKE MICHIGAN
LAKE HURON
GEORGIAN BAY
LAKE ST. CLAIR
LAKE ERIE
LAKE ONTARIO
68.3
1 cm = 2 Fluorescent Units/mg protein/ 20 min.
Open Lake
Harbours, Bays, Rivers

Figure 3

Mixed function oxidase (aryl hydrocarbon hydroxylase) activity in white suckers (*Catostomus commersoni*) from the Great Lakes captured between 1982 and 1988 (fluorescent units/mg postmitochondrial supernatant protein/20 min.). From Hodson *et al.*, 1989.

Note: White suckers were not found at sites 4, 16, 19, 21, and 26.

inducers such as PCBs and dioxins from yolk fat produce higher quantities of mutagenic metabolites and have an increased risk of tumour induction.

5.1.2 Amino Levulinic Acid Dehydratase (ALA-D)

Amino levulinic acid dehydratase (ALA-D) occurs in most bacteria, plants and animals. Its primary role is to catalyze the synthesis of porphobilinogen, a precursor of hemoglobin. In fish, ALA-D is found in the liver, spleen, kidney, and blood (erythrocytes). Hodson *et al.* (1984b) summarized the results of several years of laboratory and field research demonstrating the relationships between ALA-D activity, blood lead concentrations and the adverse effects of lead exposure. These adverse effects included cellular changes, deformities, neurotoxicity and anaemia. They found that ALA-D activity was a sensitive indicator of inorganic lead. High concentrations of inorganic lead in water or food caused a corresponding increase in blood lead levels and a decrease in blood ALA-D activity.

The inhibition of blood ALA-D has been used to measure lead exposure in lake trout, carp and white suckers from Lake Ontario and the St. Lawrence River (Hodson *et al.*, 1984b). Laboratory relationships were confirmed in the field. Blood ALA-D activity in lake trout was negatively correlated with blood lead levels and showed a trend from low lead exposure (high ALA-D activity) in the less contaminated eastern end of the lake to high lead exposure (ALA-D inhibition) in the more contaminated western end. However, when the study was repeated the following year, there were no differences in blood lead or ALA-D levels in lake trout from the eastern and western basins. Lead levels remained low and ALA-D activity remained uniformly high (Hodson *et al.*, 1984c).

Although the lake trout data were not consistent between years, the results demonstrate the difficulty applying sensitive health indicators in the field. Sensitive, specific diagnostic tools such as ALA-D respond to small changes in environmental concentrations. The results may be confounded by several factors which affect environmental exposures, such as fluctuations in contaminant emissions, the chemical state of the contaminant, changes in water current and the immigration of unexposed fish into the area.

Several of these factors influenced the results of an ALA-D study conducted near an industrial source of lead in the St. Lawrence River.

Although ALA-D levels in carp and white suckers were reduced near the source, there were contaminated and uncontaminated fish at sites upstream and downstream (Hodson *et al.*, 1984b). Furthermore, the source was emitting alkyl lead. This is an organolead that accumulates in fish tissue but does not inhibit blood ALA-D. As a result, nearshore benthivores such as carp and white suckers had high concentrations of blood lead and variable ALA-D activity. The level of activity was affected by exposure history and the ratio of organolead to inorganic lead. Fish with high blood lead levels and severe ALA-D inhibition indicated that there had been exposure to inorganic lead; fish with high blood lead levels and no ALA-D inhibition indicated exposure to alkyl lead. Both the organic and inorganic forms of lead are extremely toxic to fish.

The work of Hodson *et al.* (1984b) demonstrated that fish in the vicinity of an industrial lead discharge were exposed to high levels of lead. They confirmed that nearshore benthic fish were the best indicators for monitoring health effects and they provided good evidence that uncontrollable field variables can confound the applicability of sensitive diagnostic health indicators.

5.2 EFFECTS OF CONTAMINANTS AT THE INDIVIDUAL AND POPULATION LEVELS

5.2.1 Lake Trout Reproduction in Lake Michigan

The long-term investigation (12 years) of increased mortality of lake trout and chinook salmon fry from Lake Michigan is one of the best examples of suspected chemical effects on fish populations. This investigation attempted to identify chemical contaminants in lake trout from Lake Michigan and link them to the excessive mortality observed during the early stages of development. Although the causative agents were not positively identified, circumstantial evidence strongly suggested that chemicals were involved. The results of this investigation have been reviewed by Mac (1988).

Mortality of coho salmon and rainbow trout fry was first reported in Lake Michigan in 1969. Although the exact causes were unknown, the symptoms were similar to those reported in lake trout fry exposed to high levels of DDT (Burdick *et al.*, 1964). Research to identify the potential effects of DDT and

PCBs on reproduction in lake trout from Lake Michigan began in 1972. Normal reproductive success (survival rates of between 50 to 78%) of Lake Michigan lake trout fry were reported in 1972, 1974 and 1975. However, in 1978 the fry survival dropped to 20%. When the study was repeated in 1980, only 3% of the fry survived to 139 days post-hatch. Excessive mortality continued until 1982. Survival since 1982 has been normal.

At the same time (1981), an unusual mortality rate (18%) was recorded for chinook salmon stocks. The symptoms were similar to those observed in lake trout. The fry hatched, but failed to feed; they swam in circles, lost equilibrium and died over several weeks. The similarity of symptoms in these two species and those reported by Burdick *et al.* (1964) combined with the coincidental timing of mortality (occurring when the fry were 90 to 110 days old at 10°C) suggested a chemical etiology.

Efforts to correlate lake fry mortality with high levels of PCBs (11 mg/kg) and DDT (7 mg/kg) were unsuccessful (Mac *et al.*, 1981). However, Berlin *et al.* (1981) induced significant mortality (30-46%) to lake trout fry following chronic exposures to PCBs and DDE (exposure levels were up to 25 times ambient concentrations). Although PCBs and DDT caused fry mortality in laboratory experiments, it was clear that they could not have been the sole agents responsible for the mortality observed between 1978 and 1981. PCB and DDT concentrations were higher in fish from Lake Michigan during the period of normal reproduction (1972-1975) than they were during the period of high mortality (1978 to 1981).

Mac (1988) concluded that chemicals were involved in the reproductive impairment of Lake Michigan lake trout because:

a) Mortality was restricted to lake trout from southern Lake Michigan. This area was the most heavily contaminated;
b) Mortality occurred during the swim-up stage of development. Fry at this stage of development have the highest contaminant concentrations and are the most sensitive to chemical exposure; and
c) The syndrome peaked simultaneously in lake trout fry and chinook salmon fry (1981).

Subsequently, chemical analysis showed that there were 167 organic chemicals in the flesh of adult lake trout from lower Lake Michigan (Mac, 1988). The effects of these chemicals (either singly or in combination) on lake trout reproduction are unknown. The likelihood of separating out the effects of individual chemicals from mixtures is small since the numbers of permutations and combinations are overwhelming. These data emphasize the difficulty in establishing cause and effect relationships from field observations. Although the evidence strongly suggests a chemical etiology, the responsible chemical(s) may never be identified.

5.2.2 Pathological Anomalies and Chemical Contaminants

Pathology is the study of structural and functional changes. It offers a practical and promising tool for detecting chemical stress in fish populations. The occurrence of ulcers, lesions, fin rot, gill deformities, skeletal abnormalities and tumours in wild fish populations has been reported in both marine and freshwater species (Sindermann *et al.*, 1980; Mix, 1985; Fitchko, 1986; Black, 1988; Warwick, 1988). Field epidemiology suggests that the prevalence of pathological anomalies increases in areas of urban development and industrial pollution. This hypothesis is supported by laboratory evidence confirming that chemicals can cause skeletal deformities, induce epidermal and liver tumours and lower resistance to disease.

Fin Ray Asymmetry

Easily recognized morphological anomalies, such as asymmetry of fin rays, gill rakers and lateral line scales have been shown to be related to poor environmental quality (Valentine *et al.* 1972). Whittle *et al.* (1987) measured fin ray asymmetry in smelt, sculpin and alewife from Lakes Ontario and Huron between 1979 and 1986. They found relationships between contaminant concentrations and the occurrence of fin ray asymmetry in smelt and sculpins. The authors reported that the degree of asymmetry in sculpin populations from Lake Ontario decreased from 57% in 1979 to 18% in 1986. Sculpins are the most contaminated forage fish from Lake Ontario. The decrease in fin ray asymmetry was accompanied by decreasing whole-body contaminant burdens (sum of PCBs, DDT and mirex) from 2.32 mg/kg in 1982 to 1.24 mg/kg in 1985. There were insufficient data to conduct statistical analyses on the relationships between contaminant levels and fin ray asymmetry.

In fish, the thyroid is a diffuse gland located in the ventral anterior portion, between the base of the two opercula. It secretes thyroxin, a hormone responsible for growth regulation of muscle, bone and cartilage. It also plays a role in pigmentation during smolt transformation and energy partitioning (Leatherland and Ferguson, 1989).

The occurrence of thyroid hyperplasia (an increase in the number of thyroid cells) in Great Lakes salmon has been reviewed by Leatherland and Sonstegard (1984) and Noltie *et al.* (1988). Pacific salmon from Lakes Ontario, Erie, Huron, Michigan and Superior exhibit signs of thyroid hyperplasia and hypertrophy (larger cells). Enlarged thyroids are visible as swellings or nodules at the base of the gill arch. In severe cases, one or more nodules may be large enough to prevent the gill cover from completely closing. Visible lesions are called goiters.

Thyroid lesions in coho salmon from the Great Lakes were first recorded in the early 1970s. By 1979-80, goiters were reported in salmon from Lakes Ontario (35%), Erie (89%) and Michigan (16%) (Leatherland and Sonstegard, 1984). In addition to the high frequency of goiter reported in coho from Lake Erie, the fish also displayed poorly developed secondary sexual characteristics and poor reproductive success (Leatherland and Sonstegard, 1984). Embryo mortality in Lake Erie coho salmon (75%) was very high when compared to mortality in coho from Lakes Ontario (20%) and Michigan (20%). Leatherland and Sonstegard (1984) suggested that the combined symptoms were indicative of endocrine dysfunction.

It is not clear what agents were responsible for the goiters and reproductive failure observed in Lake Erie coho salmon. Thyroid hyperplasia may be caused by iodine deficiency. Early studies explored the hypothesis that iodine deficiency may be the causative agent. Iodine levels in Lake Erie, where goiter prevalence was 89%, were double the iodine concentrations in Lake Michigan, where goiter frequency was only 16% (Leatherland and Sonstegard, 1984). Thyroid hormone levels are lower in iodine deficient fish, and low levels of blood hormones are symptomatic of low-iodine induced goiter. Leatherland and Sonstegard (1984) analyzed thyroid hormones in coho salmon from the Great Lakes but were unable to correlate thyroid hormone levels with goiter prevalence. Their studies indicate that goiter in coho salmon was not due to iodine deficiency although in more recent work, Noltie *et al.* (1988) found that thyroid hormones were lower in Pacific pink salmon with large goiters. This suggests that hypothyroidism may be due to iodine deficiency. The role of iodine deficiency as a cause of goiter in Pacific salmon has not been resolved.

Moccia *et al.* (1978) postulated a possible role for goiterogenic contaminants, such as PCBs in the etiology of thyroid hyperplasia. However, there was no correlation between goiter frequency and contaminant levels in Lakes Ontario, Erie and Michigan. In fact, concentrations of contaminants were lowest in Lake Erie where goiter frequency was highest. Experiments to induce goiter in salmon by feeding them with food contaminated with PCBs and mirex were unsuccessful.

Thyroid hyperplasia has been induced in rats fed diets containing Great Lakes coho (Sonstegard and Leatherland, 1979; Villeneuve *et al.*, 1981). Increased thyroid histopathology in herring gulls from Lake Erie (Moccia et al, 1986) supported the observation on Lake Erie coho salmon. These data indicate that there is an unknown goiterogen in the Great Lakes. Leatherland and Sonstegard (1984) listed several goiterogens that are frequently detected in the Great Lakes. These include high concentrations of organic matter, heavy metals, phenols, sulphides, ammonia and certain minerals.

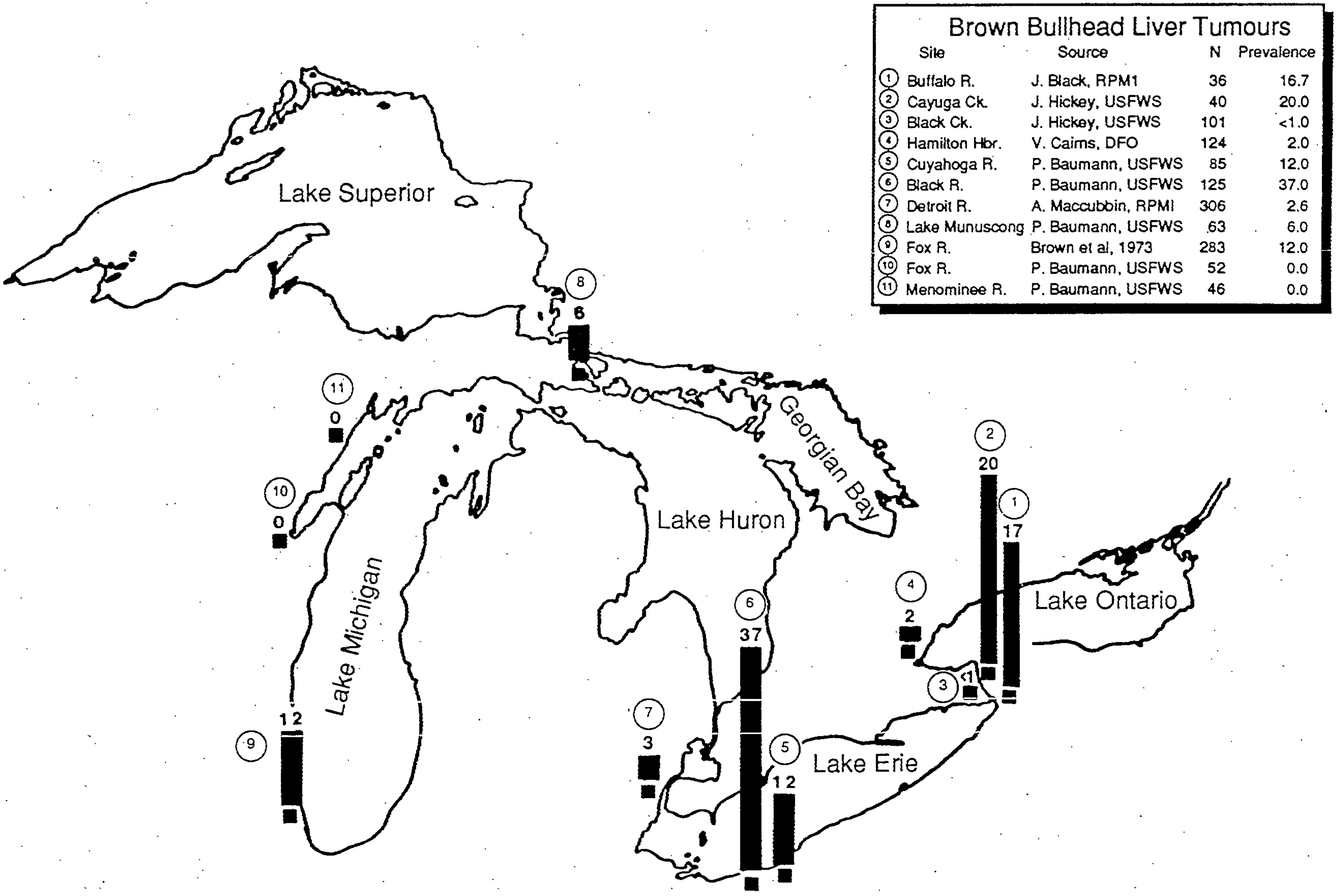
Tumours in Great Lakes Fish

Fish tumour surveys have been conducted in 18 of the 42 Areas of Concern. Three have been conducted in Lake Superior, two in Lake Michigan, two in Lake Huron, four in Lake Erie, four in Lake Ontario, and three of the five connecting channels). Surveys have not been completed for Lake St. Clair, the St. Clair River or the St. Lawrence River. In addition, several independent studies (Sonstegard, 1977; Leatherland and Sonstegard, 1984; Baumann *et al.*, 1987; Black, 1983; Cairns and Fitzsimons, 1988; Smith *et al.*, 1989b) have sampled fish from sites that were not located in Areas of Concern. The occurrence of tumours in Great Lakes fish and their relationship to environmental carcinogens has been reviewed by Black (1984), Black (1988), Fitchko (1986) and Mix (1985) .

Although the presence of tumours in Ontario fish was reported by Higgins and Hadwen as early as 1909 (Sonstegard, 1977), few systematic tumour surveys were undertaken in the Great Lakes prior to 1964. Brown *et al.* (1973) conducted one of the earliest extensive studies (from 1967-1972) in which water quality was thought to be a contributing factor. They compared the tumour frequency in 17 species of fish from the relatively contaminated Fox River, Illinois, with 17 species from the relatively uncontaminated Lake of the Woods, Ontario. Overall, tumour frequency in fish from the Fox River was significantly higher (6.3%) than in the fish from the Lake of the Woods (1.2%). The authors concluded that the increased tumour prevalence was correlated with the degree of chemical pollution. Although Brown *et al.* (1973) found tumours in several species of fish (walleye, pike, carp, brown bullhead, suckers), tumour frequency was low (less than 6.5%) in all species, except brown bullheads which had a 12% occurrence. This observation was the first of many (Baumann *et al.*, 1982; Black, 1983; Malins *et al.*, 1984; Sonstegard, 1977; Smith *et al.*, 1989b) to show that tumour frequencies were higher in fish species living in proximity to contaminated bottom sediments.

The evidence that contaminants may be involved in the etiology of fish tumours (Dawe *et al.*, 1964; Brown *et al.*, 1973) prompted several large scale investigations in the Great Lakes. In a survey conducted between 1973 and 1976, Sonstegard (1977) examined 50,000 fish from contaminated and non-contaminated sites. He reported gonadal tumours in almost 100% of the carp X goldfish hybrids, high frequencies (50%) of lip papillomas in older white suckers from western Lake Ontario and lip papillomas (80% occurrence) in brown bullheads from Hamilton Harbour in Lake Ontario.

Black *et al.* (1980) were the first to report the occurrence of liver tumours in brown bullheads from the lower lakes. They reported that 10 % of the brown bullheads from the Buffalo River were affected by liver tumours. This river is contaminated by effluents from steel and textile industries. In an extensive survey of the Black River in Lake Erie, Baumann *et al.* (1982), found that 37% of the brown bullheads that were 3 years and older had liver tumours. The authors suggested that the tumours were caused by high levels of carcinogenic PAHs in the sediments. These PAHs were discharged into the Black River from steel and coke industries. Since 1982, brown bullheads have been used extensively as a basin wide indicator of chemical



Lake Superior
Lake Michigan
Lake Huron
Georgian Bay
Lake Erie
Lake Ontario
8
6
11
0
10
0
9
12
7
3
6
37
5
12
4
2
3
<1
2
20
1
17

Brown Bullhead Liver Tumours

	Site	Source	N	Prevalence
①	Buffalo R.	J. Black, RPM1	36	16.7
②	Cayuga Ck.	J. Hickey, USFWS	40	20.0
③	Black Ck.	J. Hickey, USFWS	101	<1.0
④	Hamilton Hbr.	V. Cairns, DFO	124	2.0
⑤	Cuyahoga R.	P. Baumann, USFWS	85	12.0
⑥	Black R.	P. Baumann, USFWS	125	37.0
⑦	Detroit R.	A. Maccubbin, RPMI	306	2.6
⑧	Lake Munuscong	P. Baumann, USFWS	63	6.0
⑨	Fox R.	Brown et al, 1973	283	12.0
⑩	Fox R.	P. Baumann, USFWS	52	0.0
⑪	Menominee R.	P. Baumann, USFWS	46	0.0

Figure 4

The percentage of brown bullheads (*Ictalurus nebulosus*) with liver tumours from several sites on the Great Lakes.

carcinogenesis. To date, bullhead liver tumour surveys have been conducted at 11 sites in the Great Lakes (Figure 4).

Tumours have also been found on other species of Great Lakes fish. Sonstegard (1975) reported epidemics of lymphosarcoma (a malignant tumour caused by a virus) in populations of muskellunge and northern pike from unpolluted inland lakes. Black *et al.* (1982) reported liver tumours in sauger (100%) and walleye (30%) from Torch Lake in the Keweenaw Peninsula (Lake Superior). Torch Lake is heavily contaminated with copper mining wastes, but the agent responsible for tumour development are unknown. In a recent review, Black (1988) suggested that fish from Torch Lake have had a long history of exposure to organic agents used in copper refining that contained mammalian carcinogens, such as coal tar and creosote.

Cairns and Fitzsimons (1988) monitored the occurrence of lip and body papillomas in Lakes Ontario and Huron. They confirmed the results of Sonstegard (1977) that papilloma frequency was highest in the western end of Lake Ontario. More recent surveys (Figure 5) have indicated an increased prevalence of lip papillomas in white suckers from the Ganaraska River and Cobourg Creek area.

Papillomas were found in white suckers from Lakes Superior, Huron, St. Clair, Erie and Simcoe. Although the neoplasm occurs in fish from many parts of the Great Lakes, the prevalence is higher in fish from urban areas. Tumour surveys in fish from northern Lake Huron indicated that white suckers from non-urban environments had a low prevalence of lip papillomas. The prevalence in older fish from the Serpent River and McLennan's Creek in South Baymouth was 1% and 10% respectively. The widespread distribution of the neoplasm at relatively low levels (less than 15%) suggests an infective process and is consistent with the observation by Sonstegard (1977) that a C type virus is associated with the papilloma. Efforts to induce papillomas by abrasive contact and cell free inoculations were unsuccessful (Sonstegard, 1977). Recent studies have not been able to confirm the presence of virus particles (Smith *et al.*, 1989a) although papillomas have been shown to develop and regress in fish held in the laboratory under contaminant free conditions (Smith and Zajdlik, 1987). Papilloma prevalence between 1983 and 1987 (Figure 5) in Lake Ontario has changed very little since the first reports in 1973 by Sonstegard (1977).

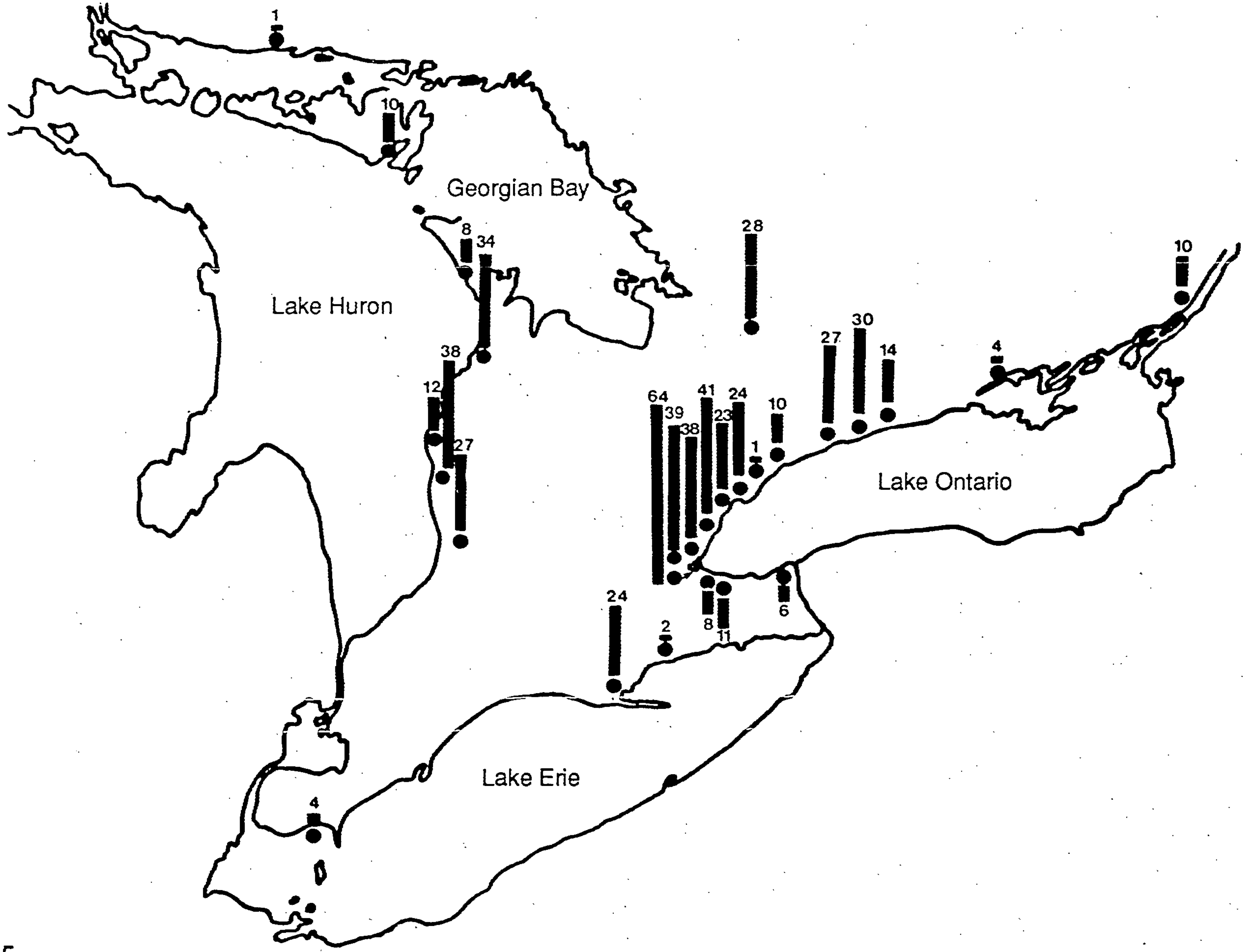

1
10
Georgian Bay
28
8
34
10
Lake Huron
27
30
14
4
38
12
64
41
24
10
39
23
38
1
27
Lake Ontario
24
2
8
11
6
4
Lake Erie

Figure 5

The percentage of white suckers (*Catostomus commersoni*) 7 years and older from Lakes Huron, Erie and Ontario with lip papillomas. Approximately 150-200 fish were collected from each site. Samples were collected between 1983 and 1987 by the Department of Fisheries and Oceans.

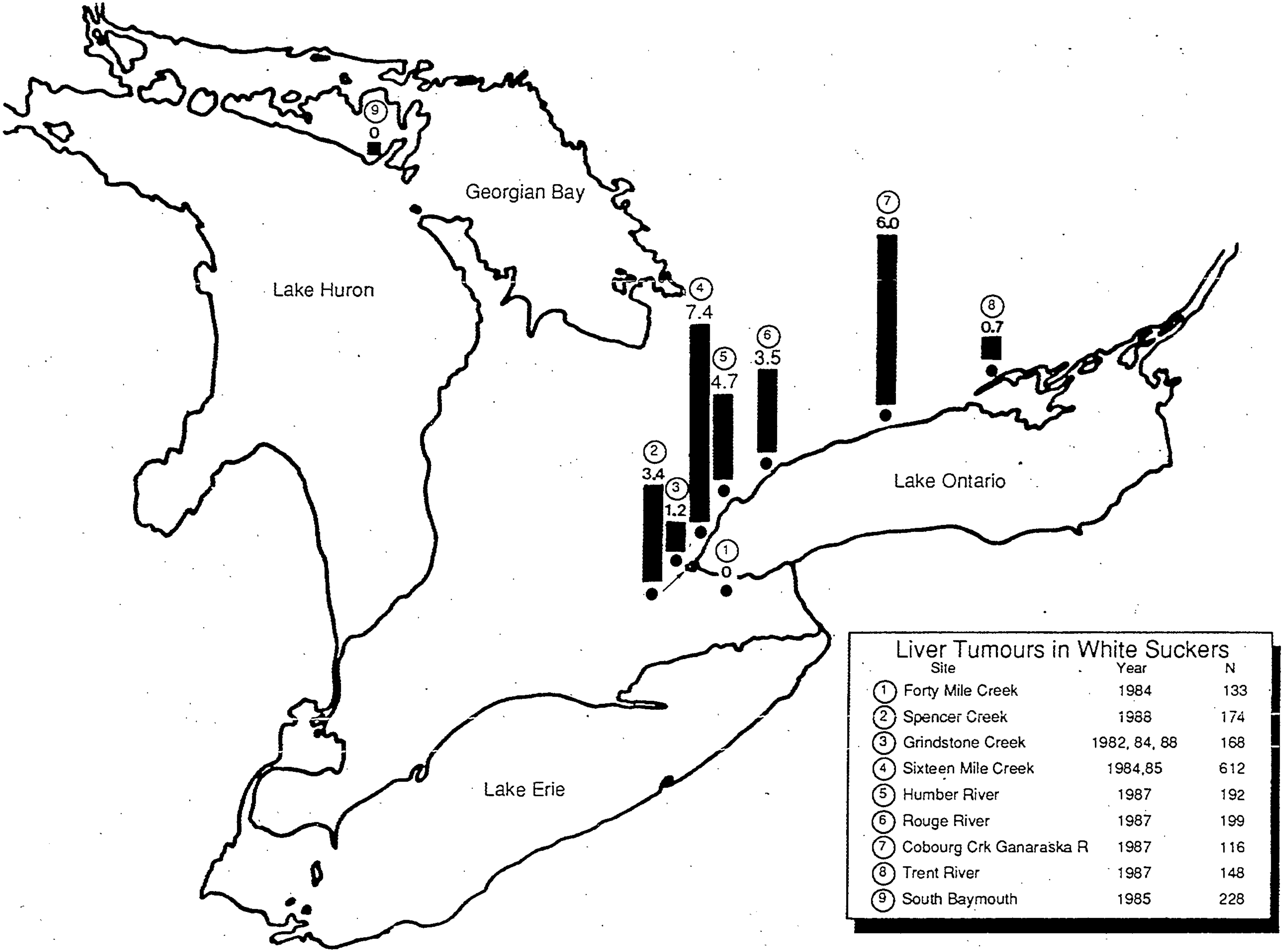
There have been no studies relating the occurrence of lip papillomas in white suckers to environmental contaminants.

In addition to benign lip papillomas, white suckers from Lake Ontario were also affected with bile duct and liver cell tumours, The distribution of liver tumours was similar to the distribution of lip papillomas. The occurrence of total liver tumours was highest in Sixteen Mile Creek (7.4%) in western Lake Ontario and lowest in Forty Mile Creek (0%) (Figure 6). Liver tumour frequency decreased from west to east (3.5%) and then suddenly increased in the Ganaraska River/Cobourg Creek area (Figure 6). There were no liver tumours present in white suckers from South Baymouth, a control site on Lake Huron.

A large proportion of the white suckers from all sites studied were affected by bile duct hyperplasia (an increase in the number of bile ducts) This suggests that non-chemical agents (such as liver parasites) may be responsible. The relationship between liver parasites and liver tumours is unknown. Hayes (personal communication) suggests that parasites may be involved in tumour promotion. Fish in uncontaminated environments respond to bile duct damage (caused by parasites) by replicating more bile ducts (hyperplasia). Chemical carcinogens may cause effects on DNA. If parasites and carcinogens occur together, DNA may be replicated before normal mechanisms can repair it. As a result, the altered DNA may be permanently incorporated into the cell, an important step in chemical carcinogenesis.

The occurrence of liver tumours in white suckers was reported by Dawe *et al.* (1964) in 3 of 12 fish from Deep Lake, Maryland. However, extensive surveys of white suckers conducted in 1973-1974 found no liver tumours in 3208 individuals from several locations in the Great Lakes (Dawe *et al.*, 1976).

Hepatocellular carcinomas (liver cell tumours) were found in white suckers from western Lake Ontario only (Hamilton Harbour, Sixteen Mile Creek, Humber River, the Rouge River and Toronto Harbour (Cairns and Fitzsimons, 1988). Their occurrence in such a geographically restricted area strongly suggest a chemical etiology especially as this type of tumour is often associated with chemical exposure. Although no specific carcinogen has been associated with liver tumours in white suckers, Kirby *et al.* (1988) have


Georgian Bay
Lake Huron
Lake Ontario
Lake Erie
⑨ 0
⑦ 6.0
⑧ 0.7
④ 7.4
⑥ 3.5
⑤ 4.7
② 3.4
③ 1.2
① 0

Liver Tumours in White Suckers

Site	Year	N
① Forty Mile Creek	1984	133
② Spencer Creek	1988	174
③ Grindstone Creek	1982, 84, 88	168
④ Sixteen Mile Creek	1984,85	612
⑤ Humber River	1987	192
⑥ Rouge River	1987	199
⑦ Cobourg Crk Ganaraska R	1987	116
⑧ Trent River	1987	148
⑨ South Baymouth	1985	228

Figure 6

The percentage of white suckers (*Catostomus commersoni*) from Lake Ontario with liver tumours. Samples were collected between 1982 and 1987 by the Department of Fisheries and Oceans.

suggested that exposure to PAHs may play a role in the later stages of cancer development. They reported increased levels of mutagenic B[a]P metabolites in the bile of white suckers from western Lake Ontario, compared to fish from Lake Huron (Kirby *et al.*, 1989). These results strongly suggest a chemical etiology.

The Case for Chemical Carcinogenesis

The stages of chemical carcinogenesis have been described by Hayes and Ferguson (1989). The process in fish is similar to carcinogenesis in mammals. It begins with exposure to a carcinogen resulting in lesions in the DNA. If the damaged DNA is replicated before it can be repaired, the lesion persists. This is the 'initiation' stage of carcinogenesis. The promotion stage is characterised by the selective growth of the altered cell. This may be caused by chemical exposure or physical damage (such as parasites) and it results in cell transformation leading to expansive growth and possibly to chromosomal alterations and malignancy.

Several reviews have examined the relationship between chemical carcinogens in the environment and the presence of tumours in fish (Black, 1984; Black, 1988; Mix, 1985). Not surprisingly, there has been much difficulty proving that chemical exposure in the Great Lakes is definitely responsible for tumours observed in the field. Despite the absence of "proof", the circumstantial evidence is overwhelming. Mammalian carcinogens have caused papillomas and liver tumours in fish in the laboratory. Chemicals extracted from contaminated sediments have successfully induced tumours in fish brought into the laboratory: Tumour frequencies are higher at locations contaminated by chemical carcinogens; and analyses of bile indicates that fish are metabolizing chemicals such as B[a]P into carcinogens. This "proof" is confounded by incomplete knowledge of fish movement (exposure patterns) and the prolonged latency period between exposure and tumour development. For example, white suckers are rarely affected by papillomas or liver tumours before maturity at 4 years of age and tumour frequency increases until approximately 10 years of age.

Given the large number of contaminants in the Great Lakes, it is unlikely that carcinogenesis in wild fish populations can be definitely linked to a specific chemical. Mammalian epidemiologists rely on several criteria to separate environmentally induced neoplasia from genetic predisposition. Dawe (1987) suggested that factors such as diet, geographic distribution and life style influence exposures to carcinogens, and are responsible for the broad range of tumour types observed and the increasing tumour prevalence in older animals. He concluded that "a relatively high prevalence (which means as little as 1% or greater) of one or more tumour histiotypes in a feral (wild) species in a habitat where there is no reason to suspect constrictions on the gene pool, and where known xenobiotics exist, favours an environmental etiology and justifies expenditure of effort towards identifying carcinogens."

Using these criteria, it is possible to identify several tumour types that are not likely to be associated with chemical carcinogens. For example, lymphosarcoma (a malignant tumour of Great Lakes muskellunge) is caused by a virus and occurs in fish from contaminated and uncontaminated sites (Sonstegard, 1975). Similarly, the occurrence of gonadal tumours in carp X goldfish hybrids is unlikely to be related to chemical exposure since the tumour occurs in almost all older males and prevalence does not seem to be related geographically to areas of high or low chemical contamination. Gonadal tumours have been found in hybrids from all sites sampled on the Great Lakes, including a relatively uncontaminated site at Kincardine on Lake Huron (Sonstegard, 1977). Mix (1985) has suggested that the tumours in carp X goldfish hybrids may be associated with genetic factors because hybridization is associated with gonadal tumours in other species (Japanese carp hybrids). The appearance of papillomas in white suckers removed from environmental exposure (Smith and Zajdlik, 1987) suggests that the papilloma may not be a useful indicator of contaminated environments, even though it meets most of Dawe's criteria.

Perhaps the most convincing evidence for chemical carcinogenesis in Great Lakes fish has been the occurrence of liver and skin tumours on brown bullheads exposed to PAHs. The increased tumour prevalence in brown bullheads from sites contaminated with PAHs combined with the presence of carcinogens in bullhead diets (Maccubbin *et al.* 1985), the occurrence of a broad variety of tumour types ranging from squamous cell carcinoma to bile and liver cell carcinomas and an increased prevalence with age (Baumann *et al.*, 1987), satisfies all of the criteria proposed by Dawe (1987) for an environmental etiology. The association is further validated by laboratory studies conducted by Black *et al.* (1985). These studies demonstrated that epidermal papillomas could be induced in brown bullheads painted with concentrated sediment extract from the Buffalo River. In additional studies, brown bullheads were fed diets containing Buffalo River sediment extract. This treatment produced liver lesions and one tumour. Skin application of sediment extracts from the Buffalo River and the Black River has been shown to be a potent inducer of papillomas in Swiss mice.

TABLE 4. EVIDENCE FOR CHEMICAL CARCINOGENESIS IN BROWN BULLHEADS FROM THE GREAT LAKES USING A GENERALIZED MODEL FOR BENZO(A)PYRENE CARCINOGENESIS

Stage of Carcinogenesis	Observed in Bullheads
1. CARCINOGEN METABOLISM	
Environmental Contamination	X
Exposure to carcinogens	X
Accumulation in tissues	X
Ah receptor binding	no
Synthesis of MFO enzymes	X
PAH metabolism	X
Production of metabolic mutagens	X
2. INITIATION	
DNA binding (adducts)	X
Permanent DNA damage	no
Impaired Cell Replication	no
3. PROMOTION	
Growths stimulus (bile duct hyperplasia)	X
4. TRANSFORMATION	
Expansive growth of liver neoplasms	X
5. PROGRESSION	
Invasion of surrounding tissue	X
Metastasis	no

Laboratory experiments do not prove that contaminated sediments caused the tumours seen in the brown bullheads from the Black and Buffalo Rivers. However, they indicate that sediment bound chemicals affect fish and mammals. Given the data from other sites indicating that the chemical profiles of fish diets are similar to the contaminants found in sediments (Maccubbin *et al.*, 1985; Malins *et al.*, 1984), it is possible to conclude that the same processes that are observed in the laboratory are also occurring in the field. Recent research on brown bullheads demonstrates that fish from contaminated sites exhibit many of the stages of B[a]P carcinogenesis (Table 4). The evidence that chemical contaminants cause tumours in Great Lakes fish is overwhelming, although research is further needed to document affected species and identify the causative chemicals.

5.3 EFFECTS OF CONTAMINANTS AT THE COMMUNITY LEVEL

A community of organisms is an assembly of populations living in a prescribed area of physical habitat. Healthy aquatic communities are diverse and consist of many species that are more or less equally represented. Interactions between species are affected by many variables controlling reproduction, food availability and predator avoidance. Although it may not be possible to predict specific chemical effects at the community level, there are useful field indicators of stressed communities. As water quality deteriorates, sensitive species are eliminated and community interaction change. Contaminant tolerant species thrive in the absence of competition and predation, resulting in a community characterized by large numbers of fewer species. If water quality continues to deteriorate, the number of species decreases further and populations are reduced to low numbers of individually resistant animals. Water quality can deteriorate to the point where biotic communities are entirely absent.

This general pattern of decreasing numbers of species and increasing numbers of resistant individuals occurs at all trophic levels. Munawar *et al.* (1988) noted the loss of picoplankton in areas contaminated with heavy metals and hypothesized that sensitive species would disappear and the resistant ones would emerge. Evans and McNaught (1988) reported changes in the feeding rates of zooplankton which lead to reduction of some species in Saginaw Bay. Although there are good laboratory data describing the effects of contaminants on zooplankton reproduction, grazing rates, biomass and growth (Evans and McNaught, 1988; Fitchko, 1986), field studies in the Great Lakes have been unable to differentiate effects caused by exposure to chemicals from the effects of eutrophication and fish predation (Evans and McNaught, 1988).

Benthic organisms provide the clearest evidence of community response to environmental degradation. Warwick (1988) cites the results of five studies in the Great Lakes where benthic communities were affected by environmental contaminants. In addition to changes in species diversity and abundance, these populations also showed higher frequencies of deformed mouth parts and head capsules. Again, the relationship between chemical exposure and deformities was confirmed by laboratory studies, but field observations were unable to isolate one chemical or class of chemical as the causative agent(s).

Nalepa and Landrum (1988) reviewed the effects of contaminants on benthic communities and reported dramatic changes in community structure in shallow areas near industrial discharges. In some cases, a gradation of response in the communities was observed as distance from the contaminated area increased; but as distance increases it becomes progressively more difficult to establish cause and effect relationships. Responses of benthic communities were predictable, ranging from the absence of sensitive species of *Hexagenia* (in areas where copper concentrations exceeded 595 mg/kg and sediments were visibly contaminated with oil), to benthic communities dominated by two species of pollution-tolerant worms. The latter occurred in Hamilton Harbour, a contaminated basin at the west end of Lake Ontario. Two species of oligochaetes account for more than 90% of the benthic biomass in the harbour and worm densities frequently exceeded 10,000 worms m^{-2} (Hamilton Harbour Stage 1 Report, 1990).

6. SUMMARY

1. Since the beginning of the century, Great Lakes fish communities have changed dramatically. No one factor can be identified as the causative agent. Fish populations have responded to overfishing, the introduction of exotic species (including sea lamprey), habitat destruction, eutrophication and toxic chemicals. The effects of chemicals on fish communities has been obscured by the combined effects of all factors.

2. The effects of toxic chemicals on the sport and commercial fisheries is clearly demonstrated by the history of closed commercial fisheries and the large number of consumption advisories for top predators such as lake trout, siscowet, Pacific salmon and walleye.

3. Lake Ontario lake trout are exposed to high levels of contaminants. These are responsible for increased mixed function oxidase (MFO) activity. Although adverse health effects have not been associated with MFO induction, enzyme activity increases in contaminated environments, indicating that fish are metabolizing toxic chemicals, one of the early stages in chemical carcinogenesis.

4. Contaminants are believed to be the cause of reproductive impairment in lake trout from Lake Michigan and possibly in lake trout from Lake Ontario.

5. There is strong circumstantial evidence that environmental carcinogens are responsible for the occurrence of liver tumours in brown bullheads from the Black, the Buffalo and the Fox Rivers, and possibly in bullheads from several other Areas of Concern. There is no "proof" that chemical carcinogens are responsible for liver tumours in walleye and sauger from the Keweenaw Peninsula, or in white suckers from western Lake Ontario. However, the limited geographic distribution of the effects and their association with contaminated environments indicates a chemical etiology.

6. The occurrence of pathological deformities in fish and invertebrates

increases in contaminated nearshore environments. In severe cases, populations and communities of benthic organisms are affected by the combined effects of eutrophication, degraded habitat, and toxic chemicals.

7. The number of possible interactions between stressors is overwhelming. The small amount of data available on combined effects clearly shows that synergism and antagonism occurs. It is unrealistic to expect that science will identify all the possible effects.

7. ACKNOWLEDGEMENT

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APPENDIX 1. COMMON AND SCIENTIFIC NAMES OF FISH SPECIES

Alewife	*Alosa pseudoharengus*
American eel	*Anguilla rostrata*
American smelt	*Osmerus mordax*
Atlantic salmon	*Salmo salar*
Black crappie	*Poxomis nigromaculatus*
Black fin cisco	*Coregonus nigripinnis*
Bloater	*Coregonus hoyi*
Blue pike	*Stizostedion vitreum*
Brown trout	*Salmo trutta*
Brown bullhead	*Ictalurus nebulosus*
Burbot	*Lota lota*
Carp	*Cyprinus carpio*
Carp hybrid	*Cyprinus carpio X Carassius auratus*
Channel catfish	*Ictalurus punctatus*
Chinook salmon	*Oncorhynchus tshawytscha*
Coho salmon	*Oncorhynchus kisutch*
Deepwater cisco	*Coregonus johannae*
Emerald shiner	*Notropis antherinoides*
Gizzard shad	*Dorosoma cepedianum*
Lake chub	*Couesius plumbeus*
Lake herring	*Coregonus artedii*
Lake sturgeon	*Acipenser fulvescens*
Lake whitefish	*Coregonus clupeaformis*
Lake trout	*Salvelinus namaycush*
Largemouth bass	*Micropterus salmoides*
Muskellunge	*Esox masqinongy*
Northern pike	*Esox lucius*
Pink salmon	*Oncorhynchus gorbuscha*
Rainbow trout	*Salmo gairdneri*
Sauger	*Stizostedion canadense*
Slimy culpin	*Cottus cognatus*
Sea lamprey	*Petromyzon marinus*
Siscowet	*Salvelinus namaycush siscowet*
Smallmouth bass	*Micropterus dolomieui*
Splake	*Salvelinus namaycush X Salvelinus fontinalis*
Starry flounder	*Platichthys stellatus*
Walleye	*Stizostedion vitreum vitreum*
White bass	*Morone chrysops*
White perch	*Morone americana*
White sucker	*Catostomus commersoni*
Yellow perch	*Perca flavescens*

TOXIC CHEMICALS IN THE GREAT LAKES AND ASSOCIATED EFFECTS

VOLUME II PART 2

EFFECTS OF CONTAMINANTS ON WILDLIFE SPECIES

EXECUTIVE SUMMARY

This section of the report documents the effects of toxic substances on wildlife. Although there is a relatively large amount of data available on the spatial and temporal trends of the concentrations of individual chemicals in fish, wildlife and humans, it is difficult to relate them to specific observed effects on biota. This is because there is a complex mixture of chemicals present in the environment and it is almost impossible to relate the observed effects to exposure to individual chemicals. In addition, factors such as altered habitat, exploitation, introduction of exotic species and changes in food supplies could be associated with some of the observed effects.

Several methods have been used to study the effects on wildlife species. These include surveys of breeding populations, reproductive success, and occurrence of congenital anomalies. Recently, biochemical indicators have also been investigated in wildlife populations. These do not indicate that the health of individuals is affected, but do show that organisms have been exposed and their metabolism has been altered. They include induction of mixed function oxidase enzymes, porphyria, changes in liver vitamin A (retinol) status, changes in thyroid function and changes in the genetic material.

These methods have shown that effects in 11 species of wildlife have been attributed to contaminants in the Great Lakes. The species are mink, otter, double-crested cormorant, black-crowned night-heron, bald eagle, herring gull, ring-billed gull, Caspian tern, common tern, Forster's tern, and snapping turtle. Reproduction was most commonly affected (in 9 of the 11 species). Reproductive effects were widespread in the lower Great Lakes in the 1970s, but now are confined to a few, highly contaminated areas. Other effects included population decreases, eggshell thinning (in bird species), behavioural changes, biochemical changes, mortality, and alterations in recruitment.

The most conclusively established cause and effect relationship in Great Lakes biota is the collapse of the cormorant population caused by DDE-induced eggshell thinning. It may also be reasonably assumed that DDE caused the decrease of the bald eagle populations although data from the 1960s and 1970s on residues and eggshell thicknesses are not available for Great Lakes shoreline nests. In addition, the reproductive abnormalities of the cormorant and Forster's tern in Green Bay have been correlated with contaminant levels expressed as dioxin equivalents.

Non-avian species have also been affected. Laboratory studies in which mink were fed diets containing contaminated fish from the Great Lakes have shown that levels of PCBs in the fish can cause reproductive failure in the mink. Populations of mink close to the lakeshore have decreased although the trapping records are difficult to interpret. Otter populations also appear to have been affected. Recent studies on the reproductive success of snapping turtles show that the percentage of unhatched eggs and the percentage of deformities in embryos and hatchlings of individuals nesting on the shorelines of Lakes Erie and Ontario are significantly higher than in an inland population.

It is evident that many wildlife species are being affected by contaminants in the Great Lakes, even though it is usually difficult to attribute effects to exposures to individual chemicals. Although in many cases, populations have recovered, the continued presence of toxic chemicals in the Great Lakes is still causing effects. This indicates that efforts to eliminate toxic chemicals should be strengthened.

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1. INTRODUCTION

This part of the report documents the effects on wildlife that have been attributed to toxic chemicals; residue data are discussed in Volume I. Although there is a relatively large amount of data available on the spatial and temporal trends of individual chemicals in fish, wildlife and humans, it is difficult to relate this information to observed effects in biota. This is because there is a complex mixture of chemicals present and it is almost impossible to relate the observed effects to exposure to individual chemicals. In addition, other factors such as altered habitat, exploitation, introductions of exotic species and changes in food supplies could be associated with some of the observed effects.

There are few data on the effects of toxic chemicals on lower trophic level organisms. Sicko-Goad and Stoermer (1988) stated that "it is particularly discouraging that there is virtually no information available on phytoplankton species that, based on their known distribution, might be particularly sensitive to toxic effects." In the same volume Evans and McNaught (1988) observe that "very little information exists on the effects of toxic substances on the community structure of Great Lakes zooplankton" and Nalepa and Landrum (1988) comment that "whether or not toxic contaminants have altered the structure of Great Lakes benthic communities is difficult to assess." This lack of information makes it difficult to attribute effects in lower trophic level organisms to particular chemicals. This is in addition to the difficulty of separating chemical effects from those of numerous other factors.

Field studies demonstrate that there are correlations between exposures and effects, but the factors discussed above make it difficult to use field data alone to establish dose-dependent causal relationships between exposures and effects. Thus, to establish cause-and-effect relationships it is necessary to supplement field studies with complementary laboratory research. The most conclusive cause and effect relationship in Great Lakes biota is the collapse of the cormorant population caused by DDE-induced eggshell thinning (section 3.3). It may also be reasonably assumed that DDE caused the decrease of the bald eagle population although data on residues and eggshell thickness are not available for Great Lakes shoreline nests. It is thought that DDE also caused the decrease in the osprey population in

North America, but there is little information on populations in the Great Lakes basin.

Reproductive failure of the herring gull and Forster's tern have been linked to chemical contaminants although the specific chemical(s) causing the effects has not been identified. Terrestrial species have also experienced contaminant-related effects. For example, the peregrine falcon was extirpated from eastern North America, including the few pairs in the Great Lakes basin. However, this and other terrestrial species are not considered in this document.

Laboratory studies in which mink were fed diets containing fish from the Great Lakes have shown that the levels of PCBs in the fish can cause reproductive failure in the mink (Aulerich and Ringer, 1977). Similarly, field studies indicate that populations of mink close to the lake shore have decreased although trapping records are difficult to interpret (section 3.1). Current studies on cormorants and Caspian terns from Lakes Michigan and Huron have demonstrated a relationship between organochlorine levels in eggs (expressed as 2,3,7,8-TCDD toxic equivalents) and reduced reproductive success (sections 2.3.2 and 3.8).

This report focuses on the spatial and temporal trends of known effects. It is not comprehensive. Species have only been included in this report where substantial data sets are available. Methodologies are presented first, followed by descriptions of the effects on a species-by-species basis. A list of the species considered and a summary of the major contaminant-related effects are shown in Table 1. These changes range from trends in the size of the entire breeding population in the Great Lakes basin to physiological changes in individuals.

TABLE 1. SPECIES OF WILDLIFE CONSIDERED IN THIS REPORT AND PRINCIPAL CONTAMINANT-RELATED EFFECTS OBSERVED IN THE GREAT LAKES[1]

Species	Population decrease	Effects on reproduction	Eggshell thinning	Congenital malformations	Behavioral changes	Biochemical changes	Mortality	Alterations in recruitment
Mink	X	X	NA	NE	NE	NE	X	?
Otter	X		NA	NE	NE	NE	?	?
Double-crested Cormorant	X	X	X	(X)		X	?	?
Black-crowned Night-Heron	X	X	X	X		X	?	?
Bald Eagle	X	X	X	NE		NE	NE	?
Herring Gull		X	X	X	X	X	X	
Ring-billed Gull				X		NE	X	
Caspian Tern		X		X	NE	NE		X
Common Tern		X	X	X		X		
Forster's Tern		X		X	X	X		
Snapping Turtle	NE	X	NA	X	NE	NE	NE	NE

X = effects documented
NA = not applicable
NE = not examined
? = suspected since population declined

1. Observations marked with an X have been reported in the published literature.
2. Unpublished records of congenital malformations exist for the double-crested cormorant, great blue heron and the Virginia rail.

2. METHODOLOGIES USED TO STUDY EFFECTS ON WILDLIFE SPECIES

This report draws on information from surveys of breeding populations, studies of reproductive success, occurrence of congenital anomalies and the measurement of physiological/biochemical indicators.

2.1 BREEDING POPULATIONS AND REPRODUCTION

Censuses of colonial waterbirds in the Great Lakes have been conducted using ground and aerial counts (Blokpoel *et al.*, in press). Detailed studies of the demography of a colony are possible if a high proportion of the individuals are banded. Banding enables the survival, reproductive history and movements of individuals to be followed. Ludwig (1979) cannon-netted Caspian terns to determine the origine of terns breeding in colonies in the Great Lakes and to determine their mortality rates. However, this technique can cause considerable disturbance to the colony (Blokpoel, 1979).

The determination of reproductive success requires several visits to a colony and although greater understanding may result with each visit, the disturbance to the colony may alter its reproductive success. Fetterolf's detailed studies (1983) of the effects of disturbance on the reproductive success of ring-billed gulls suggest that this phenomenon can be significant. Mineau and Weseloh (1981) devised a protocol to obtain the gross reproductive output of herring gulls with minimal disturbance and effort which has been used for monitoring this species.

The only population data available for mink and otter are harvest statistics from commercial trappers. These can be confounded by several variables, including weather, trapping effort, fur prices, changes in habitat and trapping methods.

The adverse reproductive effects of toxic chemicals may result from their toxicity to the embryo, alterations in the strength and porosity of the eggshell and/or effects on the behaviour of the attending adults. It is possible to separate these effects using egg exchange experiments in which fresh eggs are removed from the contaminated colony and are substituted with eggs from uncontaminated colonies and vice versa (Table 2). It is also possible to incubate eggs from contaminated and uncontaminated colonies artificially and to examine them for embryotoxic effects. While this method is not as realistic as egg exchange experiments, it overcomes the logistical difficulties and permits detailed studies on embryo development in a common, controlled environment. Detailed studies of nest attentiveness have been conducted using radio telemetry. This records the core and surface temperatures and the amount of light falling on a wax-filled egg, substituted for a normal egg. From this information it is possible to determine the amount of time that the eggs are incubated and the extent to which they are allowed to cool (Fox *et al.*, 1978).

TABLE 2. COMPARISON OF EGG EXCHANGE AND ARTIFICIAL INCUBATION EXPERIMENTS FOR HERRING GULLS ON LAKE ONTARIO (1976) AND FORSTER'S TERN IN LAKE MICHIGAN (1983)

Herring Gull, Lake Ontario 1976

Source of adult	Source of egg	percent hatched	Information obtained
Clean	Clean	86	normal reproduction
Clean	Dirty	10	strong intrinsic factors
Dirty	Clean	7	strong extrinsic factors
Dirty	Dirty	2	both intrinsic and extrinsic factors
Artificial	Clean	60	normal reproduction
Artificial	Dirty	37	intrinsic factors

Source: D. Peakall, 1980

Forster's Tern, Lake Michigan, 1983

Source of adult	Source of egg	percent hatched	Information obtained
Clean	Clean	88	normal reproduction
Clean	Dirty	94	no intrinsic factors
Dirty	Clean	11	strong extrinsic factors
Dirty	Dirty	55	both intrinsic and extrinsic factors
Artificial	Clean	75	normal reproduction
Artificial	Dirty	37	strong intrinsic factors

Source: Kubiak et al., 1989

Eggs which fail to hatch can be opened to determine the state of the embryo. A portable detector has been developed which enables the viability of the embryo to be determined nondestructively, by amplifying the sounds resulting from embryonic movements (Mineau and Pedrosa, 1986). This is particularly useful for heavily pigmented eggs (e.g., gulls and terns) because normal "candling" procedures cannot be used. The detector has been found to have >95% accuracy for herring gulls after 16 days incubation.

2.2 CONGENITAL MALFORMATIONS

Congenital malformations (birth defects) are uncommon in most wild bird populations. Prevalence and incidence data are rarely available, even in intensively studied colonial waterbirds. This is unfortunate because these data could serve as sensitive indicators of developmental toxins in the food web. Recently, prevalence and incidence data were responsible for identifying the presence of embryotoxic levels of selenium in agricultural drainwater in the San Joaquin Valley of California (Ohlendorf *et al.*, 1986; Hoffman *et al.*, 1988). Congenital malformations have been reported in the young of the following seven species of fish-eating birds from nesting locations in the Great Lakes: herring gull, ring-billed gull, common tern, Caspian tern, Forster's tern, black-crowned night-heron, and bald eagle (Scharf and Buckingham, 1976; Gilbertson *et al.*, 1976; Ryder and Chamberlain, 1972; Hoffman *et al.*, 1987; Kubiak *et al.*, 1989; Grier, 1968). In addition, there are unpublished records of congenital malformations in three other species: double-crested cormorant, great blue heron and Virginia rail.

The temporal and spatial distributions of congenital malformations in young fish-eating birds from the Great Lakes have been summarized for 1971 to 1975, 1975 to 1980, and 1981 to 1985 (Figure 1). Five definite clusters of birth defects involving several individuals of multiple species have been observed:

(i) In Lake Ontario, defects have been observed in all six species of fish-eating birds which commonly breed there. Although the largest number of defective individuals was observed between 1971 and 1975, abnormal young were found as recently as 1988;

(ii) In the North Channel of Lake Huron four defective individuals of two species were found between 1976 and 1985. Defective cormorant chicks were observed in this area as recently as 1988;

(iii) In Georgian Bay (Lake Huron), a cluster of four individuals of three species was observed between 1975 and 1985;

(iv) In Lake Huron, off Michigan's Presquile and Alpena Counties, defects were observed in two species of gulls in two colonies between 1971 and 1975. A great blue heron chick with a crossed bill was observed off Alpena County in 1988;

(v) In Green Bay (Lake Michigan) over 80 individuals of six species were found with a variety of defects between 1971 and 1985. Abnormalities continue to be found in this area at a significantly higher rate than to the rest of the Great Lakes, particularly in young cormorants (section 3.3.). Figure 2 shown the spatial variation in the incidence and prevalence of cormorant chicks with crossed bills from 1979 to 1987.

Although no systematic multi-species surveys have been conducted in Green Bay, malformations have been recorded in the young of seven species, all of which are waterbirds and partially, if not totally piscivorous. This suggests a common vector (i.e., the aquatic food web) and a nutritional, microbial or chemical etiology. This is because it is highly improbable that the gene pools of seven species would be similarly and simultaneously affected. Cormorants in Green Bay, as elsewhere in the Great Lakes, are becoming increasingly abundant and widely distributed, and are successfully raising their young to fledging. Hence, the availability and nutritional quality of cormorant food (forage fish) is adequate, suggesting that a biological or chemical agent is responsible for the documented congenital anomalies. When the etiology of the poor hatching success and congenital malformations was investigated, the U.S. Fish and Wildlife Services National Wildlife Health Laboratory found no evidence of infectious disease in eggs or embryos of Forster's terns in Green Bay (Hoffman *et al.*, 1987). However, a factor(s) was present which reduced the survival and growth of embryos and resulted in congenital abnormalities (including crossed bills), increased liver to body mass ratios, and high levels of hepatic microsomal aryl hydrocarbon hydroxylase (AHH) activity (Hoffman *et al.*, 1987; Kubiak *et al.*, 1989). These data implicate a chemically rather than a microbially mediated etiology (i.e., developmental toxicity) and indicate that agent(s) which induce AHH, such as polyhalogenated aromatic hydrocarbons, were involved.

Supernormal clutches resulting from female-female pairing were observed in Lake Ontario (CWS unpublished) and Lake Michigan (Shugart, 1980) herring gull colonies during the 1970s. There is histological and anatomical evidence that when environmentally relevant concentrations of DDT and estrogenic isomers are injected into herring gull eggs, feminization of male embryos is induced (Fry and Toone, 1981). A retrospective examination by Fry of preserved embryos and newly hatched chicks collected by G. Fox from Lake Ontario in 1975 and 1976 showed that five of the seven males were significantly feminized and that five of the ten females had abnormally enlarged oviducts (Fox and Weseloh, 1987). A detailed study (Fry *et al.*, 1987) concluded that supernormal clutches occurred when the sex ratio is skewed towards females and that nest sites are available for female-female pairs or polygamous trios. It is possible that male embryos were feminized during the peak of Great Lakes contamination in the early 1970s.

2.3 BIOCHEMICAL INDICATORS

2.3.1 Introduction

Biochemical indicators are a sensitive and essential component of modern environmental assessments and they complement chemical analyses. If the identity and amounts of chemicals present in the tissues are known and toxicological studies are available, the likely biological effects can be predicted. This is seldom the case, and rarely, if ever, is an organism exposed to a single contaminant. Biochemical measurements and other forms of biological monitoring allow us to:

(1) Identify and quantify bioavailable contaminants;
(2) Determine whether exposure to the mixture of chemicals in the environment results in a biological response and if the response has exceeded homeostatic capacity, and
(3) Assess when contaminant-induced stress has detrimentally affected individuals, populations, or the structure and function of ecosystems.

If a good battery of biochemical tests is available covering the major functions of an organism, then it is possible to assess whether the parameters measured for an individual are within normal limits for the species.

The biochemical markers that have been measured in wildlife in the Great Lakes are listed in Table 3. Part 1 of this volume discussed equivalent biochemical markers that have been used in fish.

This approach circumvents the problem of the effects of mixtures of chemicals and of unknown chemicals in the environment. It is limited by the size, nature and sensitivity of the battery of tests, and the availability of unexposed control populations. It is not possible to test all species, and some species can be tested more easily than others. A similar approach can be used at the community and ecosystem levels, although the diagnostic tools for doing this are currently poorly developed.

TABLE 3. BIOMARKERS OF TOXICITY STUDIED IN WILDLIFE OF THE GREAT LAKES

Biomarker	Species/date	Major Finding
Hepatic mixed function oxidase	Black-crowned night-heron, 1984, 1985 Herring gull, 1981 Forster's tern, 1983 Common tern, 1984, 1985 Cormorant, 1986, 1987, 1988	Elevated enzyme activity
Hepatic porphyrins	Herring gull, 1985	Increased levels
Hepatic Vitamin A	Herring gull, 1982	Decreased levels
Thyroid	Herring gull, 1974, 1980-1982	Enlargement & structural changes
Genetic alterations	Herring gull 1981, 1987 Cormorant, 1987	Decrease in degree of methylation of DNA

Details and references are given in Section 2.3

2.3.2 Mixed Function Oxidase Enzymes

The mixed function oxidase (MFO) enzymes are a major component of the biological defences that protect living organisms from toxic chemicals in the environment. These enzymes add oxygen to a wide range of compounds, rendering them more water soluble, and thus more readily excreted. They are found in many organisms, and evolved to metabolize various endogenous compounds, such as steroids, prostaglandins and fatty acids. Although the induction of MFOs is considered an adaptive response, the metabolites of some xenobiotic chemicals are more toxic than the parent compounds so that metabolism by mixed function oxidases is not always beneficial. Exposure to PCBs and other chemicals may cause enzyme induction which may lead to the increased or altered metabolism of endogenous compounds and other contaminants with production of more toxic metabolites.

Walker (1982) has shown that the activity of MFO enzymes decreases from mammals to birds to fish, although there is some overlap in activity. Levels are higher in herbivores and omnivores than in predators. This may be because omnivores and herbivores have to metabolize natural toxins. Since predators eat herbivores and omnivores, there is less need to metabolize natural toxins and so MFO levels are lower.

The central role of MFO enzymes in detoxication should make them good biomarkers of exposure to xenobiotic detoxification chemicals (Rattner *et al.*, 1989). The mixed function oxidase system has been used in wildlife studies in two ways. First, the MFO system in fish and wildlife collected from the Great Lakes basin has been studied as a biological response to contaminant exposure. Second, the induction of the MFO enzymes aryl hydrocarbon hydroxylase (AHH) and ethoxyresorufin-o-deethylase (EROD) in cultured rat hepatoma cells has been used as a biological measure of the relative toxicity of a tissue extract or chemical introduced into the culture.

Significantly elevated AHH activity was found in the livers of herring gull embryos collected from Lake Ontario and Saginaw Bay in Lake Huron in 1981, when compared to those in embryos from a marine colony (Ellenton *et al.*, 1985). There were no significant differences in activity between livers of embryos from colonies on Lake Superior and southern Lake Huron. There was a good correlation between AHH activity and the concentration of 2,3,7,8-TCDD in eggs from the same colonies. Subsequent studies conducted in 1982, in which the activities of MFO enzymes other than AHH were measured, showed no significant increases in activity between the livers of embryos from Great Lakes colonies and those from the marine colony (Boersma *et al.*, 1986). However, the activity of one enzyme was depressed in seven of eight Great Lakes colonies. Liver tissue from Forster's tern embryos collected in 1983 from Green Bay in Lake Michigan had a mean AHH activity three times higher than the livers of embryos from an inland colony (Hoffman *et al.*, 1987). These authors also reported that induction of liver microsomal AHH was also found during 1984 and 1985 in common tern embryos and chicks and late stage black-crowned night-heron embryos from Green Bay and Saginaw Bay. Livers of day-old cormorant chicks collected in 1988 from Spider Island in Green Bay had a mean EROD activity which was three times higher than that in livers of chicks collected from Lake Winnipegosis (D. Hoffman, personal communication), whereas older chicks from a Lake Ontario colony did not show elevation over those from a marine colony (Peakall, unpublished data).

The toxicity of PCBs, PCDFs and PCDDs has been correlated with their ability to induce AHH (Poland and Glover, 1973; Safe, 1986). Safe (1987) has found good correlations between the *in vitro* induction of AHH in the rat hepatoma cell line and *in vivo* effects, such as inhibition of body weight gain and thymic atrophy. Casterline *et al.* (1983) described a sensitive bioassay for polyhalogenated aromatic hydrocarbons based on the ability of extracts of fish tissue to induce AHH activity in rat hepatoma cells in culture relative to that induced by 2,3,7,8-TCDD. The results are expressed as TCDD equivalents (TCDD-EQs). The advantages of this technique are that it represents an integrated measure of the biological activity of these extremely complex mixtures and it is relatively inexpensive compared to congener-specific analysis of PCBs, PCDFs and PCDDs.

Tillitt *et al.* (in press) measured the EROD induction capability (in the rat hepatoma cell line) of extracts of composite samples of 217 eggs, collected from 41 colonies of fish-eating birds in the U.S. waters of the upper Great Lakes. They found the EROD-based TCDD-EQs in these samples to be the highest in samples from Green Bay and Saginaw Bay and lowest in samples from northwestern Lake Huron and southeastern Lake Superior. The relative ranking of the colonies correlates well with known areas of contamination. Currently, these investigators are using the bioassay as part of investigations of contaminant-related problems in Caspian terns, cormorants and bald eagles. Tillitt *et al.* (in prep.) are finding an extremely high degree of correlation between the EROD induction capability (TCDD-EQs) of egg extracts and certain measures of developmental toxicity and reproductive success in colonial birds. However, the correlations between these measurements and levels of any single chemical measured in conventional residue analyses is very poor (Tillitt *et al.*, in prep). Congener-specific chemical analysis suggests that over 90% of the biological activity of these extracts is contributed by three co-planar PCB congeners. This agrees with the calculations of Kubiak *et al.* (1989), who applied relative TCDD-EQs derived from the rat hepatoma cell bioassay for various PCB congeners, to the results of congener-specific PCB analysis of Forster's tern eggs from a failing colony in Green Bay.

2.3.3 Porphyria

Among the many important functions of the liver is the production of heme. In addition to hemoglobin, heme is also incorporated into several critical enzymes and its production rate is dependent on the requirements for these enzymes. Many toxic chemicals enhance the rate of heme biosynthesis to meet the requirement for mixed function oxidases (section 2.3.2).

Some toxic chemicals have another effect on heme biosynthesis: they cause porphyrias. In porphyrias, the deregulation of heme biosynthesis results in the accumulation of porphyrins. Porphyrins are precursors of heme and are normally present in very small quantities. Alterations in the normal porphyrin pattern can be used as an indicator of exposure to toxic chemicals (Marks, 1985; Silbergeld and Fowler, 1987). Exposure to several organochlorines (including PCBs, HCB and TCDD) produces a form of porphyria which is manifested by the accumulation of highly carboxylated porphyrins (HCPs).

The results of an investigation into the porphyrin patterns in the livers of herring gulls collected in 1985 from several colonies in the Great Lakes are shown in Figure 3. A comparison of material from the Great Lakes with that from two colonies on the Atlantic coast shows that the HCP concentrations in most of the gulls from the Great Lakes were much higher than those from the Atlantic coast. The highest HCP levels were found in gulls from Green Bay (Lake Michigan), Saginaw Bay (Lake Huron) and Lake Ontario. Several porphyrinogenic compounds with widely different potencies are present in the Great Lakes. Although possible correlations between the concentrations of these compounds and the HCP levels were examined, it was not possible to determine which of the potentially porphyrinogenic compounds was responsible for the observed porphyria (Fox *et al.*, 1988)

The argument that high levels of HCPs in Great Lakes herring gulls are caused by exposure to organochlorines is based on the fact that there are very few other known causes of high liver HCPs. In humans, the only other reported causes are rare genetic forms of porphyria, steroid therapy, genetically predisposed individuals and in extremely rare cases, alcoholism. In experimental mammals, the only reported cause of high liver HCPs, other than organochlorines, is aflatoxin B_1. Organochlorines are the only known cause of high levels of HCP in birds. While there is no evidence that the high porphyrin levels observed in herring gulls on the Great Lakes are harmful to their health, HCPs are a sensitive indicator of a biological change induced by exposure to organochlorines.

2.3.4 Liver Vitamin A (Retinol) Status

Retinol is the main natural form of vitamin A. Vitamin A is fat-soluble and it is necessary for vision, reproduction and the maintenance of differentiated epithelia and mucous secretion in higher animals. Over 95% of the body's retinoid reserves are stored in the liver, predominantly as the fatty acid ester retinyl palmitate. Retinoid homeostasis (vitamin A uptake, storage and mobilization) is a highly regulated process. Dietary exposure to a variety of xenobiotic chemicals including cadmium, PAHs, insecticides, PCBs, PBBs and TCDD can alter vitamin A homeostasis in mammals and birds. Brouwer and Van den Berg (1984) reported that the effects on retinoid levels in various strains of mice that had been dosed with 2,4,3',4'- tetrachlorobiphenyl were more pronounced than other biological effects. They also suggested that reduction in retinoid levels is a very sensitive marker for PCBs.

Retinoid concentrations were compared in livers of herring gulls collected from three Great Lakes sites with an Atlantic coast colony (Kent Island, New Brunswick) in 1982 (Spear *et al.*, 1985). The levels in the herring gulls from the Great Lakes were significantly lower than those from the coastal colony. In the Great Lakes, the levels were lowest in samples from Lake Ontario and highest in samples from Lake Superior. The analysis of a larger group of samples over a wider temporal and geographic range has now been completed (Fox and Trudeau, in preparation). They found that liver retinoid levels varied widely in gulls from the Atlantic coast colonies, both between locations and between years. However, levels were generally higher than those in gulls from the Great Lakes (Table 4). Although Great Lakes gulls ingest enough retinoids so that some contain levels equivalent to those in many individuals from the maritime colonies, levels in gulls from several locations in the Great Lakes were very low. This suggests that the homeostasis of vitamin A is altered.

TABLE 4. DISTRIBUTION OF RETINOL AND RETINYL PALMITATE CONCENTRATIONS IN µg/g IN LIVERS OF HERRING GULLS COLLECTED BETWEEN 1980-1984 AND POOLED BY LAKE

		PERCENT OF SAMPLES WITHIN CONCENTRATION RANGE						
FORM	CONCENTRATION RANGE	Atlantic Coast (n=39)	Lake Ontario (n=33)	Lake Erie (n=23)	Detroit River (n=10)	Lake Huron (n=28)	Lake Superior (n=19)	Lake Michigan (n=17)
RETINOL								
	<10	0	15.1	45.8	50.0	17.9	0	11.8
	10-100	15.4	54.5	50.0	50.0	46.4	0	5.3
	101-500	41.0	30.3	4.2	0	25.0	73.7	23.5
	501-1000	20.5	0	0	0	10.7	21.0	11.8
	>1000	38.5	0	0	0	0	5.3	0
	Statistics*	A	BCE	BCE	BCE	BCE	ADF	BCE
RETINYL PALMITATE								
	<100	5.1	18.2	95.8	80.0	3.7	5.3	17.6
	101-500	15.4	81.8	4.2	10.0	42.9	68.4	58.2
	501-1000	15.4	0	0	10.0	28.6	26.3	17.6
	1001-2000	25.6	0	0	0	21.4	0	0
	2001-4000	25.6	0	0	0	3.7	0	0
	>4000	12.8	0	0	0	0	0	0
	Statistics*	A	BCE	BDF	BDF	BDE	BCE	BCE

* Locations not sharing common letters are significantly different ($P<0.05$) on the basis of the Kolmogorov-Smirnov two-sample test.
Source: G. Fox and S. Trudeau (In prep).

Retinol levels in livers of gulls from the Atlantic coast and Lake Superior did not differ significantly, but were higher than those from the other Great Lakes. The concentrations of retinyl palmitate in livers of gulls from the Atlantic coast were significantly higher than in gulls from any of the Great Lakes. Within the Great Lakes, the retinoid stores of gulls from the Detroit River and western Lake Erie were lower than those in gulls from the other locations. The most severe retinoid depletion was observed in colonies on the western basin of Lake Erie and in the Detroit River, followed by those located on Lake Ontario. Figure 4 shows the spatial variation in the total retinoids stored in livers of gulls from Great Lakes colonies in 1985, compared to those from a colony in Newfoundland. The spatial trends are consistent with the larger data set. The relatively low amounts of stored retinoids in gull livers from Lakes Erie and Ontario are very obvious. Information within the larger data set suggest that the degree of depletion was more severe in 1985 collections from Lake Ontario than the 1982 collections. In Lake Huron, depletion of retinol levels in gulls from Saginaw Bay was greater in 1985 than in 1982. Similarly, the degree of retinoid depletion in gulls from Green Bay in Lake Michigan was greater in 1985 than in 1983. A retrospective analysis suggests that the degree of vitamin A depletion in Lake Ontario herring gulls in the mid-1970s was greater than in 1985. This is not surprising because the food web was more contaminated in the mid 1970s. Vitamin A stores increased between 1975 and 1982-83 and were lower in more recent collections.

The most severe retinoid depletion was observed in the collections from the western basin of Lake Erie. These were the same collections that had the most severe thyroid pathology (Moccia *et al.*, 1986). This correspondence is not unexpected because vitamin A deficiency causes biochemical hyperthyroidism in rats (Morley *et al.*, 1978) and recent laboratory studies have implicated abnormalities in the homeostasis of the retinol-thyroxin carrier protein complex in mammalian toxicity of coplanar PCB isomers (Brouwer and Van den Berg, 1986; Brouwer *et al.*, 1986).

Vitamin A deficiency in wildlife has been associated with decreased hatchability and chick survival, birth defects, reduced growth, visual and nervous disorders and bone abnormalities (Robbins, 1983). Brouwer *et al.* (1989) reported that common seals fed PCB-contaminated fish from the Wadden Sea had significantly lower plasma concentrations of retinol and thyroxine than seals fed fish from the north-east Atlantic that were low in PCBs. These authors suggest that PCB-induced reductions in plasma retinol and thyroxine may have resulted in increased susceptibility to infectious disease, reproductive disorders and other lesions in seal populations in the Wadden Sea and may be involved in the reproductive disorders (Reijnders, 1986) and lethal viral infections recently reported in seals in the Wadden, North and Baltic Seas (Dietz *et al.*, 1989). It is therefore possible that some of the pathophysiological and reproductive problems observed in various species of fish-eating birds at various locations in the Great Lakes in the last decade (sections 2.2, 2.3.5,, 3.3, 3.6, 3.8) have resulted from contaminant-induced alterations in vitamin A homeostasis in birds. Spear *et al.* (1990) present data suggesting that the relative amounts of retinol and retinyl palmitate in the yolks of Great Lakes herring gull eggs varies with location of the colony and is correlated with several indices of PHAH contamination. Takase and Goda (1990) suggest that retinol, not retinyl palmitate, is concentrated in the lungs of embryonic chicks late in incubation, and that this may be functionally connected to cellular differentiation and maturation within the lungs. Contaminant-induced lowered retinol availability could therefore decrease the degree of differentiation and maturation of the embryo's lungs at a time that it begins to breathe, thus compromising its chances of hatching or surviving. The fact that the degree of depletion in some locations has increased in recent years is reason for concern and suggests that further investigations and monitoring, at the biochemical, individual and population level, are warranted.

2.3.5 Changes in Thyroid Function

The thyroid is an initiator, integrator and modulator of various physiological processes in vertebrates, particularly those involving metabolism, development, differentiation and growth. The thyroid's physiological role and sensitivity to environmental factors make it a potential indicator of ecosystem health, as has been suggested by Moccia *et al.* (1981) for goitered Pacific salmon in the Great Lakes.

Blood and thyroids were collected from adult herring gulls nesting in the Great Lakes between 1980 and 1983. Material was also collected from a marine colony (Kent Island in the Bay of Fundy), from 1977 to 1982. Thyroids from gulls collected on Scotch Bonnet Island in eastern Lake Ontario in 1974 have been examined (Moccia *et al.*, 1986). Herring gulls throughout the Great Lakes suffer from thyroid enlargement (goiter) and varying degrees of epithelial hyperplasia (Moccia *et al.*, 1986) (Figure 5). The thyroids of gulls collected for western Lake Erie were the most enlarged, but those from the relatively clean northern shore of Lake Superior were the next highest while the least degree of enlargement was found in birds from Saginaw Bay. Epithelial hyperplasia was not reported in the thyroids from gulls from the Atlantic coast but it was observed in 11 of 12 collections from the Great Lakes. It was most prevalent in gulls from Saginaw Bay, Green Bay, Lake Ontario and western Lake Erie. It was most severe in the 1974 collection from Scotch Bonnet Island. Goiter occurs whether or not the free iodide content of the plasma is similar to that of gulls from the Atlantic coast, suggesting that the endemic goiter in Great Lakes gulls is the result of exposure to goitrogenic substances, rather than iodine deficiency (Fox and Moccia, in preparation). The thyroxine levels in plasma from gulls from some of the Great Lakes locations were lower than the median of any of the Atlantic coast collections, whereas the levels in gulls from other locations exceeded the highest of the Atlantic coast medians.

In Lake Ontario, the severity of goiter has decreased in more recent collections. It is highly unlikely that this reflects a corresponding increase in the availability of iodine, but it is consistent with the trends in environmental contaminant burdens in these birds. In recent years, similar trends in goiter prevalence have been observed in Pacific salmon stocks in the Great Lakes (J. Leatherland, personal communication). Sonstegard and Leatherland (1976) and Moccia *et al.* (1977, 1978, 1981) offer several arguments supporting the hypothesis that factors other than, or in addition to, iodine deficiency are responsible for goiter in Pacific salmon stocks in the Great Lakes. Several widespread environmental contaminants including DDT, dieldrin, PCBs, PBBs, TCDD, mirex, octachlorostyrene and chlorinated benzenes alter thyroid function in laboratory rodents. Experimental studies in both rats and birds suggest that the mode of action of PCBs and dioxins is different from that of DDT and dieldrin. PCBs and TCDD apparently displace thyroxine from the retinol-thyroxine carrier protein complex in plasma, and irreversibly bind to the thyroxine receptors of tissues (McKinney *et al.*, 1985). This results in very low levels of circulating thyroxine, which stimulates chronic secretion of thyroid stimulating hormone by the pituitary and leads to thyroid hyperplasia. At the same time, the full-time occupancy of the tissue receptors results in a variety of metabolic abnormalities. Some of these are normally associated with hypothyroidism and others with hyperthyroidism. Exposure to DDT and dieldrin may result in hyper- or hypothyroidism, depending on the dose. It is therefore inappropriate to interpret the thyroid-related abnormalities in wildlife exposed to environmental mixtures in these terms, and simplistic to expect a simple relationship. Several bird species developed goiter, experienced changes in thyroid histology and in many cases, changes in the metabolic rate when they were fed diets containing DDT, DDE, dieldrin and PCBs. The degree of thyroid enlargement and the prevalence of epithelial hyperplasia in Great Lakes herring gulls is significantly correlated with liver PCB levels (Fox and Moccia, in preparation). Recent laboratory studies of the effects of coplanar PCB isomers and TCDD on rodents suggest that there are changes in the homeostasis of the retinol-thyroxine carrier protein complex (section 2.3.4). The herring gull collections with the most severe thyroid pathology also had the most severe retinoid depletion. Moccia *et al.* (1981) reported that salmon from Lake Erie had the most severe pathology and highest goiter frequency of all Great Lakes collections. This interspecies correspondence is consistent with the hypothesis that a forage fish-borne factor is responsible for goiter induction in both gulls and salmon.

Herring gulls nesting in eastern Lake Ontario in 1976 and Forster's terns nesting in Green Bay in 1983 demonstrated low incubation attentiveness and other indications suggestive of thyroid-mediated toxicity (Fox *et al.*, 1978; Kubiak *et al.*, 1989). Incubation attentiveness was altered in ring doves fed diets containing PCBs (Peakall and Peakall, 1973) and a PCB - organochlorine mixture (McArthur *et al.*, 1983). The alteration in thyroxine levels in doves in the latter study was dose dependent. The metabolic rate of PCB-fed mourning doves was decreased and this was reflected in an inability to thermoregulate at low temperatures (Tori and Meyer, 1981).

Hypothyroidism has been found in rats fed diets containing coho salmon from the Great Lakes (Sonstegard and Leatherland, 1979). The authors found that serum thyroxine levels were markedly reduced in rats fed salmon from Lakes Ontario, Michigan or Erie for two months, and thyroids of those fed salmon from Lakes Ontario and Michigan were enlarged. Villeneuve *et al.* (1981) also noted mild, but statistically significant histological changes in the thyroids of rats fed salmon from Lake Ontario. Another experiment noted marked effect on the weight gain of young rats fed salmon from the Great Lakes (Sonstegard and Leatherland, 1978). The mean body weights of rats fed salmon from Lakes Michigan and Erie were approximately 55% less than controls which had been fed Pacific salmon, while the weight gain of rats fed salmon from Lake Ontario was not affected. Villeneuve *et al.* (1981) also noted effects on weight gain, but attributed their findings to the nutritional characteristics of the diet. Clearly, goiterogenic substances are present in Great Lakes fish.

2.3.6 Changes in Genetic Material

Chemicals capable of altering the integrity of DNA are present in the Great Lakes environment. Exposure of somatic cells to such genotoxic agents may cause cell death, birth defects or neoplasia. Exposure of germ cells may cause mutations which can be transmitted to subsequent generations. Several tests are available to assess chemically-induced damage to genetic material (Kohn, 1983). Three measures have been applied to the tissues of fish-eating birds from the Great Lakes. These are the frequency of sister chromatid exchange, the amount of DNA strand breakage and the degree of DNA methylation. However, these studies were initiated after the peak of chemical contamination in the mid 1970s and after most health and biological effects were observed. It is therefore impossible to assess the role of genotoxic chemicals in the pathophysiological lesions observed in Lake Ontario in the early 1970s.

Sister chromatid exchange (SCE) is the reciprocal interchange of DNA between chromatids and can be easily visualized and detected in metaphase chromosomes. SCE occurs following DNA damage and it is more frequently observed than chromosome aberrations or strand breakage. The rate of SCE was measured in 7-day herring gull embryos from eggs collected in 1981 from five colonies in the Great Lakes basin and one Atlantic coast colony (Ellenton and McPherson, 1983). There were no significant differences between the colonies. Extracts of herring gull eggs from each of these colonies were also tested in the Ames test for induction of point mutations and in Chinese hamster ovary cells for the induction of chromosome aberrations and SCEs (Ellenton *et al.*, 1983). The extracts were not mutagenic in the Ames test, but all gave positive results when tested for chromosome aberrations and SCEs in cultured mammalian cells. The extract from the Lake Ontario colony produced a significantly higher frequency of chromosome aberrations at one quarter the dose required to do so for any other extract but did not exhibit a strict dose-response. Since the extract from the Atlantic coast colony exhibited similar genotoxicity to those from more contaminated colonies, it was concluded that at least some of the observed effects could be attributed to biological lipophilic compounds, such as carotenes.

The formation of a reactive metabolite-DNA adduct is often the significant event in the toxicity of chemicals and frequently results in DNA strand breakage. The quantitation of these strand breaks is a useful measure of DNA damage. The alkaline unwinding assay measures the rate at which the DNA double helix unwinds under alkaline conditions. The rate of unwinding is dependent on the number of breaks present in the strands (Shugart, 1988). The numbers of DNA strand breaks in liver samples from young double-crested cormorants and herring gulls collected in 1987 from Lake Ontario and the Atlantic coast were similar (Shugart and Peakall, unpublished data).

Some chemical carcinogens have been shown to alter the normal inheritable patterns of DNA methylation. Hypomethylation of DNA can be a biological response to genotoxic agents and it may be a useful biological marker of exposure because it is easily measured (Shugart, 1990). The DNA from livers of young herring gulls collected from Lake Ontario in 1987 contained a significantly lower percentage of 5-methyl deoxycytidine than that in livers from gulls collected from the Atlantic coast. These differences were not seen in young cormorants from the two sites (Shugart and Peakall, unpublished data). Studies on the integrity of liver DNA of cormorants collected from Green Bay are currently under way (section 3.3).

3. SPECIES ACCOUNTS

3.1 MINK

Mink occupy a wide variety of wetland habitats, including streams, rivers, lakes, freshwater and saltwater marshes and coastlines throughout most of Canada and the U.S. In freshwater habitats, their numbers reflect the abundance of permanent wetland habitat that have ample shorelines and emergent vegetation. Such habitats are also important for muskrats, whose bank burrows provide denning sites for mink, and upon which mink prey.

Mink are generalists and prey on locally available food sources. Mammals are clearly the most important class of prey throughout the year, regardless of habitat. Muskrats, rabbits and small rodents are commonly taken. The importance of fish, birds (particularly waterfowl and marsh-nesting song birds) and invertebrates (particularly crayfish) changes seasonally (Eagle and Whitman, 1987).

In 1965, Hartsough suggested that diets containing fish from Lake Michigan were causing reproductive failure in ranch mink. It was later shown that this could be attributed to PCBs, rather than rancidity, mercury or chlorinated hydrocarbon insecticides (Aulerich and Ringer, 1977). Aulerich, Ringer and co-workers have conducted numerous toxicological studies which have shown the mink to be one of the most sensitive, if not the most sensitive, mammalian species to PCBs, PBBs, HCB and TCDD. Reproductive failure in ranch mink has been associated with dietary levels of PCBs from 0.3 to 5 ppm and mortality in adults has been caused by 3.6 to 20 ppm. The PCB concentrations in mink livers ranged from 0.8 to 2 ppm (wet weight basis) in mink experiencing reproductive problems and averaged 4.2 ppm in those that died. Since forage fish in the Great Lakes are contaminated with these chemicals, mink that live in shoreline habitats may be exposed to toxicologically significant amounts of PCBs.

If PCBs are present in the food web of wild mink and have affected their survival and/or reproduction, one would expect to see changes in mink populations. The only available measure of the relative abundance of mink are the harvest statistics. However, these are confounded by several variables including fur prices, weather and trapping effort. Overall, the harvest of mink in Ontario has decreased between 1920 and 1988. The current harvest is 25-30,000 per year (Wren, in prep.). The number caught per trapper has also decreased, suggesting that the population has decreased. According to statistics tabulated by Novak *et al.* (1987), over 2 million wild mink were harvested in Ontario between 1944 and 1983. The harvest began to decline in the mid 1950s and was at its lowest in the early 1970s (Figure 6). In the 1980s it recovered somewhat. This temporal trend is similar to that seen in populations of several fish-eating and predatory birds. Muskrat numbers reflect the availability and quality of wetland habitat, the relative availability of denning sites and prey for mink. In unperturbed conditions, their numbers should be closely correlated with mink numbers. Although the muskrat harvest was also depressed during this period, the relative decline was less severe (Figure 6). A similar trend was seen in the Wisconsin and Michigan mink harvests (although data for the critical period 1968-78 are missing for the latter). This trend was not obvious in the Ohio harvest.

A study of Ontario mink and muskrat harvests from 1970/71 to 1984/85 has been recently completed (Jones, unpublished). This compared the harvests in four areas. Two areas had a high risk of exposure to PCBs and two had a low risk of exposure. Area 1, a high risk area, included townships bordering the north shores of Lakes Erie and Ontario. Area 3, the other high risk area, included townships in the Cornwall district along the north shore of the St. Lawrence River. Area 2, a low risk area, consisted of townships removed from the north shore of Lakes Erie and Ontario. Area 4, the other low risk area, included townships within the Minden and Bancroft districts, in central Ontario. Areas 1 and 2 are of comparable size, as are areas 3 and 4.

Records for the number of mink and muskrat harvested by each trapper each year were used to prepare summary harvest data. The total number of mink harvested in area 1 from 1970/71 to 1984/85 was significantly lower ($P<0.02$) than in area 2. The difference does not appear to be due to intensity of trapping, since the average number of mink caught per trapper was also significantly lower ($P<0.01$). The total number of muskrat harvested from area 1 was also significantly lower ($P<0.01$) than from area 2, but the number of muskrat caught per trapper was lower in only seven out of 15 years. Both the total number of mink harvested and the average per trapper were significantly lower (P<0.01) in area 3 than in area 4. On an annual basis, the individual harvests of mink were significantly lower in area 3 for 11 of the 15 years. The total number of muskrat harvested and the average number per trapper did not vary significantly. On an annual basis, where significant differences occurred, the muskrat harvest was higher in area 3 than area 4. When combined, the two high PCB risk areas had a significantly lower mink harvest than the two low risk areas combined, in 12 out of 15 years. Individual harvests of muskrats were only significantly different in seven out of 15 years and in three of these it was the high risk areas that had the higher muskrat harvest. Thus, in the areas where the risk of PCB exposure was high, significantly fewer mink were harvested than in areas where the risk of PCB exposure was low. In contrast, these areas did not necessarily have lower muskrat harvests.

In Ohio, mink harvests were generally low between the late 1940s and 1970. The mink harvest in Ohio was monitored for each county from 1981/82 to 1986/87. There is a dramatic difference in the total harvest of mink from the eight counties bordering Lake Erie, compared to that in the adjacent counties remote from Lake Erie. The harvest was substantially lower in the counties bordering Lake Erie for each of the years. The total harvest for the six-year period was 2353 mink from counties bordering Lake Erie and 5089 from the counties remote from the lake. In 1985/86, the mink harvest was lower in the county adjacent to Lake Erie in seven of eight comparisons between the harvest for counties adjacent to Lake Erie and the next bordering county with no shoreline on Lake Erie (Wren, in prep.). Bednarik (1956) reported that marsh owners and trappers destroy mink throughout the year as they are regarded as the primary predators of muskrat. If this still occurs it may also reduce mink harvests in the Lake Erie marsh region.

There is insufficient information on residue burdens of wild mink from the Great Lakes basin. The available data differ in the tissue sampled and the basis of measurement, making it difficult to compare the results. PCB levels in the muscle tissue of 20 mink trapped in Simcoe County (on the southern shore of Georgian Bay) in 1972-1974 averaged 32 ppm on a lipid weight basis (Frank *et al.*, 1979). The liver of a single mink found dead in

1983 on Green Bay, Lake Michigan, contained 5.7 ppm PCBs on a wet weight basis (U.S. Fish and Wildlife Service, unpublished data). The PCB content of carcasses of mink collected from four townships on the Ontario shoreline of Lake Erie during the winter of 1978-79 ranged from 0.11 to 7.37 ppm on a wet weight basis, and 1.4 to 102 ppm on a lipid weight basis (Proulx *et al.*, 1987). The township averages were 29.2, 26.1, 10.7 and 6.1 ppm (lipid weight basis), respectively. Livers of ten mink collected from the north shore of the upper St. Lawrence River during the winter of 1976-79 contained 1 ppm on a wet weight basis and 57 ppm on a lipid weight basis (Canadian Wildlife Service, unpublished data) and livers of five mink from the Whitby--Cobourg area on the northern shore of Lake Ontario contained 21 ppm on a lipid weight basis. Livers of mink trapped in Oswego County on the New York shore of Lake Ontario between 1982 and 1984 contained 0.3 ppm on a wet weight basis, and adipose tissue contained 7 ppm on a lipid weight basis (Foley *et al.*, 1988). In one experimental study, adipose tissue levels of 10-43 ppm PCBs (lipid weight basis) were associated with significant decreases in reproduction and 100% kit mortality at three weeks of age (Hornshaw *et al.*, 1983). Liver and muscle levels above 1 ppm (wet weight basis) have been associated with reduced reproduction in other studies. In a recent study, the number of kits whelped and the body mass of kits at 3 and 6 weeks were reduced when 10% contaminated carp from Saginaw Bay was incorporated into their diet (Heaton *et al.*, 1989). This was equivalent to 0.19 mg PCBs/kg body weight per day or 4.50 ng TCDD-EQ/kg body weight/day. Thus, burdens of PCBs in most mink collected from Great Lakes shorelines are sufficient to induce adverse effects on reproduction and are consistent with the apparent population changes described above. PCB levels in mink from New York state were significantly correlated with concentrations in fish on a restricted geographic scale (Foley *et al.*, 1988). In New York, this relationship may cause suppression or elimination of the population in areas where concentrations of PCBs in fish are highest. Elimination may be occurring in shoreline habitats around the eastern end of Lake Ontario and along portions of the Hudson River. However, the harvest data available are not adequate to confirm this hypothesis.

3.2 OTTER

The river otter inhabits aquatic ecosystems throughout North America. As its broad geographic distribution would suggest, the river otter is able to adapt to diverse aquatic habitats. Otter habitat consists of riparian vegetation adjacent to lakes, streams and other wetland areas. Beaver create foraging and denning sites for otter.

Unlike the mink, the river otter is a specialist, feeding almost entirely on aquatic prey. Although it varies seasonally, the river otter's diet is composed primarily of fish. Crustaceans, reptiles, amphibians, birds, insects, and mammals are of lesser importance (Melquist and Dronkert, 1987). Thus, if wild mink populations have been affected by environmental contaminants, and assuming that the closely related otter has similar sensitivity, it is likely that river otters have also been affected.

River otter have few natural enemies and human activities are a major source of mortality. Overharvest in the early 19th century resulted in the near destruction of most furbearing populations. During the first half of the 20th century, river otter and beaver populations were widely protected (Melquist and Dronkert, 1987). Populations of these two species probably recovered more slowly than mink and muskrat because of their low reproductive potential. This is reflected in the harvest statistics (Novak *et al.*, 1987). The relative abundance of both river otter and beaver increased continuously from 1944 to 1983 in parallel, in Ontario, Michigan, Wisconsin and New York (Figure 7). There is no commercial harvest of river otter in Ohio. Unlike the mink, it is not possible to detect any decrease in otter numbers that would correspond with the peak contamination of the Great Lakes food web in the early 1970s. However, the rate of population growth may have been suppressed relative to areas outside the Great Lakes basin. There are areas in Minnesota, Wisconsin and Michigan where the harvest data suggest that very few or no otter have been trapped in recent years. There have been no otter harvests along the entire Lake Michigan shorelines of Wisconsin and Michigan, including Green Bay, the Saginaw Bay region of Lake Huron, and much of the Lake Superior shoreline of Michigan and Minnesota. In general, inland tributaries which support healthy otter populations become relatively devoid of otter as they approach Lake Michigan.

There have been no controlled studies of the effects of PCBs on otter.

It is therefore not possible to determine the levels of chemicals in food tissues that are associated with adverse effects. However, a study of the toxicity of methyl mercury in captive otter (O'Connor and Nielson, 1980) suggests that ranch mink and river otter have similar sensitivities to this chemical. Although mercury contamination is probably not a problem for otter and mink in the Great Lakes basin, dead, mercury poisoned wild mink and otter have been found near heavily contaminated rivers (Wobeser and Smith, 1976; Wren, 1985). This confirms that these species can be killed by environmental contaminants in the aquatic food web. In four studies comparing mercury burdens in mink and otter caught in the same regions, the burdens in otter generally exceeded those in mink, although there was some overlap (O'Connor and Nielsen, 1980; Sheffy and St. Amant, 1982; Kucera, 1983; Wren *et al.*, 1986), reflecting the larger proportion of aquatic prey in their diet. In the two studies where PCB burdens of mink and otter caught in the same regions were compared, the burdens in otters exceeded those of mink (Henny *et al.*, 1981; Foley *et al.*, 1988). PCB levels in tissues of otters captured in New York were associated with the concentrations in fish on a restricted geographic scale, but not on a major watershed basis.

In his recent review on water pollution and otter distribution in Britain and Europe, Mason (1989) hypothesised that PCBs were associated with the decrease of European otter populations. Four endangered or decreasing populations had mean tissue levels above 2 ppm, whereas five stable or thriving populations had levels less than 2 ppm. The lowest levels were from thriving populations. Mason regards 2 ppm as the tissue concentration associated with reproductive failure in PCB-dosed mink. Mason concludes that "the evidence from these studies strongly implicates the role of PCBs in the decline of otter populations". In a declining population in East Anglia, a road-killed otter cub, born in the wild to a mother released as part of a restocking program, had accumulated over 2 ppm in its liver by 11 weeks of age (Mason, 1989). Two adults found dead of unknown causes had residues of 10 and 2 ppm PCBs. One of these otters suffered from a leiomyoma of the reproductive tract and adrenocortical hyperplasia. Olsson *et al.* (1981) concluded that rapid decreases in Swedish otter populations were probably due to PCBs. Henny *et al.* (1981) found that PCB levels in livers of all otters from the Lower Columbia River in Oregon exceeded 2 ppm, and the mean for males was 9 ppm. They suggested that this high level of PCB contamination may explain the decreased harvest in this area. Foley *et al.* (1988) were unable to obtain otter from the Lake Ontario shoreline of New York.

The presence of thriving populations of otter and mink could serve as a biological indicator of the "virtual elimination" of PCBs from shoreline wetland habitats in the Great Lakes basin. Efforts should be made to regulate and accurately monitor harvests of these species to enable their use in biological monitoring programs.

3.3 DOUBLE-CRESTED CORMORANT

Breeding populations

This species was not known to nest in Ontario until the early 1920s. It became established first on Lake Superior. Baillie (1947) stated that "at present time there are additional nesting groups in Lake Huron, Georgian Bay, Lake Erie, Lake Ontario and the upper St. Lawrence River and there is abundant evidence that the bird is increasing as well as expanding its breeding range." The available information on the population changes is shown in Table 5. The best estimate of the total Great Lakes population in the 1940s and 1950s is somewhat less than a thousand pairs. By the early 1970s, the population had crashed on all of the lakes, except Lake Erie. By 1979, the population had begun to recover and since then there has been rapid expansion on all lakes. Currently the breeding population is approaching 20,000 pairs.

The rapid changes in the breeding population on Lake Ontario are shown in Figure 8. In the late 1940s and 1950s, the population was about 200 pairs, falling to only three pairs by 1973. Between 1979 and 1990, the Lake Ontario cormorant population increased from 315 pairs to 6700 pairs. During this period, they expanded from the eastern end of the lake through the central portion and into the western end. There are now seven active colonies on Lake Ontario.

There are many gaps in the data for Lake Erie, but since 1979 the cormorant population has increased dramatically, from 87 pairs to 1800 pairs in 1990. The population is centred on the Canadian side of the western basin and all three active colonies are located there. This probably reflects the distribution of undeveloped islands in the lake.

Data on the population changes in Lake Huron proper, Georgian Bay and the North Channel are shown in Table 5. Georgian Bay and the North Channel are both long established cormorant nesting areas dating back to before the 1940s. Lake Huron proper has only become a nesting area since the mid 1980s. Populations of cormorants nesting in Georgian Bay and the North Channel decrease by 86% and 78% respectively between the 1950s and 1972. Since 1979, both populations have increased about 40-fold. In Lake Huron proper, the increase in population has also been dramatic, increasing from 21 pairs in 1984 to over 1500 in 1990. The number of active colonies has increased from seven to at least 24.

TABLE 5. LAKE-WIDE POPULATION LEVELS OF DOUBLE-CRESTED CORMORANTS ON THE GREAT LAKES, 1940-1990

Year	Lake Ontario	Lake Erie	Lake Huron	Georgian Bay	North Channel	Lake Michigan	Lake Superior	Total
1940s-1950s*	220	22	3	145	255	100	120	873
1971	19 (2)	43 (2)	?	28 (3)	41 (2)	?	?	131 (9)
1972	20 (2)	50 (2)	0	21 (3)	55 (4)	0	0	148 (11)
1973	3 (2)	39 (1)	?	18 (2)	40 (4)	?	?	100 (9)
1979**	315 (2)	87 (1)	3 (1)	97 (3)	103 (4)	68	71 (3)	676 (14)
1980	375 (3)	114 (1)	0	176 (4)	159(5)	163	123 (3)	947 (16)
1981	641 (3)	148 (3)	-	193 (4)	199 (5)	311	191 (7)	1372 (22)
1982	770 (2)	212 (3)	-	318 (4)	253 (5)	-	223 (5)	1776 (19)
1983	1173 (4)	242 (4)	-	445 (4)	309 (5)	-	211 (4)	2380 (21)
1984	1172 (4)	230 (3)	21 (1)	558 (4)	710 (10)	-	368 (6)	3059 (28)
1985	2162 (7)	217 (3)	200 (3)	679 (5)	1011 (9)	-	536 (7)	4805 (34)
1986	2190 (7)	354 (3)	257 (3)	721 (5)	1226 (9)	1038	682 (7)	5430 (34)
1987	3471 (7)	631 (3)	557 (5)	721 (5)	2033 (9)	1676	717 (7)	8283 (36)
1987***	3471 (7)	631 (3)	976 (6)	877 (6)	2033 (12)	1646 (8)	1075 (8)	10,709 (50)
1988	3938 (7)	1125 (3)	882 (5)	1082 (7)	2619 (11)		847 (9)	10,493 (42)
1989	5961 (8)	1365 (3)	1011 (5)	3407 (24)	3159 (12)		843 (13)	15,747 (65)
1990	6704 (8)	1807 (3)	1545 (5)	4190 (23)	4179 (18)		1422 (14)	19,847 (65)

* Data for 1940s-1950s through 1973 are from Postupalsky 1978 and are for all five of the Great Lakes.

** Data since 1979 are for all of Lakes Ontario and Erie and Canadian portions only of Lakes Huron and Superior; from CWS (unpubl.)

*** Data for 1987 for all of Great Lakes except Wisconsin portion of Lakes Superior and Michigan; data from CWS (unpubl.) and Kurita et al. 1987.

Note:

Figures refer to the number of nests censused on each lake (and the number of colonies examined).

There is a similar pattern in Lakes Michigan and Superior. There was a dramatic decrease with the species failing to breed in Lake Michigan by the early 1960s, and in Lake Superior by 1972. More recently, there has been an equally dramatic increase with populations increasing twenty fold from 1979. In recent years, only the small colony in Saginaw Bay (Charity Is.) has failed to produce any fledglings. Between 1985 and 1988, nests have been built, but have been abandoned.

Productivity

The available productivity data are summarized in Table 6. Although data are available for most lakes and for most years, the extent of the coverage and the colonies visited has varied considerably. No detailed studies were conducted on the productivity of the cormorant on the Great Lakes before the 1970s, but it is likely that the productivity of the expanding population in the 1940s and 50s was within the normal range for this species, viz 1-2 young/nest/year.

TABLE 6. PRODUCTIVITY OF THE DOUBLE-CRESTED CORMORANT ON LAKE ONTARIO

Year	Productivity (young/pair)	No. of Nests Assessed	Colonies Included*
1940s	1 - 2	Productivity assumed "normal"	
1971	0.0	19	S, PI
1972	0.0	10	S, Pe
1973	0.0	3	S, Pe
1978	0.3	6	S
1979	1.3	37	PI
1980	1.7	97	PI
1981	1.5	168	PI
1982	1.5	175	PI
1983			
1984	1.5	410	PI
1985	1.6	502	PI
1986	2.3	785	LG
1987			
1988	1.8	1,592	LG

* S = Scotch Bonnet Island, PI = Pigeon Island, Pe = Peter Rock, LG = Little Galloo Island.
Source: Data prior to 1982 are from Postupalsky (1978) and/or Price and Weseloh (1986); data after 1982 are from Weseloh (unpublished).

There was no known successful breeding of cormorants in Lake Ontario between 1954 and 1977 (Price and Weseloh, 1986). It is possible that some young were produced in the two small colonies on the U.S. side, although both had been abandoned by the end of the period. In 1978, productivity was low on Scotch Bonnet Island but it has been normal on Pigeon Island since 1979.

Detailed investigations of reproductive problems of this species were carried out on the Georgian Bay population in 1972 and 1973 (Weseloh *et al.*, 1983). There was almost total reproductive failure caused by the breakage and disappearance of eggs. In 1972, 79% of the eggs were lost (broken or disappeared) from nests within the first eight days after laying. This figure increased to 95% by the end of the incubation period. The productivity data for both the North Channel and Georgian Bay suggest a small but definite increase in 1973 which continued subsequently. The increase in productivity in 1973 appears to be the first biological indication of decreased chemical contamination of the Great Lakes. However, residue monitoring programs were not in place in the early 1970s. For example, the Canadian Wildlife Services herring gull egg program started in 1974 and the Ontario Ministry of the Environment juvenile fish contaminants surveillance program in 1975 (see Volume I, Part 2). Therefore, peak concentrations of organochlorines probably went unrecorded in biota.

Egg breakage occurred because of eggshell thinning. Cormorants are particularly sensitive to eggshell thinning because they incubate their eggs by standing on them. The phenomenon of eggshell thinning has been extensively studied in many species (Risebrough, 1986). In several species, eggshell thinning between 15 and 20% is enough to cause population declines. DDE contamination is known to cause eggshell thinning.

There can be little doubt that the decline and subsequent initial recovery of the cormorant in the Great Lakes were directly related to reduced contaminant levels. Nevertheless, it is unlikely that decreases in contaminant levels alone are responsible for the unprecedented increases in population. By 1990, there were more than twenty times the number of cormorants nesting throughout the Great Lakes than at any time in recorded history. The magnitude of these population changes probably reflects major changes in the forage fish population of the Great Lakes. The current rate of increase of about 30% per year would bring the cormorant population to over a third of a million by 2000, if it were sustained.

Congenital anomalies

The double-crested cormorant is the only species for which adequate data are available to statistically analyse congenital anomalies (Fox *et al.*, submitted). Between 1979 and 1987, a few observers provided relatively consistent coverage from year to year. Observations of birth defects and the number of chicks banded by location and year were used to calculate rates of incidence and prevalence. The prevalence in hatched chicks was calculated for each region as the sum of all chicks with bill defects found in all visits within a region divided by the sum of all chicks examined in all visits within that region.

Although anomalies, such as club feet, supernumerary digits, eye and skeletal abnormalities were observed, only hatched chicks with bill defects (crossed or deflected bills and bills in which the two mandibles differed in length) were considered. Bill malformations are clearly developmental and are not the result of post-hatching trauma.

Between 1979 and 1987, 31,168 cormorant chicks were examined during 147 visits to 42 colonies in the Great Lakes (Table 7). Seventy chicks with bill defects were reported. During the same period, 20,962 chicks were examined during 82 visits to 35 colonies in the two reference areas. The first was Lake Nipigon and Lake-of-the-Woods, and the second was Alberta and Saskatchewan. Bill defects were observed in only two (6%) of the 35 reference colonies. This proportion was exceeded in six of eight geographic regions in the Great Lakes. It was the highest (73%) in birds from Green Bay (Table 2), where bill defects were observed in eight of 11 colonies. Thirty-two (53%) of the 60 malformations observed in birds from Green Bay occurred on Spider Island, the largest colony in the region. The prevalence of malformed chicks in Green Bay (52.1 per 10,000) was markedly greater than all other regions during this time period (Table 2), and was 22 to 87 times that of the reference areas. The prevalence in Green Bay was significantly higher than in all other regions, other than Lake Ontario.

There is ample evidence that the food web in Green Bay is severely contaminated with a variety of toxic organic contaminants (Sullivan *et al.*, 1983; Heinz *et al.*, 1985; Fox *et al.*, 1988; Hudson 1987; and Kubiak *et al.*, 1989). PCBs may be responsible for the observed malformations. All three of the more toxic non-ortho PCB congeners have been isolated from tissues of cormorant chicks with crossed bills collected from Green Bay (Stalling *et al.*, 1985). Two of these congeners (Marks *et al.*, 1981; Wardell *et al.*, 1982), like TCDD (Pratt *et al.*, 1984), are known to cause abnormalities in craniofacial development in laboratory mammals. Kubiak *et al.* (1989) make a strong case for the involvement of PCB congeners in the reproductive problems observed in Forster's terns from Green Bay.

TABLE 7. OCCURRENCE OF BILL DEFECTS IN THE YOUNG OF DOUBLE-CRESTED CORMORANTS BETWEEN 1979 AND 1987

Geographical Region	# of colonies	# of chicks with defects	# of chicks examined	Frequency of defects (per 10,000 chicks examined)
Lake Superior	5	1	1,825	5.5
Green Bay Lake Michigan	11	60	11,520	52.1
Beaver Islands Sts. of Macinac	4	4	3,528	12.3
Alpena Lake Huron	2	0	363	-
Georgian Bay Lake Huron	5	2	3,250	6.1
North Channel Lakes Huron & Superior	12	2	7,342	2.7
Lake Erie	1	0	728	-
Lake Ontario	1	1	2,882	3.5
Lake Nipigon & Lake of the Woods	17	1	4,184	2.4
Alberta & Saskatchewan	18	1	16,778	0.6

Note: Colonies in Lake Nipigon, Lake of the Woods and Alberta and Saskatchewan serve as reference areas to the Great Lakes colonies.
Source: After Fox, *et al.* (submitted)

3.4 BLACK-CROWNED NIGHT-HERON

This heron is widely distributed in both Europe and the Americas. However, its Canadian range is quite limited. In Ontario, most of the heronries are located near the shores of Lakes Erie, Ontario and Huron. It is most common in western Lake Erie, although between 1930 and 1970, this species expanded its range to the north and east. However, population trends are difficult to follow because of its tendency to move nesting sites. In the late 1960s and early 1970s, several populations decreased. This was coincident with DDE-induced eggshell thinning. The number of pairs nesting along the Toronto waterfront declined over this period but increased steadily during the 1980s to over 600 pairs in 1988 (Burgess, unpublished).

Between 1961 and 1963, the colony on Pigeon Island in eastern Lake Ontario consisted of approximately 125 pairs. However, after 1964 it decreased to a low of 17 in 1973, but recovered to 71 pairs by 1977. Members of the Kingston Field Naturalists visited Pigeon Island every year between 1961 and 1973. Their observations were reported in the club's publication and summarized by Edwards (1970). These records provide a rough reproductive history of the night-heron colony. Between 1966 and 1968, fewer nests contained young and in 1969, the 27 pairs remaining experienced complete reproductive failure. In 1972, the 23 pairs produced only 5 young (0.22 young/pair). More detailed studies by the Canadian Wildlife Service between 1972 and 1976 confirmed that the reproductive success of this colony was poor. Although more eggs were laid per pair than average for this species, the number of young fledged per active pair ranged from 0.5 to 1.4 (Price, unpublished data). This is considerably lower than the 2 young per pair necessary to maintain a stable population. The poor reproductive success was a result of loss of eggs from the nest (many were found punctured on the ground), failure of the eggs to hatch, and reduced survival of young. Of 72 young which survived to banding in 1972 and 1973, one had a severely crossed bill (Gilbertson *et al.*, 1976). The differences between the reproductive success of the Pigeon Island colony between 1972 and 1976 and the successful colony on the Toronto waterfront in 1988 are shown in Table 8.

The eggshells of night-herons from the Pigeon Island colony were

14-16% thinner from 1972 to 1976 than the thickness prior to the widespread use of DDT (Price, unpublished data) and fragments of shells of eggs which were thought to have hatched were 8-12% thicker than those of eggs which failed to hatch. Several North American studies have reported significant negative correlations between the DDE content of night-heron eggs and shell thickness. Henny *et al.* (1984) reported that night-heron productivity decreased and the incidence of cracked eggs increased when DDE levels exceeded 8 ppm. Custer *et al.* (1983) found that 10 ppm DDE resulted in a 50% decrease in hatching success. The yearly mean DDE content of night-heron eggs collected on Pigeon Island ranged from 4.5 ppm to 12.4 ppm in the mid 1970s (Price, unpublished data) and 4.8 ppm in 1982 (Weseloh *et al.* in preparation). This decrease in DDE content was accompanied by an increase in eggshell thickness. For example, eggs in the 1982 sample were only 6% thinner than the thickness prior to the widespread use of DDT.

TABLE 8. A TEMPORAL COMPARISON OF REPRODUCTIVE SUCCESS OF BLACK-CROWNED NIGHT-HERONS NESTING ON LAKE ONTARIO

	Pigeon Island			Toronto Waterfront
	1972	1973	1974	1988
Number of active pairs	20	17	65	33
Eggs laid per year	4.9	6.0	4.6	4.0
Eggs hatched per egg laid	0.36	0.39	0.56	0.78
Young raised per pair		(0.5 - 1.4)		2.0
Percent of eggs which				
disappeared	27	12	16	11
failed in nest	23	6	8	5
were found outside of nest	3	42	8	6
Percent of nests in which				
eggs hatched	60	76	>85	91
young fledged	ND	ND	ND	77

Source: I. Price, Canadian Wildlife Service, unpublished (Pigeon Island data)
N. Burgess, unpublished (Toronto Waterfront data)

The PCB content of eggs from the Pigeon Island colony was also very high (mean approximately 10-60 ppm, maximum 150 ppm). It decreased significantly between 1972 and 1976. By 1982, eggs from this colony contained an average of 24 ppm (range 9-44 ppm) PCBs. Although PCBs are not responsible for eggshell thinning, they are embryotoxic and are capable of altering parental behaviour. PCBs have been associated with impaired embryonic growth in night-herons in San Francisco Bay (Hoffman *et al.*, 1986) where the mean PCB content in the eggs was 4.1 ppm (range 0.8-52 ppm).

The shells of night-heron eggs collected in 1980 from lower Green Bay, Lake Michigan were 10% thinner than the pre 1944 mean, and their mean DDE content was 5.4 ppm (2.4 - 14 ppm). Their mean PCB content was 24 ppm (16-36 ppm) (Heinz *et al.*, 1985). This suggests that this subpopulation also experienced reproductive problems in the 1970s.

In general, the effects seen in the black-crowned night-heron in Lake Ontario were similar to those found in the double-crested cormorant, although the degree of egg breakage and the decrease in reproductive success was not as large. Egg puncturing has also been observed in the grey heron in Great Britain in the 1960s (Milstein *et al.*, 1970) and recently in the great blue heron in the Fraser River estuary in British Columbia. This may be a contaminant-induced behaviour characteristic of the Ardeadae.

3.5 BALD EAGLE

The bald eagle is almost entirely restricted to Canada, the U.S. and Mexico. The species was flourishing when it became the national emblem of the U.S. in 1792 and populations were relatively stable throughout the early decades of the 20th century. However, the bald eagle can now be found in near-pristine numbers only in Alaska, coastal British Columbia, boreal Canada and parts of the Maritime provinces. Since the introduction of persistent toxic contaminants into the Great Lakes food web, the Great Lakes subpopulation decreased dramatically. In areas where suitable habitat still exists and contaminant levels have decreased sufficiently, eagles have successfully re-established themselves.

Charles L. Broley, a Canadian banker and naturalist, first noted a drastic decrease in their populations during the 1950s. Within one year of the widespread introduction of DDT in 1946, Broley also noted nesting failures in the Florida subpopulation which were characterized by failure of the adults to return to claim a nest, failure of adults to nest, and failure of the eggs to hatch (Broley, 1952). In 1951, bald eagle nesting success near Delta (eastern Ontario) decreased significantly, with only one of 12 active nests under observation producing young (Broley, 1952). Broley (1958) documented a significant reduction in the number of breeding pairs and the reproductive rate of the remaining pairs in Florida. In response to increasing concern about this species, the National Audubon Society began to investigate its status and ecology over most of its range in 1960 and the U.S. Fish and Wildlife Service listed the southern subspecies as "endangered" in 1967. In their report on the outcome of 2036 nestings in six distinct populations over a 7-12 year period between 1961 and 1972, Sprunt *et al.* (1973) concluded that in any given bald eagle population, at least 50% of the breeding pairs must be successful in raising one or more young in any year, and that the population as a whole must produce at least 0.7 fledged young per active nest to maintain population stability. Of the six populations they studied, those nesting close to the shores of the Great Lakes in Michigan and Wisconsin had by far the lowest production. Only 16 of 156 nesting attempts were successful and, in any year, production ranged from 0.05 to 0.27 fledged young per active nest.

The bald eagle was probably the first species that was widely affected by the introduction of DDT. Two extensive studies have examined the statistical relationship of contaminant residues to eggshell thinning and reproduction. These studies used over 100 eagle eggs collected from more than 12 states (but not from nests on Great Lakes shorelines) in the 1970s (Wiemeyer *et al.*, 1984; Nisbet, 1989). The authors suggest that contaminants have played a major role in population declines, that DDE caused eggshell thinning and reproductive impairment, and that dieldrin (and perhaps DDT) resulted in excess adult mortality. PCBs may have contributed to reproductive problems but the evidence on this is unclear. In their studies of the closely related white-tailed sea eagle in Sweden, Helander *et al.* (1982) concluded that along the heavily contaminated Baltic coast, reproductive success was negatively correlated with egg residues of both DDE and PCBs.

According to the U.S. Fish and Wildlife Service, bald eagle numbers have recovered from a low of approximately 400 pairs nationwide in 1964 to 2,700 pairs in 1989, with productivities of 0.6 and approximately 1.0 young per occupied territory, respectively. At Besnard Lake in northern Saskatchewan, 73% of occupied breeding areas were successful between 1973 and 1981 and 1.17 young were produced per occupied breeding area (Gerrard *et al.* 1983). Using data on productivity and residues for a single population in northwestern Ontario, over the period 1966 to 1981, Grier (1982) has confirmed the negative association between DDE and bald eagle reproduction and population size. While encouraged by the rapid recovery of reproduction following the restrictions imposed on the manufacture and use of DDT, Grier cautions that reproduction is only one factor influencing population dynamics and that survival rates and habitat are also important. Using models of theoretical bald eagle populations, both Young (1968) and Grier (1980) have shown that differences in survival rates may be more important to the population status of this long- lived, slow-breeding species, than similar differences in reproduction. Although there is information available on reproduction, data on post-fledging and adult survival and the proportion of adults that are attempting to breed are needed. A high turnover of breeding eagles may effectively flush contaminants out of the population, but it also signifies potentially undesirable changes in the survival and age structure of the breeding population.

In the late 1960s, eggshells thinned in the Lake Superior population by more than 20% This was associated with DDE levels of more than 50 ppm. These were the highest levels found in any known subpopulation (Postupalsky, 1971). By 1971, the population on the Ontario shoreline of Lake Superior had declined from about 14 pairs to 3 or 4 pairs. On the Wisconsin shoreline of Lake Superior, the nesting population decreased to only one pair in 1973. In the Apostle Islands off the Wisconsin shore of Lake Superior, the first young were produced in 1983 and between 1983 and 1988, 57% of the nests were successful, producing an average of 0.8 young per occupied nest while 77% of the pairs in inland Wisconsin were successful and produced an average of 1.3 young per occupied nest (K. Kozie, personal communication). There is a high turnover of adults in the Apostle Island nests and this affects productivity significantly. Many of the Apostle Island eagles feed on herring gulls.

The number of bald eagles nesting on the Michigan shoreline of the

Great Lakes has increased from 10 breeding pairs in 1977 to 41 in 1989. In 1989, the Great Lakes shoreline subpopulation represented 25% of the total Michigan breeding population. Breeding pairs nesting on the Great Lakes shoreline and on river systems accessible to anadromous fish from the Great Lakes have significantly lower reproductive success than those nesting in inland Michigan (Table 9). Reproductive performance varies between the lakes, being highest on Lake Superior and lowest on Lake Michigan. Reproduction of pairs nesting on the shores of Lakes Michigan and Huron approaches complete failure within five years of establishment and considerable turnover of the adults is suspected. Addled eggs collected between 1985 and 1988 from shoreline nests contained higher levels of PCBs, DDE and dieldrin than in these from inland nests. These levels were high enough to cause embryo mortality. PCB and DDE levels in plasma of nestlings from Great Lakes shoreline nests collected in 1987 and 1988 were six times higher than those in plasma of nestlings from inland nests (W.W. Bowerman, personal communication).

TABLE 9. BALD EAGLE REPRODUCTIVE SUCCESS IN THE GREAT LAKES BASIN

LOCATION	INTERVAL	NESTING ATTEMPTS	PERCENT SUCCESSFUL	YOUNG PER ATTEMPT
LAKE & SHORELINE				
Lake Erie				
Ontario	1985-1989	32	69	1.1
Ohio and Michigan	1981-1988	58	53	0.79
Lake Superior				
Michigan	1981-1988	134	55	0.83
Wisconsin	1983-1988	?	57	0.80
Lake Huron				
Michigan	1981-1988	26	35	0.46
Lake Michigan				
Michigan	1981-1988	54	30	0.44
INLAND				
Michigan's upper pen.	1981-1988	474	63	1.0
Wisconsin	1983-1988	?	77	1.3
Minnesota	1982-1986	?	70	1.2
Michigan's lower pen. and Ohio	1981-1988	287	68	1.1

Source: Best *et al.*, (in prep.)

There are at least 90 historical nesting locations on the Canadian shores of the Great Lakes, including 60 on the lower lakes in eastern Lake Ontario and the upper St. Lawrence River, Lake Erie, Lake St. Clair and Lake Huron (McKeating, 1983). The historical nesting density was very high, particularly along some stretches of the Lake Erie shoreline. However, the last successful nesting on Lake Ontario occurred in 1951 (Quilliam, 1965) and from 1969 to 1972, only 10 active sites were found during extensive surveys of this historical breeding range (Weekes, 1974). Of 31 nesting attempts at active sites between 1969 and 1974 (Weekes, 1975), only 8 (26%) were successful and only 9 young fledged (0.29 per active nest). All known active nests on Great Lakes shoreline are on Lake Erie, and in the south-western extremity of Ontario. The population and distribution of nesting bald eagles expanded along the Lake Erie shoreline in the 1980s, and increasing numbers of nesting attempts are successful (McKeating, 1983; P. Hunter, personal communication). The role of the reintroduction projects at Long Point, and in Ohio and New York is unclear. However, there were no nesting attempts on Lake Ontario shorelines in the 1980s, despite the presence of suitable habitat.

It has been recommended that the International Joint Commission use the bald eagle as one of its "ecosystem objectives" as an indicator of overall habitat quality in the Great Lakes. As a tertiary level predator, it feeds on large fish, waterfowl, turtles and muskrats. Like the human subpopulations which consume Great Lakes biota, this long-lived, slowly reproducing species is subject to the chronic effects of contaminant exposure. As a tertiary predator, it provides an additional bioconcentration step and is therefore useful in detecting low level and trace contaminants. Where suitable unoccupied nesting habitat exists, the successful re-establishment, normal reproduction, and population expansion of the bald eagle can be used as a biological indicator of "virtual elimination" of persistent toxic contaminants in the local Great Lakes ecosystem.

3.6 HERRING GULL

The herring gull has been used as one of the major indicator species of ecosystem health in the Great Lakes. The monitoring program which is based on egg collection is described in Volume I. The detailed investigations carried out during the 1970s have been summarized by Mineau *et al.* (1984). Recent studies have measured biochemical changes caused by toxic contaminants. These are summarized in section 2 and have recently been reviewed elsewhere (Peakall and Fox, 1987).

Detailed population studies of this species have not been conducted. Rather biological studies have focused on productivity. The species has been widely studied in various parts of the world. A normally reproducing colony, with stable population dynamics, will fledge between 0.8 and 1.0 young per nesting pair, per year. Productivity over 1.0-1.2 young per pair usually indicates an expanding population. In the early 1960s, studies on the herring gull reproduction were conducted as part of an investigation of the effects of pesticides on Lake Michigan (Keith, 1966). The reproductive success was low (0.3 - 0.4 young/pair), largely because of poor hatching success. This was caused by both high embryonic mortality and high egg loss. Keith noted that some eggshells were chipped, although eggshell thickness was not measured.

The productivity of herring gulls in the Canadian Great Lakes between 1972 and 1985 is shown in Table 10. Between 1972 and 1975, reproductive success in the Lake Ontario colonies was very low. All colonies studied were producing 0.2 or fewer young/nest. The causes paralleled those from Lake Michigan: high embryonic mortality and high egg loss. The pathophysiological findings in embryos have been described by Gilbertson (1986). These include ascites, increased pericardial fluid volume, subcutaneous edema, shorter tarsal length, liver enlargement and necrosis. In contrast to the double-crested cormorant and many species of raptorial birds, the herring gull is quite resistant to DDE-induced eggshell thinning (Peakall, 1975). Eggshell thinning of only 4 - 8% was found, well below the degree considered to be critical. No cause and effect relationship could be established for any specific chemical and reproductive impairment. However, the presence of intrinsic (embryotoxic) and extrinsic (adult behaviour) factors were demonstrated by egg exchange experiments. The data obtained in 1976 are shown in Table 1. In 1977 for the first time in several years, productivity was above the 0.8 required for population stability on all three Lake Ontario colonies investigated. It has continued at good levels since, and the population has increased from 520 pairs in ten colonies in 1976 to 1540 pairs in 15 colonies in 1987 (Blokpoel *et al.*, in press). In Lake Erie, the only colony that was investigated during the early 1970s was within foraging distance of both the Niagara River and Lake Ontario. Hence the effects seen in this colony may represent contaminant conditions from these areas rather than from Lake Erie. Since 1978, productivity at both monitor colonies has been in or above normal range, indicating stable or growing populations. The wide fluctuations seen between 1983 and 1984 are probably due to natural conditions e.g., weather and predation. Depressed productivity levels were not recorded in colonies on Lake Huron, although no data are available before 1975. These findings are consistent with the lower levels of contaminants in the eggs of this species from Lake Huron compared to those from the lower lakes (Volume I).

TABLE 10. TEMPORAL AND GEOGRAPHIC VARIATIONS OF PRODUCTIVITY OF GREAT LAKES HERRING GULLS, 1972-1985

Location	1972	1973	1974	1975	1976	1977	1978	1979	1980	1981	1982	1983	1984	1985
Lake Ontario														
Snake I.	0.21					1.01	0.86	1.60	1.49	1.73		1.34		
Scotch Bonnet I.	0.12	0.06		0.15		1.10	1.01			2.13				
Brother's I.	0.10													
Presqu'Ile Pk.	0.06													
Black Ant. I.	0.08													
Mugg's I.						1.52	1.47	1.56		1.40			1.17	
Lake Erie														
Big Chicken I.	0.45													
Port Colborne			0.48	0.65	0.79		1.45			1.60				
Middle I.							1.70	1.63	1.62	2.10		2.17	0.95	1
Lake Huron														
Chantry I.				1.48		1.12	1.40	2.17	2.17	2.16		1.84		
Double I.							1.57	2.17	2.25	2.23		1.25	2.33	
Lake Superior														
Agawa Rk.				1.32	1.55		1.66	0.88	0.40	0.37	0.14	0.37	0.85	1.30
Granite I.							1.12	1.70	1.40	0.46	1.39		1.39	

Note:

Productivity is defined as number of young reaching 21 days of age per pair of nesting adults. Data for 1972-1981 from Mineau et al. (1984); subsequent data are from CWS (unpublished).

Monitoring of productivity in Lake Superior started in 1975 and, as in Lake Huron, there was no sign of depressed productivity at this time. Starting in 1980, there was repeated reproductive failure at one monitor colony (Agawa Rock) and occasional failure at the other (Granite Island). Poor reproductive success was associated with egg and chick disappearance. Investigations of the food supply (Fox *et al.*, 1990) showed that the proportion of fish in chick regurgitations was only half that in other areas of the Great Lakes. Under these adverse conditions of reduced food supply both members of the pair were frequently absent from their territory which lead to cannibalization of eggs and chicks by other herring gulls.

Gilbertson (1988) provides a flow diagram for demonstrating that a biological anomaly is caused by a suspected agent. He divides the sequence into three sections: initial signals, epidemiology and etiology. The initial signals were clear in the herring gull and data are certainly available for the epidemiology. Information on the etiology is much weaker. Although it is clear that a chemical agent(s) was involved, the specific agent(s) has not been identified by laboratory studies and there is no information on the toxicology of many organochlorines in the target species. The recent occurrence of the same type of problem in Forster's terns in Michigan, considered in section 3.10, has provided an opportunity to investigate the etiology of the problem further.

3.7 RING-BILLED GULL

At the beginning of the 20th century the ring-billed gull was very rare on the Great Lakes. The population expanded after about 1925 and by 1940 it was approximately 20,000 pairs. Between 1940 and 1960, the population in the Great Lakes was between 20 and 27,000 pairs (Ludwig, 1974) and by 1967 it had increased to 141,000 pairs. In 1976, the population was estimated at 281,000, increasing to 650,000 by 1984 (Blokpoel and Tessier, 1986). While details of the population changes between the late 1960s and early 1970s are lacking, the population has expanded at about 10% per year over the last 30 years.

Although the residue levels of organochlorines in the eggs were not monitored to the same extent as herring gull eggs, measurements made in the 1970s show that the levels were one half to one third of those in eggs of the herring gull. Altered reproductive success was not observed although congenital anomalies were recorded between 1971 and 1975 (section 2.2). There were two major die-offs of this species in southern Ontario in the late summer of 1969 and 1973 (Sileo *et al.*, 1977). Disease was considered to be a factor in only eight of the 138 gulls examined. The levels of DDE, dieldrin and PCBs were 30 - 90 times higher in the brains of moribund and dead gulls than those in healthy gulls. The levels of organochlorines were comparable to those associated with mortality from these compounds in experimental studies. The dead and dying gulls were severely emaciated, their body weights being only 75 - 80% of controls. The sequence of events that led to death is not clear, but it is clear that ring-billed gulls were, at that time, carrying potentially lethal burdens of organochlorines.

Major die-offs of ring-billed gulls were recorded on Lake Huron in 1965 and 1966, involving 3000 and 4000 individuals, respectively. These events were correlated with the die-off of alewife by Ludwig and Bromley (1988). The primary cause of death was botulism (113/123). In seven cases, organochlorine poisoning was considered to be the cause of death although no residue data were cited.

It is unfortunate that the only species for which there are numerous records of death of mature individuals is one in which reproductive success was not studied. However, casual observations and the population increase suggest there were no significant reproductive problems. Perhaps the rapidly expanding population (the mortality events took place at the end of the breeding season when the numbers would be maximal) caused a food shortage so that residues in fat were mobilised. However, there is no evidence of special environmental conditions in these years.

3.8 CASPIAN TERN

In 1980, there were 10 colonies of this species on the Canadian side of the lakes (Huron and Ontario) totalling approximately 2450 pairs. Between 1970 and 1980, an increase of 50% in the population was reported (Blokpoel *et al.*, in press). By 1987, the population had increased to 2950 pairs. On Lake Michigan the population was 1145 pairs in 1965, 1605 in 1976, 1640 in 1978, 2240 in 1985, 2850 in 1986 and 2720 in 1987.

The reproductive success of a colony of Caspian terns in Lake Huron was studied in 1972 (Snedden and Weseloh, in preparation). Hatching success was high (87%) and overall productivity was 1.1 chicks per pair. At this time nearby double-crested cormorants were experiencing almost complete reproductive failure (section 3.3). The residue levels of DDE in eggs was about half that found in the neighbouring cormorant eggs, but the levels of PCBs were approximately the same. The critical difference was probably the degree of eggshell thinning; 11% in the Caspian tern and 24% in the cormorant. In 1987, reproductive success in the Lake Michigan colonies (Kurita *et al.*, 1987) was poor. In the Saginaw Bay colony the hatching success was only 28% and no chicks were known to have survived to fledging. In the north central part of the lake the hatching rate was 58%, with a mean fledging rate of 0.79.

Ludwig (1979) put forward the hypothesis that although the U.S. colonies were maintaining their size, this was only accomplished by heavy recruitment from Canadian colonies. The basis of this hypothesis is the recovery data produced by cannon-netting (section 2.1) during 1966/67, 1969/73 and 1976/78. Ludwig also reported that preliminary analysis of banding data suggests that the mortality rate of adult birds in U.S. colonies was 1.4 - 2.2 times higher than in Canadian colonies. These findings point out the importance of detailed investigations using marked individuals, since on the surface the population appears to be healthy.

In 1987 and 1988, Caspian tern eggs were examined late in the breeding season, using an embryonic viability detector (Ludwig *et al.*, 1988). In the first year, 77 eggs were considered dead and were opened; four embryos had a crossed bill or mandible deformity and 15 had other structural defects. In the second year, 335 eggs were opened and 26 had crossed bills and 25 had other structural abnormalities. These data cannot be used to calculate the overall incidence of abnormalities since the total number of eggs initially present in the colonies was not known. Based on the breeding population of Caspian terns in Lake Michigan the maximum number would be approximately 6,000.

Wildlife toxicology is now moving into the examination of the effects of specific PCB, PCDD and PCDF congeners. The reproductive success of Caspian tern colonies has been plotted against TCDD-EQs (section 2.3.2.). The result shows a highly significant correlation between TCDD equivalents and percentage of embryonic mortality. Recent findings, reviewed in Umbreit and Gallo (1988), indicate that these compounds interact with hormones and hormone receptors. The TCDD-EQ factor currently used is based on studies using a rat cell line, but recent studies by Elliott (1989) on quail show that the relative toxicity of some PCB congeners in some species of birds may be quite different from that in rats. Further studies are needed so that the data generated in laboratory mammalian studies can be used to understand the toxicity of these materials to birds and other wildlife.

3.9 COMMON TERN

Common terns nest throughout the Great Lakes except for northern Lake Superior. In the lower Great Lakes, at least 58 colonies have existed between 1900 and 1980. The estimated population increased from 4000-7000 nesting pairs between 1900 and 1920, to a peak of 16,000 pairs in the early 1960s. In 1965 it began to decrease and by the late 1970s, there were 5,000 pairs with the greatest decrease occurring in the early 1970s. More recently, the population on Lake Ontario decreased further from 1300 pairs in 1976 to 940 pairs in 1987 (Courtney and Blokpoel, 1983). Similar, but less pronounced decreases occurred in Lakes Huron, Michigan and Superior (Blokpoel and Scharf, in press). Analysis of band recoveries indicates that the Great Lakes common tern population is relatively discrete and that there is little exchange with other breeding areas. This implies that the size of the population will be largely determined by reproductive success and mortality. Reproductive success has been affected by predation by birds and mammals, human disturbance and contamination of the aquatic food web (Courtney and Blokpoel, 1983).

Although perturbations such as flooding, encroachment and successional changes in vegetation and earlier nesting by gulls resulting in loss of nesting space may have affected common terns throughout their history in the Great Lakes, Courtney and Blokpoel (1983) suggest that these factors have resulted in permanent colony desertions. Great Lakes water levels have been unstable and high for the last 20 years, and this has forced many terns to abandon their preferred nesting habitat on small, sparsely vegetated gravel islands at low elevations for suitable man-made habitat on islands, peninsulas or the mainland. In 1980, about 70-80% of common terns nesting in the Great Lakes used man-made sites (Courtney and Blokpoel, 1983; Shugart and Scharf, 1983). These sites are often vulnerable to human disturbance, frequently heavily vegetated (some have trees), and are often occupied by gulls and visited by night-herons, owls and mammalian predators. Although these sites are sub-optimal habitats, they are very important because of their stability and the high proportion of the population which they support.

Gilbertson's observations of common tern colonies on two man-made islands in Hamilton Harbour in 1970 initiated the Canadian investigations of the impact of toxic chemicals on fish-eating birds in the Great Lakes. Gilbertson (1975) described what he saw as follows: "As I wandered about on one of these islands, many birds whirled around my head and swooped down upon me again and again to prevent me from approaching their nests.... I soon noticed something was fundamentally wrong with the colony. While some young of varying age were found in the nests, the eggs in most had failed to hatch. On examining one of these eggs, I found that the young chick had died before it could completely crack open the shell. Several other eggs contained dead embryos. At the edge of a grass tussock, I also noticed an abnormal two-week old chick, its upper and lower bill crossing over without meeting - a deformity which would result in certain starvation". In 1971, Gilbertson's visits to these same islands indicated that "these terns had a very low breeding success, with a high proportion of the eggs failing to hatch" (Gilbertson and Reynolds, 1972). In the Gull Island colony in Presqu'ile provincial park in 1970 and 1971, many chicks had deformed bills and feet and many adults showed unusually little aggression towards people visiting the colony (unpublished observations of J.M. Richards).

These findings were confirmed by a systematic study of colonies in the lower Great Lakes in 1972 (Morris *et al.*, 1976). As can be seen from Table 11, the reproductive success of the Lake Ontario colonies in 1972 was significantly lower than in the Lake Erie colony. This was due to egg failure and the disappearance of eggs and chicks. Similarly, the reproductive success of the failing colony on Gull Island in Presqu'ile provincial park, in 1976, was lower than the Lake Erie colony and other Lake Ontario colonies, despite the fact that this colony was "managed" to control vegetation and prevent encroachment by gulls (Morris *et al.*, 1980). Nisbet (1978) has

TABLE 11. REPRODUCTIVE SUCCESS OF COMMON TERNS IN THE GREAT LAKES, 1972-1983

LAKE & LOCATION	Year	Nests studied	x clutch size	Eggs hatched / Eggs laid	Causes of hatching failure (% of loss)	Chicks fledged / Chicks hatched	Causes of Chick loss	Chicks fledged per pair	Source
ONTARIO									
Hamilton Harbour	1972	98	2.59	0.35	Dp(27), E(23)	0.15	?	0.13	1
Toronto Harbour	1972	214	2.62	0.56	Dp(57), E(24), F	0.06	P	0.09	1
Gull I., Presqu'ile	1976	53	2.51	0.53	P(69), E(14)	0.41	P	0.55	2
Eagle W., St. Lawrence, R.	1976	179	2.55	0.58	Dp+B(88)	0.62	?	0.92	3
Toronto Harbour	1977	62	2.85	0.88		0.47	?	1.18	4
ERIE									
Port Colborne Breakwater	1972	348	2.61	0.80	Dp(57), E(24)	0.45	P?	0.94	1
	1976	300	2.40	0.34	E(49), Ds(27)	0.60	P	0.78	5
SUPERIOR									
Duluth Harbour	1983	171	2.62	0.19	Dp(67), Ds(23)	0.25	P,H	0.12	6

Dp: Disappearance
E: Rotten, cracked, hatching failure
Ds: Deserted
B: Broken
F: Flooded
P: Predation
H: Human disturbance

Sources:
1. Morris *et al.* 1976.
2. Morris *et al.* 1980
3. Waltz 1976
4. Blokpoel 1978
5. Courtney 1977
6. McKernan and Cuthbert 1989

calculated that a productivity of at least 1.1 chicks/pair is necessary to maintain a stable population of this species. Egg-related factors (failure of the eggshell to remain intact or of the embryo to survive and hatch) accounted for over 20% of the eggs which failed to hatch in Hamilton and Toronto Harbours, and at Port Colborne in 1972 and 1976. This suggests a chemical etiology. Egg disappearance was also important in Hamilton and Toronto Harbours in the early 1970s. This is similar to observations in Lake Ontario herring gulls (section 3.6). Eleven of 844 common tern chicks handled in five Lake Ontario colonies between 1971 and 1973 had crossed bills or other gross defects, compared with 2 of 1733 chicks handled in the Port Colborne colony on Lake Erie (Gilbertson *et al.*, 1976). The prevalence of birth defects in common tern chicks in Lake Ontario in this period (130 per 10,000 chicks) is over twice that observed in cormorants from Green Bay (section 3.3) and is the highest observed in any species in any location to date. Clearly, toxic contaminants have had a significant negative effect on the reproduction of this species in the lower Great Lakes. Common tern chicks with birth defects have been found in colonies in U.S. waters at the mouth of the St. Lawrence River as recently as 1987 and a severely deformed embryo was found in Hamilton Harbour in 1989 (D.V. Weseloh, personal communication). Chicks of this species are still experiencing contaminant-related problems in Saginaw Bay on Lake Huron and Green Bay in Lake Michigan (J.P. Ludwig and T. Erdman, personal communication).

PCB residues in 13 eggs from the Hamilton Harbour colonies which failed to hatch in 1970 averaged 90 ppm and were as high as 176 ppm (Gilbertson and Reynolds, 1972). The maximum HCB concentration was 6.5 ppm and the mean was 3.5 ppm (corrected for methodological loss). The LD50 for HCB for herring gull embryos is 4.3 ppm (Boersma *et al.*, 1986). The mean DDE concentration was 16 ppm and the maximum was 32 ppm. Studies of common terns in Alberta in the early 1970s found a highly significant negative correlation between shell thickness and DDE content of the eggs. Shells dented if they were 13-14% thinner than the mean prior to the widespread use of DDT and a sample of eggs with dented shells which averaged 17% thinning contained 7 ppm DDE (Fox, 1976). In 1971, eggs were collected over a 60 day period throughout the breeding season (Gilbertson, 1974). The terns immigrated with significant burdens of PCBs but negligible burdens of DDE and subsequently they became grossly contaminated with PCBs, DDE, HCB and dieldrin from the fish they consumed near the colony. PCBs were accumulated the most rapidly. In 1972, mean PCB and DDE residues in eggs collected from the Toronto Harbour were 65 and 13 ppm, respectively (Morris *et al.*, 1976) and 10 and 2.5 ppm in 1981 (Weseloh *et al.*, 1989). Similarly, mean PCB and DDE residues in eggs from the Port Colborne colony decreased from 79 and 11 ppm, respectively, in 1971 (Morris *et al.*, 1976) to 5 and 0.9 ppm in 1981 (Weseloh *et al.*, 1989).

There is widespread concern for the common tern in the Great Lakes. It is hoped that efforts to lower and stabilize lake levels will increase the amount of preferred habitat available, and that remedial actions which have reduced contamination of the food web will result in higher reproductive success. The common tern appears to be very sensitive to the reproductive effects of persistent contaminants and warrants continued surveillance.

3.10 FORSTER'S TERN

During most of this century, Forster's terns were uncommon and scattered nesters in southern Lake Huron, Lake St. Clair and the lower Great Lakes. This is no longer true. A survey conducted in 1980 found 100 nests in five colonies. When it was repeated in 1982, there were 850 pairs in 10 colonies. All of these were in marsh habitat in Saginaw Bay and Lake St. Clair (Scharf and Shugart, 1984). Although no actual population figures are available, data collected between 1981 and 1985 for the Ontario Breeding Bird Atlas suggest a range expansion and population increase in recent years for the Ontario portion of the Great Lakes (Cadman *et al.*, 1987).

The majority of Forster's terns breeding in Lake Michigan nest in colonies in Green Bay where reproductive success has been relatively low for several years (Trick, 1982). In 1983, a detailed study of reproductive success was undertaken. This compared a Green Bay colony with approximately 200 pairs with a nearby colony of 50 pairs on Lake Poygan in inland Wisconsin which was reproducing normally (Kubiak *et al.*, 1989). Hatching success in the Green Bay colony was 26% in contrast with 88% in the inland colony. None of the young hatched in the Green Bay colony were known to have fledged whereas 55% of the young produced in the inland colony fledged. Nest abandonment and egg disappearance were substantial in Green Bay but were not observed in the inland colony. The minimum incubation period in the Green Bay colony was 8.25 days longer than in the inland colony. The hatching success of artificially incubated eggs from the Green Bay colony was 52% that of eggs from the inland colony (Kubiak *et al.*, 1989). An egg exchange experiment confirmed that both intrinsic and extrinsic factors were contributing to the poor hatching success of the Green Bay colony (Table 2). Chicks artificially hatched from the eggs from the Green Bay colony weighed less, had an increased liver to body mass ratio, had shorter femurs and greater hepatic AHH activity (section 2.3.2) than those from the nearby inland colony (Hoffman *et al.*, 1987). Several of the Green Bay embryos which failed to hatch were malformed.

In general, the findings of Kubiak *et al.* and Hoffman *et al.* were similar to those reported for Lake Ontario herring gulls in the mid-1970s (section 3.6). The latter investigations were never completed because of the rapid increase in reproductive success that occurred in the late 1970s.

3.11 COMMON SNAPPING TURTLE

The snapping turtle occurs throughout eastern and central North America in both polluted and clean habitats. It is a long-lived, omnivorous species and has a very limited area of activity throughout the year. These characteristics make the snapping turtle a good integrator of pollutants and an indicator of contamination within a local area.

An investigation to determine the hatching success of turtle eggs and occurrence of deformities in hatchling turtles has been conducted at seven sites in Ontario during 1986-1989. Eggs from snapping turtle nests were collected for contaminant analysis and artificial incubation. Six wetlands on the shores of the Great Lakes were sampled: Big Creek Marsh and Rondeau Provincial Park, Lake Erie; Trent River, Lynde Creek, Cranberry Marsh and Cootes Paradise, Lake Ontario; and a seventh site in Algonquin Park, an area with very low contamination and hence a "control site" (Fig. 9).

The percentage of unhatched eggs at Algonquin Park ranged from 3.7 to 18.6% and the percentage of deformed hatchlings was from 2.6 to 5.0% (Fig. 10). The incidence of unhatched eggs and deformed young from the Lake Ontario populations were significantly higher than that at Algonquin Park in all years. However, the rates of deformity and unhatched eggs in the Lake Erie population were not significantly different from those at Algonquin Park (Fig. 10).

Logistic regression analysis of developmental anomalies and contaminant levels in eggs in 1986-1988 indicates that sites with the highest contamination tended to have the poorest developmental success in eggs.

4. CONCLUSIONS

1. Eleven wildlife species in the Great Lakes basin have experienced reproductive or other problems and/or population decreases since the 1960s. These have been attributed to chemical contaminants. The list of species comprises two mammals (mink and river otter); eight species of birds (double crested cormorant, bald eagle, black-crowned night-heron, herring gull, ring-billed gull, common tern, Caspian tern and Forster's tern); and one species of reptile (snapping turtle). All are long-lived, fish-eating species. Other species, such as the osprey, great blue heron and some rails, have probably been affected, but definitive data from populations in the Great Lakes basin are lacking. The lack of information on wildlife species other than mammals and birds is a serious concern.

The epidemiology of these reproductive failures and/or population decreases has been examined by Gilbertson (1988b). He concluded that "the case histories, concerning reproductive dysfunction in populations of fish eating mammals and birds, formally demonstrate the causal interrelationship between the observed epidemics and contamination of Great Lakes fish with organochlorine chemicals". The history of these effects indicates that at least two distinct contaminant-related phenomena are involved.

2. The reproductive failure of double-crested cormorants and bald eagles started to occur by the late 1950s and was associated with DDE-induced eggshell thinning. Eggshell-related problems were observed in herring gulls in Lake Michigan in 1964 and in Lake Ontario in 1970. In addition to these species, biologically significant eggshell thinning has been observed in black-crowned night-herons and common terns.

3. In the late 1960s and early 1970s, manifestations of developmental toxicity were noted in herring gulls, black-crowned night-herons and common terns from Lake Ontario. DDE levels in the food web have decreased in the 1970s and 1980s. Eggshell thickness has returned to near normal and is no longer biologically significant in cormorants and most bald eagles. These species have resumed breeding in the Great Lakes and this has been associated with manifestations of developmental toxicity. These manifestations are very similar to those seen in PCB-dosed birds, and have been most

prominent in the areas most highly contaminated with PCBs and related compounds; Green Bay in Lake Michigan, Saginaw Bay in Lake Huron, and in Lake Ontario.

4. In the last decade, there have been significant recoveries in reproductive success and increases in populations for most of the affected bird species. Serious contaminant-related reproductive problems in colonial fish-eating birds are now confined to specific "hot spots", such as Green Bay and Saginaw Bay, although manifestations of developmental toxicity are still observed in one or more species in Lakes Michigan, Superior, Huron, and Ontario.

5. The population increases of two species, the double-crested cormorant and the ring-billed gull, have been outside the range of variation normally recorded for vertebrate species. The populations are now 30- to 40-fold greater than any historical record. These unprecedented increases indicate that some fundamental alterations in the Great Lakes ecosystem have occurred, upsetting the ecological equilibrium of the system.

6. In contrast, populations of the three tern species have fluctuated within modest limits. The observation of reproductive failure and birth defects in common terns in 1970 was a major stimulus for the Canadian Wildlife Service's investigations of toxic chemical problems in the Great Lakes. Populations have fallen on the lower Great Lakes and productivity has been highly variable. Encroachment of vegetation and nesting gulls and disturbance by humans are thought to be contributory factors. The Committee on the Status of Endangered Species in Canada has reviewed the status of the common tern because of the decrease in its numbers.

Caspian tern populations in the Great Lakes have been increasing and reproductive success in Canadian colonies has been consistently good. Productivity of Caspian terns in some colonies in Lakes Michigan and Huron is poor and detailed studies indicate that the observed developmental toxicity is highly correlated with PCB-related TCDD-EQs in the eggs. There is evidence that the population in the U.S. colonies is maintained largely by immigration from the Canadian colonies.

There is little historic data on populations of the Forster's tern in the Great Lakes basin, but their numbers increased in the 1980s. In 1983, studies of the reproductive success of this species in Green Bay showed severe PCB-related developmental toxicity.

7. Although there has been some improvement in the productivity of the bald eagle and some expansion of its populations in the Great Lakes basin, it is still absent from many regions where it nested historically. Only a small fraction of the numbers that used to be on the lower Great Lakes are now there. Pairs nesting on the shores of Lakes Michigan and Huron continue to reproduce very poorly and no nesting attempts have been made on Lake Ontario since 1951. Lack of suitable nesting habitat may limit this species' recovery in some areas.

8. A survey of the rates of birth defects in young fish-eating birds showed that they have been recorded in 10 species in the Great Lakes basin, and that the maximum prevalence rates and the largest numbers of affected species have been in locations heavily contaminated with PCBs and related compounds, particularly Lake Ontario, Saginaw Bay and Green Bay. The prevalence of crossed bills in cormorant chicks in colonies in Green Bay was 22 to 87 times that of reference areas outside the Great Lakes. Chicks of terns and cormorants with birth defects have been found in Lake Ontario as recently as 1988.

9. Studies of captive mink have shown this species is one of the most sensitive, if not the most sensitive, mammalian species to PCBs, HCB and TCDD. Comparison of this information with the residue levels in fish from the Great Lakes suggests that the reproduction of mink inhabiting Great Lakes shorelines could be affected. Although harvest data are difficult to interpret, there is evidence of a decrease in mink populations along the north shore of Lake Ontario and the Ohio shoreline of Lake Erie. PCB burdens of most mink collected from Great Lakes shorelines are sufficient to induce adverse effects on reproduction.

Toxicological data and regional harvest statistics are not available for the river otter. However, there are several gaps in the distribution of otter harvests along the entire Lake Michigan shorelines of Wisconsin and Michigan, the Saginaw Bay region of Lake Huron, much of the Lake Superior shoreline of Michigan and Minnesota and the shoreline of Lake Ontario. There is no obvious reason why the otter should be significantly less sensitive to PCBs than its close relative, the mink. Otter feed predominantly on fish and other aquatic prey and therefore have a greater exposure to contaminants in the aquatic food web than mink. In studies comparing the contaminant burdens of mink and otter, the otter had higher burdens. It is therefore likely that PCBs have contributed to population decreases of this species in the Great Lakes basin.

10. Recent studies on the reproductive success of snapping turtles show that the percentage of unhatched eggs and the percentage of deformities in embryos and hatchlings of individuals nesting on the shorelines of Lakes Ontario and Erie are significantly higher than in an inland population. No data are available on any population effects on this or any other reptile species.

11. Normal physiological functioning can be considered as a criterion for a healthy ecosystem. Biological changes, such as alterations in porphyrin patterns and retinol levels, thyroid function and the activity of mixed function oxidase enzymes, indicate the continued presence of sufficient amounts of contaminants such as PCBs and related polyhalogenated aromatic hydrocarbons in fish to influence the physiology of herring gulls over much of the Great Lakes basin.

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7. APPENDIX

Common and scientific names of species referred to in the text.

Beaver (*Castor Canadensis*)
Mink (*Mustela vison*)
Otter (*Lutra canadensis*)
Double-crested Cormorant (*Phalacrocorax auritus*)
Great Blue Heron (*Ardea herodias*)
European Grey Heron (*Ardea cinera*)
Black-crowned Night-Heron (*Nycticorax nycticorax*)
Golden Eagle (*Aquila chrysaeetos*)
Bald Eagle (*Haliaeetus leucocephalus*)
Peregrine Falcon (*Falco peregrinus*)
Osprey (*Pandion haliaetus*)
Herring Gull (*Larus argentatus*)
Western Gull (*Larus occidentalis*)
Ring-billed Gull (*Larus delawarensis*)
Caspian Tern (*Sterna caspia*)
Common Tern (*Sterna hirundo*)
Forster's Tern (*Sterna forsteri*)
Virginia Rail (*Rallus limicola*)
Snapping Turtle (*Chelydra serpentina*)

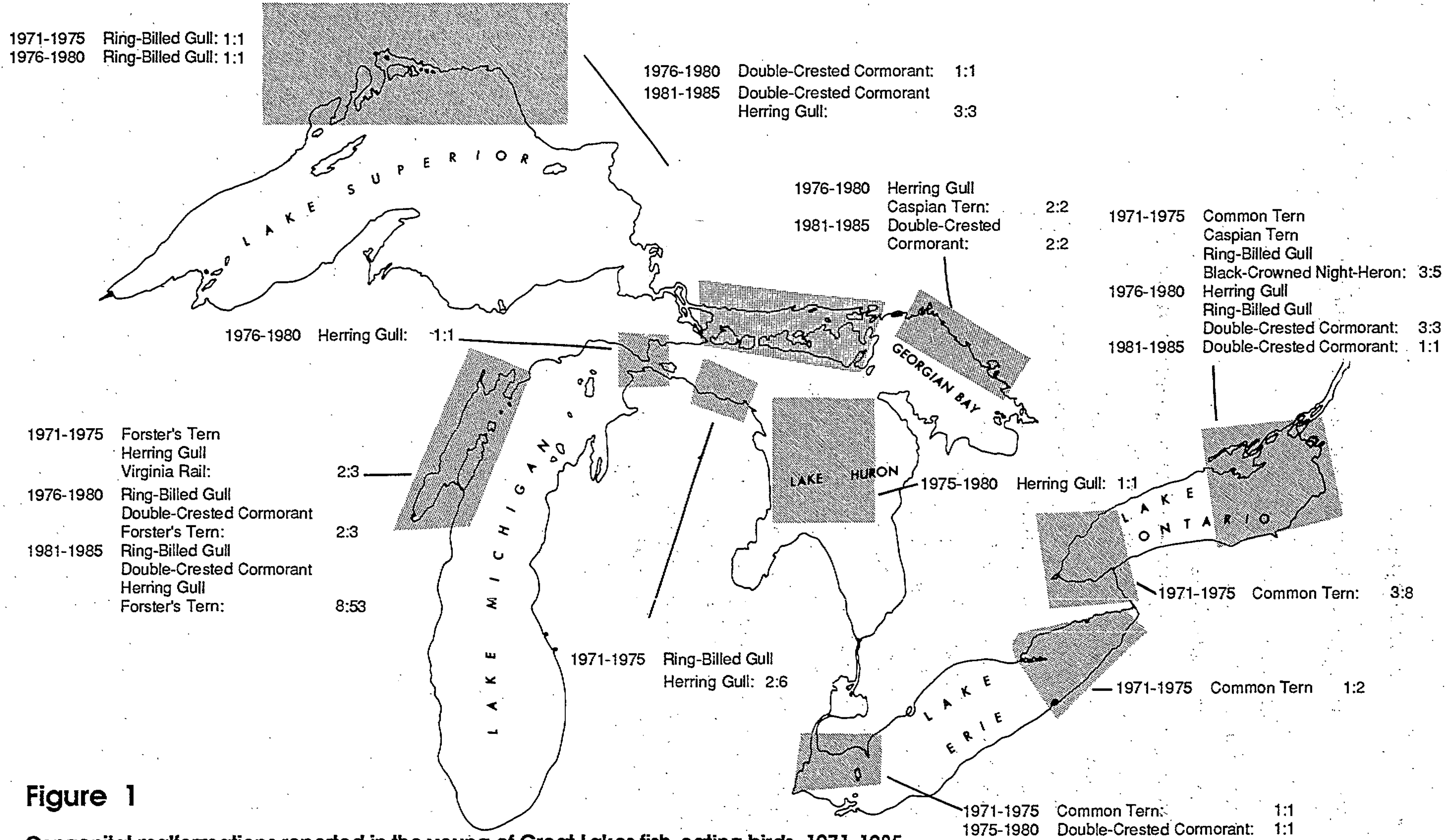

1971-1975 Ring-Billed Gull: 1:1
1976-1980 Ring-Billed Gull: 1:1
1976-1980 Double-Crested Cormorant: 1:1
1981-1985 Double-Crested Cormorant
Herring Gull: 3:3
LAKE SUPERIOR
1976-1980 Herring Gull
Caspian Tern: 2:2
1981-1985 Double-Crested
Cormorant: 2:2
1971-1975 Common Tern
Caspian Tern
Ring-Billed Gull
Black-Crowned Night-Heron: 3:5
1976-1980 Herring Gull
Ring-Billed Gull
Double-Crested Cormorant: 3:3
1981-1985 Double-Crested Cormorant: 1:1
1976-1980 Herring Gull: 1:1
GEORGIAN BAY
1971-1975 Forster's Tern
Herring Gull
Virginia Rail: 2:3
1976-1980 Ring-Billed Gull
Double-Crested Cormorant
Forster's Tern: 2:3
1981-1985 Ring-Billed Gull
Double-Crested Cormorant
Herring Gull
Forster's Tern: 8:53
LAKE MICHIGAN
LAKE HURON
1975-1980 Herring Gull: 1:1
LAKE ONTARIO
1971-1975 Common Tern: 3:8
1971-1975 Ring-Billed Gull
Herring Gull: 2:6
1971-1975 Common Tern 1:2
LAKE ERIE
1971-1975 Common Tern: 1:1
1975-1980 Double-Crested Cormorant: 1:1

Figure 1

Congenital malformations reported in the young of Great Lakes fish-eating birds, 1971-1985. For each geographical area, the species affected, the number of colonies in which defects were observed and the total number of affected individuals observed are listed sequentially.

Source: G. Fox (Canadian Wildlife Service).

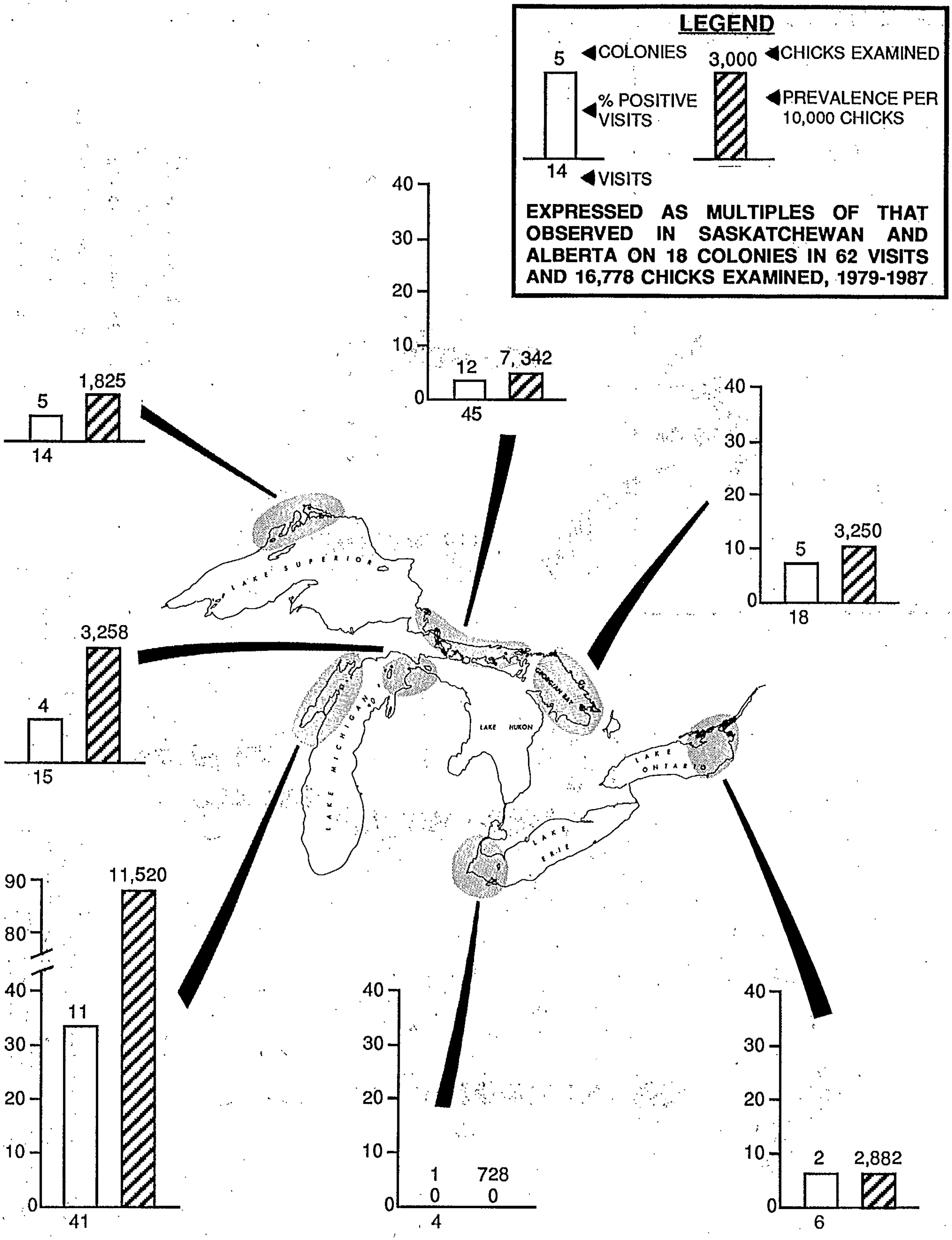
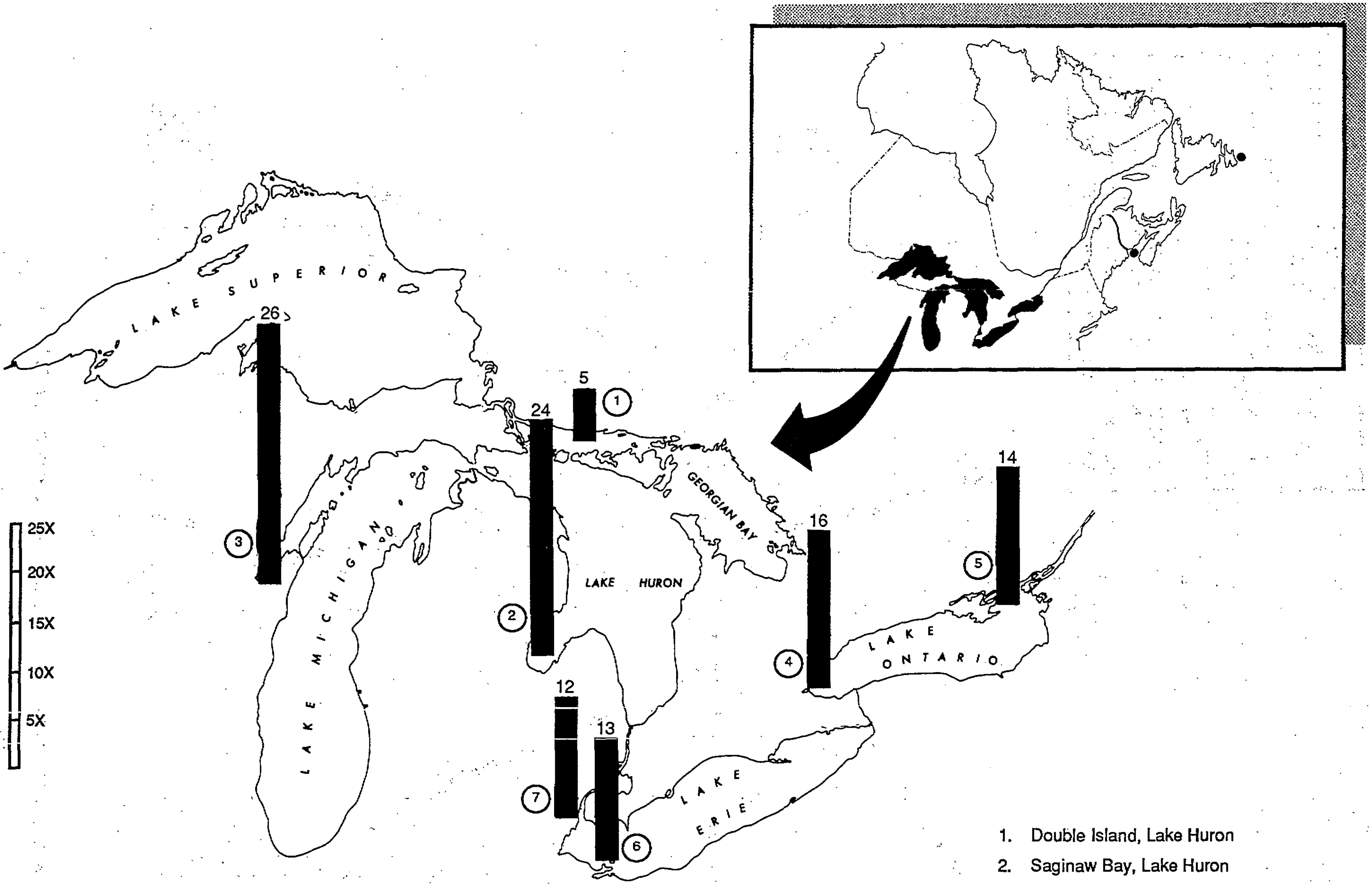


Figure 2

Occurrence and prevalence of cormorant chicks with crossed bills, 1979-1987.

Source: G. Fox et al, in press.



LAKE SUPERIOR
LAKE MICHIGAN
LAKE HURON
GEORGIAN BAY
LAKE ONTARIO
LAKE ERIE
26 >
24
5
16
14
12
13
①
②
③
④
⑤
⑥
⑦
25X
20X
15X
10X
5X

Figure 3

Porphyrin levels in livers of Great Lakes herring gulls in 1985. Median levels of highly carboxylated porphyrin levels in livers collected from seven Great Lake colonies are expressed as multiples of the median levels in herring gulls from the atlantic coast. Location of Great Lakes sites are identified with circled numbers.

Source: G. Fox (Canadian Wildlife Service).

1. Double Island, Lake Huron
2. Saginaw Bay, Lake Huron
3. Green Bay, Lake Michigan
4. Hamilton Harbour, Lake Ontario
5. Snake Island, Lake Ontario
6. Middle Island, Lake Erie
7. Detroit River

Atlantic Coast Sites are Shown in Map Insert

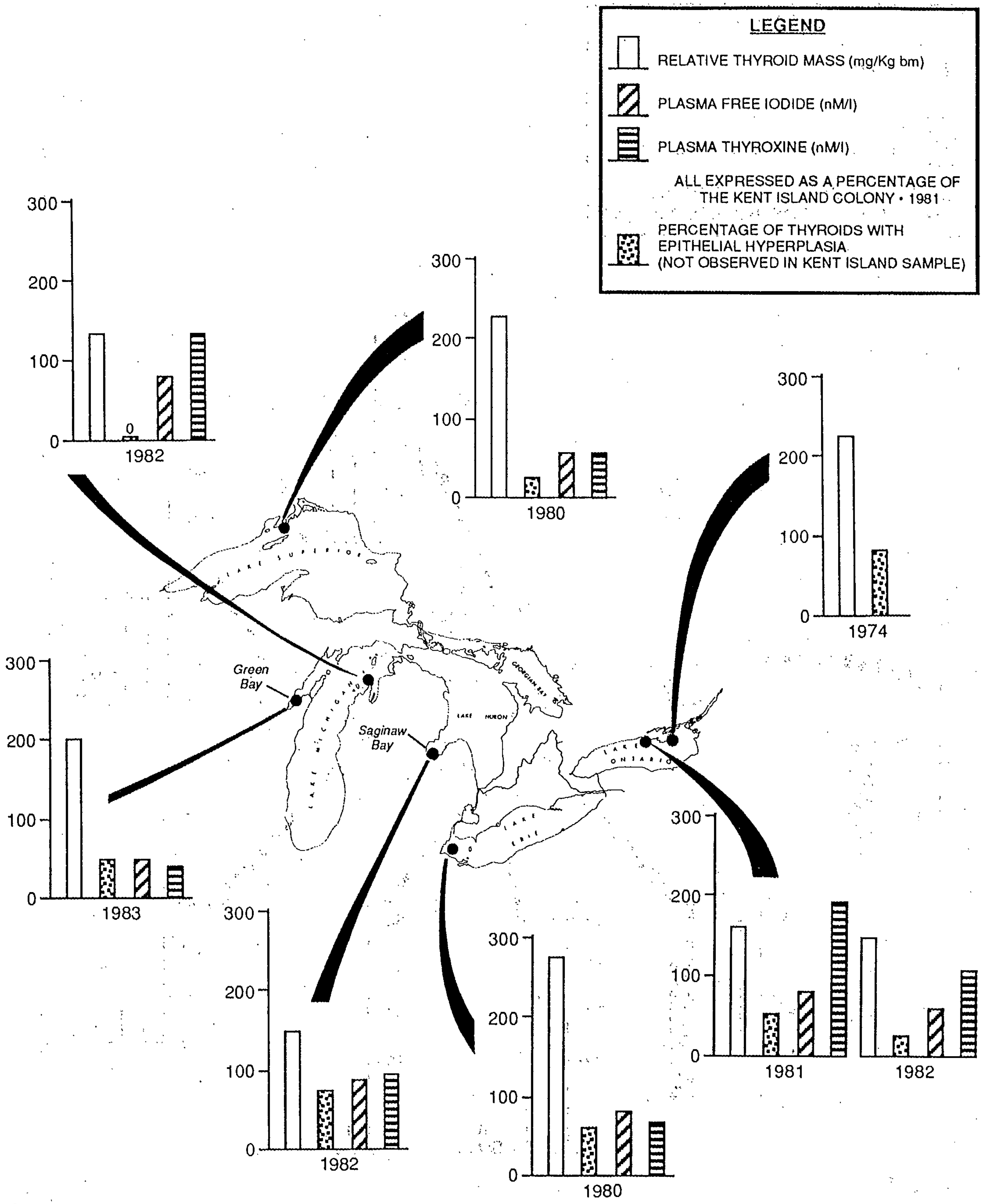


Figure 4

Spatial variation in thyroid-related measures in Great Lakes herring gulls in 1985.

Source: G. Fox (Canadian Wildlife Service).

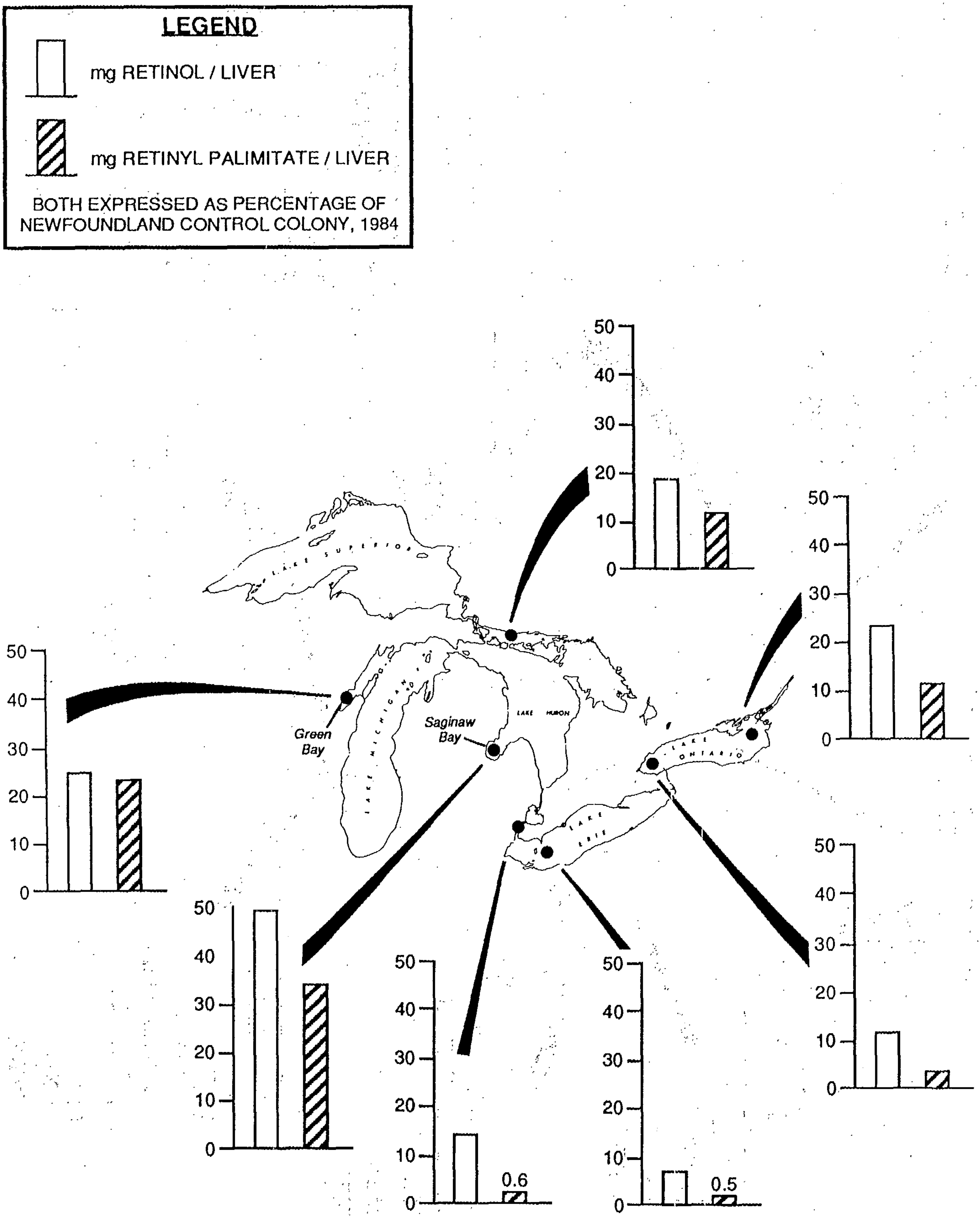
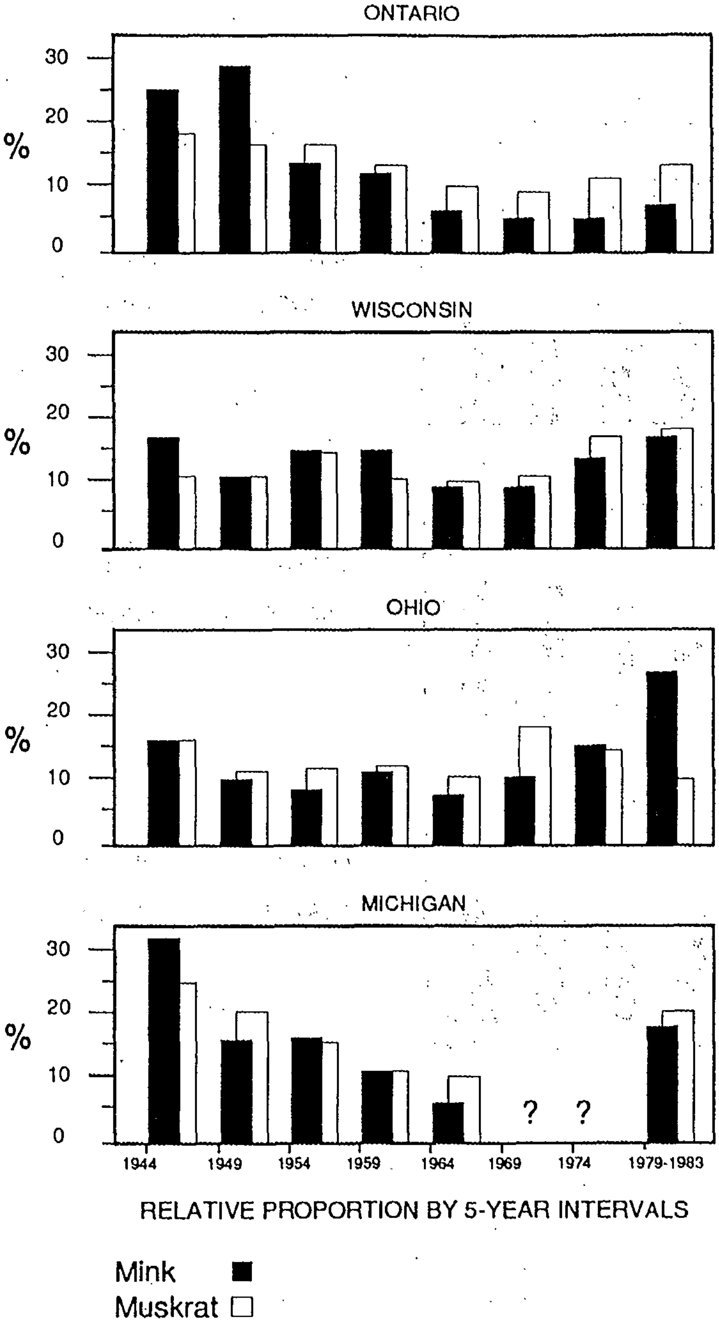


Figure 5

Spatial variation in retinoid stores of Great Lakes herring gulls.

Source: G. Fox (Canadian Wildlife Service).



TOTAL HARVEST

MINK = 2,179,731
MUSKRAT = 22,100,625

MINK = 1,085,977
MUSKRAT = 22,253,614

MINK = 410,252
MUSKRAT = 19,852,820

MINK = 609,593
MUSKRAT = 11,921,028

Figure 6

Temporal variation in the relative proportion of total harvest of mink and muskrat for the 40-year period 1944-1983 divided into 5-year intervals for Ontario, Wisconsin, Ohio and Michigan.

Source: G. Fox (Canadian Wildlife Service) using data from Novak et al, 1987.

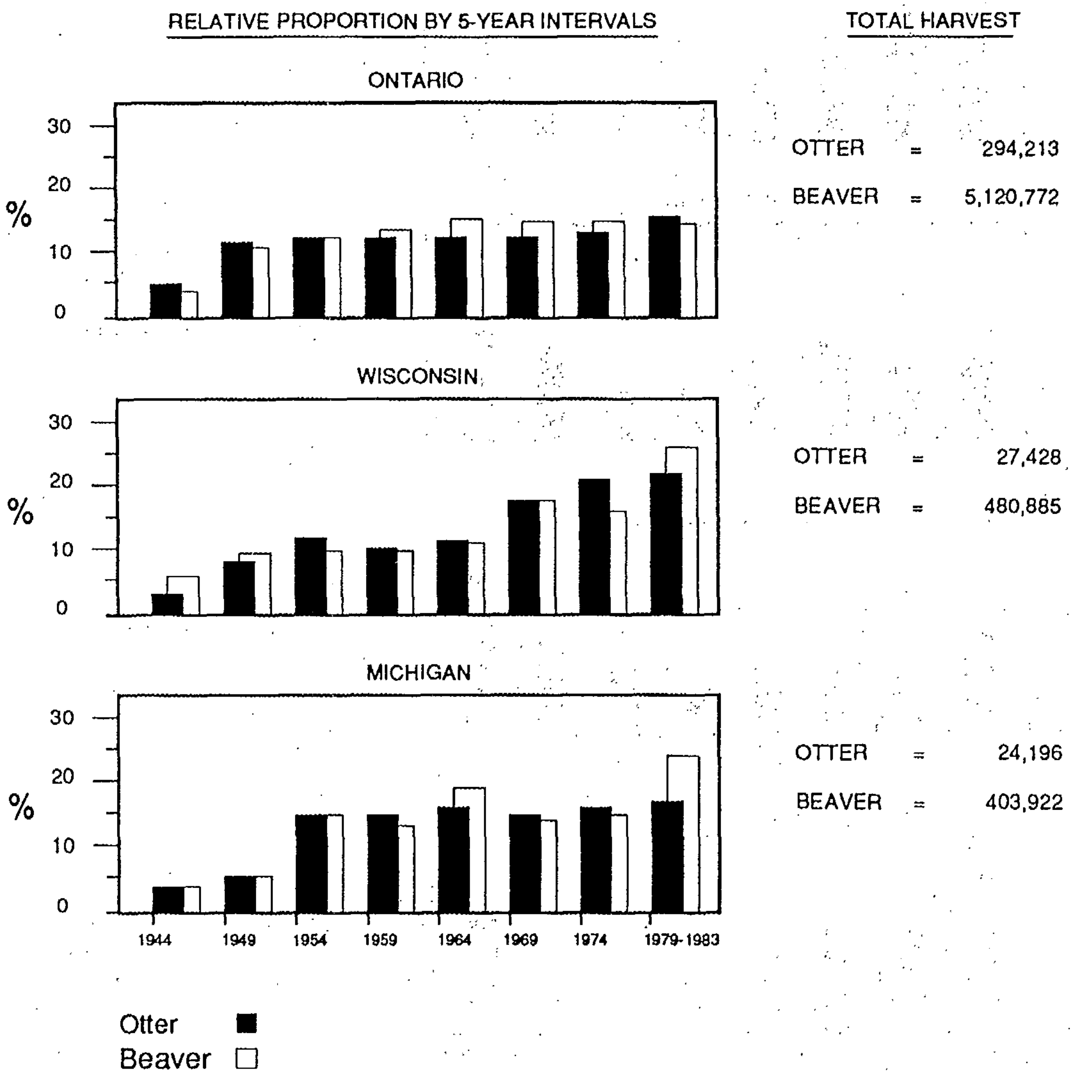


Figure 7

Temporal variation in the relative proportion of total harvest of otter and beaver for he 40-year period 1944-1983 divided into 5-year intervals, for Ontario, Wisconsin, and Michigan.

Source: G. Fox (Canadian Wildlife Service) using data from Novak et al. 1987.

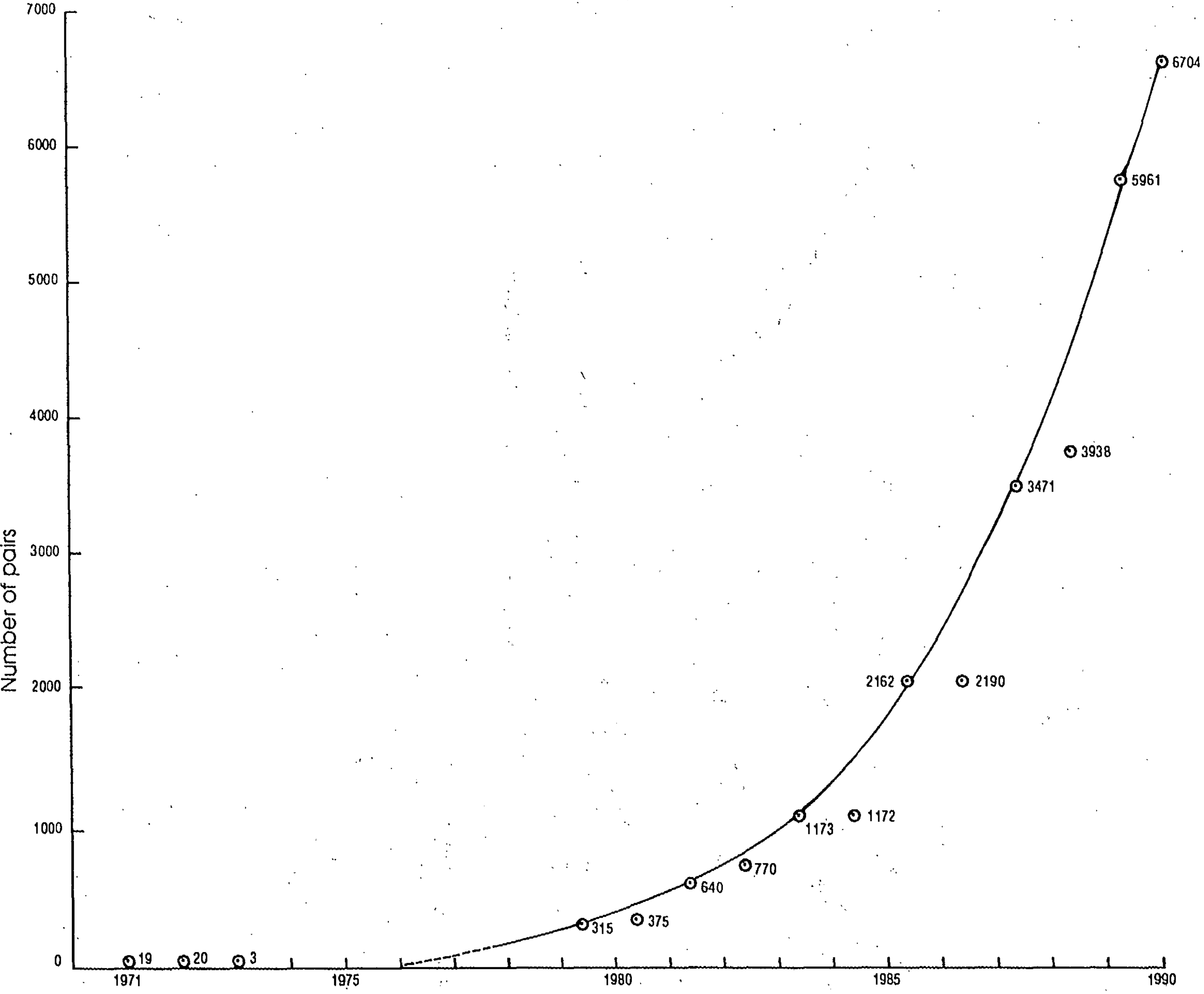


Figure 8

Temporal changes of the population of the double-crested cormorant on Lake Ontario, 1971-1990.

Source: S. Postupalsky, 1978.
D.V. Weseloh (Canadian Wildlife Service). Unpublished Data.

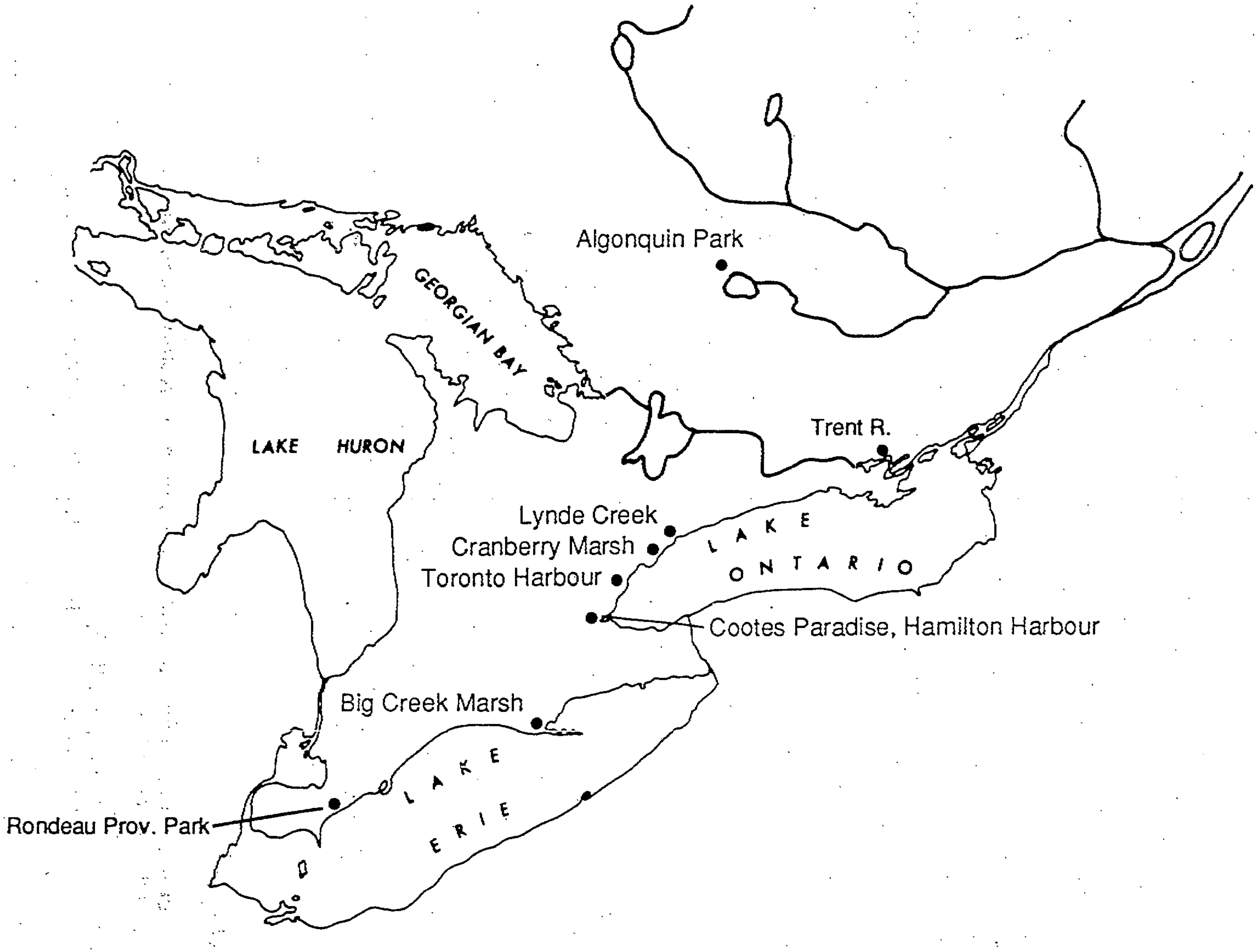

Algonquin Park
GEORGIAN BAY
LAKE HURON
Trent R.
Lynde Creek
Cranberry Marsh
Toronto Harbour
LAKE ONTARIO
Cootes Paradise, Hamilton Harbour
Big Creek Marsh
LAKE ERIE
Rondeau Prov. Park

Figure 9

Location of collection sites for snapping turtle eggs.

Source: C. Bishop (Canadian Wildlife Service).

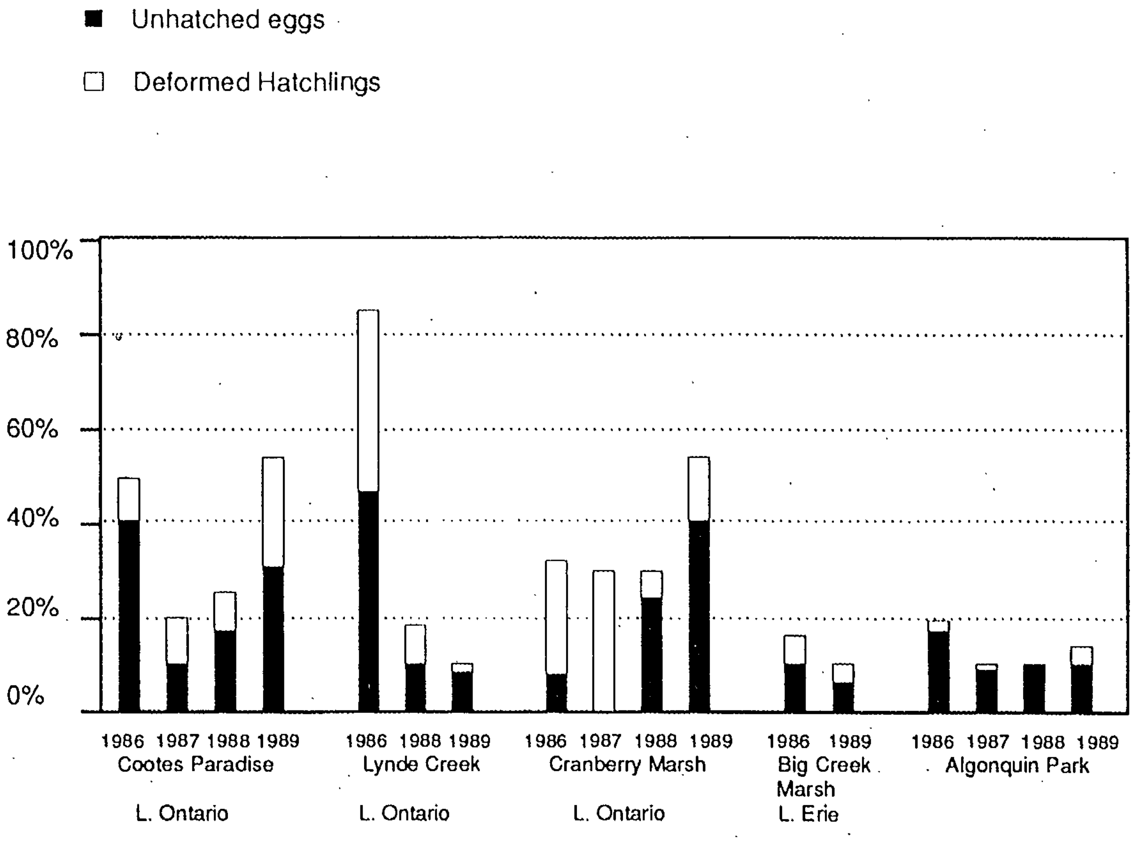


Figure 10

Incidence of abnormal development in snapping turtles (1986-1989).

Source: C. Bishop, Canadian Wildlife Service, unpublished data.

TOXIC CHEMICALS IN THE GREAT LAKES AND ASSOCIATED EFFECTS

VOLUME II PART 3

EFFECTS ON HUMAN HEALTH

EXECUTIVE SUMMARY

This part of the report describes what is known about the effects of toxic chemicals in the Great Lakes basin on human health. It focuses on the IJC's Critical Pollutants, although information on other toxic chemicals has been included when appropriate.

Multimedia analyses of national and Great Lakes regional data indicate that the majority (80-90%) of human exposure to persistent chlorinated organics comes from food, and a lesser amount from air (5-10%) and minute amounts from water (less than 1%). The limited amount of human tissue residue data suggest that residents of the Great Lakes basin are probably not exposed to higher levels of the Critical Pollutants than people residing elsewhere, although some subpopulations may receive higher than average exposures. Estimates indicate that most exposures to the Critical Pollutants are below tolerable and/or acceptable daily intakes; however individuals consuming large amounts of contaminated fish and wildlife, especially native peoples and sportsmen, have greater exposure to several persistent contaminants.

Human populations in the Great Lakes basin and elsewhere in Canada have been, and continue to be, exposed to many chemical contaminants and other agents that could cause adverse health effects. Many chemicals cause the same effects and these can also be caused by non-chemical factors. It is virtually impossible to control for all the confounding variables that could influence the occurrence of health effects. Studies often indicate that environmental contaminants are **associated** with certain health effects, but this is not equivalent to a conclusion that these contaminants **cause** the effect.

There have been few human population studies carried out in the Great Lakes basin. The few available studies have examined cancer incidence and mortality, adverse reproductive outcomes and the effects of air pollution. A number of these studies indicate that some groups in the Great Lakes basin have experienced adverse health outcomes associated with exposure to chemical contaminants.

- The incidence of total cancers in North America and the Great Lakes basin is increasing over time. This increase is closely linked with a

marked increase in lung and skin cancer and their respective associations with cigarette smoking and ultraviolet radiation. Cancer incidence rates also appear to have increased slightly over the last ten years. There has been inadequate study of the incidence of specific cancers among residents of the Great Lakes basin.

- Cancer is a significant cause of death in North America and the Great Lakes basin. Although the mortality rate due to all cancers has increased since 1950, it has actually decreased for cancers other than lung cancer. There have been very few studies of cancer mortality in specific areas of the Great Lakes basin. The limited data available indicate that cancer mortality in Niagara County municipalities does not appear to be significantly different from provincial averages. Information for municipalities in other Great Lakes counties has not been adequately assessed.

- Studies of reproductive outcomes in the general population show no consistent geographic pattern of different rates of birth defects in Ontario residents. There are insufficient data to determine whether or not the incidence of these outcomes in the Great Lakes basin is different from that in other regions of Canada. A study on reproductive outcomes of women residing in counties which use the St. Clair River as a source of drinking water does not provide any obvious indication that adverse outcomes are associated with chemical spills in the river.

- An ongoing study of the offspring of women who consumed large amounts of Lake Michigan fish has indicated that contaminant exposures of this type may cause several effects. Fish consumption was associated with significant decreases in birth weight, gestational age, head circumference and cognitive and motor deficits in the infants. These findings are important but further work is needed to clarify the relationships between exposure, blood and cord serum levels of contaminants and developmental effects in other exposed cohorts. Research is also needed to identify the causative agents more precisely.

- Air pollution in southern Ontario has been linked to increased hospital admissions for respiratory conditions and to decrements in lung function in some children and adults. However, the role of confounding exposures and other variables is important and should always be taken into account, even though this is sometimes difficult.

- The concentration of lead in the blood of children in the Great Lakes basin are similar to those in the blood of children living elsewhere. Current average blood lead concentrations may have some adverse effects on neurological development.

- Evaluations of contaminants exposures in native Indian populations in the Great Lakes basin have failed to reach substantive conclusions on cause and effect relationships. Knowledge of the degree of contamination of fish, water and air has resulted in serious concern among residents of some communities.

Studies on the effects of exposure to mixtures of toxic chemicals found in highly contaminated Great Lakes fish show that laboratory animals experience many different types of health effects. Animals exposed to mixtures of chemicals in drinking water at levels well above Great Lakes Water Quality Agreement Objectives showed no effects. It is unlikely that synergism is a widespread phenomenon; however, the additive nature of the effects must be considered.

Toxic chemicals found in the Great Lakes can have subtle effects on cellular metabolism. While these effects may not be adverse health effects in themselves and their ability to predict the eventual occurrence of adverse health effects is unclear, they are undesirable and support the need for a reduction in our exposure to such substances.

Human and wildlife populations in the Great Lakes basin are exposed to similar chemicals, although there may be considerable differences in the levels of exposure. Since there are also some biological similarities, it is useful to compare data on effects in humans with those in wildlife. While there are only limited data on human health effects available, there is more information on effects in wildlife. Both datasets suggest that developmental effects occur in the offspring of exposed parents, rather than in the parents themselves. Studies of wildlife populations suggest that more emphasis should be placed on studying effects on embryonic development, biochemical processes, reproduction and neurobehavioural development in humans.

There are sufficient data to conclude that some highly exposed or very sensitive human populations in the Great Lakes basin are at risk even if the precise nature and the extent of the threat to health are unclear. Residents of the Great Lakes consuming large amounts of contaminanted fish or wildlife should be aware of these concerns and reduce their intake of sportfish and wildlife in accordance with current advisories.

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1. INTRODUCTION

This part of the report describes what is known about the effects of toxic chemicals in the Great Lakes on human health. It is based on a review of the relevant scientific data and focuses on the effects of the IJC's Critical Pollutants, although information on other toxic chemicals has been included when appropriate.

Section 2 summarizes the available data on human exposure to toxic chemicals in the Great Lakes. This information was discussed in detail in Volume I of this report, but a brief review has been included here to emphasize that exposure is an essential antecedent to health effects. Results from analyses of human tissue samples from residents of the Great Lakes basin and elsewhere indicate that there is widespread exposure to many toxic chemicals, thus the potential for human health effects exists.

Section 3 reviews studies on the health records (births, deaths, cancer mortality, cancer morbidity) of human populations in the Great Lakes basin. The results of epidemiological studies of specific sub-populations in the basin are also discussed. Although some conclusions can be drawn, these data bases and studies are not sufficient to assess the effects of toxic chemicals in the Great Lakes on human health comprehensively.

Section 4 and the Appendix review information on several individual toxic chemicals, including the IJC's Critical Pollutants. The section also addresses effects at the cellular level as well as the potential for interactive effects.

Section 5 discusses the scientific evidence that suggests that there may be links between the effects of toxic chemicals in wildlife and human populations.

Conclusions are presented in section 6.

2. PATHWAYS AND TRENDS: HUMAN EXPOSURE AND MONITORING DATA

2.1 HUMAN EXPOSURE IN THE GREAT LAKES BASIN

The routes of exposure to environmental contaminants are the inhalation of air and airborne particulates, skin contact with water or airborne particulates, the ingestion of water, food or beverages, the handling of contaminated sediments, or the inadvertent ingestion of contaminated soil. Information on the magnitude of different exposure pathways is based mainly on what is known about ingestion of chemicals in food, drinking water, air and soil. Multimedia analyses indicate that the majority (80-90%) of human exposure to chlorinated organics comes from the food pathway, a lesser amount from air (5-10%) and minute amounts from water (less than 1%)(Birmingham *et al.*, 1989; Newhook, 1988). Most of the data available on human exposure to toxic chemicals in the Great Lakes comes from the analysis of contaminant levels in drinking water and fish. Consumption of contaminated fish and wildlife can significantly increase human exposure to chlorinated organics (e.g., PCBs, mirex) and organometals (e.g., methyl mercury, alkyl lead). For example, human consumers of contaminated Lake Michigan fish had significantly higher blood serum levels of most contaminants than non-fish consumers (Humphrey, 1983; Jacobson *et al.*, 1989).

Residents of the Great Lakes basin are exposed to toxic chemicals from many sources originating within and outside the basin. These sources include combustion processes, such as incineration and automobile exhausts, agricultural practices, leaking waste disposal sites and some industrial processes. Thus, human exposure to contaminants in the water of the Great Lakes constitutes only a small proportion of total exposure. The main routes of human exposure to contaminants in the water of the Great Lakes are the ingestion of fish and to a much lesser extent, drinking water.

2.2 A BRIEF REVIEW OF MONITORING DATA

Monitoring data on the presence of toxic chemicals in the environment and wildlife have already been reviewed in Volume I. In addition, the Appendix contains some exposure data on the IJC's Critical Pollutants. It is difficult to interpret these data because of inconsistencies in sampling and analytical protocols. There are also many data gaps. Despite these problems, the results of environmental and wildlife monitoring suggest that during the 1970s the levels of many toxic chemicals decreased significantly. Since then, however, many have remained at approximately similar concentrations indicating that levels in the ecosystem are equilibrating.

The organochlorine contaminants discussed in this report are detected only infrequently in drinking water and when they do occur, they rarely exceed existing standards or guidelines. Metals are detected more frequently, however, the levels rarely exceed existing standards or guidelines.

Residue levels of some of the most common contaminants (e.g., PCBs, HCB, dioxins) in residents of the Great Lakes basin are similar to those in people living elsewhere in North America. However, the data are insufficient to permit an assessment of spatial or temporal trends. Despite the shortage of information, there are likely to be subpopulations with higher than average exposures and residue levels. It is also likely that residue levels of the persistent organochlorine contaminants in humans increase with age.

3. STUDIES OF HUMAN HEALTH

3.1 STUDIES OF THE GENERAL POPULATION

There is limited information available on mortality, morbidity and disease incidence in residents of the Canadian side of the Great Lakes. Mortality information (i.e., death certificate codings) is collected provincially; the place of death is recorded, but this may not correspond to the place of residence. Morbidity data (i.e., incidence and prevalence) are much scarcer. Cancer incidence data are collected by the Ontario Cancer and Research Treatment Foundation (OCTRF) and are available for research purposes. The Ontario Ministry of Health maintains part of a national congenital anomalies database and permits access to that information. The only other incidence data are the few sentinel physician systems that exist and the hospital admissions information (HMRI) which is largely confidential. The only prevalence data collected is through community health surveys, such as the City of Toronto's (1982 and 1988), the results of which are publicly available. Several other factors reduce the effectiveness of public health records. For example, the quality of records varies considerably between jurisdictions, and depends upon physicians' diagnoses, bias in reporting, errors in recording information and diligence of the reporting authorities.

It is difficult to interpret retrospective population data in terms of cause (exposure to a specific contaminant or contaminants) and effects for several reasons. First, humans have been, and continue to be, exposed to many chemical contaminants and other agents that could cause adverse health effects. An individual chemical does not necessarily cause a unique health effect. Many cause the same effects and the same effects can also be caused by non-chemical factors. It is virtually impossible to control for all the confounding factors that could influence the occurrence of a health effects. Such studies often indicate that specific environmental contaminants are "*associated*" with certain health effects, but this is not equivalent to a conclusion that these contaminants "*cause*" the effect. However, if such studies describe a general association between exposure to chemicals and health effects, and they are combined with other information from other types of scientific studies, i.e., human exposure assessments, wildlife research, laboratory animal studies etc., the weight of evidence may indicate an immediate need to reduce or eliminate human exposure and/or to conduct more extensive research.

One of the major difficulties in determining the human health effects of long-term, low-level, multiple exposure to environmental contaminants is the fact that the subtle health effects likely to be occurring are very difficult to measure. Traditional health outcomes, such as cancer or birth defects, are relatively severe health effects and well recorded but may be insensitive health indicators for exposure to environmental chemicals. It is important therefore to measure less severe, more subtle endpoints such as neurobehavioural deficits, growth abnormalities, as well as cancer and reproductive or developmental effects.

3.1.1 Cancer Incidence

Cancer incidence data for Ontario are available from 1964 to 1982 (Marrett *et al.*, 1986). Using these data projections of future rates have been made through to 1994 for age and gender (Marrett *et al.*, 1986). The actual and predicted incidences increased. This is consistent with a general increase in cancer incidence (total number of cancer cases) and cancer mortality (total number of cancer deaths) in Canada and the U.S. from the 1950s through to the 1980s (Bailar, 1987; Bailar and Smith, 1986; Wigle *et al.*, 1986). These national increases are due, in large part, to an increase in population size and individual life expectancy, better reporting of cancer cases and increases in the rates of several cancers (cases per 100,000 population) associated with smoking, e.g. lung and bronchus cancer, and ultraviolet radiation, e.g. skin cancer. U.S. data indicate that overall incidence rates for all cancers and all cancers except lung and bronchus cancer (Table 1) increased from 1975 to 1984 by approximately 6% and 4% respectively (Bailar, 1987).

An analysis of the New York state cancer registry found no evidence for higher liver cancer, lymphoma, and leukaemia rates among residents living near Love Canal, when compared with residents living elsewhere in the state, excluding New York City (Janerich *et al.*, 1981). However, a higher rate of respiratory cancer was noted for all of Niagara Falls, New York, not just Love Canal (Janerich *et al.*, 1981).

The incidence of total cancers in North America and the Great Lakes basin is increasing over time. This increase is closely linked with a marked increase in lung and skin cancer and their respective associations with cigarette smoking and ultraviolet radiation. Cancer incidence rates also appear to have increased slightly over the last ten years. There has been inadequate study of the incidence of specific cancers in residents of the Great Lakes basin.

TABLE 1. CANCER INCIDENCE RATES IN THE U.S. POPULATION FOR 1975 AND 1984 (after Bailar, 1987)

	Incidence Rate (Cases per 100,000 population per year)	
	1975	**1984**
Lung, bronchus	45.2	55.3
Colon, rectum	47.3	50.3
Breast	47.7	51.8
Prostate	28.5	33.2
Pancreas	9.5	9.5
Leukemias	10.3	9.3
Non-Hodgkin's lymphoma	9.3	12.0
Stomach	9.2	8.1
Ovary	7.5	7.3
Brain, nervous system	5.4	5.4
All cancers	330.5	351.8
All cancers (except lung, bronchus)	285.3	296.5

3.1.2 Cancer Mortality

In Canada, cancer is "....the leading cause of premature death in women and the third leading cause (of death)....... in men" (Wigle *et al.*, 1986). In the U.S., it was the second leading cause of death (USDHHS, 1989).

Among Canadian men the risk of death from all cancer increased by 32% between 1951 and 1983. Among Canadian women the risk fell 12% by 1976 and then increased slightly. However the risk of death from cancers other than those of the lung actually decreased among both men and women in Canada from 1951 to 1983 (Wigle *et al.*, 1986). These data are essentially the same as those reported in the U.S. (Table 2) (Bailar, 1987 and National Cancer Institute, 1988).

A comparison of cancer mortality rates for 1981 to 1983 for type of cancer and for each province indicated that rates vary substantially by region (Wigle *et al.*, 1986). The only cancer mortality rate that was highest in Ontario (1981-1983) was for breast cancer. The mortality rates for breast cancer in the provinces do not vary as much as the mortality rates from other cancers.

TABLE 2. CANCER MORTALITY RATES FOR ALL AGE GROUPS IN THE U.S. POPULATION (after National Cancer Institute, 1988)

	Death Rate[1] (Deaths per 100,000 population per year)		
	1950	1975	1985
All cancers	157.0	162.2	171.3
All cancers (except lung, bronchus)	144.0	125.4	124.8

1. Data for all races, both sexes combined.

The Mortality Atlas of Canada (1980) compares cancer mortality in each county to the national average for 1966 to 1976. There is no evidence that total male and female mortality rates from all cancer sites are significantly different in residents of the Great Lakes basin, compared to residents living elsewhere. There are higher rates in certain Quebec counties bordering the St. Lawrence River. While the information provided in the Atlas does not permit an evaluation of the significance of these differences, the increases are associated with densely populated municipalities and could be due to several factors including lifestyle and socioeconomic factors.

The only study of mortality data on a county and municipal basis in the Great Lakes basin was initiated in response to concern about the leakage of chemicals into the Niagara River from hazardous waste disposal sites. The study used information from the National Mortality Data Base of Statistics Canada for 1951-1981 for Niagara County, and compared the mortality rates with rates for the whole province (NH&W, 1984b). The results showed that women living in Niagara County experienced significantly higher lung and liver cancer mortality, compared with provincial averages. A follow-up investigation found that there was no association with environmental variables. Instead, the increase was associated with a higher than average proportion of women smokers. After controlling for smoking, there were no differences compared to the provincial rates.

Total cancer mortality was five percent higher in males in Niagara

County, when compared with the provincial rates; however, this increase was not statistically significant (Holoweaty, 1989). The mortality rates for stomach, digestive tract, prostate, and bladder cancer in men in the City of Welland were significantly higher than provincial rates. Mortality from cancer of the tongue, mouth, and pharynx was also greater than expected for males in Niagara Falls and Fort Erie, Ontario. Rates for Niagara-on-the-Lake were similar to provincial rates. A decreased rate of lymphoid cancer and an excess of mortality from circulatory diseases were experienced by Port Colbourne men. County and province-wide rates of digestive tract cancers decreased over the same period of time. It was concluded that Niagara-on-the-Lake experienced fewer cancer deaths per 100,000 than all of Ontario (NH&W, 1984b). These data suggest that there is no consistency in cancer mortality rates and types of cancer between municipalities in Niagara County. To link cancer mortality rates to any environmental factors, it would be necessary to control for many variables including age, smoking behaviour, occupation and socioeconomic status.

Cancer is a significant cause of death in North America and the Great Lakes basin. Although the mortality rate due to all cancers has increased since 1950, it has actually decreased for cancers other than lung cancer. There have been very few studies of cancer mortality in specific areas of the Great Lakes basin. The limited data available indicate that cancer mortality in Niagara County municipalities does not appear to be significantly different from provincial averages. Information for municipalities in other Great Lakes counties has not been assessed.

3.1.3 Reproductive Outcomes

There have been only a few studies of reproductive outcomes in the general populations of residents of the Great Lakes basin. These are reviewed below.

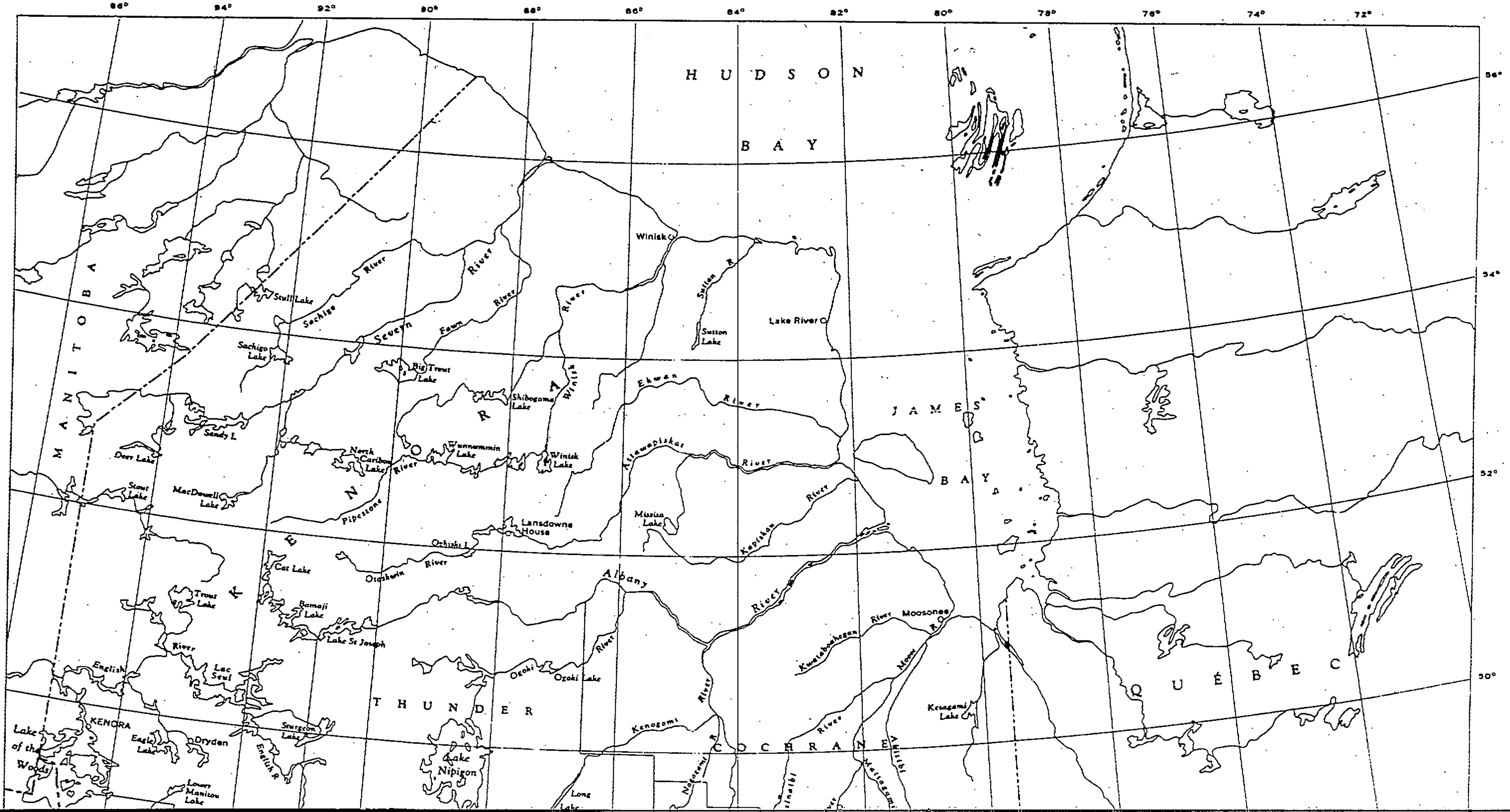
A report on human reproductive outcomes (see Figure 1 and Table 3) indicates that in central-east Ontario the rates of anencephaly, spina bifida, hypospadiasis, and congenital hip dysplasia for 1973-1983 were significantly lower than provincial rates (McIntyre, 1985). Central-west Ontario had significantly elevated rates of cleft palate/cleft lip and hypospadiasis.

Congenital anomaly rates for Southwest Ontario were not significantly different from provincial averages. Other regional differences can be seen in Table 3 (McIntyre, 1985).

Between 1973 and 1983, the provincial rate of anencephaly has decreased (McIntyre, 1985). This has also been observed in other countries where there have been improvements in prenatal healthcare. However, cleft lip/ cleft palate and hypospadiasis rates are increasing with time in Ontario (McIntyre, 1985). This may be due to improved diagnoses, changes in the ethnic structure of the population or the presence of an unspecified teratogen or teratogens. Stillbirth rates were significantly higher in Northwest Ontario, an area remote from the Great Lakes basin, compared with the provincial average (McIntyre, 1985).

A study of adverse birth outcomes between 1970 and 1983 in five Ontario municipalities in the Niagara region showed that there were differences from the provincial congenital anomaly rates, but there was no consistent pattern from year to year (DCES, 1986). An example of this can be seen in the Niagara region which showed a significantly decreased rate of stillbirths in 1970 while a significantly increased rate of Down's syndrome was seen in 1980. None of the mothers of the Down's syndrome children lived in areas which used the Niagara River as a source of drinking water. Babies born in Fort Erie had a higher rate of spina bifida and reduction deformities, but the rate of spontaneous abortions was lower, compared to other municipalities. Regional rates of cleft palate were higher in 1978 and 1982 and the rate of low birth weight babies in the municipality of Niagara was significantly lower than the provincial rate from 1980-1983. However, without considering more demographic data and clear hypotheses regarding associations between exposures and effects, it is difficult to interpret trends in reproductive outcomes.

These studies of reproductive outcomes in the general population show no consistent geographic pattern of different rates of birth defects in Ontario residents. There are insufficient data to determine whether or not the incidence of these outcomes in the Great Lakes basin are different from those of other regions in Canada.



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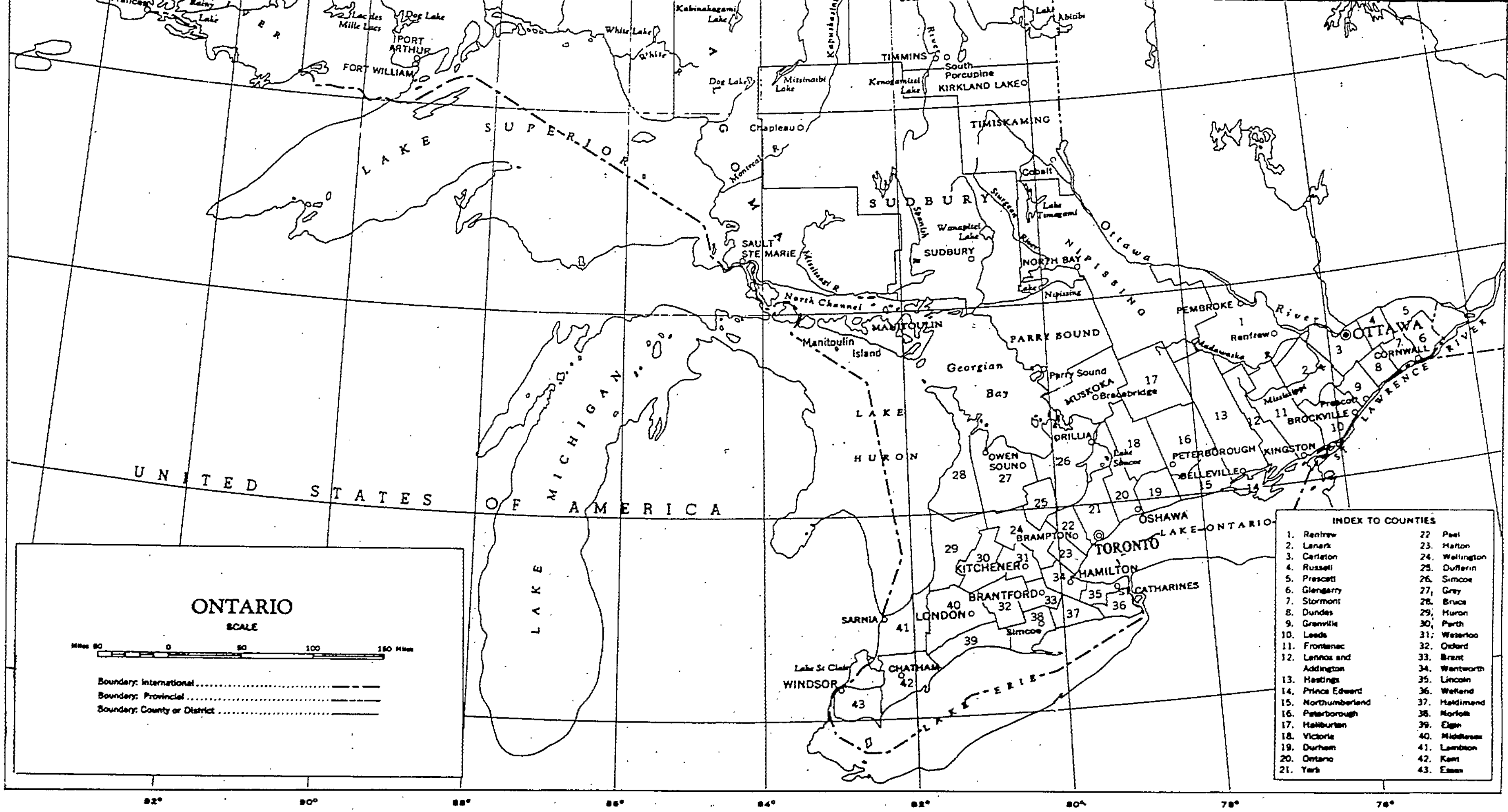
Sachigo River
Severn River
Fawn River
Sachigo Lake
Big Trout Lake
Shibogama Lake
Winisk River
Ekwan River
Attawapiskat River
Sandy L.
Deer Lake
North Caribou Lake
Wunnummin Lake
Winisk Lake
Stout Lake
MacDowell Lake
Pipestone River
Lansdowne House
Missisa Lake
Kapiskau River
Otoskwin River
Ozhiski L.
Albany River
Cat Lake
Trout Lake
Bamaji Lake
Lake St Joseph
English River
Lac Seul
Ogoki River
Ogoki Lake
Moosonee
Kwataboahegan River
Moose River
Kesagami Lake
Kenogami River
KENORA
Lake of the Woods
Eagle Lake
Dryden
Sturgeon Lake
English R.
Lake Nipigon
Lower Manitou Lake
Long Lake
Nagagami R.
Abitibi
Mattagami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7. Stormont
8. Dundas
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10. Leeds
11. Frontenac
12. Lennox and Addington
13. Hastings
14. Prince Edward
15. Northumberland
16. Peterborough
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22 Peel
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38. Norfolk
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43. Essex
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LAKE ONTARIO
LAKE ERIE
UNITED STATES OF AMERICA
PORT ARTHUR
FORT WILLIAM
TIMMINS
South Porcupine
KIRKLAND LAKE
TIMISKAMING
Cobalt
Chapleau
SUDBURY
NIPISSING
NORTH BAY
Lake Nipissing
SAULT STE MARIE
North Channel
MANITOULIN
Manitoulin Island
PARRY SOUND
Parry Sound
MUSKOKA
Bracebridge
PEMBROKE
Renfrew
OTTAWA
CORNWALL
Prescott
BROCKVILLE
KINGSTON
PETERBOROUGH
BELLEVILLE
ORILLIA
Lake Simcoe
OWEN SOUND
OSHAWA
TORONTO
BRAMPTON
KITCHENER
HAMILTON
ST CATHARINES
BRANTFORD
LONDON
Simcoe
SARNIA
CHATHAM
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WINDSOR
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Montreal R
Sturgeon River
Madawaska R
Mississippi R
Spanish R
Wanapitei Lake
Lake Timagami
Missinaibi Lake
Kenogamissi Lake
Kapuskasing R
Dog Lake
Lac des Mille Lacs
White Lake
White R
Kabinakagami Lake
Abitibi Lake
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88°
86°
84°
82°
80°
78°
76°
48°
46°
44°
42°

Produced by the Surveys and Mapping Branch, Ottawa, Canada, 1962.

Figure 1

Map of Ontario by county and region.

TABLE 3. SIGNIFICANT INCREASES OR DECREASES IN CONGENITAL ANOMALY RATES IN REGIONS OF ONTARIO (1973-1983)[1]

DEFORMITIES[2]	REGIONS OF ONTARIO[3]					
	S.W.	C.W.	C.E.	E.	N.E.	N.W.
Anencephaly	-	-	L	-	-	-
Spina bifida	-	-	L	-	H	-
Cleft lip/palate	-	H	-	-	-	-
Hypospadiasis	-	H	L	H	-	-
Congenital hip dysplasia	-	-	L	L	-	H
Reduction Deformity (Limbs)	-	-	-	-	-	-
Down's Syndrome	-	-	-	-	-	-

1. Regional figures compared to provincial averages (adapted from McIntyre, 1985)
2. H Rate is higher than the provincial rate.
   L Rate is lower than the provincial rate.
   - Rate is no different than the provincial rate.
3. Regions of Ontario
   S.W. Southwest Ontario (counties Bruce, Elgin, Grey, Huron, Kent, Lambton, Essex, Middlesex, Oxford, Perth)
   C.W. Central-West Ontario (counties Brant, Haldimand/Norfolk, Halton, Hamilton/Wentworth, Niagara, Waterloo, Wellington, Dufferin)
   C.E. Central-East Ontario (counties Durham, Haliburton, Metro Toronto, Northumberland, York Region, Peel, Peterborough, Simcoe, Victoria)
   E. Eastern Ontario (counties Frontenac, Hastings, Lanark, Leeds/Grenville, Lennox/Addington, Ottawa/Carleton, Prescott/Russell, Prince Edward, Renfrew, Stormont/Dundas/Glengary)
   N.E. Northeastern Ontario (counties Algoma, Cochrane, Manitoulin, Muskoka, Nipissing, Parry Sound, Sudbury District, Sudbury, Timiskaming)
   N.W. Northwestern Ontario (counties Kenora, Rainy River, Thunder Bay).

3.2 STUDIES OF SPECIFIC POPULATIONS

Studies of human populations attempt to link exposures to ill-health or death. Some focus on individuals in a group with a common exposure and then establish common health effects; others focus on individuals with similar health outcomes and attempt to determine a common cause (exposure).

Despite the limited number of human population studies to date, these investigations indicate that some groups in the Great Lakes basin have experienced adverse health effects.

3.2.1 Reproductive Outcomes of Women Who Consumed Lake Michigan Fish

An ongoing study of the offspring of women who consumed Lake Michigan fish has demonstrated the occurrence of several effects. The children were born to women who had consumed at least 11.8 kg of Lake Michigan fish over at least a six year period. The average consumption was 6.7 kg per year, which is equivalent to between two and four fish meals a month. Initially, a statistical correlation was found between the maximum yearly intake of Lake Michigan fish and maternal serum PCB levels (Aroclor 1260), although there was no correlation with cord serum PCB levels (Schwartz *et al.,* 1983). A further study of this group of 242 exposed individuals showed statistically significant decreases in infant birth weights (160-190 g lighter than controls), gestational age (average 4.9 days less than controls) and head circumference (average 0.6 cm smaller than controls) (Fein *et al.,* 1984). In addition the infants were assessed using the Brazelton Neonatal Scale. Contaminated fish consumption predicted motoric immaturity, poorer lability of states, a greater amount of startle and more abnormally weak reflexes. The most highly exposed infants were more likely than controls to be classified as "worrisome" (Jacobson *et al.,* 1984). These effects were associated with increasing cord serum PCB levels. Control analyses showed that these effects were not attributable to any of 37 potentially confounding variables, although the control group was much smaller than the exposed group (71 compared to 242) and there were differences between the control group and exposed mothers in terms of alcohol and caffeine consumption prior to and during pregnancy and the use of cold medications during pregnancy.

The infants were re-tested at seven months old and a significant decrease in the visual recognition of novel visual stimulus was associated with increasing cord serum PCB levels. This decrement did not correlate with fish consumption during pregnancy or lactation (Jacobson *et al.*, 1985).

Further studies of this group have demonstrated that PCBs are still detected in a significant proportion of the serum samples from the children (age 4 years) and that mothers' milk was the primary source of these exposures. Congener specific analyses have shown the presence of at least one moderately toxic PCB congener (Jacobson *et al.*, 1989). However, prenatal exposure (as indicated by cord serum PCB level) predicted poorer short term memory on both verbal and quantitative tests, in a dose-dependent manner (Jacobson *et al.,* 1990). Therefore, although much larger quantities of contaminants are transferred postnatally via lactation, prenatal transplacental exposure seems more important in terms of developmental effects.

These studies are important because they provide evidence of effects in human populations. However, it is difficult to interpret the relationships between fish consumption, maternal blood serum PCB levels, and cord serum PCB levels precisely, although cord serum PCB levels can be consistently correlated with the observed effects.

Another study of the offspring of women who consumed Lake Michigan fish has recently been completed, although it has not yet been published or peer reviewed (Dar, 1989). This study had a larger sample size (1112) and investigated associations between PCB levels of maternal and cord blood with fetal wastage and stillbirths, birth weight and length of the newborn, head circumference, ponderal index and birth weight percentiles for live births. The results suggest that birth length was associated with PCB exposure in shorter mothers, but that fish consumption did not affect "birth size". It is difficult to compare the results of this study with those of the Jacobsons because the available documentation does not contain many details on the study design or the exposed and control populations, such as the quantities of fish consumed or the duration of consumption, or what confounding variables were taken into account.

Another study of prenatal exposure to toxic chemicals showed that there was no association between maternal PCB levels and birth weight or head circumference (Rogan *et al.*, 1986b). There was an association between the highest concentrations of PCBs and DDE and decreases in two of seven neonatal reflex clusters (Rogan *et al.*,1986b). A further assessment of this group of children when they were six and twelve months old indicated that there was a small decrease in the psychomotor development index associated with increasing transplacental PCB exposure, although PCB exposure did not affect scores on the mental development index (Gladen *et al.*, 1988). Unfortunately, the maternal blood PCB levels cited in the Jacobsons' and Rogan's studies are not equivalent because of differences in the analytical methodologies, but the median PCB levels reported are markedly higher in the Rogan *et al.*, (1986a) study (9.06 ppb vs. 4.6 ppb). Thus, although Rogan *et al.*'s findings provide some support to the Jacobsons' work, there are important differences which should not be overlooked.

There have been several studies of accidental exposure to cooking oil contaminated with PCBs in Japan and Taiwan. In the Taiwan incident, the offspring of pregnant women who had ingested contaminated rice oil had higher rates of infant mortality and hyperpigmentation in newborns (Hsu *et al.*, 1985). A further study of the surviving children indicated other clinical symptoms (including eye discharge, pneumonia, bronchitis), lower scores on various scales of mental development and slowness at reaching various developmental milestones such as walking and talking (Rogan *et al.*, 1988). These findings are similar to some of those of Jacobson and Fein but there were significant differences in the exposures of the two groups. In contrast to the long term, low level exposure of the Lake Michigan women, the Taiwanese women were exposed to high doses of PCBs over a very short period of time (0.77 g to 1.84 g over 3 to 9 months) and this oil was also contaminated with other chemicals, such as polychlorinated dibenzofurans and polychlorinated quaterphenyls. It is possible that the effects observed in the Taiwanese children were related more to exposure to the furan microcontaminants than to the PCBs themselves.

The findings of Jacobson *et al.*, are important but further work is needed to clarify the relationships between exposure, blood and cord serum levels of contaminants and developmental effects in other exposed cohorts. Research is also needed to identify the causative agents more precisely.

3.2.2 Reproductive Outcomes of Women Possibly Exposed to Contaminants from the St. Clair River

Between 1980 and 1985, there were several chemical spills in the St. Clair River and a study of the reproductive outcomes of women who used the river as a source of drinking water has been undertaken (Robertson and Chan, 1986). Results indicate that the rate of stillbirths and perinatal mortality in counties using the St. Clair River as a source of drinking water was significantly less than regional and provincial rates. But there was a significantly higher rate of low birth weight infants. There were also several findings that were not statistically significant. This preliminary ecological study has not given an indication of any major effects of these chemical spills on human reproduction. A more thorough case control study has recently been completed and is being reviewed by the Ontario Ministry of Health. There were several problems associated with this study, including a low response rate and some of the data manipulation techniques. Nevertheless, it concluded that exposure to drinking water from the St. Clair River was not associated with stillbirths, congenital anomalies, low birth weight or infant deaths (Robertson and Chan, 1990), but the data presented do not justify this conclusion sufficiently.

An ecological study and a case control study conducted by Robertson and Chan on reproductive outcomes of women residing in counties which use the St. Clair River as a source of drinking water do not provide any obvious indication that adverse outcomes are associated with chemical spills in the river, although there are problems with the sample sizes and methodologies used.

3.2.3 Studies of the Effects of Air Pollution in Specific Populations in the Great Lakes Basin

Current knowledge of the health effects of air pollutants in the Great Lakes basin is limited, although four studies have been conducted. These are reviewed below.

Bates and Sitzo (1987) examined acute care hospital admissions for respiratory conditions and ambient air pollution levels in southern Ontario from the mid 1970s to the mid 1980s. They reported highly significant associations between respiratory related admissions and levels of SO2 and O3 and temperature in July and August. The results of this study were later confirmed in a report prepared by Ontario Hydro (Plagiannokos and Parker, 1988). This study also demonstrated that age and smoking behaviour are important confounding variables, although it did not take occupational history into account.

Respiratory health has been studied in adults and children in Hamilton. Cecilioni (1976) reported a five-fold difference in lung cancer rates for 1967-71 between different areas of the city, with the highest rates in neighbourhoods adjacent to heavy industry where levels of air pollution were probably greatest. However, there was no control for age, sex or other confounding variables. A further ecologic study linked lung cancer deaths with information from living residents on age, sex, residential, smoking and occupational histories (Shannon *et al.*, 1988). It concluded that air pollution was positively associated with lung cancer mortality, but that the effects were small when the results were adjusted for age and smoking behaviour.

In another study, a cohort of between 3200 and 3500 school age children in the 1980s were examined. The epidemiological design and methodology are described in Kerigan *et al.*, (1986), the air pollution data and the exposure assessment in Pengelly *et al.*, (1987) and the distribution of confounding variables in Pengelly *et al.*, (1984). Pengelly *et al.*, (1989) reported that children in the cohort with asthma who were exposed to concentrations of SO2 above a median of 10.6 ppb showed increased airway obstruction, compared to those exposed below this level. Pengelly *et al.*, (1986) also demonstrated an association between exposure to fine particulates and decreases in pulmonary function, but the effects were not associated with levels of SO2 or total suspended particulates. However, the results of studies on the whole cohort showed no association between air pollution and respiratory health and it was found to be strongly influenced by maternal smoking behaviour and hospitalization during infancy. This demonstrates the importance of confounding variables.

Lung function decrements have also been reported in children in the Great Lakes region. Studies conducted on children attending summer camps have reported that transient decrements in lung function were associated with air pollution measurements (ozone, sulphates, nitrates and sulphuric acid) (Raizenne *et al.*, 1989). Similarly, in two studies which assessed the effect of long term exposure to air pollutants, the lung function of children residing in southern Ontario were compared to children residing in Saskatchewan. After controlling for known confounders, the southern Ontario children were observed to have, on average, a 2% lower lung function (Stern *et al.*, 1989). Children living in southern Ontario were also observed to have more symptoms and a higher prevalence of inhalant allergies. Although this research focused on specific pollutants such as ozone, sulphur dioxide and acidic aerosols, the two regions studied probably differ with respect to other airborne toxic pollutants. Their role has not been assessed.

In summary, air pollution in southern Ontario has been linked to increased hospital admissions for respiratory conditions and to decrements in lung function in children and adults. However, the role of confounding exposures and other variables is important and should always be taken into account, even though this is sometimes difficult.

3.2.4 Studies of Inorganic Lead Exposure in Ontario Children

Exposure to lead can be a health hazard. It can cause toxic effects in several body systems including the hematopoietic system, the central and peripheral nervous systems, the kidneys, the gastrointestinal tract and the reproductive organs at blood lead levels of 40 µg/dL or more in adults. Children and the developing fetus are more sensitive to the effects of lead exposure than adults. The major exposure pathway is the ingestion of food and liquids but the inhalation of lead from automobile exhausts and the inadvertent ingestion of contaminated soil by children also contribute significantly to total body burdens. In addition, the ingestion of lead-containing paint by children may be important (Tenenbein, 1990).

Recent studies of the effects of lead have focused on neurological effects on children and the developing fetus (Needleman *et al.*, 1979; Needleman, 1982; 1983; Smith *et al.*, 1983). Taken together, they suggest that there is an association between adverse cognitive and behavioural effects in children at blood lead levels above 30 µg/dL (NH&W, 1987b). Canada has decreased its "intervention" level for children and women capable of bearing children to 20-25 µg/dL in blood.

Studies designed to assess neurobehavioural effects in children should take account of maternal exposure to lead because of the link between prenatal exposure to lead and deficits in neurological development.

There have been two studies of blood lead levels in children in the Great Lakes basin. Ellis *et al.*, (1985) reported average blood lead levels of 14.0, 15.8 and 15.5 µg/dL for the years 1982 to 1984 respectively, in children living in lead polluted areas of Toronto. Blood lead levels in other Ontario children living in urban, suburban and rural areas were 12.0, 10.0 and 8.9 µg/dL respectively (Ontario Ministry of Health, 1985). These levels are similar to those in children living in other parts of Canada (NH&W, 1987b), Europe and the U.S. (WHO, 1980b), i.e., 10-30 µg/dL blood. Blood lead levels are likely to decrease as the use of leaded fuels is reduced.

Exposure to lead can cause neurobehavioural effects in children and the fetus. Blood lead levels in children in the Great Lakes basin are similar to those in children living elsewhere. Current average blood lead concentrations may have some adverse effects on neurological development.

3.2.5 Studies of Native Peoples in the Great Lakes Basin

An extensive study of the exposure of Indian residents of Akwasasne to the mix of contaminants in the St. Lawrence (concentrating on methylmercury, PCBs and mirex as "indicator contaminants") and fluoride air emissions from a nearby factory was carried out in 1980 by Mount Sinai University, New York, under contract with the Department of National Health and Welfare (NHW, 1986). The study produced large volumes of data but failed to reach any substantive conclusions. Blood levels of contaminants were generally within the accepted "normal" range or slightly elevated.

Another study of methylmercury levels in native people across Canada showed some blood mercury levels above the acceptable range (less than 20 ppb) in Indian residents of the Great Lakes basin (Wheatley, 1979). No clinical effects were found (Wheatley *et al.*, 1979).

4. A REVIEW OF LABORATORY STUDIES ON THE EFFECTS OF TOXIC CHEMICALS

4.1 STUDIES OF THE EFFECTS OF SPECIFIC CONTAMINANTS

There is sufficient toxicological information available on only approximately 300 of the 800 chemicals identified in the water, fish and/or sediment of the Great Lakes (WQB, 1989b) to permit even a preliminary assessment of human health effects. Toxicology profiles for these chemicals are being prepared by the Department of National Health and Welfare. Profiles of the 11 IJC Critical Pollutants (PCB, mirex, hexachlorobenzene, dieldrin, DDT and its metabolites, 2,3,7,8-tetrachlorodibenzodioxin, 2,3,7,8-tetrachlorodibenzofuran, benzo(a)pyrene, alkylated lead, toxaphene and mercury) (WQB, 1985) and several other important toxic chemicals in the Great Lakes basin are presented in the Appendix. These profiles review information on the production, use, exposure and effects of these chemicals, as well as relevant standards and guidelines.

The information in the Appendix indicates that all of the chlorinated organic chemicals can bioaccumulate and many can cause cancer in laboratory animals. Several have been associated with effects on reproduction, development and the immune system in laboratory animals. The heavy metals (alkyl lead, mercury and tributyl tin) can also bioaccumulate and adversely affect reproduction and the nervous system.

4.2 STUDIES OF THE EFFECTS OF EXPOSURE TO MIXTURES OF GREAT LAKES CONTAMINANTS

The toxicity testing of chemicals detected in the Great Lakes and elsewhere is usually conducted on a chemical by chemical basis. This is for reasons of economy, ease of scientific interpretation of results and convenience. However, the general population is exposed to many contaminants through air, water and food. Thus, it is difficult to estimate the potential health effects of exposure to these mixtures. It is also possible that exposure to mixtures may be associated with synergistic or antagonistic effects, rather than simple additive effects. Synergism occurs when the effects of a mixture of chemicals is greater than would be expected from the addition of the effects associated with the individual chemicals. In contrast, antagonism occurs when the effects of a mixture are less than would be expected if they were added together. Additivity occurs when the effects are equal to the sum of the effects of the individual chemicals.

There have been some studies on the interactive effects of chemicals. Studies on pairs of organophosphorus pesticides, many of which are used in the Great Lakes basin, showed that 21 pairs had additive effects, 18 pairs had antagonistic effects and 4 pairs had synergistic effects (DuBois, 1961). In addition, mixtures of organochlorine pesticides have been investigated (Keplinger and Deichmann, 1967). Synergistic, additive and antagonistic effects were all observed although the synergistic effects were usually only about double the predicted additive effects. It has also been reported that exposure to organochlorine insecticides protects against the acute toxic effects of organophosphorus insecticides (DuBois, 1969; Murphy, 1969). These studies used relatively high exposure levels not usually encountered in the environment.

Several studies have examined the health effects of exposure to mixtures of toxic chemicals in the Great Lakes. Most of these have been conducted on laboratory animals. Following the discovery of epizootics of thyroid hyperplasia in Great Lakes coho salmon containing toxic chemicals (Moccia *et al.*, 1977), several studies were conducted to determine whether similar effects might occur in mammals exposed to the mixture of contaminants found in the fish. In one study, rats were fed diets containing coho salmon from Lakes Ontario, Michigan, and Erie. The results were compared with effects in rats fed diets containing Pacific salmon. The salmon from all three lakes caused hypothyroidism, and hyperplasia and hypertrophy of the thyroid in the rats (Sonstegard and Leatherland, 1979). In a separate study, rats fed salmon from Lake Ontario showed a dose-dependent association between consumption of Lake Ontario salmon and induction of liver mixed function oxidase enzymes. Mild histological changes were observed in the liver and thyroid and were attributed to the contaminants in the Lake Ontario fish (Villeneuve *et al.*, 1981). These effects were reversed when feeding of the contaminated fish ceased (Chu *et al.*, 1984).

In another study, groups of rats were offered drinking water containing concentrations of metals at or above the Great Lakes Water Quality Objectives (GLWQA, 1988) for 13 weeks (Chu *et al.*, 1981). No untoward haematological, histological, or biochemical effects were noted in the rats even at 25 times the Great Lakes Water Quality Objectives. In a separate study, a mixture of 15 persistent organohalogen chemicals found in the Great Lakes was administered to rats in drinking water for 13 weeks. The concentrations were up to 1000 times the Great Lakes Water Quality Objectives for each of the chemicals. No significant effects were observed (Côté *et al.*, 1985).

Captive ranch mink have been shown to experience severe reproductive failures when fed diets containing some species of PCB-contaminated Great Lakes fish (Aulerich *et al.*, 1971; Aulerich *et al.*, 1977). Subsequent studies in which PCBs were added to the diet indicated that PCBs were the likely causal agent (Ringer *et al.*, 1981; Hornshaw *et al.*, 1986). Mink are now known to be extremely sensitive to the effects of PCBs. This is discussed further in Part 2, Volume II.

More recently, Daly *et al.* (1989) examined behavioural changes associated with the consumption of fish. In this study, rats were fed diets containing salmon from Lake Ontario or the Pacific Ocean (control) for 20 days. The results demonstrated that ingestion of Lake Ontario salmon increased reactivity to aversive events, including mild electric shocks.

The studies reviewed in this section indicate that synergism in the Great Lakes and elsewhere probably occurs only infrequently. This conclusion is supported by a committee of the U.S. National Research Council which stated that "On the basis of theoretical considerations and its examination of some epidemiologic studies, the committee noted that effects of exposure to agents with low response rates usually appear to be additive. The only examples of interaction that were considered greater than additive occurred in humans exposed to agents such as cigarette smoke, that alone produced a high incidence of effects" (NRC, 1988).

In summary, studies on the effects of exposure to mixtures of toxic chemicals found in highly contaminated Great Lakes fish show that laboratory animals experience many different types of health effects. Animals exposed to mixtures of chemicals in drinking water at levels well above Great Lakes Water Quality Objectives showed no effects. It is unlikely that synergism is a widespread phenomenon; however, the additive nature of effects must be considered.

4.3 STUDIES OF EFFECTS AT THE CELLULAR LEVEL

There are several commonalities in the effects of many organohalogens and as research tools have become more sensitive, some effects have been observed at extremely low concentrations. Three of the most frequently observed effects are: interference with cell-to-cell communication, enzyme induction, and disruption of endocrine hormonal control. The significance of these cellular effects to predict the eventual occurrence of adverse health effects is unclear.

Cell-to-cell communication is necessary for tissue homeostasis, synchronization of tissue growth and regeneration. If communication is altered, subsequent development could be adversely affected. Among the pollutants found in the Great Lakes, chlordane, DDT and its metabolites, dieldrin, heptachlor and heptachlor epoxide, lindane, mirex, some PCBs (2,4,5,2',4',5'-hexachlorobiphenyl, Aroclor 1254), and toxaphene have been shown to affect cell to cell communication (Trosko and Chang, in press). It has been demonstrated that these chemicals prevent communication between cells by blocking the flow of nutrients, electrolytes, hormones and other vital materials necessary for the normal metabolism and development of the cell (Trosko, personal communication, 1989; Warner *et al.*, 1984; Trosko *et al.*, 1982).

Enzyme induction occurs as an adaptive response by the cell to the presence of a foreign or toxic chemical. 2,3,7,8-TCDD (dioxin), furans, B[a]P, chlordane, DDE, dieldrin, HCB, HCH (lindane isomer), mirex, PCBs, and toxaphene induce enzyme activity in many body tissues, most notably, the liver. One class of enzymes induced in response to the presence of these chemicals are the monooxygenase enzymes which are involved in the metabolism and excretion of foreign chemicals. During induction, however, other natural products of normal body function and xenobiotics (such as caffeine) can also be metabolized and excreted. Although enzyme induction has been correlated with toxic effects for some chemicals, i.e. TCDD, the full significance of this phenomenon in terms of its implications for health is not clear.

Many organochlorines are capable of upsetting the normal balance of endocrine hormones in laboratory animals. DDT/DDE are believed to have this effect because their structures are quite similar to alpha-estradiol, an effeminizing hormone. In addition, DDE is an enzyme inducer and influences the production of enzymes that break down male hormones, thus enhancing effeminization. Dioxin and some PCB congeners, whose structures resemble the estrogen moiety, have been shown to act as proestrogens in several animal species and in animal tissue. Dioxin can act as an anti-estrogen as well, although this depends on the dose, the timing and the duration of exposure. Heptachlor, chlordane, HCB, dieldrin, toxaphene, lindane, and DDE have been shown to induce from two to four different testosterone hydroxylase enzymes which enhance the metabolism of testosterone (Haake *et al.*, 1987; Elissalde and Clark, 1979).

These studies show that toxic chemicals found in the Great Lakes can have subtle effects on cellular metabolism. While these effects may not be adverse health effects in themselves and their ability to predict the eventual occurrence of adverse health effects is unclear, they are undesirable and support the need for a reduction in our exposure to such substances.

4.4 ESTABLISHING STANDARDS AND GUIDELINES

The state, provincial and federal governments in the Great Lakes basin have established standards and guidelines for toxic chemicals which are intended to protect human health. For the most part, these standards and guidelines are for individual chemicals in specific media (food, air, drinking water). However, human populations are exposed to many chemicals through multiple exposure pathways. Normally standards and guidelines for individual chemicals do not consider potential interactive effects (see section 4.2). However, they usually contain wide margins of safety to take account of these and other uncertainties. Despite this, there is a need to address interactive effects in the development of guidelines. There is currently one set of guidelines which specifically recommends the use of an additive model to estimate guidelines for exposure to mixtures (ACGIH, 1987).

Standards and guidelines are established using information from human studies and toxicological studies in laboratory animals. These studies are used to derive Acceptable Daily Intakes (ADI) or Tolerable Daily Intakes (TDI), i.e., levels at which a substance can be ingested on a daily basis by an individual for a lifetime without significant adverse health effects. ADIs and TDIs are generally derived by dividing the dose that produces no adverse health effects in laboratory animals by a safety factor of between 100 and 5000. The safety factor accounts for differences between humans and test species, the potential for differences in individual sensitivity, the adequacy of the laboratory data and the seriousness of the effects; it can take account of the potential interactions between chemicals if they are known. Permitted levels for contaminants in foods, drinking water guidelines and air quality standards are then established by apportioning the ADI and TDI to various media, following a case-by-case evaluation of the potential exposure via all possible routes. This relatively new approach is now being applied in the development of standards and guidelines in Canada.

Occasionally, guidelines and standards are based on estimates of cancer risk obtained from mathematical models and high-dose lifetime exposure studies in laboratory animals. Conservative assumptions are made throughout the process to ensure that the health of human populations is adequately protected. Upper bound estimates used to establish guidelines for carcinogens are in the range of 10^{-5} to 10^{-6}. This implies that risks of 1 (or less than one) additional cancer per 100,000 or 1,000,000 people is acceptable to most individuals.

Fish consumption guidelines and advisories have been developed by many different agencies in the Great Lakes basin (Table 4). They are intended to provide for the protection of human consumers of fish (based on the edible portion) and for the protection of fish consuming birds (based on whole fish).

Fish consumption advisories established by the federal government are set using the TDI and the Probable Daily Intake (PDI). The PDI is based on exposure to the chemical from all sources. Fish intake values take account of average and high consumption rates and expsure of specific sub-groups such as children or the elderly. If the PDI exceeds the TDI a fish consumption advisory may be proposed. Alternatively, restrictions on the sale or distribution of fish or recommendations for changes in dietary habits can be made. For example, the Ontario Ministry of the Environment publishes a "Guide to Eating Ontario Sport Fish" every year that translates fish consumption advisories into numbers of meals per year, month or week by fish species caught and catch location using known contaminant concentrations. The guide also contains specific recommendations for children and pregnant women and address exposure to mixtures of toxic chemicals in fish.

TABLE 4. FISH CONSUMPTION CRITERIA[1] (PPM, WET WEIGHT EXCEPT DIOXIN PPT, WET WEIGHT)

Parameter	**Great Lakes Quality Agreement Specific Objective[2]**	**National Health & Welfare Regulatory Limit[3]**	**U.S. FDA Action Level[4]**	**Ontario Sport Fish Consumption Guideline[5]**
Aldrin/Dieldrin	0.3	0.1	0.3	-
DDT (total)	1.0[6]	5.0	5.0	5.0
Dioxin (2,3,7,8-TCDD)	-	20	25[7]	20
Endrin	0.3	0.1	0.3	-
Heptachlor/Heptachlor Epoxide	0.3	0.1	0.3	-
Hexachlorobenzene (HCB)	-	0.1	0.3	-
Kepone	-	0.1	0.3	-
Lead	-	-	-	1.0
Lindane	0.3	0.1	0.3	-
Mercury	0.5[6]	0.5	1.0	0.5[8]
Mirex	Substantially absent[6]	0.1	0.1	0.1
PCBs	0.1[6]	2.0	2.0	2.0
Toxaphene	-	0.1	5.0	-

1. Criteria based on skinless fillet unless otherwise footnoted.
2. Based on the protection of the most sensitive species, this accounts for lower values for some compounds.
3. NH&W regulatory limits apply to fish in commerce only. The Province of Ontario applies these guidelines to sport fish consumption.
4. U.S. Food and Drug Administration (FDA) action levels based on fillet with skin on.
5. Ontario guidelines refer to restricted frequency of consumption of fish: if level of a single contaminent in a skinless dorsal fillet is below the guideline then unrestricted consumption is allowed; if the level exceeds the guideline then restriction in frequency of fish meals is advised. For women of child-bearing age and children under 15 years, restrictions apply below the guideline levels and no consumption is recommended for levels that exceed the guideline.
6. Criteria based on whole fish.
7. No consumption where TCDD levels exceed 50 ppt.
8. No consumption is recommended if the level for mercury exceeds 1.5 ppm.

The reduction of fat in fish flesh can decrease the amount of fat-soluble (lipophilic) contaminants in the portions of fish consumed (Skea *et al.*, 1979). Fats in fish flesh can be reduced by trimming fatty areas, puncturing or removing skin prior to cooking, cooking so that fats are drained (e.g., baked, broiled or grilled on a rack), or deep frying. These methods do not reduce the mercury content in fish flesh since this chemical is stored primarily in muscle tissue.

Concentrations of organochlorines in a fillet will always be lower than concentrations in whole fish because whole fish have a higher proportion of lipids than fillets. For a fish of 538 mm in length, the U.S. standard fillet produces concentrations of PCBs and organochlorine pesticides that are approximately 80% of the concentrations in whole fish, while the Canadian methodology results in concentrations that are approximately 50% of those in whole fish. Data on dioxins and furans in samples of fish from Lake Ontario indicate that concentrations of most congeners in fillets are approximately one-tenth of those in equivalent samples of whole fish (for low lipid species such as walleye). In lake trout (high lipid species) the fillet concentration may be as much as 80% of the whole fish level. Further study of the fillet: whole fish ratio is required.

By 1982, all eight U.S. Great Lakes states and the province of Ontario were operating or planning a program to sample sport fish and determine contaminant levels in the edible portions (Johnson, 1988). At that time, advisories on consumption of sport fish were often provided to the public using press releases or special notices in the state booklet of angling regulations that was issued to licensed anglers. As discussed earlier, Ontario provides a special publication, 'The Guide to Eating Ontario Sport Fish'. By 1987, the publication of special fish consumption advisory leaflets and books had expanded considerably. U.S. agencies issue an advisory only when the concentration of a contaminant exceeds a consumption guideline. In contrast, the Ontario guide is an annual summary of data on contaminant levels in fish. This provides advice on the frequency of consumption, even if no federal contaminant guideline is exceeded.

Different jurisdictions in the Great Lakes basin have established different guideline levels for fish consumption advisories. This has confused the public. However, a common format for developing sport fish consumption advisories is being developed as part of the Great Lakes Toxic Substance Control Agreement, signed in May 1986 by the governors of the Great Lakes states. These efforts should result in a binational agreement on common consumption guidelines that would be issued as a single document for recreational fishing throughout the Great Lakes.

5. EFFECTS IN WILDLIFE AS PREDICTORS OF HEALTH EFFECTS IN HUMANS IN THE GREAT LAKES BASIN

Since both human and wildlife populations are exposed to similar types of contaminants and have many biological similarities (physiology, biochemistry, cellular organization), it is not unreasonable to anticipate that at equivalent levels of exposure there could be some similar effects in both groups. The relationship between levels of exposure in human and wildlife populations have not been thoroughly examined. Generally, wildlife populations demonstrating the most serious and widespread effects have had the highest tissue concentrations of chlorinated organic chemicals such as PCBs, dioxin, furan, mirex, HCB, DDT, and organometals such as methyl mercury and alkyl lead. The most seriously affected animal populations are those in closest proximity to the most contaminated lakes and which ate primarily fish (for example, turtles, gulls, terns, cormorants, herons, mink and large predatory fish).

Many effects on wildlife (birds, fish, reptiles and mammals) have been reported. They include effects on reproduction and development, as well as effects on individual tissues and on specific biochemical parameters. They have been described in detail in parts 1 and 2 of this volume. The similarity of the effects across species is striking. This has led to the suggestion that humans, who consume from the top of the food chain, might also be at risk of developing similar types of effects, although the magnitude of the risk is likely to be less and directly related to exposure (IJC, 1989).

The limited studies of public health records have not indicated any consistent or increased trends in mortality, cancer incidence or adverse reproductive outcomes in human populations in the Great Lakes basin (see section 3). However, studies of the offspring of women who consumed Lake Michigan fish found that on average the infants were lighter than controls, had a shorter gestational age and a smaller head circumference. The infants also demonstrated behavioural decrements. Follow-up studies of the children continue to indicate behavioural decrements (see section 3.2.1). This suggests that parental exposure can affect offspring. Similarly, in wildlife populations effects have been observed in the offspring of adults who experienced long term, low level exposure to toxic chemicals, rather than in the adults themselves (IJC, 1989).

Behavioural deficits have been reported in birds (see Volume II, Part 2) exposed to heavy metals and organochlorines. Learning and deficits in motor skill development have also been reported in children and correlated with maternal tissue concentrations of mercury, lead and chlorinated organic chemicals. While the case for a direct cause and effect relationship between human exposure to lead and mercury and neurobehavioural effects has been established (NH&W, 1986; WHO, 1976), the same strong relationship for other chemicals, such as PCBs and dioxins, has yet to be confirmed. Studies of enzyme induction and thyroid hypertrophy in wildlife species and laboratory animals have shown that mixtures of chemicals in the environment and individual chemicals in the laboratory cause similar effects in fish, birds and rats consistently (IJC, 1989). These endpoints have not yet been adequately investigated in human populations.

Physical deformities have been reported in the offspring of adult birds, reptiles and fish that were heavily exposed to toxic chemicals. Current assessments of human health records of adverse reproductive outcomes in the Great Lakes basin have not indicated an excess of such effects (see section 3.1). However, the studies that have been conducted to date cannot be considered sufficiently rigorous.

In summary, human and wildlife populations in the Great Lakes basin are exposed to mixtures of similar chemicals, although there may be considerable differences in the levels of exposure. Since there are also some biological similarities, it is useful to compare data on effects in humans with those in wildlife. While there are only limited data on human health effects available, there is more information on effects in wildlife. Both data sets suggest that developmental effects occur in the offspring of exposed parents, rather than in the parents themselves. Studies of wildlife populations suggest that more emphasis should be placed on studying effects on embryonic development, biochemical processes, reproduction and neurobehavioural development in humans.

6. CONCLUSIONS

1. The major route of exposure of Canadians to most persistent chemical contaminants is through the consumption of food. Residents of the Great Lakes basin consuming large amounts of contaminated fish and wildlife may have higher than average intakes of these contaminants. Exposure to persistent contaminants from breathing the air and drinking or bathing in the water is not likely to exceed 10-15% of the total exposure.

2. The limited human tissue residue data available indicate that the general population residing in the Great Lakes basin is probably not exposed to higher levels of most persistent contaminants than people residing elsewhere in North America. Some specific sub-groups within the basin who consumed contaminated fish or wildlife have higher than average levels of several persistent chemical contaminants. There are insufficient data to draw any conclusions about temporal trends of tissue residue concentrations in the general population residing in the Great Lakes basin. Wildlife studies have shown that contaminant levels in the basin have declined significantly in many areas since 1970 but now have equilibrated.

3. The limited number of published studies available indicate that rates of cancer incidence and mortality and adverse reproductive outcomes in the Great Lakes region are no higher than would be expected in any highly industrialized areas of the country. However, a more thorough assessment of the data is desirable. More information is needed about lifestyles and occupation before overall mortality and morbidity trends can be related to specific environmental exposures.

4. The limited data available do not permit a thorough assessment of the effect of toxic chemicals in the Great Lakes on human health. The few epidemiological studies available show an association between the maternal consumption of fish from Lake Michigan and adverse effects in developing children. Others show effects associated with airborne pollutants. These are important findings and require replication and validation. Despite the uncertainties associated with these studies and that it may be never be possible to unequivocally identify any single contaminant or mixture of

contaminants as the cause of adverse health effects, it would be prudent to reduce the release of contaminants to the environment and thus reduce the potential for exposure and adverse human health effects.

5. Estimates of exposure for the general adult population to the Critical Pollutants are usually below established acceptable and/or tolerable intakes. These assume that fish consumption advisories are followed. Specific subpopulations residing in the Great Lakes basin which consume large amounts of relatively contaminated fish or wildlife (especially native peoples and sportsmen and their families) or which live in highly contaminated areas may be exposed to higher levels of toxic chemicals than the general population. Fetuses and breast-fed infants could be exposed to higher levels of contaminants than the general population through placental transfer and breast milk. Although these exposures are for a relatively short period of time, they occur at a critical period in development. Other groups of individuals may be more sensitive to the effects of contaminants than the general population. In some cases, individuals may show a hypersensitive response to low concentrations of a range of chemical substances. In other cases individuals may be suffering from a disease that compromises their health. For example, individuals who have respiratory diseases may be at greater risk from the effects of airborne pollutants in the basin.

6. There is limited data to show that the Critical Pollutants are more likely to act in an additive manner, rather than synergistically. Since most exposure guidelines and standards are based on individual chemicals and not mixtures, exposure to all synthetic chemicals should be as low as possible. Standards and guidelines intended to protect human health should take account of concurrent exposure through all relevant pathways and the potential additive nature of the effects.

7. Wildlife studies have shown that developmental and reproductive effects can occur in a wide range of species including birds, reptiles, fish and mammals exposed to mixtures of contaminants in the Great Lakes basin. While differences exist in behaviour and exposure between humans and wildlife, these findings suggest that these kinds of subtle health effects

should be investigated in human populations.

8. Highly exposed populations in the Great Lakes basin (and elsewhere in Canada) are at risk even if the nature and the extent of the threat to health are unclear. Resources and adequate appropriations are required for a long-term commitment for Great Lakes research to resolve the uncertainties associated with the implications for health of exposure to toxic chemicals and the means to reduce health risks.

9. Studies are required to clarify effects of contaminants on human health in the Great Lakes basin and to improve remedial and regulatory strategies:

(a) The relative importance of different exposure pathways (air, drinking water, food, soil, fish) for a wider range of contaminants should be quantified for human populations residing in the Great Lakes basin. This should include accurate information on the types and amounts of fish consumed. There is also a need to establish an ongoing monitoring program for residues of toxic chemicals in human tissues of the residents of the Great Lakes basin. The program should provide data for temporal and spatial analyses of concentration trends and should be correlated with equivalent data from wildlife populations. Part of this should include the establishment of a comprehensive human tissue bank.

(b) Monitoring of human subpopulations thought to be at higher risk (developing fetuses, breast-fed infants, disease-compromised individuals and individuals consuming large amounts of Great Lakes fish or aquatic wildlife) is needed. If higher exposure (based on body burdens or estimates of intake) is confirmed then recommendations should be developed promptly to assist these groups to reduce their exposure.

(c) There is a need to develop more sensitive endpoints to measure adverse effects on the reproductive, immune and nervous systems in both human populations and animals to assess the effects of long-term, low level exposure to toxic chemicals. This will not only provide direction for researchers and regulatory officials but also for physicians who need guidance in the identification of environmentally induced disease.

(d) Routine analysis for PCBs should be conducted on a congener-specific basis. Additional toxicity studies in laboratory animals using specific PCB congeners are required to permit meaningful hazard assessments of this large family of chemicals.

(e) Analyses for lead should include the identification of the lead species present because alkyl lead is at least 100 times more toxic than inorganic lead.

(f) Mercury occurs in food (primarily fish) and drinking water at low levels. Elevated blood levels in humans have been associated with adverse effects on the nervous system. Studies to determine whether more subtle effects are associated with long-term, low level exposure to mercury are needed.

(g) More information is needed on the interactive effects of toxic chemicals, particularly with respect to long-term, low-level exposure to mixtures.

7. APPENDIX: INFORMATION ON CRITICAL POLLUTANTS

This section summarises information on the 11 Critical Pollutants identified by the IJC (WQB, 1985). Six other chemicals are briefly discussed because they are important contaminants of the Great Lakes basin. This Appendix is not intended to constitute a complete review, rather, it highlights the most important effects produced under laboratory conditions in the most sensitive species. It also provides a general assessment of current exposure for Canadian residents of the Great Lakes basin. The standards and guidelines cited were all established to protect human health.

The potential human exposure to each of the Critical Pollutants from contaminated fish is illustrated using an example. The example assumes a concentration of each Critical Pollutant in fish. The levels selected are arbitrary but are intended to represent those in fish in the Great Lakes. They are not average concentrations. Concentrations in fish vary considerably depending on fish size, species and location caught; they have also changed over the past 15 years (comparing fish of the same age). Older fish bioaccumulate higher concentrations. Therefore, no single figure can accurately reflect the range of concentrations of these chemicals in fish.

Recent studies on fish consumption by sportfishermen and their families suggest that the estimated amount of fish consumed at a single meal (114 g) used to develop fish advisories, and in the calculations presented here, may underestimate the average amount of Great Lakes fish consumed per meal. Nevertheless, the examples demonstrate the calculation procedure for estimating intake and approximate ingestion of the Critical Pollutants.

In 1983 and 1986 the Ontario Ministry of the Environment surveyed Ontario fishermen about how much fish caught by angling in Ontario waters they would eat at a single meal (Cox *et al.*, 1987). The majority of respondents indicated that they consumed 227 g or more of fish per meal. The calculated mean meal size was 284 g in 1983 and 295 g in 1986. In addition, approximately 86% of the respondents in both years consumed more than the 114 g used to develop fish advisories at a single meal. However, the majority of survey respondents also indicated that they consumed fish caught by angling less frequently than the once a week assumed by the calculations in this report.

7.1 PCBs

7.1.1 Production and Use

PCBs are a group of chemicals (there are 209 possible congeners) that have been used as dielectric fluids in capacitors and transformers, hydraulic fluids, adhesives, plasticizers, heat transfer fluids, wax extenders, lubricants, cutting oils and flame retardants (Newhook, 1988). This is because they are chemically stable, unreactive and have high dielectric constants.

In the 1970s, concerns over the environmental effects of PCBs led to bans on their manufacture and importation and on most non-electrical uses in Canada and the U.S. In many cases, they have been replaced by other substances. Closed system electrical uses of PCBs are now being phased out, and there are stringent controls on handling, storage, transportation and disposal (NH&W, 1989a). Despite these actions, it has been estimated that there are still more PCBs in use today than have been released to the environment to date (Colborn, 1989). Trace levels of PCBs have been found throughout the global environment. This is thought to be due to improper disposal practices and accidental releases together with long-range atmospheric transportation. PCBs are persistent and bioconcentrate in food chains because of their stability and lipophilicity.

7.1.2 Standards and Guidelines

NH&W Tolerable Exposure Guideline:	1 µg/kg bw/day (EPS, 1987)
NH&W Guideline Fish:	2 ppm edible portion (UGLCCS, 1988)
Ontario Drinking Water Guideline (Interim MAC):	0.003 ppm (mg/L) (CPHA, 1986)
OMOE Guideline Fish:	Unrestricted Consumption: < 2.0 ppm edible portion (UGLCCS, 1988). Fish with 2 ppm or more should be eaten by adults only on an

occasional basis as provided in guidelines, excluding women of child bearing age.

GLWQA Specific Objective: Whole fish: 0.1 ppm (UGLCCS, 1988)

FDA Action Level: 20 ppm, edible portion (UGLCCS, 1988)

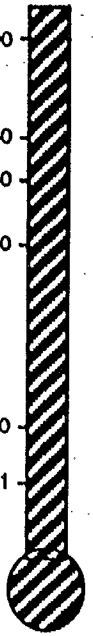
7.1.3 Effects

PCBs are only moderately acutely toxic in laboratory animals. The LD_{50}s for commercial mixtures range from 1 to 20 g/kg bw (Newhook, 1988). However, they cause many effects in laboratory animals, including decreased longevity (100 µg/kg bw/day), hepatotoxicity (105 µg/kg bw/day), developmental toxicity (100 µg/kg bw/day), reproductive toxicity (380 µg/kg bw/day) and cancer (3.45 mg/kg bw/day) (ATSDR, 1987a). The International Agency for Research on Cancer (IARC) has designated PCBs as Group 2A carcinogens (limited human and sufficient animal evidence). However, PCBs are not thought to be genotoxic and so are considered as promoters, rather than initiators of carcinogenesis. There have been several studies on the toxicity of specific congeners (McNulty, 1985; Safe, 1987). The results suggest that the 'coplanar isomers', without mono-ortho chlorine substitution, are the most toxic (McNulty, 1985). In one study, isomers, such as 3,4,3',4'-tetrachlorobiphenyl and 3,4,5,3'4'5'-hexachlorobiphenyl, were the most toxic to monkeys (approximately 50 ng/kg bw/day) (McNulty, 1985).

Occupational exposure to high levels of PCBs has occasionally been associated with skin irritations and liver effects (ATSDR, 1987a). Similar effects resulted from accidental exposures to PCB-contaminated rice oil, but other more severe effects from these exposures have been attributed to dibenzofuran contamination (Safe, 1987). Figure 2 shows the effects mentioned above, as well as the TDI for PCBs.

Several studies have correlated serum PCB levels in humans with biochemical parameters, such as increased plasma triglycerides, increased serum cholesterol and elevated blood pressure (ATSDR, 1987a). In addition, umbilical cord serum levels of PCBs have been associated with reduced birth weights, size of the offspring, shorter gestation, and neonatal behavioral effects (Fein *et al.*, 1984; Jacobson *et al.*, 1984; 1985; Rogan *et al.*, 1986a), although the results of these studies require clarification. However, just because PCBs have been associated with particular effects does not mean that they are the causative agents. These studies are discussed in section 3.2.1 above.

FIGURE 2 SUMMARY OF EFFECTS FOR PCBs



Dose Level (mg/kg bw/day)	Compound, Species and Effect
1,000-20,000	Commercial PCBs, Rats, Lethality
3.450	Aroclor 1260 Fed in Diet for 29 Months, Rats, Liver Cancer
0.380	Aroclor 1254 Fed for 4 Months Prior to Mating and During Gestation, Mink Reproductive Failure and Fetal Deaths
0.100	Aroclor 1248 Fed for 173 or 310 Days, Monkeys, Hepatotoxicity and Decreased Longevity
	Aroclor 1254 Fed for 9 Months, Mink, Decreased Longevity
	Aroclor 1248 Fed for 7 Months Prior to and During Pregnancy, Monkeys, Developmental Toxicity
0.050	3, 4, 3', 4', - Tetrachlorobiphenyl and 3, 4, 5, 3', 4', 5' Hexachlorobiphenyl Fed for 200 Days, Monkeys, Clinical Symptoms
0.001	HWC Tolerable Daily Intake for Total PCB

7.1.4 Exposure

Food, and particularly contaminated fish, are the major sources of exposure for most Canadians and residents of the Great Lakes basin. Estimates of dietary intake of PCBs range from 0.011 µg/kg bw/day for residents of Toronto (Brecher *et al.*, 1989) to 0.16 µg/kg bw/day (Newhook, 1988).

The levels of PCBs found in Great Lakes fish vary from lake to lake and from species to species (see Volume I, Part 2). In some instances, they exceed fish consumption advisories. Fish from Lakes Michigan and Ontario have the highest concentrations of total PCBs. Total PCB concentrations in fish from Lakes Erie, Huron and Superior are generally comparable and less than one-half of those in Lake Ontario fish. In 1988, mean whole fish concentrations in top predator species from Lakes Ontario and Erie were 1 ppm or above. Mean concentrations of total PCBs from dorsal filets of coho salmon collected at the Credit River (the most popular fishing site in the Great Lakes) remained above 1 ppm in 1988. PCBs tend to accumulate in the hepatopancreas of fish, so if residues are expressed on a whole fish basis, consumption may be overestimated (Kimbrough, 1987).

To illustrate the potential human exposure to PCBs from the consumption of contaminated fish one could use the following hypothetical example: If a person consumed a fish meal (114 g once per week) containing 1.0 ppm (µg/g) PCB, then s/he would ingest 114 µg/person/week. Assuming the individual weighed 70 kg, then s/he would ingest 0.23 µg/kg bw/day. This is approximately five times less than the HWC TDI of 1 µg/kg bw/day.

PCBs are rarely detected in Ontario drinking water supplies. In 1986, 1020 samples were analyzed by the Ontario Ministry of the Environment and no PCBs were found at a minimum detection level of 20 ng/L (Brecher *et al.*, 1989). Atmospheric concentrations of PCBs in the Great Lakes region have been reported to be 0.5 ng/m^3 for all of the Great Lakes except Lake Huron, for which there were no data available (IJC, 1988). Although exposure through this medium is low, it will probably continue for a long time because of cycling of PCBs in the environment (ATSDR, 1987a).

There are few data available on PCB levels in human adipose tissue. They show no obvious temporal trends and no differences between residents of the Great Lakes basin and other parts of Canada (Brecher *et al.*, 1989; Williams *et al.*, 1988). Similarly, residues of PCBs in human milk do not show any temporal trends. Concentrations range from 17-33 ppb (µg/kg whole milk) (Brecher *et al.*, 1989). Therefore, breast-feeding infants could consume more than the TDI for PCBs. For example, if breast milk contained 33 ppb (0.033 µg/g) of PCBs, and an infant ingested 130 g of milk per day/kg body weight, then it would ingest 4.29 µg/kg bw/day. This is approximately four times the HWC TDI. However, the TDI of PCBs is based on life-time exposure. Infants would only be exposed to levels higher than the TDI for a much shorter period of time. Also, there is no evidence that levels of PCB currently found in breast milk have caused any adverse effects in nursing infants. In fact, both the Michigan fish-eater study (Jacobson *et al.*, 1985) and the study of neonates in North Carolina (Rogan *et al.*, 1986a) showed that effects in infants were not associated with concentrations of PCBs in breast milk. HWC has established a 'concern level' for PCBs in breast milk at 50 µg/L (NH&W, 1978). In most cases, current health professional advice is that the known benefits of breast feeding outweigh the potential risks that may be associated with PCBs in human milk (NH&W, 1989a).

7.1.5 Summary

PCBs are persistent, ubiquitous environmental contaminants that are still being released to the environment, although they are no longer manufactured and there are restrictions on their use. The main sources of PCBs to the environment are thought to be accidental point source releases and leakage from waste disposal sites. Although they are only moderately acutely toxic, there is concern that long term, low level exposure could cause adverse effects. The main route of exposure to PCBs is the diet, particularly the consumption of fish containing PCB residues. Fish concentrations of PCBs vary from lake to lake and between species. In some instances, they exceed fish consumption advisories. Several individual PCB congeners have been shown to be considerably more toxic than the original commercial formulations. Current research is focusing on the residues and toxicity of specific PCB congeners, but sufficient information is not yet available to permit the development of regulations on a congener-specific basis.

There is an association between maternal consumption of Lake Michigan fish and some measurements of cognitive and motor function of their offspring. More information is needed to confirm these findings and to determine if the effects are causally linked to PCBs. Nursing infants could consume PCBs above the TDI, and studies should be undertaken to determine if such exposures could have adverse effects.

7.2 MIREX

7.2.1 Production and Use

Mirex is a cyclodiene contact insecticide, although it has had several other uses. All uses have been banned in Canada under the Environmental Contaminants Act since 1978 (Canada Gazette, Vol. 12, No. 23, p.43, 1978).

Mirex has never been permitted for use in Canadian agriculture, but between 1963 and 1976 at least 146 tonnes were imported as dechlorane for other applications. Mirex was used extensively in the southeast U.S. (NH&W, 1980), but its use as an insecticide was banned in the U.S. in 1978.

Mirex is primarily a contaminant of Lake Ontario because of industrial discharges to the Niagara and Oswego Rivers (Thomas *et al.*, 1988). It is one of the most stable chemicals known, but it does slowly break down to photomirex (8-monohydromirex), a toxic chemical that contains one less chlorine atom than mirex.

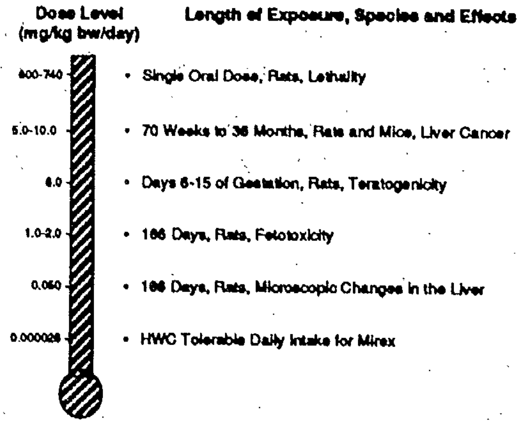
7.2.2 Standards and Guidelines

Canadian Tolerable Daily Intake	0.028 µg/kg bw /day (D. Clegg, 1989)
NH&W Guideline Fish:	0.1 ppm for fish in commerce only (UGLCCS, 1988)
FDA Fish Action Level:	0.1 ppm (LOTMP, 1989)
OMOE Guideline (provisional):	Fish consumption only on an occasional basis according concentrations above 0.1 ppm excluding women and children. Unrestricted consumption below this guideline except for women of child-bearing ages and children under 15 years of age (UCLCCS, 1988).
GLWQA Specific Objective:	Less than detection limit in edible portion of fish wet weight (UGLCCS, 1988).

7.2.3 Effects

Single doses of mirex are moderately acutely toxic in laboratory animals. The acute oral LD_{50} of mirex in rats is 740 mg/kg bw in males and 600 mg/kg bw in females (WHO, 1984a). Mirex causes several effects in laboratory animals, including morphological changes in the liver (0.05 mg/kg bw/day), fetotoxicity (1-2 mg/kg bw/day), teratogenicity (6.0 mg/kg bw/day) and carcinogenicity (5-10 mg/kg bw/day) (WHO, 1984a). Figure 3 shows these effects, as well as details on the length of exposure required to produce them.

FIGURE 3 SUMMARY OF EFFECTS FOR MIREX



There are no data on human health effects of either accidental poisonings or occupational exposure (WHO, 1984a).

Mirex bioaccumulates and biomagnifies in the food chain. It is also extremely persistent because of its resistance to breakdown (WHO, 1984a).

7.2.4 Exposure

The main route of exposure to mirex for residents of the Lake Ontario basin is from the consumption of Lake Ontario fish. Since 1976, the levels in coho salmon and lake trout have decreased, however, in 1988 they were above 0.1 ppm in lake trout (whole fish) and slightly less than 0.1 ppm in coho salmon (dorsal filet) (see Volume I, Part 2). A person who consumed Lake Ontario fish (114 g of fish per meal, once a week) at a concentration of 0.10 ppm (μg/g) would ingest 5.7 μg of mirex per person per week. This is equivalent to 0.022 μg/kg bw/day for a 70 kg person and is close to the TDI established for mirex by Health and Welfare Canada.

Mirex is hardly ever detected in drinking water samples taken from the Great Lakes (NH&W, 1980). Mirex was detected in only five out of 1147 samples taken of Ontario drinking water in 1987. The highest concentration was 5 ng/L (Brecher *et al.*, 1989). Mirex is unlikely to be detectable in air (IJC, 1988).

Adipose tissue levels of mirex in Canadian residents of the Lake Ontario basin vary between 10 and 20 ppb (ng/g) (Brecher *et al.*, 1989). Mirex and photomirex are rarely detected in breast milk samples from Ontario residents (Brecher *et al.*, 1989).

7.2.5 Summary

There are now no permitted uses of mirex in the Great Lakes Basin and it is no longer being commercially formulated. Levels are decreasing in top predator fish species from Lake Ontario. It is unlikely that current levels of mirex in Great Lakes fish are a risk to human health, if fish consumption advisories are followed.

7.3 HEXACHLOROBENZENE

7.3.1 Production and Use

HCB is one of the most persistent and ubiquitous chemicals known. Prior to the mid 1970s, hexachlorobenzene (HCB) was manufactured for use primarily as a fungicide (Morris and Cabral, 1986). Since then, it has not been manufactured in either Canada (Holliday, 1988) or the U.S. (Morris and Cabral, 1986). HCB is, however, generated as a by-product in the synthesis of several industrial chemicals and pesticides in both Canada and the U.S. (Holliday, 1988), as well as during the electrolytic production of chlorine, caustic soda and sodium chlorate. Since 1983, HCB has not been imported into Canada, although restricted fungicidal uses are still permitted (Holliday, 1988).

7.3.2 Standards and Guidelines

WHO Drinking Water Guideline (MAC):	0.01 µg/L (Tentative, CPHA, 1986)
EPA Drinking Water Health Advisory:	Longer-term non-carcinogenic: 0.175 mg/L for (proposed) a 70 kg adult Longer-term non-carcinogenic: 0.050 mg/L for a 10 kg child (U.S. EPA, 1989)

U.S. FDA Fish Action Level:	0.3 ppm (LOTMP, 1989)

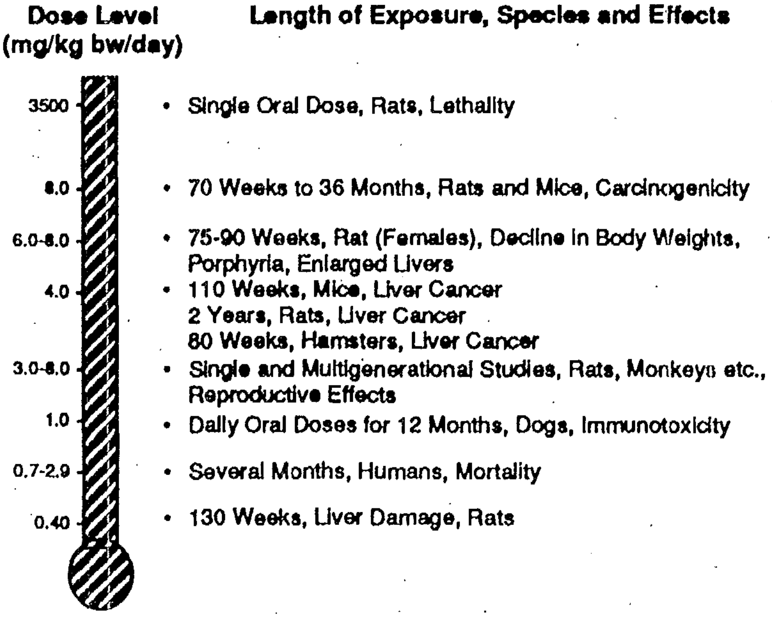
7.3.3 Effects

The acute toxicity of HCB is relatively low in laboratory animals, and the oral LD_{50} for rats has been reported to be 3,500 mg/kg bw (Strik, 1986). Acute intoxication in animals is accompanied by neurotoxic manifestations, such as trembling, ataxia and paralysis (Krewski *et al.*, 1986). HCB has also been shown to cause liver damage (0.4 mg/kg bw/day) (Arnold *et al.*, 1985), decreased body weight gain, porphyria and liver enlargement (8.0 mg/kg bw/day) (Smith and Cabral, 1980), reproductive effects (3-8 mg/kg bw/day) (Kociba, 1986), and immunotoxic effects (1.0 mg/ kg bw/day) (Gralla *et al.*,1977). HCB also produces cancer in mice (6.0 mg/kg bw/day) (Cabral and Shubik, 1986) and rats (4.0 mg/kg bw /day) (Erturk *et al.*, 1986; Arnold *et al.*, 1985; Arnold and Krewski, 1988) and liver tumours in hamsters (4 mg/kg bw/day)(Cabral and Shubik, 1986).

The consumption of HCB-treated seed grain was responsible for a widespread poisoning incident in Turkey in the late 1950s. The effects in adults included dermatological symptoms (porphyria cutanea tarda), enlarged liver and hyperpigmentation (Morris and Cabral, 1986). Pink sores were noted in infants exposed through breast feeding and the exposure was fatal in children under 2-3 years old (Morris and Cabral, 1986). The precise doses that produced these effects were not determined, but it was estimated that extended exposure to 50-200 mg HCB/ person/day produced 10% mortality among the population exposed (Cam and Nigogosyan, 1963; Courtney, 1979). Apart from an epidemiological study of this population, no other human studies have been reported (Morris and Cabral, 1986). Figure 4 summarizes these effects and as well as the length of exposure necessary to produce them.

HCB has become ubiquitous in the environment because of its resistance to degradation and its tendency to accumulate in lipid tissues. It breaks down slowly in water (half-life= 70 days) and air (half-life = 80 days) to produce chlorinated phenols and other chlorinated benzenes (Mill and Haag, 1986).

FIGURE 4 SUMMARY OF EFFECTS FOR HCB


Dose Level (mg/kg bw/day)
Length of Exposure, Species and Effects
3500
• Single Oral Dose, Rats, Lethality
8.0
• 70 Weeks to 36 Months, Rats and Mice, Carcinogenicity
6.0-8.0
• 75-90 Weeks, Rat (Females), Decline in Body Weights, Porphyria, Enlarged Livers
4.0
• 110 Weeks, Mice, Liver Cancer
2 Years, Rats, Liver Cancer
80 Weeks, Hamsters, Liver Cancer
3.0-8.0
• Single and Multigenerational Studies, Rats, Monkeys etc., Reproductive Effects
1.0
• Daily Oral Doses for 12 Months, Dogs, Immunotoxicity
0.7-2.9
• Several Months, Humans, Mortality
0.40
• 130 Weeks, Liver Damage, Rats

7.3.4 Exposure

Food is the major exposure pathway for residents of the Great Lakes basin (and other Canadians) (Holliday, 1988). Total daily intakes of HCB for several age categories have been estimated by Holliday (1988):

Adult (assuming a body weight of 70 kg): 0.002 µg/kg bw/day
Toddler (15 kg bw): 0.0043 µg/kg bw/day
Infant (8 kg bw): 0.0058 µg/kg bw/ day
Breast-fed infant: 0.260 µg/kg bw /day

For the breast-fed infant it was assumed that the concentration of HCB in breast milk was 2 ppb (2 µg/kg whole milk) and that 130 g milk/kg bw/day would be ingested. Residue levels in human breast milk from Ontario residents range from 0.26 ppb (µg/kg whole milk) to 2.0 ppb (Brecher *et al.*, 1989) for 1975-1985. There were no consistent trends over this time period (see Table 3.3 of Volume I, Part 4). However, residues do occur in nearly all samples (Brecher *et al.*, 1989).

Consumption of Great Lakes fish contributes to exposure through food, although HCB is not often reported as a contaminant in fish and in 1984

Lake Michigan coho salmon contained less than 0.05 ppm (WQB, 1989a). Concentrations in U.S. lake trout were also reported to range from 0.01-0.11 ppm. If a 70 kg adult consumed 114 g fish containing HCB at 0.10 ppm, then s/he would consume 11.4 µg HCB/person/week or 0.023 µg HCB/kg bw/day. This is approximately 2000 times less than the lowest dose known to produce toxic effects in laboratory animals (Figure 4).

Although HCB is found throughout the Great Lakes basin in ambient water and sediments (Part 1, Volume I), it is hardly ever detected in drinking water (Holiday, 1988) and is unlikely to contribute significantly to total exposure. For instance, in 1986 HCB was detected in only two of 1147 samples of Ontario drinking water. The highest concentration was 1 ng/L (Brecher *et al.*, 1989). Although there are no precise data on HCB concentrations in ambient air in the Great Lakes basin, levels are not expected to exceed 0.1 ng/m^3 and therefore would only contribute a maximum of 1-2% of the total body burden (Holliday, 1988).

In 1984, HCB levels in adipose tissue samples from residents of the Great Lakes basin ranged from 71 to 114 ppb (ng/g) (Williams *et al.*, 1984). These values are similar to those from other parts of Canada and are generally lower than those in residents of western Europe (Holliday, 1988). An estimated 98.8% of U.S. residents carry detectable levels of HCB in their adipose tissue (Robinson *et al.*, 1986).

7.3.5 Summary

HCB is one of the most persistent and ubiquitous chemicals known. Although it is no longer manufactured in Canada or the U.S. for use, it is generated in significant quantities as a by-product. Ingestion by the general population appears to be quite low. Limited data on residues in fish show that levels are generally below 0.1 ppm. If average amounts of such fish are consumed there is a considerable margin of safety in relation to the levels known to cause adverse health effects in laboratory animals.

7.4 DIELDRIN

7.4.1 Production and Use

Dieldrin is a persistent cyclodiene insecticide which was used in Canada and U.S. for control of soil insects and mosquitos and for mothproofing (ATSDR, 1987b). It is also produced in the environment and in biota by the metabolic oxidation of aldrin. Because of this close relationship, aldrin and dieldrin are usually considered as a single chemical (ATSDR, 1987b). Most uses of aldrin and dieldrin were banned in the U.S. in 1975 (ATSDR, 1987b). In Canada, all food uses for aldrin and dieldrin were prohibited in 1978 and only limited applications are now permitted (Bennett, 1989).

7.4.2 Standards and Guidelines

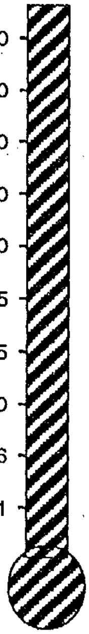
WHO Guidelines:	Acceptable Daily Intake (ADI): 0.1 µg/kg bw Food (maximum residue): 0.02-0.2 mg/kg Drinking water: 0.03 µg/L (ATSDR, 1987b)
FDA Fish Standard:	0.3 ppm (µg/g) (LOTMP, 1989)
Canada Drinking Water Guideline (MAC):	0.0007 ppm (mg/L) (CPHA, 1986)
Ontario Drinking Water Guideline (MAC):	0.0007 ppm (mg/L) (CPHA, 1986)
GLWQA Specific Objective:	Ambient Water: 0.001 ppb (µg/L) (UGLCCS, 1988) Fish: 0.3 ppm edible portions wet weight (UGLCCS, 1988)

7.4.3 Effects

Single doses of dieldrin have a high acute toxicity in laboratory animals and the acute oral LD_{50} in rats has been reported to be 46 mg/kg bw (ATSDR, 1987b). Dieldrin causes several effects in experimental animals including decreased longevity in mice, rats and dogs (the effect level for monkeys, the most sensitive species for this endpoint, is 0.105 mg/kg bw/day) (ATSDR, 1987b). The most sensitive target organ in chronic studies is the liver (0.05 mg/kg bw/day). Dieldrin also produces developmental effects (fetotoxicity) in mice and rats at 6.0 mg/kg bw/day (ATSDR, 1987b). Reproductive toxicity has been observed in rats at 0.016 mg/kg bw/day (ATSDR, 1987b). Dieldrin is also carcinogenic in mice at 19 mg/kg bw/day (ATSDR, 1987b), but the International Agency for Research on Cancer (IARC) has concluded that evidence for its carcinogenicity in humans is "inadequate" (IARC, 1987). Dieldrin also causes several other effects, such as neurotoxicity (5.0 mg/kg bw/day) and immunotoxicity (0.75 mg/kg bw/day) (ATSDR, 1987b).

Hayes (1982) has estimated that the lethal single oral dose for humans is probably between 30 and 100 mg/kg bw. The main target organ in acute poisoning (> 5 mg/kg bw/day) is the nervous system (ATSDR, 1987b). Information on the effects of long-term human exposure is limited and inconclusive (ATSDR, 1987b). Figure 5 shows the effects mentioned above, as well as the route and length of exposure necessary to produce them.

FIGURE 5 SUMMARY OF EFFECTS FOR DIELDRIN



Dose Level (mg/kg bw/day)	Length of Exposure, Species and Effects
30-100	Estimated Single Oral dose, Humans Lethality
46.0	Single Oral Dose, Rats, Lethality
19.0	Lifetime Oral Dose, Mice, Liver Cancer
6.0	Oral Dose Days 6-16 of Gestation, Rats Fetotoxicity
5.0	Oral Dose 8 Weeks, Rats Neurotoxicity
0.75	Oral Dose 6-10 Weeks, Mice, Immuntoxicity
0.105	Lifetime Feeding, Monkeys, Decreased Longevity
0.050	2 Year Feeding Study, Rats, Liver Toxicity
0.016	300 Days Feeding, Rat, Reproductive Toxicity
0.0001	WHO Acceptable Daily Intake

7.4.4 Exposure

Food is likely to be the major exposure pathway to dieldrin. The total dietary intake has been estimated to be 0.002 µg/person/day in a Canadian total diet study conducted from 1976 to 1978 (McLeod *et al.*, 1980). This is consistent with other data (Brecher *et al.*, 1989) and is well below the ADI of 0.1 µg/kg (see above).

Fish consumption may contribute to exposure, although residues in most fish are below 0.2 ppm (see Volume I, and WQB, 1989a). Assuming a worst case concentration of 0.3 ppm (the FDA fish guideline), a person consuming 114 g once per week would ingest 34.2 µg of dieldrin per week. This is equivalent to 0.069 µg/kg /day for a 70 kg individual. This is below the WHO ADI.

Drinking water is not a significant route of exposure (ATSDR, 1987b). It was not detected in any drinking water samples analyzed by OMOE for the Ontario 1987 drinking water surveillance program (Lachmaniuk, 1989). Atmospheric exposure is not thought to be a significant pathway (ATSDR, 1987b).

In the early 1970s, levels of dieldrin in the environment and in humans decreased, but more recently they have levelled out (WQB, 1985; Brecher *et al.*, 1989). Samples taken in 1984 indicate that residents of the Great Lakes basin have adipose tissue levels ranging from 7 to 63 ppb (Brecher *et al.*, 1989). Data from Mes *et al.* (1982), suggest that dieldrin concentrations in human adipose tissue decreased significantly between 1969 and 1976 and that levels in Ontario residents were no different from those in residents from other parts of Canada. Dieldrin levels in human breast milk for 1985 were reported to be <0.1 ppb. This compares to a Canadian average of 0.4 ppb for 1987 (Brecher *et al.*, 1989).

7.4.5 Summary

Dieldrin is no longer manufactured in Canada or the U.S. and most of its uses have been cancelled. Although levels in the environment and in humans decreased significantly in the 1970s, since then concentrations have levelled off. Dieldrin levels in the Great Lakes are unlikely to be a risk to human health, providing that fish consumption advisories are followed.

7.5 DDT

7.5.1 Production and Use

DDT was developed in the late 1930s and it was subsequently used extensively as a broad spectrum contact insecticide. Its use peaked in the early 1960s, and in 1963 176 million pounds were produced in the U.S. (NRC, 1977). In 1973, DDT was banned in the U.S. except for essential public health uses. In the early 1970s, most uses of DDT were banned in Canada, but the last remaining product registration was only discontinued in 1989.

Although DDT is relatively stable, it undergoes a relatively complex series of biological degradative changes. The most important of these is dehydrochlorination to DDE. DDE is more lipid-soluble and much less toxic to higher animals than DDT (NRC, 1977), however it is almost non-degradable biologically and hence is the predominant residue stored in tissues.

7.5.2 Standards and Guidelines

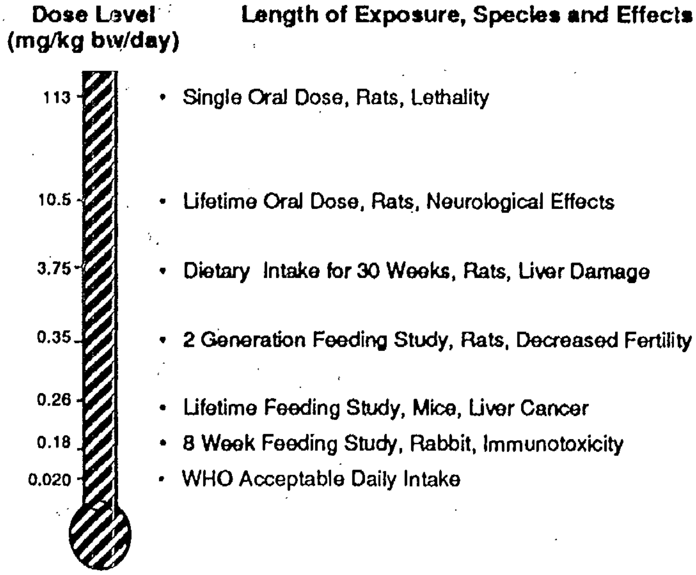
WHO Acceptable Daily Intake from food:	0.02 mg/kg bw/d (FAO/WHO, 1984)
NH&W Fish Maximum Residue Limit:	5.0 ppm (DDT + Metabolites) (Food and Drug Regulations, Table II).
FDA Fish Action Level:	5.0 ppm (LOTMP, 1989)
OMOE Fish Guideline:	5.0 ppm or more only on an occasional consumptin basis by adults; women of childbearing age and children under 15 should refrain from eating (OMOE, 1989).
Canada Drinking Water Guideline (MAC):	DDT (Total) 0.03 ppm (mg/L) (CPHA 1986)
Ontario Drinking Water Guideline (MAC):	DDT Total) 0.03 ppm (mg/L) (CPHA, 1986)
WHO Drinking Water Guideline:	DDT (Total) 1.0 mg/L (CPHA, 1986)

7.5.3 Effects

The acute oral LD_{50} for DDT in rats is 113 mg/kg in males and 118 mg/kg in females (NRC, 1977). DDT produces several effects in laboratory animals, including hepatic effects (3.75 mg/kg bw/day ATSDR, 1988a p.26), immunological effects (0.18 mg/kg bw/day), neurological effects (10.5 mg/kg bw/day ATSDR, 1988a), effects on reproduction (0.35 mg/kg bw/day ATSDR, 1988a) and cancer (0.26 mg/kg bw/day ATSDR, 1988a).

Acute DDT poisoning in humans is rare, considering its widespread use. A single oral dose of 10 mg/kg bw produced effects but was not fatal in humans (NRC, 1977). There is no evidence of any adverse long-term effects resulting from small daily doses of DDT (CEC, 1981). A recent study (Rogan *et al.*, 1986a) has indicated an association between DDE and PCBs in breast milk at birth and hyporeflexia in the neonate. These effects were associated with relatively high concentrations of DDE (> 4.0 ppm [µg/g] milk fat) and PCBs (> 3.5 ppm milk fat) which are much higher than levels currently being reported for breast milk from Ontario residents (i.e., approximately 1.0 ppm - assuming that whole milk contains 3% fat) (Brecher *et al.*, 1989). Figure 6 shows these effects, as well as the route and length of exposure necessary to produce them.

FIGURE 6 SUMMARY OF EFFECTS FOR DDT



7.5.4 Exposure

Food is the major human exposure pathway to DDT and DDE. In 1979, after the use of DDT had peaked (ATSDR, 1988a), the average worldwide ingestion was estimated to be less than 50 µg/person/day (WHO, 1979). This is well below the FAO/WHO ADI of 1400 µg/person/day for a 70 kg individual (NRC, 1977). A Canadian survey for 1976-78 estimated the total dietary intake of DDT to be 1.4 µg/person/day (McLeod *et al.*, 1980).

Fish consumption is likely to contribute to exposure. In 1988, levels of total DDT in four-year old lake trout from Lake Ontario were 1 ppm and lower in Lakes Huron and Superior (see Volume I). If a person consumed 114 g of fish containing 1 ppm of DDT once per week, s/he would ingest 114 µg DDT per week. Assuming the individual weighed 70 kg, then the intake of DDT would be approximately 0.23 µg/kg bw/day. This is less than the WHO ADI of 300 µg/person/day mentioned above.

Drinking water is unlikely to be a major route of exposure for residents of the Great Lakes basin because DDT and DDE are rarely detected in treated Ontario drinking water (Brecher *et al.*, 1989). Air is also unlikely to be a major route of exposure (WHO, 1979), even though long range atmospheric transportation of these chemicals occurs and results in residues in biota which were not directly exposed.

Table 3.3 of Volume I, Part 4, shows DDT residues in breast milk from Ontario residents for 1967 to 1985. The relatively high concentration for 1967 may be an overestimate because of limitations in the analytical methods used at the time and also the possibility that PCBs interfered in the analysis. However, it is clear that concentrations of DDT in breast milk are not increasing and that a baseline level has been reached.

Analysis of human adipose tissue samples from accident victims in 1984 from six Great Lakes municipalities showed that the average concentration was approximately 3 ppm and similar to those in residents of other parts of Canada (Williams *et al.*, 1988).

7.5.5 Summary

Since most uses of DDT were banned in the early 1970s, levels of this chemical and its principal metabolite DDE have decreased significantly in the environment. However, more recently DDE levels have levelled off. This could be due to several causes including long-range atmospheric transportation of DDT from countries still using DDT. The levels of DDT and DDE currently detected in the Great Lakes basin are not likely to be a risk to human health. Studies that have associated high breast milk levels of DDE with hyporeflexia in neonates should be substantiated to determine if DDE is the causative factor.

7.6 2,3,7,8-TCDD

7.6.1 Production and Use

2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) is one of 75 dioxin congeners. It is considered to be the most toxic. 2,3,7,8-TCDD is not manufactured commercially, but is an unwanted byproduct of some chemical manufacturing processes (such as the production of several chlorophenols and phenoxy herbicides) and some industrial practices (some Kraft pulp and paper industries and smelters). It can also be produced during combustion and has been detected in emissions from incinerators and in exhaust from vehicles using leaded fuels. The Ontario Ministry of the Environment has a program to reduce or eliminate the formation of all dioxins and furans by altering industrial practices (OMAF & OMOE, 1988). 2,3,7,8-TCDD is a persistent, fat soluble contaminant that bioaccumulates in food chains. It is resistant to degradation and has a half life in humans of between 5 and 7 years (Birmingham *et al.*, 1989).

7.6.2 Standards and Guidelines

Canada (and Ontario) Tolerable Exposure Guideline:	10 pg 2,3,7,8-TCDD toxic equivalents/ kg bw/day (Birmingham *et al.*, 1989)
Canadian Food Tolerance:	20 ppt (ng/kg) for fish in commerce based on one meal per week of 114 g (Food and Drug Regulations, 1989)
Ontario Sport Fish Consumption Advisory:	20 ppt (20 ng/kg fish)(LOTMP, 1989)

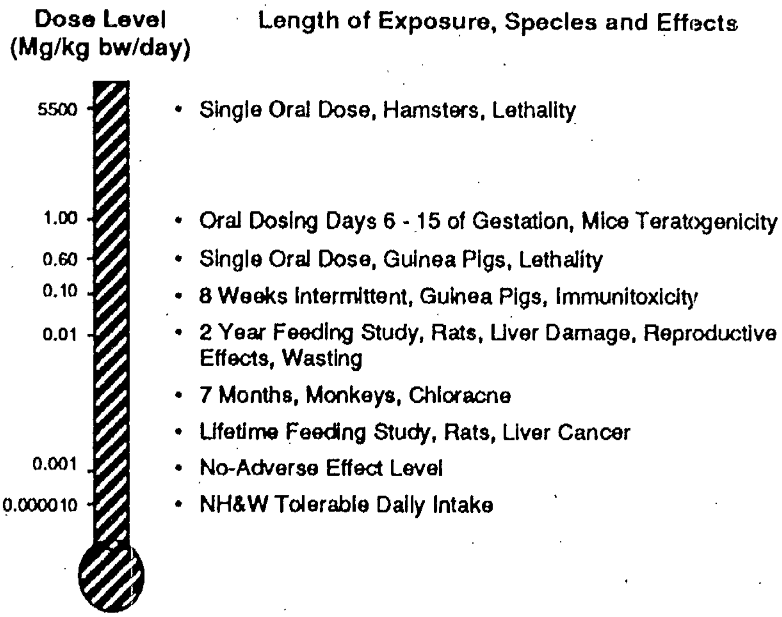
U.S. FDA Fish Advisory:	No serious concerns if levels in fish are less than 25 ppt (ATSDR, 1987c)
Ontario Ambient Air Guideline (Interim)	30 pg/m^3 air as an annual average (MMGAC, 1989)
Ontario Drinking Water Guideline (MAC):	15 ppq (15 pg/L) in 2,3,7,8-TCDD toxic equivalents (Birmingham *et al.*, 1989).
GLWQA Specific Objective:	10 ppq (pg/L) for ambient water. 10 ppt (ng/kg) for sediment or tissue or aquatic organisms (GLWQA, 1988).
U.S. EPA Drinking Water Advisories:	1 ppt (ng/L), one day advisory for a child. 0.1 ppt, ten day advisory for a child. 10 ppq (pg/L), long term advisory for a child. 35 ppq, long term advisory for an adult (ATSDR, 1987c).

Note: Many guidelines are expressed as 2,3,7,8-TCDD toxic equivalents to take account of simultaneous exposure to other dioxin and furan congeners. Since 2,3,7,8-TCDD is considered the most toxic congener, other congeners are assessed by comparing their toxicity to 2,3,7,8-TCDD using an internationally accepted approach and expressing their relative contribution in toxic equivalents of 2,3,7,8-TCDD.

7.6.3 Effects

2,3,7,8-TCDD is extremely toxic, but the response in different species is different. Guinea pigs are thought to be most sensitive in terms of acute toxicity after a single oral dose (LD_{50} 0.6 μg/kg bw), and hamsters the least sensitive (LD_{50} 5500 μg/kg bw) (ATSDR, 1987c). Short term exposures in laboratory animals cause chloracne, losses in body weight, thymic atrophy and death 3-4 weeks after exposure (ATSDR, 1987c). Longer term exposures have resulted in liver damage (0.01 μg/kg bw/day), weight loss (wasting syndrome) (0.01 μg/kg bw/day), effects on reproduction (0.01 μg/kg bw/day)(Murray *et al.*, 1979) and the immune system (0.1 μg/kg bw/day) and occasionally teratogenic effects (1 µg/kg bw/day) (ATSDR, 1987c). Most of these effects and other symptoms, such as skin disorders, loss of hair and swelling of the eyelids, have been observed in exposed monkeys (ATSDR, 1987c). Lifetime exposures in rats and mice have caused neoplastic lesions including carcinomas in the liver and other organs (0.01 µg/kg bw/day) (ATSDR, 1987c). 2,3,7,8-TCDD is not genotoxic and is thought to act as a cancer promoter, rather than as an initiator (Birmingham *et al.*, 1989). The International Agency for Research on Cancer (IARC) has designated 2,3,7,8-TCDD as a 2B carcinogen. There is general agreement that the no-observed-adverse-effect-level (NOAEL) for chronic exposure to 2,3,7,8-TCDD is 1 ng/kg bw /day (Birmingham *et al.*, 1989). This lifetime dose is based upon no adverse reproductive outcomes in monkeys and rats and no hyperplastic lesions in rats.

FIGURE 7 SUMMARY OF EFFECTS FOR 2,3,7,8-TCDD



Accidental exposures of human populations to 2,3,7,8-TCDD have resulted in chloracne, but have not been fatal or led to a detectable increase in cancer rates (ATSDR, 1987c). This indicates that humans may not be as sensitive to 2,3,7,8-TCDD as some animal species. Figure 7 shows these effects as well as the exposures necessary to cause them.

7.6.4 Exposure

2,3,7,8-TCDD is found throughout the Great Lakes ecosystem. Levels in fish and wildlife have decreased since the early 1970s, but have now stabilized. 2,3,7,8-TCDD is highly lipophilic and concentrates in fatty foods. A recent study conducted by the province of Ontario concluded that more than 90% of human exposure is from food and that 99% of this comes from animal products (OMAF & OMOE, 1988). This includes fish.

Levels of total dioxins are highest in lake trout from Lake Ontario, although there are elevated concentrations in other parts of Lakes Michigan and Huron. TCDD levels in four-year-old Lake Ontario lake trout (Part 2 of Volume I) show that levels have fluctuated between 10 and 45 ppt between 1977 and 1987. Recent data presented by Niimi and Oliver (1989) for Lake Ontario salmonids show that total dioxin congener levels ranged from 46 to 290 ppt in whole fish and from 60 to 366 ppt in muscle composite samples. Levels of the 2,3,7,8-TCDD congener in whole fish ranged from 6 to 20 ppt.

As an example of the contribution of consuming fish from the Great Lakes contaminated with 2,3,7,8-TCDD, one could use the following hypothetical situation: if an individual consumed 114 g per week of fish containing 10 ppt (pg/g tissue) 2,3,7,8-TCDD, then s/he would ingest 1140 pg, or 1.14 ng of the chemical. If the person weighed 70 kg, then on a daily basis the ingestion would be 2.33 pg/kg bw/day. This is approximately five times less than the TDI.

Air contributes approximately 10% of average daily exposure and soil; drinking water and consumer products contribute less than 1%. A recent Canadian estimate of average lifetime adult exposure from all sources is between 2 and 4 pg 2,3,7,8-TCDD toxic equivalents/kg bw/day (Birmingham *et al.*, 1989).

Levels of 2,3,7,8-TCDD in human adipose tissue in the U.S. and Canada range from non-detectable to 20 ppt, with a mean of between 5 and 7 ppt (Colborn, 1989). A pooled breast milk sample (44 donors) collected in 1986-87 from residents of Toronto and southwest Ontario contained 0.09 ppt of 2,3,7,8-TCDD on a whole milk basis (Conacher, 1988). This concentration is slightly lower than the national average (0.10 ppt) for the same time period. These levels are also similar to concentrations found in populations from other industrialized countries, such as Sweden and the U.S. (Conacher, 1988). Using data on human residue levels of 2,3,7,8-TCDD and an estimated half-life of five years, it has been calculated that current adult exposures are likely to be less than 0.5 pg/kg bw/day (Birmingham *et al.*, 1989). Estimated exposure to all dioxins and furans has been estimated to be 3 pg/kg bw/day on the basis of 2,3,7,8-TCDD toxic equivalents (Birmingham *et al.*, 1989).

7.6.5 Summary

2,3,7,8-TCDD is an extremely toxic chemical. It accumulates in the environment because of its persistence and lipophilicity. Current Canadian exposures to 2,3,7,8-TCDD and to total dioxins and furans are below the TDI established by Health and Welfare Canada. Accidental exposures of human populations to concentrations of 2,3,7,8-TCDD have resulted in chloracne but have not been fatal or resulted in an increased risk of cancer. Nevertheless, human exposure to all dioxins and furans should be kept as low as possible, because of the effects of long-term exposure reported in monkeys and rats. Efforts to control sources of dioxins and furans should continue.

7.7 2,3,7,8-TCDF

7.7.1 Production and Use

2,3,7,8-tetrachlorodibenzofuran (2,3,7,8-TCDF) is one of 135 dibenzofuran congeners. Its structure is very similar to that of 2,3,7,8-TCDD. The only difference is the presence of a single oxygen atom in the furan compared to two oxygen atoms in the dioxin molecule. Like the polychlorinated dibenzodioxins (PCDDs), PCDFs were not intentionally manufactured or used commercially. They are formed during the manufacture of several chlorophenols, chlorophenoxy herbicides, during the bleaching of some pulp fibres and during incineration (OMAF & OMOE, 1988; Birmingham *et al.*, 1989). 2,3,7,8-TCDF is highly persistent, lipophilic and bioaccumulates in animal tissues. (NRCC, 1984).

7.7.2 Standards and Guidelines

In Canada, 2,3,7,8-TCDF is controlled using the standards or guidelines for PCDDs and PCDFs, expressed as toxic equivalents of 2,3,7,8-TCDD. A toxic equivalency factor is a number assigned to an individual PCDF (or PCDD) congener that indicates its potential toxicity relative to 2,3,7,8-TCDD (the most toxic dioxin congener known). For example, 2,3,7,8-

PCDF is approximately one-tenth as toxic as 2,3,7,8-TCDD and its toxic equivalency factor is 0.1. These factors can be used as multipliers of concentrations of several individual congeners to estimate the overall toxicity of a mixture relative to 2,3,7,8-TCDD. This internationally accepted approach takes account of human exposure to the full range of 2,3,7,8- substituted PCDD and PCDF congeners in the environment. Current standards and guidelines are shown in the equivalent section for 2,3,7,8-TCDD.

7.7.3 Effects

2,3,7,8-TCDF has similar effects as 2,3,7,8-TCDD, but it is approximately ten times less toxic. The acute oral toxicity for 2,3,7,8-TCDF in guinea pigs is 7 µg/kg bw (OMAF & OMOE, 1988). LD_{50}s for monkeys, rats and mice are estimated to be 300-1000, > 1000, and > 6000 µg/kg bw respectively (OMAF & OMOE, 1988). 2,3,7,8-TCDF has been shown to be teratogenic in mice at a dose of 30 µg/kg bw administered on days 10-13 of gestation (OMAF & OMOE, 1988). PCDFs have not yet been tested for their carcinogenic effects in laboratory animals. However their structural similarity to dioxins and the ability of some 2,3,7,8- substituted PCDF congeners to induce hepatic mixed function enzymes suggests that they could be cancer promoters. The effects observed as a result of the consumption of PCB-contaminated rice oil (Yusho in Japan, 1968 and Yu-cheng in Taiwan, 1979) suggest that the causative agent could have been PCDFs (U.S. EPA, 1986; NRCC, 1984).

7.7.4 Exposure

2,3,7,8-TCDF is found ubiquitously in the environment (Birmingham *et al.*, 1989). The most important exposure pathway for humans is food, because of its lipophilicity and its propensity to bioaccumulate in animals (Birmingham *et al.*, 1989). There is only limited information on residues of 2,3,7,8-TCDF in Great Lakes fish. Data from Niimi and Oliver (1989) show that concentrations in salmonids from Lake Ontario range from 11 to 20 ppt on a whole fish basis, and from 6 to 8 ppt in muscle composite samples. If a person weighing 70 kg consumed 114 g of fish per week that contained 10 ppt of TCDF, then s/he would ingest 2.3 pg/kg bw/day of TCDF. Since the Toxicity Equivalency Factor for 2,3,7,8-TCDF is 0.1 (Birmingham *et al.*, 1989), then s/he would be exposed to 0.23 pg (2,3,7,8-TCDD toxicity equivalents)/kg bw/day, or approximately 1/50 of the TDI for total dioxins and furans.

Current levels of PCDFs in human adipose tissue range from 10 to 40 ppt. There are no discernible differences in samples from residents of different locations in Canada (Brecher *et al.*, 1989). Mean PCDF concentrations in 1981 samples of human breast milk from Canadians were 0.16 ppt (2,3,7,8-TCDF) and 0.44 ppt 2,3,4,7,8-PCDF (whole milk basis) (Brecher *et al.*, 1989). In 1986-87, average levels for Canadians were 0.16 ppt (2,3,7,8-TCDF) and 0.31 ppt (2,3,4,7,8-PCDF) on a whole milk basis (Conacher, 1988). A pooled breast milk sample from 44 residents of Toronto and southwest Ontario for 1986-87 had 0.07 ppt of 2,3,7,8-TCDF and 0.36 ppt of 2,3,4,7,8-TCDF on a whole milk basis (Conacher, 1988).

Exposure to total dioxins and furans has been estimated to be 3 pg/kg bw/day expressed as 2,3,7,8-TCDD toxic equivalents (Birmingham *et al.*, 1989).

7.7.5 Summary

2,3,7,8-substituted PCDFs are believed to have been the causative agent in at least two severe poisoning events of human populations exposed to PCBs. In general, 2,3,7,8-TCDF is 10 times less toxic than the most toxic dioxin isomer (2,3,7,8-TCDD). That they are structurally similar to dioxins and also possess similar physical, chemical and toxicological properties suggests they are of as much concern to human health as PCDDs. Currently human exposures to 2,3,7,8-TCDF and TCDD are below the Tolerable Daily Intake for total dioxins and furans.

7.8 BENZO(a)PYRENE

7.8.1 Production and Use

Benzo[a]pyrene (B[a]P) is a polynuclear aromatic hydrocarbon (PAH). It is formed from the incomplete combustion of fossil fuels, organic matter and garbage. It is also a component of many petroleum products, creosote, asphalt, cigarette smoke and vehicle exhaust (ATSDR, 1987d). Forest fires and residential wood combustion are thought to be major sources of B[a]P to urban and rural environments, respectively (Withey, 1989). It was selected as a Critical Pollutant not only because of its toxicity and presence in the environment, but also because it is a surrogate for several structurally similar PAHs that also occur in the environment.

7.8.2 Standards and Guidelines

Canadian Drinking Water Guideline (MAC):	0.01 µg/L; limit applies to the sum of all forms of each substance (PAH) present (NH&W, 1987a)
WHO Drinking Water Guideline:	0.01 µg/L (WHO, 1984b)
EPA Advisory Levels:	EPA recommended concentration for ambient water: zero. Realizing zero may be impossible to achieve, it provides an Ambient Water Quality Criterion described below (ATSDR, 1987d).
EPA Ambient Water Quality Criterion:	Total carcinogenic PAHs: 28, 2.8 and 0.28 ng/L for an estimated upper-bound lifetime excess risk at 10^{-5}, 10^{-6}, and 10^{-7} respectively (ATSDR, 1987d).

7.8.3 Effects

There are only limited data available on non-carcinogenic effects of B[a]P in laboratory animals and virtually no data on its short and long term toxicity in humans (ATSDR, 1987d). B[a]P is known to decrease longevity when fed to some strains of mice at 120 mg /kg bw/day for 6 months (ATSDR, 1987d). It also caused reproductive problems in mice when administered orally from days 7 to 16 of gestation (10 mg/kg bw/day) (ATSDR, 1987d). Respiratory tract tumours have been observed in hamsters breathing 9.5 mg/m^3 /day of B[a]P over their lifetime. In addition, forestomach tumours have been observed in mice fed 5.2 mg B[a]P/kg bw/day for 110 days and skin tumours have been reported in mice which had 0.05 mg/kg bw/day applied to their skin over their lifetime (ATSDR, 1987d).

7.8.4 Exposure

In the Great Lakes, B[a]P residues are generally associated with sediments (IJC, 1988). This is not unexpected since they have a "high propensity to bind to organic matter" (ATSDR, 1987d).

Human exposure to B[a]P in particular and to the PAHs in general occurs through drinking water, air and food. WHO (1984b) indicated that 99% of exposure is from food, while drinking water and air contribute approximately 0.1-0.3% and 0.9%, respectively. Smoking is a major route of exposure to B[a]P. Exposure can occur from direct inhalation or inhalation of sidestream smoke (ATSDR, 1987d). Data for 1986 confirm that B[a]P is rarely detected in drinking water in Ontario. B[a]P was detected in only two out of 271 samples. The highest concentration was 5 ng/L (Brecher *et al.*, 1989). One of the reasons why PAHs are rarely detected in drinking water is that they are removed by conventional treatment methods, i.e., coagulation and filtration (WHO, 1984b). B[a]P has been detected in both rural and urban air, and the levels in urban air are typically 10-100 times greater than those in rural air (ATSDR, 1987d). The concentration of B[a]P in the air over Lake Michigan has been reported to be 0.1 ng/m^3 and the concentration in air over Lake Superior has been reported to be 0.02 ng/m^3 (IJC, 1988). Very little data are available on B[a]P concentrations in fish in the Great Lakes. Concentrations of B[a]P in carp and pike from Hamilton Harbour have been reported as 51 and 29 ppt, respectively. In contrast, the same species in the Detroit River contained 0.1 and 17.3 ppt (see Volume I, Part 2).

The estimated exposure from consuming 114 g of fish containing 50 ppt once per week would be 5.7 ng/person/week or for a person who weighed 70 kg, 11.6 pg/kg bw/day. Exposure from this source would not be significant unless the fish were smoked (Withey, 1986). Exposure to B[a]P has been estimated to be 0.5 -1.7 µg/person/day for non-smoking adults and 1.5-2.7 µg/person/day for adults who smoke (Canviro, 1985). For a 70 kg individual this is equivalent to 7.1-24.3 ng/kg bw/day for non-smokers and 21.4-38.6 ng/kg bw/day for smokers. These levels are more than 1000 times smaller than those reported to cause cancer in laboratory animals.

7.8.5 Summary

B[a]P is one of several PAHs that are found in the Great Lakes ecosystem. Excluding the smoking of tobacco products, the most important human exposure pathway is food. Exposures through air and drinking water are minor. Consumption of Great Lakes fish is not expected to contribute significantly to dietary intake, unless the fish are smoked. The levels of B[a]P currently found in the Great Lakes are not thought to be any different than those elsewhere in North America and are unlikely to be risk to human health.

7.9 ALKYLATED LEAD

7.9.1 Production and Use

Alkyl lead compounds (particularly tetraethyl lead) are used mainly as gasoline additives. There is only one production facility in Canada and this is in Sarnia. The IJC included alkyl lead on its list of Critical Pollutants because residues have been detected in fish from the St. Lawrence and St. Clair Rivers, and there was concern that consumption of these fish could cause human health effects. Although there are no figures available for Canada, it is thought that the production of alkyl leads has decreased and will continue to decrease because of regulations that restrict the amount of lead in gasoline. This is also true in the U.S., where the current allowable lead content of leaded gasoline is 0.1 g/gallon (ATSDR, 1988b). Data from 1984 suggest that the manufacture of alkyl lead contributes approximately 0.6% to total U.S. lead emissions. However, it is not clear how much of this is released in the alkyl lead form (ATSDR, 1988b). Tetraethyl lead undergoes photolysis in water and in the atmosphere to triethyl lead. This may be more persistent than tetraethyl lead in the environment (ATSDR, 1988b). Tetraethyl lead has also been shown to bioaccumulate in aquatic organisms.

7.9.2 Standards and Guidelines

The only guideline for alkyl lead compounds is a tentative one in Ontario which restricts the consumption of fish containing more than 1.0 ppm (mg/kg tissue) (UGLCCS, 1988).

The relevant guidelines for 'total lead' are listed below:

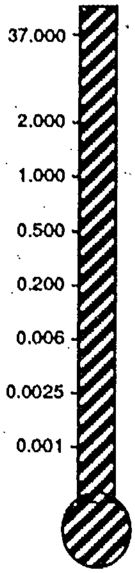
WHO ADI (for adults):	0.006 mg/kg bw/day (WHO, 1977)
WHO ADI (for infants and children):	0.0025 mg/kg bw/day (WHO, 1987)

Canadian Drinking Water Guideline:	50 ppb (µg/L) (CPHA, 1986)
Ontario Drinking Water Guideline:	50 ppb (CPHA, 1986)
WHO Drinking Water Guideline:	50 ppb (CPHA, 1986)
GLWQA Specific Objective (Ambient Water):	10 ppb (µg/L) for Lake Superior, 20 ppb in Lake Huron, and 25 ppb in the other Great Lakes (GLQWA, 1988,)
EPA Ambient Air Criteria:	Air shall not exceed 1.5 µg/m^3 averaged over a year
EPA Drinking Water: Regulations (NPDWRs)	50 ppb ("Proposed" National Primary Drinking Water 0.05 mg/L (ATSDR, 1988b).
OMOE Fish Guideline:	Unrestricted consumption: less than 1.0 ppm, (UGLCCS,1988)

7.9.3 Effects

The metabolism and toxicity of alkylated lead has not been extensively studied in humans or laboratory animals. The limited number of studies available indicate that its toxicity is qualitatively and quantitatively different from the toxicity of inorganic lead (ATSDR, 1988b). Alkyl lead compounds are rapidly and extensively absorbed through the oral and dermal routes (ATSDR, 1988b). Tetraethyl lead and tetramethyl lead are converted to triethyl- and trimethyl- compounds in laboratory animals, and in some species to diethyl- metabolites and probably to inorganic lead (ATSDR, 1988b). Data from laboratory animals shows that single doses of tetraethyl lead are at least 20 times more toxic than equivalent doses of some inorganic forms (ATSDR, 1988b). Repeated daily oral doses of tetraethyl and triethyl lead have been shown to cause symptoms of poisoning at 2.0 mg/kg bw/day after 45 and 21 days of dosing, respectively. In contrast, animals dosed with lead acetate at 200 mg/kg bw/day exhibited no symptoms after 90 days (Franklin *et al.*, 1987). The results of a subchronic study in rats dosed orally with triethyl lead on a daily basis for 91 days indicated that hematopoietic effects occurred at 1.0 mg/kg bw/day, morphological changes in the kidneys at 0.2 mg/kg bw/day and neurotoxic changes at 0.5 mg/kg bw/day (Yagminas *et al.*, 1989; Little *et al.*, 1988).

FIGURE 8 SUMMARY OF EFFECTS FOR ALKYLATED LEAD



Dose Level (mg/kg bw/day)	Compound, Species and Effect
37.000	Tetraethyl Lead, Humans, Lethality After Single Oral Exposure
2.000	Triethyl Lead and Tetraethyl Lead, Rat, Overt Toxicity After Repeated Daily Dosing for 21 and 45 Days Respectively
1.000	Triethyl Lead, Rats, Hematotoxicity After Daily Oral Dosing for 91 Days
0.500	Triethyl Lead, Rats, Neurotoxicity After Daily Oral Dosing for 91 Days
0.200	Triethyl Lead, Rats, Kidney Effects After Daily Oral Dosing for 91 Days
0.006	WHO Acceptable Daily Intake for Total Lead in Adults
0.0025	WHO Acceptable Daily Intake for Total Lead in Children
0.001	Acceptable Daily Intake for Alkyl Lead

The LD_{50} for tetraethyl lead in humans has been estimated to be 36 mg/kg bw (HHEC, 1986). The IJC's Human Health Effects Committee has proposed an Acceptable Daily Intake (ADI) for tetraethyl lead of 0.001 mg/kg bw/day (HHEC, 1986). This was based on the Threshold Limit Value recommended for the workplace by the ACGIH (American Conference of Governmental Industrial Hygienists, 1981). It is 200 times less than the lowest concentration known to cause adverse effects in laboratory animals (Figure 8).

Triethyl metabolites of tetraethyl lead and tetramethyl lead have been detected in tissues of human volunteers that had been exposed to the parent compounds (ATSDR, 1988b).

7.9.4 Exposure

Lead is routinely detected in Ontario drinking water at concentrations ranging from the minimum detection level to 0.022 mg/L on a total lead basis (Brecher *et al.*, 1989). Although there are no data on alkylated lead in drinking water, exposure through this medium is probably not significant because of the low levels of total lead. Ambient air is unlikely to be a significant route of exposure to these chemicals because atmospheric alkylleads are photoreactive, so they decay rapidly (ATSDR, 1988b). The largest airborne releases probably result from the manufacture, transport and handling of leaded gasoline (ATSDR, 1988b). Discharges in the Great Lakes basin have occurred as a result of the manufacture of tetraethyl lead at two locations, one at Sarnia on the St. Clair River and the other at Maitland, on the St Lawrence River.

Alkyl lead levels in fish from the St. Lawrence River have decreased since 1981 and in 1987 the geometric mean concentrations of most fish were below 150 ppb (µg/kg) (Wong *et al.*, 1988). The most common forms of alkyl lead found in residues are the tri- and tetraethyl forms (Wong *et al.*,1988). The IJC's Human Health Effects Committee has calculated that to exceed the TDI of 0.001 mg/kg bw/day, a person (70 kg) would have to ingest 200 g of fish containing 2.3 ppm of alkyl lead every week (HHEC, 1986). However, existing residue data suggest that alkyl lead levels rarely exceed this value. Similarly, the OMOE guideline of 1.0 ppm is rarely exceeded, so this chemical is not thought to be a risk to human health.

7.9.5 Summary

Alkyl lead compounds have been used extensively as additives to gasoline but recently their production has decreased and will continue to decrease as lead is phased out of gasoline. Contamination of fish in the Great Lakes by alkyl lead has occurred mainly in two areas, one on the St. Clair River and the other on the St. Lawrence River. This is a result of discharges associated with the manufacturing plants at these locations.

Alkyl lead compounds, especially the tetraethyl and triethyl forms, are considerably more toxic than the more common inorganic form. The consumption of contaminated fish is the most important route of exposure for residents of the Great Lakes basin. Residue data are available in fish from the two most contaminated areas (the St. Lawrence and St. Clair Rivers). There is no indication that alkyllead is a risk to human health, provided the OMOE consumption advisory is followed.

7.10 TOXAPHENE

7.10.1 Production and Use

Toxaphene is a contact insecticide and it is composed of a complex mixture of chlorinated camphene derivatives. Toxaphene has been used since 1949 and in 1975 it was the most heavily used insecticide in the U.S. (WHO, 1984c). The main applications were on cotton, cereal, grains, fruits, nuts, oil seeds and vegetables (WHO, 1984c). It has also been used extensively as a piscicide in fish eradication programs (NH&W, 1980). Since late 1982, the uses of toxaphene have been restricted in the U.S. Registered uses in Canada are minimal. Toxaphene is not manufactured in either the U.S. or Canada. However, it will continue to be found in the Great Lakes for a long time because of its previous heavy use, its persistence, and because it can be transported atmospherically.

7.10.2 Standards and Guidelines

U.S. FDA Fish Action Level:	5 ppm (LOTMP, 1989)
Canada Drinking Water Guideline (MAC):	5 ppb (CPHA, 1986)
HWC Tolerable Daily Intake:	0.5 µg/kg bw/day (Clegg, 1989)
Ontario MAC Drinking Water:	5 ppb (CPHA, 1986)
US EPA MCL:	5 ppb (CPHA, 1986)

EPA One Day Health Advisory: Office of Drinking Water: 0.5 ppb (U.S. EPA, 1987)

EPA Ten Day Health Advisory: Office of Drinking Water: 0.04 ppb (U.S. EPA, 1987)

7.10.3 Effects

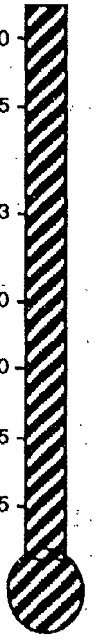
Toxaphene is moderately toxic in laboratory animals and has an oral LD_{50} of 80-90 mg/kg bw in rats (NRC, 1977). Long term exposure to toxaphene causes hepatic effects in rats (0.5 mg/kg bw/day) (Clegg, 1989), liver cancer in mice (1 mg/kg bw/day) (Clegg, 1989), degenerative changes in kidney tubules in dogs (4 mg/kg bw/day) (NRC, 1977) and thyroid tumours in rats (55 mg/kg bw/day) (Clegg, 1989).

Acute toxaphene poisoning in humans is rare and only a few cases of accidental poisoning have been reported (NRC, 1977). Dermal doses of 300 mg/person/day for 30 days and inhaled doses of 250 mg/m^3 for 30 minutes on each of 13 days did not cause any adverse effects (U.S. EPA, 1985). These effects are summarized in Figure 9.

7.10.4 Exposure

Toxaphene has rarely been detected in Canadian or Ontario drinking water (NH&W, 1980; CPHA, 1986). Atmospheric concentrations of toxaphene in the Great Lakes basin range from 0.1 to 0.3 ng/m^3 (IJC, 1988). This corresponds to an exposure of approximately 0.1 ng/kg bw/day (based on the extrapolation of calculations in U.S. EPA, 1985). Food also represents a minimal route of exposure (NH&W, 1980). However, the major source of exposure for residents of the Great Lakes basin is the consumption of fish containing toxaphene. Levels of toxaphene in lake trout from Lakes Michigan, Huron and Superior taken from 1977 to 1979 ranged from 1.9 to 10.7 ppm (average value 6.3 ppm). The highest concentrations occurred in carnivorous fish (Sullivan and Armstrong, 1985). More recent data show that levels in whole fish have decreased to 1 ppm or below. If a 70 kg person consumed 114 g of fish containing 1.0 ppm (μg/g) of toxaphene once per week, then s/he would ingest 0.23 μg toxaphene/kg bw/day. This is approximately one-half of the TDI. There are no data available on toxaphene residues in human tissues.

FIGURE 9 SUMMARY OF EFFECTS FOR TOXAPHENE



Dose Level (mg/kg bw/day)	Species and Effect
80-90	Rats, Lethality - Single Oral Dose
55	Rats, Thyroid Tumours After Long Term Oral Administration
4.3	Human Volunteers, Humans, Dermal Dose for 30 Consecutive Days, no Toxicity
4.0	Dogs, Nephrotoxicity After Long Term Oral Administration
1.0	Mice, Liver Cancer After Long Term Oral Administration
0.5	Rats, Hepatic Effects After Long Term Oral Administration
0.0005	Tolerable Daily Intake

7.10.5 Summary

Toxaphene is a complex mixture of chlorinated camphenes. It was used extensively in the U.S. and Canada in the 1970s as a general contact insecticide. Since the late 1970s and early 1980s, its use has been almost completely banned and as a result levels in the environment have decreased. The largest route of exposure for humans in the Great Lakes basin is through the ingestion of contaminated fish. It is unlikely that toxaphene is a risk to health at the levels currently being detected in the Great Lakes provided that fish consumption advisories are followed.

7.11 MERCURY

7.11.1 Production and Use

Mercury occurs naturally in the environment and can be found in geological formations and soils. The average concentration of mercury in the environment is 50 ppb (µg/kg) (NRCC, 1979). However, chlor-alkali plants associated with the pulp and paper industry were major sources of mercury to the Great Lakes basin. Mercury that is released to the environment will remain there indefinitely, although some organic mercury slowly degrades to inorganic mercury and conversely some inorganic mercury is transformed to the organic form (ATSDR, 1988c).

Mercury can occur in three forms: its elemental form, as inorganic salts, such as mercuric chloride, or as organic (alkyl) mercury, e.g., methyl mercury. Naturally occurring mercury and most industrial discharges are inorganic. Inorganic mercury can be methylated by bacteria in sediments as well as by other biota (NRCC, 1979). The organic form of mercury can bioconcentrate in fish and render the fish unacceptable for human consumption.

There are an estimated 3000 uses of mercury (Bever, 1986). In Canada, the greatest single use has been in the production of chlorine, sodium hydroxide and hydrogen from the electrolysis of brine in a mercury cell (NRCC, 1979). Mercury and its compounds have also been used in dental preparations, thermometers, pharmaceuticals, and as fungicides in paints, as well as for many other applications (NH&W, 1980). Although there are no production data available for Canada, in 1986 world production was estimated to be approximately 13,000,000 pounds (ATSDR, 1988c).

7.11.2 Standards and Guidelines

The following standards and guidelines apply to total mercury unless otherwise stated:

WHO Provisional Tolerable Weekly Intake:	0.3 mg total mercury (5 µg/kg body weight) including a maximum of 0.2 mg methyl mercury, as mercury (3.3 µg/kg body weight) (WHO, 1989)

NH&W Guideline for Commercial fish:	Maximum acceptable level 0.5 ppm (NH&W, 1979)
NH&W Guideline for those eating large quantities of fish:	Maximum acceptable level 0.2 ppm (NH&W, 1979)
OMOE Fish Guideline:	Unrestricted consumption: less than 0.5 ppm. Restricted consumption by adults in limited amounts: 1.5 ppm. Recommendation: Children under 15 and women of childbearing age should consume only fish with a Hg content of 0.5 ppm or less (UGLCCS, 1988).
U.S. FDA Action Level:	1.0 ppm whole fish wet weight (UGLCCS), 1988)
Canadian Drinking Water Guideline:	1 ppb (µg/L) Hg (NH&W, 1987a)
OMOE Drinking Water Objective:	1 ppb (µg/L) (CPHA, 1986)
WHO Drinking Water Guideline:	1 ppb (µg/L) (CPHA, 1986)
GLWQA Specific Objective:	Water: 0.2 ppb (µg/L) (filtered) (UGLCCS, 1988) Fish: 0.5 ppm (mg/kg) whole fish wet weight
EPA Drinking Water Regulation:	2 ppb (µg/L) (ATSDR, 1988c)

7.11.3 Effects

Metallic mercury is poorly absorbed and oral doses of 100-500 g have been administered to humans with no significant effects (NRCC, 1977). However, exposure to low levels of mercury vapour have been associated with neuropsychiatric effects (Clarkson, 1983; 1986). The oral LD_{50} of mercuric chloride in rats ranges from 35 to 105 mg/kg bw, and doses of between 29 and 50 mg/kg bw caused lethality in humans (ATSDR, 1988c).

FIGURE 10 SUMMARY OF EFFECTS FOR MERCURY

Dose Level (mg/kg bw/day)	Compound, Species and Effect
35-105	Inorganic Mercury Single Oral Dose, Rats Lethality
29-50	Inorganic Mercury Single Oral Dose, Humans, Lethality
35	Inorganic Mercury Single Oral Dose, Hamsters, Fetotoxicity
1.2700	Inorganic Mercury Oral Feeding for 2 Years, Rats, Nephrotoxicity
0.8000	Inorganic Mercury Single Oral Dose, Rats, Neurological Damage
0.1000	Methyl Mercury Fed for 12 Weeks, Rats, Nephrotoxicity
0.0500	Methyl Mercury Oral Dosing from Birth for 3-4 Years, Monkeys, Neurobehavioural Effects
0.0007	WHO Acceptable Daily Intake for Total Mercury Including Methyl Mercury
0.0005	WHO Acceptable Daily Intake for Methyl Mercury

Organic mercury is the most toxic form. Alkyl mercury compounds are readily absorbed by the gastrointestinal tract, the lungs and the skin (Bever, 1986). There are no acute toxicity data for organic mercury in either humans or laboratory animals (ATSDR, 1988c). However, both inorganic mercury and methyl mercury can cause kidney damage in laboratory animals (ATSDR, 1988c). The rat is the most sensitive species for these effects and 0.1 mg/kg bw/day of methyl mercury administered to female rats for 12 weeks caused ultrastructural changes in kidney tubules (ATSDR, 1988c). Methyl mercuric chloride causes dysfunction of the blood-brain barrier when it is administered to laboratory animals in a single dose of 0.8 mg Hg/kg bw (ATSDR, 1988c).

Inorganic mercury salts can cause neurotoxic effects in humans, but these are often the result of occupational exposure or abuse of therapeutic agents (ATSDR, 1988c). Neurotoxic effects have also been reported in laboratory animals following the administration of 1 mg $HgCl_2$/kg bw/day for up to 11 weeks (ATSDR, 1988c).

Organic mercury is also known to cause neurotoxic effects in humans following the consumption of contaminated fish or grain or of animals fed with contaminated grain. The first known incident occurred in the Minimata area of Japan in the late 1960s. The observed symptoms of mercury poisoning included a prickling, tingling sensation in the extremities, tunnel vision and slurred speech. In Canada, the potential effects of exposure to mercury from contaminated fish and game have been investigated in all Indian and Inuit communities (NH&W, 1979; 1984a). Although severe methyl mercury poisoning (Minimata Disease) was not found, symptoms associated with milder forms of mercury poisoning were reported. Eighteen communities were identified as being "at risk". This was defined as communities where some residents had blood (or hair) levels of 100 ppb or greater of mercury (NH&W, 1984a).

Both inorganic and organic mercury have been associated with developmental effects. Mercuric chloride has been shown to cause increased fetal resorptions in hamsters at doses of 35 mg/kg bw. In addition, monkeys which had been dosed with 0.05 mg methyl mercury/kg bw/day from birth to 3-4 years old displayed impaired spatial vision (ATSDR, 1988c). Other studies have shown that methyl mercuric chloride caused altered behavioral performance in 4-month-old rats when their mothers were fed 0.05 mg/kg bw/ day between days 6 and 9 of pregnancy (ATSDR, 1988c) and organic mercury has been associated with neurological damage in human infants whose parents had consumed mercury-contaminated food (ATSDR, 1988c) although no precise information was available on the dose levels which caused these effects.

Figure 10 shows these effects.

In summary, long term exposure to inorganic or organic mercury can cause irreversible damage to the brain, kidneys or the developing fetus. Inorganic mercury is more likely to damage the kidneys, while organic mercury is more likely to effect the brain and the developing fetus (ATSDR, 1988c). Bever (1986) has suggested that information from poisoning incidents in Iraq and Japan show that the fetus is between 2 and 7 times more sensitive to methyl mercury than its mother.

7.11.4 Exposure

Food is the largest route of exposure to mercury for the general population. Overall exposure in Canada has been reported to range from 0.01 to 0.02 mg/person/day for a 70 kg adult. This is equivalent to 0.15-0.29 µg mercury/kg bw/day (NH&W, 1980). If guidelines on the consumption of mercury-contaminated fish are not followed, health effects may occur, particularly if diets contain a large proportion of fish or seafood (NH&W, 1980).

If a person ate 114 g of fish containing 0.5 ppm (µg/g) per week then s/he would consume 57 µg of methyl mercury per week (based on a 70 kg body weight) or 0.81 µg methyl mercury/kg bw/ week, this is approximately 30% of the weekly intake recommended by WHO.

Concentrations of methyl mercury in fish from the Great Lakes are now usually less than the value used in the example above. In 1985, the concentrations of mercury in the edible portions of walleye, northern pike, white bass and yellow perch from Lake St. Clair were approximately 0.3 to 1.0 ppm (UGLCCS, 1988). In 1987, Lake Huron rainbow smelt contained 0.04 ppm (whole fish), Lake Huron walleye 0.18 ppm (whole fish), and Lake Ontario trout 0.14 ppm (see part 2 of Volume I). Concentrations in Lake St. Clair fish have decreased in recent years (WQB, 1989a) and the commercial walleye fishery in Lake St. Clair has been restored.

Mercury is detected relatively frequently in samples of Ontario drinking water, although the levels reported do not exceed the provincial drinking water guideline. In 1986, 78.6% of 687 samples tested contained detectable levels. The concentrations ranged from non-detectable to a maximum of 0.38 µg/L (Brecher *et al.*, 1989). Only a small proportion of the mercury in drinking water is in the more toxic organic form (NRC, 1977), and thus drinking water contributes very little to the overall exposure to methyl mercury. Air is also unlikely to be a major contributor to the overall exposure of residents of the Great Lakes basin. Atmospheric concentrations have been reported to be 2.0 ppt (2 ng/m^3) for all five of the Great Lakes (IJC, 1988). This is consistent with levels reported in the U.S. (ATSDR, 1988c).

7.11.5 Summary

Mercury is a naturally occurring element. It is used for many purposes and thus is found as a ubiquitous contaminant in the environment. The most hazardous form is methyl mercury which results from the bacterial transformation of inorganic mercury. Food is the main source of exposure for humans. Fish and seafood are the major food items which contribute to exposure because they bioaccumulate methyl mercury. The consumption of fish contaminated with methyl mercury could cause health effects, if fish consumption advisories and guidelines are not followed. Mercury also occurs in drinking water and air in the Great Lakes basin but the concentrations detected are similar to those found in the U.S. and are unlikely to be a risk to human health.

All three types of mercury cause health effects in humans and laboratory animals. These include effects on the kidneys, developmental effects, including fetal toxicity, and neurological effects.

7.12 OTHER CHEMICALS

The IJC designated the eleven chemicals described above as Critical Pollutants because of their persistence and toxicity in the Great Lakes basin. However, many other chemicals have been identified in the region. The IJC's Toxic Substances Coordinating Committee listed 362 chemicals in the Great Lakes (WQB, 1987). There are insufficient data on the environmental distribution and/or the toxic effects of most of these to permit any meaningful conclusions about their potential hazard to human health. These contaminants are at present at levels less than the Critical Pollutants, except when there have been point source discharges. The following are a few examples.

7.12.1 Tributyltin

Tributyltin (TBT) is a biocide that has been used in anti-fouling paints applied to boat hulls and marine and aquaculture facilities in the Great Lakes (WQB, 1989b). They are of concern because of their acute toxicity to aquatic organisms (WQB, 1989b). There is no information on the concentrations of tributyltin or any other organotin in drinking water or in fish. The limited toxicological data available suggest that organotins are only moderately toxic in laboratory animals (WHO, 1980a). The U.S. Environmental Protection Agency has recently enacted restrictions on the use of organotins in antifouling paints. It is therefore likely that the production and use of these chemicals will decrease. Exposure is also likely to decrease. However, in the absence of data on levels of tributyltin in fish and drinking water it is impossible to ascertain if there is a risk to human health, and if so its magnitude.

7.12.2 Tetrachloroethylene

Tetrachloroethylene is a volatile organic chemical that is manufactured in large quantities in the U.S. and Canada. It is used for many purposes, including dry cleaning and textile processing (Colborn, 1989). A manufacturing plant is located at Sarnia on the St. Clair River. The IJC's Human Health Effects Committee identified tetrachloroethylene as being of possible concern for human health. This was based on concentrations detected in surface waters (HHEC, 1986). However, it is not often detected in Ontario drinking water (CPHA, 1986). Although there are no data available on concentrations in fish, it is unlikely to bioaccumulate. In 1985, there was an industrial spill of tetrachloroethylene in the St. Clair River and levels of the chemical in local drinking water reached 4 ppb. However, two weeks after the spill, levels had decreased to below the minimum detection level (St. Clair River Pollution Investigation, DOE and OMOE, 1986). Tetrachloroethylene has also been implicated in several groundwater contamination problems across Canada.

Tetrachloroethylene is unlikely to be a risk to human health in the Great Lakes basin because it is unlikely to bioaccumulate in fish and is not often detected in drinking water.

7.12.3 Octachlorostyrene

Octachlorostyrene (OCS) is a by-product of some industrial processes and it does not have any commercial applications (Chu *et al.*, 1986).

OCS is only moderately toxic in laboratory animals. Based on acute, subacute and chronic toxicity data in the rat, the no-adverse-effect level has been estimated as 31 µg OCS/kg bw/day (Chu *et al.*, 1986). Chu *et al.* have also calculated the exposure of a 60 kg person from ingesting fish containing 0.3 ppm OCS, assuming a fish consumption of 114 g/week. They concluded that the daily human ingestion of OCS would be 0.078 mg/kg bw/day. This is approximately 400 times less than the no-adverse-effect level in laboratory animals.

Concentrations in fish (young-of-the-year spottail shiners and yellow perch) are highest just below Sarnia and persist downstream (WQB, 1987). More recent data (WQB, 1989b) confirm that most OCS contamination is restricted to the St. Clair River. The data also show that concentrations of OCS in spottail shiners decreased significantly between 1985 and 1987 in this area (WQB, 1989b). However, spottail shiners from the St. Clair River which contain levels ranging from 30-40 ppb (ng/g tissue) are above the NYDEC Fish Flesh Objective of 20 ppb (WQB, 1989b). OCS has been detected in five out of 141 human cadaver adipose tissue analyzed. None of the levels exceeded 15 ppb (ng/g) (Williams *et al.*, 1988).

In summary, at the levels currently found in the environment, OCS is not a risk to human health in the Great Lakes basin.

7.12.4 Di(2-ethylhexyl)phthalate

Di(2-ethylhexyl)phthalate (DEHP) is used extensively in North America in the manufacture of plastics. It is a phthlate ester. Approximately 1 billion pounds of phthlate esters are produced annually and in 1977, 400 million pounds of DEHP were produced (Menzer and Nelson, 1986). In 1981, there was an international conference on phthlates and the papers presented were published in a special 1982 issue of Environmental Health Perspectives (Conference on Phthalates, 1982).

Phthlate esters in general, and DEHP in particular, are known to be ubiquitous environmental contaminants. However, it is difficult to obtain accurate results from analytical methodologies if normal laboratory equipment is used. This is because plastic laboratory equipment contains phthlates, so sample contamination occurs. Accurate results can only be obtained if all sample collection, preparation and analysis is conducted using clean glassware.

DEHP has a low acute toxicity, but high doses are carcinogenic in laboratory animals (Conference on Phthlates, 1982). Other subtle chronic effects have been reported (Rubin and Jaeger, 1973) and low concentrations may be detrimental to the reproduction of some aquatic organisms (Mayer and Sanders, 1973).

Exposure of the general population to DEHP has been estimated to average 0.3 to 2 mg/person/day with food (non-fish) sources identified as the primary route (ATSDR, 1987e).

There are few reliable data on DEHP in fish and drinking water in the Great Lakes Basin and no data on DEHP in food. Therefore, it is difficult to assess the extent of the risk to human health in the Great Lakes basin from exposure to DEHP.

7.12.5 Chlordane

Chlordane is another cyclodiene insecticide that was widely used to control soil insects in the 1970s. It breaks down to oxychlordane. There are now restrictions on the use of chlordane in the U.S. and Canada. As a result, levels in various types of environmental biota have decreased significantly. Levels in fish are now generally less than 0.1 ppm (WQB, 1989a). Chlordane was not detected in 1987 in Ontario drinking water (Brecher *et al.*, 1989). Based on an Acceptable Daily Intake of 0.001 mg/kg bw/day established by FAO/WHO (WHO, 1984d), a 70 kg person would have to consume more than 700 g of fish per day with a chlordane concentration of 0.1 ppm to exceed the ADI. Residues of oxychlordane in fish are generally low (<0.1 ppm) and its subchronic toxicity is not substantially different from chlordane itself (WHO, 1984d).

Chlordane and oxychlordane are not likely to be a risk to human health in the Great Lakes basin because of the low levels of exposure from drinking water and fish.

7.12.6 Hexachlorocyclohexane

Hexachlorocyclohexane (gamma-HCH or BHC) is another persistent organohalogen insecticide that was used widely in the 1960s and 1970s. Its use has now been severely restricted in Canada and the U.S. It has been estimated that 100 tonnes were used in agriculture in Canada in 1976, and that only 110 kg were used in 1983 for agricultural applications in Ontario (CPHA, 1986).

Of the eight possible isomers, the alpha and gamma forms are most often detected in surface waters and fish from the Great Lakes (WQB, 1989a).

The main routes of exposure to HCH for residents of the Great Lakes basin are drinking water and food. In 1986, lindane (gamma-HCH) was detected at approximately 20% of the sites at which drinking water was sampled in Ontario. The levels ranged from less than the minimum detection level to 11 ppt (ng/L) (Brecher *et al.*, 1989). Drinking water levels are well below the provincial drinking water guideline of 4.0 ppb (µg/L) (CPHA, 1986). Levels of HCH in fish are generally less than 0.1 ppm (WQB, 1989a). Assuming a concentration of 0.1 ppm in fish, a 70 kg person who consumed 114 g per week would ingest 0.000023 mg of lindane/kg bw/day. This is approximately 1/500 of the WHO guideline. The toxic properties of alpha- and gamma-HCH are similar so the current FAO/WHO ADI for HCH of 0.010 mg/kg bw/day (WHO, 1984b) could be applied to both isomers.

The beta isomer is most frequently detected in samples of human adipose tissue from residents of the Great Lakes basin (Brecher *et al.*, 1989). The reported levels of beta-HCH in human adipose tissue are in the range of 70 to 110 ppb (ng/g tissue). The alpha and gamma isomers are rarely detected.

HCH is therefore unlikely to be a risk to human health in the Great Lakes basin.

Several other chemicals in the Great Lakes basin may be of concern for human health. These include: alachlor, a pesticide that has now been banned in Canada; carbon tetrachloride, a volatile industrial solvent that is manufactured in large quantities, does not bioaccumulate, but can contaminate groundwater; hexachlorobutadiene, an industrial chemical that is manufactured in North America in relatively large quantities; and trichlorophenols, chemicals that are precursors of dioxins, but are no longer manufactured in Canada (HHEC, 1986).

The shortage of adequate monitoring and/or toxicity data precludes a meaningful assessment of the potential of these chemicals to cause adverse health effects in the residents of the Great Lakes basin.

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TOXIC CHEMICALS IN THE GREAT LAKES AND ASSOCIATED EFFECTS

VOLUME II PART 4

SYNTHESIS

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1. INTRODUCTION

The Great Lakes basin is a contaminated ecosystem. Since the early 1970s, monitoring data have shown the presence of toxic chemicals in the water, air, sediments and biota. These chemicals have been associated with effects in many fish and wildlife species and, to a degree, in human residents of the Great Lakes basin.

In 1987, an internal Environment Canada report concluded that "fish and wildlife in the Great Lakes basin have been hurt by toxic chemicals and that people are being affected as well. The scale of the effects of toxic chemicals on human health cannot be determined...but...is significant and warrants concern".

Since then, there have been many other expressions of concern about the health of the Great Lakes ecosystem. For example, in 1989, the Health Committee of the Science Advisory Board recommended to the International Joint Commission (IJC) that "pending the availability of more information on human health, the results of studies on health effects in wildlife populations be used as a basis for decisions regarding the effects of chemical exposure on human populations". Subsequently, in its fifth biennial report on Great Lakes Water Quality, the IJC concluded that "there is a threat to the health of our children". A similar conclusion was reached in a consensus report from an international conference on toxic chemicals and health in the Great Lakes basin which stated that "persistent toxic chemicals due to their nature ... pose threats to the health of individuals within the Basin".

This report has reviewed much of the same scientific data as discussed in these earlier reports. This synthesis interprets the significance of the technical data discussed in the earlier sections.

2. CONCENTRATIONS AND TRENDS OF TOXIC CHEMICALS IN WATER, SEDIMENTS, FISH, WILDLIFE AND HUMANS

The Great Lakes basin is the largest and most-studied freshwater aquatic ecosystem in the world. There are data available on toxic chemicals in water, sediments, fish, wildlife and humans. Water quality data suggest that levels of toxic inorganic (e.g., mercury) and organic (e.g., PCBs) chemicals have decreased significantly. However, concentration trends in the water phase are difficult to verify because of the extremely low concentrations and because 'clean' analytical and sampling methodologies have provided data which are not comparable with earlier data sets. Analyses of sediment cores show that peak concentrations of many toxic chemicals, including mercury, PCBs, DDT, chlorinated benzenes and mirex, occurred in sediments deposited in the 1960s and 1970s. Since then, concentrations of most toxic chemicals in bottom sediments have decreased as a result of regulatory controls and improvements in industrial practices. Bottom sediment levels of some toxic compounds such as PCDDs and PCDFs have not declined to the same extent. Concentrations in fish and bird species (e.g., spottail shiners, lake trout and herring gulls) indicate contaminants have also decreased on a lake-wide basis; however, there are still heavily contaminated nearshore areas, harbours and channels. Although it is difficult to generalise, Lake Ontario biota are more contaminated than those from the other Canadian Great Lakes, in terms of the number of chemicals detected and their concentrations.

While residue levels in biota today are generally lower than in 1975, these declines should be understood in the following context.

1) Levels of many toxic chemicals in biota are no longer decreasing and have equilibrated, as indicated by monitoring data. This is partly because the Great Lakes basin is still receiving loadings of organochlorines from industrial and municipal sewage systems, atmospheric deposition, and leaking hazardous waste disposal sites.
2) Contaminated bottom sediments will continue to be a major internal source of toxic chemicals to biota in the Great Lakes basin until these sediments are buried, treated or removed.
3) Organochlorines are extremely persistent and continue to cycle through the different components of the biosphere. It is likely that levels of many of these toxic chemicals will decrease very slowly over the next 10 to 20 years.
4) Total PCB levels have decreased in most species; however, the concentrations of some individual congeners in predatory birds and mammals at the top of the food chain have not decreased to the same extent due to their selective accumulation. Often these are the more toxic congeners.

5) Contaminant levels measured annually in lake trout of the same age have decreased. Contaminant burdens in fish increase with age in relation to the duration of exposure. Although direct data are lacking for fish eating birds and mammals, older animals of these long-lived species are likely to have elevated levels of contaminants also.

Humans in the Great Lakes basin are exposed to toxic chemicals through food, air, drinking water and the use of a variety of consumer products. Approximately 80% to 90% of human exposure to many persistent organochlorines is through food. This proportion may be even higher for consumers of large quantities of contaminated Great Lakes fish and wildlife. Air and drinking water are minor exposure pathways for the persistent contaminants. Great Lakes residents are exposed to many chemicals originating from outside the basin. A significant proportion of food consumed by people living in the basin is grown outside the region. In addition, cross boundary transport of air pollutants from sources outside the basin has lead to increased human exposure and health effects. Standards and guidelines have been established for many toxic chemicals to reduce emissions and exposure in order to protect human health. These include air emission standards, ambient air guidelines, fish consumption advisories, drinking water guidelines and restrictions on the use of specific consumer products or product formulations.

Organochlorine contaminants have been measured in adipose tissue, mother's milk and blood from people living in the Great Lakes basin. These data confirm that humans are exposed to persistent, bioaccumulative chemicals. Residue levels of some of the most commonly occurring contaminants (e.g., HCB, dioxin) in residents of the Great Lakes basin are similar to those in people living elsewhere. The data are insufficient to permit a detailed assessment of the trends of the contaminants over time or by regions. However, there are sub-populations with higher than average exposures (nursing infants and people who eat relatively large amounts of contaminated fish or wildlife such as native peoples, sportmen and their families) and they are likely to have higher than average residue levels. Human tissue residue levels will probably decrease slowly, providing that discharges to the environment are reduced and contaminated areas are cleaned up. Contaminant concentrations in humans are likely to increase with age due to prolonged exposure, although there are no data for human populations in the Great Lakes basin to support this conclusion.

3. SIMILARITIES BETWEEN EXPOSURE OF FISH, WILDLIFE AND HUMANS

Like fish-eating birds, aquatic mammals and fish, humans who consume Great Lakes fish and wildlife are top predators in the Great Lakes trophic system. As a result, humans are exposed to many of the same toxic chemicals as wildlife, although exposures vary between species and between individuals of a species. Contaminant concentrations vary with diet, metabolic rate and fat content and the ability to excrete contaminants. Exposure to single chemicals is rare; more often humans and wildlife are exposed to mixtures of many chemicals over a lifetime. Exposures to persistent chemical contaminants are reflected as residues in human and wildlife tissues (e.g, 2,3,7,8-substituted dioxins).

In recent years, the number of toxic chemicals detected in human, fish and wildlife tissues has increased, as has the frequency of detection. This is a result of the increased sensitivity of current analytical techniques now available to scientists.

4. SIMILARITIES OF THE EFFECTS OF CONTAMINANTS ON FISH, WILDLIFE AND HUMANS

Biota in the Great Lakes basin are exposed to hundreds of chemicals. Many of these chemicals administered singly or in combination are toxic to laboratory animals and have been associated with effects in fish, wildlife and humans. Laboratory studies have also shown that many of the chemicals in the Great Lakes basin cause similar toxic responses such as reproductive disorders, enzyme induction, and immunotoxicity. Health outcomes are also affected by many interacting variable factors, such as age, diet, sex, nutrition and stress. These factors are more complex in human populations than in fish and wildlife. For example in human populations, lifestyle variables such as socioeconomic status, occupation, smoking behaviour, alcohol consumption and use of medications often confound the results. As a result of these factors it is usually difficult to establish cause and effect relations in fish, wildlife or humans. Instead, it is more common for "associations" to be established between exposure and effects.

Toxic chemicals are associated with many biological effects. A contaminant may not adversely affect an organism's ability to function, but may cause a change that is beyond the range of normal physiological responses. Biological effects can range from enzyme induction, changes in hormone metabolism or alterations in cell-to-cell communication to changes in lung function, loss of weight or rapid cell growth. Many toxic chemicals have similar biological effects across different species at equivalent levels of exposure. They may also have dissimilar effects due to differences in sensitivity. Nevertheless, effects on behaviour, reproduction, and fetal development have been observed in four orders of biota (fish, reptiles, birds and mammals) in the Great Lakes basin. Reproductive effects have been observed in fish, for example, lake trout from Lake Michigan. Eleven other species, including two species of mammals, eight bird species and one species of reptile, have experienced reproductive or other effects that have been attributed to exposure to organochlorine chemicals. Some of these species (double-crested cormorant, black-crowned night-heron, bald eagle, mink and otter) have also experienced population declines in the Great Lakes basin. All of these species are long-lived and eat fish. Tumours have been reported in bottom-dwelling fish and the spatial distribution of the affected populations indicates that pollution is associated with these effects. For example, there is strong circumstantial evidence that polyaromatic hydrocarbons are responsible for liver tumours in brown bullheads at several locations. Laboratory studies have also shown that many toxic chemicals affect the immune system, suggesting that these effects should be examined in fish, wildlife and human populations.

Traditional measures of human mortality and disease incidence have not indicated that the health of Canadians living inside or outside the Great Lakes basin is affected by toxic chemicals. However, exposure to contaminants is a risk to human health, even though the nature of the risk is unclear and cannot yet be quantified. An important epidemiological study has shown that there were adverse effects in the offspring of women who ate contaminated fish from Lake Michigan. It is likely that there are people who are at greater risk than average because they are more heavily exposed (e.g., nursing infants and people who eat relatively large amounts of contaminated fish and wildlife, such as native peoples, sportsmen and their families) or are more susceptible (e.g., the developing fetus, newborns, the elderly and those whose health is already compromised).

5. UNCERTAINTIES

Our understanding of the Great Lakes ecosystem is sufficient to conclude that further reductions in contaminant levels and improvements in the health of species depend on control of sources and clean-up activities. There remain however a number of uncertainties regarding the interactions occurring within the ecosystem; the resolution of these uncertainties would better enable us to restore the Great Lakes ecosystem.

A new lake bottom sediment survey for all of the lakes is needed for comparison with the results from the initial survey conducted in the late 1960s and early 1970s. New sediment cores should also be collected, dated and analyzed to provide more up-to-date information on contaminant trends. In addition, rates of contaminant exchange between sediments, water, air and biota should be quantified for whole lake and nearshore environments. Monitoring and surveillance programs on chemical residues in fish and wildlife should be coupled with programs on water quality and the sediment survey mentioned above so that uptake rates can be studied.

Previous biological programs have placed emphasis on determining levels of toxic chemicals, but in the future, monitoring of effects should be routinely conducted on fish and wildlife populations. This has already occurred to a limited extent in bird populations and there have been studies of tumours in fish populations. This type of monitoring should examine any adverse effects on population size, structure and health. Effects monitoring should be correlated with routine residue monitoring programs and with studies on laboratory animals so that the effects of exposure can be better understood.

There is no routine monitoring or surveillance of contaminant levels in human tissues. Individual research studies are often difficult to compare because of differences in the selection of study populations and in the analyses of contaminants. To date, the sizes of the exposed and control populations studied in the Great Lakes basin have been relatively small and the importance of such factors as age, gender, location and socioeconomic status have not been fully investigated.

A comprehensive human health monitoring and surveillance strategy should relate residue levels, biomarkers and effects in the human populations to each other and to equivalent data for wildlife populations and to levels in the environment. It should also examine spatial and temporal trends and the importance of such factors as age, gender, socioeconomic status and routes of exposure.

6. SOLUTIONS

The goal of the Canada-U.S. Great Lakes Water Quality Agreement is the virtual elimination of persistent toxic substances in the Great Lakes basin ecosystem. This will only be achieved if attitudes and behaviours change. All residents of the basin need to take responsibility for making virtual elimination a reality. It will require individual action, just as much as strategies from the boardroom or regulatory initiatives from government departments. These actions must include waste reduction, reuse and recycling as well as the use of substances that are less toxic and persistent. Pending the achievement of virtual elimination, ecosystem objectives should be established for each lake to protect the most sensitive species.

In addition to reducing inputs to the Great Lakes basin, it will also be necessary to clean up the areas that have already been contaminated (i.e., the 42 Areas of Concern). This is the ultimate goal of Remedial Action Plans. Many of the Remedial Action Plans have defined this as making the water "drinkable, fishable and swimmable". Although water from many parts of the Great Lakes is drinkable and swimmable, the unrestricted consumption of fish will require further reduction of inputs of toxic chemicals. Implementation of Remedial Action Plans would be a large step towards the restoration of the entire Great Lakes basin. Contaminated sediments are a problem in nearly all of the Areas of Concern and remediation cannot be complete until they have been buried, treated or removed.

Another large step towards restoration of the Great Lakes basin would be the implementation of the Lakewide Management Plans. The relationship between the reduction of nutrient inputs and the fate of toxic chemicals is an important component of these Plans. The preparation of both Remedial

Action Plans and Lakewide Management Plans is required in the 1987 Protocol Amending the 1978 Great Lakes Water Quality Agreement. To achieve these goals it will be necessary to quantify the inputs and effects of toxic chemicals so that appropriate remedial strategies can be selected and implemented in the appropriate sequence.

The goal of virtual elimination is analogous to the principle of preventive health care. Preventive health care has long been recognised as a vital component of good health. In simple terms, it is easier and better to prevent diseases from occurring than to cure them. The same philosophy could be applied to 'health care' for the Great Lakes basin. The introduction of any new chemical or process to the Great Lakes basin will probably have effects on the local environment and on the whole biosphere. These should be anticipated and prevented.

This report has described a complex set of facts, interactions and problems related to toxic chemicals in the Great Lakes basin. The role of science is to provide solutions wherever appropriate. The clean-up of a contaminated ecosystem is not a simple undertaking, but the solution can be simply stated:

> "What difference can a three-letter word make in changing the attitudes of a civilization? Can a change in our attitude make a difference in the health of our planet? Whose environment is it, anyway?
>
> "We must acknowledge that it is indeed OUR environment. We must put this small, three-letter word into our everyday vocabulary. "The" environment is far too impersonal a term to have any real effect on our deep emotions, and it is our deep emotions that will move us to action.
>
> "Our environment = our earth, our health, our future."

Viki Mather, Wahnapitae, Ont.

-- from *Seasons*, a publication of the Federation of Ontario Naturalists, Autumn 1990

APPENDIX

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* Mr. David Fairbairn and Mr. Ron Shimizu served as chairpersons during the latter stages of this project.

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