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

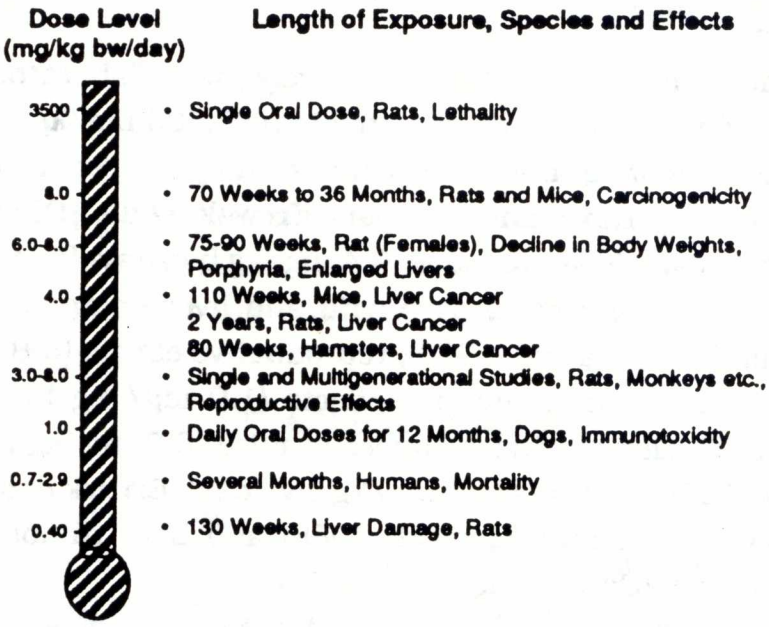
7.3.3 Effects

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



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 mothproof-

ing (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)

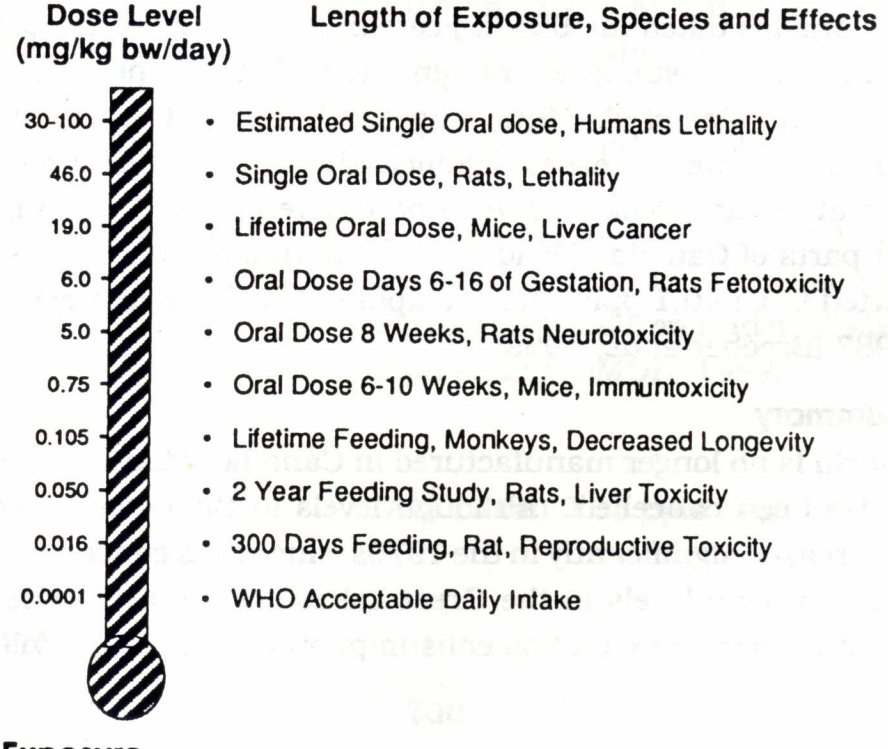
7.4.3 Effects

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



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

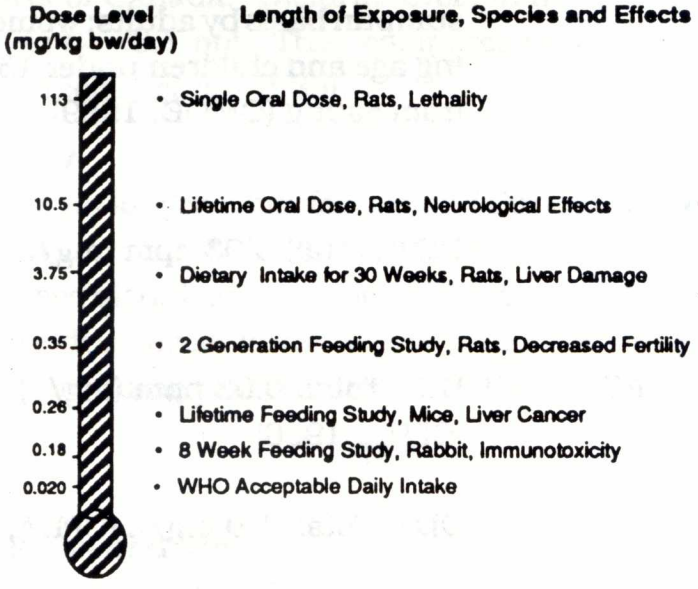
WHO Acceptable Daily Intake from food:	0.02 mg/kg bw/d (FAO/WHO, 1984)
NH&W Fish Maximum Residue Limit:	5.0 ppm (DDT + Metabolites) (Food and Drug Regulations, Table II).
FDA Fish Action Level:	5.0 ppm (LOTMP, 1989)
OMOE Fish Guideline:	5.0 ppm or more only on an occasional consumptin basis by adults; 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).

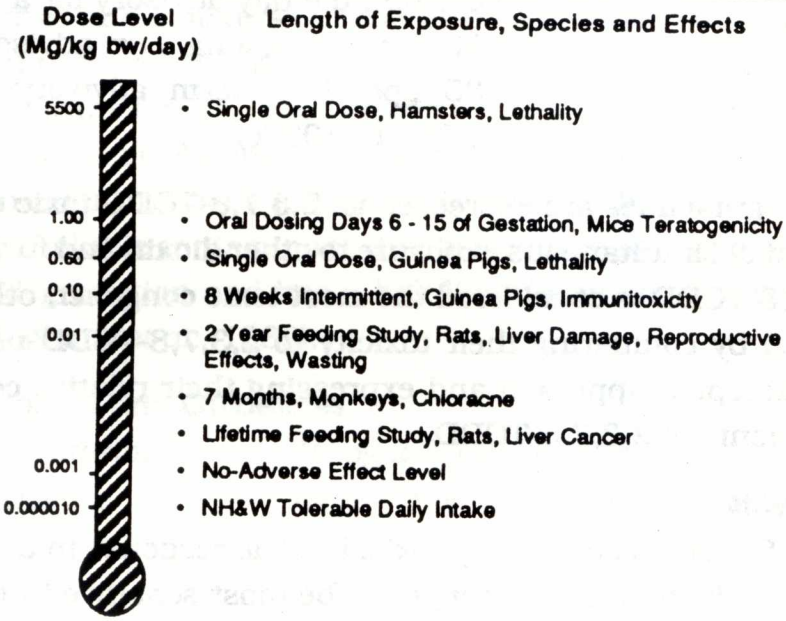
Note: Many guidelines are expressed as 2,3,7,8-TCDD toxic equivalents to take account of simultaneous exposure to other dioxin and furan congeners. Since 2,3,7,8-TCDD is considered the most toxic congener, other congeners are assessed by comparing their toxicity to 2,3,7,8-TCDD using an internationally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

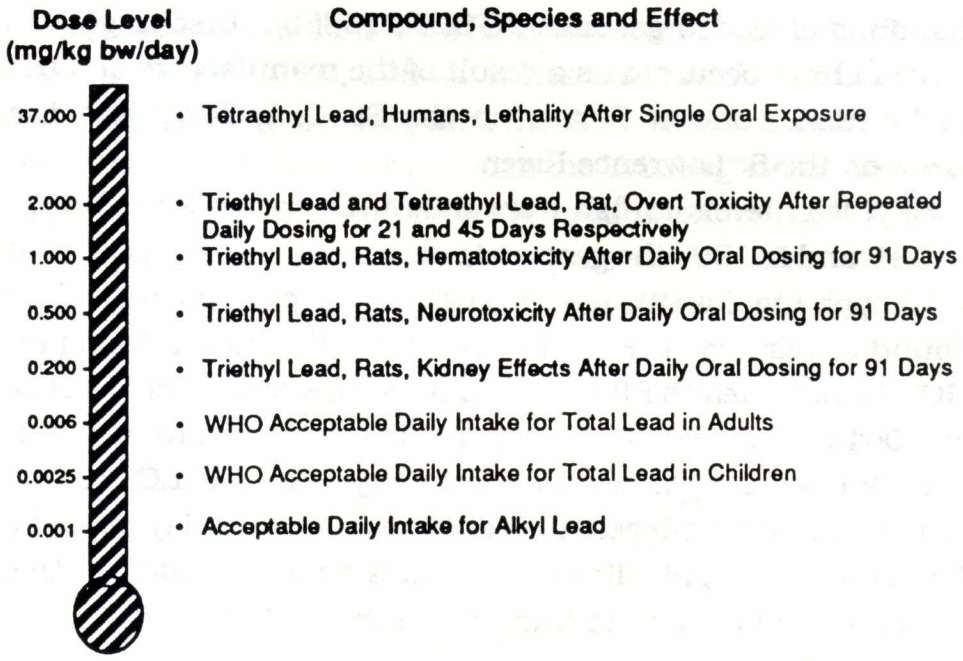
Canadian Drinking Water Guideline:	50 ppb (µg/L) (CPHA, 1986)
Ontario Drinking Water Guideline:	50 ppb (CPHA, 1986)
WHO Drinking Water Guideline:	50 ppb (CPHA, 1986)
GLWQA Specific Objective (Ambient Water):	10 ppb (µg/L) for Lake Superior, 20 ppb in Lake Huron, and 25 ppb in the other Great Lakes (GLQWA, 1988,)
EPA Ambient Air Criteria:	Air shall not exceed 1.5 $\mu g/m^3$ 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



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).

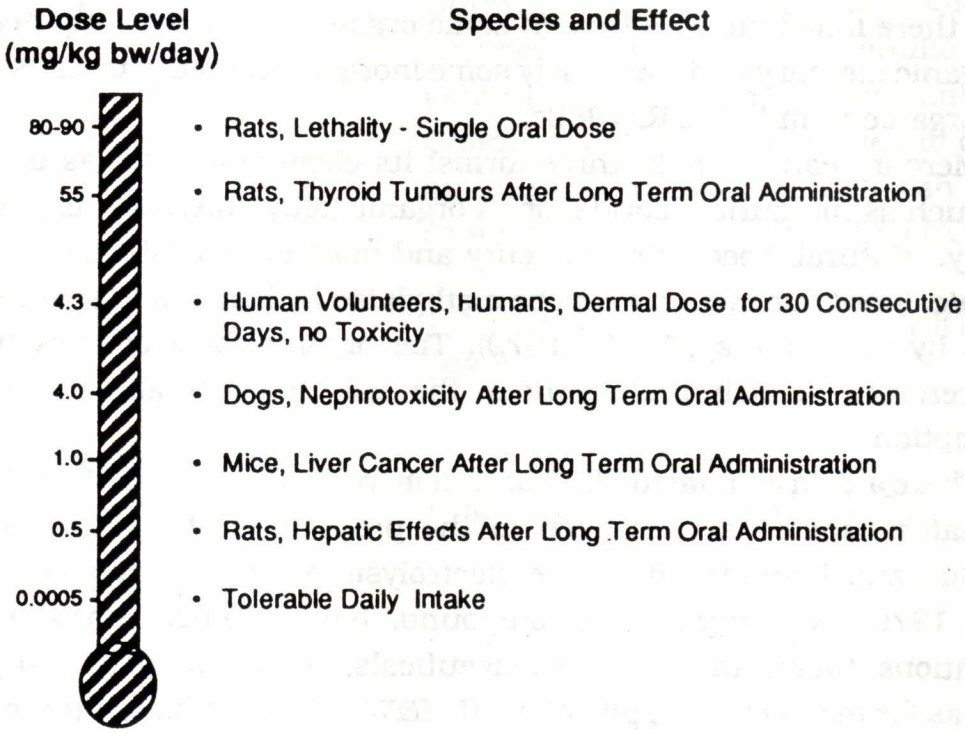
Acute toxaphene poisoning in humans is rare and only a few cases of accidental poisoning have been reported (NRC, 1977). Dermal doses of 300 mg/person/day for 30 days and inhaled doses of 250 mg/m^3 for 30 minutes on each of 13 days did not cause any adverse effects (U.S. EPA, 1985). These effects are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



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:

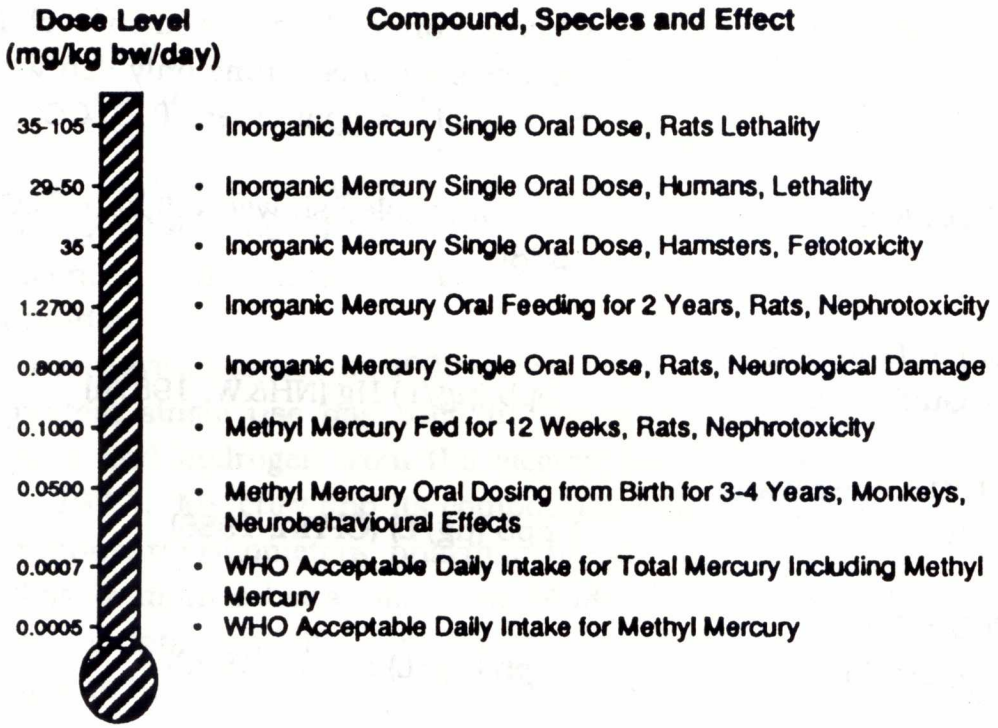
WHO Provisional Tolerable Weekly Intake:	0.3 mg total mercury (5 µg/kg body weight) including a maximum of 0.2 mg methyl mercury, as mercury (3.3 µg/kg body weight) (WHO,1989)

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



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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