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Niagara River Biota Contamination Project: Fish Flesh Criteria for Piscivorous Wildlife

by
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July, 1987

New York State/Department of Environmental Conservation

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TABLE OF CONTENTS

TABLE OF CONTENTS (Continued)

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EXECUTIVE SUMMARY

The Niagara River Toxics Committee (NRTC 1984) recommended that environmental criteria should be established for chemicals for which none exist. This study focused on the 19 organochlorine chemicals or chemical groups that have been found in spottail shiners from the Niagara River (Tables 3 and 4). The two primary objectives of this report are 1) to develop fish flesh criteria that will protect piscivorous wildlife, and 2) to evaluate a methodology for deriving such criteria where toxicology data is unavailable for wildlife species of concern. Both carcinogenic and non-carcinogenic effects are considered in development of criteria.

A list was compiled of 18 piscivorous species including mammals, birds and a reptile which are known to occur or have occurred along the Niagara River (Table 2). For each of these species body weight, daily food consumption by weight and food habits were determined. It is concluded that exposure of any of these target species to the 19 chemicals or chemical groups would be as great from any aquatic animal consumption as it would be from a 100 percent fish diet. From all the target species data, the mammal and bird with the greatest ratios of daily food consumption to body weight were selected for use in calculation of fish flesh criteria. Mink was selected with an average weight of 1 kg and food consumption of 0.15 kg/day. Several birds consume about 20 percent of their weight per day, thus, for calculation of criteria a "generic" bird was selected with a weight of 1 kg and food consumption of 0.2 kg/day.

In the past, fish flesh criteria for protection of wildlife have generally been derived from feeding studies with wildlife. However, few chemicals have been tested with wildlife species. The method proposed

in this report is to utilize the extensive laboratory animal toxicology data base employed by human health scientists to derive criteria for protection of human health, but instead extrapolate from that lab animal data to criteria for wildlife. The major advantage of this approach is that many more of the chemicals of concern in the environment have been tested with lab animals than with wildlife.

Results of lab animal tests are extrapolated to fish flesh criteria for wildlife with the following general formula:

NOEL/LOEL/Cancer Risk dose (mg/kg/day) X AF/UF(s)

X Target Species Weight (Kg) ÷ Target species daily intake (kg/day)

= Criterion (mg/kg)

Where:

NOEL (no observed effect level), LOEL (lowest observed effect level), or Cancer Risk dose is the result of a chronic or subacute toxicity test, or, the lower 95 percent confidence limit for the 1 in 1,000 or 1 in 100 risk calculated from dose-response data from a carcinogenicity assay with a lab species, AF/UF is one or more application or uncertainty factors.

After review of the scientific literature the following AF or UF are proposed for use where a chronic NOEL for a sensitive species is unavailable:

AF = 0.1, used to estimate a chronic NOEL from subacute data.

AF = 0.2, used to estimate a chronic NOEL from a chronic LOEL.

UF = 0.1, interspecies uncertainty factor when chronic data is available from only one or two species in the same class.

When extrapolating from lab to wildlife species, lab mammal data was used only to extrapolate to a wildlife mammal species (i.e. mink), and lab bird data was used only to extrapolate to a wildlife bird species (i.e. the "generic" bird).

Fish flesh criteria to prevent non-carcinogenic effects were derived for 16 of the 19 organochlorine chemicals or chemical groups; cancer risk criteria were derived for ten (Table 26). For five of the chemicals/chemical groups sufficient toxicity data was available to calculate and compare non-carcinogenic based criteria, derived directly from tests with target wildlife species, with criteria derived from lab animal tests. For four of the five criteria based on lab animal data the final criterion is somewhat less than the criterion derived directly from target wildlife data. It is concluded that the method proposed to derive non-carcinogenic based criteria from lab animal data, including the several AF/UFs used, is adequate to protect target wildlife species.

All of the ten 1 in 100 cancer risk criteria calculated are within an order of magnitude of the non-carcinogenic based criteria. It is tentatively concluded that a 1 in 100 risk is an adequate level of protection for wildlife populations, although this conclusion is not fully justified at this time.

Data on residues of the 19 organochlorines in Niagara River and Lake Ontario fish are available for comparison with fish flesh criteria derived in this report (residues detailed in Tables 3-7 and summarized in Table 26). In spottail shiners from the Niagara River only PCBs clearly exceed the fish flesh criteria. Fish flesh criteria for dioxin (2,3,7,8-TCDD) are less than the detection limit; dioxin was detected in Niagara River spottail shiners at all of five stations sampled in 1981 and at 2 of 13 stations in 1982.

Residues of PCB, dioxin and several other organochlorines found in a number of other fish species taken from the Niagara River and Lake Ontario exceed the fish flesh criteria. In one or more fish species

residues of each of dieldrin, DDT and metabolites, mirex and photomirex, chlordane, and octachlorostyrene exceed one or both of the non-carcinogenic based and 1 in 100 cancer risk criteria. Hexachlorobenzene residues in two fish species collected in the Niagara River in 1977 exceeded the criteria, but in fishes collected in the Niagara River and Lake Ontario in 1984 and 1985 none of the residues exceeded either criterion.

Exceedance of fish flesh criteria in some species at some locations suggests that the potential exists for toxic effects in wildlife from consumption of Niagara River and Lake Ontario fish. Actual occurrence of effects would depend on the extent to which individual animals consume those fish species with residues in excess of criteria and the duration for which those species are consumed.

1.0 INTRODUCTION

Contaminants in fish remain a concern for fish consumers. Composite samples of fish collected from major United States watersheds in 1976 (Veith, Kuehl, Leonard, Puglisi, Lemke 1979) contained PCB in 93% of all samples and 53% contained more than 5 mg/kg (whole fish basis). The current tolerance level is 2 mg/kg PCBs, set by the Food and Drug Administration (FDA), U.S. Department of Health, Education, and Welfare (Fed. Reg. 49 (100):21514-21520). The sum of DDT concentrations, hexachlorobenzene, and chlordane were also identified in a high percentage of samples. The first epidemiological studies of human health impacts indicate that contaminated fish consumption may reduce neonatal weight and motor skills (Fein et al. 1985). Fish eating wildlife species are particularly at risk, as some species depend almost entirely on consumption of fish and other aquatic organisms which may equal or exceed fish in levels of contaminants (Whittle and Fitzsimons 1983) (See Section 3.1).

Gilbertson (1985b) believes that toxic chemicals pose an "extremely complex hazard to the Great Lakes" because they are persistent and move into many compartments in the environment, crossing state, provincial and international boundaries. Experiments in the late 60's and 70's demonstrated reproductive failure in ranch mink fed Great Lakes fish (Aulerich et al. 1973). Wild mink are very dependent on the aquatic habitat for food and it appears the Niagara River area and western Lake Ontario mink populations are now non-existent (Robert Foley, NYS Dept. Envir. Cons. pers. comm.). There are at present no ospreys or bald eagles nesting in the vicinity of Lake Ontario (Gilbertson 1975a). It is likely that loss of habitat is a major factor affecting the reduction in numbers of some of the wildlife species discussed in this report. There is no conclusive evidence that ingestion of contaminated fish or other aquatic life contributed to the decline of these speices. Nevertheless, there is substantial evidence in the scientific literature that wildlife are sensitive to low level exposures of some contaminants.

The Niagara River Toxics Committee (NRTC 1984) summarized analytical data of residues in spottail shiners of 19 organochlorine chemicals or groups (Tables 3 & 4). NRTC recommended that criteria be developed for chemicals found in the Niagara River in order to determine the significance of the monitoring results.

The majority of these chemicals are on the national priority pollutant list and are persistent organochlorines. Contaminant concentrations in young-of-the-year spottail shiners (Notropis hudsonius) from the Niagara River were available for a number of stations along this waterway. Due to their restricted nearshore habitat, young-of-the-year spottail shiners have been useful and sensitive biomonitors and could be representative of local pollution influences (Suns et al. 1985). Since wildlife consumers do not utilize small forage exclusively, other whole fish and aquatic life residue data were obtained to compare with estimated effect levels for laboratory animals and wildlife.

Very little data on effects of the 19 NRTC chemicals/chemical groups on target wildlife species are available. Several reasons that dietary tests have not been conducted include cost, complexity, and the unsuitability of many wildlife species to laboratory culture and testing. Hudson (1984) and Wiemeyer et al. (1986) have addressed various aspects of estimating effects of toxicants on untested species (which may even be necessary in the case of valuable endangered species) through the use of comparisons of relative tolerances of test species to a chemical and use of surrogate species. Extrapolation of test data to untested species has become routine practice for estimating toxicity to humans, and predictions from such comparisons are now used for many risk assessments.

Chronic no effect levels of contaminants in the diets of laboratory animals can be extrapolated to estimate fish residue levels which will not effect wildlife. Chronic no effect levels of contaminants on the few wildlife species

tested in the lab will provide a form of validation of the extrapolation. Thus, the two primary objectives of the report are 1) to develop fish flesh criteria that will protect piscivorous wildlife from a number of contaminants found in the Niagara River, and 2) to evaluate a methodology for deriving such criteria where toxicology data are unavailable for wildlife species of concern. In addition to developing criteria to prevent non-carcinogenic effects, as generally described above, fish flesh criteria based on cancer risk to wildlife are also developed.

Criteria developed in this report and criteria for other chemicals to be developed in the future by these methods, can be used either to assess risks to wildlife from contaminants in their food at specific sites or, in conjunction with bioaccumulation factors, to calculate water quality criteria. Some assessment of the risk to piscivorous wildlife along the Niagara River, from the chemicals in Tables 3 and 4, is presented in this report. Water quality criteria will <u>not</u> be developed in the report.

2.0 METHODOLOGY

Wildlife which are fish consumers and current or former inhabitants of the Niagara River were listed and a population status assessment made through literature search and by contacting wildlife experts including Gordon Batcheller, the NYSDEC regional wildlife biologist. Feeding habits, body weights, and other data about these target species were gathered (Table 1) and narratives about those species follow in Section 3.0. In addition to information on wildlife species, similar data for laboratory animals (Table 2) was collected in order to make toxicological calculations when dose, dietary concentration or some other factor was not presented by the original author, or when needed for comparative purposes.

Methodologies proposed to calculate acceptable daily intake and fish flesh criteria are presented in the following pages.

Fish residue data for the Niagara River and western Lake Ontario and presented in Tables 3 & 4 (NRTC 1984), Table 5 (FDA 1977), Table 6 (Norstrom et al. 1978) and Table 7 (DEC in prep.). Toxicity tests and criteria calculations are presented in narratives for each of the 19 chemicals/chemical groups listed by NRTC.

2.1 General Risk Assessment Considerations

2.1.1. Calculating Acceptable Daily Intake (ADI)

Various researchers favor selecting a no observed effect level (NOEL) for ADI calculations from a test regime which demonstrated a lowest observed effect level (LOEL) just above the NOEL (Dourson and Stara 1983). Therefore, the NOEL presented in the scientific literature by the original researcher will be used if available.

The basic formula (Dourson and Stara 1983) for acceptable daily intake for humans or other animals other than the tested species is:

$$ADI = \frac{\text{no observed effect level}}{\text{uncertainty factor}}$$

Also, an extrapolation from laboratory animals to wildlife species has to take into account body weight and food intake compared to the test species.

Dourson and Stara (1983) describe a number of situations where uncertainty factors are used to account for variables, but note that the factors also incorporate a degree of safety (specifically in human health risk assessment). In Section 2.2 of this report, factors used in this study are discussed. To distinguish the wildlife risk assessment in this report from human risk assessment, the term application factor is used where estimating a chronic threshold or ADI from the toxicity data for the tested species requires a factor, and the term uncertainty factor is used where the objective is to provide some safety because of uncertainty about the data. Use of the term application factor in this way is consistent with how it is used in aquatic toxicology; i.e. a factor is applied to estimate a chronic threshold with no specific objective of incorporating a safety factor.

In a few cases it may be necessary to apply two factors as is commonly practiced in human health criteria development (Kim and Stone 1981).

2.1.2 Applying Laboratory Animal Studies to Wildlife

Differences in metabolism, exposure, distribution, storage, reabsorption, longevity, age to maturation, etc., result in considerable interspecies variation in tolerance to a given chemical. Due to some of the above factors, species that are sensitive to one contaminant, may be relatively more tolerant to another. This phenomenon is illustrated by examining the dietary feeding tables for the contaminants and noting that species' relative tolerance vary considerably (Table 7 through 23). As an example, the guinea pig is more sensitive to 2,3,7,8-TCDD than is the mouse (LD50s of 2.0 ug/kg and 114-284 mg/kg, respectively) although the guinea pig is more tolerant of pentachlorophenol exposure (LD_{lo} of g.p. = 250 mg/kg, LD_{lo} of mouse = 164 mg/kg).

2.1.3 Weight to Surface Area

Generally, larger animals have a lower metabolic rate and slower distribution of chemicals through their systems, and more cells exist which may be susceptible to some adverse effect (Kim and Stone 1981). On a body weight basis, humans are often more vulnerable than experimental animals (Doull et al. 1980). In developing the methodology applied in this report comments were solicited from a number of toxicologists, wildlife biologists, and water quality experts. Experts were queried about conversions or corrections when making wildlife risk assessments from laboratory animal data. It was asked if an application factor or a cube root correction for body to surface area should be used. Michael Dourson of the USEPA, Cincinnati (pers. comm.) responded that "This extrapolation is sometimes but not often used. For example, U.S. EPA uses a 10-fold uncertainty factor in lieu of this equation when estimating ADI's [for humans]." To examine this weight to surface ratio Klaassen and Doull (1980) provided the table presented below.

COMPARISON OF DOSAGE BY WEIGHT AND SURFACE AREA (100 mg/kg) DOSE

Species	Weight (g)	Surface Area (cm^2)	Dose by Weight (mg)	Dose by Surface (mg)	Ratio
Mouse	20	36	2	2	1
Rat	200	325	20	14	1.43
Guinea pig	400	564	40	24	1.65
Mink	1000				
Rabbit	1500	1272	150	55	2.74
Cat	2000	1381	200	60	3.46
Monkey	4000	2975	400	128	3.12
Otter	4500				
Dog	12,000	5766	1200	248	4.82
Man	70,000	18,000	7000	776	9.08

On a dose per unit of body surface, toxic effects in man and experimental animals are usually in a narrower range than effects expressed as a dose per unit of weight. However, the ratio of dose by weight to dose by surface is greater in humans than lab animals. When predicting the toxicity to humans of a drug tested in lab animals, a conversion factor to account for the difference in these species' weight to surface ratios is deemed necessary. Most wildlife as noted in the above table are in an even narrower range of dose by weight to dose by surface ratios, and in this study interspecies comparisons will be for animals of similar surface area such as rats to mink, chickens to ducks, or mallards to other ducks, etc. Therefore, a surface to weight conversion is not included in this method.

2.1.4 Selection of Toxicity End Points

Toxicity end points in the literature range from mortality through cholinesterase-depression. Weil and McCollister (1963) studied toxicity tests of over 50 chemicals and concluded that body weight gain, liver and kidney weight (as a percentage of body weight), and liver and kidney micropathology were the most reliable indicators of toxicity in acute and two year chronic tests, other than mortality. Reproductive losses are also an important toxicity factor that will be used to measure effect levels.

Long term, multi-generation toxicity tests are often not available. Where available, long-term dietary exposures to contaminants have been used; tests with a variety of species are reported to establish a range in interspecies tolerance.

Many factors cause variation in toxicity effect levels for any given endpoint such as 1) changes in the formulation of the toxic agent, 2) nominal versus actual exposure experienced by the test animal or target species, and 3) test animals selected (test lot health, genetics, etc.). To account for these experimental variation effects in studies with the Coturnix quail, the U.S. Fish and Wildlife Service includes positive controls (the chemicals dicrotophos and dieldrin) which are used along with negative controls (no chemical treatment, just the carrier or appropriate zero treatment) (Hill and Camardese 1986).

2.2 Application and Uncertainty Factors

2.2.1 Interspecies Adjustments

Results of many toxicity tests demonstrate that some species are more vulnerable than others (NAS 1977; Doull et al. 1980). Evans et al. (1944-in Doull et al. 1980) found humans were more sensitive on a mg/kg basis than rats to a number of metallic poisons. Ratios of toxic doses between rats and humans varied between 2.5 and 152, with a geometric mean of approximately 12. Hayes (1967) compared the smallest acute dose or largest acute non-fatal dose of six

pesticides between rats and humans. Ratios varied from 1.9 to 100 with a geometric mean of 11. Variation in toxicity for various birds and mammals presented in this paper strongly supports a 10-fold or more range in sensitivity to thoroughly tested organochlorines. The range from highest tolerance to greatest sensitivity usually exceeds this magnitude. If three or more species NOELS in a class exist, the lowest NOEL could probably serve as an estimate of a wildlife NOEL. If only one or two species NOELS in a class exist, an uncertainty factor of 0.1 appears appropriate to compensate for the unknown range of species sensitivities.

2.2.2 Short-term Versus Long-term Adjustments

Assessments of hazards to wildlife for the selected pollutants are limited by lack of data on chronic toxicity to Niagara River piscivorous wildlife. Optimally, multiple generation tests should be used for toxicity comparisons. Weil and McCollister (1963) presented evidence that short-term or subacute studies (30-90 days) can be used to predict no effect levels in longer trials (up to 2 years) with a fair degree of accuracy. These authors found that a 10-fold factor would cover 95% of the chemicals tested for short-term versus long-term exposure. Therefore, this acute to chronic application factor of 0.1 will be used when appropriate to estimate a chronic NOEL from subacute data. The term "application" is used here to denote that with use of this factor, a best estimate of the NOEL is made, as opposed to the fully acknowledged uncertainty underlying the interspecies adjustment.

2.2.3 LOEL to NOEL Adjustment

The EPA (1980a) recommends a factor from 1 to 10 for adjusting LOELS to NOELS based on the severity of the adverse effect of the LOEL. Dourson and Stara (1983) used the following example: if the LOEL is liver cell necrosis, a higher value is suggested for the factor (perhaps 10), but if the LOEL is fatty

infiltration of the liver then these authors suggest a lower value (perhaps 3). Stokinger (1972 - in Dourson and Stara 1983) use similar application factors in deriving threshold limit values for industrial chemical exposure. Weil and McCollister (1963) present data to justify using factors for LOEL to NOEL ratios all of which were 10 or less, and 92% were 5 or less. In this study an uncertainty factor of 0.2 is used to convert a LOEL to NOEL.

2.3 Steps in Calculating Wildlife NOELS

1. Adjusting laboratory species dose rate to representative bird or mammal.

Rationale: The wildlife NOEL is calculated from a chronic NOEL of a sensitive lab species by adjusting the daily food intake (d.f.i.)/body weight (b.w) ratio of the lab animal to the d.f.i/b.w. ratio of the wildlife species. If both mammal and bird data are available, the lowest fish flesh criterion derived using both bird and mammal data will be the final criterion.

Bird NOELS will be calculated for the bird that consumes 100% fish in its diet and consumes 20% of its body weight each day. For avian species, this will represent a realistic exposure to contaminants in fish. A number of target wildlife bird species are known to consume 20% of their body weight per day (Table 2). For simplicity in the calculation, a typical "sensitive" bird weighing 1 kg and consuming 0.2 kg/day will be used.

Mammalian NOELS will be calculated for a mammal that consumes 100% fish in its diet and consumes 15% of its body weight each day. For mammalian species this will represent a somewhat high, but realistic exposure to contaminants in fish for risk assessment purposes. The mink for example averages 1 kg b.w. and consumes 150 grams/day.

2. Interspecies adjustment factor when only one or two species were tested:

   0.1 X chronic lab animal NOEL = Wildlife NOEL

3. Acute data or subchronic (single dose to 30 day exposures) to chronic NOEL:

   0.1 X Acute LOEL = Estimate of chronic NOEL

4. LOEL to NOEL:

   0.2 X LOEL = Estimate of chronic NOEL

In conclusion the basic ADI formula of Dourson and Stara (1983) is modified to:

$$\text{Wildife ADI} = \frac{\text{NOEL of most sensitive animal adjusted by weight and food intake of wildlife species}}{\text{application/uncertainty factors (if applicable)}}$$

As an example of the review of literature on contaminant toxicity testing, the first chemical narrative on PCBs (Section 3.2.1) illustrates a well researched, thoroughly tested chemical. The proposed method is applied to toxicity tests on lab animals extrapolated to wildlife NOEL. The research on sensitive wildlife species is then reviewed to validate the proposed extrapolation for contaminants which have not been thoroughly researched.

To calculate no effect levels of contaminants in fish to protect piscivorous wildlife (wildlife NOELS) toxicity tests of laboratory animals were reviewed. These test results are presented in tabular form for each contaminant selected by the NRTC (1984). Acute toxicity tests with the rat were available in most cases, although a number of contaminants have not been evaluated for chronic or for carcinogenic effects.

Acute and chronic effect levels of a toxicant in the diet vary from test to test as noted in Hill and Camardese (1986) hence their inclusion of dieldrin as a positive control in a contaminant testing program. Hudson et al. (1984) note in their Handbook of Toxicities of Pesticides to Wildlife that the 1984 reported

results supersede previous values. Preliminary NOELS reported in the literature may fail to consider effects such as reproductive impacts which affect species survival. When several authorities present a NOEL the lowest was selected to calculate the ADI.

All dietary concentrations were converted to metric equivalents for the sake of uniformity. To calculate dosage for the test animal on a mg/kg/day basis, body weight (b.w.), daily food intake (d.f.i.), and dietary concentration of the contaminant are used. If the author failed to note one or several of the above items, animal weights and food consumption by NIOSH (1982) were used for calculating dose from non-specific data for laboratory animals (Table 1), or for wildlife, the Niagara River Wildlife Data (Table 2).

2.4 Epidemiological Studies

Epidemiological studies attempt to quantify risk by comparing two populations, one of which has been exposed to a substance and one which has not (Kim and Stone 1981). Reliable evidence of an adverse chronic health effect is a properly conducted epidemiological study in combination with well conducted animal experiments (Rall 1979). Several wildlife case histories will be presented which illustrate this approach including PCB effects in mink, DDT effects in birds and dieldrin effects in eagles.

2.5 Cancer Risk Assessment

Uncertainty factors are not recommended for carcinogenic data (NAS 1977). Kim and Stone (1981) trace development of cancer risk based on the one gene, one hit theory (as compared to assuming cancer induction requires a certain threshold level). Using results of the most sensitive test animal and the most frequent tumor, a dose/response multiple regression is developed and confidence limits set. In the development of water quality standards, the New York State Department of Health uses a lower 95% confidence level of dose response experimental data to extrapolate to a 1×10^{-6} increased cancer risk lifetime exposures of the experimental animals (Kim and Stone 1981).

The results of the DOH cancer risk for a chemical are then extrapolated to a human lifetime cancer risk. The 1×10^{-6} increased cancer risk calculation for the experimental animals is based on the lab animals life span. It is assumed that target species (e.g. human, mink, otter) would experience the same risk if exposed to the same daily dose over their lifetime.

In addition to calculation of cancer risk fish flesh criteria, cancer risk to wildlife estimated for contaminant levels found in Niagara River and western Lake Ontario fish are presented. Risk of increased cancer in experimental animals was calculated by the DOH with the Global 82 program as presented in the DOH fact sheet for water quality standards references in the appropriate chemical narrative sections of this report. The 1 in one million risk dose in experimental animals was then converted to 1 in 100 and 1 in 1,000 cancer risk fish flesh criteria for wildlife. These criteria are compared with criteria derived to prevent non-carcinogenic effects, and a rationale for selecting a particular cancer risk is discussed in Section 4.

3.0 SPECIES AND CHEMICAL NARRATIVES

3.1 Species Narratives

3.1.1. Mammals

3.1.1.1. Mink (Mustela vison)

Mink are distributed throughout most of North America (Linscombe et al. 1980). They occur in all the United States except Arizona. They are abundant in New York, including the western part of the state. However, they are rare if not absent from the Niagara River at present (G. Batcheller, NYS Dept. Env. Cons. pers. comm.); the same seems to be true for the entire lower Hudson River and Mohawk River (R. Foley, NYS Dept. Env. Cons. pers. comm.). Mink were present during the periods of exploration and settlement in these major river corridors. Regardless of the reason for their current absence or low population levels along the Niagara River (i.e. relative contribution from habitat loss, contaminants, etc.) mink will be considered in this report on appropriate representative piscivorous wildlife species.

Mink prey heavily on aquatic organisms for food; 50% of the aquatic diet is attributed to fish (Sealander 1943; Korshagen 1958). While other authors also suggest the diet is almost 100% aquatic food depending on season and feeding location, normal fish content in the diet is deemed closer to 30% than 50% (Aulerich 1973; Linscombe et al. 1982). Aulerich et al. (1973) used 30% fish in their mink feeding studies because it is the percentage used in mink ranching to yield optimal development. Frogs, crayfish, invertebrates and muskrats are important aquatic items in mink diet (Sealander 1963). However, mink utilize a diversified array of prey items and will feed on any animal they can find and kill (Linscombe et al. 1982). Mink are primarily

carnivorous; ingestion of plant debris is incidental to feeding on other prey items (Sealander 1943; Waller 1962). Regardless of the type of food eaten, mink consume large quantities of food per kilogram of body weight, more than does the otter (O'Connor and Nielson 1980).

Adult male mink range in weight from 0.9 to 1.6 kilograms (kg) and adult females weigh 0.7 to 1.1 kg (Linscombe et al. 1982). Amounts of food given to mink in feeding studies averaged 150 grams for an adult usually weighing about 1.00 kg (Aulerich et al. 1973).

Mink do not appear to suffer significant mortality due to predators other than humans, although fisher, red fox, grayfox, bobcat, lynx, wolf, alligators, and great horned owl are occasional predators of mink (Linscombe et al. 1982). Disease and environmental contaminants rank very high along with habitat degradation and human predation in limiting mink populations (Linscombe et al. 1982)

3.1.1.2 River Otter (Lutra canadensis)

The northern river otter was found historically over much of the North American continent (Hall and Kelson 1959). Along with the beaver (Castor canadensis) and the timber wolf (Canis lupus), it occupied one of the largest geographical areas of any North American mammal (Toweill and Taber 1982). Toweill and Tabor (1982) report that the northern river otter were found in all major waterways of the United States and Canada until the eighteenth century. Settlement and attendant changes in habitat, and perhaps overharvest, resulted in their extirpation from some areas. However, the otter is rare or absent from the Niagara River (T. Moore, NYS Dept. Env. Cons. pers. comm.).

Otter rely almost exclusively on fish in their diet and the remainder is almost entirely aquatic. Fish average about 90% of the otter diet (Lagler and Ostenson 1942; Greer 1955; Toweill 1974).

Toweill and Taber (1982) present tables of authorities on otter diet by geographic region and the results are overwhelmingly fish diets with crustaceans and amphibians also present. Otters consume less per kilogram than mink, but it may be erronous to conclude that they are less sensitive to contaminants. It simply may take otter longer to accumulate a toxic dose than mink (O'Connor and Nielson 1980).

Adult northern river otter range in weight from 5.0 to 13.7 kg (Harris 1968). Harris (1968) also found that otters in captivity required about 700 to 900 gm/day of prepared food. In favorable wild habitat observers have frequently noted that otter are highly successful at diving for and catching food, suggesting that maintenance diets of captive otter are comparable to those in the field.

O'Connor and Nielsen (1980) felt that otter would be as sensitive to methyl mercury poisoning as mink, but that the clinical course of the disease was faster in mink due to higher food intake per kilogram body weight than the otter. Henny et al. (1981) investigated the impact of PCB's and organochlorines on mink and otter in Oregon. The river otter harvest has declined for the last three decades in the Lower Columbia River whereas the statewide harvest trend is upward. Henny et al. (1981) conclude that PCB may have caused part of the otter decline in the lower Columbia River. Body residues of the otter from the lower Columbia reported in Henny et al. (1981) exceeded those of experimental animals of other species that died on PCB dosage. However, there are no laboratory studies on the sensitivity of otter to PCB's and other organochlorines. Otters do represent a species dependent on aquatic prey species. Toweill (1974) found that Cottidae (31%), Salmonidae (24%) and Cyprinidae (24%) were present in the otter diets from 75 river otters from Oregon. Other food items of importance in the diet were crustaceans, amphibians, birds and muskrats.

3.1.2. Birds

3.1.2.1 Bald Eagle (Haliaetus leucocephalus)

The bald eagle was much more numerous and widespread in the early part of the 19th century than it is now. They were active along the Niagara River, especially in the area of the falls (Beardslee and Mitchell 1965). The birds nested on Goat Island before the bridge connected it with the mainland, and various accounts spoke of an "abundance" of eagles at Niagara Falls in the 1800's (Beardslee & Mitchell 1965). The species was breeding chiefly in northern and western parts of the state, nesting wherever its principal food (fish) was abundant (Bull 1975). Peter Nye (NYS Dept. Envir. Cons. pers. comm.) of the Endangered Species Unit also asserts that bald eagles were prevalent along southern Lake Ontario and the Niagara River. At least four nest sites were active in 1910 along the Niagara River (Navy, Grand, and Goat Islands and Youngstown and Porter in Niagara and Erie Counties). The date of last active nesting for the Niagara was 1957 on Navy Island, which has been selected as a hacking site for an upcoming reintroduction attempt according to Peter Nye. Currently, the bald eagle and golden eagle are extremely rare in the Niagara Frontier (G. Batcheller, NYS Dept. Envir. Cons. pers. comm.).

The bald eagle is "a typical sea eagle" and flies along the coastline or waterways (Brown and Amadon, 1968). They prey heavily on fish, but routinely catch waterfowl and feed on carrion. Bald eagles frequent open waterways during the critical times in winter when severe weather can limit populations in their natural habitat. Peter Nye (NYS Dept. Envir. Cons. pers. comm.) reported that during bad weather New York wintering eagles may go for 4 or 5 days without food in Sullivan County near Mongaup Reservoir.

Although fish are an important component of the bald eagle diet, food varies with availability. Fish ranged from 6% to 90% of the diet (Krog 1953; Sherrod et al. 1980). Sherrod et al.(1980) felt the percentage of fish taken was probably far greater than shown by collection of prey remains because fish remains do not persist in nests as often as other food remains. Large concentrations of eagles feed on spawning salmon in the Pacific Northwest (Neuhold et al. 1971). On Amchitka Island, food availability was a major factor regulating the constantly changing population (Sherrod et al. 1980). During winter, carrion of big game was an important food item near western reservoirs, as were whale and sea otter carcasses in Alaska. In the Niagara River, fish would probably be a high percentage of the diet. Both extensive alewife and salmon die-offs in the Niagara River and nearby Lake Ontario area would seasonally furnish a great abundance of dead fish if eagles are reestablished.

Eagles consume 450 to 750 gms/day as fledglings, and gorged older young or adults can consume as much as 300 to 1200 grams in one day. Eagles mature at 4 to 5 years old (P. Nye, pers. comm.) and range from 3.5 to 7 kg in body weight. This study uses a body weight of 4.5 kg and food consumption of 900 gm/day.

Studies by Patuxent Wildlife Research Center at Laurel, Maryland, appear to show a decreasing level of DDD and dieldrin residues in bald eagle eggs between earlier data, 1969-1974, and later data, 1975-1979. DDE, heptachlor epoxide, PCB and mercury levels showed no definite trend in the overall data (although the DDE figures may be partially explained by DDD having a shorter half life than DDE) (Wiemeyer et al. 1984). Geometric mean residues of DDE, DDD, dieldrin and PCBs in eagle eggs from Alaska and Minnesota illustrates this trend: Alaska had 1.8, 0.09,

0.08, 2.1 ppm respectively in 1969 as compared to 0.94, 0, 0, 0.69 (where 0=none detected) in 1975. The Minnesota data for 1969-1972 was 8.5, 0.70, 0.90, 10.0 ppm respectively as compared to 2.5, 0.1, 0.15 2.7 in 1978 (Wiemeyer et al. 1984).

Although environmental contaminants are not the primary cause of death in eagles (Stickel et al. 1966) the eggshell thinning effect of contaminants continues to influence reproductive success and remains a valid concern. Study data corresponding to aforementioned contaminant levels around 1975 still demonstrated that 13, 27, 31 and 20% of eggs from breeding areas were experiencing more than 20% thinning of eggshells.

3.1.2.2 Belted Kingfisher (Ceryle alcyon)

The belted kingfisher is common along the Niagara River and is a widespread breeder in suitable habitat in New York (Bull 1975). Kingfishers are dependent on suitable cutbacks along streams for nesting. The bird migrates south during the winter and is rare to uncommon during the winter. The belted kingfisher is familiar in New York and a representative of a well defined (90 species) family. Members of the kingfisher family are highly specialized but are all clearly of common descent (Fry 1980). The North American belted kingfisher is a common inhabitant, occupying all types of waters from estuaries and lakes to rocky, swift mountain streams.

The kingfisher often hovers when fishing, scanning the water from as high as 10 to 12 meters and making a straight or spiral dive directly downward. Fish predominate in the diet but they also feed on frogs, crayfish, and aquatic reptiles (Fry 1980). Alexander (1977) found the kingfisher stomach contents to be highly variable by water type. Birds of Michigan's North Branch of the Au Sable River ate 63% fish, with

trout comprising 29%. Eighty-six percent of their diet from streams in the Hunt Creek Area was fish, 80% of which was trout. Virtually all of the kingfisher diet is aquatic.

The average adult weight of the belted kingfisher is 0.15 kg (Fry 1980). The kingfisher's food consumption per day is a very high percentage of its body weight. White (1936) found that the kingfisher consumed 1 to 1 3/4 times their weight per day from hatching to flight stage, with consumption decreasing as the birds grew older. Alexander (1977) concluded that kingfishers consumed 50% of their body weight per day. Rorig (1905 - in Seibert 1949) made intensive investigation of food consumed by small wild birds and concluded that the smaller a bird was, the relatively more food it consumed. Therefore, the kingfisher is a pisciverous bird with a high food intake.

Some contaminant levels have been measured in New York kingfishers and the body burdens are relatively high. An analysis of a kingfisher from Westchester County in 1976 found 4.8 ppm chlordane and 4 ppm PCB (Aroclor 1254). Death seemed directly linked to the chlordane contamination (Ward Stone, NYS Dept. Envir. Cons. pers. comm.). The tolerance of kingfishers to contamination is unknown.

3.1.2.3. **Bufflehead (Bucephala albeola)**

This small duck is common on the Niagara River. Bull (1974) stated that "the bufflehead seems to have increased in recent years, especially within the last decade." The bufflehead winter inland maxima on the Niagara River was 2200 on December 7, 1968. Banding data from buffleheads suggest that they move around extensively and that they spend their summers in western Canada.

Bufflehead feed primarily on small animals (70-90%) (Erskine 1972 - in Palmer 1976) and the reported percentages of fish in the diet range

from 3% to over 20%. Stott and Olson (1973) reviewed the food habits of sea ducks including bufflehead and concluded that bivalves, crabs, shrimp, and small fry make up the bulk of the food. Erskine (1972 - in Palmer 1976) reports bufflehead feeding mostly on small animals including aquatic insects, molluscs, crustaceans, and gastropods. R. Foley (NYS Dept. Envir. Cons. pers. comm.) concluded that about 20% of the Niagara River bufflehead's diet was fish. For calculations in this project the average percentage of fish in the diet was assumed to be 20%.

Adult bufflehead males averaged 0.45 kg (Erskine 1972 - in Palmer 1976) and the adult females averaged .33 kg. The food intake in grams per day is estimated at 90 for adult males. Bufflehead mature at age two (Palmer 1976).

Body burden measurements of a number of persistent organochlorines from Niagara River bufflehead have been performed (R. Foley, NYS Dept. Envir. Cons. pers. comm.) which show body burdens of 47 ppm for PCBs, 0.188 ppm for DDT, 0.198 ppm for dieldrin and 0.027 ppm for chlordane and metabolites. Erskine (1972 - in Palmer 1976) notes that significant amount of the species' western Canadian habitat has been lost in the summer and will probably limit the upper population numbers to a level well below that which existed a hundred years ago.

3.1.2.4 Common Goldeneye (Bucephala clangula)

The common goldeneye is an abundant diving duck in the Niagara River area. The common goldeneye is a common to very abundant winter visitant on the coast and on the Great Lakes, and is especially numerous on eastern Long Island (Bull 1975). All of the recorded inland maxima in New York are for the Niagara River or western Lake Ontario. The species is generally rare before November and after early April.

Food items of the common goldeneye are markedly different depending on the habitat (Stott and Olson 1973). The bulk of common goldeneye diet is animal matter (74% animal, 26% plant). Common goldeneye taken in harbors by Stott and Olson (1973) had eaten seeds of eel grass and sand shrimp, and birds taken along the coastline proper had eaten isopods, amphipods, and rock crab. Foley and Batcheller (in press) found 6.4% fish in the stomach and lower intestine of common goldeneye collected on the Niagara River. Cottam (1939 - in Palmer 1976) examined the stomachs of 395 "adult" common goldeneye. Crustaceans, amphipods, shrimp, and insects were frequently found common goldeneye food items. During spring, 60% of the food taken by U.S.S.R. goldeneye were small fish (Dementiev and Gladkov 1952).

Common goldeneye adult males average 1.1 kg and females average 0.9 kg (Palmer 1976). An estimate of consumption each day is 200 grams based on a diet percentage equaling 20% of bodyweight per day.

Foley and Batcheller (in press) measured contaminant levels in common goldeneye along the Niagara River and western Lake Ontario. These levels average about 5 ppm PCB and were considerably lower than the highly piscivorous mergansers.

3.1.2.5. Common Tern (Sterna hirundo)

The common tern is common in the Niagara River area, although it is apparently negatively impacted by contaminants and competition with other species (Gilbertson 1985a). Common terns breed from Canada south to the Bahamas (Bent 1963a). The main wintering range is in South America, all along both coasts, but will winter north to South Carolina.

The food of the common tern consists almost wholly of small fish, not over 3" to 4" long. Adult common tern average 0.14 kg (Whittow and Rahn 1984) and consume about 20% of their body weight in food per day.

The food of common terns nesting on the lower Great Lakes was studied by Courtney and Blokpoel (1980). In western Lake Ontario, 90% of the diet was comprised of alewife and smelt. In the Niagara River the principal food items were smelt, emerald shiner, common shiner, and bluntnose minnow. In eastern Lake Erie smelt, emerald shiner and trout perch were principal items. In all of these locations non-fish material was rarely observed. The young are fed by their parents until they are fully grown and able to fly.

Declines of common tern in the late 1800's, early 1900's, have been mentioned in the literature. MacKay (1891 - in Bent 1963a) describes an astonishing abundance of terns in the 1870's. But, at that time tern eggs were taken in large numbers, their plumage became fashionable, and the numbers of terns declined noticeably. However, these were not the only reasons for decline. Jones (1906 - in Bent 1963a) found evidence of great mortality among young tern chicks on muskeg, probably killed by exposure to prolonged, cold, easterly rainstorms. Stringent laws were subsequently passed for common tern protection (and fashion changed) and by the 1960's it had practically regained its former abundance (Bent 1963a). Thus, common tern populations have varied as influenced by predation and weather.

Recently, common tern breeding on the Great Lakes has decreased (Morris et al. 1980; Haymes and Blokpoel 1978). This reduction may be due to environmental contaminants (Fox 1976; Gilbertson 1974; Gilbertson et al. 1976). This data is discussed as a case history in the toxicology section of this paper since it illustrates PCB and DDT-DDE contaminant effects.

3.1.2.6 Common Merganser (Mergus merganser)

The common merganser is abundant on the Niagara River. Bull (1975) reports that the common merganser is a frequent to very abundant winter vistant on the Niagara River, the Great Lakes, and the larger lakes of interior New York. The common merganser is primarily a freshwater species.

The common merganser is a fish eating bird. Palmer (1976) states that there "is little point in giving details of the names of food items and percentage occurrence for mergansers as they eat what is available to them in their particular habitats." Although young common mergansers consume a fair percent of insects, the young soon start to catch fish (White 1957). In waters with trout or salmon common mergansers feed on a high proportion of these gamefish. Alexander (1977) reports that trout made up 84% of the total common merganser diet and the remainder consisted of other fish species. Common mergansers will resort to eating other items if that particular water is fished out (White 1957).

An adult common merganser typically weighs 1.5 kg (Palmer 1976) with males ranging from 1.5 to 2 kg and females from 1.05 to 1.4 kg. Alexander (1977) calculated that an average 1.41 kg common merganser consumed 0.47 kg of fish/day when feeding on good trout waters in the north central Lower Peninsula of Michigan. This calculation assumes 33% of body weight eaten per day. Data presented in both Avian Energetics (Paynter 1974) and Seabird Energetics (Whittow and Rahn 1984) seem to indicate that 20% would be a more appropriate percent of body weight eaten daily or 300 grams of food/day for 1.5 kg birds.

The total volume and the size of the individual prey fish consumed by the common merganser exposes them to contaminants in fish. R. Foley (NYS Dept. Envir. Cons. pers. comm.) is monitoring residue levels in common and red-breasted mergansers on the Niagara River and is finding

high PCB and other organochlorine levels. Owls, man, and the bald eagle are predators of the common merganser (Palmer 1976) although due to the body burden of organochlorines in birds sampled in New York, this consumption would constitute a health hazard.

3.1.2.7 Red-Breasted Merganser (Mergus serrator)

The red-breasted merganser is a common to very abundant migrant on the New York coast and on Lake Ontario. The red-breasted merganser is much more marine than the common merganser, and is just as numerous on the Niagara River.

Red-breasted mergansers are estimated to consume 235g of food per day. But, since the red-breasted and common mergansers are so alike in habits, the section on the common merganser should suffice. The bulk of the diet consists of fishes (Palmer 1976). At 1.15 kg average, red-breasted mergansers weigh somewhat less than the common merganser.

3.1.2.8 Common Loon (Gavia immer)

The common loon is rare in the Niagara River area at present (G. Batcheller pers. comm.). Bull (1974) lists Oswego County as the solitary breeding record in western New York. The fast flowing Niagara River would not seem to be preferred habitat. However, G. Batcheller suggests that several areas adjacent to Grand Island with still, quiet waters, may have been suitable nesting habitat in historic times. In addition, Beardslee and Mitchell (1965) report that most winter records in New York are from Lake Ontario and the Niagara River. Summer records are also found for the Niagara River mainly from the gorge below the falls; most birds from these records are immatures (Beardslee and Mitchell 1965). The common loon is included in calculations of fish flesh criteria as a representative of a large obligatory piscivore.

The common loon is an excellent diver and its food is mostly aquatic. Approximately 80% of the diet is fish according to most reports (Warren - in Bent 1963; Parker 1985). The diet of loon varies considerably from lake to lake since highland lakes vary in fauna. The typical Adirondack Lake has a very small number of fish species (George 1981) and contains brook trout alone, or brook trout and a few other species. Common loons feeding in these lakes consume many trout. Alexander (1977) found the diet of the loon to be predominatly fish, with 80% being trout. The Michigan lakes studied by Alexander were managed for trout and were treated periodically with chemicals to remove non-game fish populations.

Common loons have been reported to subsist on plant material during periods of captivation. Common loons in lakes devoid of fish due to increasing lake acidification feed largely upon crayfish and macroinvertebrates (Parker 1985). In waters with fish present, fish comprise from 50% to 100% of the loon diet.

The common loon is a large and heavy bird ranging from 3 to over 6 kg, with 4.5 kg being an average adult weight. The common loons daily consumption is reported to be rather high by many accounts. Alexander (1977) reported that the common loon consumes nearly 2.4 pounds of trout per day and calculated that they consume 33% of their body weight per day. Parker (1985) estimated that 430 kg of food is required to support a pair of adult common loons on their territory for six months and to rear a chick to the fledging stage at 15 weeks of age. The developing common loon chick consumes 40% to 80% of its body weight per day depending upon activities (Parker 1985).

3.1.2.9 Herring gull (Larus argentatus)

Herring Gulls are abundant in New York, and on the Niagara River they are abundant in winter. Herring gulls have been chosen (Gilman et al. 1985; Gilberston 1985a) as a prime species for routine monitoring of trends in reproductive success, of levels of organochlorine compounds, and for detailed etiological research. Gilbertson (1985a) states that among the most important reasons for the choice was the relatively non-migratory habits of the adult breeding population in the Great Lakes.

The herring gull is a seabird and is widely distributed. Fish are the single most important food item in the herring gull diet, but they also consume carrion of almost any kind, shellfish, crustaceans, insects, smaller birds and mammals, insects and earthworms (Tinbergen 1960). Herring gulls open shelled invertebrates by dropping them (Kent 1981). They also readily accept food offered by humans such as stale bread and viscera from cleaned fish. Fish constitute 50% of diets reported in the literature, but the herring gull is the epitome of opportunistic feeding, seizing whatever food is available. Body weights of herring gulls range from over 0.5 kg to 1.3 kg (Whittow and Rahn 1984). The birds mature at about 2 years old. Research on seabird energetics (Whittow and Rahn 1984) indicates that in order to maintain themselves herring gulls must consume about 20% of their body weight or about 200 gm/day.

The selection of herring gulls as indicators of contaminants in the Great Lakes is based on several important points (Gilman et al. 1985). First, it feeds at the highest trophic level of both aquatic and terrestrial food chains. Secondly, the herring gull is a year-round resident of the Great Lakes (Moore 1976). Apparently there is little

movement of these gulls from lake to lake. Gulls wintering along the Niagara River may range to Lake Ontario or Lake Erie. Gilman et al. (1977) showed via banding recoveries that Lake Superior gulls overwinter in Lake Michigan. Within a lake the gulls seem to be wide ranging, and Gilman et al. (1985) suggest the herring gull may be integrators of pollution, largely from aquatic food chains.

Third, the herring gull nests colonially. Tinbergen (1960) describes the colonial nesting behavior and movement of herring gulls which concentrates large sectors of the breeding population in one place at one time. Gilman et al. (1977) point out that colonial nesting allows reproductive success, behavior, and levels of contamination to be easily assessed. Organochlorine levels can be measured in the gull's egg, with second laying and other mechanisms compensating for the loss, thus maintaining population levels.

The fourth point that Gilman et al. (1985a) cite is the wide holoarctic distribution of the herring gull, allowing researchers to compare contaminant levels, reproductive rates, and behavioral characteristics of Great Lakes gulls with coastal and European populations.

Although abundant along the Niagara River and the Great Lakes in general, data from Lakes Michigan, Heron and Ontario points to "clear, easily-observed signals [that] chemically-induced epizootics were occurring" in herring gull populations between 1964 and 1970 (Gilbertson 1985a). Reproductive problems have also been noted in terns, herons, and cormorants (Gilbertson 1985a).

Keith (1966) presented some of the first data on reproductive success as it relates to pesticides residues, analyzing residues in eggs and adult birds in Green Bay, Wisconsin. Gulls which were collected

contained as high as 2,000 mg/kg organochlorine residues in body fat. His work, along with Lake Ontario data in 1966, evidenced "severe reproductive failures" in colonies previously appearing normal, linking the presence of organochlorine chemicals with the subsequent reduction in the number of eggs progressing to fledging (Gilbertson 1985a). Effects of pesticides have been noted throughout the life stages, including egg viability, hatchability and adult survival (Gilberston 1985a). The probable causal agents and supporting studies are presented in several of the chemical narratives.

3.1.2.10 Ring-Billed Gull (Larus delawarensis)

The ring-billed gull is smaller (averages 0.45 kg compared to 1.0 kg for the herring gull) and has few of the brown specks which frequently mark the herring gull. The differences in size are most noticable when both species are present. The black ring around the bill slightly ahead of center towards the tip, identifies the ring-bill. Bull (1975) lists the ring-billed gull as a common to abundant migrant and winter visitant on the Great Lakes and Niagara River, and much less numerous on the coast and on larger lakes and rivers of the interior. Banded ring-billed gulls from New York are frequently recovered in the southern United States or Central America. Eaton (1910 - in Bull 1975) spoke of it as a rare to uncommon visitant to upstate New York chiefly during migrations. Few species in the state have increased in numbers as dramatically as the ring-billed gull (Bull 1975). Goat Island, near Niagara Falls, had 400 pairs in 1959. New York DEC wildlife biologist Gordon Batcheller lists the ring-billed gull as abundant (pers. comm.).

The food habits of the ring-billed gull do not differ much from the herring gull. Approximately 50% of their diet is fish and the food is

primarily aquatic. Consumption is about 95 gms/day for the ring-billed gull. Whittow and Rahn (1984) list the average intake as 75 gms/day in their article on eggs, yolk and embryonic growth rates of sea birds. Sileo et al. (1977) recovered a high number of emaciated dead and dying ring-billed gulls during the fall migrations of 1969 and 1973. Gilberston (1985a) relates how autopsies and residue analysis of these specimens, tested at the University of Guelph, resulted in an improved method for testing the significance of multiple residues by calculation of an organochlorine index for residues in the brain. At times of stress such as post nuptial or post juvenile molt, contamination from Dieldrin, DDE and PCB's caused gull deaths (Gilbertson 1985a).

3.1.2.11 Great Blue Heron (Ardea herodias)

The great blue heron is our largest U.S. heron; in erect stance it is about 4 feet tall (Palmer 1976). The great white heron is of similar size. The great blue heron ranges throughout the United States and southern Canada. Breeding occurs as far south as Central America (Bull 1975). The great blue heron winters from South Carolina to extreme northern South America. The great blue heron is fairly common along the Niagara River, but is less common in New York during the winter (Palmer 1976).

About 85% of the great blue heron diet is fish according to an examination of 189 stomachs collected throughout the U.S. (Cottam and Uhler 1945-in Palmer 1976). Although great blue herons are most frequently found near rivers, swamps, or lakes, numerous reports of great blue heron are received during statewide surveys of upland forests. However, in habitat like the Niagara River or rivers and lakes in Michigan, a large part of their diet will be fish, often of good size (Alexander 1977).

Weights on great blue heron are rare in the literature. Cameron (1906 - in Palmer 1976) lists 2 kg as the weight of one bird, but 3 kg is a reported average (Alexander 1977) used to estimate volume of trout consumed. Based on a 3 kg average weight, it is calculated that the great blue heron consumes 600 grams per day, using the 20% food to body weight ratio as discussed in methods. Alexander (1977) uses a 33% food to body weight ratio for a daily intake estimate. Because captive birds of several species fell closer to 20%, we continue to use this number.

3.1.2.12 Green-backed Heron (Butorides virescens)

The green-backed heron is the second smallest U.S. heron. (Palmer 1976). Green-backed heron range throughout the neoarctic and neotropical regions, breeding from southeastern Canada, the United States (absent from the Northern Great Plains and Rocky Mountains) through the West Indies and Middle America to Panama and northern South America. It winters occasionally in New York and south to South Carolina (Bull 1975). The green-backed heron is fairly common along the Niagara River, is a widespread breeder in New York, but is rare at higher elevations in the Adirondacks and Catskills. It is very rarely seen in New York during the winter.

According to the results of a U.S. Fish and Wildlife study of 255 birds collected over a wide territory about half of the green-backed heron diet is composed of fish, white crustaceans (20%), insects (24%), and miscellaneous invertebrates made up the remainder (Cottam and Uhler 1945 - in Palmer 1976).

Size variation in green-backed heron is rather slight (Palmer 1976), and adults average about 0.25 Kg. Although daily intake of food is not presented in the literature, it is estimated that 50 grams per day of largely aquatic food is consumed. Virtually all of the

green-backed heron diet is animal material; seasonally, terrestrial insects can be important food items (Bryant 1914 - in Palmer 1976).

The average time span for young birds to go from nestling to flight stage is about 20 days (Palmer 1976). Meyerriecks (1960 - in Bull 1975) studied green-backed heron in New York and found that many green-backed heron raised double broods.

3.1.2.13 Mallard (Anas platyrhynchos)

The mallard is common in the Niagara River. "Mallards are among [New York's] most numerous ducks, especially in the western part of the State" (Bull 1975). Montezuma Refuge has by far the largest concentration of this species. Marsh land is the preferred habitat type. The fast flowing Niagara River is not ideal habitat, but mallards are year-round residents and raise broods there.

Mallards will on occasion eat great quantities of fish (3 times to 5 times normal), if available, and are opportunistic feeders. However, their normal diet is 90% plant material (10% aquatic animals), and the normal diet is 5% fish or less (Palmer 1976). McAtee (1918 - in Palmer 1976) examined 1578 stomachs in North America from 22 states and 2 Canadian provinces. Over half of the stomachs contained aquatic macrophytes, as either vegetative parts or seeds. Mallards eat many varieties of grains. The mallard, therefore, represents a year-round breeding resident which feeds primarily on plants, and is included in these biotic contamination calculations for comparison with fish eaters.

Mature mallards vary seasonally in weight but fall birds in Illinois averaged 1.25 kg for drakes and 1.08 kg for hens (Bellrose and Hawkins 1947). Other authorities have recorded similar weights although barnyard and captive birds are heavier on the average. Weight of ducklings increases very rapidly. Kear (1965) noted that birth

weight of ducklings doubles in a week and quadruples in two weeks. By the time the juvenile ducks weigh 50% of adult weight they consume amounts equal to adults. Mallards consume about 250 grams per day to maintain body weight. There are many cases of consumption of several times this amount which can often prevent mallards from flying until the food is digested. An author of this report has also witnessed mallards, during alewife die-offs on Lake Ontario, eating so much they could not fly. Similarly, he has witnessed mallards rendered immobile after gorging on dead trout that had been cleared off outlet screens in fish hatcheries.

Niagara Frontier mallards have been analyzed for several organochlorines: PCBs, 2.2 ppm; DDT, 0.707 ppm; dieldrin, 0.01 ppm; chlordane and metabolites, 0.115 ppm (R. Foley, NYS Dept. Envir. Cons. pers. comm.).

The mallard has received much attention in toxicological research with contaminants. Direct feeding studies with organochlorines and organophosphates have been conducted to establish acute toxicity to mallards (Hudson et al. 1984). These acute toxicities have been used to estimate the risk of mallards consuming large amounts of fish containing contaminants in amounts found in Lake Ontario and the Niagara River. However, data on chronic feeding still does not exist for each of the chemicals tested by Hudson et al. (1984). It is quite possible that since the mallard diet is largely herbivorous, the result is lower exposure to contaminants in the Niagara River than is experienced by piscivorous ducks.

3.1.2.14 Oldsquaw (Clangula hyemalis)

This small diving duck is abundant on the lower Niagara River during the winter (G. Batcheller pers. comm.). Bull (1975) lists them as a common to very abundant New York winter visitant along the coast and on the larger inland lakes, being most numerous in the Great Lakes

area and at the eastern end of Long Island. The oldsquaw was also abundant in former years as evidenced by the large numbers taken in gill nets from great depths on the Great Lakes -15,000 were found in nets from a haul on Lake Erie in May, 1917 (Palmer 1976). Most of these ducks were taken at 15 fathoms (about 90 feet deep).

As shown by gill netting data, oldsquaw most frequently dive for food which averages about 20% fish. Examination of the stomachs of 190 adults showed 88% animal matter consisting primarily of crustaceans, along with mollusks, insects, and fish (Palmer 1976). The remaining 12% vegetable matter is comprised of mainly grass seeds and pond weeds. Juvenile diets were similar to the adult diet. Fish percentage of the diet ranged from 10% to almost 100%. Loring (1880) found 52 small pike in a stomach from a N.Y. bird and Hull (1914 - in Palmer 1976) reported finding 140 two inch long shiners (Notropis atherinoides) in an oldsquaw stomach near Lake Michigan. Lake Michigan oldsquaw consumed 99% animal food of which 77% were amphipods and 18% were fish (Lagler and Wienert 1948). As discussed in the introduction the contaminant levels of the crustaceans may be as high or higher than fish (Whittle and Fitzsimmons 1983).

Bellrose (1976) presented a number of weights, recorded by Ellarson (1956), of oldsquaw removed from gillnets in Lake Michigan. Adult male oldsquaws averaged 0.91 kg-1.0 kg, adult females averaged 0.50 kg to 0.83 kg (the first figure represents birds with dry plummage, the latter represents birds with wet plumage). These averages were based on over 1300 birds. The weight for oldsquaw used in this report is 0.83 kg. Oldsquaw mature at 2 years and spend their summers on the arctic breeding grounds. Food intake to maintain body weight is estimated at 190 gm/day for the average adult male.

Organochlorine compounds were monitored in oldsquaw and their food from Lake Michigan between 1969-72 by Peterson and Ellarson (1978). Average residues in oldsquaw carcasses from L. Michigan ranged from 4 to 107 ppm PCB's, 2 to 42 ppm DDE, and 0.1 to 0.7 ppm endrin. Residues were relatively low in oldsquaw foods from Lake Michigan with a concentration factor from the food to the ducks calculated to be between 1X and 22X depending on the date and the compound. Peterson and Ellarson (1978) reported that organochlorines were significantly lower for arctic food than Lake Michigan food samples.

Residue levels in paired male and female oldsquaws were highly correlated, as were females and their egg clutches. DDE and PCB increased at a relatively constant rate throughout the winter, however, the food samples did not reflect the apparent build up of these residues. Part of this anamoly may be due to changes in amounts of fat. Mobilization of contaminants during periods of starvation were thought to threaten breeding females as well as the developing embryos (Peterson and Ellarson 1978).

3.1.2.15 Osprey (Pandion haliaetus)

Although almost cosmopolitan in distribution, osprey are now rare or absent in the Niagara River area. Bull (1975) lists the osprey as a "fairly common migrant along the coast and on Lake Ontario." The large fish hawk is the size of a small eagle and breeds on Long Island, in the Adirondacks, and along the St. Lawrence River. Osprey populations in general have declined since the 1940's. Northern ospreys migrate to warmer climates during winter. Banding recoveries from New York tagged ospreys are from North Carolina to South America (Bull 1975). Many ospreys winter in Central or South America where they may be exposed to

a considerable variety of organochlorines including DDT (Henny and Wright 1969; Henny and VanVelzew et al. 1972; Johnson, et al. 1975).

Osprey food is almost entirely fish (Sprunt 1955; Grossman and Hamlet 1964). Osprey are very skilled at fishing and have a number of anatomical features which allow them to catch and hold fish and to plunge into water. Fish such as saltwater catfish, Tomcod, carp, perch, sunfish and sucker are among those fish commonly taken. A variety of sizes are caught, some weighing up to four pounds (Grossman and Hamlet 1964). Osprey adults range in weight from 1.22 kg to 1.6 kg for the male to 1.25 to 1.9 kg for females. An estimate of 300 grams of food per day for a 1.5 kg osprey would appear appropriate based on a 20% food intake to adult body weight formula, although accounts of osprey feeding indicate that short-term intake exceeds this. Alexander (1977) suggested selecting a 33% food to body weight ratio for piscivorous birds.

The organochlorine threat to ospreys is particularly high. The contaminant levels of fish in the Niagara River for several compounds probably exceeds the tolerance level of the species. Even ospreys living in less contaminated areas of New York such as the Adirondacks, may be exposed to high contaminant levels in their wintering areas in Central or South America. Nearly all of northern osprey winter in the Caribbean Islands and in Central and South America (Henny and VanVelzew 1972). They disperse widely across South America, inhabiting coastal and inland river systems. The first year ospreys stay in the South and return in their second or third year to the area they were hatched.

Although osprey population declines have been attributed to various causes such as habitat destruction, human disturbance or decreased food supply, studies confirm that effects of environmental contaminants can

be important factors in the decline of this species (Wiemeyer et al. 1978). In a New Jersey study, Barnegat Bay and Avalon-Stone Harbor, high DDE residues and moderate PCB levels were both found in Osprey eggs and seem to have been the cause of reproductive problems exhibited by the birds (Wiemeyer et al. 1978). An Idaho population has also experienced a decline due to residues of DDT and PCB (Johnson et al. 1975). Eggshell thinning and embroyonic death due to these residues may result from exposures on nesting grounds, during migration or on wintering grounds; this points out that the osprey's life habits may make them particularly vulnerable to contaminant poisoning (Johnson et al. 1975).

Wiemeyer et al. (1975) tested the hypothesis that the decline in reproductive success was caused by something in the external environment of the eggs. High levels of dieldrin, DDE, and PCB's were found in Connecticut osprey eggs and chicks. The Connecticut osprey eggs were reared by Maryland parents and failed to hatch. When Maryland eggs with low contaminant levels were reared by Connecticut birds there was normal hatching success. Second batches of eggs were laid by the Maryland birds which were raised by their own parents at a normal hatching rate. This experiment provided further proof that osprey declines are due to contaminant burdens acquired by consuming fish contaminated with pesticides and other toxic chemicals.

Wiemeyer et al. (1975) measured contaminant levels in fish from both the Connecticut and Maryland study areas (Table 1.) and concluded that the basic difference was that the fish consumed by Connecticut ospreys were generally much higher in contaminants than the fish consumed by Maryland birds. It is logical to conclude that

environmental conditions on the breeding grounds will have a great bearing on breeding success.

The trend in contaminant residues in ospreys has not been favorable, no declines in DDT and DDE are apparent (Johnston 1978). The exposure of ospreys in their first and often second year in southern foraging areas is also a critical factor. Organochlorine and land use patterns in Central and South America may further limit osprey populations as previously suggested.

3.1.2.16. Other Birds

As this paper was nearing completion, unpublished data obtained from the N.Y. State Breeding Bird Atlas project (DEC in prep. a.) suggested that the pied-billed grebe, a confirmed breeder in the Niagara River area, and a consumer of smallfish and other aquatic life (Palmer 1976), may also be species of concern. According to Robert Miller (NYS Dept. of Envir. Cons. pers. comm.) double-crested cormorants and black-crowned night herons, while not confirmed breeders on the Niagara River, are both piscivorous species and vistants to the area that might also be considered species of concern.

3.1.3. Reptiles

3.1.3.1 Snapping Turtle (Chelydra serpentina)

"The common snapping turtle is more widely distributed in North America than any other turtle" (Carr 1952). They are probably common on the Niagara River. Snapping turtles are believed to be quite resistant to contaminants and therefore serve to monitor pollutants in aquatic systems (Stone et al. 1980; Helwig and Hora 1983). Hammer (1969) felt that snapping turtles were good indicators of local environmental conditions because they are long-lived, relatively sedentary, and tolerant of contaminants.

Snapping turtles eat both plant and animal material (Pell 1940 - in Carr 1952; Lagler 1943). Pell (1940 - in Carr 1952) found considerable variation in diet from one habitat to another, and noted that plant and animal material was almost equally represented in specimens from New York and Massachusetts. Lagler (1943) noted that larger snappers used very few small forage fish, concentrating on sub-legal game fish. Captive turtles are frequently fed vegetable material only. In addition to fish, plants, crustaceans and invertebrates, snapping turtles are well known predators on ducklings and almost any waterfowl they can catch.

Volume of food consumed by snapping turtles per day is estimated to be 10% of the body weight per day, as Alexander (1977) assumed for water snakes. A 9 kg adult snapper then might consume 900 grams of food per day or 450 grams of fish if they were 50% of the diet.

Snapping turtles in New York have high contaminant residues; measurements in the 800 to 1600 ppm range for DDE and PCB have been recorded (Stone 1980). Levels of DDE and PCB averaged less than 0.08 ppm in loggerhead turtles eggs and even lower in green turtle eggs sampled in Florida Island (Clark and Krynitsky 1980). These low Florida contaminant levels in sea turtle eggs suggest relatively uncontaminated food supplies.

3.2 Chemical Narratives

3.2.1 Polychlorinated Biphenyl (PCB)

PCBs are organic compounds containing from 18 to 79% chlorine, which are formed by the chlorination of biphenyls. The principal commercial PCBs have a chlorine content from 42 to 60% (Hammond et al. 1972) with the extent of chlorination depending on their intended use. These compounds are highly stable. They are not hydrolyzed in water, an acid medium, or an alkaline medium (Hascoet et al. 1978). Between 1930 and 1975, more than 630 million kg of PCBs were manufactured domestically (Safe 1984). There are 209 synthetic organochlorines classed as PCBs and they have been used extensively as heat transfer agents, lubricants, dialectric agents, flame retardants, plasticizers, and water proofing materials (Roberts et al. 1978).

Due to human activities and the chemical characteristics of the products, PCBs are now distributed world-wide, with measurable concentrations reported in aquatic organisms and wildlife of North America, Europe, the United Kingdom, and the Atlantic and Pacific Oceans (Eisler 1986a). Eisler (1986a) has produced a synoptic review of PCB hazards to fish, wildlife and invertebrates in which he details a variety of biological and toxic effects including death, birth defects, reproductive failure, liver damage, tumors, and a wasting syndrome. PCBs are known to bioaccumulate and biomagnify within the food chain and have been banned from all U.S. use and manufacture since 1979 (Eisler 1986a).

Biological activities of PCB isomers differ substantially (Eisler 1986a). Aroclor toxicity has been found to be positively related to chlorine percentage (last two digits of Aroclor number) by Heath et al. (1970). In the rat the single oral LD50 is 1,010 mg/kg, with a LD_{lo} of

188 mg/kg (NIOSH 1982). Rats fed diets of Aroclor 1254 totalling 1,000 mg/kg all died in 53 days (Hudson et al. 1984). Eisler (1986a) concluded that the total (sum of exposures) rat lethal dietary level of Aroclor 1254 is from 500 to 2,000 mg/kg for 1 to 7 week exposures. Bio-test Laboratories (1970) exposed rats to a diet of 6.25 mg/kg/day (Aroclor 1254) for 2 years without significant mortality, establishing this as a NOEL for mortality. The exposure of 28 mg/kg/day Aroclor 1254 (NCI 1978a) resulted in stomach lesions and cancer in rats exposed for 2 years. Spencer (1982) however, reported reduced fetal survival from 3.14 mg/kg of Aroclor 1254 in the daily diet of female rats during 9 days of pregnancy.

Marks et al. (1981) reported that mice exposed to 3,3',4,4',5,5'-hexachlorobiphenyl in gastric doses of 2 mg/kg/day had significantly more deformed offspring and fewer offspring per litter. Mice exposed to gastric doses as low as 1 mg/kg/day showed discolored livers in Marks et al.'s (1981) research. Talcott and Koller (1983) reported higher NOEL and LOELs with Swiss-Webster mice which appear to be PCB resistant.

Mink have been exposed to PCB in the laboratory. The commercial fisheries of the Great Lakes had provided the mink ranching industries of the North Central U.S. and Canada with an inexpensive supply of fish for mink feeding (Aulerich and Ringer 1977). However, in 1965, Hartsough reported reproductive complications and excessive kit mortality in mink fed these fish. A number of years of research have established that PCB is toxic to mink (Aulerich and Ringer 1977).

Aulerich and Ringer (1977) found 10 ppm PCBs in Great Lakes salmon and demonstrated that diets of even 2 mg/kg (0.48 mg/kg/day) for 4 months, resulted in nearly complete reproductive failure of mink.

Further research has proven that PCBs, and not some other factor, are the cause of these problems in mink. Ringer et al. (1973) found that reproduction was impaired with 16 week exposures to Great Lakes contaminated fish, with a LOEL of 0.225 mg/kg/day (1 ppm) and placed the NOEL at 0.1 mg/kg/day.

PCB toxicity varies with isomers. Some isomers are of low toxicity and others are considerably more toxic. Therefore, the approximate composition of a PCB mixture by isomer groups is required to estimate toxicity. Aroclor 1016 and Aroclor 1254 are the most prevalent PCB's in the Hudson River with Aroclors 1254 determined to be much more persistent than 1016 (DEC 1986a). Over 50% of the total PCBs in Niagara River and Lake Ontario fish is 1254 and the next most prevalent is 1260. Fortunately very little is present as 3,3',4,4',5,5'- hexachlorobiphenyl as it has proven very toxic relative to many other PCB isomers.

Exposure of mink to hexachlorobiphenyls such as 3,3',4,4',5,5' -hexachlorobiphenyl as low as 0.1 mg/kg produced an LD50 in 3 months and completely inhibited reproduction (Aulerich et al. 1985). No adverse reproductive effects were noted with 2,3,6-HCBP or 2,4,5-HCBP. Aulerich et al. (1985) concluded that even 0.1 mg/kg (0.0225 mg/kg/day) 3,3',4,4',5,5'-Hexachlorobiphenyl produced a number of toxic effects . Mink are among the most sensitive species to PCBs, and are the most sensitive wildlife species tested to date (Eisler 1986a).

The European ferret is at least three times more tolerant of PCB's than mink (Bleavins et al. 1984) even though they are closely related. Bleavins et al. (1984) found complete reproductive failure at 4.8 mg/kg/day Aroclor 1254 after a 4 month feeding trial, with a LD50 estimated at 20 mg/kg/day.

Zepp and Kirkpatrick (1976) report 1 mg/kg/day as the NOEL for the cottontail rabbit, with a LD50 of about 10 mg/kg/day Aroclor 1254 for a 12 week period. Domestic rabbits (Koller and Zinkl 1983) and raccoon proved more tolerant (Montz et al. 1982).

Birds were more resistant to acutely toxic effects of PCBs than mammals (Eisler 1986a). LD50's for birds varied from 604 to more than 6,000 mg/kg (Eisler 1986a). The LD50 for the mallard was greater than 2000 mg/kg total dose and depended on chlorine content of the toxicant (Heath et al 1972). When PCB residues in the brain reach 310 mg/kg there is an increased likelihood of death from PCB poisoning (Eisler 1986a). Residues of PCB in the brain of greater than 310 mg/kg can probably be used to identify PCB killed birds in the field (Stickel et al. 1984).

Although birds may be resistant to acute short term exposures to PCB, chronic dietary trials have been remarkable for demonstrating adverse effects at low levels. Nine week exposure of Aroclor 1248 in the diet of the white leghorn chicken caused reproductive losses with doses as low as 2.24 mg/kg - the 0.224 mg/kg/day dose level can be selected as the NOEL (Britton and Huston 1973). Subsequent studies with chickens have confirmed these approximate LOELs and NOELs (Platonow and Reinhart 1973; Lillie et al. 1975).

Mallards fed PCB at concentrations as low as 7.8 mg/kg/day (25 mg/kg in diet) for 10 days suffered no apparent clinical intoxication. However, when these birds were challenged with duck hepatitis virus they suffered significantly higher mortality than birds not exposed to PCB's (Friend and Trainer 1970). Loose et al. (1977) investigated the apparent reduction in lot health and lack of resistance to disease in birds exposed to PCBs and attributed the effects to suppressed immune

response. The suppressed host resistance in birds exposed to PCBs, followed by disease, may be associated with the suppressed immune response which Loose et al. (1977) demonstrated.

Calculation of Wildlife NOEL of total PCBs in Fish

PCBs have been rather extensively tested for toxicity to both laboratory birds and mammals, and several wildlife species. Thus far the mink has been the most sensitive species tested. However the list of Niagara River wildlife which consume fish contains several species which have not been tested under laboratory conditions. Review of residue literature concerning these species indicates that mink would still be the species to first develop clinical signs of PCB intoxication, and that some species, especially the snapping turtle, would prove highly tolerant. Furthermore, only a portion of the 209 PCB isomers have been toxicologically tested, and of these 3,3',4,4',5,5'-hexachlorobiphenyl is emerging as one of the most toxic isomers to mink.

The mink data from Platonow and Karstad (1973) is the basis of the fish flesh criterion of 0.13 mg/kg calculated below. Treatment levels used by Platonow and Karstad (1973) did not include diets lower than the 0.64 mg/kg in the mink diet. The criterion of 0.13 mg/kg is considerably less conservative than 1.5 ug/kg bodyweight (about 0.01 mg/kg diet) which Eisler (1986a) estimated as the tolerable daily limit for mink. Eisler derived this criterion using the Platonow and Karstad LOEL of 0.64 mg/kg, study mink weights and food consumption, and a safety factor of 100. It is recommended in this study to apply a factor of 0.2 to estimate a NOEL from a LOEL. The mink data from Ringer et al. (1983) established 0.1 mg/kg/day as the NOEL (about 0.67 mg/kg diet), about five times greater than the estimated fish flesh criterion of 0.13 mg/kg.

Table 8 summarizes data from dietary exposures of PCB in birds and mammals. For comparison with the above empirically derived PCB wildlife NOEL, several other NOELS could be used and appropriate risk factors applied to calculate criteria for comparison with the mink based criterion.

1. Determining wildlife application uncertainty factors for PCB dietary exposure based on target and non-target birds and mammals. Refer to Table 8 for details of data selected below.

Study	Duration	Effect at LOEL	NOEL/LOEL mg/kg/d (mg/kg-diet)	Recommended Application(AF)/ Uncertainty(UF) Factor
Mink	4 months	Reproduction impaired	0.64 (LOEL)	0.2 (LOEL to NOEL AF)
Cottontail Rabbit	12 weeks	No higher treatments used	1.0 (NOEL)	0.1 (Sub-acute to chronic AF)
Chicken	9 weeks	Reproduction loss at LOEL	0.224 (NOEL)	0.1 (Interspecies UF)
Mouse-1	28 days	Some mortality and deformed offspring	2.0 (LOEL)	0.2 (LOEL to NOEL AF)
Rat-4	9 days during pregnancy	Fetal survival potential	3.14 (LOEL)	0.2 (LOEL to NOEL AF)

2. Calculation of criteria:

-Rabbit NOEL

1.0 mg/kg/day X 0.1 (AF) X 1 kg (mink weight) ÷ 0.15 kg/day (mink daily intake) = 0.66 mg/kg

-Chicken NOEL

0.224 mg/kg/day X 0.1 (UF) X 1 kg ÷ 0.2 kg/d = 0.11 mg/kg.

-Mouse LOEL

2.0 mg/kg/day X 0.2 (AF) X 1 kg ÷ 0.15 kg/d = 2.7 mg/kg

-Rat LOEL

3.14 mg/kg/d X 0.2 (AF) X 1 kg ÷ 0.15 kg/d = 4.2 mg/kg.

-Mink LOEL

0.64 mg/kg X 0.2 (AF) = 0.13 mg/kg.

The chicken based criterion is comparable to the criterion derived using the target species, mink data. The other tentative criteria would almost certainly result in reproductive impairment in the mink and any other highly sensitive species yet untested. The far less conservative rat based criteria without interspecies adjustment would probably cause reproductive failure and outright mortality. The International Joint Commission (a United States-Canada Treaty Organization 1981), has set a PCB objective in fish of 0.1 ug/g to protect piscivorous wildlife. The objective was derived by applying a factor of 0.2 to the LOEL of 0.64 ug/g found by Platonow and Karstad (1973).

Carcinogenic Data for PCBs

PCBs have been determined to be carcinogenic (IARC 1978; NCI 1978a; Kimbrough et al. 1975). Twenty-one month exposures of laboratory rats from Kimbrough et al. (1975) were used for extrapolation to a lifetime 1 X 10^{-6} cancer risk for the experimental animals of 0.0017 ug/kg/day (DOH

1985a). Conversion of this dose to the dose that would correspond to the 1×10^{-3} and 1×10^{-2} risks of cancer involves the following steps.

1. 1×10^{-6} increased cancer risk dose in the rats = 0.0017 ug/kg/day = 1.7×10^{-6} mg/kg/day.
2. 1×10^{-3} increased cancer risk dose in rats = 1.7×10^{-3} mg/kg/day.
3. Assume that the same dose will result in equal risk for rats and mink. Then convert the rat dose to a mink dietary criterion:
   1.7×10^{-3} mg/kg/d X 1 kg ÷ 0.15 kg/d = 11×10^{-3} mg/kg = 0.011 mg/kg, 1 in 1000 cancer risk in diet.
4. The 1 in 100 cancer risk in diet = 0.11 mg/kg.

Comparison of Criteria with Residue Data

The median for PCB in spottail shiners in the Niagara River in 1981 and 82 was 0.327 mg/kg, with a maximum of 1.683 mg/kg (Tables 3,4, and 25).

FDA measurements of total PCBs in Niagara River white bass were reported as 18.0 mg/kg (Table 5) (FDA 1977). Aulerich and Ringer (1977) found 10.0 mg/kg in Great Lakes salmon. Norstrom et al. (1978) reported 2.21 mg/kg in alewives and smelt from western Lake Ontario in 1976, and 8.17 mg/kg in coho salmon muscle, and 6.16 mg/kg total PCBs in coho salmon liver (See Table 6). Recent measurements of PCB in several salmonid species from Lake Ontario ranged from 1.14 to 9.31 mg/kg; concentrations in non-salmonid fish in the Niagara River ranged from 0.18-5.29 mg/kg (Table 7).

Spottail shiner PCB residues are probably toxic to mink. Many stations exceeded the 0.13 mg/kg wildlife criterion in 1982 and the median of 0.327 mg/kg is well above the non-carcinogenic based

criterion. For other fish species total PCB residues exceeded the estimated the criterion by 15 to over 100 times. Residues in many fish species exceed dietary NOELS for a number of species tested. All fish species, including spottail shiners, exceed the 1 in 100 cancer risk level. The firm conclusion is that the sensitive wildlife fish consumers are at risk from eating fish from the Niagara River based on PCB residues alone, and that PCB exposure should be reduced. Examination of several species NOELs and LOELs suggest that more tolerant species at present PCB levels may be subject to marginal toxicity, also.

3.2.2 DDT, DDD, DDE

DDT (1,1,1-trichloro-2,2-bis [p-chlorophenyl]ethane) is one of the few insecticides which has a strong potential for food chain magnification (Macek and Korn 1970). In 1938 a Swiss chemist, Paul Mueller, discovered that DDT was a very potent insecticide and was soon widely used in that capacity. Technical grades are a mixture of several similar compounds which all have insecticidal properties; the technical grade was not refined for commercial use (Berg 1983).

However, in the 1960's, evidence of DDT persistence and toxicity to non-target species began to surface. Eggs of lake trout lost viability when the DDT concentration in the eggs reached levels of 2.9 mg/kg or above (Burdick et al. 1964). Increasing bioaccumulation of DDT with successive trophic levels has been reported in field surveys (Woodwell et al. 1967) and laboratory studies (Macek and Korn, 1970; Grzenda et al. 1970). Food is believed to be a primary source of DDT to non-target species (Eberhart et al. 1971). Reinhart (1970) reported residue accumulations of up to 2 million times background water concentrations (1-5 ng/l) in Lake Michigan coho salmon.

DDT is also made up of DDD and DDE in technical grades and DDT metabolizes to these products to varying degrees (Mitjavila, Carrera and Fernandez 1981). Often DDE is the highest quantity recovered in DDT related compound residue analysis. Radomski et al. (1968) showed DDE was accumulated in preference to DDT in man. When DDT exposures are administered episodically or at very low concentrations, DDE accumulates most (Durham et al. 1961), although Mitjavila, Carrera, Biogegrain, and Derache (1981) found DDT was the primary storage contaminant in chronic feeding studies with the rat.

Macek et al. (1970) found that fish accumulate a considerable amount of DDT residues from food -- for wildlife species, food may also be the primary source. Reinert et al. (1971) suggested that cooking and eating lean muscle regions of Lake Michigan salmon removed a large part of contaminants in fish used for human consumption, since DDT and several other organochlorines are stored in the high oil content portions. Obviously, wildlife fish consumers do not consume just fillets, and may be required by a changeable environment to consume the fatty and high energy portions, which in turn lead to higher contaminant exposure. The contaminants will in turn be deposited in fat which can mobilize during periods of greater energy demands or periods of starvation.

The single dose acute oral LD50 for the rat exposed to technical grade DDT is 113 mg/kg (Verschueren 1983). Rats exposed to 14.5 mg/kg/day up to 52 days evinced few effects on growth, food intake, body composition and activities of various enzymes, but total lipid levels fell 30% and the weight of the liver rose 20% due to cellular hypertrophy induced by DDT (Mitjavila, Carrera, Biogegrain, and Derache 1981). Chadwick et al. (1975) exposed rats to 5.0 mg/kg in their diet (dose of 0.375 mg/kg/day) and found increased enzyme induction. The LOEL for the rat, therefore, is equal to or lower than 14.5 mg/kg/day

and possibly close to 0.375 mg/kg/day, depending on the interpretation of the severity of these non-lethal effects. It can be concluded from examining toxicity tests of other species, that the rat is a species of average DDT sensitivity (Table 8).

Relatively low doses of DDT induce the mixed-function oxidase system of the endoplasmic recticulum, which is believed to be a factor in thinning egg shells of a number of bird species (Hickey and Anderson 1968; Longcore and Samson 1963).

Black duck hens fed a diet of 10 mg/kg DDE laid eggs with shells about 30% thinner than controls (dose of approximately 2 mg/kg/day) and produced 1/5 as many ducklings as the control hens (Longcore and Samson 1973). The resultant egg concentration was 64.9 mg/kg (wet weight) DDE which the EPA (1976) interpreted as a 10-fold increase over the concentration in the food. Heath et al. (1969) reported similar eggshell thinning with mallards exposed to DDE at the same dose levels of 2 mg/kg/day.

Human volunteers have been exposed to dietary concentrations of DDT of up to 35 mg/kg (dosage of 0.61 mg/kg/day) for periods of up to 21 months with no apparent symptoms (Hayes et al. 1971).

Reports of illness in humans from DDT exposure were absent despite the widespread dependence on DDT as an insecticide. Many toxicity tests were conducted with DDT before evidence of ecosystem contamination lead to restrictions in use. As a result of numerous DDT tests we can compare laboratory bird and mammal results with those of wildlife species tested under controlled conditions.

Field Studies of DDT Applications

Accidental DDT contamination of the Wheeler National Wildlife Refuge by a U.S. Army installation at the Redstone Arsenal resulted in

high DDT biotic contamination (Shea et al. 1980). Cormorants and herons declined at the Wheeler Refuge. Nesting eagles disappeared. High residues in biota on the refuge furnished some proof that DDT contamination was a large factor in this degradation. A 13 ha plot of crop land was treated experimentally at a low rate of 0.22 kg/ha DDT in Great Britain (Rudd et al. 1981). A two compartment mode of uptake occurred, one fast, the other slow. The carnivorous shrew was totally absent after DDT application, suggesting that its high metabolism and high trophic level placed the shrew at risk even though mammals are considered less sensitive to DDT than birds.

Lab Studies of Wild Species

House sparrows fed a diet of 100 mg/kg of DDT contaminated food (approximate dosage of 20 mg/kg/day) began to die after 41 days of exposure, although several survived 90 days of exposure. Sacrificed birds were generally found to have less than 50 ug DDT/g in the brain, while those that died before 90 days had more than this amount (Bernard 1973). Starvation in DDT exposed house sparrows significantly reduced exposure time required to kill birds; DDT is apparently released from less sensitive tissues of the body to more vital sites under starvation conditions (Bernard 1963). Non-captive house sparrows dying with tremors on the Michigan State University Campus closely matched the signs of intoxication (tremors) of experimentally poisoned birds. Stickel et al. (1966) experimentally fed Alaskan bald eagles at dietary concentrations of 5, 83, 414, and 2070 mg/kg mixed with ground salmon and other waste fish products. The eagles fed the 5 mg/kg diet (0.3 mg/kg/day dosage) were not visibly effected. Mortality and gross intoxication was typical at higher feeding levels (including the 83 mg/kg treatment level which constitutes a 4.98 mg/kg/day dosage).

One eagle of the five tested died at the 0.3 mg/kg/day dosage, but Stickel et al. (1966) believed this to be due to other factors. The authors concluded that these direct feeding studies with a key wildlife species indicate that the bald eagle is not overly susceptible to DDT poisoning compared to other species tested at the Patuxent Wildlife Research Center by the same authors and their colleagues.

Long-term tolerance limits for the mallard duck is at or below 8.0 mg/kg/day and is 5.0 mg/kg/day for Coturnix and bobwhite quail (Stickel et al. 1966). The long term tolerance limit is a LD_{lo} and certainly not a chronic LOEL comparable to the 2.0 mg/kg/day reported as a LOEL for the mallard by Heath et al. (1969), and the same LOEL also reported for the closely related black duck by Longcore and Samson (1973).

Stickel et al. (1966) concluded that the hazard zone of DDT residues in eagle tissues is about 30 mg/kg. These authors also concluded that a number of species (meadowlark, cottontail rabbit, teal, lesser scaup, and shoveler) were about as sensitive as eagles. DDT levels of about 30 mg/kg brain residue are lethal to birds.

Blus et al. (1971, 1972) examined eggshell thinning in the brown pelican. Eggs were collected from 12 colonies in South Carolina, Florida, and California. The level of DDE in the eggs which did not cause thinning was estimated to be 0.5 mg/kg. However, EPA (1976) concluded that a conservative estimate of the NOEL in eggs was 2.0 mg/kg based on the data of Blus et al. (1972). The EPA (1976) then reasoned that a 10-fold increase from food to egg residues used for black duck (Longcore and Samson 1973) could be used to estimate a NOEL diet for the brown pelican of 0.2 mg/kg. Blus et al. (1971) consider the brown pelican to be extremely susceptible to DDE-induced eggshell thinning.

Although direct feeding studies of key wildlife species such as mink and bald eagle are very valuable for the purposes of this study, use of some species regarded as rare or endangered (e.g. bald eagle) is more unlikely now than the study by Stickel et al. (1966) in the 1960s. Wiemeyer et al. (1986) used surrogate species to examine the contamination role in the decline of the California condor. Likewise surrogate species will have to be used to monitor contaminant levels of valuable species in the Niagara River and Great Lakes area such as sampling herring gull eggs, nestlings, or adults.

Calculation of DDT, DDD, and DDE Wildlife Fish Flesh Criteria

A variety of toxicity tests could be employed to calculate the wildlife fish flesh criteria. Table 9 summarizes data from dietary exposures of DDT in birds and mammals.

1. Determining wildlife application/uncertainty factors for DDT and DDD or DDE dietary exposure based on target and non-target birds and mammals. Refer to Table 9 for details of data selected below.

Study	Duration	Effect at LOEL	NOEL/LOEL mg/kg/d (mg/kg-diet)	Recommended Application (AF)/ Uncertainty (UF) Factor
Mallad/Black duck	6 months & 2 laying seasons	fewer ducklings & egg shell thinning	2.0 (LOEL)	0.2 (LOEL to NOEL AF)
Bald eagle	120 days	mortality	0.3(5.0) (NOEL)	None
Brown Pelican	8 weeks	reproductive impairment	(0.2) (NOEL)	None
Rat	6 months	MFO induction	0.375 (LOEL)	0.2 (LOEL to NOEL AF)

2. Calculation of criteria:

-Mallard/Black duck LOEL

10 mg/kg X 0.2 (AF) = 2 mg/kg

-Bald Eagle NOEL

NOEL = 5 mg/kg in diet

OR

0.3 mg/kg/d X 1 kg (bird weight) ÷ 0.2 kg/d (bird intake) = 1.5 mg/kg.

-Brown Pelican NOEL

2.0 mg/kg in eggs ÷ 10 (Biomagnification factor) = 0.2 mg/kg

-Rat LOEL

0.375 mg/kg/d X 0.2 (AF) X 1 kg (mink weight) ÷ 0.15 kg/d (mink intake) = 0.5 mg/kg

This example of possible criteria developed from four species (mallard/black duck, bald eagle, brown pelican, and laboratory rats) illustrates the variability in species sensitivity to DDT and metabolites. The most protective criteria is derived from the brown pelican data with the rat based criterion only slightly higher. The mallard/black duck or bald eagle derived criteria would be the least protective but are not greatly different from the brown pelican and rat based criteria. It is concluded that the safe fish flesh criterion to protect sensitive species would be 0.2 mg/kg in whole fish supported by the brown pelican study.

Calculation of Cancer Risk Criteria

The above calculations are for non-carcinogenic effects. DDT and its metabolites have been found to be carcinogenic (Thorpe and Walker 1973). The lower 95% confidence limit value of the DDE dose corresponding to an increased lifetime cancer risk of 1×10^{-6} for the experimental animals was 0.004 ug/kg/d (DOH 1983). Conversion of this dose to the doses that correspond to the 1×10^{-3} and 1×10^{-2} risks of cancer involves the following steps:

1. 1×10^{-3} risk for the experimental animals is = 0.004 mg/kg/day.
2. Assume that the same dose will result in equal risk for mice and mink then convert the mouse dose to a mink criterion: 0.004 mg/kg X 1 kg ÷ 0.15 kg contaminated fish/day = 0.0266 mg/kg, 1×10^{-3} risk level.
3. A 1/100 increased lifetime cancer risk for mink due to DDE contaminated diet would be 0.266 mg/kg.

Comparison of Criteria with Residue Data

The median DDT residue in spottail shiners in the Niagara River in 1981 and 1982 was 0.031 mg/kg, with a maximum of 0.189 mg/kg (Tables 3,4, and 26).

Alewife and smelt DDE levels averaged 0.47 mg/kg and coho salmon 0.97 mg/kg (muscle) and 0.41 mg/kg (liver) DDT levels as reported by Norstrom et al. (1978) for western Lake Ontario (Table 6). Recent measurements of DDT in several salmonid species from Lake Ontario ranged from 0.38-2.77 mg/kg; concentrations in non-salmonid fish in the Niagara River ranged from 0.02-0.81 mg/kg (Table 7).

Spottail shiners do not exceed the 0.2 mg/kg non-carcinogenic based fish flesh criterion. However, residues in a number of other fish species are 2-10 times the criterion.

Spottial shiners in the Niagara River contain total DDT and metabolites in excess of the 1 in 1000 cancer risk criterion (NRTC 1984). Alewives, smelt and coho salmon in western Lake Ontario contain DDE in excess of the 1 in 100 cancer risk criterion (Norstrom et al. 1978). ≤ DDT measurements of salmonids from Lake Ontario, eel and some of the smallmouth bass from the Niagara River, exceed the 1 in 100 cancer risk criterion based on DDE effects (Table 7).

3.2.3 Aldrin and Dieldrin

Aldrin and dieldrin are members of the chemical family called chlorinated cyclodienes. Aldrin is the common name for a technical grade product containing at least 95% pure aldrin. Dieldrin is the common name for a technical-grade product containing at least 85% pure dieldrin. Since 1974, the use of aldrin and dieldrin has been restricted to underground termite control (NIOSH 1978).

Both compounds are readily absorbed after ingestion, inhalation or dermal exposure (IARC 1974). Aldrin applied as an insecticide is readily converted to dieldrin via epoxidation in a number of animals (NIOSH 1978). Dieldrin is the primary metabolite stored in fat tissue.

Surveys of humans in the U.S. demonstrate the widespread nature of aldrin and dieldrin contamination. Kutz et al. (1974) found dieldrin in about 99% of 7,000 human fat tissue samples from 48 states, with mean levels of 0.27 to 0.30 mg/kg. A number of reports of human illness have been recorded (NIOSH 1978), including death. Estimates of dosage of approximately 10 mg/kg (single exposure) for human mortality have been made (Hayes 1967).

Aldrin is extensively converted to dieldrin in all ecosystem components according to microcosm sudies (Metcalf et al. 1973). In Metcalf et al.'s (1973) model, ecosystem residues of aldrin/dieldrin in fish were 95.9% dieldrin in 33 days. Therefore, the majority of aldrin/dieldrin residues are as dieldrin. Results of the following animal toxicity tests indicate that both the parent compound and metabolites are highly toxic and that fish contaminant levels should be based on the sum of aldrin/dieldrin.

Dieldrin residues in experimentally poisoned birds versus residues found in wild birds.

There have been many instances of acute poisoning resulting from wildlife eating food contaminated with dieldrin (Stickel et al. 1969; Flickinger 1972). The level of dieldrin in the brain that causes death has been determined in several laboratory studies, and averages 6.8 mg/kg (Heinz and Johnson 1981). Nationwide monitoring of bald eagles which were autopsied and found to have died from various causes, points to dieldrin (brain levels of 6.8 mg/kg were considered diagnostic of dieldrin poisoning) as a leading lethal contaminant (Prouty et al. 1977). Heinz and Johnson (1981) concluded that brain levels as low as 1 mg/kg dieldrin in highly sensitive individuals may prove hazardous to birds by triggering irreversible starvation. Dieldrin is stored in body fats and even sub-lethal levels can cause starvation. Once the starvation process has begun, mobilization of dieldrin to the brain could lead to death.

Animal Laboratory Studies

Acute, subacute and chronic studies of experimental animals exposed to aldrin and dieldrin have been summarized by Hodge et al. (1967). For

twelve species of animals the acute lethal doses (LD50) for both compounds ranged between 20-70 mg/kg.

There are numerous long-term dietary studies of aldrin and dieldrin in mammals and birds. Since dieldrin is the primary residue, dietary toxicity of that contaminant is emphasized.

Subacute and chronic dietary toxicities of aldrin/dieldrin

Induction of liver microsomal enzymes has been selected as a toxicity endpoint in several subacute animal feeding studies. Male rats were fed dieldrin levels of 2, 5, 10, 20 and 50 mg/kg for two weeks (den Tonkelaar and vanEsch 1974). The lowest effect level for statistically significant enzyme induction was 5 mg/kg diet and the NOEL dietary level was 2 mg/kg.

Long-term dietary exposures of laboratory animals to dieldrin have resulted in liver damage at quite low levels. Liver histopathology was found at 1.0 mg/kg diet in the rat (Treon et al. 1955; FAO/WHO 1978).

Aldrin and dieldrin have caused diverse reproductive effects in animals including birth defects at higher dose levels, reduced fertility of dams and reduced survival of offspring (DEC 1986b). Chernoff et al. (1975) exposed female rats from day 7 to 16 of gestation during reproductive tests of dieldrin. The NOEL was reported as 3 mg/kg/day (Table 10). Harr et al. (1970) exposed rats at 0.08 to 40 mg/kg dietary levels in a long term study. In rats fed 0.31 to 1.25 mg/kg there was a slight reduction in survival of litters and a marked reduction in conception (73% - 1st mating, 33% - 2nd mating). At a higher dietary level of 2.5 to 10 mg/kg, females survived, but the nursing pups starved or died of convulsions. Birds are also sensitive to aldrin/dieldrin. Hungarian partridge and mallard exhibit reproductive LOELs of 1 and 3 mg/kg in diet, respectively (Neill et al. 1969 - in EPA 1976 and Lehner and Egbert 1969).

Calculation of Aldrin/Dieldrin Wildlife NOELS

Tables 10 and 11 summarize data from dietary exposures of aldrin and dieldrin in birds and mammals. Chronic dietary exposure of rats to dieldrin represent the lowest NOEL levels (Harr et al. 1970; FAO/WHO 1978). Data for several birds and mammals representing the range of sensitivity are presented below and used to calculate criteria.

1. Determining wildlife application/uncertainty factors for aldrin/dieldrin dietary exposure based on target and non-target animal data. Refer to Table 11 for details of data selected below.

Study	Duration	Effect at LOEL	NOEL/LOEL mg/kg/day (mg/kg-diet)	Recommended Application (AF)/ Uncertainty (UF) Factor
Rat-2	4 months	Decreased survival of young	0.018 (NOEL)	None
Rat-3	7-16 day Gestation	Histopathology reduced survival of young	0.30 (NOEL)	
Dog-1	1 year	Liver damage	0.025 (NOEL)	None
Dog-2	1 year	reduced survival of pups	0.2 (NOEL)	None
Monkey	6 years	Liver enzyme induction	0.1 (NOEL)	None
Mallard	4 months	20% eggshell thinning	(3.0) (LOEL)	0.2 (LOEL to NOEL AF)
Hungarian partridge	1 year	Reduced reproduction	1.0 (LOEL)	0.2 (LOEL to NOEL AF)

2. Calculation of criteria:

-Rat-2 NOEL

0.018 mg/kg/d X 1 kg (mink wt) ÷ 0.15 (mink intake) = 0.12 mg/kg.

-Rat-3 NOEL

0.3 mg/kg/d X 1 kg (mink wt) ÷ 0.15 kg/d (mink intake) = 2.0 mg/kg.

-Dog-1 NOEL

0.025 mg/kg/d X 1 kg (mink wt) ÷ 0.15 kg/d (mink intake) = 0.16 mg/kg.

-Dog-2 NOEL

0.2 mg/kg/d X 1 kg (mink wt) ÷ 0.15 kg/d (mink intake) = 1.33 mg/kg.

-Monkey NOEL

0.1 mg/kg/d X 1 kg (mink wt) ÷ 0.15 kg/d (mink intake) = 0.67 mg/kg.

-Mallard LOEL

3.0 mg/kg X 0.2 (AF) = 0.6 mg/kg

-Hungarian partridge LOEL

1.0 mg/kg X 0.2 (AF) = 0.2 mg/kg

(Note: Mallard data was left as dietary concentration because conversion to dose and then back to dietary criterion would use the same constant; data was unavailable to convert the Hungarian partridge data to the dose form).

The value of 0.12 mg/kg is selected as the final non-carcinogenic based criterion.

Aldrin has not been shown to be carcinogenic in rats, but aldrin and dieldrin were shown to be carcinogenic in mice (Walker et al. 1972).

The bioassay of Walker et al. (1972) is the basis of New York State's ambient surface water quality guidance value for sources of drinking water (DOH 1984b). Dose-response data from Walker et al. (1972) were used for extrapolation. The lower 95% confidence limit value for the dieldrin dose corresponding to an increased lifetime cancer risk of 1 X10^{-6} for the experimental animals was 3.3 X 10^{-4} ug/kg/day. Conversion from the 10^{-6} risk as a dose in the experimental animals to a 1 in 1000 and 1 in 100 risks in diet for a wildlife consumer involves the following steps.

1. 1 X 10^{-6} increased cancer risk in mice = 3.3 X 10^{-4} ug/kg/d
2. 1 X 10^{-3} risk in mice = 3.3 X 10^{-1} ug/kg/d
3. 3.3 X 10^{-1} ug/kg/d = 0.00033 mg/kg/day, 1 in 1,000 increased lifetime cancer risk in the mouse.
4. Assume that the same dose will result in equal risk for a mouse and mink. Then convert the mouse dose to a mink dietary criterion:

   0.00033 mg/kg/d X 1 kg (mink wt) ÷ 0.15 kg/d (mink intake)

   = 0.0022 mg/kg, 1 in 1000 cancer risk in diet.
5. The 1 in 100 cancer risk in diet = 0.022 mg/kg.

Comparison of Criteria with Residue Data

Aldrin was not measured in Niagara River fish by the NRTC (1984). Dieldrin levels in spottail shiners for 1981 varied from below detection to trace, and 1982 young-of-the-year shiners varied from ND to 0.009 mg/kg with a median for both years of 0.002 mg/kg (Tables 3,4 and 26). Norstrom et al. (1978) present dieldrin levels of 0.029 mg/kg in western Lake Ontario alewives and smelt which are about 10 times higher than the

spottail shiner dieldrin residues. Coho salmon muscle averaged 0.087 mg/kg and coho salmon liver averaged 0.06 mg/kg or about 2.5 times higher than their food, the alewives and smelt (Table 6). Dieldrin residues for herring gull eggs sampled from 4 Lake Ontario colonies averaged 0.32 mg/kg (wet weight), i.e., 10 times higher than the alewife and smelt forage and 4 times higher than the piscivorous coho salmon (Norstrom et al. 1978). Recent measurements of dieldrin in several Lake Ontario salmonid species ranged form 0.008-0.14 mg/kg; concentrations in non-salmonid fish in the Niagara River ranged from less than 0.01 (below detection) to 0.08 mg/kg (Table 7).

Niagara River and western Lake Ontario fish were from 1.3 to 13 times lower than the non-carcinogenic based fish flesh criterion of 0.12 mg/kg. Eggs of herring gulls reported by Norstrom et al. (1978) are 2.6 times higher the criterion. Among all species sampled recently in Lake Ontario and the Niagara River only older lake trout from Lake Ontario slightly exceeded the criterion (Table 7). From these calculations it appears that in general, dieldrin levels in fish are not hazardous to wildlife consumers of fish in or near the Niagara River based on non-carcinogenic data, but concentrations in some species of fish approach hazardous levels.

The spottail shiner levels (NRTC) of 0.002 mg/kg are about equal to the 1 in 1,000 increased lifetime cancer risk for piscivorous wildlife. The Norstrom et al. (1978) alewife, smelt and coho salmon residues exceed an estimated 1 in 100 increased lifetime cancer risk for wildlife. Based on recent data of dieldrin residues, three of four salmonids in Lake Ontario and eel from the Niagara River exceed the 1 in 100 cancer risk criterion.

3.2.4 Chlordane

Chlordane is an insecticide used in termite and carpenter ant control and its use has been restricted recently in the U.S. Technical grade chlordane may have as many as 20 components. Khasawinah (1982) estimated chlordane to contain 19% cis-chlordane, 24% trans-chlordane, 7% trans-nonachlor, 10% heptaclor, 21.5% chlordene isomers, and 19.5% micellaneous. Feeding studies reported here have used technical grade chlordane and therefore represent toxicity of a mixture. Chlordane is lipid soluble and highly persistent. The metabolic products of chlordane are more toxic than chlordane. Tashiro and Matsumura (1977) and Brimfield et al. (1978) reported that the metabolites of chlordane include oxychlordane, several chlordane isomers, glucuronides, and heptachlor.

The storage and accumulation of chlordane has been investigated by Balba and Saha (1978). The metabolites of chlordane accumulate in the fatty tissues of animals. Dietary studies of rats, rabbits and dogs have been performed (Table 12) with technical grade chlordane and indicate that chlordane is highly toxic. Liver damage occurred in the dog at 0.075 mg/kg/day.

Chlordane is also carcinogenic to laboratory animals, with chronic exposure resulting in significant increase in the incidence of liver tumors in male and female mice (NCI 1977; Epstein 1976). The New York State Department of Health (DOH 1985c) used dose-response data from the National Cancer Institute to calculate a lower 95% confidence limit for the 1 in a million risk in mice of 5.8×10^{-3} ug/kg/day.

Calculation of Chlordane Wildlife Fish Flesh Criteria

Table 12 summarizes data from dietary exposures and one injection of chlordane in birds and mammals.

1. Determining wildlife application/uncertainty factors for chlordane based on non-target and mammal data. Refer to Table 12 for details of data selected below.

Study	Duration	Effect at LOEL	NOEL mg/kg/day (mg/kg-diet)	Recommended Application (AF)/ Uncertainty (UF) Factor
RAT-2	2 years	Kidney & lung damage	0.25	None
RAT-3	2 weeks	Increased enzyme induction	0.25	None
DOG	2 years	Liver damage	0.075	None

2. Calculation of criteria:

-Rat-2 and -3 NOELs

0.25 mg/kg/d X 1 kg (mink wt) ÷ 0.15 kg/d (mink intake)

= 1.67 mg/kg.

-Dog NOEL

0.075 mg/kg/day X 1 kg (mink wt) ÷ 0.15 kg/d (mink intake)

= 0.5 mg/kg, dietary criterion.

The value of 0.5 mg/kg is selected as the final non-carcinogenic based criterion.

Conversion of the 1 X 10^{-6} cancer risk dose in the experimental animals to a 1 X 10^{-3} cancer risk in diet for a wildlife consumer involves the following steps:

1. 1 X 10^{-6} increased cancer risk in mice = 5.8 X 10^{-3} ug/kg/day
2. 1 X 10^{-3} risk in mice = 5.8 ug/kg/day = 5.8 X 10^{-3} mg/kg/day.

3. 5.8×10^{-3} mg/kg/day = 0.0058 mg/kg/day

4. Assume that the same dose will result in equal risk for mice and mink. Convert the mouse dose to a mink dietary criterion: 0.0058 mg/kg/d X 1 kg (mink wt) ÷ 0.15 kg/day (mink intake) = 0.037 mg/kg, 1 in 1000 cancer risk in diet.

5. The 1 in 100 risk level in diet = 0.37 mg/kg.

Comparison of Criteria with Residue Data

Chlordane residues were found in spottail shiners at 16 of 27 Niagara River Stations with a median of 0.0075 mg/kg, ranging from trace to 0.048 mg/kg (Tables 3,4 and 26). Examination of the Niagara River spottail data would suggest that the chlordane residues (median = 0.0075 mg/kg) represents less than a 1 in 1000 cancer risk and are considerably less than the non-carcinogenic based criterion. However, age 10+ lake trout from Lake Ontario average 0.52 mg/kg chlordane, exceeding both the 1 in 100 cancer risk criterion and the criterion of 0.5 mg/kg based on non-carcinogenic effects. Age 7+ lake trout averaged 0.32 mg/kg chlordane which is at about the 1 in 100 cancer risk level. Among non-salmonid fish from the Niagara River, only eel exceed the 1 in 100 cancer risk and non-carcinogenic based criteria (Table 7).

3.2.5 Dioxin (2,3,7,8-TCDD)

Polychlorinated dibenzo-para-dioxins (PCDDs) are present as trace impurities in some manufacturing chemicals and industrial wastes. PCDDs are environmentally stable and have a tendency to accumulate in fat. Eisler (1986b) has produced a synoptic review of dioxin hazards to fish, wildlife and invertebrates, in which he notes there are 75 PCDD isomers; some are extremely toxic, while others are believed to be relatively innocuous. Eisler (1986)links high levels of PCDDs to hazardous waste

dumps, industrial discharges, or application of PCDD-contaminated herbicides.

Attention was drawn to dioxins by effects noted during and after extensive application of Agent Orange in South Vietnam, a phonoxy herbicide with troublesome levels of contaminants such as the PCDD isomer 2,3,7,8-tetrachlorodibenzo-para-dioxin (2,3,7,8-TCDD). The 2,3,7,8-TCDD isomer is the most toxic (Eisler 1986b). An accident occurred at a trichorophenol production facility in Italy, and a cloud containing 2,3,7,8-TCDD settled over Seveso. Many weeks passed before the public was alerted. Humans exposed to this incident suffered from chloracne, increased spontaneous abortion, and many animals died (Reggiani 1978). Eisler (1986b) states that in the United States and elsewhere, accidental contamination of the environment by 2,3,7,8-TCDD has resulted in deaths of many species of wildlife and domestic animals.

Acute and chronic toxicity studies of 2,3,7,8-TCDD in mammals and birds demonstrate the severity of even low levels of exposure to the contaminant. It causes severe liver damage in rats, mice,and rabbits, chloracne-type skin lesions in man and monkeys and edema formation in birds (Gilbertson 1983). The LD50 for a single oral dose for the guinea pig is 0.2 to 2.5 ug/kg, 22 to 45 ug/kg for the rat, and 1,157 to 5,051 ug/kg for the hamster (Kociba and Schwetz 1982; McConnell et al. 1978). The range of variation of acute toxicity (up to 8,400X) may relate to different rates of metabolism of the parent compound (Eisler 1986b). The parent compound is considerably more toxic than the metabolites (Neal 1985).

The main targets of TCDD appear to be the liver in rats and the thymus in rats, guinea pigs, and mice according to Gupta et al. (1973). Atrophy of the thymus is a consistent finding in mammals poisoned by

2,3,7,8-TCDD, and supression of thymus-dependent cellular immunity, particularly in young animals, may contribute to their death (Eisler 1986b).

Chronic exposure tests with TCDD on rats (Harris et al. 1973; Kociba et al. 1978; Kociba and Schwetz 1982) confirm the severe toxicity of the contaminant. Harris et al. (1973) reported 0.1 ug/kg was the NOEL in a 31 day study, but subsequent 3 generation rat tests by Kociba and Schwetz (1982) found that even this level reduced litter size at birth, increased stillborns, and reduced survival and growth in F_1 and F_2 generations - 0.001 ug/kg/day was selected as the NOEL. Long-term studies in rhesus monkeys (EPA 1985) seem to indicate that even 5 ng/kg diets (0.4 mg/kg/day dose) resulted in effect levels although the toxicity endpoints were bone marrow and axial lymph node deficiencies. Higher treatment levels (50 ng/kg or dose of about 1.7 ng/kg/day) resulted in abortion and weight loss in the rhesus monkey (Barsotti et al. 1979-in Eisler 1986b) with 7 to 29 month exposures; one year exposure at dietary level of 0.5 ug/kg resulted in death of 60% of the experimental animals. The dose of 0.4 ng/kg/day, reported by USEPA, 1985, will be used as the NOEL; Barsotti et al. (1979 - in Eisler 1986b) reported a NOEL of 0.017 ng/kg/day, but it was two orders of magnitude below the LOEL.

The effect of 2,3,7,8-TCDD on birds is also characterized by marked differences in sensitivity. Hudson et al. (1984) tested bobwhite quail, mallards, and ringed turtle doves and report LD50 single oral doses of 15 ug/kg for the bobwhite, 108 ug/kg for the mallard, and 810 ug/kg for

the ringed turtle dove (Table 12). All three species showed similar signs of intoxiciation. Domestic chickens are even more sensitive (Kociba and Schwetz 1982; Gilbertson 1983). Chick edema disease developed in the domestic chickens at 1-10 ug/kg in the diet after 21 days (Gilbertson 1983). These effects are similar to those noted by NRCC (1981 - in Eisler 1986 b) for fish eating bird populations of the Great Lakes in the 1960's and 1970's. Edema signs include pericardial, subcutaneous and peritoneal edema, also liver enlargement and frequent death.

Gilbertson (1983) argues that there are only a small number of chick edema active compounds which include a few of the chlorinated biphenyls, dibenzo-p-dioxins, dibenzofurans, azobenzenes, and azoxybenzenes. The chick-edema active compound, 2,3,7,8-TCDD, is also "the most embryotoxic, teratogenic, hepatoxic, porphyrinogenic" of the chemicals affecting chick embryos (IJC 1986). Herring gull chicks showed signs of edema in the 1970's and it has declined since then; concentrations in herring gull eggs have declined from about 1,000 ug/kg to less than 80 ug/kg in 1981 (Gilberston 1983). The improvement in reproductive success and the decrease in congenital anomalies seen on the Niagara River are most likely the result of decreased production of TCP on the river. The principal manufacturer of TCP stopped production on the Niagara River in the early 1970's; the resultant decrease in TCP production correlates with the observed decrease in 2,3,7,8-TCDD (a by-product of TCP production) in herring gull eggs. According to the IJC (1986) review, 2,3,7,8-TCDD was probably the principal agent responsible for reproductive and pathological effects observed in herring gulls.

Eisler (1986b) concluded that 2,3,7,8-TCDD had a greater effect on growth, survival, and reproduction than on tumor formation, because it exerts non-carcinogenic toxicity at such very low levels at or below actual environmental exposure levels. The NYS Dept. of Health calculated a lower 95% confidence limit value of the 2,3,7,8-TCDD dose corresponding to an increased lifetime cancer risk of 1×10^{-6} for humans of 6.4×10^{-12} mg/kg/day (K. Bogden pers. comm.). Dividing the human dose by the weight to surface factor of

$$\left(\frac{0.45 \text{ kg (rat wt)}}{70 \text{ kg (human wt)}}\right)^{0.33}$$

results in a rat dose of 1.2×10^{-12} mg/kg/d.

Calculation of 2,3,7,8-TCDD Wildlife Fish Flesh Criteria

Table 13 summarizes data from dietary exposures of 2,3,7,8-TCDD in birds and mammals. Eisler (1986b) selected 10 to 12 ng/kg (ppt) in food items of birds and other wildlife as the NOEL. The only target species for which we have extensive etiological data (Gilbertson 1983, 1985) is the herring gull in the Great Lakes area. Chick edema disease in the gulls has declined since 2,3,7,8-TCDD residues in the herring gull eggs have declined to 80 ng/kg or less. As residue levels in birds and mammals freguently reach levels about 10 times higher than the daily intake (Fries and Marrow 1975) the wildlife NOEL is estimated at 8 ng/kg or less. Using several long term toxicity tests from Table 13 on TCDD criteria can be calculated:

1. Determining wildlife application/uncertainty factors for TCDD dietary exposure based on target and non-target animal toxicity data. Refer to Table 13 for details of data selected below.

Study	Duration	Effects at LOEL	NOEL/LOEL ug/kg/day (ug/kg-diet)	Recommended Application (AF)/ Undertainty (UF) Factor
RAT-6	13 weeks	Decreased litter size, reduced survival & growth of young	0.01 (sub-acute)	0.1 (sub-acute to chronic AF)
RAT-7	multi generation	Heptatic toxicity and. histopathology	0.001 (NOEL)	none
Guinea Pig-4	8 weekly doses	Thymus effects	0.1 (sub-acute)	0.1 (sug-acute to chronic AF)
Monkey-2	8 months	Bone marrow & axial lymph node deficiencies	0.0004 (NOEL)	None

2. Calculation of criteria:

-Rat-6 data

0.01 ug/kg/d (sub-acute) X 0.1 (AF) X 1 kg (mink wt.) ÷ 0.15 kg/d (mink intake) = 0.007 ug/kg.

-Rat-7 data

0.001 ug/kg/d (NOEL) X 1 kg (mink wt) ÷ 0.15 kg/d (mink intake) = 0.007 ug/kg

-Guinea pig-4 data

0.1 ug/kg/d (sub-acute) X 0.1 (AF) X 1 kg (mink wt.) ÷

0.15 kg/d (mink intake) = 0.07.

-Rhesus Monkey-2 data

0.0004 ug/kg/d (NOEL) X 1 kg (mink wt) - 0.15 kg/d (mink intake) = 0.003 ug/kg.

The value of 0.003 ug/kg is selected as the final non-carcinogenic based criterion.

Conversion of the dose that corresponds to a 1×10^{-6} risk of cancer to experimental animals to a level in the diet of wildlife that would correspond to 1×10^{-3} and 1×10^{-2} risks of cancer involves the following steps:

1. 1×10^{-6} increased cancer risk dose in rats = 3.4×10^{-11} mg/kg/day.
2. 1×10^{-3} increased cancer risk dose in rats = 3.4×10^{-8} mg/kg/day.
3. Assume that the same dose will result in equal risk for rats and mink. Then convert the rat dose to a mink dietary criterion:

   3.4×10^{-8} mg/kg/d X 1 kg - 0.15 kg/day = 2.3×10^{-7} mg/kg = 0.23 ng/kg. 1 in 1,000 cancer risk in diet.
4. The 1 in 100 cancer risk in diet = 2.3 ng/kg.

Comparison of Criteria with Residue Data

Spottail shiners are somewhat lower in TCDD on the average than the fish reported by Stolzenburg and Sullivan (1983). Larger (older) spottail shiners and especially larger, older fish of more predatory species would be expected to have higher TCDD levels due to longer exposure, more consumption, and higher lipid content. The spottail shiners sampled were approximately 1% lipid content compared to 6%-10%

for other area fish species. Ryan et al. (1984) found that 2,3,7,8-TCDD accumulates with age in Lake Ontario fish and that TCDD levels are associated with elevated PCB levels. Eisler (1986b) points out that this demonstrates the need for checking interaction kinetics with other contaminants.

Bottom feeding fish contained higher levels of TCDD than surface feeding fish in Michigan Rivers (Harless et al. 1982), probably the result of contact with contaminated sediments. Some fish residue levels reviewed have exceeded either Eisler's (1986b) 10 to 12 ng/kg diet or the estimated wildlife fish flesh criterion to prevent non-carcinogenic toxicity of 3 ng/kg (0.003 ug/kg) diet. The median dioxin concentration in spottail shiners in the Niagara River in 1981 and 1982 was "not detectable," but with a maximum of 120 ng/kg; residues in other fish species in the river ranged from 162-870 ng/kg (Tables 3-6, 26). Stolzenburg and Sullivan (1983) reported that fish from the Niagara River and parts of Lake Ontario ranged from 0.087 ug/kg to 0.162 ug/kg 2,3,7,8-TCDD. It is concluded that 2,3,7,8-TCDD levels represent a significant non-carcinogenic toxicity risk to sensitive, piscivorous wildlife in the Niagara River and western Lake Ontario.

The spottail shiner 2,3,7,8-TCDD median residue level of "not detectable" is not interpretable, but the maximum of 120 ng/kg and the residues of other species exceed the 1 in 100 cancer risk dietary criterion of 2.3 ng/kg by about 50 to several hundred times; this corresponds to a cancer risk between 1 in 10 and 1. It would appear that Niagara River wildlife (or at least the mammals) using Niagara River fish for most of their sustenance throughout their lifetime have a high chance of developing cancer from exposure to 2,3,7,8-TCDD. To

validate use of this risk assessment approach, it would be valuable to determine the extent of cancer in Niagara River wildife compared to wildlife from unpolluted areas.

3.2.6 ENDRIN

Endrin is the most toxic of the cyclodiene pesticides among the widely used organochlorine pesticides; the cyclodiene group is the most toxic to mammals (Allen et al. 1980). A variety of human health impacts from exposure to endrin during manufacture and use have been reported. The lowest dose reported to have caused death in humans is 5 mg/kg (NIOSH 1978).

Quail fed 1 mg/kg of endrin in their diet produced no eggs during a chronic treatment (NRC 1980)

Endrin was fed to rats at 2, 6, and 12 mg/kg in the diet for 2 years without producing increased tumor incidence in any organ (NRC 1980). Groups of 50 rats of each sex were administered one or two doses of endrin for 80 weeks, tumor incidence was not significantly different from controls (NCI 1979b).

A variety of species have been tested for endrin acute toxicity. LD50's ranged from about 1.78 mg/kg to 5.64 mg/kg (Table 14). Hudson et al. (1984) exposed mallard ducks to both a single dose (LD50 5.64 mg/kg) and to a 30 day exposure (0.25 mg/kg diet each day) which caused 50% mortality. Treon et al. (1955) fed rats diets containing 1, 5, 25, 50, or 100 mg/kg endrin. Endrin diets of 25 mg/kg and higher caused significant mortality. At the dietary level of 100 mg/kg, only 5% of the males survived beyond 2 weeks (6.5 mg/kg/day dose level). The livers of male rats fed 5 mg/kg were significantly greater in relation to body weight than those of controls. The 1 mg/kg dietary level was the NOEL level for the rat in the Treon et al. (1955) study, representing a dose level of 0.065 mg/kg/day.

Dogs fed on diets containing toxic concentrations of endrin regurgitated their food, became lethargic, salivated, and later refused to eat. Dogs fed at 4, 3, or 1 mg/kg dietary level exhibited no signs of intoxication (Treon et al. 1955). Dogs fed at 3 and 1 mg/kg dietary endrin levels also showed no organ damage, establishing 3 mg/kg as the NOEL level (.075 mg/kg/day).

Screech owls fed 0.75 ppm endrin produced 43% fewer fledged owlets than controls (Fleming et al. 1982). Hatching success appeared to be the main variable affected by endrin. Estimates of harmful levels of endrin in screech owl eggs is 0.3 mg/kg or more (Fleming et al. 1982). Blus et al. (1979) estimated that 0.5 mg/kg in eggs of brown pelican was the critical level, and if exceeded, caused reproductive impairment.

Two bald eagles lived 13 and 20 days on diets containing 20 mg/kg endrin (dry weight), therefore it is clear that bald eagles are at least as tolerant as other species (Stickel et al. 1979). The eagles were not repelled by the endrin blended into the meat diet, which is not true for many species. Brains of the two eagles contained 1.2 and 0.92 mg/kg endrin (wet weight), well within the ranges Stickel et al. (1979) found for blackbirds and ducks. A number of wild eagles found dead have contained brain residues of endrin in this probably lethal range.

A number of dietary studies have established the relative toxicity of endrin to birds and mammals, but without providing calculation of LOELs or NOELs. The dietary study with the 2 bald eagles (Stickel et al. 1979) is a good example, yet this study estimated brain residues of endrin which are lethal to eagles and probably other birds.

Calculation of Endrin Wildlife NOEL

Dietary effect levels of endrin in animals is presented in Table 14..

1. Determining wildlife application/uncertainty factors for endrin based on target and non-target animal dietary toxicity tests. Refer to Table 14 for details of data selected below.

Study	Duration	Effect at LOEL	NOEL/LOEL mg/kg/day (mg/kg-diet)	Recommended Application (AF)/ Uncertainty (UF) Factor
Rat-3	2 year	Enlarged kidney heart liver	0.065 (NOEL)	None
Dog	2 year	Enlarged liver	0.075 (NOEL)	None
Screech owl	8 weeks	43% fewer owlets	(0.75) (LOEL)	0.2 (LOEL to NOEL AF)
Mallard-2	30 days	50% mortality	(0.25) (subacute)	0.1 (sub-acute to chronic AF)

2. Calculation of criteria:

-Rat-3 data

0.065 mg/kg/d X 1 kg (mink wt) ÷ 0.15 kg/day (mink intake)

= 0.433 mg/kg.

-Dog data

0.075 mg/kg/d X 1 kg (mink wt) ÷ 0.15 kg/d (mink intake)

= 0.5 mg/kg.

-Screech owl data (using the dietary LOEL directly)

0.75 mg/kg X 0.2 (AF) = 0.15 mg/kg.

-Mallard-2 data

0.25 mg/kg X 0.1 (AF) = 0.025 mg/kg.

The value of 0.025 mg/kg is selected as the final non-carcinogenic based criterion.

A cancer risk assessment for endrin was not available.

Comparison of Criteria with Residue Data

Niagara River spottail shiner median endrin concentrations were "not detectable" in 1981 and 1982 (detection limit = 1 ug/kg) with a maximum of 0.007 mg/kg (Tables 3,4, and 26). Endrin was also less than detection (i.e. 0.01 mg/kg) in six other fishes in the Niagara River. It appears that endrin is not a problem for Niagara River piscivorous wildlife.

3.2.7 Heptachlor and Heptachlor Epoxide

Heptachlor is a "white crystalline solid" used for a number of years in commercial preparations as a "broad spectrum insecticide" (EPA 1980b). Technical grade heptachlor is approximately 73% heptachlor, 21% trans (gamma) chlordane, 5% heptachlor epoxide or various metabolic products of heptachlor, and 1% chlordane isomers (EPA 1980b).

Heptachlor is quite stable, but does degrade via microbial, biochemical, and photochemical reactions (Feroz and Kahn 1979). Heptachlor epoxide is the primary metabolite, photoheptachlor III the predominant photo isomer (EPA 1980b). Interestingly, the photoheptachlor III metabolite is 20 times more toxic to rats and 264

times more toxic to goldfish than heptachlor itself (Podowski et al. 1979). Information is currently unavailable to determine the actual likelihood of photoheptachlor III production in surface waters, and its subsequent environmental fate and effects. Thus, only heptachlor and its epoxide will be discussed in detail.

Experimental evidence from goldfish injected with heptachlor at a dose of 38 ug/44 g fish, showed 18% elimination of the dose within 10 days (Feroz and Kahn 1979). At the end of 10 days, 91% of the retained dose occurred as heptachlor and most of the remainder was metabolized to heptachlor epoxide.

The U.S. Fish and Wildlife Service (1978) found heptachlor poisoning of Pacific Northwest wildlife including pheasant, quail, Canada geese, magpies, and even a golden eagle. Die-offs which occurred were due to heptachlor coated seed grains.

Dietary tests of heptachlor and heptachlor epoxide are fairly extensive. Single dose acute toxicity for the rat is 40 mg/kg heptachlor and 62 mg/kg heptachlor epoxide (NIOSH 1982). The LD50 for mallard heptachlor toxicity is much higher, exceeding 2000 mg/kg (NIOSH 1982; Hudson et al. 1984). Dietary effect levels of heptachlor and heptachlor epoxide are presented in Table 15. Harbison (1975) found that neonatal Sprague-Dawley rats were more resistant than adult rats (single dose LD50=150 mg/kg vs. 120 mg/kg) on a statistically significant basis. Miranda and Webb (1974) found that lower protein levels in the diet reduced heptachlor toxicity, presumably because it slowed metabolism of heptachlor to more toxic forms, such as heptachlor epoxide and photoepoxide.

Wagstaff et al. (1980) estimated the NOEL in laboratory chickens exposed to heptachlor and DDT at 0.3 mg/kg heptachlor during the first

eight weeks of life, and noted that DDT storage was reduced when heptachlor was present.

The results of several bioassays with rats and mice indicate that heptachlor is likely to increase the incidence of tumors (Davis 1965; Reuber 1977). Epstein (1976) reviewed several of these bioassays and concluded that the contaminant should be regarded as carcinogenic. The NYS Department of Health calculated a lower 95% limit value of the heptachlor and heptachlor epoxide dose corresponding to an increased lifetime cancer risk of 1×10^{-6} to mice of 3.1×10^{-3} ug/kg/d (DOH 1985e). DOH 1985e noted that in mammals heptachlor is rapidly converted and metabolized to heptachlor epoxide..

Metcalf and Sandborn (1975) reported 70% of the fish they measured in the U.S. contained heptachlor residues. The lake trout from Lake Superior were found to contain heptachlor and heptachlor epoxide (Parejko and Wu 1977).

Calculation of Heptachlor and Heptachlor Epoxide Fish Flesh Criteria

Table 15 summarizes data from dietary exposures of birds and mammals to heptachlor and heptachlor epoxide.

1. Determining wildlife application uncertainty factors for heptachlor and heptachlor epoxide dietary exposure based on non-target animal toxicity data. Refer to Table 15 for details of data selected below.

Study	Duration	Effect (at LOEL)	Recommended NOEL/LOEL mg/kg/day (mg/kg-diet)	Application (AF)/ Uncertainty (UF) Factor
RAT-6	8 months	Induced enzymes	0.075 (NOEL)	None
RAT-7	8 months	Induced enzymes	0.075 (NOEL)	None
Chicken	8 weeks	LOEL not established	0.05 (NOEL)	0.1 (inter-species UF)
Calf	100 days	Kidney disorders	(0.2) (NOEL)	None

2. Calculation of criteria:

-Rat data

0.075 mg/kg/d X 1 kg (mink wt) ÷ 0.15 kg/d (mink intake)

= 0.5 mg/kg.

-Chicken data

0.05 mg/kg/d X 0.1 (UF) X 1 kg (bird wt) ÷ 0.2 (bird intake)

= 0.025

mg/kg.

-Calf data - factors to convert the calf dietary NOEL of 0.2 mg/kg to a dose are unavailable so this dose will be used directly as a candidate wildlife fish flesh criterion.

The chicken based criterion is the lowest, but without a LOEL it cannot be determined whether the NOEL approaches a threshold for chronic effects. Therefore, the calf based value of 0.2 mg/kg is selected as the final non-carcinogenic based criterion.

Conversion of the 1 X 10^{-6} cancer risk dose in the mouse to a fish

flesh criterion with 1 X 10^{-3} and 1 X 10^{-2} cancer risk for a wildlife consumer involves the following steps:

1. 1 X 10^{-6} increased cancer risk dose in mice = 3.1 X 10^{-3} ug/kg/d = 3.1 X 10^{-6} mg/kg/d.
2. 1 X 10^{-3} increased cancer risk dose = 3.1 X 10^{-3} mg/kg/d.
3. Assume that the same dose will result in equal risk for mice and mink. Then convert the mouse dose to a mink dietary criterion:
   3.1 X 10^{-3} mg/kg/d X 1 kg - 0.15 kg/d = 2.1 X 10^{-2} mg/kg, 1 in 1000 cancer risk in diet.
4. The 1 in 100 cancer risk diet = 0.21 mg/kg.

Comparison of Criteria with Residue Data

Residues of heptachlor/heptachlor epoxide in spottail shiners and six other non-salminid fish species in the Niagara River exceed neither the non-carcinogenic criterion of 0.2 mg/kg nor the 1 X 10^{-3} cancer risk criterion of 2.1 X 10^{-2} mg/kg (Tables 3-7).

Metcalf and Sandborn (1975) reported 70% of the fish they measured in the U.S. contained heptachlor resiudes. The lake trout from Lake Superior were found to contain heptachlor and heptachlor epoxide (Parejko and Wu 1977).

3.2.8 Mirex

Mirex, a polycyclic organochlorine, has been used to treat vast areas of the southeastern United States to control the imported fire ant (Hill and Dent 1985). Most of the mirex was applied aerially using 1.4 kg/ha of 0.3% technical mirex in corncob grits (Hill and Dent 1985). The compound was also used as a flame retardant in electronic

components, plastics, and fabrics (Eisler 1985). In 1978, the U.S. Environmental Protection Agency banned all further use of mirex, partly because of the hazards it imposed on non-target biota (Eisler 1985). Eisler (1985) has produced a synoptic review of mirex hazards to fish, wildlife and invertebrates that reviews mirex impact on non-target species.

Mirex is composed of 22% carbon and 78% chlorine and is highly resistant to chemical, thermal, and biochemical degradation (Eisler 1985). Mirex has a long half-life and may be present in Great Lakes sediment for 200-600 years (Scrudato and DelPrete 1982). Mirex residues have been found in a variety of wild fauna (Hill and Dent 1985). Mirex residues have also been found in domestic animals used for human food. Coho salmon from Lake Ontario (Norstrom et al. 1978) averaged 230 ug/kg in a 1976 sample. Fat from slaughtered beef from treated areas in Georgia and Mississippi average 25 ug/kg (Ford et al. 1973). Mirex residues were found to be absent in Tennessee and Iowa beef fat (Ford et al. 1973) showing that mirex had not become a contaminant in regions with little use.

Toxicity of Mirex

Acute toxicity to aquatic organisms, mammals, and birds is quite low (Eisler 1985). This resistance of animals to mirex in short term toxicity tests and effectiveness as a toxicant for the imported fire ant was undoubtedly the factor that led to such widespread use.

Schafer et al. (1983) summarized the acute oral toxicity, repellency, and hazard potential of 998 chemicals to birds. Mirex acute toxicities to birds was low, 100 mg/kg did not affect the red-winged blackbird, although it proved to be the most sensitive bird in Schafer

et al.'s (1983) review. Acute oral toxicities to mammals are similarly low, with 400 mg/kg being the lowest fatal dose in rats (NAS 1978). Gaines and Kimbrough (1969) found that the acute toxicity of mirex in rats was low, with 50% of the test animals dying 14 days after a single exposure to 365 mg/kg. However, when Gaines and Kimbrough (1969) exposed rats over a 24 month period, dietary levels of 25 mg/kg caused enlargement of liver cells which led them to select a NOEL of 5 mg/kg for rats. Enlargement in liver cells in parent rats (Gaines and Kimbrough 1969) was followed by fewer and less viable offspring.

Chu et al (1980) fed rats containing organohalogens alone or in various combinations for 28 days. They concluded that mirex-related compounds at dose levels studied (usually 1 to 20 mg/kg PCB) did not potentiate the effects produced by halogenated biphenyls and vice versa. Chu et al. (1981) found reduced litter size and histopathological effects in rats fed 5 mg/kg mirex for one year.

A significant effect of prenatal exposure to mirex is fetal edema (Grabowski 1981). Dosage of pregnant female rats with 6 mg/kg on each of 8 successive days induced slight weight loss of dams, but no mortality. However, fetuses had high incidences of edema and cardiovascular disorders.

Long-term feeding studies with mirex demonstrate the impact of mirex on non-target biota (Table 16). Hyde (1972) exposed old field mice to 1.8 mg/kg dietary mirex for 60 weeks and reported 20% mortality. Prairie voles were also sensitive in a 90 day test with some mortality at 5 mg/kg diet and 100% dead at the 25 mg/kg dietary mirex level.

Eisler (1985) did not select a safe dietary level of mirex to protect wildlife consumers, but did suggest that this level should be less than 0.1 mg/kg.

Eighteen month exposures of laboratory mice as low as 26 mg/kg mirex in the diet caused 40% hepatomas (Innes et al. 1969). A carcinogenicity assay of mirex in rats (Ulland et al. 1977) was positive and it is the basis of New York State's ambient surface water quality guidance value for sources of drinking water (DOH 1985f). Dose response data from Ulland et al. (1977) were used for extrapolation. The lower 95% confidence limit value of the mirex dose corresponding to an increased lifetime cancer risk of 1×10^{-6} for the experimental animals was 5.6×10^{-3} ug/kg/day.

To some extent mirex has been found to degrade to photomirex and some other chemicals, all of which appear to be stable and about as biologically active as mirex (Eisler 1985 and IJC 1981). A criterion for mirex should probably be expressed as "mirex and its degradation products."

Calculation of Mirex Wildlife Fish Flesh Criteria

Table 16 summarizes data from dietary exposures of mirex in birds and mammals.

1. Determining wildlife application/uncertainty factors for mirex dietary exposure based on target and non-target animal toxicity data. Refer to Table 16 for details of data selected below.

Study	Duration	Effect at LOEL	NOEL/LOEL mg/kg/day (mg/kg-diet)	Recommended Application (AF)/ Uncertainty (UF) Factor
Rat-7	1 year	Enlarged liver, decreased litter size	0.25 (LOEL)	0.2 (LOEL to NOEL AF)
Prairie vole	13 weeks	100% Dead	0.8 (NOEL)	0.1 (sub-acute to chronic AF)
Old field mouse	60 weeks	20% Mortality	0.28 (LOEL)	0.2 (LOEL to NOEL AF)
Mallard	25 weeks	Adult mortality, reduced survival of ducklings	(100) (LOEL)	0.1 (interspecies UF) 0.2 (LOEL to NOEL AF)

2. Calculation of criteria

-Rat-7 data

0.25 mg/kg/d X 0.2 (AF) X 1 kg (mink wt) ÷ 0.15 kg/d (mink intake) = 0.33 mg/kg.

-Prairie vole data

0.8 mg/kg/d X 0.1 (AF) X 1 kg (mink wt) ÷ 0.15 kg/d (mink intake) =0.53 mg/kg.

-Old field mouse data

0.28 mg/kg/d X 0.2 (AF) X 1 kg (mink wt) ÷ 0.15 kg/d (mink intake) = 0.37 mg/kg.

-Mallard

100 mg/kg X 0.2 (AF) X 0.1 (UF) = 2 mg/kg.

The value of 0.33 mg/kg is selected as the non-carcinogenic based

criterion. This is very similar to the value derived from old field mouse data.

Conversion of the dose that corresponds to a 1×10^{-6} risk of cancer to experimental animals to a level in the diet of wildlife that would correspond to 1×10^{-3} and 1×10^{-2} risks of cancer involves the following steps:

1. 1×10^{-6} increased cancer risk dose in rats = 5.6×10^{-3} ug/kg/day = 5.6×10^{-6} mg/kg/day.
2. 1×10^{-3} increased cancer risk dose in rats = 5.6×10^{-3} mg/kg/day.
3. Assume that the same dose will result in equal risk for rats and mink. Then convert the rat dose to a mink dietary criterion:
   5.6×10^{-3} mg/kg/day X 1 kg $\stackrel{\circ}{\sim}$ 0.15 kg/d = 37.3×10^{-3} mg/kg = 0.0373 mg/kg, 1×10^{-3} risk.
4. The 1 in 100 cancer risk in diet = 0.373 mg/kg.

Comparison of Criteria With Residue Data

The median mirex concentration in spottail shiners from the Niagara River in 1981 and 1982 was "not detectable", with a maximum of 0.018 mg/kg (Tables 3,4 and 26). White bass in the Niagara River had a mirex concentration of 0.51 mg/kg (Table 5). Alewives, smelt and coho salmon had higher mirex residues in 1976 as would be expected of higher lipid content fish (Norstrom et al. 1978). Alewives and smelt averaged 0.09 mg/kg and also had photomirex residues averaging 0.03 mg/kg (Table 6). Coho salmon mirex residues averaged 0.23 mg/kg and 0.11 mg/kg for photomirex. Recent measurements of mirex and photomirex in several salmonids from Lake Ontario ranged ranged from 0.115-0.633 mg/kg;

concentrations of mirex (photomirex was not measured in non-salminid fish in the Niagara River ranged from less than detection to 0.17 mg/kg (Table 7).

Residues of mirex in spottail shiners are less than both the non-carcinogenic and 1 in 1000 cancer risk criteria. Residues in alewives, smelth and coho salmon liver in western Lake Ontario are less than the non-carcinogenic based criterion, but exceed the 1 in 1000 cancer risk criterion. Residues of mirex in white bass from the Niagara River and the combined residues of mirex and photomirex in coho salmon muscle from western Lake Ontario exceed both the non-carcinogenic and 1 in 100 cancer risk criteria (Tables 3-6). Recent residue data demonstrates that among fish sampled in Lake Ontario only lake trout exceed the non-carcinogenic and 1 in 100 cancer risk based criteria and none of the fish collected in the Niagara River exceed the criteria (Table 7).

3.2.9 **Hexachlorobenzene (HCB)**

Hexachlorobenzene is a crystalline substance which is insoluble in water. It is most frequently used in dust form as a fungicide to control fungal diseases (Vos et al. 1971). The occurance and effects of HCB have been reported in many organisms, e.g. birds (Vos et al. 1971; Gilbertson and Reynolds 1972; Crotmartie et al. 1975), rats (Kimbrough and Linder 1974), man (Cam and Nigogosyn 1963; Currier et al. 1980), and fish (Johnson et al. 1974, Niimi and Cho 1981). HCB residues have been found in human food (Booth and McDowell 1975) and in the food of laboratory animals (Yang et al. 1976). HCB is highly persistant (Metcalf et al. 1973).

Long-term ingestion of HCB-treated grain poisoned several thousand people in Turkey. Human victims had enlarged livers and porphyria, loss

of appetite, weight loss and wasting of skeletal muscles (Clayton and Clayton 1981).

The acute toxicity of HCB is low. The LD50 single oral dose for the rat is 3,500-10,000 mg/kg (Booth and McDowell 1975; NIOSH 1982). The single oral lethal dose (50%) for Coturnix quail is greater than 1,000 mg/kg (Vos et al. 1971). However, the sub-acute and chronic toxicity of HCB is much lower. Vos et al. (1971) established 1 mg/kg as the LOEL (due to histopathological effects) for Coturnix in 3 month feeding studies, although no mortality occurred.

Kimbrough and Linder (1974) established a LOEL of 7.5 mg/kg for HCB in a four month feeding study with rats, based on increased liver size as a toxicity endpoint, concluding that part of the damage resulted from HCB impurities. The technical grade used in agriculture is reported to contain 98% HCB, 1.8% pentachlorobenzene and 0.2% 1,2,4,5 tetrachlorobenzene (Berg 1983). Villeneuve et al. (1974) found evidence of chlorodibenzo-p-dioxin and chlorodibenzofuran in commercial HCB preparations. Upon microscopic examination, the organs primarily affected by HCB were liver, heart, lungs, and adrenals (Vos et al. 1971; Kimbrough and Linder 1974).

The pig proved to be the most sensitive animal tested (Fassbender et al. 1977) with a NOEL of 0.05 mg/kg/day and porphyria and liver damage at higher treatment levels (0.5 mg/kg/day). Aside from acute toxicity data on mallards no data were found on HCB dietary effects in target species.

Calculation of HCB Wildlife Fish Flesh Criteria

Table 17 summarizes data from dietary exposures of HCB in birds and mammals.

1. Determining wildlife application/uncertainty factors for HCB dietary exposure based on non-target bird and mammal data. Refer to Table 17 for details of data selected below.

Study	Duration	Effect at LOEL	NOEL/LOEL mg/kg/day (mg/kg-diet)	Recommended Application (AF)/ Uncertainty (UF) Factors
Rat-3	4 months	Increase in liver weight	7.5 (LOEL)	0.1 (LOEL to NOEL AF)
Pig	3 months	Porphyria, increased liver weight, mortality	0.05 (NOEL)	None
Dog	21 days	Liver enlargement	1.25 (sub-acute)	0.1 (sub-acute to chronic AF)
Coturnix	3 months	Increased liver & weight damage	0.2 (NOEL)	None
Cat	4 1/2 months	Susceptibility to respiratory infection	4.5 (LOEL)	0.2 (LOEL to NOEL AF)

2. Calculation of criteria:

-Pig data

0.05 mg/kg/day X 1 kg (mink wt) ÷ 0.15 kg/day (mink intake) = 0.33 mg/kg

-Rat-3 data

7.5 mg/kg/d X 0.1 (AF) X 1 kg (mink wt) ÷ 0.15 kd/d (mink intake) = 5 mg/kg.

-Dog data

1.25 mg/kg/d X 0.1 (AF) X 1 kg (mink wt) ÷ 0.15 kg/d (mink intake) = 0.83 mg/kg.

-Coturnix quail data

0.2 mg/kg/d X 1 kg (bird wt) ÷ 0.2 kg/d (bird intake) = 1 mg/kg

-Cat data:

4.5 mg/kg/d X 0.2 (AF) X 1 kg (mink wt) ÷ 0.15 kg/d (mink intake) = 6 mg/kg.

It is concluded that mammal data suggests lower wildlife NOEL, therefore 0.33 mg/kg diet is the estimated wildlife flesh criterion for non-carcinogenic data.

HCB is a carcinogen (Courtney 1979; Lambrect et al. 1983). The bioassay of Lambrect et al. (1983) is the basis of New York State ambient surface water quality guidance value for sources of drinking water (DOH 1985g). Dose-response data from Lambrect et al. (1983) were used for extrapolation. The lower 95% confidence limit value for the HCB dose corresponding to an increased lifetime cancer risk of 1×10^{-6} for the experimental animals (rat) was 3.0×10^{-3} ug/kg/day.

Conversion of the 1 X 10 $^{-6}$ risk in the experimental animals to a fish flesh criterion with 1 X 10^{-3} cancer risk for wildlife consumer involves the following steps:

1. 1 X 10^{-6} increased cancer risk in rats = 3.0 X 10^{-10} ug/kg/day.
2. 1 X 10^{-3} risk in rats = 3.0 ug/kg/day.
3. 3.0 ug/kg/day = 0.003 mg/kg/day, 1 X 10^{-3} increased lifetime cancer risk in the rat.
4. Converting rat dose to mink dietary level:
   0.003 mg/kg/day X 1 kg (mink) $\frac{e}{e}$ 0.15 kg/day = 0.02 mg/kg, 1 in 1000 cancer risk in diet. The 1 in 100 cancer risk in diet = 0.2 mg/kg.

Comparison of Criteria with Residue Data

HCB residues have been found in the biota of Niagara River. This contamination is due in large part, to the high volume production of chlorobenzenes by industry. Hooker Electrochemical Company began operation of their chlorobenzenes plant in the United States with a capacity of 8,200 metric tons/year at Niagara Falls, New York in 1915 (Oliver and Nicol 1982). Compared to other chlorobenzenes, HCB predominates in many fish residues in Lake Ontario. Oliver and Nicol (1982) speculate that the higher HCB residues compared to other CBs is due to HCB's high octanol/water coefficient, and to the lower CB's higher metabolism by fish.

Young-of-the-year spottail shiners (Tables 3 and 4) from Lake Erie and the Niagara River in 1981 and 1982 combined HCB ranging from ND to 261 ug HCB/ kg fish, with a median of 2.5 ug/kg. Niimi and Cho (1981) reported that concentrations of HCB in Lake Ontario fish generally range from 1 to 100 ug/kg. The FDA measured 350 ug/kg in Niagara River white

bass, and 240 ug/kg in smallmouth bass for fish samples taken by N.Y.S. in 1976 (Table 5). Nimi (1979) reported 70 ug/kg HCB in Lake Ontario salmonids. Recent measurements of HCB in several salmonids from Lake Ontario ranged from 0.005-0.1 mg/kg; concentrations in non-salmonids in the Niagara River were all less than detection (Table 7).
Spottail shiner levels (NRTC 1984) of up to 0.008 mg/kg are less than the 1 in 1000 cancer risk criterion of 0.02 mg/kg and less than the estimated wildlife non-carcinogenic based criterion of 0.33 mg/kg. The FDA measurement of 0.03-0.95 mg/kg in several species represents a greater than 1 in 100 increased life time cancer risk to wildlife consumers and also exceeds the non-carcinogenic based criterion for some of the species. However, recent residue data collected by NYSDEC (in prep.) demonstrates that among salmonid sampled in Lake Ontario and non-salmonids, sampled in the Niagara River, none currently exceed the non-carcinogenic or 1 in 100 cancer risk based criteria; rainbow trout and spring brown trout from Lake Ontario exceed the 1 in 1000 cancer risk criterion (Table 7).

3.2.10 Hexachlorocyclohexane (α, β, and γ and Δ isomers)

The persistent organochlorine insecticide, hexachlorocyclohexane (HCH), popularly known as lindane or benzenehexachloride (BHC), has eight stereo isomers of which four (alpha, beta, gamma, and delta) predominate in the technical product because of relatively strainless bonds (Deo et al. 1982). Of the isomers, only gamma HCH is highly insecticidal. The half life of the four predominant isomers varied from 4 to 22 days when exposed to sunlight, although it can be as long as 50 days in submerged soils (Deo et al. 1982). HCH isomers degrade to chlorophenols at different rates in order of their solubilities in fat (delta > gamma > alpha > beta).

The acute toxicity of these isomers are listed in Table 18. Beta HCH is the least toxic isomer to the rat with an LD50 of 6,000 mg/kg. Gamma HCH is the most acutely toxic isomer to the rat with an LD50 of 76 mg/kg. Short term feeding studies of alpha, beta, and gamma HCH isomers conducted by Muller et al. (1981) led these authors to conclude that beta and gamma HCH may exert neurotoxic effects. When chickens were fed at levels of 0.1 to 10 mg/kg gamma HCH, Sauter and Steele (1972) found significantly reduced hatchability. In another study Whitehead et al. (1972) did not find reduced hatchability at 100 mg/kg dietary level, although they did note decreased egg production. The NOEL reported by Whitehead et al. (1972) was 64 mg/kg dietary level as compared to the 10 mg/kg dietary level reported by Sauter and Steele (1972).

The NYS Department of Health calculated a lower 95% limit value of the gamma HCH dose corresponding to an increased lifetime cancer risk of 1×10^{-6} to mice of 0.0076 ug/kg/day (DOH, 1985h-using data from Thorpe and Walker 1973). DOH applied the value as the 1×10^{-6} risk dose for the sum of all HCH isomers.

Calculation of Combined HCH Wildlife Fish Flesh Criteria

Table 18 summarizes data from dietary exposures of HCH in birds and mammals.

1. Determining wildlife application/uncertainty factors for HCH dietary exposure based on laboratory animal non-carcinogenic toxicity data. Refer to Table 18 for details of data selected below.

Study	Duration	Effect at LOEL	NOEL/LOEL mg/kg/day (mg/kg-diet)	Recommended Application (AF) / Uncertainty (UF) Factor
Rat-5	30 days	Neurotoxic	9.37 (sub-acute LOEL)	0.1 (sub-acute to NOEL AF)
Dog	4 months	Neurotoxicity	0.3 (NOEL)	None
Chicken-2	3 months	Reduced hatchability decreased	0.02 (NOEL)	None
Chicken-1	27 days	Decreased egg production	12.8 (NOEL)	0.1 (sub-acute to NOEL AF)
Coturnix-2	30 days	Reduced hatchability	5.0 (sub-acute LOEL)	0.1 (sub-acute to NOEL AF)

2. Calculation of criteria:

-Rat-5 data

9.37 mg/kg/d X 0.1 (AF) X 1 kg (mink wt) ÷ 0.15 kg/d (mink intake) = 6.25 mg/kg.

-Dog data

0.3 mg/kg/d X 1 kg (mink wt) ÷ 0.15 kg/d (mink intake) =2 mg/kg.

-Chicken-2 data

0.02 mg/kg/d X 1 kg (bird wt) ÷ 0.2 kg/d (bird intake) = 0.1 mg/kg.

-Chicken-1 data 12.8 mg/kg/d X 0.1 (AF) X 1 kg (bird wt) ÷ 0.2 kg/d (bird intake) = 6.4 mg/kg.

-Coturnix-2 data

5.0 mg/kg/d X 0.1 (AF) X 1 kg (bird wt) ÷ 0.2 kg/d (bird intake)

= 2.5 mg/kg.

The value of 0.1 mg/kg is selected as the final non-carcinogenic based criterion.

To assess cancer risk to wildlife, the gamma HCH data will be used to derive a criterion for the sum of all HCH isomers. Conversion of the dose that corresponds to a 1×10^{-6} risk of cancer to experimental animals to a level in the diet of wildlife that would correspond to 1×10^{-3} and 1×10^{-2} risks of cancer involves the following steps:

1. 1×10^{-6} increased cancer risk dose in mice = 0.0076 ug/kg/day = 0.0076×10^{-3} mg/kg/d
2. 1×10^{-3} increased cancer risk dose in mice = 0.0076 mg/kg/day
3. Assume that the same dose will result in equal risk for mice and mink. Then convert the mouse dose to a mink dietary criterion:

   0.0076 mg/kg/day X 1 kg ÷ 0.15 kg/day = 0.051 mg/kg, 1 in 1000 cancer risk in diet
4. The 1 in 100 cancer risk in diet = 0.51 mg/kg

Comparison of Criteria with Residue Data

NRTC (1984) reported a median of "not detected" for HCH in spottail shiner in the Niagara River with a maximum of 0.034 mg/kg (Tables 3,4, and 26). The FDA (1977) reported levels of alpha HCH (alpha BHC in Table 5) of from 0.05 mg/kg to 0.43 mg/kg in white bass, smallmouth bass, and coho salmon. Residues in alewives, smelt and salmon from Lake Ontario and several non-salmonid fish from the Niagara River were less than or equal to 0.05 mg/kg (Tables 6 & 7).

Except for residues in some fish reported by FDA (1977) (Table 5) all residues in Niagara River and Lake Ontario fish, including the most recent data reported by NYS Dept. of Envir. Cons. (in prep.), are less than the 1 in 100 cancer risk and non-carcinogenic based criteria. Some recent measurements in carp and eel in the Niagara River are about equal to the 1 in 1000 cancer risk criterion (Table 7).

3.2.11 Hexachlorobutadiene

Hexachlorobutadiene (HCBD) is a by-product of certain processes associated with the chlorination of hydrocarbons. HCBD is toxic to experimental animals when inhaled, ingested, injected, or absorbed through the skin. It affects the central nervous system and causes hepatic disorders (IARC 1979). The kidney is the most sensitive organ. The acute toxicity LD50 for the rat is 90 mg/kg (NIOSH 1982). Feeding 20-30 mg/kg/day to rats for 30 days caused renal degeneration, necrosis, and regeneration (IARC 1979).

Lifetime ingestion of 0.2 mg/kg/day caused no discernible ill effects in rats (Kociba et al. 1976; Schwetz et al. 1977). The LOEL was established as 2.0 mg/kg/day for the rat due to increased urinary excretion and increased hyperplasia of the renal system. At the 20 mg/kg/day treatment level, a variety of toxic effects including mortality were reported (Kociba et al. 1977).

The NYS Department of Health calculated a lower 95% limit value for the HCBD dose corresponding to an increased lifetime cancer risk of 1 X 10^{-6} to rats of 0.068 ug/kg/day (DOH 1985i - using data from Kociba et al. 1977).

Calculation of HCBD Wildlife Fish Flesh Criteria

Table 19 summarizes data from dietary exposures of HCBD in animals.

1. Determining wildlife application/uncertainty factor for HCBD exposure based on animal data. Refer to Table 19 for details of the data selected below.

Study	Duration	LOEL or NOEL mg/kg/day (mg/kg-diet)	Recommended Application (AF)/ Uncertainty (UF) Factor
Rat-2	2 years	0.2 (NOEL)	None

2. Calculation of non-carcinogenic criterion with rat data (although only one NOEL was available the rate was the most sensitive species among three acute tests, justifying use of the NOEL without a UF).

   0.2 mg/kg/day X 1 kg (mink weight) ÷ 0.15 kg/day (mink intake) = 1.3 mg/kg.

Conversion of the dose that corresponds to a 1×10^{-6} risk of cancer to experimental animals to a level in the diet of wildlife that would correspond to 1×10^{-3} and 1×10^{-2} risks of cancer involves the following steps:

1. 1×10^{-6} increased cancer risk dose in rats = 0.068 ug/kg/day = 0.068×10^{-3} mg/kg/day.
2. 1×10^{-3} increased cancer risk dose in rats = 0.068 mg/kg/day.
3. Assume that the same dose will result in equal risk for rat and mink. Then convert the rat dose to a mink dietary criterion:

0.068 mg/kg/day X 1 kg - 0.15 kg/day = 0.45 mg/kg, 1 in 1000 cancer risk in diet.

4. The 1 in 100 cancer risk dietary criterion = 4.5 mg/kg.

Comparison of Criteria with Residue Data

HCBD does not represent a current threat to fish eating wildlife. Low residues have been detected in several Niagara River and western Lake Ontario fish. As indicated by criteria derived from toxicity tests with laboratory animals, current exposure to HCBD is below both carcinogenic and non-carcinogenic criteria. Only five of 23 stations were found to have HCBD residues in the Niagara River spottail shiners in 1982, with the highest residue of Q.029 mg/kg at Station N-15 on the lower river (Table 4).

The maximum residue reported by FDA (1977) was 0.08 ppm for Lake Ontario coho salmon collected in the Salmon River (Table 5).

3.2.12 Hexachloroethane (HCE)

Hexachloroethane is used in organic synthesis, as a retarding agent in fermentation, as a substitute for camphor in nitro cellulose, in pyrotechnics and smoke devices, and in the manufacture of explosives, solvents and medicines (EPA 1975). HCE is used to control liver and stomach flukes in domestic animals (Berg 1983). HCE has only been studied to a limited degree. No studies have been conducted to examine the acute, subchronic, or chronic effects of hexachlorethane in humans.

HCE was detected as a metabolite of carbon tetrachloride in rabbits following a 1 ml/kg dose in olive oil (Fowler 1969). Fat contained the highest concentration of HCE, muscle the lowest; tissue concentrations reached a peak in 24 hours, and persisted for as long as 44 hours (Fowler 1969).

The LD50 for acute toxicity in the rat is 6000 mg/kg (NIOSH 1982). Little chronic testing has been conducted with HCE. It is likely that there is fairly high uncertainty about the derived criterion. No cancer risk has been calculated for HCE, although it is possibly carcinogenic in mice (NCI 1978c).

Calculation of HCE Wildlife Fish Flesh Crieria

1. Determining wildlife application/uncertainty factor for HCE. Refer to Table 20 for details of data selected below.

Study	Duration	LOEL or NOEL mg/kg/day (mg/kg-diet)	Recommended Application (AF)/ Uncertainty (UF) Factor
RAT-2	5 1/2 months	0.05 (NOEL)	None
RAT-3	1 year	212 (LOEL)	0.1 (sub-acute to NOEL AF because no important sublethal or reproductive effects were studied) and 0.1 (interspecies UF)
Mouse-1	91 weeks	212 (LOEL)	0.2 (LOEL to NOEL AF) 0.1 (interspecies UF)

2. Rat-3 is selected to derive the fish flesh criterion with considerable reservation as reproduction was not studied; a LOEL was not determined for Rat-2.

212 mg/kg/d X 0.1 (AF) X 0.1 (UF) X 1 kg (mink wt) ÷ 0.15 kg/day (mink intake) = 14 mg/kg.

Comparison of Criterion with Residue Data

HCE residues are not usually detected in Niagara River fish. Spottail shiner HCE residues were not detectable for all samples in 1981 and 1982 (Table 3 and 4) except for one station with 4 ug/kg. The criterion of 14.1 mg/kg indicates that at present HCE residues in Niagara River fish have no effect on fish eating wildlife.

3.2.13 Octachlorostyrene

Octachlorostyrene (OCS) is an environmental contaminant identified in Great Lakes fish (Kuehl et al. 1976). Fish eating great blue heron were found to have OCS residues of 0.01-0.43 mg/kg within the U.S. (Reichel et al. 1977). The chemical has also been found in fish in the fjords of Norway (Ofstad et al. 1978) but the source of OCS is unknown. OCS may be produced during the manufacturing of magnesium (Chu et al. 1982).

Because of the presence of OCS as an environmental pollutant, concern has been raised over its toxicity and possible bioaccumulation. Strik and Koeman (1975) reported OCS as a potent porphyrinogen in rats and Coturnix quail. Porphyria is a disease typified by brittle skin, extreme light sensitivity, and deposition of porphyrins to the liver. Chu et al. (1982) found that OCS can produce hepatic changes even at low dietary levels and possessed some of the same toxic properties as hexachlorobenzene. Both chemicals exhibit low acute toxicity to rats. Chu et al.'s (1982) work is the most complete OCS toxicity study to date, but did not deal with reproductive effects. A listing of the few dietary effect studies of OCS in animals is presented in Table 21.

Although the toxicity of OCS may be comparable to HCB, Tarkpea et al. (1985) found the depuration half-life of OCS is twice that of HCB (143 days versus 81 days). The bioaccumulation potential of OCS is very

high. This seems to be borne out by OCS concentrations in fish and sediments as compared to accompanying waters in the German Bight (Ernst et al. 1984).

Calculation of OCS Wildlife Fish Flesh Criteria

Table 21 summarizes data from dietary exposures of octachlorostyrene in laboratory rats.

1. Determining wildlife application uncertainty factor for OCS dietary exposure based on laboratory animal data. Refer to Table 21 for details of data selected below.

Study	Duration	LOEL or NOEL mg/kg/day (mg/kg-diet)	Recommended Application (AF)/ Uncertainty (UF) Factor	
Rat-3	28 day	Liver damage	0.314 (sub-acute)	0.1 (sub-acute to NOEL AF) and 0.1 (interspecies UF)

2. Calculation of criterion

0.314 mg/kg/d X 0.1 (AF) X 0.1 (UF) X 1 kg (mink wt) ÷ 0.15 kg/d (mink intake) = 0.02 mg/kg.

Although no reproductive studies have been conducted, the estimated wildlife fish flesh criterion is based on organ damage that did not cause mortality. OCS toxicity has been compared to hexachlorobenzene (HCB) toxicity. Hexachlorobenzene has been found to be carcinogenic whereas OCS has not been adequately tested. Applying the method described in this report results in a fish flesh criterion for OCS about

an order of magnitude lower than the 1 $X10^{-2}$ cancer risk and non-carcinogenic criteria derived for HCB.

Comparison of Criterion with Residue Data

OCS has been identified in Niagara River spottail shiners in both 1981 and 1982 (NRTC 1984). The median OCS concentration in Niagara River spottail shiners in 1981 and 82 was 0.002 mg/kg; the maximum was 0.536 mg/kg (Tables 3,4, and 26). Suns et al. (1985) reported a similarly high (0.560 mg/kg) OCS residue for spottail shiners in 1983 samples in the St. Clair River, which suggested active OCS inputs to the St. Clair. These authors concluded that in general, residues in Great Lakes spottail shiners have been low, including Niagara River sites. No other OCS fish residue data have been obtained.

Based on non-carcinogenic toxicity, OCS is not a current hazard to fish eating wildlife on the Niagara River with the exception of station N-15 on the lower river and station N-13 in the Tonawanda - North Tonawanda section of the river.

3.2.14 Sum of Trichlorobenzenes

Trichlorobenzene (TCB) is present in the environment as a result of a variety of industrial processes. It is used as a dye carrier, dielectric and solvent, herbicide intermediate, fire retardant, and an oil (Robinson et al. 1981). It is also used as an herbicide and for termite control. TCB and other chlorinated benzenes also result from the breakdown of less stable pesticides such as hexachlorobenzene (Jondorf et al. 1955) and lindane (Saha and Burrage 1976). Several isomers of TCB exist including 1,2,4-trichlorobenzene, and 1,3,5-trichlorobenzene.

The acute toxicity of 1,2,4-TCB in the rat is 756 mg/kg for the single dose LD50 and for the mouse between 300 mg/kg (NIOSH 1982) and

766 mg/kg (Brown et al. 1969). TCBs are reported to have a slight effect on the liver compared to monochlorobenzene and 0-dichlorobenzene (Koch-Weser et al. 1953). Oral doses of TCB are excreted as phenolic derivatives (Jondorf et al. 1955).

FDA (1977) reported residues up to 0.36 mg/kg in Lake Ontario fish and from 0.49-1.0 in fish from the Niagara River (Table 5).

Calculation of Sum of Trichlorobenzene Wildlife Fish Flesh Criteria

Table 22 summarizes data from dietary exposure of 1,2,4-trichlorobenzene in lab animals. The rat studies of Robinson et al. (1981) and Carlson and Tardoff (1976) represent the only chronic exposures.

1. Determining wildlife application/uncertainty factors for TCB dietary exposure based on laboratory animal toxicity data. Refer to Table 22 for details of data selected below.

Study	Duration	Effect at LOEL	NOEL/NOEL mg/kg/day (mg/kg-diet)	Recommended Application (AF)/ Uncertainty (UF) Factor
Rat-2	1 year, 2 generations	Significant adrenal enlargement	10	0.2 (LOEL to NOEL AF and 0.1 (interspecies UF)
Rat-3	1 year	Xenobiotic induced liver metabolism	10	0.2 (LOEL to NOEL AF) and 0.1 (interspecies UF)

2. Calculation of criterion

10 mg/kg/d X 0.2 (AF) X 0.1 (UF) X 1 kg (mink wt) ÷ 0.15 kg/d (mink intake) = 1.33 mg/kg.

Trichlorobenzene has not been shown to be carcinogenic (EPA 1980c), therefore, no increased lifetime cancer risk in wildlife is calculated. Acute toxicity of TCB to animals can be summarized as moderate; lowest effect levels reported in the literature for chronic toxicity did not adversely effect survival or reproduction (Robinson et al. 1981). The relatively sparse data on TCB toxicity should indicate that there is a high degree of uncertainty about the fish flesh criterion of 1.33 mg/kg.

Comparison of Criterion with Residue Data

Although none of the reported residues exceed the criterion the uncertainty about the criterion suggests that the relatively high residues found may be of concern.

3.2.15 Pentachlorophenol

Pentachlorophenol (PCP) and its salts were used as biocides (Rao 1978) but U.S. production and sale was halted in 1985, and both allowed to resume on a restricted basis in 1986. Used mainly as a wood preservative, their anti-microbial, anti-fungal, herbicidal, insecticidal, and molluscidal properties led to widespread application of PCP formulations. U.S. production of PCP was about 80 million pounds in 1977 and expanding (Cirelli 1978). Residues of PCP had become one of the most ubiquitous contaminants worldwide (RAO 1978). In most of the U.S. and countries that have discontinued PCP usage these residues will probably decline. However, in the Niagara frontier, there are a number of manufacturing processes that can inadvertantly generate PCP.

Impurities produced during PCP production are contained in technical or commercial grade PCP. These impurities have also increased the toxicity of PCP containing diets for laboratory animals. PCP was most commonly available as a sodium salt, as a 5% emulsifiable concentrate or as a 3-40% solution in oil or grease. The NRC Drinking Water and Health (1977) lists commercial grade PCP as a containing 88.4% PCP, 4.4% tetrachlorophenol, 6.2% higher-chlorinated phenoxy phenols, and less than 1% trichlorophenol and various chlorinated dibenzo-p-dioxins and dibenzofurans. Jansson and Sundstrom (1978) found that manufacture and combustion variables could result in undesirable impurities which had earlier led to the ban on PCP formulations in Sweden.

The environmental fate, stability, and environmental significance of PCP has been reviewed by Arsenault (1976). PCP is readily lost from animal tissues, but many of its precursors and metabolites are persistent (Conklin and Fox 1978). The principal matabolites such as pentachloroanisole have not been researched for dietary toxicity.

The recorded aquatic impacts of PCP causing fish kills include accidents involving: Flooding of wood treatment tanks which ordinarily contained 10,000 mg/kg PCP or tetrachlorophenol, spraying of telephone poles near water, and discarding of wastes containing high concentrations of PCP in landfills (Conklin and Fox 1978). Vermeer et al. (1974) documented a large fish kill and heavy mortality of snail kites after use of PCP as a molluscicide in rice fields. The snail kite feeds solely on Pomacea snails which contain about 32 ug/g PCP.

Investigations of the dietary effects of PCP have been limited to a few laboratory animals (Table 25). Schwetz et al. (1977) maintained rats on diets containing PCP, characterized by low content of

non-phenolic properties, for up to 24 months. PCP was found to be non-carcinogenic at dose levels high enough to cause mild signs of toxicity (1, 3, 10, and 30 mg/kg/day). Schwetz et al. (1977) concluded that 3 mg/kg/day had no effect on neonatal growth, survival, or development. Male rats were more tolerant than females to 10 mg/kg/day without adverse effect.

PCP dietary exposures of piscivorous wildlife species found along the Niagara River have not been performed.

Calculating PCP dietary Wildlife Fish Flesh Criteria

PCP has not been extensively tested in birds or mammals. Table 23 summarizes data from dietary exposures of PCP. Chronic feeding studies which included histopathological and reproductive aspects have been conducted with the laboratory rat (Johnson et al. 1973; Schwetz et al. 1977).

1. Determining wildlife application/uncertainty factors for PCP dietary exposure based on animal data. Refer to Table 23 for details of data selected below.

Study	Duration	Effect at LOEL	Recommended NOEL/LOEL mg/kg/day (mg/kg-diet)	Application (AF)/ Uncertainty (UF) Factor
Rat-4	90 days	Increased liver weight	3.0 (NOEL)	0.1 (interspecies UF)
Rat-5	2 years	Darkening of the liver	3.0 (NOEL)	0.1 (interspecies UF)

2. Calculation of criterion

3.0 mg/kg/day X 0.1 (UF) X 1 kg (mink) ÷ 0.15 kg/day (mink intake) =

2.0 mg/kg.

Schwetz et al. (1977) reported that PCP had not been found to be carcinogenic in the rat, therefore, only non-carcinogenic effects were considered.

Comparison of Criteria with Residue Data

Pentachlorophenol was found in the majority of spottail shiner samples from the Niagara River (Table 4). Levels of PCP ranged from non-detectable to 70 ug/kg with a median of 10 ug/kg in the young of the year spottails. FDA measurements of samples of Niagara River composites of several fish species contained 50 ug/kg (ppb) pentachloroanisole (Table 5) which is a major degradation product of PCP under aerobic conditions, and appears to be a persistent compound.

The highest spottail concentration reported for the Niagara River is 70 ug/kg at the Wheatfield upper river station (M-11). Therefore, the current PCP contaminant levels do not pose a risk to wildlife consumers. The FDA (1977) reported 50 ug/kg pentachloroanisole, the primary metabolite of PCP. Without data on the toxicity of pentachloroanisole it can not be determined whether a fish flesh criterion should be for PCP alone or if it should also include PCP degradation products.

3.2.16 2,3,4,6-Tetrachlorophenol

Tetrachlorophenol (TCP) probably arises from the incomplete chlorination of phenol during manufacturing processes. Tetrachlorophenol (TCP) is the major impurity in the commercial production of pentachlorophenol (Bruns and Currie 1980). The acute toxicity of TCP via single intraperitoneal dose was 130 mg/kg (NIOSH 1982). The oral LD50 for the rat was 140 mg/kg; for the guinea pig it was 250 mg/kg (NIOSH 1982).

The short term toxicity of TCP is almost entirely focused on the liver (Bruns and Currie 1980). Rats were exposed daily by gavage to TCP in olive oil at 0, 10, 50, and 100 mg/kg/day dosage level for 55 days. Some liver damage occured above 10 mg/kg/day dosage. At dosages of 50 and 100 mg/kg/day residues as high as 50 mg/kg were measured in spleen and kidney, with the lowest residues in the muscle and brain. Severe necrosis of the liver was noted in rats exposed to the higher dosage levels (Hattula et al. 1981).

Carcinogenicity of TCP has not been directly evaluated (EPA 1980c).

Calculation of TCP Wildlife Fish Flesh Criteria

Table 24 summarizes data from the few dietary exposures of TCP to rats. The single dietary (gavage) exposure appropriate for

extrapolation to a wildlife NOEL is the 55 day subacute NOEL of 10 mg/kg (Hattula et al. 1981).

1. Determining wildlife application/uncertainty factors for 2,3,4,6-tetrachlorophenol dietary exposure based on laboratory rat data. Refer to Table 24 for details of data selected below.

Study	Duration	Effect	NOEL/LOEL mg/kg/day (mg/kg-diet)	Recommended Application (AF)/ Uncertainty (UF) Factor
Rat-3	55 days	Liver damage	10 (sub-acute LOEL)	0.1 (sub-acute to NOEL AF) and 0.1 (interspecies UF)

2. Calculation of criterion

10 mg/kg/d X 0.1 (AF) X 0.1 (UF) X 1 kg (mink wt) ÷ 0.15 kg/d (mink intake) = 0.67 mg/kg.

The dearth of dietary studies of other species and studies of carcinogenicity lends uncertainty to the criterion. Since TCP is a major impurity in pentachlorophenol (PCP) technical grade, dietary studies of PCP have included up to 30% TCP. The estimated wildlife criterion of PCP is 2.0 mg/kg which is fairly close to the estimated TCP wildlife criterion of 0.67.

Comparison of Criterion with Residue Data

Niagara River fish residues of TCP were generally not detectable although several samples of 0.004 to 0.007 mg/kg were reported (Tables 3 & 4) (NRTC 1984). TCP has rarely been reported in Niagara River and Lake Ontario fish.

The TCP criterion of 0.67 mg/kg is about two orders of magnitude above the spottail shiner residues. Fish with higher lipids could be expected to contain greater amounts of TCP than the spottails, but possibly less than the fish flesh criterion.

3.2.17. OTHER CHEMICALS

Insufficient toxicity data was available for the remaining chemicals reported by NRTC (1984) (Tables 3 & 4) known to occur in the Lake Erie/Niagara River spottail shiners (tetrachlorobenzenes, pentachlorobenzene and trichlorophenols) to derive fish flesh criteria.

4.0 SUMMARY OF RESULTS AND DISCUSSION

Criteria to establish no-effect (non-carcinogenic) levels in fish to protect piscivorous wildlife were derived for 16 of 19 organochlorine chemicals or chemical groups that have been found in Niagara River spottail shiners; cancer risk criteria were derived for 10 of the 19 chemical groups. The risk assessment methods used to derive the criteria are discussed below, including some limitations or factors not included in the methods. In addition, criteria are compared to contaminant residues in several fish species in the Niagara River.

4.1 Summary of Application and Uncertainty Factors

There are several application and uncertainty factors that may need to be applied when extrapolating from limited animal tests to estimation of no effect levels in target species. The following is a summary of these factors and how they have been applied in this study.

4.1.1 Acute to Chronic Toxicity Adjustment

All of the chemicals reviewed in this study are toxic at much lower levels on a chronic basis than on an acute basis. Where appropriate and after careful review, short-term (about 30 days or less) effect levels were multiplied by 0.1 to estimate the chronic NOEL. The selection of the application factor of 0.1 was based on evidence presented by Weil and McCollister (1963).

4.1.2 LOEL to NOEL Adjustment

All of the chemicals for which there is extensive data and which are reviewed in this study have no effect levels well below the lowest effect levels, although the severity of the effect in the LOEL should be considered. LOELS were multiplied by 0.2 to estimate the NOEL based on evidence presented in Dourson and Stara (1983) and Weil and McCollister (1963).

4.1.3 Inter-species Adjustment

Final criteria were based on species from dietary feeding studies which resulted in the most protective criteria. Trial calculation of criteria using toxicity test results for a number of species and for five chemicals demonstrates interspecies variation in tolerance to the contaminants (Table 25).

The ratio of the least protective to the most protective of the non-carcinogenic based trial criteria was 3:1 for PCB's, 10:1 for DDT and metabolites, 11:1 for dieldrin, 17.2:1 for endrin, and 6:1 for mirex.

In addition, the most sensitive species for each chemical varied. Such interspecies variation in sensitivity supports the use of an uncertainty factor in developing criteria so that less tolerant species will be protected. The remaining contaminants were not tested extensively enough to develop such ratios.

For a contaminant for which only one animal species has been tested in dietary feeding studies (such as octachlorostyrene or hexachloroethane) it would be probable that more sensitive species exist. Therefore a 0.1 interspecies adjustment factor should be employed to calculate the final fish flesh criteria for the relatively unstudied chemicals.

4.2 Comparison of Target and Non-Target Species Based Criteria

Sufficient toxicity data with target species (any of the species listed in Table 2) was available to derive fish flesh criteria for five of the chemicals evaluated in this study. In addition, criteria were derived for each of these chemicals based on toxicity data with three or more lab or other non-target species. Table 25 summarizes all candidate criteria derived for these five chemicals, along with

application/uncertainty factors used in deriving the criteria. For each of the five chemicals, except endrin, at least one of the lab species-based non-carcinogenic criteria was lower than target species criteria. In the case of endrin, the mallard exhibits atypically high sensitivity to the chemical. In general, the methods used in this study result in non-carcinogenic criteria derived using lab species that will protect target species. Therefore, it is concluded that for the remainder of the NRTC (1984) chemicals of concern for which fish flesh criteria were derived based only on non-target species, the criteria can be expected to be adequate to protect target species.

All of the ten 1 in 100 cancer risk criteria that are available for the NRTC chemicals are within an order of magnitude of the non-carcinogenic based criteria. It is proprosed that a 1 in 100 cancer risk is an adequate level of protection for wildlife populations to ensure that there will be virtually no reduction in a population from toxic-induced cancer. In the case of humans, no cancer risk is considered acceptable, and there are no recognized safe concentrations either; thus, attaining a zero concentration may be infeasible. Consequently, a one in one million risk concentration is often considered virtually "safe". For wildlife it is not clear that a goal of virtually no toxic induced cancer is reasonable, that cancer is even a significant occurrence in wildlife populations, or that cancer significantly affects wildlife population levels or use of wildlife. The selection of an acceptable level of cancer risk should receive more study to determine whether the preliminary conclusion, that a 1 in 100 risk is appropriate, is justified.

4.3 Relationship Between Food Habits and Exposure to Contaminants

The diet of wildlife feeding almost entirely on aquatic animal food other than fish was considered to be as contaminated as the diet of wildlife feeding predominantly on fish. To arrive at this conclusion some studies of contaminant uptake in fish and invertebrates were reviewed. Metcalf et al. (1973) studied a model ecosystem and found that the bioaccumulation factor of bis(2-ethylhexyl) phthalate in mosquitofish was 130 and 108,000 in mosquito larvae. In another model ecosystem Lu et al. (1977-in Neff 1979) studied uptake of benzo(a)pyrene (BaP) in the presence of a mixed function oxidase inhibitor which enhances retention of BaP in fish and, to some extent, insects. After three days BaP levels in snails were greater than mosquito larvae which were greater than fish. In two waters of Norway, Bjerk and Brevik (1980) found that DDT, PCB and pentachlorobenzene were at uniform levels on a percent lipid basis in several marine fishes and invertebrates. Unpublished data on the Moreau Marsh in New York, adjacent to an inactive hazardous waste site containing PCB (E. Horn, NYS Dept. Envir. Cons. pers. comm.) also illustrates relatively similar PCB contamination of food items (Table 27).

Shorebirds such as herring gulls on the average contain even higher residues in relation to fish (See Chemical and Wildlife Narratives). Thus, carnivores which feed high on the food chain in the Niagara River-Lake Ontario ecosystem may be at even more risk than strict fish eaters.

The bald eagle was found to be more tolerant to endrin and dieldrin than a number of commonly tested species (Stickel et al. 1969), however, the total exposure levels are expected to be higher. If an eagle consumes an occasional herring gull at from 10 to 50 times the contaminant level of fish it might accumulate lethal residues as quickly as a less tolerant animal feeding at lower trophic levels.

In general, plants appear to accumulate organochlorines to a lesser extent than animals. Cattail rhizomes in the Moreau marsh had PCB residues comparable to animals, but Carex and pondweed were lower than all animals, except for PCB in muskrat muscle (Table 27). In the Ft. Edwards-Thompson Island section of the upper Hudson River, Bush et al. (1987) found PCB in Valisineria, Elodea and algal mats at 0.32, 0.42 and 0.1 mg/kg, respectively. Average PCB in seven species of fish from the same area in the river ranged from 7.2-123.3 mg/kg (DEC in prep.). For the purpose of the assessments in this report the diet of wildlife that eat aquatic plants was considered to contain lower organochlorine residues than the diet of wildlife that eat animals. Therefore, only risk to carnivorous wildlife was assessed. If herbivorous wildlife consume more food per day than carnivorous wildlife, then their exposure to organochlorines may not be lower than carnivorous wildlife.

4.4 Some Limitations to the Methodology

4.4.1. Exposure in Nature Versus the Laboratory

Most wildlife experience times with decreased availability of food and water—periods of starvation or high energy demands. Lab animals, in contrast, usually have unlimited access to food and water. IJC (1986) suggests that this is an important concept to consider, since the stress of these situations may result in NOELS for wildlife that are

lower than shown by lab animal studies. Therefore, wildlife in nature may be more susceptible to effects of toxics.

In addition, many of the contaminants studied accumulate in fat and can be mobilized during starvation, migration, etc. (Mitjavila, Carrera, and Fernandez 1981). Weights of many wildlife species vary with the season (Seibert 1949). DDT and its metabolites rapidly mobilized from the fat to the body organs in tests with the rat, although no major toxic signs developed (Mitjavila, Carrera and Fernandez 1981). Fatter individual birds survived longer in dieldrin tests due to fat storage of contaminants, but then irreversible cessation of feeding occurred and dieldrin mobilization to the brain caused death. Wildlife may be exposed to contaminants at one location, but not suffer effects until some time later at another location.

4.4.2 Diagonostic Brain Levels of Contaminants in Wildlife

Lethal levels of several contaminants in the brain have been established for some laboratory and wildlife species (Heinz and Johnson 1981). These diagnostic brain levels are presented in the chemical narratives. DDT brain levels of about 30 mg/kg are likely to be lethal in birds, with lethal dieldrin brain levels at about 8 mg/kg. Dietary levels of the contaminants required to achieve the diagnostic brain level need to be better established using surrogate species if necessary. The fish residue levels for DDT and dieldrin in Tables 3-6 were probably insufficient to cause lethal brain levels in birds.

4.4.3 Possibility of Synergistic or Unexpected Effects Due to Contaminant Combination

Michael Gilbertson of Fisheries and Oceans, Canada (1986 pers. comm.) advanced three reasons why calculation of acceptable levels of organochlorine pollutants in fish for safe consumption by wildlife is so problematic:

1. as biologically active chemicals the organochlorines may cause adverse, sublethal effects that are currently unobservable;
2. the organochlorines generally occur as a mixture and effects of mixtures have not been accounted for in setting criteria; and
3. seemingly acceptable levels of a chemical have been known to alter susceptability to other toxics.

4.5. Comparison of Criteria with Niagara River/Lake Ontario Fish Residues

To assess risk to wildlife fish consumers, fish flesh criteria (based on non-carcinogenic data and 1 in 100 cancer risk) were compared to residues in Niagara River fish. A variety of stations were sampled for spottail shiners (NRTC 1984); for each contaminant criteria were compared to the residues in spottail shiners. For each contaminant fish flesh criteria were developed in the chemical narrative section, and they are summarized in Table 26. Other fish residue data for alewives, smelt, and coho salmon (Norstrom et al. 1978, composite fish samples of white bass and smallmouth bass and residues in several salmonids from Lake Ontario and non-salmonids from the Niagara River (NYS Dept. Envir. Cons. in prep.; FDA 1977) were also compared to the fish flesh criteria.

Residues in alewives and smelt, some salmonids, white bass, black bass and eel were invariably higher than those in young-of-the-year spottail shiners. Alewives and smelt average about 5% lipid content. Spottail shiners average about 2% lipid content (L. Skinner, NYS Dept. Envir. Cons. pers. comm.; DEC in prep.). Extrapolation from residues in spottail shiners to other higher lipid content species furthers the utility of monitoring spottails, but

if a chemical is not detected in the spottail shiner one may not safely conclude that it does not occur in other species.

Median PCB residues in spottail shiner exceed the 0.11 mg/kg dietary fish flesh criterion for non-carcinogenic effects, and exceed the 1 in 100 cancer risk criterion of 0.11 mg/kg (Table 26; Table 6). All other species analyzed exceeded the criteria by 2 to over 100 times (Table 26). Dieldrin residues in spottail shiner are lower than the non-carcinogen fish flesh criterion and the 1 in 1000 cancer risk. Residues in other fish species exceed the 1 in 100 cancer risk, but were lower than the dieldrin non-carcinogenic based criterion. DDT, DDD, and DDE also pose a present risk to wildlife consumers of some fish species, exceeding the 1 in 100 cancer risk and non-carcinogenic based criteria. However, spottail shiner residues are below both criteria.

Some other fish residues exceed the criteria for both non-carcinogenic effects and the 1 in 100 cancer risk. Dioxin (2,3,7,8-TCDD) poses a risk to wildlife fish consumers, with the maximum residues in spottail shiners exceeding both the non-carcinogenic based and cancer risk fish flesh criteria as do other fish (FDA 1977) by as much as 50 times. The fish flesh criteria for dioxin are less than the detection limit; dioxin was detected in spottail shiners at all of five stations sampled in 1981 and at 2 of 13 stations in 1982.

Median hexachlorobenzene (HCB) residues in spottail shiners were below the criteria for non-carcinogenic effects and the 1 in 100 cancer risk. Other fish (Table 5) residue averages from the Niagara River exceeded both HCB criteria and therefore pose some risk to wildlife fish consumers. However, recent measurements of HCB in Lake Ontario and Niagara River fish are all less than both criteria (Table 7). Mirex

(including photomirex) poses a marginal risk to consumers. Spottail shiner residues were less than both criteria. Fish of some other species exhibit combined mirex and photomirex residues in excess of criteria (Tables 5-7). Chlordane residues in spottail shiners are less than criteria (Tables 3 and 4); residues in lake trout from Lake Ontario and eel from the Niagara River exceed both the 1 in 100 cancer risk and the non-carcinogenic based criteria (Table 7). Octachlorostyrene in spottails exceeded the criterion at two sites, and residues in other fish exceed the criterion by as much as ten times. Other contaminants are apparently not present in levels high enough to threaten wildlife consumers of fish. See the chemical narratives for results and discussion of the individual contaminants.

Spottail shiners would be marginally toxic for fish eating wildlife from the standpoint of contamination, due to the amount the criteria are exceeded on the average. However, a number of stations on the river have spottail residues which exceed the criteria considerably for the above mentioned problem contaminants. Reproduction impairment and organ damage could occur with chronic exposure to spottails from the more polluted locations. Other fish species (alewives, smelt, several salmonids, white bass, black bass and eel) levels are high enough to predict mortality, reproductive impairment, and organ damage in sensitive wildlife species. Actual occurrence of effects would depend on the extent to which individual animals consume those fish species with residues in excess of criteria and the duration for which those species are consumed.

REFERENCES

Alexander, G. 1977. Food of vertebrate predators of trout waters in north central lower Michigan. Michigan Academician. 10:181-195.

Allen, J., W. Hargraves, M. Hsia, and F. Lin. 1980. Comparative toxicology of chlorinated compounds on mammalian species. Pharmac. Ther. 7:513-547.

Anderson, D. and J. Hickey. 1976. Dynamics of storage of organochlorine pollutants in herring gulls. Environ. Pollut. 10:183.

Arsenault, R. 1976. The environmental fate, stability and environmental significance of pentachlorophenol. Proc. Am. Wood-Preserv. Assoc. 122pp.

Aulerich, R., S. Bursian, W. Breslin, B. Olsen, and R. Ringer. 1985. Toxicological manifestation of 2,4,5,2',4',5'-2,3,6,2',3',6'-and 3,4,5,3',4',5'-hexachlorobiphenyl and Aroclor 1254 in mink. J. Toxicol. Environ. Health. 15:63-79.

Aulerich, R.J. and R. Ringer. 1977. Current status of PCB toxicity in mink and effect on their reproduction. Arch. Environ. Contam. Toxicol. 6:279-292.

Aulerich, R., R. Ringer, and S. Iwamoto. 1973. Reproductive failure and mortality in mink fed on Great Lakes fish. J. Reprod. Fert., Suppl. 19:365-376.

Balba, H.M. and J.G. Saha. 1978. Studies on the distribution, excretion, and metabolism of a cis-and trans-isomers of [C-14] chlordane in rabbits. J. Environ. Sci. Health. B13(3):211-233.

Barsotti, D.A., L. Abrahamson, and J. Allen. 1979. Hormonal alterations in female rhesus monkeys fed a diet containing 2,3,7,8 tetrachloro dibenzo-p-dioxin. Bull. Environ. Contam. Toxic. 21(4-5):463-469.

Beardslee, C.S. and H.D. Mitchell. 1965. Birds of the Niagara Frontier Region. Bull. of the Buffalo. Soc. of Nat. Sci., Buffalo, N.Y. Vol. 22. 478pp.

Bellrose, F. 1976. Ducks, Geese and Swans of North America. Stackpole Books, Harrisburg, Pennsylvania. 544pp.

Bellrose, F. and A. Hawkins. 1947. Duck weights in Illinois. Auk 64:422-430.

Bent, A. 1963a. Life histories of North American Gulls and Terns. Dover Publications, Inc., New York. 337pp.

Bent, A. 1963b. Life Histories of North American Diving Birds. Dover Publications, Inc., New York. 292pp.

Berg, G. (ed.). 1983. Farm Chemicals Handbook. Meister Publ. Co., Willoughby, Ohio. Section C, 264pp.

Bernard, R. 1963. Studies on the effects of DDT on birds. Mich. State Univ. pp. 161-191.

Bio-Test Laboratories, 1970. Two year chronic oral toxicity study of Aroclor 1254 in albino rats. A status summary prepared for Monsanto Co. 3 pp.

Bjerk, J.E. and E.M. Brevik. 1980. Organochlorine compounds in aquatic environments. Arch. Environ. Contam. Toxicol. 9:743-750.

Blakely, B. 1982. Lindane toxicity in pigeons. Can. Vet. J. 23:267-268.

Bleavins, M.R., W.J. Breslin, R.J. Aulerich, and R.K. Ringer. 1984. Placental and mammary transfer of a polychlorinated biphenyl mixture (Aroclor 1254) in the European ferret (Mustella putorius furo). Environ. Toxic. and Chem. 3:637-644.

Blus, L. 1982. Further interpreation of the relation of organochlorine residues in brown pelican eggs to reproductive success. Environ. Pollut. 28A:15-33.

Blus, L. 1972. Further analysis of the logarithmic relationship of DDE residues to nest success. Nature 240:164.

Blus, L., E. Cromartie, L. McNease, and T. Joanen. 1979. Brown pelican: Population status, reproductive success, and organochlorine residue in Louisiana. Bull. Environ. Contam. Toxic. 22:128-135.

Blus, L., R. Heath, C. Gish, A. Belisle, and R. Prouty. 1971. Eggshell thinning in the brown pelican: Implication of DDE. Bioscience 21: 1213-1215.

Booth, N. and J. McDowell. 1975. Toxicity of hexachlorobenzene and associated residues in edible animal tissues. J. Am. Vet. Med. Assoc. 106:591-595.

Brimfield, A., J. Street, J. Futrell, and D. Chatfield. 1978. Identification of products arising from the the metabolism of cis- and trans-chlordane in rat liver microsomes in vitro: Outline of a possible metabolic pathway. Pest. Biochem. and Physiol. 9:84-95.

Britton, W. and T. Huston. 1973. Influence of polychlorinated biphenyls in the laying hen. Poult. Sci. 52(4):1620.

Brown, V., C. Muir, and E. Thorpe. 1969. The acute toxicity and skin irritant properties of 1,2,4-trichlorobenzene. Ann. Occup. Hyg. 12:209-212.

Brown, L. and D. Amadon. 1968. Eagles, Hawks, and Falcons of the World, Vol. I. McGraw Hill, publ., New York. 414 pp.

Bruns, G. and R. Currie. 1980. Extraction of pentachlorophenol and tetrachlorophenol residues from field-contaminated carrots and potatoes: Comparison of several methods. J. Assoc. Off. Anal. Chem. 63(1):56.

Bryant, H. 1914. Auk 31:168-77. In R. Palmer (ed.), Handbook of North American Birds. Yale Univ. Press, New Haven.

Buckley, J. and P. Springer. 1965. Waterfowl Tomorrow. Doubleday Publ., New York. 655pp.

Bull, J. 1975. Birds of the New York Area. Dover Publ., New York. 534pp.

Burdick, G., E. Harris, H. Dean, T. Walker, J. Skea, and D. Colby. 1964. The accumulation of DDT in lake trout and the effect on reproduction. Trans. Am. Fish. Soc. 93(2):127-136.

Bush, B., C.A. Shane and M. Wahlen. 1987. Sedimentation of 74 PCB Congeners in the Upper Hudson River. Chemosphere 16:733-744.

Cam, C. and G. Nigogosyan. 1963. Acquired toxic porphyria cutanea tarda due to HCB. J. Amer. Med. Assoc. 183:88-91.

Cameron, E. 1906. Nesting of the Great Blue Heron in Montana. Auk 23:252-262. In: R. Palmer (ed.), Handbook of North American Birds. Yale Univ. Press, New Haven.

Carlson, G.P. and R.G. Tardiff. 1976. Effect of chlorinated benzenes on the metabolism of foreign organic compounds. Toxicol. Appl. Pharmacol. 36:383-394.

Carr, A. 1952. Handbook of turtles. Cornell Univ. Press. Ithaca, N.Y. 540pp.

Cecil, H.C., J. Bitman, R.J. Lillie, G.F. Fries, and J. Verrett. 1974. Embryotoxic and teratogenic effects in unhatched fertile eggs from hens fed polychlorinated biphenyls (PCBs). Bull. Environ. Contam. Toxicol. 11:489-495.

Chadwick, R., R. Linko, J. Freal, and A. Robbins. 1975. The effect of age and long-term low-level DDT exposure on the response to enzyme induction in the rat. Toxic. and Appl. Pharmacol. 31(3):469-480.

Chapman, J. and G. Feldhammer. 1982. Wild Mammals of North America. John Hopkins Univ. Press., Baltimore. 1123pp.

Chernoff, N., R. Linder, T. Scotti, E. Rodgers, B. Carver and R. Kaulock. 1979. Fetotoxicity and carcinogenicity of mirex in rats and mice with notes on Kepone. Environ. Sci. Res. 18:257-269.

Chernoff, N., R.J. Kavlock, J.R. Kathrein, J. M. Dunn, and J.K. Haseman. 1975. Prenatal effects of dieldrin and photodieldrin in mice and rats. Toxicol. Appl. Pharmacol. 31:302-308.

Chu, I., D.C. Villeneuve, G.C. Becking, F. Iverson, L. Ritter, V.E. Valli, and L.M. Reynolds. 1980. Short-term study of the combined effects of mirex, photomirex, and kepone with halogenated biphenyls in rats. J. Toxicol. Environ. Hlth. 6:421-432.

Chu, I., D.C. Villeneuve, V. Valli, V. Secours, and G. Becking. 1981. Chronic toxicity of photomirex in the rat. Toxicol. Appl. Pharmacol. 59:268-278.

Chu, I., V. Secours, and D. Villeneuve. 1982. Acute and subacute toxicity of octachlorostyrene in the rat. J. Toxicol. Environ. Hlth. 10:285-296.

Cirelli, D. 1978. Patterns of pentachlorophenol usage in the United States of America - An overview In: Pentachlorophenol. Environ. Sci. Res. 12:13-18.

Clark, D. and A. Krynitsky. 1980. Organochlorine residues in eggs of loggerhead and green sea turtles nesting at Merrit Island, Florida-July-August, 1976. Pest. Monit. J. 14(1):7-10.

Clarke, M.L., D.G. Harvey and D.J. Humphreys. 1981. Veterinary Toxicology, 2nd ed. B. Tindall, London. 328pp.

Clayton, G.D. and F.E. Clayton (ed.). 1981. Patty's Industrial Hygiene and Toxicology. Three volumes. John Wiley and Sons, N.Y.

Conklin, P. and F. Fox. 1978. Environmental impacts of pentachlorophenol and its products. In Pentachlorophenol. Environ. Sci. Res. 12:389-394.

Cottam, ____ and ____ Uhler. 1945. U.S. Fish and Wildlife Service leaflet 272. In R. Palmer (ed.), Handbook of North American Birds. Yale Univ. Press, New Haven (paper not seen).

Cottam, ____. 1939. U.S. Dept. Agric., Tech. Bull. No. 643 In R. Palmer (ed.), Handbook of North American Birds. Yale Univ. Press, New Haven. (paper not seen).

Courtney, P. and H. Blokpoel. 1980. Food and indicators of food availability for common terns on the lower great lakes. Can. J. Zool. 58:1318-1323.

Courtney, K. 1979. Hexachlorobenzene (HCB): A review. Environ. Res. 20:225-266.

Cranmer, J.M., M.F. Cranmer, and P.T. Goad. 1984. Prenatal chlordane exposure: Effects on plasma corticosterone concentrations over the lifespan of mice. Environ. Res. 35: 204-210.

Cromartie, E., W. Reichel, N. Locke, A. Belisle, T. Kaiser, T. Lamont, B. Mulhern, R. Prouty, and D. Swineford. 1975. Residues of organochlorine pesticides and polychlorinated biphenyls and autopsy data for bald eagles, 1971-72. Pest. Monit. J. 9(1): 11-14.

Currier, M., C. McClimans, and G. Barna-Lloyd. 1980. Hexachlorobenzene blood levels and the health status of men employed in the maufacture of chlorinated solvents. J. Toxic. and Environ. Health. 6:367-377.

Davis, K. 1965. Pathology report on mice fed aldrin, dieldrin, heptachlor, or heptachlor epoxide for two years. International memorandum to Dr. A.J. Lehman. USFDA.

DEC (NYS Dept. of Environmental Conservation). 1986a. Contaminants in Hudson River striped bass: 1978-1985. Tech. Rep. 86-2, Bureau of Environmental Protection, Division of Fish and Wildlife, NYS Dept. of Environmental Conservation, Albany, N.Y.

DEC (NYS Dept. of Environmental Conservation). 1986b. Draft Env. Impact Statement on Amendments to 6NYCRR Part 326. Relating to the Restriction of the Pesticides Aldrin, Chlordane, Chlorpyrifos, Dieldrin, and Heptachlor. Bureau of Pesticides management, Division of Lands and Forests, NYS. Dept. of Environmental Conservation, Albany, N.Y.

DEC (NYS Dept. of Environmental Conservation). In prep. Toxic substances in Fish and Wildlife, Volume VI, Bureau of Environmental Protection, Division of Fish and Wildlife, NYS Dept. of Environmental Conservation, Albany, N.Y.

DEC (NYS Dept. of Environmental Conservation). In prep. (a). Breeding Bird Atlas data 1980-1985, Bureau of Wildlife, Division of Fish and Wildlife, NYS Dept. of Environmental Conservation, Delmar, N.Y.

Dementiev, G. and N. Gladkov (eds.). 1952. Birds of the Soviet Union. Transl. Publ. Israel program for scientific translations, Jerusalem. Vol. 4. 683pp.

denTonkelaar, E. and G. van Esch. 1974. No effect levels of organochlorine pesticides based on induction of microsomal liver enzymes in short-term toxicity experiments. Toxicology 2:371-380.

Deo, P.G., S.B. Hasan and S.K. Majumder. 1982. Interconversion of hexachlorocyclohexane isomers. J. Food Science and Technology 19:221-227.

DeWitt, J. and J. George. 1957. Bureau of Sport Fisheries and Wildlife. Pesticide-Wildlife Rev. U.S. Fish and Wildlife Service, U.S. Dept. of Interior, Wash., D.C.

DOH (NYS Department of Health). 1985a. Ambient water quality standards fact sheet for polychlorinated biphenyls (PCBs). Prepared for Criteria and Standards Section, Division of Water, New York State Department of Environmental Conservation. 3pp.

DOH (NYS Department of Health). 1985b. Ambient water quality standards fact sheet for DDT; DDD; DDE. Prepared for Criteria and Standards Section, Division of Water, NYS Dept. of Environmental Conservation. 5pp.

DOH (NYS Department of Health). 1985c. Ambient water quality standards fact sheet for dieldrin. Prepared for Criteria and Standards Section, Division of Water, NYS Dept. of Environmental Conservation. 3pp.

DOH (NYS Department of Health). 1985d. Ambient water quality standards fact sheet for chlordane. Prepared for Criteria and Standards Section, Division of Water, NYS Dept. of Environmental Conservation. 2pp.

DOH (NYS Department of Health). 1985e. Ambient water qualtiy standards fact sheet for heptachlor and heptachlor epoxide. Prepared for Criteria and Standards Section, Division of Water, NYS Dept. of Environmental Conservation. 3pp.

DOH (NYS Department of Health). 1985f. Ambient water quality standards fact sheet for mirex. Prepared for Criteria and Standards Section, Division of Water, NYS Dept. of Environmental Conservation. 3pp.

DOH (NYS Department of Health). 1985g. Ambient water quality standards fact sheet for hexachlorobenzene. Prepared for Criteria and Standards Section, Division of Water, NYS Dept. of Environmental Conservation. 3pp.

DOH (NYS Department of Health). 1985h. Ambient water quality standard fact sheet for hexachlorocyclohexane (HCH) and the isomers alpha-HCH, beta-HCH, gamma-HCH (Lindane), delta-HCH and epsilon-HCH. Prepared for Criteria and Standards Section, Division of Water, NYS Dept. of Environmental Conservation. 4pp.

DOH (NYS Department of Health). 1985i. Ambient water quality standard fact sheet for hexachlorobutadiene (HCBD). Prepared for Standards and Criteria Section, Division of Water, NYS Dept. of Environmental Conservation. 3pp.

Doull, J., C.D. Klaassen, and M.O. Amdur (eds.). 1980. Casarett and Doull's Toxicology: Basic Science of Poisons, 2nd ed., MacMillan Publ., N.Y. 778 pp.

Dourson, M. and J. Stara. 1983. Regulatory history and experimental support of uncertainty (safety) factors. Reg. Toxicol. and Pharmacol. 3:224-238.

Durham, W.F., J.F. Armstrong, W.M. Upholt, and C. Heller. 1961. Insecticide content of diet on body fat of Alaskan natives. Science 134:1880.

Eaton, E. 1910. Birds of New York, Vol. 1. Univ. State of New York, Albany. In J. Bull, Birds of the New York Area. Dover publ., N.Y.

Eberhart, L., R. Leemeeks, and T. Peterle. 1971. Food chain model for DDT kinetics in a freshwater marsh. Nature. 230:60-62.

Eisler, R. 1986a. PCB hazards to fish, wildlife and invertebrates: A synoptic review. USFWS Biol. Rep. 86(1.7). 72pp.

Eisler, R. 1986b. Dioxin hazards to fish, wildlife, and invertebrates: A synoptic review. USFWS Biol. Rep. 86(8). 37pp.

Eisler, R. 1985. Mirex hazards to fish, wildlife and invertebrates: a synoptic review. USFWS Biol. Rep. 85(1.1). 42pp.

Ellarson, R. 1956. A study of the oldsquaw duck on L. Michigan. Univ. of Wisconsin Ph.D. Thesis. In R. Palmer (ed.), Handbook of North American Birds. Yale University Press, New Haven (paper not seen).

EPA (Environmental Protection Agency). 1985. Status of Dioxin Research in the Environmental Protection Agency: A report prepared by the office of Research and Development. August 1985. 51pp.

EPA (Environmental Protection Agency). 1980a. Guidelines and Methodology used in the preparation of health effects assessment chapters of the consent decree water quality criteria. Fed Regist. 45,79347-79357.

EPA (Environmental Protection Agency). 1980b. Ambient water quality criteria for heptachlor. EPA 440/5-80-052, USEPA, Wash., D.C.

EPA (Environmental Protection Agency). 1980c. Ambient water quality criteria for chlorinated benzenes. EPA 440/5-80-028, USEPA, Wash., D.C.

EPA (Environmental Protection Agency). 1980d. Ambient water quality criteria for chlorinated phenols. EPA 440/5-80-032, USEPA, Wash., D.C.

EPA (Environmental Protection Agency). 1980e. Ambient water quality criteria for hexachlorocyclohexane. EPA 440/5-80-054, USEPA, Wash., D.C.

EPA (Environmental Protection Agency). 1976. Quality criteria for water. EPA 440/9-76-023. USEPA, Wash., D.C. 256pp.

EPA (Environmental Protection Agency). 1975. Update of food factor tables. Memorandum of R.D. Schmitt, Ph.D. Toxicology Branch. 20pp.

EPA (Environmental Protection Agency). 1985. Status of dioxin research in the U.S. Environmental Protection Agency. Unpubl. report by Office at Research and Development for Science Advisory Board. 51pp.

Epstein, S. 1976. Carcinogenicity of heptachlor and chlordane. Sci. Total. Environ. 6:103-154.

Ernst, W., V. Weighelt, and K. Weber. 1984. Octachlorostyrene. A permanent micropollutant in North Sea. Chemosphere 13:161-168.

Erskine, A. 1972. Buffleheads. Canadian Wildlife Serv. Monograph, Ser. No. 4. 240pp. In R. Palmer (ed.), Handbook of North American Birds. Yale Univ. Press, New Haven (paper not seen).

FAO/WHO (Food Agriculture Organization/World Health Organization). 1978. Evaluations of some pesticides in food. Food and Agric. Organ. Plant Production and Protection Paper. Suppl. 10, Rome.

Fassbender, V.C., A. Alvarez, and S. Wenzel. 1977. Experimentelle Untersuchungen zur festlegung von geeigneten Probenentnahmes-tellen fur Hexachlorobenzol (HBC). Ruckstande bei Mastschweinen. Archiv fur Lebensmittelhygiene. 28:201-240.

FDA (Food and Drug Administration). 1977. Letter to Dr. Theodore Hullar, Dep. Commissioner NYS Dept. of Environmental Conservation, from Pasquale Lombardo, Dept. of Health, Education and Welfare, Wash., D.C.

Fein, G., Jacobson, S. Jacobson, P. Schwartz and J. Dowler. 1984. Prenatal exposure to polychlorinated biphenyls: Effects on birth size and gestational age. J. Pediatr. 105:315-320.

Feroz, M. and M. Khan. 1979. Metabolism of ^{14}C-heptachlor in goldfish (Carassius auratus). Arch. Environ. Contam. Toxic. 8:519-531.

Fish and Wildlife Service. 1978. Fish and Wildlife News Bulletin. October, 1978.

Fleming, W., M. McLane, and E. Cromartie. 1982. Endrin decreases screech owl productivity. J. Wildl. Manage. 46(2): 463-468.

Flickinger, E. 1979. Effects of Aldrin exposure on snow geese in Texas rice fields. J. Wildl. Manage. 43(1):94-101.

Foley, R.E. and G.R. Batcheller. In press. Organochlorine contaminants in common goldeneye wintering on the Niagara River. J. Wildlife Manage.

Ford, J., J. Hawthorne, and G. Markin. 1973. Residues of Mirex and certain other chlorinated hydrocarbon insecticides in beef fat, 1971. Pest. Monit. J. 7(2):87-94.

Fowler, J.S.L. 1969. Carbon tetrachloride metabolism in the rabbit. Br. J. Pharmacol. 37:733-737.

Fox, G. 1976. Eggshell quality: Its ecological and physiological significance in a DDE-contaminanted common tern population. Wilson Bull. 88:459-477.

Friend, M. and D. Trainer. 1970. Polychlorinated biphenyl: Interaction with duck hepatitus virus. Science 170:1314.

Fries, G. and G. Marrow. 1975. Retention and excretion of 2,3,7,8-tetrachlorodibenzo-p-dioxin by rats. J. Agric. Food Chem. 23(2):265-269.

Fry, C. 1980. The evolutionary biology of kingfishers (Alcedinidea). In The Living Bird, 1979-1980. The Laboratory of Ornithology, Cornell Univ., Ithaca. pp. 113-160.

Gaines, T. and R. Kimbrough. 1969. The oral toxicity of mirex in adult and suckling rats. Toxicol. Appl. Pharmacol. 14:631-632.

Gaines, T.B. 1960. The acute toxicity of pesticides to rats. Toxicol. Appl. Pharmacol. 2:88.

George, C. 1981. The fishes of the Adirondack Park. Department of Environmental Conservation, New York. 87pp.

Gilbertson, M. 1985a. Epidemics in Great Lakes birds and mammals caused by chemicals. In Symposium on Persistent Toxic Substances and the Health of Aquatic Communities. Minn., Minnesota. 33pp.

Gilbertson, M. 1985b. The Niagara labyrinth-the human ecology of producing organochlorine chemicals. Can. J. Fish. Aquat. Sci. 42:1681-1692.

Gilbertson, M. 1983. Etiology of chick edema disease in herring gulls in the lower Great Lakes. Chemosphere 12:357-370.

Gilbertson, M., R. Morris, and R. Hunter. 1976. Abnormal chicks and PCB residue levels in eggs of colonial birds on the lower Great Lakes (1971-73). Auk. 93:434-442.

Gilbertson, M. and G. Fox. 1977. Pollutant-associated embryonic mortality of Great Lakes herring gulls. Environ. Pollut. 12:211-216.

Gilbertson, M. 1974. Seasonal changes in organochlorine compounds and mercury in common terns of Hamilton Harbour, Ontario. Bull. Environ. Toxicol. 12(6):726-732.

Gilbertson, M. and L. Reynolds. 1972. Hexachlorobenzene (HCB) in the eggs of common terns in Hamilton Harbour, Ontario. Bull. Environ. Contam. Toxic. 7(6)371-373.

Gilman, A.P., D.B. Peakall, D.J. Hallet, G.A. Fox, and R.J. Norstrom. 1979. Herring gulls (Larus argentatus) as monitors of contamination in the Great Lakes. In Animals as Monitors of Environmental Pollutants. Natl. Academy of Sciences Press, Wash., D.C. pp. 280-289.

Gilman, A.P., G.A. Fox, D.B. Peakall, S.M. Teeple, T.R. Carroll and G.T. Haymes. 1977. Reproductive parameters and egg contaminant levels of Great Lakes herring gulls. J. Wildl. Manage. 41(3)458-468.

Grabowski, C. 1981. Pesticide effects on prenatal cardiovascular physiology-An analysis of the causes of perinatal deaths induced by prenatal exposure to mirex. EPA Project Summary. pp. 4-7.

Greer, K. 1955. Yearly food habits of the river otter in the Thompson Lake region, northwestern Montana, as indicated by scat analysis. Am. Midl. Nat. 54(2):299-313.

Grossman, M. and J. Hamlet. 1964. Birds of Prey of the World. Bonanza Books, N.Y. 495pp.

Grzenda, A., D. Paris, and W. Taylor. 1970. The uptake, metabolism, and elimination of chlorinated residues by goldfish (Carassius auratus) fed a ^{14}C-DDT contaminated diet. Trans. Am. Fish. Soc. 99(2):385-396.

Gupta, B., J. Vos., J. Moore, J. Zinkl, and B. Bullock. 1973. Pathological effects of 2,3,7,8-tetrachlorodibenzo-p-dioxin in laboratory animals. Environ. Hlth. Persp. pp. 125-140.

Hall, E. and K. Kelson. 1959. The Mammals of North America. Ronald Press Co., New York. 1083 pp.

Hammer, D. 1969. Parameters of a marsh snapping turtle population, La Creek Refuge, South Dakota. J. Wildl. Manage. 33:995-1005.

Hammond, P.B., I.C.T. Nisbet, A.F. Sarofim, N.H. Drury, N. Nelson and D.P. Rall. 1972. Polychlorinated biphenyls-Environmental impact. Environ. Res. 5:249-362.

Hansen, L.G., S.B. Dorn, S.M. Sundlof and R.S. Vogel. 1978. Toxicity, accumulation and depletion of hexachlorobenzene in laying chickens. J. Agric. Food Chem. 26(6):1369.

Harbison, R. 1975. Comparative toxicity of selected pesticides in neonatal and adult rats. Toxicol. Appl. Pharmacol. 32:443.

Harleman, J.H. and W. Seinen. 1979. Short-term toxicity and reproduction studies in rats with hexachloro-(1,3)-butadiene. Toxicol. Appl. Pharmacol. 47(1):1.

Harless, R.L., E.O. Oswald, R.G. Lewis, A.E. Dupuy, Jr., D.D. McDaniel, and H. Tai. 1982. Determination of 2,3,7,8-tetrachlorodibenzo-p-dioxin in freshwater fish. Chemosphere 11:193-198.

Harr, J., R. Claeys, J. Bone, and T. McCorcle. 1970. Dieldrin toxicosis: Rat reproduction. Am. J. Vet. Research. 31:181-187.

Harris, M., J. Moore, J. Vos, and B. Gupta. 1973. General biological effects of TCDD in laboratory animals. Environ. Hlth. Per. pp. 101-109

Harris, C. 1968. Otters: A Study of the Recent Lutrinae. Weidenfield & Nicholson, London. 397pp.

Hartsough, G. 1965. Great Lakes fish now suspect as mink food. Am. Fur Breeder 38:25.

Hascoet, M., E. LaVaur, and B. Jomard. 1978. Environment and quality of life: A study of the contamination of continental fauna by organochlorine pesticides and PCBs. Publ. by Commission of the European Communities, Luxembourg. 174 pp.

Hattula, M., V. Wasenius, R. Krees, A. Arstila, and M. Kihlstrom. 1981. Acute and short-term toxicity of 2,3,4,6-tetrachlorophenol in rats. Bull. Environ. Contam. Toxicol. 26:795-800.

Hayes, W. 1967. Toxicity of Pesticides to Man: Risks from present levels. Proc. Roy. Soc. London 167(1007): 101-127.

Hayes, W., E. Dale, and C. Pirkle. 1971. Evidence of safety of long-term, high oral doses of chlorophenothane (DDT) in man. Arch. Environ. Hlth. 22:119-135.

Haymes, G.T. and H. Blokpoel. 1978. Seasonal distribution and site tenacity of the Great Lake common tern. Bird Banding 19(2); 142-151.

Health, R., J. Spann, Hill and J. Kreitzer. 1972. Comparative dietary toxicities of pesticides to birds. USFWS Special Scientific Report pp. 475-485.

Heath, R. J. Spann, J. Kreitzer, and C. Vance. 1970. Effects of polychlorinated biphenyls on birds. Proceedings XV International Ornithological Congress, Bureau of Sport Fisheries and Wildlife. pp. 745.

Heath, R., J. Spann, and J. Kreitzer. 1969. Marked DDE impairment of mallard reproduction in controlled studies. Nature 224: 47-48.

Heinz, G. and R. Johnson. 1981. Diagnostic brain residues of dieldrin: Some new insights. Avian and Mammalian Wildlife Toxicology: Second Conference, ASTM STP 757, D. Lamb and G. Kenaga (eds.), American Society of Testing and Materials pp. 72-92.

Helwig, D. and M. Hora. 1983. Polychlorinated biphenyl, mercury, and cadmium concentrations in Minnesota snapping Turtles. Bull. Environ. Contam. Toxicol. 30:186-190.

Henny, C., L. Blus, S. Gregory, and C. Stafford. 1981. PCB's and organochlorine pesticides in wild mink and river otters from Oregon. Worldwide Furbearer Conference Proceedings, Laurel, Md. 3:1763-1780.

Henny, C. and W. VanVelzew. 1972. Migration patterns and wintering localities of American ospreys. J. Wildl. Manage. 36(1):1133-1141.

Henny, C. and H. Wright. 1969. An endangered osprey population: Estimates of mortality and production. Auk 86:156-198.

Hickey, J. and D. Anderson. 1968. Chlorinated hydrocarbons and eggshell changes in raptorial and fish-eating birds. Science 162:271-273.

Hill, E. and M. Camardese. 1986. Lethal dietary toxicities of environmental contaminants and pesticides to Coturnix. USFWS, Report #2, Dept. of Interior, Wash., D.C. 147pp.

Hill, E. and D. Dent. 1985. Mirex residues in seven groups of aquatic and terrestrial mammals. Arch. Environ. Contam. Toxicol. 14:7-12.

Hodge, H., A. Boyce, W. Deichmann, and H. Kraybill. 1967. Toxicology and no-effect levels of aldrin and dieldrin. Toxicol. Appl. Pharmacol. 10:613-675.

Hudson, R., R. Tucker, and M. Haegele. 1984. Handbook of toxicity of pesticides to wildlife. Second Edition. USFWS Resources Publication No. 153. 90pp.

Hull, ___. 1914. Wilson Bulletin 26:116-123. In R. Palmer (ed.), Handbook of North American Birds. Yale Univ. Press, New Haven (paper not seen).

Hyde, K. 1972. Studies of the responses of selected wildlife species to mirex bait exposure. Ph.D. thesis, Louisiana State University, Baton Rouge. 170pp. In R. Eisler, Mirex hazards to fish, wildlife and invertebrates: A synoptic review. U.S. Dept. of the Interior, Fish and Wildlife Service Biological Report No. 85.

IARC (International Agency for Research on Cancer). 1979. International Agency for Research on Cancer (IARC) Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. 18:43-103; Suppl. 4:112-114.

IARC (International Agency for Research on Cancer). 1974. International Agency for Research on Cancer (IARC) Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. 5:25-38; Suppl. 4:238-243.

IARC (International Agency for Research on Cancer). 1977. International Agency for Research on Cancer (IARC) Monographs on the Evaluation of the Carcinogenic risk of Chemicals to Humans. 20:187.

IJC (International Joint Commission). 1981. Report of the Aquatic Ecosystems Objectives Committee. International Joint Commission, Windsor, Ontario. 48 pp.

IJC (International Joint Commission. 1986. Literature review of the effects of persistent toxic substances on Great Lakes biota. Report of the Health of Aquatic Communities Task Force. International Joint Commission, Windsor, Ontario. 255pp.

Innes, J., B. Ulland, M. Valerio, L. Petrocelli, L. Fishbein, E. Hart, A. Pallotta, P. Bates, H. Falk, J. Gart, M. Klein, I. Mitchell, and J. Peters. 1969. Bioassay of pesticides and industrial chemicals for tumorigenicity in mice: A preliminary report. J. Nat. Cancer Inst. 42:1101-1114.

Jang, H. and F. Talamantes. Effects of chlordane on ova implantation in the mouse. Bull. Environ. Contam. Toxicol. 17(5): 559-563.

Jansson, B. and G. Sundstrom. 1978. Formation of polychlorinated dibenzo-p-dioxins during combustion of chlorophenol formulations. Science of Total Environ. 10:209-217.

Johnson, D., W. Melquist, and G. Schroeder. 1975. DDT and PCB levels in Lake Coeur d'Alene, Idaho, osprey eggs. Bull. Environ. Contam. Toxicol. 13(4): 401-404.

Johnson, J., D. Stalling, and J. Hogan. 1974. Hexachlorobenzene (HCB) residues in fish. Bull. Environ. Contam. Toxicol. 11(5): 393-398.

Johnson, R.L., P.J. Gehring, R.J. Kociba, and B.A. Schwetz. 1973. Chlorinated dibenzo-dioxins and pentachlorophenol. Environ. Hlth. Perspect. 5:171-175.

Johnston, D. 1978. Organochlorine pesticides residues in Florida birds of prey, 1969-76. Pest. Monit. J. 12(1):8-15.

Jondorf, W., D. Park, and R. Williams. 1955. Studies in detoxification: Metabolism of halogenobenzenes 1,2,3-,1,2,4-, and 1,3,5-trichlorobenzenes. Biochem. J. 61:512-521.

Jones, L. 1906. A contribution to the life history of the common and roseate terns. The Wilson Bulletin 18:35. In A. Bent, Life Histories of North American Gulls and Terns. Dover Publ. N.Y.

Kaiser, T., W. Reichel, L. Locke, E. Cromartie, J. Krynitsky, T. Mulhern, R. Prouty, C. Stafford, and D. Swineford. 1980. Organochlorine pesticide residues and necropsy data for bald eagles from 29 states, 1975-1977. Pest. Monit. J. 13(4): 145-147.

Kear, J. 1965. The internal food reserves of hatching mallard ducklings. J. Wildl. Manage. 29: 523-528.

Kendall, R.J., R. Noblet, L.H. Senn, and J.R. Holman. 1978. Toxicological studies with mirex in bobwhite quail. Poult. Sci. 57:1539-1545.

Kent, B. 1981. Prey dropped by herring gulls (Larus argentatus) on soft sediments. Auk 98:350-354.

Keith, J.A. 1966. Reproduction in a population of herring gulls (Larus argentatus) contaminated by DDT. J. Appl. Ecol. 3(suppl): 57-70.

Keplinger, M.L., W.B. Deichmann, and F. Sala. 1970. Effects of combinations of pesticides on reproduction in mice. In Pesticides Symposia. Halos and Assoc., Miami. pp. 125-138.

Khasawinah, A.M. 1982. Metabolism of chlordane/heptahclor in animals. Prepared for the Velsicol Chemical Corp., Chicago (unpubl.)

Kim, N. and D. Stone. 1981. Organic chemicals in drinking water. NYS Dept. of Health, Albany, N.Y. 140pp.

Kimbrough, R. 1984. The epidemiology and toxicology of TCDD. Bull. Environ. Contam. Toxicol. 33:636-647.

Kimbrough, R., R. Squire, J. Strandberg, R. Montali, and V. Burse. 1975. Induction of liver tumors in Sherman strain female rats by polychlorinated biphenyl Aroclor 1260. J. Nat'l Cancer Inst. 55:1453.

Kimbrough, R. and R. Linder. 1974. The toxicity of technical hexachlorobenzene in the Sherman strain rat: A preliminary study. Res. Commun. Chem. Pathol. Pharmacol. 8:653-664.

Kinoshita, F.K. and C.K. Kempf. 1970. Quantitative measurement of hepatic microsomal enzyem induction after dietary intake of chlorinated hydrocarbon insecticides. Toxicol. Appl. Pharmacol. 17:288.

Klaassen, C. and J. Doull. 1980. Evaluation of safety; toxicological evaluation. In The Basic-science of Poisons, 2nd ed. MacMillan Publ. Co., N.Y. pp. 11-27.

Koch-Weser, D., J. delaHuerga, C. Yesinich, and H. Popper. 1953. Hepatic necrosis due to bromobenzene as an example of conditioned amino-acid deficiency. Metabolism 2:248-260.

Kociba, R.J. and B. Schwetz. 1982. A review of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) with a comparison to the toxicity of other chlorinated dioxin isomers. Assoc. of Food and Drug Officials Quarterly Bulletin 46(3):168-188.

Kociba, R., D. Keyes, J. Beyer, R. Carreon, C. Wade, D. Dittenber, R. Kalnins, L. Frauson, C. Park, S. Barnard, R. Hummel, and G. Humiston, and B. Schwetz. 1978. Results of a two year chronic toxicity and oncogenicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin study in rats. Applied Pharmacol. 46:279-303.

Kociba, R.J., D. Keyes, G. Jersey, J. Ballard, A. Dittenber, J. Quast, C. Wade, C. Humiston, and B. Schwetz. 1977. Results of a two-year chronic toxicity study with hexachlorobutadiene in rats. Am. Indus. Hyg. Assoc. J. 38:589-602.

Koller, L.D. and J.G. Zinke. 1973. Pathology of polychlorinated biphenyls in rabbits. Amer. J. Pathol 70:363.

Korschgen, L. 1958. December food habits of mink in Missouri. J. Mammal. 39:521-527.

Krog, J. 1953. Notes on the birds of Amchitka Island, Alaska. The Condor 55(6):299-304.

Kuehl, D., H. Kopperman, G. Veith, and G. Glass. 1976. Isolation and identification of polychlorinated styrenes in Great Lakes. Fish. Bull. Environ. Contam. Toxicol. 16:127-132.

Kutz, F.W., A.R. Yobs, W.G. Johnson and G.B. Wiersma. 1974. Pesticide residues in adipose tissue of the general population of the U.S. FY1970 Survey. Bull Soc. Pharmac. Environ. Pathol. 2:4-10.

Lagler, K. and C. Wienert. 1948. Food of the Oldsquaw in Lake Michigan. Wilson Bull. 60:118.

Lagler, K. 1943. Food habits and economic relations of the turtles of Michigan with special reference to game management. Amer. Midl. Nat. 29:257-312.

Lagler, K. and B. Ostenson. 1942. Early spring food of the otter in Michigan. J. Wildl. Manage. 6(3):244-254.

Lambrecht, R. 1983. Renal tumors in rats (R) chronically exposed to hexachlorobenzene (HCB). Proc. Am. Assoc. Can. Res. 24:59 (Abstract).

Larson, P.S., J.L. Egle, G.R. Hennigar, and J.F. Borzelleca. 1979. Acute and subchronic toxicity of mirex in the rat, dog, rabbit. Toxicol. Appl. Pharmacol. 49:271-277.

Lehman, A.J. 1965. Summaries of pesticide toxicity. Assoc. of Food and Drug Officials of the U.S. Topeka, Kans. pp.1-40. In EPA, Quality Criteria for Water. EPA 440/9-76-023 USEPA, Wash. D.C.

Lehner, P. and Egbert, ____. 1969. Dieldrin and eggshell thickness in ducks. Nature 224:1218.

Lillie, R.J., H.C. Cecil, J. Bitman, and G.F. Fries. 1975. Toxicity of certain polychlorinated and polybrominated biphenyls on reproductive efficiency of caged chickens. Poult. Sci. 54:1550-1555.

Linscombe, G., N. Kinler, and R. Aulerich. 1982. Mink. In J. Chapman and G. Feldhamer (eds.), Wild Mammals of North America: Biology, management, and economics. John Hopkins Univ. Press, Baltimore. pp. 629-643.

Longcore, J.R. and F.B. Samson. 1973. Eggshell breakage by incubating black ducks fed DDE. J. Wildl. Manage. 37(3): 390-394.

Loose, L., A. Pittman, K. Benitz, and J. Silkworth. 1977. Polychlorinated biphenyl and hexachlorobenzene induced humoral immunosupression. J. Reticuloendothelial Society 22(3):253-271.

Loring, _____. 1890. Ornithol and Ool. 15:81-87. In R. Palmer (ed.), Handbook of North American Birds. Yale Univ. Press, New Haven (paper not seen).

Lu, P.Y., R.L. Metcalf, N. Plummer, and D. Mandel. 1977. The environmental fate of three carcinogens: benzo[a]pyrene, benzidine, and vinyl chloride evaluated in laboratory model ecosystems. Arch. Environ. Contam. Toxicol. 6:129-142.

Macek, K. and S. Korn. 1970. Significance of the food chain in DDT accumulation by fish. J. Fish. Res. Bd. Can. 27:1496-1498.

Macek, K., C. Rodgers, D. Stalling, and S. Korn. 1970. The uptake, distribution, and elimination of dietary ^{14}C-DDT and ^{14}C-dieldrin in rainbow trout. Trans. Amer. Fish. Soc. 99:689-695.

Mackay, G. 1898. The terns of Muskeget Island, Massachusetts, pt 4. Auk 15:168.

Marks, T.A., G.L. Kimmel, and R.E. Staples. 1981. Influence of symmetrical polychlorinated biphenyl isomers on embryo and fetal development in mice. I teratogenicity of 3,3',4,4',5,5'-hexachlorobiphenyl. Toxicol. Appl. Pharmacol. 61:269-276.

McAtee, _____. 1918. U.S. Department of Agriculture. Bull No. 720. In R. Palmer, (ed.), Handbook of North American Birds. Yale University Press, New Haven (paper not seen).

McConnell, E., E. Moore, J. Haseman, and M. Harris. 1978. The comparative toxicity of chlorinated dibenzo-p-dioxins in mice and guinea pigs. Toxic. Appl. Pharmacol. 44:335-356.

McNulty, W.P. 1977. Toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin for Rhesus monkey: Brief report. Bull. Environ. Contam. Toxicol. 18:108-114.

Mestitzova, M. 1967. On reproduction studies on the occurrences of cataracts in rats after long-term feeding of the insecticide heptachlor. Experienta 23:42.

Metcalf, R.L., G.M. Booth, C.K. Schuth, D.J. Hansen, and P.Y. Lu. 1973. Uptake and fate of di-2-ethylhexyl phthalate in aquatic organisms and in a model ecosystem. Environ. Hlth. Persp. 4:27-34.

Metcalf, R. and J. Sanborn. 1975. Pesticides and environmental quality in Illinois. Ill. Natur. Hist. Survey. Bull. 31:381.

Metcalf, R., I. Kapoor, P.Lu, C. Schuth, and P. Sherman. 1973. Model ecosystem studies of the environmental fate of six organochlorine pesticides. Environ. Hlth. Persp. 4:35-44.

Miranda, C. and R. Webb. 1974. Effect of diet and chemicals on pesticide toxicity in rats. Phillipp. J. Nutr. 27:30.

Mitjavila, S., G. Carrera and Y. Fernandez. 1981. Evaluation of the toxic risk of accumulated DDT in the rat: During fat mobilization. Arch. Environ. Contam. Toxicol. 10:471-481.

Mitjavila, S., G. Carrera, R. Biogegrain, and R. Derache. 1981. Evaluation of the toxic risk of accumulated DDT in the rat: During accumulation. Arch-Environ. Contam. Toxicol. 10:459-469.

Montz, W., W. Card, and R. Kirkpatrick. 1982. Effects of PCBs and nutritional restriction on barbituate-induced sleeping times and selected blood characteristics in raccoons (Procyon lotor). Bull. Environ. Contam. Toxicol. 28:578-583.

Moore, F. 1976. The dynamics of seasonal distribution of Great Lakes herring gulls. Bird banding 47(2):141-159.

Morris, R., I. Kirkham, and J. Chardine. 1980. Management of a declining common tern colony. J. Wildl. Manage. 44(1):241-245.

Muller, D., H. Klepel, R. Macholz, J. Lewerenz, and R. Engst. 1981. Electroneurophysiological studies on neurotoxic effects of hexachlorocyclohexane isomers and gamma-pentachlorocyclohexane. Bull. Environ. Contam. Toxic. 27:704-706.

Myerriecks, A. 1960. Comparative breeding behavior of four species of North American herons. Publ. Nuttal Ornithol. Club, No. 2. In J. Bull, Birds of New York State. Dover Publ., New York.

NAS (National Academy of Sciences). 1977. Scientific and technical assessments of environmental assessment. Nat. Acad. Sci., Wash. D.C. 73 pp.

NCI (National Cancer Institute). 1978a. Bioassay of Aroclor 1254 for possible carcinogenicity. Technical Report Series No. 38.

NCI (National Cancer Institute). 1978b. Bioassay of DDT, TDE, and p,p'-DDE for possible carcinogenicity. Technical Report Series No. 131. 117pp.

NCI (National Cancer Institute). 1978c. Bioassay of hexachloroethane for possible carcinogenicity. Technical Report Series No. 68. 46pp.

NCI (National Cancer Institute). 1977. Bioassay of chlordane for possible carcinogenicity. Technical Report Series No. 8.

NCI (National Cancer Institute). 1979a. Bioassay of dibenzo-p-dioxin for possible carcinogenicity. Technical Report Series No. 122. 103 pp.

NCI (National Cancer Institute). 1979b. Bioassay of endrin for possible carcinogenicity. Technical Report Series No. 12. 110pp.

Neal, R. 1985. Mechanisms of the biological effects of PCBs, polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans in experimental animals. Environ. Hlth. Persp. 60:41-46.

Neal, R. 1973. Mechanisms of the biological effects of PCB's, polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans in experimental animals. Environ. Hlth. Persp. 60:41-46.

Neff, J.M. 1979. Polycyclic aromatic hydrocarbons in the aquatic environment. Applied Sciences Publ., Ltd., Essex, England. 262. pp.

Neill, D.D. et al., 1969. 58th Annual Meeting of the Poultry Assoc. p. 130. In EPA, Quality criteria for water, U.S. Environmental Protection Agency, Wash. D.C.

Neuhold, J.M., W.T. Helm, and R.A. Valdez. 1971. Freshwater vertebrate and invertebrate ecology of Amchitka Island. Amchitka Bioenvironmental Program, Annual Progress Report, Battelle Columbus Laboratories, BMI-171-142.

Nimi, A. and C. Cho. 1981. Elimination of hexachlorobenzene (HCB) by rainbow trout (Salmo garrdneri), and an examination of its kinetics in Lake Ontario salmonids. Can. J. Fish. Aquat. Sci. 38(1): 1350-1356.

Nimi, A. 1979. Hexachlorobenzene (HCB) levels in Lake Ontario salmonids. Bull. Environ. Contam. Toxicol. 23:20-24.

NIOSH (National Institute of Occupational Safety and Health). 1978. Registry of Toxic Effects of chemical substances. R.J. Lewis and R.L. Tatken (eds.), publ. U.S. Department of Heath Education and Welfare (HEW), NIOSH, Cinncinatti, OH. DHEW (NIOSH) Publ. No. 79-100.

NIOSH (National Institute of Occupational Safety and Health). 1982. Registry of Toxic Effects of Chemical Substances. R. L. Tatken, and R.J. Lewis (eds.), publ. U.S. Dept. Health and Human Services (DHHS), NIOSH, Cincinatti, OH. DHHS (NIOSH) Publ. No. 83-107.

Norstrom, R.J., Hallett, D.J., and R.A. Stonstegard. 1978. Coho salmon (Oncorhynchus kisutch) and herring gulls (Larus argentatus) as indicators of organochlorine contamination in Lake Ontario. J. Fish. Res. Bd. Can. 35:1401-1409.

NRC (National Research Council). 1980. Drinking Water and Health, Vol. 2. Safe Drinking Water Committee, National Academy Press, Wash. D.C. 393 pp.

NRCC. 1981. Polychlorinated dibenzo-p-dioxins: Criteria for their effects on man and his environment. Nat. Res. Counc. Canada, Publ. NRCC. No. 18574, 251pp. In R. Eisler, Dioxin hazards to fish, wildlife and invertebrates: A synoptic review. USFWS Biological Report 85(1.8).

NRTC (Niagara River Toxics Committee). 1984. Report available from Inland Waters Directorate, Canada Centre for Inland Waters, Burlington, Ont. LTR. 4A6.

O'Connor, ___ and ___ Nielson. 1980. Environmental Survey of methylmercury levels in wild mink (Mustela vison) and otter (Lutra canadensis) from the northeastern United States and experimental pathology of methylmercurism in the otter. North American Fur Breeders Conference Pro. Aug. 1980. Maryland. pp. 1728-1745.

OECD (Organization for Economic Cooperation and Development). 1980. Chemical Trends in Wildlife. Publ. OECD, Paris. 245pp.

Ofstad, E., G. Lunde, K. Martinsen, and B. Rygg. 1978. Chlorinated aromatic hydrocarbons in fish from an area polluted by industrial effluents. Sci. Total Environ. 10(3):219-230.

Oliver, B. and K. Nicol. 1982. Chlorobenzenes in sediments, water, and selected fish from Lakes Superior, Huron, Erie, and Ontario. Environ. Sci. Tech. No. 1. 16(8):532-536.

Ottolenghi, A.D., J.K. Haseman, and F. Suggs. 1974. Teratogenic effects of aldrin, dieldrin, and endrin in hamsters and mice. Teratology. 9:11-16.

Palmer, R.S. (ed.). 1976. Handbook of North American Birds, Vol. 1, 2, and 3. Yale Univ. Press, New Haven. 567pp., 515 pp., and 526 pp.

Parejko, R. and C. Wu. 1977. Chlorohydrocarbons in Marquette fish hatchery lake trout (Salvelinus namaycush). Bull. Environ. Contam. Toxicol. 17:90.

Parker, K. 1985. Foraging and reproduction of the common loon (Gavia immer) on acidified lakes in the Adirondack Park, New York. M.S. thesis, S.U.N.Y. College of Environ. Sci. and Forestry, Syracuse. 144pp.

Paynter, R.A. 1974. Avian Energetics. Publ. Nuttal Ornithology Club No. 15, Museum of Comparative Zoology, Harvard University, Cambridge, MA. 334pp.

Pell, S. 1940. Notes on the food habits of the common snapping turtle. Copeia 2:31. In A. Carr, Handbook of Turtles. Cornell Univ. Press, New York.

Peterson, S. and R. Ellarson. 1978. p, p^1-DDE, polychlorinated biphenyls and endrin in oldsquaws in North America, 1969-73. Pest. Monit. J. 11:170-181.

Platonow, N. and C. Karstud. 1973. Dietary effects of polychlorinated biphenyls on mink. Can. J. Comp. Med. 37:391-400.

Platonow, N. and B. Reinhart. 1973. The effects of polychlorinated biphenyls (Aroclor 1254) on chicken egg production, fertility and hatchability. Can. J. Comp. Med. 37(4): 341-350.

Podowski, A., B. Banerjee, M. Feroz, M. Dudek, and R. Willey. 1979. Photolysis of heptachlor and cis-chlordane toxicity of their photoisomers to animals. Arch. Environ. Contam. Toxicol. 8:509-518.

Porter, R. and S. Wiemeyer. 1972. DDE at low dietary levels kills captive American Kestrals. Bull. Environ. Contam. Toxicol. 8(4):193-199.

Post, G. 1952. The effects of aldrin on birds. J. Wildl. Manage. 16(4):490-497.

Prouty, R. W. Reichel, L. Locke, A. Beliste, E. Cromartie, T. Kaiser, T. Lamont, B. Mulhern, and D. Swineford. 1977. Residues of organochlorine pesticides and polychlorinated biphenyls and autopsy data for bald eagles, 1973-1974. Pest. Monit. J. 11(3):134-137.

Radomski, J.L., W.B. Deichmann, and E.E. Clizer. 1968. Pesticide concentration in the liver, brain, and adipose tissue of terminal hospital patients. Fd. Cosmet. Toxicol. 6:209.

Rall, D. 1979. Relevance of animal experiments to humans. Environ. Hlth. Persp. 32:297-300.

Rao, K. (ed.). 1978. Pentachlorophenol. Plenum Press, New York. 122pp.

Reggiani, G. 1978. Medical problems raised by the TCDD contamination in Seveso, Italy. Arch. Toxicol. 40:161-188.

Reichel, W., R. Prouty, and M. Gay. 1977. Identification of polychlorinated styrene compounds in heron tissues by gas-liquid chromatography-mass spectometry. J. Assoc. Off. Anal. Chem. 60:60-62.

Reinart, R., D. Stewart, and H. Seagran. 1971. Effects of dressing and cooking on DDT concentrations in certain fish from Lake Michigan. J. Fish. Res. Bd. Can. 29:525-529.

Reinhart, R. 1970. Pesticide concentrations in Great Lakes fish. Pestic. Monit. J. 3:233-240.

Reuber, M. 1977. Histopathology of carcinomas of the liver in mice ingesting heptachlor or heptachlor epoxide. Expl. Cell. Biol. 45:147.

Ringer, R. 1983. Toxicology of PCBs in mink and ferrets. In F. D'Itri and M. Kamrin (eds.), PCBs: Human and Environmental Hazards. Butterworth Pub., Woburn, MA. pp. 227-240.

Roberts, J., D. Rodgers, J. Bailey, and M. Rorke. 1978. Polychlorinated biphenyls: Biological criteria for the assessment of their effects on environmental quality. Nat. Res. Council. Can. Rep. 16077. 172pp.

Robinson, J. 1967. Residues of aldrin and dieldrin in wildlife of Britain. Symposium on Science and Technology of Residual Insecticides in Food. Shell Co., Md. pp.196-223

Robinson, K., R. Kaulock, N. Chernoff, and L. Gray. 1981. Multigeneration study of 1,2,4-trichlorobenzene in rats. J. Toxicol. Environ. Hlth. 8:489-500.

Rorig, G. 1905. Studien uber die wirtschaftliche Bedeutung insektenfressenden Vogel. Arb. Biol. reichsanstait Land-und Forst wirtschaft (Biol. Abteil. Land-und Forstwirtschaft am kais Gesunahelts) 4:1-50. In Siebert, 1949, Diff. between migrant and non-migrant birds in food and water intake at various temp. and photo periods. Auk 66(2):128-153.

Rudd, R., R. Craig, and W. Williams. 1981. Trophic accumulation of DDT in a terrestrial food web. Environ. Poll. Ser. A. 25:219-228.

Ryan, J.J., P.Y. Lau, J.C. Pilon, D. Lewis, H.A. McLeod, and A. Gervais. 1984. Incidence and levels of 2,3,7,8-tetrachlorodibenzo -p-dioxin in Lake Ontario commerical fish. Environ. Sci. Technol. 18:719-721.

Safe, S. 1984. Polychlorinated biphenyls (PCBs) and polybrominated biphenyls (PBBs): Biochemistry, toxicology, and mechanisms of action. CRC Crit. Rev. Toxicol. 13:319-393.

Saha, J.G. and R.H. Burrage. 1976. J. Environ. Sci. Health Pestic., Food Contam., Agric. Wastes, 311(1):67. In A. Szokolay, A. Madaric, and J. Uhnak, Relative cumulation of -BHC in ecological and Biological System. J. Environ. Sci. Health B12(3):193-212.

Sauter, E. A. and E.E. Steele. 1972. Poultry Science. 51:71. In S. Tullet, G. Ravie, and P.E. Lake, Possible effects of gamma benzene hexachloride on developing turkey and chicken embryos. Veterinary Record 110:499-500.

Schafer, E. Jr., W. Bowles, and J. Hulbert. 1983. The acute oral toxicity, repellancy and hazard potential of 998 chemicals to one or more species of wild and domestic birds. Arch. Environ. Contam. Toxicol. 12:355-382.

Schwetz, B.A., J.E. Quast, P.A. Keeler, C.G. Humiston, and R.J. Kociba. 1977. Chronic toxicity and reproduction studies in rats given pentachlorphenol by the dietary route. Toxicol. Appl. Pharmac. 40:138.

Scrudato, R. and A. Delprete. 1982. Lake Ontario sediment mirex relationships. J. Great Lake Res. 8:695-699.

Sealander, J. 1943. Notes on some parasites of the mink in sourthern Michigan. J. Parisitol. 29:361-362.

Seibert, H. 1949. Differences between migrant and non-migrant birds in food and water intake at various temperatures and photoperiods. Auk 66(2): 128-153.

Shannon, V.C. 1976. The effects of mirex on the reproductive performance and behavioral development of the prairie vole Micropterus ochrogaster, Ph.D. Thesis, Iowa State Univ., Ames. 318pp. In R. Eisler, Mirex hazards to fish, wildlife and invertebrates: A synoptic review. U.S. Dept. of Interior, Fish and Wildlife Service Biological Report No. 85.

Shea, T., J. Flemming, and E. Cromartie. 1980. DDT contamination at Wheeler National Wildlife Refuge. Science 209:_____.

Sherrod, S.K., C.M. White, and F. S.L. Williamson. 1980. Biology of the bald eagle on Amchitka Island, Alaska. The Living Bird. Cornell Univ. Press.

Sidell, N., G.V. RamaRao, W.A.F. Tompkins, and L.G. Hansen. 1979. Phagocytic and Fc receptor functions of alveolar macrophages in cats following ingestion of hexachlorobenzene. Vet. Hum. Toxicol. 21(4):254-257.

Sileo, L., L. Karstad, R. Frank, M. Holdrinet, E. Addison, and H. Brown. 1977. Organochlorine poisoning of ring billed gulls in southern Ontario. J. Wildl. Diseases. 13:313-322.

Sloan, R. and E. Horn (eds.). 1986. Contaminants in Hudson River Striped Bass: 1978-1985. DEC Technical Report 86-2, Bureau of Environmental Protection, Div. of Fish and Wildlife. New York.

Smith, C.C., S.T. Cragg, and G.F. Wolf. 1978. Subacute toxicity of 1,2,4-trichlorobenzene (TCB) in subhuman primates. Fed. Proc 37:248. In Robinson et al., Multigeneration study of 1,2,4-trichlorobenzene in rats. J. Toxicol. Environ. Hlth. 8:489.

Spencer, F. 1982. An assessment of the reproductive potential of Aroclor 1254 in female Sprague-Dawley rats. Bull. Environ. Contam. Toxicol. 28:290-297.

Sprunt, A. 1955. North American Birds of Prey. Harper Bros, Publ. New York. 227pp.

Stickel, L.E., N. Chura, D. Stewart, C. Menzie, R. Prouty, and W. Reichel. 1966. Bald eagle-pesticide relations. N. Am. Wildlife Conf. Wildlife Management Institute, Wash., D.C. 31:190-200.

Stickel, W., L. Stickel, R. Dryland, and D. Hughes. 1984. Aroclor 1254 residues on birds: Lethal levels and loss rates. Arch. Environ. Contam. Toxicol. 13:7-13.

Stickel, W., W. Reichel, and D. Hughes. 1979. Endrin in birds: Lethal residues and secondary poisoning. In W.B. Deichman, Org., Toxicology and Occupational Medicine. Elsevier, N.Y. pp. 397-406.

Stickel, W., L.E. Stickel, and J. Spann. 1969. Tissue residues of dieldrin in relation to mortality in birds and mammals In N. Miller and G. Berg (eds.), Chemical Fallout. Current research on persistent pesticides. Chas. C. Thomas, Springfield, Ill. pp. 174-204.

Stokinger, H.E. 1972. Concepts of thresholds in standard setting. Arch. Environ. Hlth. 25:153-157.

Stolzenburg, T. and J. Sullivan. 1983. Dioxin: a cause for concern? Publ. WIS-SG-83-141. Univ. of Wisconsin, Madison. 21pp.

Stone, W.B., E. Kiviat, and S. Butkas. 1980. Toxicants in snapping turtles. N.Y. Fish and Game J. 27:39-50.

Stott, R.S. and D.P. Olson. 1973. Food-habitat relationship of sea ducks on the New Hampshire coastline. Ecology 54(5): 996-1007.

Strik, J. and J. Koeman. 1975. Prophyrinogenic action of hexachlorobenzene and octachlorostyrene. In M. Doss and M.A.D. Lahn (eds.), Porphyrin in Human Disease. Basel, Switzerland. pp. 418-423.

Sundalof, S.F., L.G. Hansen and M.E. Welborn. 1976. Pharmacokinetics of hexachlorobenzene in male laboratory beagles. Pharmacologist 18:149.

Suns, K., G. Crawford, D. Russel, and R. Clement. 1985. Temporal trends and spatial distribution of organochlorine and mercury residues in Great Lakes spottail shiners (1975-1983). Ontario, MOE. 24pp.

Talcott, P. and L. Koller. 1983. The effect of inorganic lead and/or a polychlorinated biphenyl on the developing immune system of mice. J. Toxicol. Environ. Hlth. 12:337-352.

Tarkpea, M., I. Hagen, G.E. Carlberg, P. Kolsaker, and H. Storflor. 1985. Mutagenicity, acute toxicity, and bioaccumulation potential of six chlorinated styrenes. Bull Environ. Contam. Toxicol. 35:525-530.

Tashiro, S. and F. Matsumura. 1977. Metabolic routes of cis- and trans-chlordane in rats. J. Agric. Food Chem. 25:872.

Thorpe, E. and A. Walker. 1973. The toxicology of dieldrin (HEOD). II. In Comparative long-term oral toxicity studies in mice with dieldrin, DDT, phenoaarbitone, - BHC and BHC. Food Cosmet. Toxicol. 11:433-442.

Tinbergen, N. 1960. The Herring Gull's World. Basic Books, Inc., New York. 255 pp.

Toweill, D. 1974. Winter food habits of river otters in western Oregon. J. Wildl. Manage. 38(1):107-111.

Toweill, D.E. and J.E. Tabor. 1982. River otter. In J. Champman and G. Feldhammer (eds.), Wild mammals of North America. John Hopkins Univ. Press, Baltimore. pp. 688-703.

Treon, J., F. Cleveland, and J. Cappel. 1955. The toxicity of hexachlorocyclopentadiene. A.M.A. Archives of Industrial Health. 11:459-472.

Tugarinova, N.I., V.E. Miklaskevskii, N.P. Alekseeva, and G.P. Yakovleva. 1960. Experimental determination of limits of allowable tetrachloroethane (TCLE) and hexachlorethane (HCLE) concentration in water. USSR literature on water supply and pollution control. 5:145-154.

Ulland, B., N. Page, R. Squire, E. Weisberger, and R. Cypher. 1977. A carcinogenic assay of mirex in Charles River CD rats. J. Nat. Cancer Inst. 58:133-140.

Veith, G., D. Defoe, and B. Bergstedt. 1979. Measuring and estimating the bioconcentration factor of chemicals in fish. J. Fish. Res. Bd. Canada 36:1040-1048.

Veith, G., D. Kuehl, E. Leonard, F. Puglisi, and A. Lemke. 1979. Polychlorinated biphenyls and other organic chemical residues in fish from major watersheds of the United States, 1976. Pest. Monit. J. 13(1):1-20.

Vernineer, K., R. W. Risenbrough, A.L. Spaans, and L.M. Reynolds. 1974. Pesticides effects on fishes and birds in rice fields of Surinam, South America. Environ. Poll. 7(3):217-236.

Verschueren, K. 1983. Handbook of Environmental Data on Organic Chemicals. Van Norstrand Reinhold Publ. 1310pp.

Villeneuve, D.C., W. Phillips, L. Panopio, C. Mendoza, G. Hatina, and D. Grant. 1974. The effects of phenobarbital and carbon tetrachloride on the rate of decline of body burdens of hexachlorobenzene in the rat. Arch. Environ. Contam. Toxicol. 2:243-252.

Villeneuve, D.C., M.J. vanLoglen, E.M. denTonkelaar, P.A. Greve, J.G. Vos, G.J.A. Speijers, and G.J. vanEsh. 1977. Combined effect of food deprivation and hexachlorobenzene feeding in rats. Toxicol. Appl. Pharmac. 40:202.

Vos, J., L. VanderMaas, A. Musch, and E. Ram. 1971. Toxicity of hexachlorobenzene in Japanese quail with special reference to porphyria, liver damage, reproduction and tissue residues. Toxicol. Appl. Pharmacol. 18:944-957.

Wagstaff, D., J. McDowell, and H. Paulin. 1980. Heptachlor residue accumulation and depletion in broiler chickens. Am. J. of Vet. Res. 40(5): 765-768.

Waller, D. 1962. Feeding behavior of minks at some Iowa marshes. M.S. thesis. Iowa State Univ. 90 pp.

Walker, A., E. Thorpe, D.E. Stevenson, J. Robinson, and M. Roberts. 1972. The toxicity of dieldrin (HEOD). I. Longterm toxicity studies in mice. Food Cosmet. Toxicol. 11:415.

Warren, B. 1890. Report on the birds of Pennsylvania. In A. Bent, Life Hist. of N.A. Diving Birds. Dover Publ., N.Y.

Weil, C. and D. McCollister. 1963. Relationship between short and long-term feeding studies in designing an effective toxicology test. Agric. Food Chem. 11:486-491.

White, H. 1957. Fisheries Res. Bd. Can., Bull. No. 116. In R. Palmer (ed.), Handbook of North American Birds. Yale University Press, New Haven.

White, H. 1936. The food of Kingfishers and mergansers on the Margaree River, Nova Scotia. J. Biol. Bd. Can. 2:299-309. In R. Palmer (ed.), Handbook of North American Birds. Yale Univ. Press, New Haven.

Whitehead, C., A. Downing, and R. Pettigrew. 1972. The effect of lindane on laying hens. Br. Poult. Sci. 13:291-299.

Whittle, D. and J. Fitzsimons. 1983. The influence of the Niagara River on contaminant burdens of the Lake Ontario biota. J. Great Lakes Res. 9(2):295-302.

Whittow, G. and H. Rahn. 1984. Seabird Energetics. Plenium Press, N.Y. 328pp.

Wiemeyer, S., R. Jurek, and J. Moore. 1986. Environmental Contaminants in surrogates, foods, and feathers of California condors (Gymnogyps californianus). Environ. Monit. and Assess. 6:91-111.

Wiemeyer, S.N., T.G. LaMont, C.M. Bunck, C.R. Sindelar, F.J. Gramlich, J.D. Faser, and M.A. Byrd. 1984. Organochlorine pesticide, polychlorobiphenyl and mercury residues in bald eagle eggs, 1909-79, and their relationships to shell thinning and reproduction. Arch. Environ. Contam. Toxicol. 13:529-549.

Wiemeyer, S.N., D.M. Swineford, P.R. Spitzer, and P.D. McLain. 1978. Organochlorine residues in New Jersey Osprey eggs. Bull. Environ. Contam. Toxicol. 19:56-63.

Wiemeyer, S.N., P.R. Spitzer, W.C. Krantz, T.G. Lamont, and E. Cromartie. 1975. Effects of Environmental pollutants on Connecticut and Maryland Ospreys. J. Wildl. Manage. 39:124-139.

Windholz, M. (ed.). 1983. The Merck Index. Merck and Co., Rahway, N.J. 1461pp.

Woodwell, G., C. Wurster, and P. Issacson. 1967. DDT residues in an east coast estuary: A case of biological concentration of a persistent insecticide. Science 156:821-824.

Wright, A.S., C. Donninger, R.D. Greenland, K.L. Stemmer, and M.R. Zavon. 1978. The effects of prolonged ingestion of dieldrin on the livers of male Rhesus monkeys. Ecotox. Environ. Safety. 1:477-502.

Yang, R., W. Mueller, H. Grace, L. Golberg, and F. Couston. 1976. Hexachlorobenzene contamination in laboratory monkey chow. J. Agric. and Food Chem. 24(3):563-565.

Zepp, R. and R. Kirkpatrick. 1976. Reproduction in cottontails fed diets containing a PCB. J. Wildl. Manage. 40(3):491-495.

TABLE 1. Laboratory animal live weights and food consumption for toxic dose calculations*.

Species	Age At Testing	Consumption Weight	Food gm/day
Cat, adult		2 kg	100
Cattle, horse		500 kg	10,000
Chicken, adult (male/female)	8W (weeks)	800 gm	140
Dog, adult	52W	10 kg	250
Domestic goat/sheep		60 kg	2,500
Duck, adult (domestic)	8W	2,500 gm	250
Frog, adult		33 gm	
Gerbil		100 gm	5
Guinea pig, adult		500 gm	30
Hamster	14W	125 gm	15
Human	Adult	70 kg	
Monkey	2.5Y (years)	5 kg	400
Mouse	8W	32 gm	5
Pig		60 kg	2,400
Pigeon	8W	500 gm	
Quail (laboratory)		100 gm	
Rabbit, adult	12W	2 kg	100
Rat, adult female	14W	200 gm	10
Rat, adult male	14W	250 gm	15
Rat, adult, sex unspecified	14W	200 gm	15
Rat, weanling	3W	50 gm	15
Turkey	18W	5 kg	

*[1] Source - NIOSH RTECS 1982

TABLE 2. Feeding habits and live weights for toxic dose calculations for Niagara River wildlife.

Species	Age to Reproduction	Body Weight kg	Food kg/day	% Fish in diet	Comments on Overall Diet
Reptile					
Snapping Turtle	?	9.0	0.9	50%	60% animal food
Mammals					
Raccoon	2 years	6.5	0.5	5% fish	15% crustacean food 40% animal food
Mink	1 year	1.0	0.015	31% fish	mostly aquatic food
Northern River Otter	2 years	6.35	0.8	90% fish	mostly aquatic food
Birds					
Common Goldeneye	2 years	1.0	0.2	10% fish	Juveniles consume more animal matter
Mallard	1	1.25	0.25	5% fish	90% plant matter
Old Squaw	2	0.83	0.19	20% fish	88% animal 12% plant matter
Bufflehead	2	0.45	0.09	20% fish	80% animal matter
Common Merganser	2	1.5	0.3	95% fish	obligatory piscivores
Redbreasted Merganser	2	1.15	0.235	95% fish	obligatory piscivores
Herring Gull	2	1.0	0.2	50% fish	opportunistic feeders
Ringbilled Gull	2	0.46	0.095	50% fish	opportunistic feeders
Common Tern	2	0.14	0.03	80% fish	mostly aquatic food
Belted Kingfisher	1	0.15	0.075	95% fish	
Common Loon	2	4.5	1.5	80% fish	remainder mostly aquatic
Green-backed Heron	1	0.25	0.05	50% fish	remainder mostly aquatic
Great Blue Heron	2	3.0	0.6	85% fish	almost all aquatic food
Bald Eagle	4.5	4.5	0.9	65% fish	may consume quite a few shore birds
Osprey	3	1.5	0.3	100%	obligatory piscivores

*The sources for all information summarized in this table are presented in Section 3.1 of th report.

Table 3. Taken from NRTC 1984.

TABLE C.26

CONTAMINANT CONCENTRATIONS IN 1981 YOUNG-OF-THE-YEAR
SPOTTAIL SHINERS (Notropis hudsonius) FROM LAKE ERIE AND THE NIAGARA RIVER
(ng/g)

PARAMETER	Detection Limit	RIVER SEGMENT/SUB-AREA: Fort Erie M-6 (5)	Chippawa M-21 (7)	Wheatfield - Upper River M-11 (7)	M-12 (4)	Lower River M-28 (5)	M-29 (6)	M-32 (7)	M-36 (7)
PCBs, Total	20	164 ± 56	124 ± 14	327 ± 53	573 ± 84	405 ± 87	329 ± 120	309 ± 90	327 ± 62
BHC (α, β, γ)	1	1 ± 1		31 ± 11	34 ± 9	9 ± 3	4 ± 3	Tr	
Chlordane (α, γ)	2	Tr		11 ∓ 3	18 ∓ 4	ND	47 ∓ 20	10 ± 14	11 ± 4
Total DDT & Metabolites	5	37 ± 19	30 ± 5	9 ∓ 4	23 ∓ 4	74 ± 17	107 ∓ 57	189 ∓ 62	73 ∓ 15
Dieldrin	2	ND		ND	ND	ND	Tr	ND	Tr
Endrin	1	Tr		ND	ND	6 ± 4	7 ± 3	Tr	6 ± 11
Heptachlor	1	ND	ND	ND	ND	ND	ND	ND	ND
Heptachlor epoxide	1	Tr		ND	ND	ND	Tr	Tr	Tr
Mirex	5	ND	ND	17 ± 6	18 ± 4	12 ± 3	15 ± 8	6 ± 3	10 ± 2
						(3)		(3)	(3)
Trichlorobenzenes	1	-	-	-	-	3 ± 6	-	3 ± 6	ND
Tetrachlorobenzenes	1	-	-	-	-	11 ∓ 1	-	5 ∓ 1	4 ± 1
Pentachlorobenzene	1	-	-	-	-	7 ∓ 0	-	5 ∓ 1	7 ∓ 7
Hexachlorobenzene	1	-	-	-	-	7 ∓ 2	-	6 ∓ 1	5 ∓ 2
Hexachloroethane	1	-	-	-	-	ND	-	ND	ND
Octachlorostyrene	1	-	-	-	-	3 ± 1	-	3 ± 1	3 ± 0
2,4,6-Trichlorophenol		-	-	-	-	-	-	-	-
2,3,5-Trichlorophenol		-	-	-	-	-	-	-	-
2,3,4,6-Tetrachlorophenol		-	-	-	-	-	-	-	-
Pentachlorophenol	5	-	-	-	-	-	-	-	-
		(1)		(2)	(2)			(2)	(2)
2,3,7,8-TCDD	0.001	0.015	-	0.008	0.059	-	-	0.007	0.014

NOTES: Data source: Sub-project 30 (MOE) and Suns et al. (1983). Stations correspond to locations in Fig. 4.5 (Chapter IV)
Concentrations are mean and standard deviation in ppb (ng/g, wet weight) of number of composite samples indicated at tops of columns in brackets. Each composite sample was composed of 10 fish (PCBs and pesticides), 15 fish (chlorinated aromatics) or 20 fish (2,3,7,8-TCDD).
A dash (-) indicates no data available.
ND = Not detected at detection limit indicated; Tr = Trace (mean below detection limit).

Table 4. Taken from NRTC 1984.

TABLE C.27

CONTAMINANT CONCENTRATIONS IN 1982 YOUNG-OF-THE-YEAR SPOTTAIL SHINERS (Notropis hudsonius) FROM LAKE ERIE AND THE NIAGARA RIVER (ng/g)

			RIVER SEGMENT									
	DETECTION LIMIT		FORT ERIE			CHIPPAWA	LAKE ERIE		BLACK ROCK CANAL		BIRD ISLAND - RIVERSIDE	
PARAMETER	MOE	NYSDEC	N-1	N-5	N-6	N-21	N-1	N-2	N-4	N-5	N-6	N-7
			(7)	(6)	(7)	(7)	(5)	(5)	(5)	(5)	(5)	(4)
PCBs, Total	20		60±17	181±69	216±34	124±14	83±19	40±6	673±	1683±477	646±173	93±14
BHC (α, β, γ)	1	2-6	ND	4±1	6±1	ND	ND	ND	ND	ND	ND	ND
Chlordane (α, γ)	2	1-8	ND	6±4	8±6	10±2	ND	ND	ND	17±4	Tr	ND
Total DDT & Metabolites	5		19±8	31±12	57±10	30±5	13±4	13±4	30±8	112±28	40±7	12±
Dieldrin	1		Tr	2±1	4±3	5±2	Tr	Tr	4±1	9±	3±2	3±0.4
Endrin	1	1-2	-	-	-	-	Tr	ND	ND	ND	ND	ND
Heptachlor	1	1-2	ND	ND	ND	ND	ND	ND	ND	Tr	ND	ND
Heptachlor expoxide	1	1-2	ND	ND	1±0	ND	ND	ND	ND	2±0.4	ND	ND
Mirex	5	1-5	ND	ND	ND	ND	ND	ND	ND	ND	Tr	ND
							(5)	(5)	(5)	(5)	(5)	(4)
ΣTrichlorobenzenes	1	3-6	ND	ND	-	25±6	Tr	ND	ND	ND	13±	ND
ΣTetrachlorobenzenes		-	ND	Tr	-	ND	-	-	-	-	-	-
Pentachlorobenzene		-	ND	ND	-	ND	-	-	-	-	-	-
Hexachlorobenzene	1	2-8	Tr	Tr	-	1±0	ND	ND	Tr	2±0.6	2±	ND
Hexachloroethane		-	ND	ND	-	ND	-	-	-	-	-	-
Hexachlorobutadiene	1	1-2	ND	ND	-	ND	ND	ND	ND	ND	ND	ND
Octachlorostyrene	1	1-2	ND	ND	ND	ND	ND	ND	ND	10±	8±	ND
							(2)	(2)	(2)	(2)	(2)	(2)
2,4,6-Trichlorophenol			ND	ND	-	ND	3	2	5	7	2	11
2,3,5-Trichlorophenol		1	ND	ND	-	ND	ND	ND	ND	ND	ND	ND
2,3,4,6-Tetrachlorophenol		1	-	-	-	-	ND	ND	ND	ND	4	ND
Pentachlorophenol	5		33±11	ND	-	17±20	8	9	4	5	18	5
				(1)	(1)	(1)				(1)	(1)	(1)
2,3,7,8-TCDD	0.001		-	ND	ND	ND	-	-	-	ND	0.001	ND

NOTES: Data Sources: Sub-projects 30 (MOE) and 4 (NYSDEC). MOE stations prefixed by "M"; NYSDEC stations prefixed by "N" (see Fig. 4.5 for locations). (Chapter IV)
Concentrations are mean and standard deviation in ppb (ng/g, wet weight) of number of composite samples indicated at tops of columns in brackets.
A dash (-) indicates no data available.
ND = Not detected at applicable detection limit (MOE or NYSDEC); Tr = Trace (calculated mean below detection limit).
Rock bass substituted for spottail shiners at station N-15 in lower river.

Table 4. Continued.

TABLE C.27 (Continued)

PARAMETER	RIVER SEGMENT/SUB-AREA																		
	TONAWANDA-NORTH TONAWANDA							WHEATFIELD-UPPER RIVER			LOWER RIVER								
	N-8	N-9	N-10	N-11	N-12	N-13	N-14	N-11	N-12	N-16	N-22	N-23	N-15	N-28	N-29	N-32	N-36		
	(5)	(5)	(5)	(5)	(3)	(5)	(5)	(6)	(6)	(5)	(5)	(5)	(1)	(5)	(5)	(6)	(5)		
PCBs, Total	331±89	457±345	560±170	918±101	458±	426±	394±64	512±143	880±136	1091±351	96±5	187±45	500±	180±45	245±21	260±56	255±24		
BHCs (α,β,γ)	ND	ND	ND	ND	ND	ND	ND	28±11	29±10	7±3	Tr	Tr	ND	ND	3±1	3±1	4±1		
Chlordane (α , γ)	ND	ND	ND	17±5	ND	ND	ND	15±6	19±7	13±6	7±3	10±5	48±	8±2	8±2	7±3	17±7		
DDT & Metabolities	Tr	Tr	Tr	84±7	34±	65±	23±4	18±6	50±4	14±7	36±12	19±15	91±	26±10	61±19	47±22	62±14		
Dieldrin	8±10	Tr	3±1	8±1	4±	3±	2±1	ND	4±2	2±2	4±2	ND	5±	4±3	Tr	2±1	-		
Endrin	ND	ND	ND	ND	ND	ND	ND	-	-	-	-	-	ND	-	-	-	-		
Heptachlor	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND		
heptachlor epoxide	ND	ND	ND	2±0.2	ND	ND	ND	ND	ND	ND	2±3	ND	ND	3±2	ND	ND	-		
Mirex	ND	ND	ND	ND	ND	3±	ND	ND	6±2	5±3	ND	ND	7±	6±4	7±6	6±2	6±2		
	(5)	(5)	(5)	(5)	(3)	(5)	(5)	( )	( )	( )	( )	( )	(1)	( )		( )	( )		
Trichlorobenzenes	ND	ND	ND	ND	Tr	428±	ND	9±1	-	ND	-	50±1	86±	ND	-	ND	2±1		
Tetrachlorobenzenes	-	-	-	-	-	-	-	4±3	-	Tr	-	ND	-	3±3	-	3±4	ND		
Pentachlorobenzene	-	-	-	-	-	-	-	ND	-	ND	-	ND	-	ND	-	ND	ND		
Hexachlorobenzene	2±0.4	Tr	2±	8±5	3±	261±	4±1	8±3	-	6±4	-	1±0	31±	3±1	-	3±1	4±1		
Hexachloroethane	-	-	-	-	-	-	-	ND	-	ND	-	ND	-	ND	-	4±4	ND		
Hexachlorobutadiene	ND	ND	ND	ND	ND	14±	ND	Tr	-	7±3	-	Tr	29±	2±2	-	ND	2±2		
Octachlorostyrene	18±17	ND	ND	8±	Tr	94±	6±3	ND	-	6±3	-	Tr	536±	1±1	-	2±1	4±1		
	(2)	(2)	(2)	(2)	(2)	(2)	(2)						(2)						
2,4,6-Trichlorophenol	11	2	4	2	14	52	9	ND	ND	ND	-	ND	20	ND	ND	ND	ND		
2,3,5-Trichlorophenol	ND	ND	ND	ND	ND	5	ND	ND	ND	ND	-	ND	5	ND	ND	ND	ND		
2,3,4,6-Tetra-chlorophenol	ND	1	2	2	ND	ND	ND	-	-	-	-	-	7	-	-	-	-		
Pentachlorophenol	6	8	10	14	7	12	7	70±35	23±15	43±6	-	42±29	4	45±23	25±23	Tr	20±11		
	(1)	(1)		(1)	(1)	(1)	(1)			(1)									
2,3,7,8-TCDD	ND	ND	-	ND	ND	0.120	ND	-	-	ND	-	-	-	-	-	-	-		

Table 5. Residues found in New York State fish taken from the Niagara River, Salmon River area of Lake Ontario, exerpted from FDA (1977).

	Niagara River White Bass (ppm)	Salmon River Coho Salmon (ppm)	Niagara River Smallmouth Bass (ppm)	Niagara River Bass (ppm)	Niagara River White Bass (ppm)	Niagara River Yellow Perch (ppm)
Hexachlorobutadiene	0.03	0.08	0.04	0.05	0.03	+
Trichlorobenzenes	0.38	0.23	0.68	1.0	0.49	*
Tetrachlorobenzenes	0.59	0.12	0.60	0.82	0.52	*
Pentachlorobenzene	0.10	0.03	0.18	0.40	0.12	*
Hexachlorobenzene	0.35	0.11	0.24	0.95	0.14	*
Pentachloroanisole	0.05	0.05	0.05	0.05	0.05	*
-BHC	0.15	0.08	0.06	0.43	0.16	*
Mirex	0.51	0.18	-	-	-	-
PCBs	18	5.2	-	-	-	-
Octachlorostyrene	0.3	0.15	-	-	-	-

* No analysis.
+ Present but not quantitated.

Table 6. Taken from Norstrom et al. 1978.

TABLE 3. Levels of organochlorine compounds in coho salmon muscle, liver, and pooled salmon stomach contents (alewives and smelt) collected from western Lake Ontario in 1976. Relative standard deviations (%) are in parentheses.

Species and tissue	No. in sample	Lipid (%)	mg/kg wet wt									
			PCBs[a]	*p,p'*-DDE	Mirex	Photomirex[b]	HCB	β-HCH	Oxychlordane	Heptachlor epoxide	Dieldrin	*p,p'*-DDD
Alewives and smelt[c]	50	2.34	2.21	0.47	0.09	0.03	0.024	0.002	0.010	0.003	0.029	0.047
Coho salmon muscle[d]	28	8.17 (55)	5.77 (47)	0.97 (45)	0.23 (42)	0.11 (41)	0.097 (39)	0.012 (72)	0.016 (54)	0.015 (56)	0.087 (44)	0.110 (38)
Coho salmon liver[d]	28	6.16 (32)	2.31 (39)	0.41 (44)	0.10 (56)	0.04 (55)	0.065 (34)	0.010 (70)	0.013 (50)	0.007 (42)	0.060 (36)	0.075 (43)

[a]Calculated as 1/1 Aroclor® 1254/1260.
[b]8-monohydromirex.
[c]Duplicate analysis of pooled sample.
[d]Mean of individual analyses.

Table 7. Contaminants in Fish from Lake Ontario in 1985 and the Niagara River in 1984, ppm (mg/kg) on a wet weight basis*.

	Average in Lake Ontario Salmonids, 1985					Average or range of averages for species at 4 sites in the Niagara River, 1984					
	Lake trout 7+	Lake trout 10+	Rainbow trout	Brown trout Spring/Fall	Coho salmon	Smallmouth bass	Rockbass	Yellow perch	Carp	Brown bullhead	American eel
PCB	4.91	9.31	1.93	1.14/1.62	1.74	1.76- 3.16	0.3 - 1.41	0.18 - 0.6	1.92- 2.52	2.10	5.29
∑DDT	1.32	2.77	0.46	0.38/0.73	0.70	0.13- 0.38	0.05- 0.12	0.02- 0.07	0.16- 0.27	0.07	0.81
Mirex	0.39	0.58	0.13	0.09/0.12	0.13	<0.01- 0.07	<0.01- 0.03	<0.01- 0.02	<0.01-<0.01	<0.01	0.17
Photomirex	0.032	0.053	0.037	0.025/0.036	0.035	-	-	-	-	-	-
∑Chlordane	0.32	0.52	0.09	0.09/0.12	0.08	0.05- 0.09	0.02- 0.04	0.01- 0.02	0.14- 0.18	0.05	0.63
Dieldrin	0.08	0.14	0.04	0.044/0.045	0.008	0.01- 0.02	<0.01-<0.01	<0.01-<0.01	0.01- 0.01	0.01	0.08
Hexachlorobenzene	-	-	0.10	0.02/0.015	0.005	<0.01-<0.01	<0.01-<0.01	<0.01-<0.01	<0.01-<0.01	<0.01	<0.01
Endrin	-	-	-	-	-	<0.01-<0.01	<0.01-<0.01	<0.01-<0.01	<0.01-<0.01	<0.01	<0.01
Lindane (Hexachlorocyclohexane)	-	-	-	-	-	<0.01- 0.06	<0.01- 0.01	0.01-<0.01	0.01- 0.05	0.01	0.05
Heptachlor epoxide	-	-	-	-	-	<0.01-<0.01	<0.01-<0.01	<0.01- 0.01	<0.01- 0.01	<0.01	<0.01

*Data excerpted from DEC (in prep.)

Dietary effect levels of PCB s in animals.

Species, Sex, Age	Dose Level in mg/kg/day+ (mg/kg dietary conc. in food) NOEL	LOEL	Exposure Period	Toxicity End Point	Reference	Comments
Mink-1	0.24 (1.0)	0.48 (2.0)	8 months	Nearly complete reproduction failure	Aulerich and Ringer 1977	Found 10 ppm in Great Lakes Salmon
Mink-2	1.0 (4*)	2.0 (8*)	16 weeks	Loss of offspring	Ringer 1983	1254 mixed in food
Mink-3	-	0.375 (1.5)	16 weeks	Reproduction impaired	Aulerich et al. 1985	1254 mixed in food
Mink-4	0.1	0.225(1.0)	16 weeks	Reproduction impaired	Ringer 1983	Great Lakes Fish Contaminated with 1254
Mink-5		0.096(0.64)	16 weeks	Reproductive failure	Platonow and Karstad 1973	1254 mixed w/food
European Ferret		4.8 (20)	16 weeks	Complete reproductive failure	Bleavins et al. 1984	Dietary conc. was 20 ppm
Otter, wild				Have declined in Columbia River	Henny et al. 1980	Field study without feeding trial
Raccoon		3.85* (50)		Voluntary food restriction, loss in weight gain	Montz et al. 1982	
Rabbits	1.0	12.5	28 days of gestation	Embryotoxicity	Koller and Zinkl 1973	
Cottontail Rabbit	(1.0)		12 weeks	None	Zepp and Kirkpatrick 1976	Cottontails taken off PCB diet before breeding.
Mouse-1		2.0 (10)	28 days	Some mortality & deformed offspring	Mark et al. 1981	
Mouse-2	3.0	5.0	30 days	mortality & reproductive effects	Talcott and Koller 1983	Swiss strain PCB resistant

Table 11-2. Continued.

Species, Sex, Age	Dose Level in mg/kg/day+ (mg/kg dietary conc. in food) NOEL	LOEL	Exposure Period	Toxicity End Point	Reference	Comments
Rat-1			Single dose	188 mg/kg LDLO	NIOSH 1982	Aroclor 1254
Rat-2		1.0	Longterm	Tumorigenic agent	NIOSH 1982	Aroclor 1254
Rat-3	6.25 (100)	28.0	2 years	Stomach lesions, cancer in LOEL	Bio-Test Laboratories 1970	Chronic test
Rat-4		3.14 (50)	9 days during pregnancy	Fetal survival potential	Spencer 1982	Aroclor 1254
Chicken, white leghorn	0.224 (2)	2.24 (20)	9 weeks	Reproduction loss at LOEL	Poult. Sci. 53(2):726-32, 1974	Aroclor 1248
Mallard			5 days	LD50 = 3182 mg/kg	Heath et al. 1972	Aroclor 1242
Mallard			5 days	LD50 = 2699 mg/kg	Heath et al. 1972	Aroclor 1254
Mallard			5 days	LD50 = 1975 mg/kg	Heath et at. 1972	Aroclor 1260
Pheasant			5 days	LD50 = 1091 mg/kg	Heath et al. 1972	Aroclor 1254
Mallard	7.8 (25)		10 days		Heath et al. 1972	Mortality not significantly different than control
Mallard	7.8 (25)		2 years	Higher mortality to duck hepatitus virus	Friend & Trainer 1970	
Bobwhite quail	0.1 (3.2*)		10 weeks	None	Heath et al. 1972	
European starling			4 days	LD50 = 150 mg/kg	Stickel et al. 1984	

Table [illegible] ntinued.

Species, Sex, Age	Dose Level in mg/kg/day+ (mg/kg dietary conc. in food) NOEL	LOEL	Exposure Period	Toxicity End Point	Reference	Comments
Redwinged blackbird			6 days	LD50 = 1500 mg/kg	Stickel et al. 1984	
Brown headed cowbird			7 days	LD50 = 1500 mg/kg	Stickel et al. 1984	
Coturnix quail			5 days	LD50 = 2898 mg/kg	Heath et al. 1972	

+ Values without parentheses are doses in mg/kg/day; values in parentheses are dietary levels in mg/kg.
* Calculated by these authors.

Dietary effect levels of DDT and DDE in animals.

Species, Sex, Age	Dose Level in mg/kg/day* (mg/kg dietary conc. in food) NOEL	LOEL	Exposure Period	Toxicity End Point	Reference	Comments
Rat-1			Single dose	LD50 87 mg/kg	NIOSH 1982	
Rat-2		6.6 (88)	2 years	Neoplasms	NCI 1978b	
Rat-3		18.7 (250)	2 years	Reproductive effects	NCI 1978b	
Rat-4		0.375 (5.0)	6 months	Increased enzyme induction	Chadwick et al. 1975	
Bald Eagle-1	0.3(5)	(83)	120 days	Mortality at LOEL	Stickel et al. 1966	
Bald Eagle	(Egg residues, 5 mg/kg in egg)		Full generation field study	10% eggshell thining	Wiemeyer et al. 1984	
American Kestrel		(2.8)	2 years	Significant mortality	Porter & Wiemeyer 1972	Brain residues of 212 to 30 mg/kg
Black duck		2.0 (10)	6 months	1/5 as many ducklings as controls, 30% thinning in eggshells	Longcore & Sampson 1973	
Mallard-1		2.0 (10)	2 laying seasons	24% fractured eggs	Heath et al. 1969	
Mallard-2			5 day diet	LD50 = 2240 mg/kg	Hudson et al. 1984	
Pheasant			5 day diet	LD50 = 1334	Hudson et al. 1984	
Coturnix quail			5 day diet	LD50 = 841 mg/kg	Hudson et al. 1984	
House sparrow		20 (100)	90 days	Mostly all dead	Bernard 1963	

Continued.

Species, Sex, Age	Dose Level in mg/kg/day (mg/kg dietary conc. in food) NOEL	LOEL	Exposure Period	Toxicity End Point	Reference	Comments
Brown Pelican	0.2		8 weeks	Significant reproductive impairment	Blus et al. 1972 and EPA 1976	NOEL from EPA (1976) using Blus et al. (1972) data
Chicken		2.0 (10)	4 weeks	Chick mortality of 7.6%	Britton et al. 1973	Higher dose increased mortality greatly
Chicken	10 (50)		28 weeks	None	Cecil et al. 1974	Residues in eggs were almost 50 mg/kg

* Values without parentheses are doses in mg/kg/day; values in parenthese are dietary levels in mg/kg.

Table 10. Dietary effect levels of aldrin in animals.

Species, Sex, Age	Dose Level in mg/kg/day* (mg/kg dietary conc. in food) NOEL	LOEL	Exposure Period	Toxicity End Point	Reference	Comments
Rat			Single dose	LD50 = 39 mg/kg	NIOSH 1982	
Rat	(2.5)	(12.5)	2 years	Inc. liver weight	Clayton and Clayton 1981	
Rat	0.025 (0.5)		Longterm feeding study	Liver histopathology	FAO/WHO 1978	
Snow Geese			Single dose	112 dead in Texas when rice fields treated	Flickinger et al. 1979	
Chicken			Single dose	LD50 = 10 mg/kg	NIOSH 1982	
Mallard			Single dose	LD50 = 520 mg/kg	Hudson et al. 1984	
Mallard		(5.0)	30 days	EMLD extremely high degree of cumulative toxicity	Hudson et al. 1984	
Coturnix quail	(25)	(50)	14 days	Increased mortality at LOEL	Hill and Camardese 1986	
Pheasant		(4)	10 days	Reduced survival	Post 1952	
Turkey	(3.0)	(6.25)	42 days	Rapid death of many above 12.5 ppm	Anderson et al. 1952	very heavy mortality treated birds exposed.
Starling			Single dose	LD50 = 5.0 mg/kg	Schafer et al. 1983	

10. Continued.

Species, Sex, Age	Dose Level in mg/kg/day (mg/kg dietary conc. in food) NOEL	LOEL	Exposure Period	Toxicity End Point	Reference	Comments
Rabbit	(1.25)	(2.5)	90 days	mortality at 2.5 mg/kg in diet	Borgman et al. 1952	Rabbits more sensitive than rats.
Dog			Single dose	LD50 = 65 mg/kg	NIOSH 1982	
Dog	0.025 (1.0)		Longterm feeding study	Liver histopathology	FAO/WHO 1978	

*Values without parentheses are doses in mg/kg/day; values in parentheses are dietary levels in mg/kg.

Table 11. Dietary effect levels of dieldrin in animals.

Species, Sex, Age	Dose Level in mg/kg/day* (mg/kg dietary conc. in food) NOEL	LOEL	Exposure Period	Toxicity End Point	Reference	Comments
Coturnix Quail		(10)	4 months	No effect on egg numbers or hatch. Small decrease in chick survival	Robinson 1967	20 mg/kg egg residue
Chicken	(5)	(10)	4 months	NOEL at 5 mg/kg diet 25% decreased chick survival at LOEL	Robinson 1967	20-25 mg/kg egg residue/LOEL
Hungarian partridge		(1.0)	1 year	Reduced reproduction in LOEL	Neill et al. 1969 in EPA 1976	
Mallard		(3.0)	4 months	Slight eggshell thinning, 20%	Ichner and Egbert 1969	LOEL not a serious effect due to no significant mortality
Mouse (CF-1)	1.25	2.5	23 months	Lower survival in LOEL O.K. in NOEL	Walker et al. 1972	These studies are the basis of carcinogenic estimates
Mouse	1.5	3.0	From day 7-16	Increased liver weight, osteopathological effects	Chernoff et al. 1975	Mouse twice as sensitive on dosage basis
Rat-1	3(50)	6(100)	From day 7-16	Maternal deaths and weight loss, no anomalies in offspring	Chernoff et al. 1975	Technical grade dieldrin
Rat-2	0.014(0.24)	(0.31)	From 28 day to reproduction	Slight reduction in survival of litters & marked reduction in conception	Harr et al. 1970	

Table 11. Continued.

Species, Sex, Age	Dose Level in mg/kg/day (mg/kg dietary conc. in food) NOEL	LOEL	Exposure Period	Toxicity End Point	Reference	Comments
Rat-3	0.3(5)	0.6(10)	From day 7-16	15% maternal mortality at LOEL, no other effects noted	Chernoff et al. 1975	Photodieldrin exposure
Mouse	1.5	3.0	From day 7-16	Increased liver weight, osteopathologica effects	Chernoff et al. 1975	Mouse twice as sensitive on dosage basis compared to rat.
Hamster		(30)	Single dose to 7-9 day old animal	Pathological effects, fetal deaths	Ottolenghi et al. 1974	
Mouse	(3)	(10)	6 months	Reduced fertility & lowered pup survival	Keplinger et al. 1970	
Dog-2	0.2	0.6	1 year	Increased pup mortality in LOEL	Kitselman 1949	Aldrin effects at 0.2 mg/kg/day dose which is dieldrin NOEL.
Rat-4			Single dose	LD50 = 46 mg/kg	Gaines 1969	
Rat-5	0.025(0.5)	(1.0)	1 year	Liver histopathogy	FAO/WHO 1978 Treon and Cleveland 1955	
Rat-6	(2)	(5)	2 weeks	MFO induction	den Tonkelar and Van Esch 1974	
Dog-1	0.025(1.0)		1 year	Liver weight increase at LOEL	FAO/WHO 1978	
Monkey	0.1	0.5	6 years	liver enzyme induction	Wright et al. 1978	

*Values without parentheses are doses in mg/kg/day; values in parentheses are dietary levels in mg/kg.

Table 12. Dietary effect levels of chlordane in animals.

Species, Sex, Age	Dose Level in mg/kg/day* (mg/kg dietary conc. in food) NOEL	LOEL	Exposure Period	Toxicity End Point	Reference	Comments
Rat-1		(2.5)	40 weeks	Slight liver damage	NCI 1977	
Rat-2	0.25 (5.0)	0.5 (10)	2 year	Kidney & Lung damage	Clayton and Clayton 1981	
Rat-3	0.25(5.0)	0.5(10)	2 weeks 2 weeks	Significant increase in enzyme induction	Den Tonkelaar & Van Esch 1974	
Rat-4	1.0(20)	2.0(40)	2 years	Liver histopathological effects	FAO/WHO 1983; DEC 1986	
Rat-5		1.25 (25)	3 months	Decreased enzyme at Phase System.	Drummond et al. 1980	
Rat-6			Single dose	LD50 = 283 mg/kg	NIOSH 1982	
Dog	0.075(3.0)	0.375(15)	2 years	Liver histopathology Enlarged liver	FAO/WHO 1983	
Mouse-1			Single dose	LD50 = 145 mg/kg	NIOSH 1982	
Mouse-2		2.5 (50)	Single injection	Reduced reproduction	Jang & Talamantes 1977	
Mouse-3		0.16	Single dose	Male offspring had significantly elevated plasma cortisone levels	Cranmer et al. 1984	

*Values without parentheses are doses in mg/kg/day; values in parentheses are dietary concentrations in mg/kg.

Table 13. Dietary effect levels of dioxin in animals.

Species, Sex, Age	Dose Level in ug/kg/day* (mg/kg dietary conc. in food) NOEL	LOEL	Exposure Period	Toxicity End Point	Reference	Comments
Rat-1	5.0	25	Single dose (Gastric intubation)	Weight loss compared to control.	Harris et al. 1973	Illustrates acute toxicity from single dose NOEL/LOEL established.
Rat-2	0.1	1.0	31 Consecutive days (Gastric intubation)	Sig. weight loss with high mortality at high levels.	Harris et al. 1973	Females more sensitive than males
Rat-3		4.0	13 weeks	Chromosomal abberations	IARC 1977	
Rat-4			Single dose	LD50 = 22-45 ug/kg	Kociba and Schwetz 1982	
Rat-5		2.2	2 years	Neoplasms	Kociba et al. 1978	Carcinomas noted by IARC
Rat-6	0.01 (0.133)	0.1 (1.333)	13 weeks	Increased mortality hepatic toxicity porphyria, histopathological effects.	Kociba et al. 1976	Significant mortality at higher treatment levels.
Rat-7	0.001 (.01 to .03 ng/kg)	0.01 (0.12-0.29 ng/kg)	Multigeneration	Mortality, histopathological effects, high tet. levels.	Kociba and Schwetz 1982	
Guinea Pig-1			Single dose	LD50 = 2.0 ug/kg	McConnell et al. 1978	
Guinea Pig-2			Single dose	LD50 = 2.5 ug/kg	Silkworth et al. 1978	

Table 13. Continued.

Species, Sex, Age	Dose level in ug/kg/day* (mg/kg dietary conc. in food) NOEL	LOEL	Exposure Period	Toxicity End Point	Reference	Comments
Guinea Pig-3		0.2 ug/kg	Single dose	90% mortality at 3.0 ug/kg	Harris et al.1973	Illustrates range in sensitivity to TCDD
Guinea Pig-4		0.1	8 weekly doses	thymus effects	Gupta et al. 1973	
Rabbit-1			Single dose	LD50 = 115 ug/kg	McConnel et al. 1978	
Rabbit-2		(10 ug/kg)	1 year	histopathological effects	NIOSH 1982	
Hamster			Single dose	LD50 = 1,157 to 5,051 ug/kg	Kociba and Schwetz 1982	
Mouse			Single dose	LD50 = 100-200 ug/kg	Kociba and Schwetz 1982	
Rhesus monkey-1			Single dose	LD50 = 70 ug/kg	McConnel et al. 1978	
Rhesus monkey-2	0.4 ng/kg/d	(5.0 ng/kg)	8 months	Bone marrow & axial lymph node deficiencies	EPA 1985	
Rhesus monkey-3	(0.5 ng/kg) 0.017 ng/kg/d	(50 ng/kg) 1.7 ng/kg/d	17 to 29 months	Abortion and weight loss	Barsotti et al. 1979 EPA 1985	More severe effects than previous study
Rhesus monkey-4		(1)	up to 61 days	Lethal level	McNulty 1977	
Bobwhite quail			Single dose	LD50=15 ug/kg	Hudson et al. 1984	
Mallard			Single dose	LD50=108 ug/kg	Hudson et al. 1984	
Chicken-1			Single dose	LD50 = 25 ug/kg	Kociba & Schwetz 1982	
Chicken-2		(1 to 10)	21 consecutive days	Chick edema disease	NRCC 1981 (in Gilbertson 1983)	Parallels common tern & herring gull symptoms
Ringed turtle dove			Single dose	LD50 = 810 ug/kg	Hudson et al. 1984	

*Values without parentheses are dose in ug/kg/day; values in parentheses are dietary concentrations in ug/kg, unless otherwise noted.

Table 14. Dietary effect levels of endrin in animals.

Species, Sex, Age	Dose Level in mg/kg/day* (mg/kg dietary conc. in food) NOEL	LOEL	Exposure Period	Toxicity End Point	Reference	Comments
Coturnix Quail		(1)	4 months	No eggs produced during exposure period	NRC 1980	
Rat-1			Single dose	LD50 = 3 mg/kg	NIOSH 1982	
Rat-2		(2.5)	80 weeks	Hyperexcitability and death, and other damage	NCI 1979b	
Rat-3	0.065(1)	0.325(5)	2 years	Liver, heart, kidney increased weight in LOEL	Treon et al. 1955	High mortality at treatment levels above LOEL.
Dog	0.075 (3)	(4)	2 years	Organ damage in LOEL and above	Treon et al. 1955	
Screech Owl		(0.75)**	8 weeks	43% fewer owlets than controls	Fleming et al. 1982	
Mallard-1			Single dose	LD50 = 5.64 mg/kg	Hudson et al. 1984	
Mallard-2			30 days	50% dead at 0.25 mg/kg diet	Hudson et al. 1984	
Starling			Single dose	LD50 = 2.37 mg/kg	Schafer et al. 1983	
Pheasant			Single dose	LD50 = 1.78 mg/kg	Hudson et al. 1984	
Cat			Single dose	LD50 = 5.0 mg/kg	NIOSH 1982	

* Values without parentheses are dose in mg/kg/day; values in parentheses are dietary concentrations in mg/kg.
** Information in this paper was not adequate to calculate a dose. Therefore, this dietary level was used directly to calculate candidate dietary criterion.

Table 15. Dietary effect levels of heptachlor and heptachlor epoxide in animals.

Species, Sex, Age	Dose Level in mg/kg/day* (mg/kg dietary conc. in food) NOEL	LOEL	Exposure Period	Toxicity End Point	Reference	Comments
Rat-1			Single dose	LD50=100 mg/kg	Gaines 1960	LD50 reported in 1960 higher than 1982 RTECS
Rat-2			Single dose (in food)	LD50=40 mg/kg	NIOSH 1982	
Rat-3			Acute toxicity test	LD50=71 mg/kg	Podowski et al. 1979	LD50=60 mg/kg heptachlor epoxide
Rat-4		(6)	one generation-3 generation	Marked decrease in litter size	Mestitzova 1967	Heptachlor only
Rat-5		(10)	8 weeks	Higher protein caused 2 times toxicity due to higher metabolism	Miranda & Webb 1974	
Rat-6	0.075 (1)	(2)	8 months	induced enzymes	Kinoshita and Kempf 1970	
Rat-7	0.075 (1)	(2)	8 months	induced enzymes	Den Tonkelarr & Van Esch 1974	
Rat-8			Single dose	Neonatal LD50 = 120 mg/kg and adult LD50 = 150 mg/kg	Harbison 1975	
Hamster			Single dose	LD50=100 mg/kg	NIOSH 1982	
Mallard			5 days	LD50=2,080 mg/kg	Hudson et al. 1980	Reports storage reduction of DDT with heptachlor present
Mouse				LD50=62 mg/kg	NIOSH 1982	

Table 15. Continued.

Species, Sex, Age	Dose Level in mg/kg/day (mg/kg dietary conc. in food) NOEL	LOEL	Exposure Period	Toxicity End Point	Reference	Comments
Calf	(0.2)	(50)	100 consecutive days	Pyelonnephritis Kidney disorders	Clarke et al. 1981	
Mice		(10)	8 weeks	Hepatiwein thrombosis	Reuber 1977	Both heptachlor & heptachlor epoxide; concluded heptachlor carcinogenic
Chicken	0.05(0.3)	None	8 weeks	None	Wagstaff et al. 1980	No diets 0.3 mg/kg tested.

*Values without parentheses are dose in mg/kg/day; values in parentheses are diet in mg/kg.

Table 16. Dietary effect levels of mirex in animals.

Species, Sex, Age	Dose Level in mg/kg/day* (mg/kg dietary conc. in food) NOEL	LOEL	Exposure Period	Toxicity End Point	Reference	Comments
Rat-1			Single Dose	LD50=6 mg/kg	Gaines & Kimbrough 1969	Lowest LD50 in literature
Rat-2			Single Dose	LD50=235 mg/kg	NIOSH 1982	
Rat-3	(5.0)	(25)	24 months	Fewer and less viable offspring	Gaines & Kimbrough 1969	
Rat-4		10.0 (25)	30 weeks	Some mortality	Chernoff et al. 1979	
Rat-5	(80)	(320)	2 years	Cytopathology, depressed growth	Larson et al. 1979	
Rat-6	(1.0)	(10)	14 days	Food deprivation caused mobilization of fat deposits	Villeneuve et al. 1977	
Rat-7		0.25 (5.0)	1 year	Decrease litter size histopathology	Chu et al. 1981	Reproductive, Chronic study
Rat-8		6.0	8 days	Dam weight loss and fetal edema and cardiovascular disorders	Grabowski 1981	
Old Field Mouse		0.28 (1.8)	60 weeks	20% mortality	Hyde 1972	
Prairie Vole	0.8 (5.0)	(25.0)	13 weeks	100% dead at LOEL	Shannon 1976	
Mouse		(26)	18 months	40% hepatomas	Innes et al. 1969	This study is the basis of carcinogenicity estimates
Bobwhite Quail	(40)		Egg to breeding	No mortality	Kendall et al. 1978	

Table 16. Continued.

Species, Sex, Age	Dose Level in mg/kg/day (mg/kg dietary conc. in food) NOEL	LOEL	Exposure Period	Toxicity End Point	Reference	Comments
Coturnix Quail			5 days acute	LD50=1540-2400 mg/kg	Heath et al. 1970; Hudson et al. 1984	
Pheasant			5 days acute	LD50: 1540-2400 mg/kg	Heath et al. 1979; Hudson et al. 1984	
Mallard			5 days acute	LD50 = 2400 mg/kg	Hudson et al. 1984	
Mallard		(100)	25 weeks	27.4% dead	Hyde 1972	
Mallard		(100)	25 weeks	Survival of duckling from treated adults lower than control	Hyde 1972	

*Values without parentheses are dose in mg/kg/day; values in parentheses are dietary concentrations in mg/kg.

Table 17. Dietary effect levels of hexachlorobenzene in animals.

Species, Sex, Age	Dose Level in mg/kg/day* (mg/kg dietary conc. in food) NOEL	LOEL	Exposure Period	Toxicity End Point	Reference	Comments
Rat-1			Single dose	LD50 = 10,000 mg/kg	NIOSH 1982	
Rat-2			Single dose	LD50 = 3,500 mg/kg	Booth & McDowell 1975	
Rat-3		7.5(100)	4 months	Increased liver size	Kimbrough & Linder 1974	No rats died at LOEL during exposure.
Pig	0.05	0.5	3 months	Porphyria, increased liver weight and death at higher treatment	Fassbender et al. 1977	
Dog (Beagle)		1.25 (50)	21 days	Liver & hepatocyte enlargement	Sundlof et al. 1981	
Coturnix Quail-1	0.2 (1)	1.0 (5)	3 months	Increased liver weight and damage; single dose LD50=1,000 mg/kg.	Vos et al. 1971	5 & 20 mg/kg diet quail gained more weight than the control lot.
Coturnix Quail-2		4.0 (20)	3 months	Decreases survival		
Pheasant			Single dose	LC50 = 617 mg/kg		
Mallard			Single dose	LC50 = 5000 mg/kg .	Hudson et al. 1984	
Chicken	2.0 (10)	20.0 (100)	28 to 52 days	Hepatomegaly in 100 mg/kg birds	Hansen et al. 1978	

Table 17. Continued.

Species, Sex, Age	Dose Level in mg/kg/day* (mg/kg dietary conc. in food) NOEL	LOEL	Exposure Period	Toxicity End Point	Reference	Comments
Cat		4.5 (90)	142 days	susceptibility to respiratory infection	Sidell et al. 1979	Exact mode of HCB suppression of immunity is unknown

*Values without parentheses are dose in mg/kg/day; values in parentheses are dietary concentrations in mg/kg.

Table 18. Dietary effect levels of hexachlorocyclohexane in animals.

Species, Sex, Age	Dose level in mg/kg/day* (mg/kg dietary conc. in food) NOEL	LOEL	Exposure Period	Toxicity End Point	Reference	Comments
Rat-1			Single dose	LD50 = 177 mg/kg	NIOSH 1982	Alpha HCH
Rat-2			Single dose	LD50 = 6000 mg/kg	NIOSH 1982	Beta HCH
Rat-3			Single dose	LD50 = 1000 mg/kg	NIOSH 1982	Delta HCH
Rat-4			Single dose	LD50 = 76 mg/kg	NIOSH 1982	Gamma HCH
Rat-5	9.37 (125)	19.8 (250)	30 days	Neurotoxic effects at LOEL	Muller et al. 1981	Beta & Gamma HCH produce neurotoxic effects.
Dog	0.3 (15.0)		4 months	Neurotoxic effects	Lehman 1965	Gamma HCH Technical grade HCH
Chicken-1	12.8 (64)	(100)	27 days	20-30% decrease in egg production	Whitehead et al. 1981	
Chicken-2	0.02 (0.1)	2.0 (10)	3 months	Reduced hatchability	Sauter and Steele 1972	
Coturnix Quail-1			14 days	LC50 = 49 mg/kg	Hill and Camardese 1986	
Coturnix Quail-2		5.0 (25)	30 days	Reduced hatchability	Dewitt and George 1957	
Pigeon		(150)	1 week	This was an estimate of acute LDLO in field	Blakely 1982	

*Values without parentheses are dose in mg/kg/day; values in parentheses are dietary concentrations in mg/kg.

Table 19. Dietary effect levels of hexachlorobutadiene in animals.

Species, Sex, Age	Dose Level in mg/kg/day* (mg/kg dietary conc. in food) NOEL	LOEL	Exposure Period	Toxicity End Point	Reference	Comments
Rat-1	(2.0)	(20)	30 days	Significant increase of kidney tumors	IARC 1979	
Rat-2	0.2	2.0	2 years	Loss of weight and kidney disease	Kociba et al. 1977 Schwetz et al. 1977	No effect on pregnancy or neonatal survival and development
Rat-3	2.5	2.5 to 6.3	3 months	Renal disorders, females more sensitive	Harleman & Seinen 1979	No effects on fertility
Rat-4			Single dose	LD50 = 90 mg/kg	NIOSH 1982	
Mouse			Single dose	LD50 = 110 mg/kg	NIOSH 1982	
Guinea Pig			Single dose	LD50 = 90 mg/kg	NIOSH 1982	
Coturnix quail	(30)		3 months	No effect on body weight, food consumption, egg production or survival	IARC 1977	

Note: Dermal Toxicity of HCBD is as high as oral toxicity.
*Values without parentheses are dose in mg/kg/day; values in parentheses are dietary concentrations in mg/kg.

Table 20. Dietary effect levels of hexachloroethane in animals.

Species, Sex, Age	Dose Level in mg/kg/day (mg/kg dietary conc. in food) NOEL	LOEL	Exposure Period	Toxicity End Point	Reference	Comments
Rat-1			Single dose	LD50 6000 mg/kg	NIOSH 1982	
Rat-2	0.05		5 1/2 months	No effect	Tugarinova et al. 1960	
Rat-3		212	1 year	A variety of toxic effects	NCI 1978b	Dose level was high; reproduction not studied.
Mouse-1		212	91 weeks	Increased incidence of of cancer, histopathology	NCI 1978b	Established hexachloroethane as carcinogenic to mice.

*Values without parentheses are dose in mg/kg/day.

Table 21. Dietary effect levels of octachlorostyrene in animals.

Species, Sex, Age	Dose Level in mg/kg/day* (mg/kg dietary conc. in food) NOEL	LOEL	Exposure Period	Toxicity End Point	Reference	Comments
Rat-1			Single dose	LD50 = 1300 mg/kg	Chu et al. 1982	
Rat-2	0.314 (5)	3.14 (50)	28 day oral	Liver hypertrophy and hepatic microsomal induction	Chu et al. 1982	Growth rates and consumption not affected at LOEL

*Values without parentheses are dose in mg/kg/day; values in parentheses are dietary concentrations in mg/kg.

Table 22. Dietary effect levels of trichlorobenzene in animals.

Species, Sex, Age	Dose Level in mg/kg/day* (mg/kg dietary conc. in food) NOEL	LOEL	Exposure Period	Toxicity End Point	Reference	Comments
Rat-1			Single Dose	LD50=756 mg/kg	Brown et al. 1969 NIOSH 1982	1,2,4-trichlorobenzene.
Rat-2		10 (100)	1 year, 2 generation	Adrenal gland enlargement	Robinson et al. 1981	No evidence that LOEL affects survival or reproduction in rats.
Rat-3		10	1 year	Xenobiotic metabolism	Carlson and Tardiff 1976	No evidence that LOEL affects survival or reproduction in rats.
Mouse-1			Single Dose	LD50 = 300 mg/kg	NIOSH 1982	1,2,4-trichlorobenzene
Mouse-2			Single dose	LD50 = 766 mg/kg	Brown et al. 1969	
Monkey	25		30 days	Death at 173.6 mg/kg	Smith et al. 1978	

*Values without the parentheses are dose in mg/kg/day; values in parentheses are dietary concentrations in mg/kg.

Table 23. Dietary effect levels of pentachlorophenol in animals.

Species, Sex, Age	Dose Level in mg/kg/day* (mg/kg dietary conc. in food) NOEL	LOEL	Exposure Period	Toxicity End Point	Reference	Comments
Rat-1			Single dose	LD50 210 mg/kg	NIOSH 1982	
Rat-2			Single dose	LD50 146 = mg/kg male LD50 175 = mg/kg female	Windholz 1983	
Rat-3	30.0		90 days	No gross histopathological & histopahtological effects	Schwetz et al. 1977	
Rat-4	3.0	10.0	90 days	Increased liver weight	Johnson et al. 1973	
Rat-5	3.0	10.0	2 years	An accumulation of pigment in liver & kidney	Schwetz et al. 1977	No mortality at 30 mg/kg/day dose level.
Mouse			Single dose	LD10 = 164 mg/kg	NIOSH 1982	
Rabbit			Single dose	LD50 = 328 mg/kg	NIOSH 1982	
Mallard			Single dose	LD50 = 380 mg/kg	Hudson et al. 1984	
Pheasant			Single dose	LD50 = 504 mg/kg	Hudson et al. 1984	

*Values without parentheses are dose in mg/kg/day; values in parentheses are dietary concentrations in mg/kg.

Table 24. Dietary effect levels of 2,3,4,6-tetrachlorophenol in animals.

Species, Sex, Age	Dose Level in mg/kg/day* (mg/kg dietary conc. in food) NOEL	LOEL	Exposure Period	Toxicity End Point	Reference	Comments
Rat-1			Single dose	LD50=130 mg/kg	NIOSH 1982	Intraperitoneal injection
Rat-2			Single dose	LD50=140 mg/kg	NIOSH 1982	
Rat-3	10	50	55 days	Liver damage at LOEL	Nattula et al. 1981	No residues found at NOEL
Guinea Pig			Single dose	LD50=250 mg/kg	NIOSH 1982	

*Values without parentheses are dose in mg/kg/day; values in parentheses are dietary concentrations in mg/kg.

Table 25. Summary of application/uncertainty factors used, fish flesh criteria to prevent non-carcinogenic effects where chronic or sub-acute toxicity data was available for both target and non-target species, and cancer risk fish flesh criteria.

Chemical/Species	Non-carcinogenic Criteria, mg/kg Application/Uncertainty Factors (AF & UF)	Criteria	1 in 100 Cancer Risk Criteria, mg/kg
PCB			
Rabbit	0.1 (S-C AF*)	0.66	-
Chicken	0.1 (I UF*)	0.11	-
Mouse	0.2 (L-N AF*)	2.7	-
Rat	0.2 (L-N AF)	4.2	-
Mink	0.2 (L-N AF)	0.13	
Rat	-	-	0.11
DDT			
Mallard/Black Duck	0.2 (L-N AF)	2.0	-
Bald Eagle	None	1.5	-
Brown Pelican	None	0.2	-
Rat	0.2 (L-N AF)	0.5	-
Mouse	-	-	0.27
Aldrin/Dieldrin			
Mallard	0.2 (L-N AF)	0.6	-
Hungarian Partridge	0.2 (L-N AF)	0.12	-
Rat-3	None	2.0	-
Rat-2	None	0.12	-
Dog-1	None	0.16	-
Dog-2	None	1.33	-
Monkey	None	0.67	-
Mouse	-	-	0.022
Endrin			
Rat	None	0.43	-
Dog	None	0.5	-
Screech Owl	0.2 (L-N AF)	0.15	
Mallard	0.1 (S-C AF)	0.025	-
Mirex			
Rat	0.2 (L-N AF)	0.33	-
Prairie Vole	0.1 (S-C AF)	0.53	-
Old Field Mouse	0.2 (L-N AF)	0.37	-
Mallard	0.2 (L-N AF) 0.1 (I UF)	2.0	-
Mouse	-	-	0.37

*S-C AF = subacute to chronic NOEL application factor.
I UF = interspecies uncertainty factor.
L-N AF = chronic LOEL to chronic NOEL application factor.

Table 26. Fish flesh criteria, residues, and risk for 19 organochlorine chemicals or chemical groups.

Contaminant	Non-carcinogenic Final Fish Flesh Criteria, mg/kg	1 in 100 Cancer Risk Criteria, mg/kg	Spottail Shiner 1981 & 1982 Residues, mg/kg		Residues in Other Fish Species, mg/kg
			Median	Maximum	
PCB's	0.11	0.11	0.327	1.683	0.3-18
Aldrin/Dieldrin	0.12	0.022	0.002	0.009	<0.01-0.09
DDT, DDD, DDE	0.2	0.27	0.031	0.189	0.02-1.32
Chlordane	0.5	0.37	0.0075	0.048	0.01-0.52
Dioxin	0.000003	0.0000023	ND*	0.12 ug/kg	0.87 to 0.162 ug/kg
Endrin	0.025	-	ND	0.007	<0.01
Hexachlorobenzene	0.33	0.2	0.0025	0.261	<0.01-0.35
Hexachlorobutadiene	1.3	4.5	ND	0.029	up to 0.08
Heptachlor and Heptachlor epoxide	0.2	0.21	ND	0.003	0.003-0.015
Hexachlorocyclohexane	0.1	0.51	ND	0.034	0.002-0.05
Hexachloroethane	14.1	May be carcinogenic, but no criterion derived	ND	0.004	-
Mirex	0.33	0.37	ND	0.018	<0.01-0.58
Pentachlorobenzene	Insufficient data	Not shown to be carcinogenic	ND	0.007	-
Octachlorostyrene	0.02	Insufficient data	0.002	0.536	0.09-0.23

Table . Continued.

Contaminant	Non-carcinogenic Final Fish Flesh Criteria, mg/kg	1 in 100 Cancer Risk Criteria, mg/kg	Spottail Shiner 1981 & 1982 Residues, mg/kg		Reside in Other Fish Species, mg/kg
			Median	Maximum	
Tetrachlorophenol	0.67	Insufficient data	ND	0.007	-
Trichlorophenol and (sum of 2,4,6 - and 2,3,5)	-	Insufficient data	ND	0.052	
Trichlorobenzenes	1.3	Insufficient data	ND	0.428	-
Tetrachlorobenzenes	-	Insufficient data	Tr*-0.003	0.011	-
Pentachlorophenol	2.0	Insufficient data	0.01	0.07	ND-0.05**

ND* = not detectable, Tr = Trace.
**Pentachloroanisole residues.

Table 27. Total PCBs in various biota from Moreau marsh adjacent to an inactive hazardous waste site containing PCB (E. Horn, NYS Dept. Envir. Cons., pers. comm.)

Animal or Plant	Tissue	Total PCBs mg/kg	% Lipid
Muskrat	Muscle	0.3	1.5
	Fat	1.0	7.8
Mallard	Muscle	2.1	1.4
	Fat	73.1	31.5
Red-winged blackbird	Muscle	1.5	1.0
Fish	Whole Fish	5.8	3.6
Insects (average of orders)	Whole	2.6	0.75
Mollusks	Whole	4.8	2.5
Frogs	Whole	11.8	-
Plants			
Cattails	Rhizomes	4.2	-
Pondweed	Leaves	0.7	-
Carex	Fruit Composite	0.8	-