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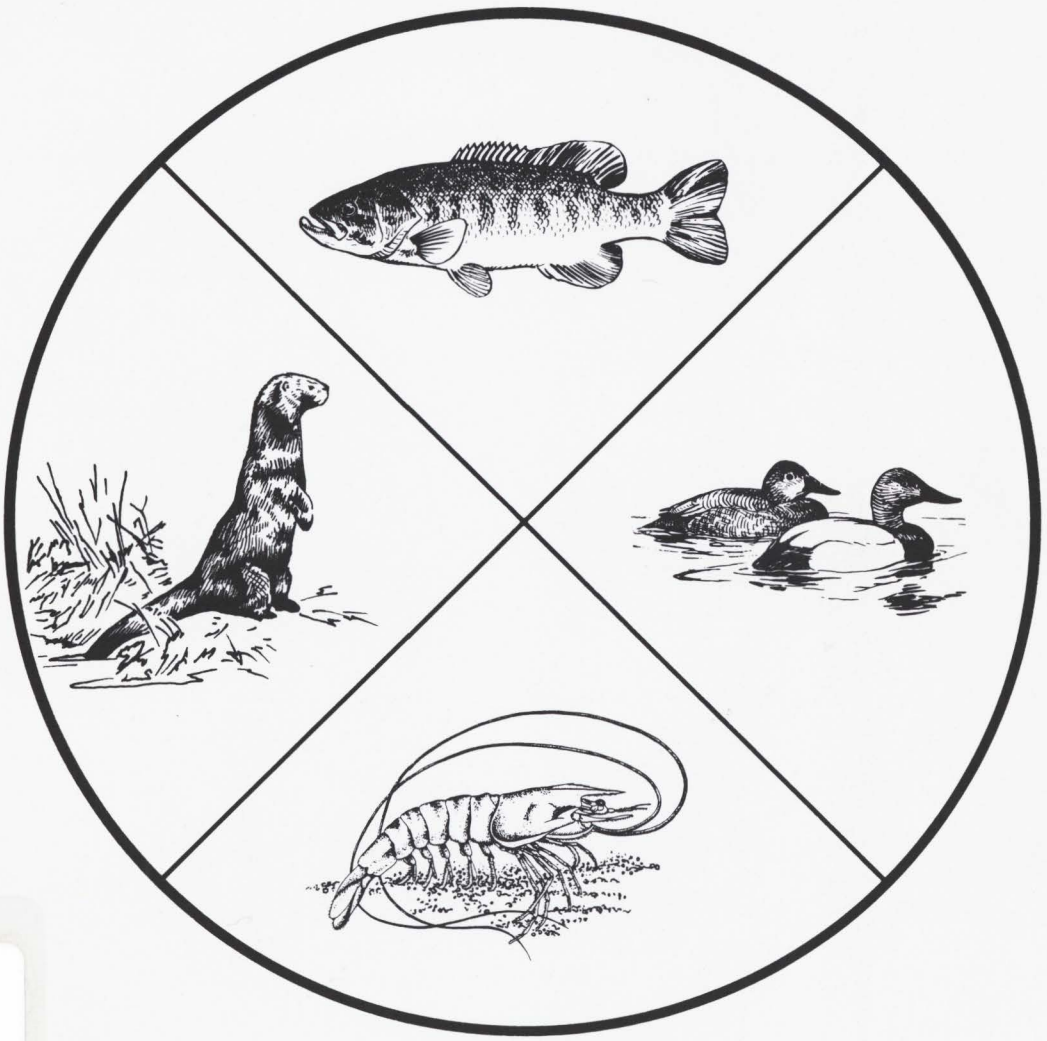
CONTAMINANT HAZARD REVIEWS
REPORT NO. 1

MIREX HAZARDS TO FISH, WILDLIFE, AND INVERTEBRATES: A SYNOPTIC REVIEW



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Contaminant Hazard Reviews
Report No. 1

MIREX HAZARDS TO FISH, WILDLIFE, AND INVERTEBRATES: A SYNOPTIC REVIEW

by

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SUMMARY

Mirex (dodecachlorooctahydro - 1,3,4 - metheno - 2H - cyclobuta(c, d) pentalene) has been used extensively in pesticidal formulations to control the imported fire ant (Solenopsis invicta), and as a flame retardant in electronic components, plastics, and fabrics. One environmental consequence of mirex was the severe damage recorded to fish and wildlife in nine Southeastern States and the Great Lakes, especially Lake Ontario. In 1978, the U.S. Environmental Protection Agency banned all further use of mirex, partly because of the hazards it imposed on nontarget biota. These included delayed mortality and numerous birth defects in aquatic and terrestrial fauna; tumor formation; histopathology; wildlife population alterations; adverse effects on reproduction, early growth, and development; high biomagnification and persistence; degradation into toxic metabolites; movement through aquatic and terrestrial environmental compartments; disrupted mammalian energy metabolism; and detection of residues in human milk and adipose tissues.

Among susceptible species of aquatic organisms, significant damage effects were recorded when concentrations of mirex in water ranged from 2 to 3 ppb. Evidence suggested that sensitive species of wildlife are adversely affected at 0.1 ppm of dietary mirex. For comparison, current tolerance limits for mirex in food for human consumption range from 0.01 ppm for raw agricultural commodities to 0.1 ppm for eggs, milk, and animal fat to 0.4 ppm for various seafood products. Additional research is needed on the fate of mirex degradation products and their effects on natural resources. Further, it is strongly recommended that environmental use of all mirex replacement compounds be preceded by intensive ecological and toxicological evaluation.

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TABLES

ACKNOWLEDGMENTS

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INTRODUCTION

Fish and wildlife resources associated with approximately 51 million hectares (125 million acres) in the Southeastern United States, and with the Great Lakes, especially Lake Ontario, have been negatively affected by intensive or widespread use of mirex, a chlorinated hydrocarbon compound (Waters et al. 1977; Bell et al. 1978; Kaiser 1978; NAS 1978; Lowe 1982). Contamination of the Southeast and of Lake Ontario by mirex probably occurred between 1959 and 1978. During that period, mirex was used as a pesticide to control the imported fire ant (Solenopsis invicta), which infested large portions of Alabama, Arkansas, Florida, Georgia, Louisiana, Mississippi, North Carolina, South Carolina, and Texas. Under the trade name of Dechlorane, mirex was used as a fire retardant in electronic components, fabrics, and plastics; effluents from manufacturing processes resulted in the pollution of Lake Ontario.

Regulatory agencies, environmentalists, and the general public became concerned as evidence accumulated demonstrating that mirex was associated with high death rates, numerous birth defects, and tumors, and that it disrupted metabolism in laboratory mammals, birds, and aquatic biota. Mirex also tends to bioaccumulate and to biomagnify at all trophic levels of food chains. Field studies corroborated the laboratory findings and showed that mirex appeared to be one of the most stable and persistent organochlorine compounds known, being resistant to chemical, photolytic, microbial, metabolic, and thermal degradation processes. Upon degradation, a series of potentially hazardous metabolites are formed, although it is generally acknowledged that the fate and effects of the degradation products are not fully understood. Mirex was also detected in human milk and adipose tissues at low concentrations, the levels related to the degree of environmental contamination.

In 1978, the U.S. Environmental Protection Agency banned all uses of mirex. It is probable, however, that mirex and its metabolites will continue to remain available to living organisms in this country for at least 12 years, although some estimates range as high as 600 years. In this account, I briefly review the evidence leading to the ban of mirex, with emphasis on afflicted natural resources, and provide current recommendations for the protection of fish and wildlife resources. It is part of a continuing series of synoptic reviews prepared in response to requests for information from environmental specialists of the U.S. Fish and Wildlife Service.

CHEMICAL PROPERTIES

Mirex is a white, odorless, free-flowing, crystalline, nonflammable, polycyclic compound composed entirely of carbon and chlorine; the empirical formula is $C_{10}Cl_{12}$, and the molecular weight 545.54 (Hyde 1972; Waters et al. 1977; Bell et al. 1978; NAS 1978; Menzie 1978; Kaiser 1978). In the United States, the common chemical name is dodecachlorooctahydro-1,3,4 - metheno - 2H - cyclobuta(c,d)pentalene; the systematic name is dodecachloropentacyclo $5.3.0.0^{2,6}.0^{3,9}.0^{4,8}$decane. Mirex was first prepared in 1946, patented in 1955 by Allied Chemical Company, and introduced in 1959 as GC 1283 for use in pesticidal formulations against hymenopterous insects, especially ants. It was also marketed under the trade name of Dechlorane for use in flame retardant coatings for various materials. Mirex is also known as ENT 25719 (Tucker and Crabtree 1970), CAS 2385-85-5 (Schafer et al. 1983), Dechlorane 510, and Dechlorane 4070 (Kaiser 1978). Technical grade preparations of mirex consists of 95.19% mirex and less than 2.58×10^{-7}% contaminants, mostly kepone $C_{10}Cl_{10}O$ (NAS 1978).

Mirex is comparatively soluble in various organic solvents, such as benzene, carbon tetrachloride, and xylene, with solubilities ranging from about 4000 to 303,000 ppm (mg/l). However, mirex has a very low solubility in water, not exceeding 1.0 ppb (ug/l) in freshwater or 0.2 ppb in seawater (Bell et al. 1978). In biological systems, mirex lipophilicity would account for the high concentrations observed in fatty tissues and reserves.

Mirex, which is composed of 22% carbon and 78% chlorine, is highly resistant to chemical, thermal, and biochemical degradation. It is reportedly unaffected by strong acids, bases, and oxidizing agents, and is resistant to photolysis in hydrocarbon solvents, but less so in aliphatic amines. Thermal decomposition begins at about 550°C and is rapid at 700°C; degradation products include hexachlorobenzene, hexachlorocyclopentadiene, and kepone. Several additional degradation products of mirex have been isolated, but not all have been identified (Holloman et al. 1975; Menzie 1978). At least one photodegradation product, the 8-monohydro analog, sometimes accumulates in sediments and animals, but the fate and effects of these photoproducts is unclear (Cripe and Livingston 1977).

Mirex is rapidly adsorbed onto various organic particles in the water column, including algae, and eventually is removed to the sediments. Not surprisingly, mirex has a long half-life in terrestrial and aquatic sediments; large fractional residues were detected at different locations 12 and 5 years

after initial application (Bell et al. 1978). Some degradation of mirex to the 10-monohydro analog was reported in anaerobic sewage sludge after 2 months in darkness at 30°C (Menzie 1978). Other studies with mirex-contaminated anaerobic soils, anaerobic lake sediments, and soil microorganisms showed virtually no bacterial degradation over time (Jones and Hodges 1974). In Lake Ontario, mirex from contaminated sediments remained available to lake organisms for many years and, as judged by present sedimentation rates, mirex may continue to be bioavailable for 200 to 600 years in that system (Scrudato and DelPrete 1982). Disappearance of mirex from baits over a 12-month period was about 41% for those exposed on the ground, 56% from those exposed in soil, and 84% from those exposed in pond water (de la Cruz and Lue 1978b). Mirex disappearance was probably related to uptake by biological organisms, as has been demonstrated in marine ecosystems contaminated with mirex (Waters et al. 1977), and not to degradation.

Mirex is a highly stable chlorinated hydrocarbon with lipophilic properties, and its accumulation and persistence in a wide variety of nontarget biological species has been well documented. The biological half-life of mirex reportedly ranges from 30 days in quail to 130 days in fish and to more than 10 months in the fat of female rats (Menzie 1978); this subject area is further developed later. At this juncture, it is sufficient to state that most authorities agree on two points: there is little evidence of significant mirex metabolism; and mirex ranks among the more biochemically stable organic pesticides known.

TOXICITY

AQUATIC ORGANISMS

Aquatic organisms are comparatively resistant to mirex in short term toxicity tests. Among various species of freshwater biota, LC-50 (96 h) values were not obtained at the highest nominal concentrations tested of 1,000 ppb for insects, daphnids, and amphipods (Johnson and Finley 1980; Sanders et al. 1981) and 100,000 ppb for five species of fish (Johnson and Finley 1980). Similar results were reported for other species of freshwater invertebrates (Muncy and Oliver 1963; Lue and de la Cruz 1978) and fishes (Van Valin et al. 1968), although waterborne mirex at concentrations of 1,000 ppb was lethal to postlarval freshwater prawns (Macrobrachium rosenbergerii) in 24 hours (Eversole 1980). It is probable that bioavailable concentrations from the water in each test did not exceed 1.0 ppb. However, delayed mortality frequently occurs for extended periods after exposure, and the potential for adverse effects at the population level remains high (NAS 1978). Latent biocidal properties of mirex were documented for fish (Van Valin et al. 1968; Koenig 1977) and crustaceans (Ludke et al. 1971; Hyde 1972; Cripe and Livingston 1977). Crustaceans were the most sensitive group examined. For example, the crayfish (Procambarus blandingi) immersed in nominal concentrations of 0.1 to 5.0 ppb mirex for periods of 6 to 144 hours died 5 to 10 days after initial exposure (Ludke et al. 1971). Immature crayfish were more sensitive than adults, and mortality patterns were similar when mirex was administered in the water or in baits (Ludke et al. 1971).

BIRDS AND MAMMALS

Acute oral toxicity of mirex to warm blooded organisms was low, except for rats and mice, which died 60 to 90 days after treatment with 6 to 10 mg mirex/kg body weight (Table 1). Birds were comparatively resistant. The red-winged blackbird (Agelaius phoeniceus) was unaffected at 100 mg mirex/kg body weight, even though it was considered the most sensitive of 68 species of birds tested with 998 chemicals for acute oral toxicity, repellency, and hazard potential (Schafer et al. 1983).

Mortality due to dietary mirex is variable among species, although high death rates were usually associated with high dietary concentrations and long exposure periods (Table 2). One significant effect of mirex fed to breeding

adult chickens, voles, and rats was a decrease in survival of the young (Naber and Ware 1965; Shannon 1976; Waters et al. 1977; Chu et al. 1981). Prairie voles (Micropterus ochrogaster) fed diets containing 15 ppm of mirex bred normally, but all pups died by day 21 (Shannon 1976). Survival of the pups of prairie voles decreased in the first litter when the diet of the parents contained 10 ppm mirex, in the second litter when it contained 5 ppm, and in the third litter when it contained 0.1, 0.5, 0.7, or 1.0 ppm (Shannon 1976).

Table 1. Acute oral toxicity of mirex to birds and mammals.

Organism	Dose, in mg/kg body weight	Mortality	Reference
Mice, *Mus* sp.	5	None, 60 days posttreatment	Gaines and Kimbrough 1969
Rat, female, *Rattus* sp.	6	50%, 90 days posttreatment	" "
Mice	10	100%, 60 days posttreatment	" "
Red-winged blackbird	100	None	Schafer et al. 1983
Mice	100-132	50% in 10 days	Fujimori et al. 1983
Common quail, *Coturnix coturnix*	300	12-30%	Stickel 1963
Rat, male	306	Some	Hyde 1972
Mice	330	50%	Waters et al. 1977
Rat, female	365	50%, 14 days posttreatment	Gaines and Kimbrough 1969
Rat, male	400	Lowest fatal dose	NAS 1978
Rat, female	500	Lowest fatal dose	" "
European starling, *Sturnus vulgaris*	562	None	Schafer et al. 1983
Rat, female	600[a]	Some	Hyde 1972
Rabbit, *Lepus* sp.	800[a]	50%	Waters et al. 1977
Dogs, *Canis* sp.	1000	None	Larson et al. 1979
Ring-necked pheasant, *Phasianus colchicus*	1400-1600	50%	Waters et al. 1977
Mallard, *Anas platyrhynchos*	2400	None	Tucker and Crabtree 1970
Japanese quail, *Coturnix coturnix japonica*	10,000	50%	Waters et al. 1977

[a] Dermal

Table 2. Dietary toxicity of mirex to vertebrate organisms.

Organism	Mirex dietary concentration, in ppm	Exposure interval	Percent mortality	Reference
Mallard	1.0	25 weeks	6.2	Hyde 1972
Old-field mouse, *Peromyscus polionotus*	1.8	60 weeks	20.0	Wolfe et al. 1979
Mice	5.0	30 days	Some	Chernoff et al. 1979
Prairie vole	5.0-15	90 days	Some	Shannon 1976
Old-field mouse	17.8	60 weeks	91.7	Wolfe et al. 1979
Beagle dog	20	13 weeks	None	Larson et al. 1979
Pinfish, *Lagodon rhomboides*	20	20 weeks	None	Lowe 1982
Prairie vole	25	90 days	100	Shannon 1976
Rat	25	30 days	Some	Chernoff et al. 1979
Rat	50	14 days	None	NAS 1978
Mice	50	14 days	100	NAS 1978
Coho salmon	50	12 weeks	None	Leatherland et al. 1979
Beagle dog	100	13 weeks	Some	Larson et al. 1979
Mallard	100	25 weeks	27.4	Hyde 1972
Channel catfish	400	4 weeks	None	McCorkle et al. 1979
Ring-necked pheasant	1540	5 days	50.0	Heath et al. 1972
Common bobwhite, *Colinus virginianus*	2511	5 days	50.0	Heath et al. 1972
Japanese quail	5000	5 days	20.0	Heath et al. 1972
Mallard ducklings	5000	5 days	None	Heath et al. 1972

SUBLETHAL EFFECTS

AQUATIC ORGANISMS

The maximum acceptable toxicant concentration (MATC) values calculated for mirex and various freshwater species were <2.4 ppb for amphipods (Gammarus sp.), based on growth inhibition at higher concentrations (Sanders et al. 1981); 2 to 3 ppb for fathead minnows (Pimephalas promelas), as judged by disruption of swim bladder hydroxyproline content, vitamin C metabolism, and bone collagen (Mehrle et al. 1981); 34 ppb for fathead minnows, based on impaired reproduction (Buckler et al. 1981); and >34 ppb for daphnids (Daphnia sp.) and midges (Chaoborus sp.), predicated on daphnid reproduction and midge emergence (Sanders et al. 1981). Other mirex-induced sublethal effects included reduced photosynthesis in freshwater algae (Hollister et al. 1975), gill and kidney histopathology in the goldfish Carassius auratus (Van Valin et al. 1968), reduced growth in the bluegill Lepomis macrochirus (Van Valin et al. 1968), cessation of reproduction in Hydra sp. (Lue and de la Cruz 1978), and disrupted behavior in the blue crab Callinectes sapidus (Shannon 1976) and the marine annelid Arenicola cristata (Schoor and Newman 1976). McCorkle et al. (1979) showed that channel catfish (Ictalurus punctatus) are particularly resistant to high dietary concentrations of mirex; juveniles fed 400 ppm mirex for 4 weeks showed no significant changes in enzyme-specific activities of brain, gill, liver, or muscle. However, yearling coho salmon (Oncorhyncus kisutch) fed 50 ppm mirex for 3 months showed significant reduction in liver weight and whole body lipid content (Leatherland et al. 1979). Additional studies with coho salmon and rainbow trout (Salmo gairdneri) fed 50 ppm mirex for 10 weeks demonstrated a significant depression in serum calcium, and significant elevation of skeletal magnesium in salmon; trout showed no measurable changes in calcium and magnesium levels in serum, muscle, or skeleton, although growth was reduced, muscle water content was elevated, and muscle lipid content was reduced (Leatherland and Sonstegard 1981). Interaction effects of mirex with other anthropogenic contaminants are not well studied, despite the observations of Koenig (1977) that mixtures of DDT and mirex produced more than additive deleterious effects on fish survival and reproduction.

BIRDS

Among captive American kestrels (Falco sparverius) fed 8 ppm mirex for 69 days by Bird et al. (1983), there was a marked decline in sperm concentration and a slight compensatory increase in semen volume, but an overall net decrease of 70% in sperm number. These investigators believed that migratory raptors feeding on mirex-contaminated food organisms could ingest sufficient toxicant to lower semen quality in the breeding season which, coupled with altered courtship, could reduce the fertility of eggs and the reproductive fitness of the individual. Altered courtship in ring-necked doves (Streptopelia capicola) fed dietary organochlorine compounds was reported by McArthur et al. (1983).

Most investigators, however, agree that comparatively high dietary concentrations of mirex had little effect on growth, survival, reproduction, and behavior of nonraptors, including chickens (Gallus sp.), mallards, several species of quail, and red-winged blackbirds. For domestic chickens, levels up to 200 ppm dietary mirex were tolerated without adverse effects on various reproductive variables (Waters et al. 1977), but 300 ppm mirex for 16 weeks was associated with reduced chick survival, and 600 ppm for 16 weeks reduced hatching by 83% and chick survival by 75% (Naber and Ware 1965). Mallard ducklings experienced temporary mild ataxia and regurgitation when given a single dose of 2,400 mg/kg body weight but not when given 1200 mg/kg or less (Tucker and Crabtree 1970). Mallards fed up to 100 ppm mirex for prolonged periods showed no significant differences from controls in egg production, shell thickness, shell weight, embryonation, hatchability, or duckling survival (Hyde 1972). However, in other studies with mallards fed 100 ppm dietary mirex, eggshells were thinned and duckling survival was reduced (Waters et al. 1977), suggesting that 100 ppm dietary mirex may not be innocuous to mallards. No adverse effects on reproduction were noted in the common bobwhite at 40 ppm dietary mirex (Kendall et al. 1978), or in two species of quail fed 80 ppm mirex for 12 weeks (Waters et al. 1977). Red-winged blackbirds were not repelled by foods contaminated with mirex, but consumed normal rations (Schafer et al. 1983); a similar observation was recorded for bobwhites (Baker 1964).

MAMMALS

Mirex has considerable potential for chronic toxicity since it is only partly metabolized, is eliminated very slowly, and is accumulated in the fat, liver, and brain. The most common effects observed in small laboratory mammals fed mirex included weight loss, enlarged livers, altered liver enzyme metabolism, and reproductive failure. Mirex reportedly crossed placental membranes and accumulated in fetal tissues. Among the progeny of mirex-treated mammals, developmental abnormalities included cataracts, heart defects, scoliosis, and cleft palates (NAS 1978).

Mirex has caused liver tumors in mice and rats and must be considered a potential human carcinogen (Waters et al. 1977; NAS 1978). Long-term feeding of 50 and 100 ppm of mirex to rats of both sexes was associated with liver lesions that included neoplastic nodules and hepatocellular carcinomas; neither sign was found in controls (Ulland et al. 1977).

Adults of selected mammalian species showed a variety of damage effects of mirex: enlarged livers in rats at 25 ppm dietary mirex (Gaines and Kimbrough 1969) or at a single dose of 100 mg/kg body weight (Ervin 1982); liver hepatomas in mice at 10 mg mirex/kg body weight daily (Innes et al. 1969); decreased incidence of females showing sperm in vaginal smears, decreased litter size, and thyroid histopathology in rats fed 5 ppm dietary mirex since weaning (Chu et al. 1981); elevated blood and serum enzyme levels in rats fed 0.5 ppm mirex for 28 days (Yarbrough et al. 1981); and diarrhea, reduced food and water consumption, body weight loss, decreased blood glucose levels, and disrupted hepatic microsomal mixed function oxidases in mice receiving 10 mg/kg daily (Fujimori et al. 1983). In studies of field mice, decreased litter size was observed at 1.8 ppm dietary mirex, and complete reproductive impairment at 17.6 ppm after 6 months (Wolfe et al. 1979). At comparatively high sublethal concentrations of mirex, various deleterious effects were observed: thyroid histopathology and decreased spermatogenesis in rats fed 75 ppm mirex for 28 days (Yarbrough et al. 1981); abnormal blood chemistry, enlarged livers, reduced spleen size, and loss in body weight of beagles fed 100 ppm for 13 weeks (Larson et al. 1979); and decreased hemoglobin, elevated white blood cell counts, reduced growth, liver histopathology, and enlarged livers in rats fed 320 ppm for 13 weeks (Larson et al. 1979).

Cataract formation, resulting in blindness, in fetuses and pups from maternal rats fed comparatively low concentrations of dietary mirex is one of the more insidious effects documented. Mirex fed to maternal rats at 6 mg/kg body weight daily on days 8-15 of gestation, or at 10 mg/kg daily on days 1-4 postpartum, caused cataracts in 50% of fetuses on day 20 of gestation, and in 58% of pups on day 14 postpartum (Rogers 1982). Plasma glucose levels were depressed in fetuses with cataracts, and plasma proteins were depressed in neonates; both hypoproteineia and hypoglycemia are physiological conditions known to be associated with cataracts (Rogers 1982). Mirex-associated cataractogenicity has been reported in female pups from rats fed 5 ppm dietary mirex since weaning (Chu et al. 1981), in rat pups from females consuming 7 ppm dietary mirex on days 7-16 of gestation or 25 ppm in diets for 30 days prior to breeding (Chernoff et al. 1979), and in mice fed 12 ppm dietary mirex (Chernoff et al. 1979). Offspring born to mirex-treated mothers, but nursed by nontreated mothers showed fewer cataracts (Waters et al. 1977). Other fetotoxic effects in rats associated with dietary mirex included: edema and undescended testes (Chernoff et al. 1979); lowered blood plasma proteins, and heart disorders, including tachycardia and blockages (Grabowski 1981); and, as noted by Kavlock et al. (1982), hydrocephaly; decreases in weight of brain, lung, liver, and kidney; decreases in liver glycogen, kidney proteins and alkaline phosphatase; and disrupted brain DNA and protein metabolism.

In prairie voles exposed continuously to dietary mirex of 0.5, 0.7, 1.0, 5.0, or 10.0 ppm, the numbers of litters produced decreased (Shannon 1976). Maximum number of litters per year were four at 1.0 ppm dietary mirex; three at 5.0 ppm; and two at 10.0 ppm. Furthermore, the number of offspring per litter also decreased progressively. Concentrations as low as 0.1 ppm in the diet of adults were associated with delayed maturation of pups and with an increase in number of days required to attain various behavioral plateaus such as bar-holding ability, hind-limb placing, and negative geotaxis (Shannon 1976). On the basis of residue data from field studies, as is shown later, these results strongly suggest that mirex was harmful to the reproductive performance and behavioral development of prairie voles at environmental levels approaching 4.2 g mirex/hectare, a level used to control fire ants before mirex was banned.

BIOACCUMULATION

AQUATIC ORGANISMS

All aquatic species tested accumulated mirex from the medium and concentrated it over ambient water levels by factors ranging up to several orders of magnitude; uptake was positively correlated with nominal dose in the water column (Table 3). Other investigators have reported bioconcentration factors from water of 8,025X in daphnids (Sanders et al. 1981), 12,200 in bluegills (Skaar et al. 1981), 56,000 in fathead minnows (Huckins et al. 1982), and 126,600 in the digestive gland of crayfish (Ludke et al. 1971). Rapid uptake of mirex by marine crabs, shrimps, oysters, killifishes, and algae was reported after the application of mirex baits to coastal marshes (Waters et al. 1977; Cripe and Livingston 1977). Mirex was also accumulated from the diet (Table 3; Ludke et al. 1971; Zitko 1980) but not as readily as from the medium. Mirex may also be accumulated from contaminated sediments by marine teleosts (Kobylinski and Livingston 1975), but such accumulation has not been established conclusively. Although terrestrial plants, such as peas and beans, accumulate mirex at field application levels, mangrove seedlings require environmentally high levels of 11.2 kg mirex/ha before accumulation occurs (as quoted in Shannon 1976).

There is general agreement that aquatic biota subjected to mirex-contaminated environments continue to accumulate mirex, and that equilibrium is rarely attained before death of the organism from mirex poisoning or from other causes. There is also general agreement that mirex resists metabolic and microbial degradation, exhibits considerable movement through food chains, and is potentially dangerous to consumers at the higher tropic level (Hollister et al. 1975; NAS 1978; Mehrle et al. 1981). Marine algae, for example, showed a significant linear correlation between amounts accumulated and mirex concentrations in the medium. If a similar situation existed in nature, marine unicellular algae would accumulate mirex and, when grazed upon, act as passive transporters to higher trophic food chain compartments (Hollister et al. 1975). The evidence for elimination rates of mirex from aquatic biota on transfer to mirex-free media is not as clear. Biological half-times of mirex have been reported as 12 hours for daphnids (Sanders et al. 1981), more than 28 days for fathead minnows (Huckins et al. 1982), about 70 days in Atlantic salmon (Salmo salar) (Zitko 1980), 130 days for mosquitofish (Gambusia affinis), and 250 days for pinfish (as quoted in Skea et al. 1981). However, Skea et al. (1981) averred that biological half-times may be much longer if organism growth is incorporated into rate

Table 3. Bioaccumulation of mirex from ambient medium or diet by selected species.

Habitat, organism, and tissue	Mirex concentration in ppb in medium (M), or in ppm in diet (D)	Exposure period	Bioconcentration factor	Reference[a]
FRESHWATER				
Fish				
Fathead minnow				
Whole	2.0 (M)	120 days	28,000	Buckler
"	7.0 (M)	" "	18,400	et al. 1981
"	13.0 (M)	" "	12,000	
"	34.0 (M)	" "	13,800	
"	2.0 (M)	120 + 56 days	12,000	
"	7.0 (M)	" "	6,860	
"	13.0 (M)	" "	5,460	
"	34.0 (M)	" "	7,880	
Bluegill				
Whole	1.3 (M)	60 days	1,540	Van Valin
"	1000.0 (M)	90 days	150	et al. 1968
Goldfish				
Skin	100.0 (M)	224 days	1,220	
Muscle	" "	" "	460	
Liver	" "	" "	370	
Gut	" "	" "	1,520	
Atlantic salmon				
Whole	0.6 (D)	15 days	0.06	Zitko 1980
"	" "	42 days	0.13	
Brook trout				
Whole	29.0 (D)	17 days	0.04	Skea
"	" "	104 days	0.22	et al. 1981
"	" "	104 + 385 days	0.07	

Table 3. (Continued)

Habitat, organism, and tissue	Mirex concentration in ppb in medium (M), or in ppm in diet (D)	Exposure period	Bioconcentration factor	Reference[a]
Crustaceans				
Crayfish				
Muscle	7.4 (M)	10-21 days	81	Minchew
Brain	" "	(interval	54	et al. 1980
Nerve cord	" "	represents	54	
Green gland	" "	appearance of	243	
Gill	" "	late symptoms	108	
Digestive gland	" "	of mirex	622	
Intestine	" "	toxicity)	257	
Muscle	74.0 (M)	7-14 days	8	
Brain	" "	(see above)	80	
Nerve cord	" "	"	84	
Green gland	" "	"	76	
Gill	" "	"	23	
Digestive gland	" "	"	105	
Intestine	" "	"	43	
MARINE				
Fish				
Diamond killifish, Adinia xenica (Exposed adults)				
Embryo	1.5 (D)	9 days	1.7	Koenig 1977
"	6.0 (D)	"	1.3	
"	24.0 (D)	"	1.2	
"	96.0 (D)	"	0.9	
Hogchoker, Trinectes maculatus				
Muscle	56.0-5000.0 (M)	4 weeks	3800-10,400	Kobylinski and Livingston 1975

Table 3. (Continued)

Habitat, organism, and tissue	Mirex concentration in ppb in medium (M), or in ppm in diet (D)	Exposure period	Bioconcentration factor	Reference[a]
Striped mullet, *Mugil cephalus*				
Whole	10.0 (M)	4 days	17-38	Lee et al. 1975
Crustaceans				
Shrimp, *Palaemonetes vulgaris*				
Hepatopancreas	0.04 (M)	4 days	9250	Schoor 1979
"	" "	13 days	16,250	
Muscle	" "	4 days	2250	
"	" "	13 days	2000	
Whole	" "	4 days	4000	
"	" "	13 days	3250	
Algae				
Whole	0.04 (M)	13 days	375	Schoor 1979
Whole, 4 spp.	0.01 (M)	7 days	3200-7500	Hollister et al. 1975
TERRESTRIAL				
Birds				
Chickens				
Fat	1.06 (D)	39 weeks	24	Waters et al. 1977
Kidney	" "	"	3	
Liver	" "	"	2	
Muscle	" "	"	0.3	
Skin (chick)	1.0 (D)	2 weeks	37	Ahrens et al. 1980
Fat (chick)	" "	"	586	
Mallards (exposed adults)				
Eggs	1 (D)	18 weeks	2.4	Hyde 1972

Table 3. (Continued)

Habitat, organism, and tissue	Mirex concentration in ppb in medium (M), or in ppm in diet (D)	Exposure period	Bioconcentration factor	Reference[a]
Eggs	100 (D)	"	28	
Fat	" "	"	29	
American kestrels, yearling males				
Muscle lipids	8.0 (D)	69 days	7	Bird
Testes lipids	" " (D)	"	6	et al. 1983
Liver lipids	" " (D)	"	3	
Common bobwhite				
Fat	1.0 (D)	36 weeks	20	Kendall
"	20.0 (D)	"	10	et al. 1978
"	40.0 (D)	"	9.5	
Breast muscle	1.0 (D)	"	0.7	
" "	20.0 (D)	"	0.6	
" "	40.0 (D)	"	0.3	
Mammals				
Rat				
Adipose fat	3.0 (D)	6 days	16	NAS 1978
" "	12.5 (D)	6 days	23	
" "	5.0 (D)	16 weeks	62	Chu
" "	10.0 (D)	"	42	et al. 1981
" "	20.0 (D)	"	43	
" "	40.0 (D)	"	18	
Liver	5.0 (D)	"	1	
"	10.0 (D)	"	1.4	
"	20.0 (D)	"	1.6	
"	40.0 (D)	"	3	

Table 3. (Concluded)

Habitat, organism, and tissue	Mirex concentration in ppb in medium (M), or in ppm in diet (D)	Exposure period	Bioconcentration factor	Reference[a]
Old-field mouse				
Liver	1.8 (D)	24 weeks	3.3	Wolfe
"	17.8 (D)	"	3.6	et al. 1979
Rhesus monkey, Macaca mulatta				
Fat	1.0 (D)	single dose	1.7-5.8	NAS 1978

[a] Reference cited applies to data in that row and to data in other rows immediately following.

elimination models. For example, brook trout (Salvelinus fontinalis) fed 29 ppm mirex for 104 days contained 6.3 mg/kg body weight or a total of 1.1 mg of mirex in whole fish. At day 385 postexposure, after the trout had tripled in body weight, these values were 2.1 mg/kg body weight, an apparent loss of 67%; however, on a whole fish basis, trout contained 1.2 mg, thus showing essentially no elimination on a total organism basis (Skea et al. 1981).

No mirex degradation products were detected in whole fathead minnow or in hydrosoils under aerobic or anaerobic conditions (Huckins et al. 1982). In contrast, three metabolites were detected in coastal marshes after mirex bait application, one of which, photomirex, was accumulated by fish and oysters (Cripe and Livingston 1977). The fate and effects of mirex photoproducts in the environment is unclear and merits additional research.

The significance of mirex residues in various tissues is unresolved, as is the exact mode of action of mirex and its metabolities. Minchew et al. (1980) and others indicated that mirex is a neurotoxic agent, with a mode of action similar to that of other chlorinated hydrocarbon insecticides, such as DDT. In studies with crayfish and radiolabeled mirex, mirex toxicosis was associated with neurotoxic effects that included hyperactivity, uncoordinated movements, loss of equilibrium, and paralysis (Minchew et al. 1980). Before death, the most significant differences in mirex distributions in crayfish were the increases in concentrations in neural tissues, such as brain and nerve cord, by factors up to 14X (or 0.4 ppm) in low-dose groups held in solutions containing 7.4 ppb mirex, and up to 300X (or 6.2 ppm) in high-dose groups held in solutions with 74.0 ppb. With continued exposure, levels in the green gland and neural tissues approached the levels in the hepatopancreas and intestine (Table 3). Schoor (1979) also demonstrated that mirex accumulates in the crustacean hepatopancreas, but suggested that other tissues, such as muscle and exoskeleton, have specific binding sites that, once filled, shunt excess mirex to hepatopancreas storage sites.

BIRDS AND MAMMALS

Like aquatic organisms, birds and mammals accumulated mirex in tissue lipids, and the greater accumulations were associated with the longer exposure intervals and higher dosages (Table 3). Sexual condition of the organism may modify bioconcentration potential. For example, in adipose fat of the bobwhite, males contained 10X dietary levels and females only 5X dietary levels; the difference was attributed to mirex loss through egg laying (Kendall et al. 1978).

Data on excretion kinetics of mirex are incomplete. Prairie voles fed mirex for 90 days contained detectable whole body levels 4 months after being placed on a mirex-free diet (Shannon 1976). Levels of mirex in voles after 4 months on uncontaminated feed were still far above levels in their mirex diets. Humans living in areas where mirex has been used for ant control contained 0.16 to 5.94 ppm in adipose fat; 60% of the mirex was excreted and

most of the rest was stored in body tissues, especially fat (28%), and in lesser amounts of 0.2 to 3% in muscle, liver, kidney, and intestines (Waters et al. 1977). Almost all excretion of mirex takes place through feces; less than 1% is excreted in urine and milk. The loss rate pattern is biphasic; the fast phase was estimated at 38 hours and the slow phase at up to 100 days. Mirex binds firmly to soluble liver proteins and appears to be retained in fatty tissues, a property that may contribute to its long biological half-life. Chickens given single doses of mirex at 30 mg/kg intravenously or 300 mg/kg orally demonstrated a biphasic decline in blood concentrations (Ahrens et al. 1980). The fast component, constituting about 25% of the total, was lost during the first 24 hours; the loss of the slow component was estimated to be at a constant rate of about 0.03% daily, suggesting a half-life of about 3 years. Growing chicks fed 1 or 10 ppm dietary mirex for 1 week lost the compound rather rapidly; disappearance half-times were 25 days for skin and 32 days for fat (Ahrens et al. 1980). It is clear that much additional research is warranted on loss rate kinetics of this persistent compound and its metabolites.

MIREX IN THE SOUTHEASTERN UNITED STATES

Between 1961 and 1975, about 400,000 kg of mirex were used in pesticidal formulations, of which approximately 250,000 kg were sold in the Southeastern U.S. for control of native and imported fire ants (Solenopsis spp.); most of the rest was exported to Brazil for use in fire ant control in that country (NAS 1978). Mirex was also used to control big-headed ant populations in Hawaiian pineapple fields (Bell et al. 1978), Australian termites (Paton and Miller 1980), South American leaf cutter ants, South African harvester termites, and, in the U.S., western harvester ants and yellow jackets (Shannon 1976). Chemical control measures for imported fire ants began in the Southeastern United States during the 1950's with the use of heptachlor, chlordane, and dieldrin. The large mounds built by ants in cultivated fields were believed to interfere with mowing and harvesting operations, the "vicious sting" of the insects presented a hazard to workers harvesting the crops, and the species was considered to be a pest in school playgrounds and homes (Lowe 1982). In 1965, the use of organochlorine insecticides to control fire ants was discontinued, due partly to their high acute toxicity to nontarget biota and their persistence. Previously used compounds were replaced by mirex 4X bait formulations, consisting of 0.3% mirex by weight, dissolved in 14.7% soybean oil, and soaked into corncob grits (85%). Initially, the 4X baits were broadcast from low flying airplanes at a total yearly rate of 1.4 kg bait/ha (1.25 lbs total bait/acre) or 4.2 g mirex/ha. Usually, three applications were made yearly. More than 50 million hectares in nine Southeastern States were treated over a 10-year period. Later, dosages were modified downward, and mirex was applied to mounds directly. Ecologically sensitive areas, such as estuaries, prime wildlife habitats, heavily forested areas, and State and Federal parks, were avoided. In 1977, for example, the formulation was changed to 0.1% mirex and the application rate lowered to 1.12 g/ha; about 8200 kg of the lower concentration bait were manufactured in 1977 (Bell et al. 1978). Under ideal aerial application conditions, about 140 particles of mirex-impregnated bait were distributed/m^2. When an infested area is treated, the bait is rapidly scavenged by the oil-loving fire ant workers, placed in the mound, and distributed throughout the colony, including queen and brood, before any toxic effects become evident; death occurs in several days to weeks. The exact mode of action is unknown, but is believed to be similar to that of other neurotoxic agents, such as DDT (Waters et al. 1977; NAS 1978).

Widespread use of mirex may lead to altered population structure in terrestrial systems, with resurgence or escalation of nontarget pests due to

selective mirex-induced mortality of predators (NAS 1978). For example, populations of immature horn flies and rove beetles, two species of arthropods normally preyed upon by fire ants, were higher in mirex-treated areas than in control areas (Howard and Oliver 1978). Conversely, other species, such as crickets, ground beetles, and various species of oil-loving ants, were directly affected and populations were still depressed or eliminated 14 months posttreatment (NAS 1978), whereas fire ants recovered to higher than pretreatment levels, as judged by mound numbers and mound size (Summerlin et al. 1977).

Field results from aquatic and terrestrial ecosystems receiving mirex bait formulations indicated, with minor exceptions, that mirex accumulates sequentially in food complexes and concentrates in animals at the higher trophic levels. In both ecosystems, omnivores and top carnivores contained the highest residues (Hyde 1972; Shannon 1976; Waters et al. 1977; de la Cruz and Lue 1978a; Hunter et al. 1980). In South Carolina, where the 4X formulation was used to control fire ants from 1969 to 1971, mirex was translocated from treated lands to nearby marshes and estuarine biota, including crustaceans, marsh birds, and raccoons (Lowe 1982). Juvenile marine crustaceans showed delayed toxic effects after ingesting mirex baits, or after being exposed to low concentrations in seawater. About 18 months posttreatment, mirex residues of 1.3 to 17.0 ppm were detected in shrimp, mammals, and birds (Table 4); however, 24 months after the last mirex treatment, less than 10% of all samples collected contained detectable residues (Lowe 1982). A similar study was conducted in pasturelands of bahia grass (Paspalium notatum) (Markin 1981). Within a month after application, the target fire ant colonies were dead. Of the 4.2 g mirex/ha applied to the 164 hectare block, 100% was accounted for on day 1, 63% at 1 month, and 3% at 1 year (Table 5). Unaccounted mirex residues could include loss through biodegradation; through movement out of the study area by migratory insects, birds, other fauna, and ground water; and through photodecomposition and volatilization (Markin 1981).

Mirex residues in bobwhites from South Carolina game management area were documented after treatment with 4.2 g mirex/ha (Kendall et al. 1977). Pretreatment residues in bobwhites ranged from nondetectable to 0.17 ppm mirex in breast muscle on a dry weight basis, and 0.25 to 2.8 ppm in adipose tissues on a lipid weight basis. Mirex residues in adipose tissue increased up to 5X within 1 month posttreatment and declined thereafter; however, another residue peak was noted in the spring after mirex treatment and corresponded with insect emergence (Kendall et al. 1977).

Heavily treated watershed areas in Mississippi were investigated by Wolfe and Norment (1973) and Holcombe and Parker (1979). After treatment, mirex residues were elevated in crayfish and stream fish; among mammals, residues were highest in carnivores and insectivores, lower in omnivores, and lowest in herbivores (Wolfe and Norment 1973). Mirex residues in liver and eggs were substantially higher in the box turtle (Terrapene carolina) an omnivorous feeder, than in the herbivorous slider turtle (Chrysemys scripta); mirex did not accumulate for protracted periods in tissues of these comparatively

Table 4. Mirex residues in water, sediments, and fauna in a South Carolina coastal marsh 18 months after application of 4.2 g/ha (after Lowe 1982).

Sample	Maximum mirex residues, in ppm	Percent samples with mirex residues
Water	<0.01	0
Sediments	0.7	1
Crabs	0.6	31
Fishes	0.8	15
Shrimp	1.3	10
Mammals	4.4	54
Birds	17.0	78

Table 5. Temporal persistence of residues for 1 year after applications of mirex 4X formulation to bahia grass pastures. Values represent rounded percentages recovered of the original 4.2 g/ha applied (after Markin 1981).

Sample	Time, postapplication						
	1 d	2 wk	1 mo	3 mo	6 mo	9 mo	12 mo
Imported fire ants	44	8	0	0	0	0	0
Grit from bait	40	35	a	-	-	-	-
Soil from mound	0.3	2	4	3	2	b	-
Pasture soil	18	18	52	26	24	5	3
Bahia grass	0.3	0.9	0.4	0.7	0.3	0.6	0.0
Invertebrates	0.3	0.2	0.3	0.2	0.0	0.1	0.1
Vertebrates	-	0.1	0.2	0.2	0.1	0.1	0.1
Not accounted for	0	35	43	69	74	94	97

[a]Grit now included with pasture soil.

[b]Mounds badly weathered, not possible to identify.

long-lived reptiles (Holcombe and Parker 1979). Among migratory reptiles, mirex was detected in only 11% of the eggs of the loggerhead turtle (Caretta caretta) and not at all in eggs of the green turtle (Chelonia mydas) collected during summer 1976 in Florida (Clark and Krynitsky 1980). However, DDT or its isomers were present in all eggs of both species, and PCB's were detected in all loggerhead turtle eggs. The low levels of mirex and other organochlorine contaminants suggest that these turtles, when not nesting, live and feed in areas remote from Florida lands treated with mirex and other insecticides (Clark and Krynitsky 1980).

A 10-5 bait formulation containing 0.1% mirex was designed to make more of the toxicant available to the fire ant and less to nontarget biota. In one study, the 10-5 formulation was applied to a previously untreated 8000-hectare area near Jacksonville, Florida, infested with fire ants (Wheeler et al. 1977). The bait was applied by airplane at 1.12 kg/ha, or 1.12 g mirex/ha. Insects accumulated mirex to the greatest extent during the first 6 months after application, and most of the mirex was lost by 12 months (Table 6); other invertebrates accumulated only low levels during the first 9 months, and no residues were detected after 12 months. Fish also showed low concentrations for 9 months and no detectable residues afterward; amphibians contained detectable residues after 12 months, but not at 24; reptiles contained measurable, but low, residues for the entire 24-month study period. Mammals had higher residue levels than reptiles, particularly in fat, whereas birds contained low to moderate residues (Table 6). After 24 months, mirex was found infrequently and only at low concentrations in birds, mammals, reptiles, and insects. It was concluded that 10-5 mirex formulations were as effective in controlling fire ants as the 4X formulation and that residues in nontarget species were reduced from that following 4X treatment, or were lacking (Wheeler et al. 1977).

Eggs of the American crocodile (Crocodylus acutus) from the Florida Everglades contained up to 2.9 ppm fresh weight of DDE and 0.86 ppm of PCB's, but less than 0.02 ppm mirex (Hall et al. 1979). Livers of the deep sea fish (Antimora rostrata) collected in 1971-74 from a depth of 2500 m off the U.S. east coast, contained measurable concentrations of DDT and its degradation products, and dieldrin, but no mirex (Barber and Warlen 1979).

Table 6. Mirex residues in fauna near Jacksonville, Florida, at various intervals posttreatment following single application of 1.12 g mirex/ha (after Wheeler et al. 1977).

Taxonomic group and time (in months posttreatment)	Maximum residue, in ppm wet weight whole organism
Insects	
1	4.1
3	19.8
6	7.8
12	1.1
24	0.8
Amphibians	
1	0.78
24	ND[a]
Reptiles	
1-9	1.2
24	0.06
Fish	
1	0.08
3-9	0.25
>9	ND
Birds	
1-12	10.0 (fat)
9-24	b
Mammals	
1-6	3.4 (fat)
12-18	0.7 (fat)
24	0.09 (fat)

[a]ND = not detectable.
[b]Pretreatment levels.

MIREX IN THE GREAT LAKES

Between 1959 and 1975, 1.5 million kg of mirex were sold, of which 74% or more than 1.1 million kg were predominantly Dechlorane, a compound used in flame-resistant polymer formulations of electronic components and fabrics (Bell et al. 1978; NAS 1978). The total amounts are only approximate because almost half the mirex sold from 1962 to 1973 could not be accounted for (NAS 1978). Kaiser (1978) reported that all fish species in Lake Ontario were contaminated with mirex, and that concentrations in half the species exceeded the Food and Drug Administration guideline of 0.1 ppm; other aquatic species had mirex residues near this level. Reproduction of the herring gull (Larus argentatus) on Lake Ontario was poor; mirex levels were an order of magnitude higher in gull eggs from Lake Ontario than in eggs from other Great Lakes locations (Kaiser 1978). It was concluded that the probable source of contamination was a chemical manufacturer that used mirex (Dechlorane) as a flame retardant, and that only Lake Ontario was contaminated (Kaiser 1978; NAS 1978).

Gilman et al. (1977, 1978) observed poor reproductive success and declines in colony size of the herring gull at Lake Ontario at a time when dramatic increases of this species were reported along the Atlantic seaboard. In 1975, herring gull reproduction in Lake Ontario colonies was about one-tenth that of colonies on the other four Great Lakes. In addition, in Lake Ontario colonies, there were reductions in nest site defense, in number of eggs per clutch, in hatchability of eggs, and in chick survival. Hatching success of Lake Ontario gull eggs was 23 to 26%, compared with 53 to 79% for eggs from other areas. Analysis of herring gull eggs from all colonies for organochlorine compounds and mercury demonstrated that eggs from Lake Ontario colonies had mean mirex levels of 5.06 ppm fresh weight (range, 2.0-18.6), or about 10X more mirex than any other colony. Mean PCB and mercury levels were up to 2.8X and 2.3X higher, respectively, in gull eggs from Lake Ontario than in those from other colonies, but only mirex levels could account for the colony declines (Gilman et al. 1977, 1978). As judged by log-linear regression models, the half-life for mirex in herring gull eggs was 1.9 to 2.1 years, or essentially none was lost during egg incubation (Weseloh et al. 1979). Reproductive success of the Lake Ontario herring gull colonies improved after the early 1970's, an improvement that was directly paralleled by a decline in mirex, other organochlorine pesticides, and PCB's (Weseloh et al. 1979).

The fate of mirex in the environment and the associated transfer

mechanisms have not been well defined (NAS 1978). One of the more significant works on this subject area was that by Norstrom et al. (1978), who documented levels of mirex and its degradation products in herring gull eggs collected from Lake Ontario in 1977 (Table 7). They concluded that photodegradation was the only feasible mechanism for production of the degradation compounds, although mirex and its photoproducts rapidly become sequestered in the ecosystem and protected from further degradation. Norstrom et al. (1980) found mirex degradation products in herring gull eggs from all of the Great Lakes, and suggested that a high proportion of mirex and related compounds in herring gull eggs from Lakes Erie and Huron originated from Lake Ontario fish, whereas lower levels in eggs from Lakes Superior and Michigan originated from other sources. Mirex in sediments was considered an unlikely source because it was not being recycled into the ecosystem at an appreciable rate.

Biomagnification of mirex through food chains was investigated by Norstrom et al. (1978). Their basic assumption was that both herring gulls and coho salmon ate alewives (Alosa pseudoharengus) and rainbow smelt (Osmerus mordax). Mirex residues in these organisms, in parts per million fresh weight, were 4.4 in gull eggs, 0.23 in salmon muscle, 0.10 in salmon liver, and 0.09 in whole alewives and smelt retrieved from stomachs of salmon. Bioconcentration factors (BCF) from prey to predator ranged up to 50, and those from water to gull egg were estimated to be near 25 million (Table 8). Norstrom et al. (1978) indicated that salmon muscle and gull eggs are complementary indicators of organochlorine contamination in the Great Lakes.

Among Great Lakes fishes, the highest mirex value recorded was 1.39 ppm in whole eels (Anguilla sp.) collected from Lake Ontario (NAS 1978). This was substantially in excess of the tolerated limit of 0.3 ppm for human consumption (NAS 1978). High mirex values were also reported in chinook salmon (Oncorhynchus tshawytscha) and coho salmon from South Sandy Creek, a tributary of Lake Ontario, during autumn 1976; as a consequence, possession of all fish from that area was prohibited by the State of New York (Farr and Blake 1979). The significance of mirex residues in salmonid fishes is unclear. Skea et al. (1981), in laboratory studies with brook trout, showed that whole body residues of 6.3 ppm fish weight were not associated with adverse effects on growth or survival and speculated that long-lived species, such as the lake trout (Salvelinus namaycush), would probably continue to accumulate mirex in Lake Ontario as long as they were exposed, and may continue to contain residues for most of their lives, even after the source has been eliminated.

There was no widespread mirex contamination of urban environments near Lake Ontario as a result of Dechlorane use, although local contamination of the Lake Ontario area was high when compared with other Great Lake areas (NAS 1978). Among humans living in the Great Lakes area, there was great concern that mother's milk might be contaminated, owing to the high lipophilicity of mirex. Bush (1983) found mirex concentrations in mother's milk from residents of New York State to be 0.07 ppb in Albany, 0.12 in Oswego, and 0.16 in Rochester, confirming that mirex was present in human milk but that concentrations were sufficiently low to be of little toxicological

Table 7. Mirex and its degradation products in herring gull eggs collected from the Great Lakes in 1977 (after Norstrom et al. 1980).

Compound	Mirex concentration, in ppm fresh weight	Percent of samples containing compound
Mirex	2.58	66.7
8-monohydro mirex (photomirex)	0.95	24.5
10-monohydro mirex	0.199	5.1
$C_{10}Cl_{11}H$(III), possibly 9-monohydro mirex	0.077	2.0
$C_{10}Cl_{12}$(II)	0.039	1.0
2,8-dihydromirex	0.016	0.4
$C_{10}Cl_{10}H_2$(II), possibly 3,8-dihydromirex	0.011	0.3
Total	3.872	100

Table 8. Biomagnification of mirex in Great Lakes food chains (after Norstrom et al. 1978).

From	To	Bioconcentration factor
Water	Whole rainbow smelt or whole alewife	500,000
Water	Muscle of coho salmon	1,500,000
Water	Egg of herring gull	25,000,000
Alewife and smelt	Salmon muscle	2.6
Alewife and smelt	Gull egg	50.0

significance. It is noteworthy that none of the mothers had eaten Lake Ontario fish or any freshwater fish, and only a few had eaten marine fishes (Bush 1983). For a 5-kg infant consuming 500 g of milk daily, this amount would approximate a dietary intake of 0.01 ug mirex/kg body weight daily (Bush 1983) or about 1/10,000 of the lowest recorded dietary value causing delayed maturation in prairie voles (Shannon 1976). It is not known if a safety factor of 10,000 is sufficient to protect human health against delayed toxic effects of mirex, but it now appears reasonable to believe that it is.

MIREX IN OTHER GEOGRAPHIC AREAS

Mirex residues were determined in birds collected Nationwide or from large geographic areas of the United States; however, aside from the Southeast and the Great Lakes, concentrations were low, considered nonhazardous, and occurred in a relatively small proportion of the samples collected (Cain and Bunck 1983).

Among wings of mallards and American black ducks (Anas rubripes) collected from the four major flyways during 1976-77, mirex concentrations were highest and percent occurrence greatest in samples from the Atlantic Flyway: mallards, 50% occurrence, 0.14 ppm fresh weight; black ducks, 19% and 0.04 ppm (White 1979). Data for mallards collected from other flyways follow: Mississippi, 29% and 0.03 ppm; Central, 14% and 0.06 ppm; and Pacific 4% and 0.03 ppm (White 1979). Carcasses of several species of herons found dead or moribund Nationwide from 1966 to 1980 were analyzed for a variety of common organochlorine pesticides by Ohlendorf et al. (1981); they detected mirex in less than 15% of the carcasses, a comparatively low frequency, and only in nonhazardous concentrations. However, about 20% of all herons found dead or moribund had lethal or hazardous concentrations of dieldrin or DDT. In bald eagles (Haliaeetus leucocephalus) found dead Nationwide, elevated mirex levels were recorded in carcass lipids (24.0 ppm) and in fresh brain tissues (0.22 ppm) (Barbehenn and Reichel 1981). Among endangered species such as the bald eagle, it was determined that the most reliable indicator for assessing risk of organochlorine compounds was the ratio of carcass to brain residues on a lipid weight basis (Barbehenn and Reichel 1981). Wings from American woodcocks (Philohela minor) collected from 11 States in 1970-71 and 14 States in 1971-72 were analyzed for mirex and other compounds by McClane et al. (1978). Mirex residues in the 1971-72 wings showed the same geographical pattern of recovery as those observed in 1970-71: residues were highest in the Southern States and New Jersey, and lowest in the Northern and Midwestern States. Mirex residues were significantly lower in 1971-72 than in 1970-71. As judged by the analysis of wings of immature woodcocks in Louisiana, mirex residues were significantly lower in immatures than in adults: 2.48 ppm lipid weight vs. 6.20 ppm (McClane et al. 1978).

Mirex and other organochlorine compounds in eggs of anhingas (Anhinga anhingal) and 17 species of waders (including herons, egrets, bitterns, ibises, and storks) were measured in various locations throughout the Eastern United States during 1972 and 1973 (Ohlendorf et al. 1979). The highest mean concentration of 0.74 ppm mirex, range 0.19 to 2.5, was found in eggs of the

green heron (Butorides striatus) from the Savannah National Wildlife Refuge in South Carolina; a single egg of the cattle egret (Bubulucus ibis) analyzed from there contained 2.9 ppm mirex. However, the overall frequency of mirex occurrence was higher in eggs collected from the Great Lakes region (24%) than in those from the South Atlantic Coast (15.6%), inland areas (10.7%), Gulf Coast (4.4%), or North Atlantic region (3.2%).

Measurable mirex residues were detected in migratory birds collected from a variety of locations, including areas far from known sources or applications of mirex. For example, 22% of all eggs from 19 species of Alaskan seabirds collected in 1973-76 contained mirex. The highest concentration was 0.044 ppm in eggs of a fork-tailed storm petrel (Oceanodroma furcata) from the Barren Islands. Mirex residues were low compared with those of other organochlorine compounds (Ohlendorf et al. 1982). Eggs from the clapper rail (Rallus longirostris) collected in New Jersey from 1972-74 contained 0.16 to 0.45 ppm mirex (Klaas et al. 1980). Eggs from the greater black-backed gull (Larus marinus) collected from Appledore Island, Maine, in 1977 contained up to 0.26 ppm, but no mirex was detected in eggs of common eider (Somateria mollissima) or herring gull from the same area (Szaro et al. 1979). The greater black-backed gull is an active carnivore; 36-52% of its diet consists of small birds and mammals, whereas these items compose less than 1% in eider and herring gull diets. The higher mirex levels in black-backed gulls is attributed to its predatory feeding habits (Szaro et al. 1979). In New England, eggs of the black-crowned night-heron (Nycticorax nicticorax) contained between 0.28 and 0.66 ppm mirex wet weight in 1973; in 1979, this range was 0.11-0.37 ppm (Custer et al. 1983). Falcon eggs contained detectable mirex; levels were highest in the pigeon hawk (Falco columbarius) (0.25 ppm) and in the peregrine falcon (Falco peregrinus) (0.43 ppm), two species that feed on migratory birds or migrate to mirex-impacted areas (Kaiser 1978). Active mirex was also found in eggs of a cormorant (Phalacrocorax sp.) from the Bay of Fundy on the Atlantic coast; the suspected source of contamination was the southern wintering range (Kaiser 1978).

Mirex residues in 20 great horned owls (Bubo virginianus) found dead or dying in New York State in 1980-82 contained concentrations of mirex and PCB's higher than those reported for great horned owls elsewhere (Stone and Okoniewski 1983). Owls in "poor flesh" contained higher residues than those in "good flesh"; these values were 6.3 vs. 0.07 for brain, and 5.56 vs. 0.12 ppm for liver (Stone and Okoniewski 1983).

CURRENT RECOMMENDATIONS

Before the banning of mirex for all uses in 1978, the tolerance limits in food for human consumption were 0.1 ppm for eggs, milk, and fat of meat from cattle, goats, hogs, horses, poultry, and sheep, and 0.01 ppm for all other raw agricultural commodities (Waters et al. 1977; Buckler et al. 1981). Higher limits of 0.3 ppm mirex in fish and shellfish and 0.4 ppm in crabs were tolerated (NAS 1978). However, mirex concentrations as low as 0.1 ppm in diets of adult prairie voles were associated with delayed maturation of pups, and with significant delays in the attainment of various early development behaviors such as bar-holding ability, hind-limb placing, and negative geotaxis (Shannon 1976). It is not known whether or not prairie voles can serve as a model for protection of health of humans or various wildlife species. In the absence of supporting data, however, it seems prudent now to establish a dietary threshold of mirex at some level lower than 0.1 ppm. A maximum concentration of 0.01 ppm total dietary mirex, which is the current recommended level for most raw agricultural commodities, appears reasonable and conservative for the protection of fish, wildlife, and human health. This value could be modified as new data become available.

Although mirex is extremely persistent in the environment, recent data indicate that some degradation occurs and that some of the degradation products, such as photomirex, are biologically active. Accordingly, additional research is warranted on the fate and effects of mirex degradation products, with special emphasis on biomagnification through aquatic and terrestrial food chains.

Alternate means of controlling imported fire ants are under consideration. One approach has been to reduce the concentrations of active mirex in bait formulations from the current 0.3% to some lower, but effective, level. Paton and Miller (1980) demonstrated that mirex baits containing 0.07% mirex were effective in controlling Australian termites, reporting a 90% kill in 9 days; baits containing as little as 0.01% mirex were also reported effective, although termite mortality was delayed considerably. Waters et al. (1977) indicated that alternate chemical control agents, such as chloropyrifos, diazinon, dimethoate, or methyl bromide may be suitable and that nonbiocidal chemicals, such as various pheromones and hormones, which are capable of disrupting reproductive behavior of fire ants, are also under active consideration. Another proposal was to modify mirex chemically to a more water soluble and rapidly degradable product (Waters et al. 1977). The formulation Ferriamicide, which consisted of 0.05% mirex, ferrous chloride, and a small amount of long-chain alkyl amines, was formulated in baits during

1978-79 for ant control (Lowe 1982). Ferriamicide degraded within a few days after initial application; however, approval was revoked in 1980 when it was learned that the toxicity of various degradation products to mammals, especially that of photomirex, exceeded that of 4X bait formulations (Lowe 1982).

In 1980, the use of Amdro (tetrahydro-5,5-dimethyl-2 (1H)-pyrimidine) was conditionally approved by the U.S. Environmental Protection Agency (Lowe 1982). Amdro reportedly has good ant control properties, degrades rapidly in sunlight, has a biological half life of less than 24 hours, is nonmutagenic, and is relatively nontoxic to other than targeted species, except fish. Amdro was more acutely toxic than mirex to fish.

Mirex replacements should not manifest the properties that led to the discontinuance of mirex for all uses; namely, delayed mortality in aquatic and terrestrial fauna; numerous birth defects; tumor formation; histopathology; adverse effects on reproduction, early growth, and development; high biomagnification and persistence; disrupted energy metabolism; degradation into toxic metabolites; population alterations; and movement through aquatic and terrestrial environmental compartments. It is emphasized that mirex replacement compounds must be thoroughly tested before widespread application in the environment; if testing is incomplete, it is almost certain that the Nation's fish and wildlife resources will be adversely affected.

LITERATURE CITED

Ahrens, F.A., D.C. Dyer, and W.E. Lloyd. 1980. Mirex kinetics in chickens. J. Toxicol. Environ. Health 6:835-842.

Baker, M.F. 1964. Studies on possible effects of mirex bait on the bobwhite quail and birds. Proc. Annu. Conf. Southeast. Assoc. Fish Game Comm. 18: 153-159.

Barbehenn, K.R., and W.L. Reichel. 1981. Organochlorine concentrations in bald eagles: brain/body lipid relations and hazard evaluation. J. Toxicol. Environ. Health 8:325-330.

Barber, R.T., and S.M. Warlen. 1979. Organochlorine insecticide residues in deep sea fish from 2500 m in the Atlantic Ocean. Environ. Sci. Technol. 13:1146-1148.

Bell, M.A., R.A. Ewing, and G.A. Lutz. 1978. Reviews of the environmental effects of pollutants: I mirex and kepone. U.S. Environ. Protection Agency Rep. 600/1-78-013. 235 pp.

Bird, D.M., P.H. Tucker, G.A. Fox, and P.C. Lague. 1983. Synergistic effects of Aroclor^{R} 1254 and mirex on the semen characteristics of American kestrels. Arch. Environ. Contam. Toxicol. 12:633-640.

Buckler, D.R., A. Witt, Jr., F.L. Mayer, and J.N. Huckins. 1981. Acute and chronic effects of kepone and mirex on the fathead minnow. Trans. Am. Fish. Soc. 110:270-280.

Bush, B., J. Snow, S. Connor, L. Rueckeit, Y. Han, P. Dymerski, and D. Hilker. 1983. Mirex in human milk in upstate New York. Arch. Environ. Contam. Toxicol. 12:739-746.

Cain, B.W., and C.M. Bunck. 1983. Residues of organochlorine compounds in starlings (Sturnus vulgaris), 1979. Environ. Monitor. Assess. 3:161-172.

Chernoff, N., R.E. Linder, T.M. Scotti, E.H. Rogers, B.D. Carver, and R. Kavlock. 1979. Fetotoxicity and cataractogenicity of mirex in rats and mice with notes on kepone. Environ. Res. 18:257-269.

Chu, I., D.C. Villaneuve, V.E. Secours, V.E. Valli, and G.C. Becking. 1981. Effects of photomirex and mirex on reproduction in the rat. Toxicol. Appl. Pharmacol. 60:549-556.

Clark, D.R., Jr., and A.J. Krynitsky. 1980. Organochlorine residues in eggs of loggerhead and green sea turtles nesting at Merritt Island, Florida - July and August 1976. Pestic. Monitor. J. 14:7-10.

Cripe, C.R., and R.J. Livingston. 1977. Dynamics of mirex and its principal photoproducts in a simulated marsh system. Arch. Environ. Contam. Toxicol. 5:295-303.

Custer, T.W., C.M. Bunck, and T.E. Kaiser. 1983. Organochlorine residues in Atlantic coast black-crowned night-heron eggs, 1979. Colonial Waterbirds 6:160-167.

de la Cruz, A.A., and K.Y. Lue. 1978a. Mirex incorporation in estuarine animals, sediment, and water, Mississippi Gulf Coast - 1972-74. Pestic. Monitor. J. 12:40-42.

de la Cruz, A.A., and K.Y. Lue. 1978b. Mirex incorporation in the environment: in situ decomposition of fire ant bait and its effects on two soil macroarthropods. Arch. Environ. Contam. Toxicol. 7:47-61.

Ervin, M.G. 1982. The role of the pituitary-adrenal cortical-liver axis in mirex-induced adaptive liver hypertrophy. Ph.D. Thesis. Mississippi State Univ., Mississippi State. 84 pp.

Eversole, A.G. 1980. Effects of water-borne mirex on the survival and production of Macrobrachium rosenbergii (de Man). Water Resour. Res. Inst., Clemson Univ., Clemson SC, OWRT Proj. B-115-SC. 44 pp.

Farr, D.H., and L.M. Blake. 1979. A creel census of the salmonid fishery in South Sandy Creek, New York. N.Y. Fish Game J. 26:1-10.

Fujimori, K., I.K. Ho, H.M. Mehendale, and D.C. Villeneuve. 1983. Comparative toxicology of mirex, photomirex and chlordecone after oral administration to the mouse. Environ. Toxicol. Chem. 2:49-60.

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

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. 41:458-468.

Gilman, A.P., D.J. Hallett, G.A. Fox, L.J. Allan, W.J. Learning, and D.B. Peakall. 1978. Effects of injected organochlorines on naturally incubated herring gull eggs. J. Wildl. Manage. 42:484-493.

Grabowski, C.T. 1981. Plasma proteins and colloid osmotic pressure of blood of rat fetuses prenatally exposed to mirex. J. Toxicol. Environ. Health 7:705-714.

Hall, R.J., T.E. Kaiser, W.B. Robertson, Jr., and P.C. Patty. 1979. Organochlorine residues in eggs of the endangered American crocodile (Crocodylus acutus). Bull. Environ. Contam. Toxicol. 23:87-90.

Heath, R.G., J.W. Spann, E.F. Hill, and J.F. Kreitzer. 1972. Comparative dietary toxicities of pesticides to birds. U.S. Fish Wildl. Serv. Spec. Sci. Rep. - Wildl. 152. 57 pp.

Holcombe, C.M., and W.S. Parker. 1979. Mirex residues in eggs and livers of two long-lived reptiles (Chrysemys scripta and Terrapene carolina). Bull. Environ. Contam. Toxicol. 23:369-371.

Hollister, T.A., G.E. Walsh, and J. Forester. 1975. Mirex and marine unicellular algae: accumulation, population growth and oxygen evaluation. Bull. Environ. Contam. Toxicol. 14:753-759.

Holloman, M.E., B.R. Layton, M.V. Kennedy, and C.R. Swanson. 1975. Identification of the major thermal degradation products of the insecticide mirex. J. Agric. Food Chem. 23:1011-1012.

Howard, F.W., and A.D. Oliver. 1978. Arthropod populations in permanent pastures treated and untreated with mirex for red imported fire ant control. Environ. Entomol. 7:901-903.

Huckins, J.N., D.L. Stalling, J.D. Petty, D.R. Buckler, and B.T. Johnson. 1982. Fate of kepone and mirex in the aquatic environment. J. Agric. Food Chem. 30:1020-1027.

Hunter, R.G., J.H. Carroll, and J.C. Randolph. 1980. Organochlorine residues in fish of Lake Texoma, October 1979. Pestic. Monitor. J. 14:102-107.

Hyde, K.M. 1972. Studies of the responses of selected wildlife species to mirex bait exposure. Ph.D. Thesis, Louisiana State University, Baton Rouge. 170 pp.

Innes, J.R.M., B.M. Ulland, M.G. Valerio, L. Petrucelli, L. Fishbein, E.R. Hart, A.J. Pallotta, P.R. Bates, H.L. Falk, J.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.

Johnson, W.W., and M.T. Finley. 1980. Handbook of acute toxicity of chemicals to fish and aquatic invertebrates. Summaries of toxicity tests conducted at Columbia National Fisheries Research Laboratory, 1965-78. U.S. Fish Wildl. Serv. Resour. Publ. 137. 98 pp.

Jones, A.S., and C.S. Hodges. 1974. Persistence of mirex and its effects on soil microorganisms. J. Agric. Food Chem. 22:435-439.

Kaiser, K.L.E. 1978. The rise and fall of mirex. Environ. Sci. Technol. 12:520-528.

Kavlock, R.J., N. Chernoff, E. Rogers, D. Whitehouse, B. Carver, J. Gray, and K. Robinson. 1982. An analysis of fetotoxicity using biochemical endpoints of organ differentiation. Teratology 26:183-194.

Kendall, R.J., R. Noblet, J.D. Hair, and H.B. Jackson. 1977. Mirex residues in bobwhite quail after aerial application of bait for fire ant control, South Carolina - 1975-76. Pestic. Monitor. J. 11:64-68.

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.

Klaas, E.E., H.M. Ohlendorf, and E. Cromartie. 1980. Organochlorine residues and shell thicknesses in eggs of the clapper rail, common gallinule, purple gallinule, and limpkin (Class Aves), Eastern and Southern United States, 1972-74. Pestic. Monitor. J. 14:90-94.

Kobylinski, G.J., and R.J. Livingston. 1975. Movement of mirex from sediment and uptake by the hogchoker, Trinectes maculatus. Bull. Environ. Contam. Toxicol. 14:692-698.

Koenig, C.C. 1977. The effects of DDT and mirex alone and in combination on the reproduction of a salt marsh cyprinodont fish, Adinia xenica. Pages 357-376 in F.J. Vernberg, A. Calabrese, F.P. Thurberg, and W.B. Vernberg (eds.). Physiological responses of marine biota to pollutants. Academic Press, New York.

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

Leatherland, J.F., and R.A. Sonstegard. 1981. Effect of dietary mirex and PCB's on calcium and magnesium metabolism in rainbow trout, Salmo gairdneri and coho salmon, Oncorhynchus kisutch; a comparison with Great Lakes coho salmon. Comp. Biochem. Physiol. 69C:345-351.

Leatherland, J.F., R.A. Sonstegard, and M.V. Holdrient. 1979. Effect of dietary mirex and PCB's on hepatosomatic index, liver lipid, carcass lipid and PCB and mirex bioaccumulation in yearling coho salmon, Oncorhynchus kisutch. Comp. Biochem. Physiol. 63C:243-246.

Lee, J.H., C.E. Nash, and J.R. Sylvester. 1975. Effects of mirex and methoxychlor on striped mullet, Mugil cephalus L. U.S. Environ. Protection Agency Rep. EPA-660/3-75-015. 18 pp.

Lowe, J.I. 1982. Mirex, fire ants, and estuaries. Pages 63-70 in Proceedings of the workshop on agrichemicals and estuarine productivity. Duke Univ. Mar. Lab., Beaufort, NC. Sept. 18-19, 1980. U.S. Dep. Comm. NOAA/OMPA.

Ludke, J.L., M.T. Finley, and C. Lusk. 1971. Toxicity of mirex to crayfish, Procambarus blandingi. Bull. Environ. Contam. Toxicol. 6:89-96.

Lue, K.Y., and A. de la Cruz. 1978. Mirex incorporation in the environment: toxicity in Hydra. Bull. Environ. Contam. Toxicol. 19:412-416.

Markin, G.P. 1981. Translocation and fate of the insecticide mirex within a bahia grass pasture ecosystem. Environ. Pollut. 26A:227-241.

McArthur, M.L.B., G.A. Fox, D.B. Peakall, and B.J.R. Philogene. 1983. Ecological significance of behavioral and hormonal abnormalities in breeding ring doves fed an organochlorine mixture. Arch. Environ. Contam. Toxicol. 12:343-353.

McCorkle, F.M., J.E. Chambers, and J.D. Yarbrough. 1979. Tissue enzyme activities following exposure to dietary mirex in the channel catfish, Ictalurus punctatus. Environ. Pollut. 19:195-199.

McLane, M.A.R., E.H. Dustman, E.R. Clark, and D.L. Hughes. 1978. Organochlorine insecticide and polychlorinated biphenyl residues in woodcock wings, 1971- 72. Pestic. Monitor. J. 12:22-25.

Mehrle, P.M., F.L. Mayer, and D.R. Buckler. 1981. Kepone and mirex: effects on bone development and swim bladder composition in fathead minnows. Trans. Am. Fish. Soc. 110:638-643.

Menzie, C.M. 1978. Metabolism of pesticides. Update II. U.S. Fish Wildl. Serv. Spec. Sci. Rep. - Wildl. 212. 381 pp.

Minchew, C.D., R.N. Hunsinger, and R.C. Giles. 1980. Tissue distribution of mirex in adult crayfish (Procambarus clarki). Bull. Environ. Contam. Toxicol. 24:522-526.

Muncy, R.J., and A.D. Oliver. 1963. Toxicity of ten insecticides to the red crawfish, Procambarus clarki (Girard). Trans. Am. Fish. Soc. 92: 428-431.

Naber, E.C., and G.W. Ware. 1965. Effects of kepone and mirex on reproductive performance in the laying hen. Poult. Sci. 44:875-880.

NAS. 1978. Scientific and technical assessments of environmental pollutants. Kepone/mirex/hexachlorocyclopentadiene: an environmental assessment. Nat. Acad. Sci., Washington, DC. 73 pp.

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

Norstrom, R.J., D.J. Hallett, F.I. Onuska, and M.E. Comba. 1980. Mirex and its degradation products in Great Lakes herring gulls. Environ. Sci. Technol. 14:860-866.

Ohlendorf, H.M., E.E. Klaas, and T.E. Kaiser. 1979. Environmental pollutants and eggshell thickness: anhingas and wading birds in the Eastern United States. U.S. Fish Wildl. Serv., Spec. Sci. Rep. - Wildl. 216. 94 pp.

Ohlendorf, H.M., D.M. Swineford, and L.N. Locke. 1981. Organochlorine residues and mortality of herons. Pestic. Monitor. J. 14:125-135.

Ohlendorf, H.M., J.C. Bartonek, G.J. Divoky, E.E. Klaas, and A.J. Krynitsky. 1982. Organochlorine residues in eggs of Alaskan seabirds. U.S. Fish Wildl. Serv. Spec. Sci. Rep. - Wildl. 245. 1-41.

Paton, R., and L.R. Miller. 1980. Control of Mastotermes darwiniensis Froggatt (Isoptera: Mastotermitidae) with mirex baits. Aust. For. Res. 10:249-258.

Rogers, J.M. 1982. The perinatal cataractogenicity of the pesticide mirex: lens changes and related systemic effects. Ph.D. Thesis, Univ. Miami, Coral Gables. 100 pp.

Sanders, H.O., J. Huckins, B.T. Johnson, and D. Skaar. 1981. Biological effects of kepone and mirex in freshwater invertebrates. Arch. Environ. Contam. Toxicol. 10:531-539.

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

Schoor, W.P. 1979. Distribution of mirex in an experimental estuarine ecosystem. Bull. Environ. Contam. Toxicol. 21:315-321.

Schoor, W.P., and S.M. Newman. 1976. The effect of mirex on the burrowing activity of the lugworm (Arenicola cristata). Trans. Am. Fish. Soc. 105:700-703.

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

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. 318 pp.

Skaar, D.R., B.T. Johnson, J.R. Jones, and J.N. Huckins. 1981. Fate of kepone and mirex in a model aquatic environment: sediment, fish, and diet. Can. J. Fish. Aquat. Sci. 38:931-938.

Skea, J.C., H.J. Simonin, S. Jackling, and J. Symula. 1981. Accumulation and retention of mirex by brook trout fed a contaminated diet. Bull. Environ. Contam. Toxicol. 27:79-83.

Stickel, L.F. 1963. Wildlife studies, Patuxent Wildlife Research Center. Pages 77-115 in Pesticide-wildlife studies, 1963. A review of fish and wildlife investigations during the calendar year. U.S. Fish Wildl. Serv. Circ. 199.

Stone, W.B., and J.C. Okoniewski. 1983. Organochlorine toxicants in great horned owls from New York, 1981-82. Northeast. Environ. Sci. 2:1-7.

Summerlin, J.W., A.C.F. Hung, and S.B. Vinson. 1977. Residues in nontarget ants, species simplification and recovery of populations following aerial applications of mirex. Environ. Entomol. 6:193-197.

Szaro, R.C., N.C. Coon, and E. Kolbe. 1979. Pesticide and PCB of common eider, herring gull and great black-backed gull eggs. Bull. Environ. Contam. Toxicol. 22:394-399.

Tucker, R.K., and D.G. Crabtree. 1970. Handbook of toxicity of pesticides to wildlife. U.S. Fish Wildl. Serv. Resour. Publ. 84. 131 pp.

Ulland, B.M., N.P. Page, R.A. Squire, E.K. Weisburger, and R.L. Cypher. 1977. A carcinogenicity assay of mirex in Charles River CD rats. J. Natl. Cancer Inst. 58:133-140.

Van Valin, C.E., A.K. Andrews, and L.L. Eller. 1968. Some effects of mirex on two warm-water fishes. Trans. Am. Fish. Soc. 97:185-196.

Waters, E.M., J.E. Huff, and H.B. Gerstner. 1977. Mirex. An overview. Environ. Res. 14:212-222.

Weseloh, D.V., P. Mineau, and D.J. Hallett. 1979. Organochlorine contaminants and trends in reproduction in Great Lakes herring gulls, 1974-1978. Trans. N. Am. Wildl. Nat. Resour. Conf. 44:543-557.

Wheeler, W.B., D.P. Jouvenay, D.P. Wojcik, W.A. Banks, C.H. Van Middelem, C.S. Lofgren, S. Nesbitt, L. Williams, and R. Brown. 1977. Mirex residues in nontarget organisms after application of 10-5 bait for fire ant control, Northeast Florida - 1972-74. Pestic. Monitor. J. 11:146-156.

White, D.H. 1979. Nationwide residues of organochlorine compounds in wings of adult mallards and black ducks, 1976-77. Pestic. Monitor. J. 13:12-16.

Wolfe, J.L., and B.R. Norment. 1973. Accumulation of mirex residues in selected organisms after an aerial treatment, Mississippi, 1971-1972. Pestic. Monitor. J. 7:112-114.

Wolfe, J.L., R.J. Esher, K.M. Robinson, and J.D. Yarbrough. 1979. Lethal and reproductive effects of dietary mirex and DDT on old-field mice, Peromyscus polionotus. Bull. Environ. Contam. Toxicol. 21:397-402.

Yarbrough, J.D., J.E. Chambers, J.M. Grimley, E.G. Alley, M.M. Fang, J.T. Morrow, B.C. Ward, and J.D. Conroy. 1981. Comparative study of 8-monohydromirex and mirex toxicity in male rats. Toxicol. Appl. Pharmacol. 58:105-117.

Zitko, V. 1980. The uptake and excretion of mirex and dechloranes by juvenile Atlantic salmon. Chemosphere 9:73-78.

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Concern about the effects of mirex (a chlorinated hydrocarbon compound) on fish and wildlife, and other environmental consequences of its use, prompted the Environmental Protection Agency in 1978 to ban all further use. This report briefly reviews the evidence leading to the ban of mirex, with emphasis on afflicted natural resources, and provides current recommendations for the protection of fish and wildlife resources from residual effects of its use. Mirex was used extensively as a pesticide in the Southeast, and as a flame retardant in electronic components, plastics, and fabrics.

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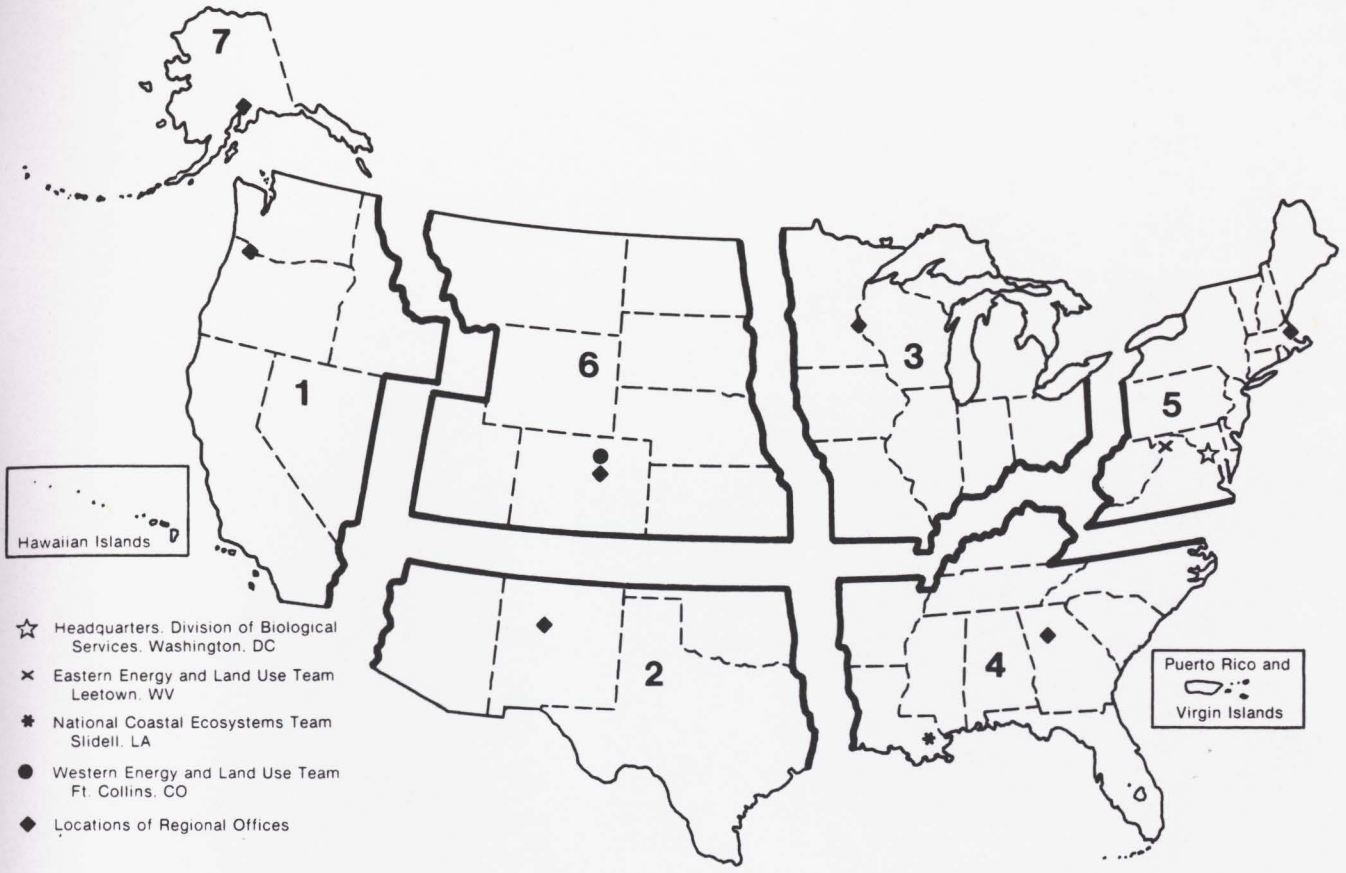

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