Endrin Toxicity and Distribution in Freshwater: A Review

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Forty-nine reports containing useful information about endrin are summarized herein. This review should be helpful in evaluating potential consequences of continued endrin use and its impact as an aquatic pollutant. These and other investigations are the basis for recommended maximal amounts of 2 pptr endrin in water if aquatic organisms are to be protected (Water Quality Criteria 1972).

ACUTE TOXICITY

Endrin is one of the most toxic of all economic poisons to fish except for cases wherein populations of a few species have developed resistance. Standard 96-h toxicity tests yield LC50's of 1 ppb (µg/l) or less in representative freshwater species: bluegills, rainbow trout, coho salmon, chinook salmon, fathead minnows, goldfish, guppies, mosquitofish, brook trout, and cutthroat trout (HENDERSON et al. 1959; KATZ and CHADWICK 1961, KATZ et al. 1961; MOUNT 1962; COPE 1965; MACEK et al. 1969; LINCER et al. 1970; and POST and SCHROEDER 1971). Increased temperature was shown to increase toxicity several-fold in bluegills and rainbow trout (KATZ and CHADWICK 1961; COPE 1965; MACEK et al. 1969). Small brook trout, cutthroat trout, and bluntnose minnows were reported to be slightly more susceptible than larger ones (MOUNT 1962; POST and SCHROEDER 1971). Tests continuously maintaining initial concentrations yielded lower LC50's i.e., indicated greater toxicity, than static tests, but both estimates were within X 10^1 range of magnitude (MOUNT 1962; LINCER et al. 1971). Differences in toxicity estimates from tests varying pH and water hardness were not appreciable.

Endrin is slightly less toxic to freshwater insects and crustacea than to fish. For example, SANDERS and co-workers reported 96-h LC50's of 0.25 to 0.76 ppb for three species of immature stoneflies (SANDERS and COPE 1968), 1.3 to 3 ppb for two species of Gammarus (SANDERS 1969, 1972), 1.8 ppb for immature damselflies (unpublished data, Fish-Pesticide Research Laboratory, Columbia, Missouri), 3.2 ppb for glass shrimp, 1.5 ppb for sowbugs (SANDERS 1972), 20 to 45 ppb (48-h LC50) for two species of Daphnia (SANDERS and COPE 1966), and 3.2 ppb for immature and 320 ppb for mature crayfish (SANDERS 1972). NEBEKER, GAUFIN, and co-workers obtained similar toxicity estimates (NEBEKER 1964; GAUFIN et al. 1965).

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SANDERS (1970) reported endrin LC50's of 180 and 120 ppb for tadpoles of the western chorus frog and Fowler's toad, respectively.

CHRONIC TOXICITY

MOUNT (1962) reported that bluntnose minnows and guppies tolerated continuous contact with endrin greater than 0.5 ppb in water only a few days, and that less than 50% exposed to 0.5 ppb survived 30 days. Exposed continuously to 0.4 ppb, about 65% survived longer than 30 days, whereas no mortality attributable to endrin occurred with exposures of 0.25 or 0.1 ppb. The effects of endrin were interpreted as noncumulative and, if fish survived endrin exposure for 30 days, they would not be seriously harmed. Symptoms at the onset of endrin intoxication were reversible.

GRANT and MEHRLE reported several potentially harmful physiologic manifestations of chronic dietary endrin in rainbow trout (GRANT and MEHRLE 1973) and goldfish (GRANT and MEHRLE 1970). Sublethal doses (4.3-145 µg/kg body wt/day, 0.215-7.25 ppm dry food) for 163 days produced abnormal serum composition and inhibited liver glycogenolysis in trout forced to swim for 1 h at a moderate rate. Endrin caused no mortalities but 7.25 ppm inhibited growth appreciably and produced behavioral symptoms of intoxication such as hypersensitivity. For example, slight movement or noise outside their tank caused them to swim into the tank walls with sufficient force to cause severe injury. Visceral fat of rainbow trout receiving 2.15-7.25 ppm endrin contained 4.8-8.7 ppm endrin residue. They concluded that the lowest "no-effect" dose was less than 14.5 µg/kg per day (0.715 ppm in food) because this dose impaired a homeostatic mechanism critical to survival of trout in nature. Rainbow trout receiving this dose did not respond normally to the moderate "loading" stress of exercise. Cortisol production was either blocked or it was cleared from circulation too rapidly to achieve normal values. Liver glycogenolysis for metabolic energy was inhibited in proportion to endrin dose, and loss of this glucogenic mechanism reflected the absence of the blood glucocorticoid. A dysfunction such as this may be interpreted as potentially harmful even though no mortalities occur. Information from controlled laboratory studies may be inherently weak in predicting impacts on natural populations. The favorable conditions under which experiments are carried out may lead to erroneously high estimates of "safe" levels of contamination.

Dietary endrin affected growth, gonad development, thyroid activity, serum characteristics, total and differential body fat, behavior, and mortality in goldfish. "Low doses" during 157 days (0.99-9.9 ppm) increased growth rates and body fat content. Higher doses (33-99 ppm) reduced growth rates and caused behavioral symptoms of endrin intoxication and mortality. These doses also decreased

thyroid cell height, gametogenesis, total body fat, and liver lipids. Fish receiving the highest dose did not regulate serum composition within viable limits.

ENDRIN UPTAKE

BRUNGS and MOUNT and MOUNT et al. demonstrated that endrin levels in blood of gizzard shad (BRUNGS and MOUNT 1967) and channel catfish (MOUNT et al. 1966) are directly correlated with lethal thresholds after exposure. When gizzard shad were exposed continuously to a range of endrin concentrations for 8 days or less, the blood level of endrin was a function of exposure time and exposure concentration. The endrin concentration in blood was greater than 0.1 ppm in gizzard shad that were killed, but less in survivors. Results of analyses of exposed and killed gizzard shad from the Lower Mississippi area indicated that the threshold value of 0.1 ppm is applicable to field situations with greater than 95% predictability. Similar exposures of channel catfish yielded a threshold value of 0.3 ppm endrin in blood.

BENNETT and DAY (1970) reported that endrin accumulated in bluegills to about 70 and 300 ppb after 24 h of continuous exposure to 0.2 and 2 ppb, respectively (biomagnification of about 350- and 150-fold).

Channel catfish receiving diets that contained 0.04-4 µg endrin/g dry food rapidly accumulated the pesticide (ARGYLE et al. 1973). Tissue levels in fish receiving 0.04, 0.4, and 4 ppm endrin were proportional to amounts in food: whole body residues after 20 days were 6, 37, and 310 ppb, respectively; average residues during 20-198 days were 8, 50, and 307 ppb, respectively. No effects on growth were observed and no mortality attributable to endrin occurred. Endrin residues in catfish given 4 ppm declined to 11 ppb after 28 days on an endrin-free diet and to nondetectable levels after 41 days. In catfish exposed to 0.5 ppb in water, mortalities began at tissue levels of 0.7 ppm (based on fresh wt) and reached 100% at levels near 1 ppm; endrin residues were 0.2 ppm on day 5 (400-fold magnification) and 0.38 ppm on day 19 (760-fold magnification). The fish exhibited hypersensitivity to sudden noise or movement from day 19 throughout the remainder of the experiment. Mortalities began on day 26, and nonmoribund fish had 0.72 ppm endrin residues (1440-fold magnification). On day 34, residue levels had increased to 1 ppm (2000-fold magnification) and did not change appreciably by day 41, when all fish had died. Catfish similarly exposed to 0.25 ppb endrin in water survived and exhibited no hypersensitivity. Uptake was highest on days 49 and 55 (average = 410 ppb); this value declined to 20 ppb after 13 days in endrin-free water and to below detectable limits after 23 days.

VANCE and DRUMMOND (1969) demonstrated that algae concentrate endrin manyfold and, in general, are more resistant to its toxic effects than organisms higher in the food web. Four species of algae, exposed to endrin in water at 1 ppm (0.02 and 0.5 ppm were also tested) for 7 days, accumulated residues of 200, 222, 156, and 140 mg/l. This represents about 140- to 222-fold magnification.

PRIESTER (1965) reported uptake of 0.46 ppb endrin by Daphnia, exposed 14 days to 0.5 ppb in water. Biotransfer of endrin from a single feeding of 400 to 600 of these organisms to individual fathead minnows was below detectable limits of his endrin assay; repeated feedings were not attempted. He reported endrin LC50's of 368 ppb (96 h) for the daphnid, and 0.59 ppb (96 h) for fathead minnows.

RESISTANCE IN FRESHWATER ORGANISMS

Endrin resistance in populations of fish from areas of heavy endrin contamination is well documented. The list includes black bullheads (FERGUSON et al. 1965), yellow bullheads (FERGUSON and BINGHAM 1966), golden shiners (FERGUSON et al. 1964; LUDKE et al. 1968), mosquitofish (BURKE and FERGUSON 1969), bluegills (FERGUSON et al. 1964), and green sunfish (FINLEY et al. 1970). For example, mosquitofish, green sunfish, and yellow bullheads have 36-h endrin LC50's as high as 1500, 200, and 75 ppb, respectively. Black bullheads were reported to be only moderately resistant. But mosquitofish are up to 1500-fold more resistant than other natural populations. At present, these populations are known only from contaminated areas, and levels of resistance generally reflect the degree of contamination. Resistance is generally highest to the most chemically stable pesticides, including the cyclodienes (FERGUSON 1968). It is genetically based but the genetic expression may be increased body lipids that provide a protective sink (FABACHER and CHAMBERS 1971). Third generation progeny of resistant fish reared in an endrin-free environment retained resistance. Endrin resistant fishes tolerate and survive massive amounts of endrin in tissues. For example, living resistant mosquitofish were reported to contain more than 1000 ppm endrin after laboratory exposure, whereas less than 10 ppm was recovered from endrin-killed susceptible fish. Resistant mosquitofish contained more than 200 ppm after a 2-week exposure to 500 ppb endrin (biomagnification of 400-fold) (FERGUSON et al. 1966).

Feeding experiments with resistant fish demonstrate potential hazards of accumulating residues. Resistant green sunfish given live mosquitofish containing 25 ppm endrin survived, but susceptible green sunfish died within 1 day or less, even though they regurgitated the mosquitofish (FERGUSON et al. 1967). Resistance and toleration of stored residues in fish-forage invertebrates (NAQVI

and FERGUSON 1968; FERGUSON et al. 1965) present an additional potential hazard, especially to top carnivores (FERGUSON et al. 1964; ROSATO and FERGUSON 1968). FERGUSON (1967) put the potential ecologic consequences of endrin resistance in perspective by concluding that resistance confers survival advantage to certain aquatic species in heavily contaminated areas. But in these areas, the number of species is restricted and top carnivores are not found associated with resistant forage fish. This potential hazard does not exclude man. Resistant fish are being eaten by people in the Delta area of Mississippi. When living green sunfish were exposed to endrin at rates simulating heavy contamination and rapid residue accumulation, and then prepared as if for cooking, the edible portion contained 11 to 26 ppm endrin (FERGUSON 1968).

FACTORS AFFECTING ENDRIN TOXICITY

BRUNGS and BAILEY (1967) reported on the influence of suspended solids (continuous flow conditions with montmorillonite clay, Brookston silty clay loam, and activated carbon) on endrin toxicity to fathead minnows. Only carbon increased the LC50's. It adsorbed approximately 95% of the endrin; about 5% of the total endrin contributed to toxicity.

EICHELBERGER and LICHTENBERG (1972) determined that no detectable degradation occurred when 10 ppb endrin in raw river water was aged for 8 weeks.

BRIDGES (1961) reported on the distribution of endrin in water, mud, vegetation, and fish after a pond received contamination from aerial spray (6 oz/acre). Four days after the aerial spray, large numbers of dead yellow perch, pumpkinseed sunfish, bluegills, black crappies, largemouth bass, and carp were observed. Endrin concentrations in the water were 40 ppb on day 4, 4 ppb on day 22, and nondetectable on day 30. Concentrations in mud were highest (800 ppb) on day 50, declined (to 350 ppb) on day 58, and were nil on day 72. Endrin in vegetation was highest (550 ppb) on day 13, persisted (370 ppb) by day 41, and was nil by day 50. Bluegills accumulated 1 ppm by day 22 but only traces were observed by day 72.

FERGUSON et al. (1965) demonstrated that muds from areas receiving runoff from treated cotton fields contained lethal amounts of endrin. Acetone extracts from muds killed fish, but under the test conditions employed, the mud did not release toxic quantities into standing water. Physicochemical factors favoring desorption of endrin from natural muds and silts are largely unknown. Slightly acid conditions were shown to decrease association of DDT and natural clays.

LEIGH (1969) concluded from laboratory studies that endrin was extremely resistant to degradation.

ENVIRONMENTAL DISTRIBUTION OF ENDRIN

GODSIL and JOHNSON (1968) reported on a survey of seasonal occurrence of pesticides in the Tule Lake National Wildlife Refuge, California, after kills of fish-eating birds. Residues in biota reflected seasonal agricultural uses of pesticides and irrigation water. Endrin concentrations in water entering the refuge were highest (50-70 ppb) in summer and declined to nondetectable levels in winter. Similarly, maximal amounts occurred in the biota during summer and declined in winter: endrin varied from winter to summer from 1.3 to 58 ppb in suspended material; about 2 to 12 ppb in vascular plants; 2 to 22 ppb in algae; 4 to 198 ppb in tui chubs; and 2 to 34 ppb in clams.

Endrin pollution has been reported extensively over the past decade. Appreciable amounts have been reported in potable, finished drinking water from the Missouri and Mississippi Rivers (SCHAFER et al. 1969), from estuaries (ROWE et al. 1971), and surface waters (LAUER et al. 1966). Also, massive fish kills typically follow accidental spills (LAUER et al. 1966) or irresponsible discharges (MOUNT and PUTNICKI 1966) of endrin into lakes or rivers.

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