Practicality of Endrin as a Fish Toxicant[1]

H. E. McReynolds, U. S. Forest Service

Abstract

When fisheries bioassays documented the lethality of endrin to fish at almost inconceivably low concentrations, the question of the suitability of this pesticide as a replacement for rotenone naturally followed. Fisheries workers immediately noted two characteristics of this chemical that would make it superior to rotenone as a piscicide: (1) it was cheaper; and (2) it was more persistent, thereby more likely to effect a complete kill.

However, many other characteristics and effects of endrin as a fish toxicant were unknown or poorly understood. Experiments were set up in selected southern Indiana ponds to test this chlorinated hydrocarbon. Laboratory bioassays with test fish established an LC_{50} (Lethal Concentration, 50%) or TL_m (Median Tolerance Limit) of about 1.3 parts per billion. Field tests indicated highly disparate LC's. After testing in a diversity of aquatic situations, it was postulated that the amount of suspended particulate matter exerted a considerable effect on the lethality of the chemical in field tests. It was surmised that the mechanism of lowered toxicity in more turbid situations was the adsorption of the chemical to organic (and possibly inorganic) suspensoids. Verification of these postulations was not pursued, since other endrin experiments of a public health nature were beginning to point out the potential dangers of endrin in potable water supplies. In addition, the duration of toxicity of the pesticide showed evidence of extending beyond that desired in the ideal fish toxicant, especially in the sediments. Later, more thorough experiments have generally tended to establish the validity of these surmisals. It was concluded that despite the economy and kill efficiency of endrin, other undesirable and dangerous characteristics outweighed the kill-cost attributes.

Introduction

At the time of these experiments, fisheries biologists were—and still are—looking for a cheaper, more effective replacement for rotenone as a fish toxicant. The use of piscicides for eliminating undesirable fish populations has become an important tool in fish management. While rotenone, a ketone of botanical origin, has certain advantages of low toxicity to mammals and low-level of danger to the applicator, it also has a number of limitations that fisheries technicians have found frustrating (difficulty in killing resistant species such as bullhead catfish; the problem of getting effective vertical dispersal through metalimnetic barriers; relatively quick oxidation to non-toxic levels; high toxicity for many fish food organisms; and relatively high cost).

When biologists began to notice that organochlorine pesticides were displaying piscicidal potencies many times greater than rotenone, first reactions were dismay and apprehension. After the first shock waves had passed, however, some biologists began to wonder if this chemical lightning might be harnessed for fish management use. Toxaphene, a chlorinated camphene, was the first to receive extensive testing as a fish toxicant, and was even used—somewhat surreptitously—in a commercial fish toxicant product. Unfortunately, a toxicity of extended duration (up to 3 years in Michigan lakes) made its use unfeasible in

[1] This study was supported in part by Federal Aid to Fish Restoration funds (Project F-4-R-8, Indiana).

most situations. Interest turned to other members of the generically similar chlorinated hydrocarbon pesticides. While Canadian biologists began testing thiodan, some American fisheries people became intrigued with endrin, an insecticide and rodenticide with toxicity thresholds for fish measured in low or fractioned parts per *billion!* This paper reports one of these earlier experiments with endrin.

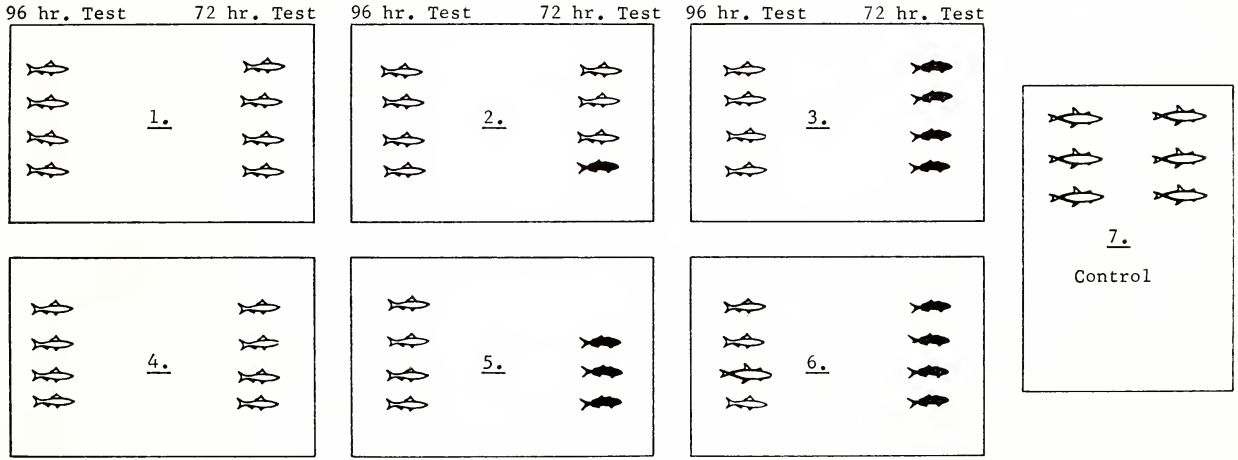
Laboratory Aquaria Tests

Methods

The preliminary tests were conducted in seven 15-gallon metal frame glass aquaria (Fig. 1) having aeration and filtration systems. Diluent waters were obtained from the two ponds on the Crosley State Fish and Game Area which were to be used for the field tests. These are designated merely as Pond 1 and Pond 2 in this paper. Two species of centrarchids were used as test animals: the bluegill sunfish (*Lepomis m. macrochirus* Rafinesque), and the redear sunfish (*Lepomis microlophus* [Gunther]). Fish were obtained by seining and were held in the aquaria for 10 days before testing began. The fish ranged in size from approximately 1 inch to 3 inches. Although this range does not meet size-uniformity recommendations (12) no grading was done since these preliminaries were merely exploratory in nature. Actually, the larger and smaller fish were purposely combined in each aquarium to observe any difference in size-susceptibility. Two bluegills and 2 redears were placed in each of 6 aquaria and 10 fish, a mixture of both species, were held in the 7th aquarium as controls.

The endrin was obtained as a 75% wettable powder and weighed on an analytical balance in the following amounts: 0.19, 0.38, 0.75, and 1.50 g. These weights are for the active ingredient and assume that the manufacturer's assay (75%) is correct. The validity of this assumption was not investigated.

The minute concentrations at which these tests must be conducted present a problem in dilution. It was decided that 10 gal of water would be used in the aquaria, and calculations of dosage began from there. To get the lowest concentration, 0.26 parts per billion (ppb), it was necessary to mix 0.19 g active ingredient of the endrin powder with 5 gal of water, take 1 g of this solution and introduce this amount into the 10 gal of aquarium water. Addition of another 0.19 g of endrin to the original solution gave the 0.52 ppb dosage, and so on up the scale. The test solution was drawn off by pipette from the center of the 5 gal bottle after extended agitation. A shell vial was weighed on a triple beam balance having a sensitivity of 0.01 g, and the weight recorded. The balance was set up one more gram and the test solution was pipetted into the vial until the correct weight was reached. The contents of the vial were then dripped into the aquarium and the aquarium water stirred with a clean glass rod. Hourly checks were made by the study leader or biologist aides, and notes were made on the reactions of the fish. Observations were made from 8:00 AM until 4:30 PM (the work



Aquarium No.	Test Water	Dosage	Result of original dosage (96 hr. Test)	Later additions[1] and results (72 hr. Test)
1.	Pond No. 1	0.26 ppb	No reaction	No additions
2.	Pond No. 1	0.52 ppb	No reaction	1 ppb; 25% survival
3.	Pond No. 1	1.00 ppb	No reaction	1 ppb; no survival
4.	Pond No. 2	0.26 ppb	No reaction	No additions
5.	Pond No. 2	0.52 ppb	No reaction	1 ppb; no survival
6.	Pond No. 2	1.00 ppb	No reaction	2 ppb; no survival
7.	Pond No. 2	None (control)	No dose; complete survival	No dose; complete survival

= Survival = Mortality

[1] Refers to amount added, not concentration at this time. Sum of original and additional dosage would give the maximum concentration present (assuming no dissipation of original concentration).

FIGURE 1. *Vertical diagrammatic view of aquaria arrangement with notes on dosages, waters, durations of tests, and results of both original and additional dosages.*

day) each day from Monday through Saturday. Water temperatures were not controlled during these tests and ranged between 63° F and 75° F.

Results

Concentrations of 0.26 ppb and 0.52 ppb were run concurrently for a period of 96 hours, and at neither level were there any mortalities. There were no indications of even slight distress in any fish. These fish were observed for several weeks after completion of the 96-hour test, and no reaction to the chemical was observed. The next dosage tested was 1.0 ppb. During a 48-hour test no mortalities occurred and the fish showed no observable evidence of discomfort. At the termination of the 48-hour test, another 1.0 ppb was added to Aquarium 3, and the concentration in Aquarium 6 was augmented by 2.0 ppb. Assuming that the original 1.0 ppb dosage had not been appreciably dissipated during the previous 48 hours, concentrations at this time were upwards to 2.0 ppb in Aquarium 3 and 3.0 ppb in Aquarium 6. After 18 hours, the first indication of distress was noted. A small bluegill in Aquarium 6 showed the initial signs of disquietude. Symptoms of distress in this fish became more intense at each 30-min observation period, and 90 min after the first manifestations the fish was dead. Soon afterward, other fish in both aquaria began to show reactions to the test solution, and 42 hours after increasing the concentration all fish in both aquaria were dead.

The physiological reactions were similar to those described by Public Health Service biologists (14). An increase in respiration and fin movements was first noted. Later, fish exhibited annoyed wanderings to the surface or into bottom corners of aquaria. As distress became more intense, darting movements were noted, with fish occasionally breaking the surface and in one instance jumping clear of the aquarium. Later, a loss of hydrostatic equilibrium was evidenced by fish swimming abruptly to the surface and settling slowly to the bottom. At this time, the test animals were oriented at an angle to the horizontal. The body axis was a line diverging from the horizontal by 20°-35°, caudal end down. Fish near expiration showed little control of their movements; some were attempting feeble swimming efforts while on their sides or backs.

These results indicated that the toxicity threshold of endrin lay between 1.0 ppb and 1.52 ppb in these experimental waters and with these test animals. However, since the first dosage of 1.0 ppb was followed for only 48 hours, there was a possibility that a more extended test period would have resulted in some mortality. Another test at this level (1.0 ppb) was run for 72 hours but no mortalities resulted. Again, 1.0 ppb was added to each aquarium and once more no fish survived an additional 72-hour test. From these exploratory tests, it was assumed that the toxicity threshold was a value between 1.0 ppb and 2.0 ppb, indicating that the field tests should begin with a concentration of 1.0 ppb (apparently a sublethal dose) and continue upward until concentration at which all test fish were killed was reached (Lethal Concentration, 100%, or simply LC_{100}). It should be noted here that where a chemical's

use as a piscicide is concerned, the LC_{100} becomes a more important figure than the more commonly used LC_{50} (also commonly termed LD_{50} Lethal Dosage, 50% or TL_m). This stems from the fact that many chemicals show a non-linear regression from the concentration producing total extermination to those of sublethal levels (LC_{100} to LC_0). Thus, where the economic feasibility of a product is one of the important criteria, it becomes of greater concern to establish this level by direct testing rather than to estimate it by projection from intermediate levels (LC_{50}).

Later checks of the 2.0 ppb solutions were made to determine if they were still toxic. Two fish were placed in each of 2 aquaria 15 days after the solutions were made. All fish failed to survive until the 19th day (96-hour test) indicating that dissipation had not progressed to any great degree.

An interesting sidelight which supports results of a Japanese study (19) was the presence of cladocerans and planarians in the 3.0 ppb solution. Two days after all fish had been killed, these organisms were noted in large numbers on the sides of the aquarium.

Crosley Pond Tests

During 1960, the endrin study suffered from a low priority, and there was no time available for this project until mid-October. On October 13, a small pond on the Crosley State Fish and Game Area was treated with endrin calculated to produce a concentration of 1.0 ppb. After 120 hours without any mortality of test fish, another 1.0 ppb of endrin was added. This presumably raised the endrin concentration in the pond to 2.0 ppb, a level that had killed all test fish in laboratory experiments.

After 144 hours of testing at this level, there were no mortalities, nor even discomfort among test fish. It was theorized that this puzzling situation resulted from lower temperatures and/or higher turbidities than had been present in laboratory tests. This study was not continued during 1961.

Driftwood Pond Tests

In 1962 the study was given a higher priority and the testing site was moved to the Driftwood Farm Pond Experiment Station where four ponds were available. It was felt that the common water source, depth, basin morphometry, and soil types, and similar thermal gradients would eliminate some of the variables encountered in the heterolimnetic environs of dissimilar farm ponds.

Three ponds were used for testing and the fourth served as a control. The study here was designed to bracket the minimum LC_{100} with a concentration below the LC_{100} (at 1 ppb) and one calculated to give a total kill (4 ppb). These figures were based on the preliminary lab tests which showed LC_{50} of 1 ppb or more, and no mortality in the Crosley pond at an assumed 2 ppb level. It was reasoned that 4 ppb in waters of higher temperature would effect a complete kill. A third pond was treated with a tremendous dose to observe the duration of toxicity in

cases of miscalculated dosages. Treatment in this pond was at 46 ppb. No treatment was made on the control pond. Ponds ranged in size from 0.62 acres to 1.37 acres. Actual testing began in mid-September when water temperatures were near 16° C.

Pond 1

Pond 1 (0.62 acres) received the initial treatment. This pond was sprayed with a solution calculated to give a 4 ppb concentration of endrin (active ingredient). Twenty-four hours previous to the spraying of the toxicant, 2 test cages each containing 10 bluegills were placed in the pond. One cage was placed in water of 18-inch depth, the other in 5 feet of water. Test cages were approximately 36″ × 36″ × 22″. Since bluegills used as test fish were from adjacent ponds, the 24-hour acclimation period seemed sufficient.

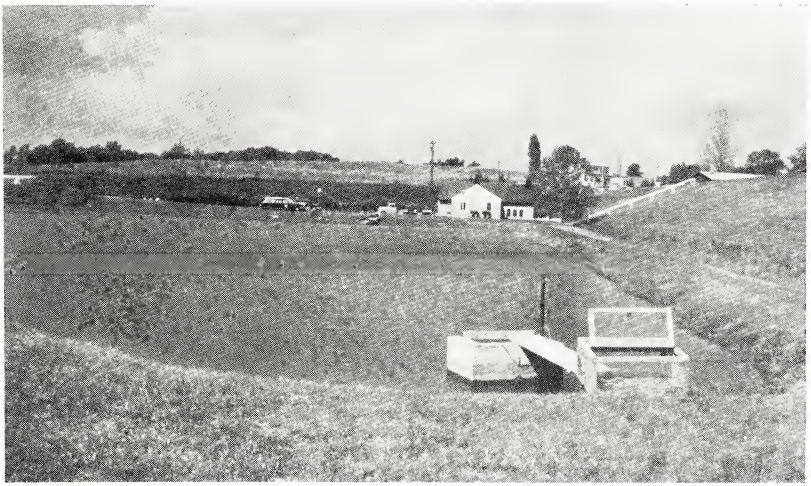


FIGURE 2. *One of the endrin test ponds at the Driftwood Station showing deepwater test cage raised for examination. Dead test fish are visible in cage.*

Test cages were checked periodically to observe fish reactions, but for the purpose of recording, data were considered only on a 24-hour interval basis (i.e., fish were recorded as having died only at the termination of a 24-hour test period). Although the last fish in a test group may actually have expired at 42 hours, the LC_{100} was recorded as having occurred at the 48-hour level. (There seems to be little justification for carrying the recording of results to a precision beyond the sophistication of the basic technique itself.)

After 24 hours, 2 fish in the deep water cage had died, but there were no mortalities in the shallow water cage although 2 bluegills were in extreme distress. At the 48-hour check, all fish in the deep cage were dead and there was but one survivor in the shallow cage. The following day (72-hour test) the last fish had expired.

Pond 2

The same procedure was followed in Pond 2 testings as had been used in Pond 1 (a deep and a shallow cage each with 10 test fish). This pond (1.07 acres) was sprayed with a dosage calculated to give a concentration of 1 ppb of endrin (active ingredient). After introduction of the toxicant, no mortalities were recorded during the first 4 days (the 24-, 48-, 72-, and 96-hour tests), nor was any distress noted in any of the test fish during this time. In view of subsequent results, the significance of this lack of mortality during the 96-hour period will be discussed in some detail later.

On the fifth day, one bluegill showed the first signs of agitation and symptoms of distress became more intense. Before the end of the 120-hour test, this fish had become the first mortality in Pond 2. The following day another fish died (144-hour test) and sporadic mortalities continued until the last fish expired 3 weeks after introduction of the toxicant (504-hour test). These events, although instructive, were somewhat disconcerting since this level (projected from aquaria and Crosley Pond tests) was intended to be a sublethal concentration. It seemed untenable to suspect disease, parasitism, debility from feeding interference, etc. as the causative agent of the mortalities. Observations showed no external parasites or disease and body forms of the dead fish displayed no noticeable emaciation. In addition, during this period, the control fish in an adjacent pond had suffered no mortalities under very similar, if not identical, conditions—with the exception of the test toxicant, of course. I was forced to the conclusion that the LC_{100} concentration in these Driftwood waters was somewhat lower than it had been in the Crosley pond waters. The major differences in the ponds were size, shading, temperature, and transparency. The Driftwood ponds were larger, unshaded, warmer, and had greater clarity. All of these physical factors have been suspect in influencing the toxicity of various chlorinated hydrocarbons in solution. These will be discussed later at some length.

Pond 4

Pond 4 was a 1.23-acre pond used for testing the residual effect of an extremely high dosage. This pond was sprayed with a quantity of endrin calulated to give a concentration of 46 ppb. The same procedure (two cages with 10 bluegills each) that had been used in Ponds 1 and 2 was used in Pond 4.

At the termination of the 24-hour test (8:00 AM, September 18, 1961) all fish were dead. Until the pond froze over, test fish (usually at 2-week intervals) were placed in the cages, and each time died within the 24-hour period. After ice cover sealed the pond, no tests were made until a warmer period opened it in January. At this time, test fish again died within a 24-hour period. It was decided to replace the water and see if the residual endrin in the sediments would produce a toxicity in previously-uncontaminated water.

The pond was drained February 1-3, 1962, and allowed to stand idle for about 3 weeks. Then it was refilled with uncontaminated water

(March 1) and allowed to stand until March 13. On this date, a new group of 10 bluegills was placed in a single cage in the pond. Two of these subsequently escaped from encagement and one was later found dead along the pond edge. Of the eight fish remaining in the test cage, the first mortalities occurred on March 26. At this time, four fish were found dead. One more bluegill was dead on March 28, another on March 29, and the final two expired on April 1.

The pond was then drained a second time, and refilled on April 13. Nine test fish were put in the cage on April 25. The first mortality was recorded on May 2 and by May 9 all remaining test fish were dead. The pond was immediately drained again and had been refilled on May 15. Ten new fish were placed in the pond on May 20, and on June 2 the first death was recorded. Another died on June 5, and two more on June 7. Although five fish were still alive, it was obvious that a long-term lethal toxicity still existed.

Once more the pond was drained and refilled, and was ready for the continuation of testing by June 15. Fish again were put in the cage, and these fish showed no mortality in a subsequent month of observations. Tests were suspended at this time since the pond appeared to have become non-toxic and was needed for another study. Apparently, the dissipation of the toxicant was finally complete since the other fish stocked in this test pond were not affected.

It should be stressed that the water source for these ponds was piped by gravity flow from adjacent Starve Hollow Lake which is located upstream from the station. Ponds drained into a small creek which also drained the lake. The source water was entirely uncontaminated by endrin and no other insecticides were known to have been used in the lake itself. In addition, no fish kills were noted in the lake. Thus it appears that the toxicity was of an autochthonous nature, and is assumed to have been recycled or brought into re-solution from the contaminated sediments.

Discussion

Only in 1962 were the endrin tests given precedence over other duties. By 1962 it was becoming apparent from personal experiences and those of other investigators that this chemical was too dangerous for use as a piscicide except in extremely isolated instances.

As data accumulated from various studies of endrin, certain problems became apparent. It has been noted that other chlorinated hydrocarbons were present in fish flesh at higher levels than were present in solution (6, 7, 8, 18, 20). This accumulative effect is indicated for endrin also in Bridges' (3) study of a Colorado fish kill, and by implication (22) in aquaria tests. The concentrating potential of lower forms in the food chain is well documented for hydrocarbons: moss (15); *Potamogeton* (8); vegetation (3, 4); earthworms (2); and plankton, frogs and fish (18). It is generally assumed that these accumulations in

fish flesh are dietary translocations of residues through ingestion of contaminated food items. The role of direct absorption of the materials from the aquatic medium through integumentary tissues has not been as convincingly demonstrated, although there are data (22) that imply direct absorption of endrin. Hoss (17) has effectively shown that both pathways (absorption and ingestion) may be important in his studies of zinc^{65} accumulations in the flounder, and Williams and Pickering (28) have shown that bluegills accumulate cesium^{137} and strontium^{85} by both ingestion and absorption. Though not conclusive, these data are insinuative.

Regardless of whether the accumulative deposition in fish flesh results from ingestion or from absorption, it forces us to re-evaluate our reliance on short term bioassays (24- to 96-hour tests) where chronic exposures to chlorinated hydrocarbons are involved. One of the two major points arising from this present study is that short term bioassays are insecure evidence of non-toxic levels in situations of chronic or long-persisting contamination and/or with materials accumulatively stored by metabolic processes. The need for studies of the effects of long term exposure has been pointed out (14), but there is a rather dramatic example in the study of Driftwood Pond 2. Had testing in this pond been suspended after a common 48-hour, 72-hour, or even 96-hour period, the assumption of a sublethal level would have appeared valid. However, the first mortality occurred on the 5th day and all test fish had expired within 3 weeks from time of exposure. There were no mortalities among the control fish during this period. Therefore, one must carefully examine over extended periods those agents of an accumulative or an insidious nature to ascertain that seeming sublethality is not actually slow response. This is particularly true as testing approaches the toxicity threshold.

The second point of interest deduced from this and other studies involves the variance in toxic limits for fish established by several workers (Table 1). Katz and Chadwick (22) obtained a 96-hour LC_{50} (their TL_m) of 0.27 ppb for coho salmon, and 0.60 ppb for bluegills. Henderson *et al.* (14) established a 96-hour LC_{50} (their TL_m) of 0.44 ppb for bluegills in hard water using the emulsible concentrate. In the present study, I failed to get any kill on test fish in the Crosley test at an assumed 2 ppb. On the other hand, there was a complete kill of test fish over a 3-week period in one Driftwood pond at a 1 ppb concentration. A resume of Michigan lake and stream rehabilitations (16) notes a survival of some fish (*Fundulus*) at 8 ppb. Bridges (3) points to a partial survival of largemouth bass, bluegills, pumpkinseed sunfish, and black crappies in a Colorado pond adjacent to a beet field which had been sprayed with an emulsible formulation of endrin. At the time of investigation (4 days after the spraying), water analyses indicated a concentration of 40 ppb of endrin. Fish were not dying at this time. Bridges, noting the disparity of his results with those of the USPHS team, points a suspicious finger at the 9.1 pH of the Colorado pond and the fact that the highest pH tested by Henderson's group (14) was 8.2.

Temperature has been shown to exert a tremendous effect on the toxicity of endrin. The studies of a Japanese group (19) observed that endrin toxicity to fish increased at higher temperatures, and Katz and Chadwick (22) found 96-hour LC_{50} levels for bluegills of 8.25 ppb at 1.0°-4.5°C and 0.33 ppb at 25°C. The data of the latter study indicate a 25-fold increase in toxicity with an increase of 20.5°-24.0°C.

There are wide differences between the toxic thresholds of endrin, DDT and dieldrin in diverse animal groups (1, 5, 9, 10, 13, 14). An instance of this is obvious in Table 1. Species susceptibility to endrin has been pointed out by numerous investigators (14, 21, 22, 25, 27). Even within a particular species there is evidence that susceptibility to endrin changes with age, with embryonic stages being more resistant than larval stages (19).

TABLE 1. *The varying toxicity levels of three chlorinated hydrocarbons in their effect on several classes of organisms (measured in mg/kg of body weight or in ppb of active ingredient).*

Organism	DDT	Dieldrin	Endrin
Estimated fatal dose for a man weighing 70 kg	30 g (5, 13) (= an estimated 428 mg/kg)	5 g (13) (= an estimated 71 mg/kg)	?
Pheasants (LD_{50})	300 mg/kg (9, 10)	50 mg/kg 9, 10)	14 mg/kg (9, 10)
Bluegills (LC_{50} for emulsible concentrate)	8.8 ppb (14)	9.1 ppb (14)	0.44 ppb (14)
Daphnia (50-hour Immobilization test)	1.4 ppb (1)	330 ppb (1)	352 ppb (1)

It has been pointed out that the tolerance of some species is different in different volumes of water even though the concentration of the active ingredient is the same. This so-called volume effect has been noted for DDT (24) and for endrin (22). This increase in toxicity in greater volumes of the same concentration as smaller volumes can be logically correlated with the previously discussed accumulative nature of this hydrocarbon. In addition to fish as accumulators of pesticidal hydrocarbons, studies have indicated the importance of macrophytes as concentrators of these chemically related insecticides (4, 8). Bridges and his co-workers (4), by use of isotope-labeled DDT (C_{14}-DDT), have found that storage of this relative of endrin is about ⅓ external (i.e., adsorption to external portions of the plant) and ⅔ internal (absorption into the tissues). This could be presumably true for endrin also. If such a presumption has any basis in actuality, it is quite conceivable that in aquatic ecosystems, where macrophytes form an important part of the biocoenosis, withdrawal of hydrocarbons by these plants would have a depressing effect on the toxicity. The result would be a balancing or a suppression of the volume effect, if one assumes the plants' retention of endrin until

degradation of the chemical. The volume effect has been reported for aquaria studies, and seldom, if ever, in natural waters. Similarly, laboratory studies have generally established lower toxicity levels than field studies, sometimes with great disparities. My experience with 1 ppb in Driftwood Pond 2 is a notable exception, but as previously pointed out this would have been considered an LC_0 at the 96-hour level.

Fish bioassayists have noted different toxicity levels produced by various formulations of the same chemical compound. These are most graphically depicted by Henderson, *et al.* (14). They compared 96-hour LC_{50s} for bluegills when tested in 2 formulations (acetone solution and emulsible concentrate) of a number of insecticides. DDT was considerably more toxic in emulsible concentrate, dieldrin was less toxic in this form, and endrin showed little difference. From other experiments, they conclude that the toxicity of wettable powders (used in the present study) was similar to that of the acetone solution. Other chemical and physical factors such as the effects of light, dissolved mineral content, suspended particulate matter, etc., are not well-understood and could have an important or contributing influence.

In spite of all these possible contributory influences, the distribution of variously-established toxic thresholds seems implausibly disparate. From these doubts is born a second—or at least, an accessory—explanation. This is simply that a lack of standardization of analytical techniques exists. Extraction and analysis of almost infinitesimal hydrocarbon residues in tissues has been a tedious, painstaking job with interfering substances having unknown effects on the results. It appears that the more exacting gas chromatography-infra-red spectrophotometric method now in use will narrow the divergence of results. However, even this advance did not produce incontrovertible evidence in the lower Mississippi River incidents.

I suspect that higher temperatures and water transparency were responsible for the total kill in Driftwood Pond 2 at a level which, in laboratory tests, was sublethal. Conversely, the failure to get a kill in the Crosley Pond at a high dosage (2 ppb) is attributed to the high turbidity in this pond, with a possible assist from the decreasing water temperatures.

Different investigators have used various designations for their toxicity measurements (LD, TL, and LC). Lethal dosage (LD) has been used for some time as the index of relative toxicity to terrestrial or avian animals, and the method generally has used milligrams of a substance per kilogram of body weight as its comparative measurement. The Subcommittee on Toxicity, of the Federation of Sewage and Industrial Wastes Associations (12) recommended the term Median Tolerance Limit (TL_m) to express the concentration at which just 50% of the test animals die. This designation was aimed at measurements of chemicals in a liquid medium with fish as the test animals. It is usually derived by a straight line graphical interpolation from tests producing higher and lower percentages of mortality. More recently, several investigators have

shown an increasing preference for Lethal Concentration (LC). Usually LD and LC tests, like TL_m, have attempted to establish the 50% level (LD_{50}, LC_{50}).

For a piscicide, as briefly mentioned previously, the minimum concentration which produces 100% mortality of the fish population (LC_{100}) constitutes a more important figure than the LC_{50}. Desirably, for field use as a fish toxicant, a material should be effective in eradicating the population within a specific, preferably short, time (LC_{100} during x time interval). It is obvious then that the LC_{100} is not a precise unity but rather a fluctuating value between time parameters (e.g., the 24-hour LC_{100} may be 4 ppb whereas the LC_{100} for a 96-hour test may be 2 ppb).

Lennon and Walker (23) have used the term Effective Concentration (EC_{100}) in their delineative screening program. If we define an effective concentration (EC) as the minimum concentration that produces a 100% mortality to problem species, this can be written without the mortality-percentage subscript numeral. This would allow contraction of the abbreviate symbols by inclusion of the time interval as a superscript numeral as has Dorris *et al.* (11) in refinery waste bioassays (TL_m 48 = TL_m for 48-hour test). This would avoid complicating the formula with both a subscript and a superscript (LC_{100}^{48}), and it would be simpler to use. While EC (or LC_{100}) is a varying quantity with time-influenced parameters, the EC^{24}, the EC^{48}, etc., are precise figures for particular test situations.

Recommendations

Among a number of desirable characteristics of a fish toxicant (low cost, easy application, homogeneous dispersal, relatively persistent toxicity, etc.), the mandatory nature of one trait makes it the primary consideration. This is its effect on human health. It must first meet regulatory standards set up to guard the public (against itself?). At the time these tests were initiated, endrin was merely the latest and most potent of an exponentially-increasing number of chlorinated hydrocarbon insecticides. Its entry into the commercial market was accompanied by the usual—at that time—incomplete testing of its biological field effects. (Erstwhile tests by chemical manufacturers might be paraphrased as the development of broad spectrum pesticides with narrow spectrum evaluations.)

It soon became apparent that endrin (for fish, at least) was the most lethiferous substance to come out of the chemists' cauldron. When certain doubts concerning its effect on human health arose, its status became immediately suspect, in spite of its use in harvesting food fish in Malaysia (26). All the ramifications of its use are still not understood, and it appears possible that authorities may belatedly prohibit its sale. In view of its possible health hazards and the probability of its imminent withdrawal from the commercial market, it seems that health considerations and unavailability have negated whatever potential endrin may have had as a fish toxicant. One cannot with any integrity abhor

the fallout from atomic testings, deplore the dispersal of heptachlor to kill some insignificant bug, and then put on the Hydean mask to espouse the widespread dissimination of endrin in our waters.

Literature Cited

1. ANDERSON, BERTIL G. 1960. The toxicity of organic insecticides to Daphnia. Robert A. Taft Sanitary Eng. Center, U. S. Public Health Serv. Tech. Rep. W60-3. p. 94-95.

2. BARKER, ROY J. 1958. Notes on some ecological effects of DDT sprayed on elms. J. Wildl. Mgt. 22:269-274.

3. BRIDGES, W. R. 1961. Disappearance of endrin from fish and other materials of a pond environment. Trans. Amer. Fish. Soc. 90:332-335.

4. BRIDGES, W. R., B. J. KALLMAN, and A. K. ANDREWS. 1963. Persistence of DDT and its metabolites in a farm pond. Trans. Amer. Fish. Soc. 92:421-427.

5. BROWN, A. W. A. 1951. Insect control by chemicals. John Wiley and Sons, Inc., New York. 817 p.

6. BURDICK, G. E., E. J. HARRIS, H. J. DEAN, I. M. WALKER, JACK SKEA, and DAVID COLBY. 1964. The accumulation of DDT in lake trout and the effect on reproduction. Trans. Amer. Fish. Soc. 93:127-136.

7. COPE, OLIVER B. 1960. The retention of DDT by trout and whitefish. Robert A. Taft Sanitary Eng. Center, U. S. Public Health Serv. Tech. Rept. W60-3. p. 72-75.

8. COPE, OLIVER B. 1961. Effects of DDT spraying for spruce budworm on fish in the Yellowstone River system. Trans. Amer. Fish. Soc. 90:239-251.

9. DEWITT, J. B., C. M. MENZIE, V. A. ADORMAITIS, and W. L. REICHEL. 1960. Pesticidal residues in animal tissues. Trans. 25th N. Amer. Wildl. and Natur. Resources Conf. p. 277-285.

10. DEWITT, J. B., and J. L. GEORGE. 1960. Bureau of Sport Fisheries and Wildlife Pesticide-Wildlife Review: 1959. Fish and Wildlife Circular No. 84, Revised. U. S. Dept. Interior, Washington, D. C.

11. DORRIS, TROY C., WILLIAM GOULD, and CHARLES R. JENKINS. 1960. Toxicity bioassay of oil refinery effluents in Oklahoma. *In* Biological Problems in Water Pollution. Trans. of the 1959 Seminar Eng. Center, Cincinnati, Ohio. p. 276-285.

12. DOUDOROFF, P., B. G. ANDERSON, G. E. BUDDICK, P. S. GALTSOFF, W. B. HART, R. PATRICK, E. R. STRONG, E. W. SURBER, and W. M. VAN HORN. 1951. Bioassay methods for the evaluation of acute toxicity of industrial wastes to fish. Sewage and Ind. Wastes 23:1380-1397.

13. DUBOIS, K. P. 1958. Insecticides, rodenticides, herbicides, household hazards. Post-grad. Med. 24:278.

14. HENDERSON, C., Q. H. PICKERING, and C. M. TARZWELL. 1959. Relative toxicity of ten chlorinated hydrocarbon insecticides to four species of fish. Trans. Amer. Fish. Soc. 88:23-32.

15. HOFFMAN, C. H., and A. T. DROOZ. 1953. Effects of a C-47 airplane application of DDT on fish-pond organisms in two Pennsylvania watersheds. Amer. Midland Natur. 50:172.

16. HOOPER, FRANK F., JOHN E. WILLIAMS, MERCER PATRIARCHE, FRED KENT, and JAMES C. SCHNEIDER. 1964. Status of lake and stream rehabilitation in the United States and Canada with recommendations for Michigan waters. Mich. Inst. for Fish. Res. Rep. 1688. 56 p.

17. HOSS, DONALD E. 1964. Accumulation of zinc-65 by flounder of the genus *Paralichthys*. Trans. Amer. Fish. Soc. **93**:364-368.

18. HUNT, ELDRIDGE G., and ARTHUR I. BISCHOFF. 1960. Inimical effects on wildlife of periodic DDD applications to Clear Lake. Calif. Fish and Game **46**:91-106.

19. IYOTOMI, KISABU, TOMOTSU TAMURA, YASNO ITAZAWA, ISAO HANYU, and SYOTORO SUGIURA. 1958. Toxicity of Endrin to Fish. Prog. Fish Cult. **20**:155-162.

20. KALLMAN, B. J., O. B. COPE, and R. J. NAVARRE. 1962. Distribution and detoxication of toxaphene in Clayton Lake, New Mexico. Trans. Amer. Fish. Soc. **91**:14-22.

21. KATZ, MAX. 1961. Acute toxicity of some organic insecticides to three species of salmoids and to the three-spine stickleback. Trans. Amer. Fish. Soc. **90**:264-268.

22. KATZ, MAX, and GEORGE G. CHADWICK. 1961. Toxicity of endrin to some Pacific Northwest fishes. Trans. Amer. Fish. Soc. **90**:394-397.

23. LENNON, ROBERT E., and CHARLES R. WALKER. 1964. Investigations in fish control: (1) Laboratories and methods for screening fish-control chemicals. U. S. Dept. of the Interior, Bureau of Sport Fisheries and Wildlife, Circular 185. 15 p.

24. PREVOST, G., C. LANNOUETTE, and F. GRENIER. 1948. Effect of volume on the determination of DDT or rotonone toxicity on fish. J. Wildl. Mgt. **12**:241-250.

25. RUDD, R. L., and R. E. GENELLY. 1956. Pesticides: their use and toxicity in relation to wildlife. Calif. Dept. Fish and Game, Game Bull. No. 7. 209 p.

26. SOONG, MIN KONG. 1960. Shell "Endrex" used as a fish toxicant. Prog. Fish-Cult. **22**:93.

27. SCREENIVASAN, A., and M. V. NATARAJAN. 1962. Use of endrin in fishery management. Prog. Fish-Cult. **24**:181.

28. WILLIAMS, LOUIS G., and QUENTIN PICKERING. 1961. Direct and food-chain uptake of cesium^{137} and strontium^{85} in bluegill fingerlings. Ecology **42**:205-206.