derm derived from the separation procedure was not contaminated by algae.

The high specific activity of carbon-14 in green hydra ectoderm, and its broad distribution in the host's major chemical fractions, supports the hypothesis that the symbiotic algae are of nutritional significance to the host. This view is also supported by recent quantitative growth studies (*11*, *14*) which show that symbiotic algae exert a favorable influence on the growth and well-being of their hosts, especially under regimes of limited food or starvation. We are currently investigating the manner in which the carbon supplied by the algae aids the host. This material might be used as a general supplemental carbon source or, alternatively, a small part, possibly in the form of essential cofactors or vitamins, might be required for enzymatic activities of the animal (*15*).

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Insecticides: Effects on Cutthroat Trout of Repeated Exposure to DDT

Abstract. *Cutthroat trout were periodically exposed to* p,p′*-DDT, in acetone solution or in the food. Excessive mortality occurred only in lots treated with high concentrations of DDT, probably as a result of decreased resistance to nonspecific stressors. Surviving fish in these lots were significantly larger than those in the control lot, or in the lots treated with low concentrations of DDT. The number and volume of eggs produced was not reduced by DDT, but mortality among sac fry appeared to be highest in the lots treated with high concentrations. The data suggest that the sublethal concentrations of DDT ordinarily encountered in the environment are unlikely to damage a fishery.*

The Fish-Pesticide Research Laboratory of the Bureau of Sport Fisheries and Wildlife organized an experiment at the National Fish Hatchery at Jackson, Wyoming, to measure some long-term effects of DDT on the Snake River cutthroat trout, (*Salmo clarki lewisi*). This study began in December 1960 and terminated in September 1962.

Eleven lots of yearling fish were used; each lot was composed of 636 fish and was divided into three sublots located randomly in raceways. Lot I included untreated control fish. Lots II to VI were exposed for 30 minutes, once every 28 days, to a bath of DDT (*1*) in acetone solution, at concentrations of 1.0, 0.3, 0.1, 0.03, and 0.01 parts per million, respectively. Lots VII to XI were given DDT once a week in a pelleted diet, in doses of 3.0, 1.0, 0.3, 0.1, and 0.03 mg/kg body weight, respectively. At 4-week intervals throughout the experiment the surviving fish were weighed to determine growth. Samples of fish were withdrawn according to a schedule and were either frozen for subsequent chemical analysis or fixed in Bouin's solution for histopathological examination. Sampled fish, and those suffering mortality during the experiment, were weighed, measured, sexed, and examined for gross pathology. Microhematocrit measurements were made on blood from sampled fish. Reproductive success was determined during artificial spawning in 1962. The effects of DDT on mortality and pathology, growth, residue levels, and reproduction will be considered separately (*2*).

Cumulative mortality over the course of the experiment is shown in Fig. 1. For simplicity, the data from the sublots have been combined and are presented as the total for each lot. Little difference in mortality occurred among sublots receiving identical treatment. To determine if mortality in the treated lots differed significantly from that in lot I, *t*-tests were made at 2-month intervals.

Three months after the start of treatment, mortality in lot VII was significantly higher than that in lot I ($p < .05$). After 4 months, mortality differed also in lots II and VIII ($p < .05$, $< .02$, respectively). At 6 months it differed in lots III and IV ($p < .001$, $< .01$, respectively). Cumulative mortality in the remaining lots did not differ significantly from that in lot I during the 20-month experimental period.

Mortality rates, calculated by dividing the number of deaths during a month by the number of living fish in the lot, varied for each lot throughout the study. The control lot, and the lots treated with low concentrations of DDT, displayed peaks of mortality at about the 6th, 11th, and 18th months after the initial treatment. The lots treated with the highest concentrations displayed peaks at about the 6th and 18th months.

The periodicity of the death rate in all lots suggests that additional factors were involved in the increased mortality of the fish exposed to higher concentrations of DDT. One such factor was disease. Lesions due to disease were most common in dead fish from the control lot and lots treated with the lower concentrations of DDT. Fish that were obviously sick survived for considerable periods, but were the first to succumb during the periods of high mortality. When fish in the lots treated with higher concentrations showed signs of illness, they seldom survived long, perhaps indicating that the DDT had reduced their resistance to disease. Another stressor operative during part of this study was artificial spawning. Mortality was highest among fish from lots II, III, VII, and VIII, again indicating reduced ability to withstand stress.

Histopathological examination of sampled fish in apparent good health, and of fish which died, revealed no

pathology ascribable to DDT. No changes in microhematocrit measurements were found. No fish, either survivors or those which died, showed the symptoms of acute DDT poisoning.

Apparently associated with the increased mortality in lots II, III, VII, and VIII, was an increase in the size of these fish as compared with those in the control lot and the lots treated with low concentrations of DDT. The average weight of surviving fish in the lots treated with high concentrations was significantly greater than that of the fish in lot I after 6 to 7 months of treatment; p values ranged from $< .025$ to $< .001$. Also, both the mortality and growth curves of those treated with high concentrations began to diverge at the same time from the curves of the other lots. Since the actual growth rates between consecutive weighings were about the same in all lots, it was concluded that this effect was due to the selective death, in the lots treated with high concentrations of DDT, of smaller, weaker, and sometimes diseased fish, possessing less than average growth potential. The data on which this conclusion is based will be presented elsewhere (*2*).

Residues of DDT and two related substances were determined by a paper chromatographic procedure after the method described by Mitchell (*3*). The other substances, found in varying proportions in almost all samples, were DDE, 1,1-dichloro-2,2-bis(*p*-chlorophenyl)-ethylene, and DDD, 1,1-dichloro-2,2-bis(*p*-chlorophenyl)-ethane. The analyses were performed on portions of homogeneous blends of whole fish and anhydrous Na_2SO_4. From one to six fish per sublot were analyzed at each sampling. Representative data are given in Table 1; these data constitute about one-fourth of the total number of samples analyzed.

Under the conditions of this experiment, the maximum uptake of DDT from water appeared to occur when the concentration was about 0.3 parts per million. Lots II and III reached a plateau of residue accumulation; lot II did so somewhat earlier than lot III. Residue buildup in lot IV occurred at a slower rate and continued throughout the study. Residues in lots V and VI were not higher than those in the control lot, indicating that an approximate equilibrium was attained between accumulation and metabolism and excretion, or all three, when the fish were exposed to 0.01 and 0.03 parts per million. The amounts of residue in lots VII and VIII increased rapidly during the early part of the experiment and then leveled off. Residues in lot IX were markedly lower than those in lot VIII, but were higher than control levels. The residues in lots X and XI were not significantly different from the controls. No correlation between the amount of residue and either the sex or size of the fish was found in any of the lots.

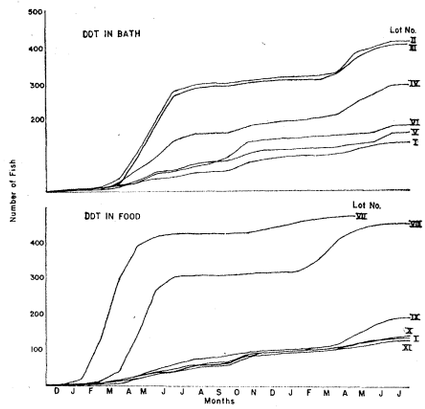

Fig. 1. Cumulative mortality among trout from December 1960 through July 1962.

The amount of chlorinated hydrocarbons found in the control fish was not entirely surprising. We have found small amounts of DDT and related substances in almost all fish analyzed from other sources, including those reared in hatcheries. Commercial fish foods have yielded similar analytical results. To determine the source of contamination of the fish in lot I, the components of the pelleted diet fed to all fish in the study were analyzed. Appreciable amounts of chlorinated hydrocarbons were found only in cod-liver oil, which contained 3.0, 17.0, and 3.7 parts per million of DDT, DDE, and DDD, respectively. This material, fed for a period of about 1 year, constituted 3 percent of the diet, by weight. It was probably the major source of residues in the control fish.

The proportions of two of the three components making up the total residues varied with the dosage of DDT. Lots treated with low concentrations of DDT, and control fish, contained larger proportions of DDE than did fish treated with high concentrations of DDT. The amount of DDT in the total residue at the end of the experiment ranged from about 90 percent in the groups fed with high concentrations of DDT to about 4 percent in lot XI after one feeding of DDT. Although the fish treated with low concentrations of DDT had the same total residues as the controls, they had relatively larger amounts of DDT and smaller amounts of DDE. Lower proportions of DDE have also been reported in fat samples obtained from humans exposed to large quantities of DDT (*4*).

The DDD found in whole bodies of trout in this study did not appear to be derived from DDT by metabolic processes. There was a decrease in the percentage of DDD present as the experiment progressed, suggesting that levels of DDT and DDE were building up at a faster rate than DDD. Sufficient DDD was present in the basic diet to account for the residues found. The presence of DDD in rat tissues as a metabolite of DDT, but not of DDE, has been determined (*5*). In our laboratory, DDD has been found inconsistently in the tissues of fish which have been exposed to commercial preparations of DDT when in their normal environment. In a warm water pond, that was treated once with p,p'-DDT, we found both DDE and DDD as major metabolites in fish and crayfish. In several types of microorganisms exposed to C^{14}-labeled DDT we have found labeled DDD as the only metabolite (*6*).

Chemical analysis of tissue and organ pools obtained from fish which died during the experiment indicated a highly uneven distribution of total chlorinated hydrocarbons and of the three components. The distribution of the separate components is probably not a function of the lipid content of

Table 1. Mean residues in whole fish of chlorinated hydrocarbons expressed as parts per million, ± standard error.

Elapsed time (days)	Control	DDT in bath			DDT in food		
	Lot I	Lot II	Lot IV	Lot VI	Lot VII	Lot IX	Lot XI
3	0.4 ± 0.2	1.2 ± 0.2	0.8 ± 0.1	0.6 ± 0	0.7 ± 0.3	0.6 ± 0.1	0.9 ± 0.1
56	0.7 ± 0.2	2.0 ± 0.1	1.4 ± 0.2	1.0 ± 0.2	7.0 ± 1.5	0.9 ± 0.4	0.7 ± 0.1
166	0.7 ± 0.1	4.3 ± 0.5	3.0 ± 0.1	0.8 ± 0.1	14.1 ± 4.8	1.4 ± 0	0.8 ± 0.1
278	0.6 ± 0.2	5.9 ± 0.6	1.7 ± 0.5	0.3 ± 0.1	—	2.1 ± 0.7	0.4 ± 0.1
391	0.9 ± 0.2	4.5 ± 0.5	3.5 ± 0.7	1.2 ± 0.1	—	3.8 ± 2.1	1.4 ± 0.3
497	1.0 ± 0.1	6.5 ± 4.7	5.7 ± 0.6	1.1 ± 0.2	—	3.9 ± 1.8	1.4 ± 0.5

the various organs, but may reflect individual rates of uptake and discharge and of metabolic formation and degradation. The fish for which data are presented in Table 2 died during a 4-month period in which whole-body residues were approximately 5 parts per million in lot II, 68 percent as DDT and 29 percent as DDE. The amount of residue was about 13 parts per million in lot VIII, 83 percent as DDT and 14 percent as DDE. Comparable figures were about 1 part per million in lots V, VI, X, and VI. In lot V, the residue was distributed 55 percent as DDT, 40 percent as DDE; in lot VI, 20 percent DDT, 75 percent DDE; in lot X, 45 percent DDT, 50 percent DDE; in lot XI, 35 percent DDT, 60 percent DDE. The data for the lots treated with large amounts of DDT indicate that the rate of metabolism of DDT may be high in the liver and gut, and low in the brain, ovary, and skeletal muscle. The comparative large amounts of DDD in several tissues again raises the question of the metabolic formation of this substance from DDT. The very large amount of DDD in livers of DDT-fed fish, as compared to those of DDT-bathed fish, is particularly indicative of such formation, since both groups of fish received the same amounts of DDD in the contaminated basic diet. Assuming that it is impractical to try to obtain fish and diet components free of DDD, the final resolution of this problem will depend on studies in vivo and in vitro with C^{14}-labeled DDT. It is possible that our method of analysis of whole-body residues was not sufficiently sensitive to detect the very slow formation of DDD from DDT, as may occur in some tissues.

These results can be compared with the findings of Holden (*7*), who exposed brown trout to lethal doses of C^{14}-labeled DDT in water. The concentrations in the tissues, calculated from the radioactivity and expressed as DDT, were generally higher in liver and muscle than in brain. Although the total amount of solvent-extractable material (lipid) in the brain was high, the amount of DDT in the brain was consistently low in this short-term study. This is in marked contrast to the results we obtained with fish treated with large amounts of DDT, both with those which were periodically exposed to the chemical, as in this study, and with fish exposed to DDT once and then analyzed after periods of up to a year. Large residues in the brain may be indicative of either long-term exposure to DDT, or a prolonged time lapse after a single exposure. The total amount of DDT encountered by the fish is also a factor to be considered; the amount of residue in the brains of fish that were treated with low concentrations of DDT were no higher than whole-body residues at comparable times (Table 2).

Many of the fish reached sexual maturity during the spring of 1962. Eggs were stripped from the females as they became ripe and were fertilized with milt from males in the same sublot. An attempt was made to rear at least three collections from each of the three sublots at each level of treatment. The number and volume of eggs produced were measured initially, and mortality was determined during the various developmental stages.

The proportion of fish reaching sexual maturity was somewhat higher in the lots treated with high concentrations of DDT than in the control lot or the lots treated with low concentrations. This was apparently due to the higher incidence of small fish in the latter groups; observation revealed no differences in sexual development among fish of like size in any lot. Considering the larger size of females in the lots treated with large amounts of DDT, egg production was similar in all lots except VII. Only one female could be spawned in this lot; egg production was lower in this individual than the average for any other lot. Survival rates of eggs during incubation were also uniform, again with the exception of lot VII. Of approximately 850 eggs produced by this fish, only five hatched and the fry died within several days. Mortality among sac fry of the other ten lots was also high, and accurate measurements of losses at this stage could not be made. The death rate was discernibly highest among fry of lot VIII, and was higher in lots II and III than in the control lot and the lots treated with small amounts of DDT. Once the fry began to feed, mortality was low in all lots, including the few survivors of lot VIII. The growth rates of the fry were variable during an additional 6 months of observation, but there was no apparent correlation with the concentrations of DDT to which the parent fish had been exposed.

Chemical analyses of eggs and fry showed that appreciable amounts of chlorinated hydrocarbons were present. The results were very variable, but larger residues were generally found in the eggs of fish treated with larger amounts of DDT. The amount of total residue ranged from 0.2 parts per million in one collection of eggs from lot I, to 20 parts per million in a sample from lot VIII. No significant pattern could be distinguished between the amounts of residue in nonviable eggs and the amounts in unfertilized ones, which consisted of a mixture of viable and nonviable eggs. Small amounts of residue were found consistently in fry collected 5 to 6 weeks after hatching.

In brief, we conclude that, under our experimental conditions, a threshold level of toxicity of DDT in the chronic poisoning of cutthroat trout exists at somewhat below 0.1 parts per million when the DDT is present in the surrounding medium, and 1.0 mg/kg body weight when taken in with food. A threshold effect is, perhaps, due to the limited ability of the fish to metabolize DDT.

It is unlikely that concentrations of DDT in water or food would reach even the lower levels used in this experiment for any length of time. Al-

Table 2. Residues of chlorinated hydrocarbons (parts per million) in the tissues of dead fish.

Tissue	DDT	DDE	DDD	Total
Lot II (8 fish)				
Brain	6.2	6.3	1.0	13.5
Skeletal muscle	3.3	1.5	0.3	5.1
Liver	1.3	1.8	2.5	5.6
Ovary	8.6	2.9	0.8	12.2
Gut	1.0	2.4	1.3	4.7
Testes	0.6	1.0	0.4	2.0
Lots V and VI, combined (15 fish)				
Brain	ND*	1.3	ND*	1.3
Skeletal muscle	0.4	0.3	0.1	0.8
Liver	0.2	0.8	0.4	1.4
Ovary	1.4	1.8	0.3	3.5
Gut	Trace	1.5	0.4	1.9
Testes	0.3	3.5	0.1	3.9
Lot VIII (8 fish)				
Brain	28.0	7.0	1.0	36.0
Skeletal muscle	7.8	2.3	0.8	10.9
Liver	1.8	1.4	7.0	10.2
Ovary	4.8	0.9	0.9	6.6
Gut	1.0	0.8	2.5	4.3
Testes	1.3	0.7	0.4	2.4
Lots X and XI, combined (15 fish)				
Brain	0.5	0.5	Trace	1.0
Skeletal muscle	0.3	0.3	0.05	0.6
Liver	0.3	0.9	0.4	1.6
Ovary	0.8	0.7	0.2	1.7
Gut	1.5	5.5	0.3	7.3
Testes	0.1	0.2	0.1	0.4

* No detectable amount.

though engorgement of DDT-poisoned insects might occur shortly after an application, it is doubtful that it could recur often enough to damage a fish population. Even after deliberate addition of DDT to a stream or pond, its rate of disappearance is rapid (*8*). Thus, it is doubtful that the concentrations of DDT capable of damaging a fishery by chronic toxicity, as determined in this experiment, would be reached frequently enough to do so, barring repeated flagrant misuse of the insecticide.

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13 August 1963

Electron Microscopy of the Replicative Form of the DNA of the Bacteriophage ϕX174

Abstract. *Electron micrographs of surface films containing the replicative form of the DNA of bacteriophage ϕX174 show ring structures, whose average contour length is 1.64 μ, which have the characteristic appearance of double-stranded DNA throughout most of their length.*

The DNA of bacteriophage ϕX174 has been shown to be single-stranded (*1*) and to have a ring structure (*2*).

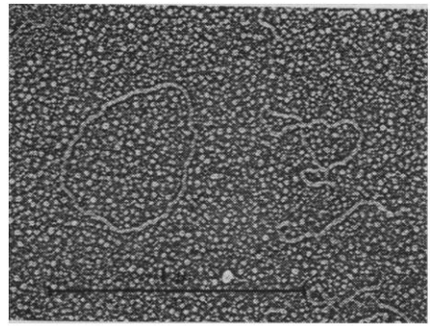

Fig. 1. Opened and twisted rings of ϕX-RF DNA with filamentous DNA of *E. coli*. Uranium contrast, negative. × 75,000.

Evidence has been presented (*3*) that during the intracellular reproduction of this virus, the viral DNA is converted to a double-stranded form, referred to as a "replicative" form, or RF, which is then multiplied. A purification of the replicative form has recently been described (*4*).

Our purification of the RF has included the use of fractional precipitation by cetyltrimethylammonium bromide (*5*) and of column chromatography, as described by Mandell and Hershey (*6*). The preparation obtained in this way is infective to protoplasts (*7*), and this infectivity has the resistance to inactivation by ultraviolet light and the buoyant density characteristic of the replicative form (*3*).

This material has been examined in the electron microscope by the monolayer technique of Kleinschmidt *et al.* (*8*). A solution of 2×10^{-6} g DNA per ml is mixed with 10^{-4} g/ml cytochrome *c* in 1*M* ammonium acetate, and 0.2 ml of the mixture is spread upon a clean surface of 0.1*M* ammonium acetate as hypophase in a Langmuir trough.

The DNA threads diffuse and part of them become adsorbed to the protein film. After full expansion to about 0.85 m² per mg of protein, the film is transferred to carbonized support films (*9*) and rotary shadowed with uranium (*10*). An appreciable fraction of the DNA threads are seen in the form of rings, either open or twisted to varying extent (Fig. 1). The appearance of these circular structures throughout most of their length is that characteristic of double-stranded DNA. Measurement of the length of the DNA in over 200 of these rings, both open and twisted, has given an average length of 1.64 ± 0.11 μ. Many of the

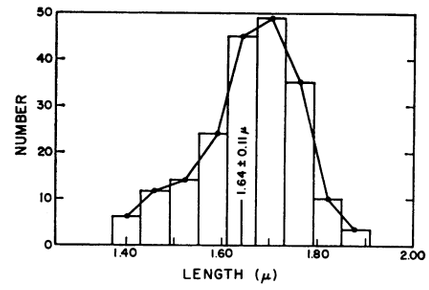

Fig. 2. Length distribution of ϕX-RF DNA of over 200 rings.

DNA threads in the preparation appear to have two ends, but only a small fraction of these are comparable in length with the rings.

If a Watson-Crick structure (*11*) is assumed for a double-stranded ϕX174 DNA, it should have a weight of 1.96×10^6 avograms (1 avogram = 1g/avogadro number) per micron (Na^+ salt). Thus the observed mean length corresponds to a molecular weight of 3.2×10^6, in remarkably good agreement with the calculated value of twice the weight of the viral DNA (1.7×10^6 avograms) (*1*).

It thus appears plausible to associate these structures with the replicative form of ϕX174 DNA and to conclude that the RF, like the viral form, occurs in a ring structure during the vegetative stage of the virus (*12*).

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