DDT: Short Term Effects on Osmoregulation in Black Surfperch (*Embiotoca jacksoni*)

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The toxicity of pesticides to fish has been attributed to various mechanisms, including suffocation, osmoregulatory failure, and damage to various organs, including liver and the central nervous system (EISLER and EDMUNDS, 1966; GRANT and MEHRLE, 1970; JOHNSON, 1968; RUDD, 1964). This report demonstrates that death due to DDT in a species of marine fish was not due primarily to osmoregulatory failure.

Osmoregulatory impairment caused by acute DDT exposure has been attributed to inhibition of certain ionic transport mechanisms and consequently to decreased intestinal water absorption in marine teleosts, such as eels (Anguilla rostrata) and killifish (Fundulus heteroclitus) (JANICKI and KINTER, 1971; KINTER et al., 1972). However, the problem of osmoregulatory failure as a direct effect of acute DDT exposure or as a consequence of DDT effects on the nervous system is still unresolved (CUTKOMP et al., 1971; KOCH et al., 1971). We attempted to determine the effects of DDT on osmoregulation in a marine teleost and to learn whether osmoregulatory failure was sufficient in itself to cause death.

MATERIALS AND METHODS

The fish used were black surfperch, Embiotoca jacksoni (family: EMBIOTOCIDAE). Populations of black surfperch living in waters subject to large amounts of DDT influx (the inshore waters from Los Angeles south to Newport Beach, California) contain rather high amounts of DDT in their tissues* (PARKHURST, 1972).

The black surfperch were trapped in the harbor at San Pedro, California and transported to UCLA, where they were held without feeding for 10 days before use. Fish were injected intraperitoneally with corn oil (0.0025 ml per gram body weight) in which 100% technical DDT (Montrose Chemical Corporation of California) had been dissolved in concentrations calculated to

* Average total DDT residues in fillets of 28 fish = 14.7 ppm

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result in total injected doses of 0, 1, 10, 100, and 200 parts per million (ppm) in the whole fish. Blood samples were procured by terminal heart puncture at 12 or 72 hours after injection. Plasma osmotic concentrations were determined by vapor pressure osmometry (Mechrolab Model 301A).

RESULTS AND DISCUSSION

The effect of the injected DDT on plasma osmotic concentration is shown in TABLE 1. These black surfperch

TABLE 1

Effect of DDT on Plasma Osmotic Concentration of Black Surfperch (Embiotoca jacksoni)

Treatment	Time (hours)	Plasma Osmotic Concentration (milliosmols ± S.E.)
Non injected	0	307.1 ± 3.5 (14)[a]
Control (corn oil only)	12	306.2 ± 12.8 (7)
	72	308.4 ± 2.8 (8)
DDT 1 ppm	12	309.8 ± 6.2 (8)
	72	No data
DDT 10 ppm	12	301.5 ± 7.8 (7)
	72	317.9 ± 7.5 (5)
DDT 100 ppm	12	310.0 ± 4.3 (8)
	72	331.3 ± 3.1 (9)
DDT 220 ppm	12	368.6 ± 3.9 (8)
	44[b]	375.4 ± 15.3 (4)

[a] Number in parentheses shows the number of fish sampled
[b] Mean survival time of 43.9 hours

presumably contained high levels of DDT already, but only the highest injected dose of DDT affected blood plasma electrolytes to any significant degree ($p < .01$). All fish survived 12 hours at any given dose. In the 72 hour experiment, all fish survived at doses below 100 ppm DDT. Mortality was noted at the 100 ppm and 200 ppm DDT doses. Three out of the twelve fish injected with 100 ppm DDT died between 60 and 72 hours. Blood samples were not taken from dead fish. Of the eight fish injected with 200 ppm DDT, four died before blood samples could be taken. The four fish from which blood was obtained were moribund and showed typical symptoms of DDT poisoning (GAKSTATTER and WEISS, 1967; JOHNSON, 1968; KINTER et al., 1972). Only one of these fish survived the full 72 hours. The mean survival time for these four fish was 44 hours.

Osmoregulatory breakdown did not seem to be the principal cause of death in these DDT-poisoned fish for the following reasons. First, the major rise in plasma osmotic concentration resulting from the 200 ppm dose occurred within 12 hours after injection, and did not increase much further with additional time, even in dying fish. Second, the mean osmotic concentration observed just before death in DDT treated fish (375 mOsm/L) was lower than that found in the same population acclimated for 10 days to 50 ^{o}/oo salinity (442 mOsm/L), and was markedly less than that at death caused by higher salinities (499 mOsm/L) (WAGGONER, 1972). Rapidity of increase of plasma osmotic concentration likewise is ruled out as a cause of death, as similar increases have been observed in fish acclimating to 50 ^{o}/oo salinity with no apparent ill effects (WAGGONER, 1972).

We do not think that the observed changes in plasma osmotic concentration indicates a disruption of osmoregulation by DDT sufficient to be primarily responsible for death in these fish over short time periods. While a continued high osmotic concentration over longer periods might prove deleterious, direct action of DDT on vital tissues (brain and spinal cord) seem more likely as causes of death.

SUMMARY

DDT injected intraperitoneally into black surfperch caused substantial increases in plasma osmotic concentration only at doses much larger than are likely to be encountered in nature. Increased plasma concentrations were below those tolerated by fish adapted to high salinities. Death of marine teleosts from DDT poisoning probably involves factors other than simply osmoregulatory failure.

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