Degradation of DDT by Goldfish

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Degradation of DDT by fish is well documented (1,2,3). Fish liver contains oxidases of the mixed-function kind which are associated with microsomes (4). In rats these enzyme systems can be induced by phenobarbital (5). Practical use of phenobarbital has been made to greatly hasten excretion of dieldrin and/or metabolites by cows and to rapidly reduce the dieldrin level in their milk (6,7,8,9). The loss of fry born of fish carrying burdens of DDT (10) led to experiments here to determine whether exposure of goldfish to phenobarbital would increase the rate of elimination of DDT, presumably by induction of appropriate enzymes.

Experimental

Sixty goldfish (Carassius auratus) were held in two 12 gallon aquaria at 20°C with glass wool filter aerators. The DDT levels in the fish were built up by feeding commercial fish food with DDT incorporated. The amount of DDT in the food was increased as follows: Days 1-5, 45 ppm; days 6-13, 90 ppm; days 14-20, 180 ppm. Four fish from each tank were taken for analysis at day 21. Sacrifice was delayed for two days to allow the guts to clear DDT. No symptoms of DDT poisoning were noted and there was no mortality during the feeding period.

The remaining fish were treated as follows: The test group was exposed to 75 ppm sodium phenobarbital in the water. The level of phenobarbital present was such that the treated fish were evidently slightly affected, being judged as somewhat less active than the controls. The barbiturate solution was replaced daily for seven days. The water in the control tank was replaced daily. Eight fish from each group were analysed for DDT at day 29, two days after cessation of exposure to phenobarbital. After two weeks (day 43) without further treatment, final samples of eight fish from each tank were taken for analysis. During the three week period from the beginning of drug treatment and termination of the experiment two fish from each group died of unknown causes and were discarded.

The liver and intestinal tracts of four fish from each group in the above experiment were used to prepare microsomes for measurement of aldrin epoxidation (11). An indication of enzyme induction was taken from the fact that the preparation from the phenobarbital treated fish gave 42% greater dieldrin production than

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that from the control group.

Chlorinated hydrocarbon insecticides are stored in fatty tissues. Differing amounts of fat could therefore influence the levels of DDT and its metabolites in the fish. Four fish which had not been fed DDT were analysed for total fat (12). A range of 0.78 to 1.2% fat, about a mean of 0.96% indicated that the lipid content was probably not an important factor in the insecticide residue differences found in these experiments.

Analysis of DDT, DDE and DDD were done by electron affinity gas chromatography as previously described for chlorinated hydrocarbons (13).

Results and Discussion

The results of the analysis are presented in Table 1. Although the fish were under DDT pressure for the first 21 days, most of the DDT was converted to DDE. Calculation of the average amount of DDT fed indicated that 40% of it was stored as DDT, DDE and DDD at the end of the feeding period. Statistical analysis of the results indicated that no significant differences existed between treated and control fish. This failure to yield evidence of enzyme induction occurred despite the possible reinforcement of the phenobarbital pressure by the presence of significant amounts of DDE which is recognized as an enzyme inducer (14). The significance, if any, of the absence of DDT in the control fish is not known.

Examination of the standard errors illustrates a trend toward higher values with increasing elapsed time after cessation of feeding DDT. The variable metabolic capabilities of the individual fish were apparently expressed after DDT feeding stopped, leading to increased variability of the residues with time.

The speed of accumulation and ultimate level of DDT and metabolites were limited in these experiments to simulate a natural situation as much as possible.

To answer more certainly the question of whether induction of DDT metabolism in fish can be effected, will perhaps require higher levels of DDT, and analysis of the more polar metabolites such as DDA.

Summary

Goldfish were fed DDT for three weeks, then exposed to phenobarbital solution for one week to study the possibility of microsomal enzyme induction and therefore possibly enhanced insecticide excretion. Analysis of DDT, DDE and DDD were made at intervals up to six weeks. Forty percent of the DDT fed was incorporated during the feeding. It was recovered mainly as DDE (68 to 96%). Significant differences between pesticide levels of phenobarbital treated and control fish were not found.

TABLE 1

DDT and metabolites in goldfish after feeding DDT and treatment with phenobarbital. (Brackets indicate standard deviations).

	Group*	weight in grams	DDT	DDE	DDD	Total
				parts per million		
(1)	after feeding DDT for 21 days	2.3 (0.5)	2.3 (1.7)	6.8 (0.7)	0.9 (0.3)	10.0 (2.0)
(2)	Two days after 7 days exposure to phenobarbital	2.8 (0.7)	0.1 (0.2)	4.7 (2.5)	0.1 (0.2)	5.0 (2.8)
(3)	Controls for (2)	2.7 (0.5)	n.d.	8.2 (2.6)	0.8 (0.3)	9.6 (3.1)
(4)	Two weeks after 7 days exposure to phenobarbital	2.7 (0.9)	0.5 (0.1)	3.8 (5.0)	0.3 (0.1)	4.1 (5.5)
(5)	Controls for (4)	3.1 (1.0)	n.d.	4.9 (2.8)	0.2 (0.3)	5.1 (5.4)

* There were eight individuals in each group.
n.d. Not detected.

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