Lethal and Reproductive Effects of Dietary Mirex and DDT on Old-Field Mice, Peromyscus polionotus

J. L. Wolfe, R. J. Esher, K. M. Robinson, and J. D. Yarbrough Department of Zoology, Mississippi State University, Mississippi State, Miss. 39762

Although the compound mirex has consistently been found in wild mammals in areas of field application (MARKIN 1972, WOLFE and NORMENT 1973), the significance of these residues is largely unknown. Mirex is stable in the environment, and appears not to be readily metabolized by mammals (ALLEY 1973, STEIN and PITTMAN 1977).

While the main objective of the study was to evaluate mirex, the experimental diets and feeding regimes were duplicated with DDT for comparative purposes. The literature suggests that the oral toxicity of mirex and DDT to laboratory animals is similar (SPECTOR 1955, GAINES and KIMBROUGH 1970, GINGELL and WALLCAVE 1974), although the LD $_{50}$ for mirex in mice is difficult to obtain due to its low solubility in corn oil, and apparently none has been reported.

Here we report findings on the effects of dietary mirex and DDT on survival and reproduction of the old-field mouse, <u>Peromyscus polionotus</u>. This species was selected because it is common in the southeastern United States, its preferred habitat is similar to that of fire ants, and it can serve as a useful model for other wild mammals. The literature on its biology, both in the field and laboratory, is quite extensive (FELLEY and SMITH 1975) and will be useful in inferring possible ecological consequences from our results.

METHODS

The animals used were first generation laboratory offspring of wild parents trapped in the Ocala (Florida) National Forest. At the age of about 60 days they were paired and placed on experimental diets. They were housed in 450 \times 240 \times 140 mm plastic cages with wire tops. Ground corncob (San-i-cel) and cotton were provided for bedding. Water and the diet formulations were available in excess.

The diets were made by dissolving technical grade mirex or DDT in hexane, mixing thoroughly with ground laboratory chow (Purina) and air drying. Controls were fed ground food mixed with an identical quantity of hexane and dried in the same fashion.

Both mirex and DDT were fed at rates of 1.8 and 17.8 ppm in the food. We previously determined food consumption at 2.0 g per day, and the consumption of treated food did not deviate significantly from this value. Thus, about 0.004 mg was consumed per day

at the low concentrations and about 0.036 at the high concentration. The mice averaged 15.1 \pm 1.4 (SD) g in body weight. The daily intake expressed as mg/kg body weight was therefore 0.24 at the low dosage and 2.40 at the high dosage.

Twelve pairs of mice were fed each diet for 15 months or until death. The cages were checked daily to record mortality and the birth of the litters. Surviving young were removed at weaning.

Another group of mice was fed identical diets and sacrificed periodically for histological and physiological examinations. At 30, 90, and 180 days, three animals at each mirex treatment level were selected for residue analysis.

RESULTS

Survival on the four experimental and the control diets is illustrated in Fig. 1. The high mirex diet resulted in significant mortality, with only 2 of 24 mice surviving the 15 mo period. Mortality in the other three groups was near that of the controls.

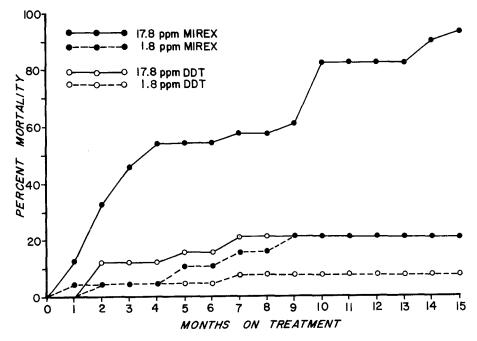


Figure 1. Mortality of mice given two concentrations of mirex and DDT in the diet. N=24 for each group.

Table 1 shows reproductive performance at 3-mo intervals. Reproduction virtually stopped (only 1 litter produced) after 3 mo on 17.8 ppm mirex. There was also a suggestion of decreased reproduction at 1.8 ppm mirex. During the 15-mo period, this group averaged 16.7 young per pair. Controls averaged 25.2 young per

TABLE 1

Reproductive performance of mice on dietary mirex and DDT for 15 months. LS = average litter size, L/Pr = litters per pair produced during interval. Means and standard errors in the last row are for the entire period.

Months of	Co	Controls	1.8 ppm Mirex	Mirex	17.6 pp	17.6 ppm Mirex	1.8 pp	1.8 ppm DDT	17.6 p	17.6 ppm DDT
Ireatment	LS	LS L/pr	57	L/pr	LS	L/pr	ΓS	LS L/pr	77	L/pr
0-3	4.0	4.0 1.1	3.8	1.2 4.0	4.0	9.0	3.8	3.8 1.4	4.0	6.0
3-6	4.4	0.8	4.0	0.8	3.0	0.2	4.0	1.3	4.5	=
6-9	4.2	1.5	3.3	1.3	ı	0	3.9	3.9 1.4	4.2	1.0
9-12	4.3	1.3	3.5	9.0	ı	0	4.3	4.3 1.5	4.6	1.3
12-15	4.6	1.2 4.1	4.1	0.8	ı	0	4.4	4.4 1.0	4.0	6.0
x + SE 4.	4+.13	1.2+.12	± SE 4.4±.13 1.2±.12 3.8±.14 0.9±.13	0.94.1	3	7	4.1+.12	1.3±.09	4.1±.12 1.3±.09 4.3±.12 1.0±.07	1.9

TABLE 2

Liver mirex residues as related to dietary level and period of treatment. Amount ingested is based on the average daily consumption of $60~\rm mice$; residues on three individuals randomly selected from each group.

	5	Length of High Concentration (17.8 ppm in Food) Low Concentration (1.8 ppm in Food)	בסת כסווכפורו פרוסוו	(500 ·
reatment cum (mg	Treatment Cum. Amt. Ingested (mg/g body weight)	Liver Residue (ppm)	Cum. Amt. Ingested Liver Residue (mg/g body weight) (ppm)	Liver Residue (ppm)
30 days	70	20-28	7	2-3
90 days	210	25-65	21	3-5
180 days	422	49-78	42	2-6

pair. Both figures are adjusted for parent mortality late in the study. DDT diets did not affect reproduction. At 17.8 ppm, 21.5 young/pair and at 1.8 ppm, 26.7 young/pair were produced. Although previous studies have shown that experiments on effects on litter size may not be consistent (WARE and GOOD 1967), there is an indication in our data that 1.78 ppm mirex may decrease litter size (\underline{t} -test on overall means, 0.1 > P > .05). Results of the residue analyses are given in Table 2.

DISCUSSION

Although comparisons are difficult, earlier investigations suggest that the toxicity of DDT and mirex is roughly equal (GAINES and KIMBROUGH 1970, WARE and GOOD 1967, GINGELL and WALLCAVE 1974). Our results indicate that, given in diet, mirex is about four times as toxic as DDT.

Mirex appears slightly less toxic to old-field mice than to laboratory mice. WARE and GOOD (1967) reported 100% mortality in laboratory mice fed 10 ppm mirex for 60 days. At 17.8 ppm, 50% mortality occurred in 105 days and 92% mortality at 450 days in old-field mice.

Liver mirex residues appear to increase in proportion to amount ingested through 180 days of treatment, and are directly proportional to dietary concentration. This substantiates earlier work (ALLEY 1973) indicating the stability of mirex in biological systems, and provides a crude means of evaluating the potential importance of residue levels found in wild mammals.

WARE and GOOD (1967) reported significant effects on reproduction of laboratory mice fed 5 ppm (specifically a decrease in litter size), although their interpretation has been questioned by ALLEY (1973). We found a virtual cessation of reproduction in pairs surviving 3 mo on 17.8 ppm, which is not surprising since 50% mortality had occurred by this time. However, we also had an indication of decreased reproduction in the 1.8 ppm group. The main effect appeared to be a decrease in litter size, although after 6 mo there was also a slight indication of fewer litters being produced. No information on the physiological mechanisms involved in the reproductive effects is provided by our data.

Previous work (WARE and GOOD 1967, FULLER and DRAPER 1975) and the data presented here clearly indicate that further work on the reproductive effects of mirex is warranted. Considering the potential for biological magnification of mirex (WOLFE and NORMENT 1973), amounts approximating our low dietary levels could be ingested by small mammals in areas of field applications. Residue levels of the same order of magnitude as found in our animals on low dietary regimes have been reported in wild mammals. Thus the potential for effects on reproduction in natural populations of small mammals exists in areas where mirex has been used.

ACKNOWLEDGMENTS

We thank Martha Robertson for assistance in the project. The work was funded by NIH grant ES00817-03.

REFERENCES

- ALLEY, E.G.: J. Environ. Qual. 2, 52 (1973).
- FELLEY, J.D. and M.H. SMITH: A bibliography of the old-field mouse,

 Peromyscus polionotus Wagner (Rodentia). Savannah River Ecol.

 Lab., Aiken, S.C. 33 p. (1975).
- FULLER, G.B. and S.W. DRAPER: Proc. Soc. Exper. Biol. Med. 148, 414 (1975).
- GAINES, T.B. and R.D. KIMBROUGH: Arch. Environ. Hith. 21, 7 (1970). GINGELL, R. and L. WALLCAVE: Toxicol. Appl. Pharmacol. 28, 385 (1974).
- MARKIN, G.P., J.H. FORD, J. HAWTHORNE, J.H. SPENCE, J. DAVIS, H.L. COLLINS, and C.D. LOFTIS: USDA, APHIS 81-3, 19 p. (1972).
- SPECTOR, W.S. (ed.): Handbook of Toxicology. NAS-NRC Wright Air Dev. Center Tech. Rep. 55-16, 408 p. (1955).
- WARE, G.W. and E.E. GOOD: Toxicol. Appl. Pharmacol. 10, 54 (1967). WOLFE, J.L. and B.R. NORMENT: Pestic. Monit. J. 7, 112 (1973).