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Uptake and Disposition of Endrin in Insecticide-Susceptible and -Resistant Mosquitofish (*Gambusia affinis*)

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Endrin uptake and tissue distribution was studied in insecticide-susceptible (S) and -resistant (R) mosquitofish (*Gambusia affinis*). Fish were exposed to equal (10.2 ppb) and equitoxic (0.6 ppb for S and 314 ppb for R) levels of [^{14}C]endrin for up to 8 h. The accumulation and tissue disposition patterns of endrin were significantly different between the two fish populations studied. A 13.5-fold greater [^{14}C]endrin concentration in gills of S fish than R fish at the equal exposure levels suggested a barrier to endrin penetration at the organismal level. S/R ratios of tissue radioactivity following 8-h exposure to 10.2 ppb [^{14}C]endrin indicative of tissue membrane barriers in the R fish included: brain, 45.9; liver, 7.7; gall bladder, 9.7; spleen, 20.9; intestine, 4.8; and ova, 4.1. The barrier to endrin penetration into the brain appeared to be most effective. The gall bladder/liver ratios suggested that the R fish could effectively excrete endrin over a wider range of exposure concentrations than S fish.

An organochlorine insecticide-resistant population of mosquitofish (*Gambusia affinis*) is the result of natural selection pressures from chronic exposure to agricultural chemical run-off in Mississippi. This unique population has provided a basis for the studies of vertebrate insecticide resistance (Boyd and Ferguson, 1964; Vinson et al., 1963; Culley and Ferguson, 1969). Similar to the investigations of insecticide resistance in insects, these studies have shown that vertebrate insecticide resistance is a multifactorial phenomenon involving: uptake, disposition, metabolism, and possible insensitivity at the target site (Wells and Yarbrough, 1972; Wells et al., 1973; Scales and Yarbrough, 1975; Watkins and Yarbrough, 1975; Fabacher and Chambers, 1976).

The purpose of this present study was to investigate the uptake and disposition of endrin (1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-*endo*,*endo*-1,4:5,8-dimethanonaphthalene) within the major organ and tissue compartments of insecticide-resistant (R) mosquitofish and -susceptible (S) mosquitofish. Endrin is an extremely toxic compound to freshwater fishes (Ferguson et al., 1966; Grant and Mehrle, 1973). Culley and Ferguson (1969) reported a 500-fold difference in the 48-h LC_{50} values for endrin between the R and S mosquitofish, which represents the greatest degree of resistance for any of the insecticides studied.

MATERIALS AND METHODS

Insecticide-resistant mosquitofish used in these experiments were collected from drainage ditches in Humphreys County, Mississippi, and susceptible mosquitofish from ponds located in Oktibbeha County, Mississippi. The fish were maintained in the laboratory for at least 1 week prior to use. Fish were treated with a 1% potassium permanganate solution to prevent fungal infections; however, no fish were used for experimentation for at least 24 h after permanganate treatments. All fish were fed daily Tetramin staple food diet.

Stock solutions of [1,2,3,4,10-^{14}C]endrin (California Bionuclear Corporation; sp act., 5.2 mCi/mM; 98% pure) were prepared in acetone. Nonradioactive endrin (recrystallized, Shell) stock solutions were also prepared in acetone. Treatment concentrations of 0.6, 10.2, and 314 ppb were selected. The 10.2-ppb treatment served as an equal concentration for direct comparisons between the R and S fish, while the 0.6 ppb treatment to S fish and the 314 ppb treatment to R fish provided equivalent toxicity concentration comparisons (based on 48-h LC_{50} values.

Sixteen adult female fish from each population were selected at random and were placed in an 8-L glass aquaria for 12-h equilibration periods before the insecticide was added. The endrin stock solutions were thoroughly mixed into the aquarium water. Aquaria were not aerated. Three fish from each tank were taken at 0.5, 2, 4, 6, and 8 h of endrin exposure. All fish were rinsed with acetone and blotted dry after removal from the aquaria.

Brain, liver, gill, spleen, gall bladder, intestine, ova, and kidney were removed from three fish, pooled, weighed, and homogenized in 1 mL of cold, glass-distilled, deionized water in a TenBroeck glass tissue grinder. The homogenates were digested in chlorox and added to a scintillation mixture consisting of: 0.05 g of 2,2′-p-phenylenebis(5-phenyloxazole), 4.0 g of 2,5-dipenyloxazole, 40.0 g of Cabosil M-5, 500 mL of scintillation grade toluene, and 500 mL of triton X-100. Radioactivity was counted in a Beckman "CPM-100" liquid scintillation counter. Data are expressed as nanograms endrin equivalents per milligram tissue wet weight. No differentiation was made

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Table I. Endrin Uptake in Tissues from Insecticide-Susceptible (S) and -Resistant (R) Mosquitofish Exposed to 10.2 ppb [^{14}C] Endrin (Values Are Expressed as the Mean ± SEM in ng of Endrin equiv/mg of Wet Weight of Tissues in Four-Six Treatments of Three Fish Each)

tissue	population	hours 0.5	2	4	6	8
brain	S	86.64 ± 4.65*[a]	119.37 ± 3.88**[a]	37.88 ± 2.61*	192.24 ± 19.21*	301.83 ± 30.63*
	R	5.70 ± 0.28	6.10 ± 0.45	6.44 ± 0.22	6.91 ± 0.47	6.58 ± 0.39
gill	S	34.32 ± 0.65*	22.11 ± 2.36*	11.38 ± 3.17*	78.04 ± 23.44*	89.08 ± 21.14*
	R	3.42 ± 0.29	3.73 ± 0.20	4.14 ± 0.39	6.50 ± 0.78	6.59 ± 1.38
liver	S	32.80 ± 4.18*	38.59 ± 3.50*	26.80 ± 3.07*	119.88 ± 23.63*	117.09 ± 25.01*
	R	5.19 ± 0.12	6.96 ± 0.42	9.08 ± 0.53	12.45 ± 0.61	15.14 ± 1.67
gall bladder	S	96.51 ± 4.49*	98.91 ± 7.76*	124.56 ± 16.73*	140.42 ± 7.12*	199.17 ± 29.60*
	R	2.42 ± 0.04	8.32 ± 0.95	14.94 ± 0.81	20.80 ± 0.66	20.64 ± 0.97
intestine	S	8.64 ± 0.65*	31.56 ± 3.39*	15.82 ± 0.43	30.09 ± 7.29*	56.67 ± 9.38*
	R	4.03 ± 0.30	9.55 ± 1.07	14.29 ± 1.00	12.78 ± 0.32	11.88 ± 0.19
spleen	S	205.55 ± 6.99*	315.18 ± 31.01*	284.99 ± 8.54*	288.11 ± 12.96*	821.16 ± 58.54*
	R	4.45 ± 0.42	14.96 ± 1.58	16.24 ± 0.89	24.08 ± 0.81	39.13 ± 1.16
kidney	S	13.51 ± 1.47	18.77 ± 0.38*	14.51 ± 0.20*	20.17 ± 0.52*	29.98 ± 0.62*
	R	10.22 ± 0.87	20.39 ± 0.46	50.96 ± 0.48	66.80 ± 1.78	52.63 ± 0.60
ova	S	89.08 ± 5.58*	75.13 ± 0.69*	52.63 ± 2.24*	80.81 ± 27.02*	53.24 ± 5.32*
	R	1.09 ± 0.01	3.90 ± 0.30	8.32 ± 0.24	10.43 ± 0.16	12.85 ± 0.76

[a] Significant difference between the means of the two populations (*) $P < 0.05$ and (**) $P < 0.01$ as determined by the t test.

Table II. Endrin Uptake in Tissues from Insecticide-Susceptible (S) Mosquitofish Exposed to 0.6 ppb [^{14}C] Endrin Values Are Expressed as the Mean ± SEM in ng of Endrin equiv/mg in Wet Weight of Tissues of Four-Six Treatments of Three Fish Each)

tissues	hours 0.5	2	4	6	8
brain	0.44 ± 0.04	0.59 ± 0.02	0.41 ± 0.07	0.54 ± 0.01	0.95 ± 0.02
gill	0.16 ± 0.01	0.21 ± 0.01	0.28 ± 0.02	0.19 ± 0.01	0.22 ± 0.02
liver	1.17 ± 0.38	0.31 ± 0.07	0.88 ± 0.02	0.91 ± 0.03	0.66 ± 0.04
gall bladder	0.51 ± 0.03	0.43 ± 0.02	0.61 ± 0.03	0.91 ± 0.04	2.08 ± 0.07
intestine	0.17 ± 0.01	0.52 ± 0.04	0.50 ± 0.07	0.49 ± 0.02	0.44 ± 0.03
spleen	2.45 ± 0.43	5.41 ± 0.19	1.38 ± 0.08	1.89 ± 0.03	1.91 ± 0.04
kidney	0.26 ± 0.01	0.35 ± 0.02	0.81 ± 0.01	1.81 ± 0.03	2.37 ± 0.02
ova	0.47 ± 0.06	0.44 ± 0.03	0.38 ± 0.05	0.83 ± 0.03	0.78 ± 0.03

between [^{14}C]endrin and possible ^{14}C metabolites of endrin.

Statistical analysis was done using the Student's t test to determine significant differences between the two populations at various sampling times.

RESULTS AND DISSCUSSION

At all endrin exposure levels differences in uptake and disposition patterns were found between the two fish populations. When exposed to 10.2 ppb [^{14}C]endrin, there was greater radioactivity in the S fish brain, liver, and gill tissues than in comparable tissues of the R fish (Table I). In addition, there were differences in distribution patterns between tissues from the two fish populations: brain in the S fish had the highest concentration of radioactivity, followed by the liver and gill, whereas liver of the R fish had higher radioactivity than did either brain or gill. In S fish, liver had more endrin than intestine, while in R fish, intestine had more endrin than the liver at all sampling times except the 8-h period. In both populations, gall bladder had the highest concentration of endrin. Except for the initial sampling period, kidney tissue levels of radioactivity were higher in R fish than in S fish. Kidney tissue from the S fish was the lowest in [^{14}C]endrin of all tissues examined and highest in all the R fish tissues examined. Ova had higher concentrations of radioactivity at every sampling time in the S fish than in R fish. Spleen tissue concentrated relatively more [^{14}C]endrin than any other tissue analyzed at the 10.2-ppb treatment in the S fish and was second only to kidney tissue at the same treatment level in the R fish. The spleens of many lower vertebrates are enriched with smooth musculature and are under autonomic control (Guyton, 1969). Any inhibition of the autonomic system could lead to an increased endrin pooling in the spleens as the musculature is relaxed.

In S fish endrin uptake increased with time of exposure at all timed intervals investigated with the exception of the 4-h sampling period. At this sampling period all S fish tissues with the exception of the gall bladder showed a decrease in [^{14}C]endrin. Although there is no ready explanation for this response, there is a definite two phasic response in the overall uptake pattern. The first phase is from initial endrin exposure to 4 h, followed by a second phase from 4–8 h. Further, there is an apparent increase in uptake/tissue disposition of endrin in the second phase than in the initial phase. No such pattern is seen in the R fish.

In comparing the [^{14}C]endrin uptake data between tissues from S and R mosquitofish, there were definite differences in endrin penetration. For example, the S/R [^{14}C]endrin ratios at 8 h of exposure in selected tissues were: brain, 45.9; spleen, 20.9; gill, 13.5; gall bladder, 9.7; liver, 7.7; intestine, 4.8; and ova, 4.1. The differences between the endrin uptake of the two populations is apparently the result of a barrier to endrin penetration at several levels of organization. Since the gill is the site of endrin absorption, it might be expected to serve as an initial barrier to endrin penetration (Ferguson et al., 1966). In the 10.2-ppb treatment at all sampling times, gill tissue from the R fish had less endrin than did the gill tissue of the S fish. This difference may be reflective of the uptake of endrin through the gill and, if so, shows the effectiveness of the gill barrier. The nature of the gill barrier may be analogous to the changes that render the membranes of the mitochondria isolated from the brain and liver tissues of the R fish less permeable to endrin (Yarbrough and Wells, 1971).

Table III. Endrin Uptake in Tissues from the Insecticide-Resistant (R) Mosquitofish Exposed to 314 ppb [^{14}C]Endrin (Values Are Expressed as the Mean ± SEM ng of Endrin equiv/mg of Wet Weight of Tissue of Four-Six Treatments of Three Fish Each)

	hours				
tissues	0.5	2	4	6	8
brain	10.31 ± 0.10	46.30 ± 5.61	74.22 ± 10.22	73.79 ± 6.13	68.26 ± 6.79
gill	7.19 ± 0.70	39.40 ± 4.35	47.14 ± 3.98	57.58 ± 6.15	66.25 ± 2.32
liver	14.90 ± 1.10	104.31 ± 9.98	220.11 ± 24.66	184.77 ± 8.06	358.92 ± 31.42
gall bladder	32.88 ± 3.03	52.02 ± 10.47	106.84 ± 6.69	105.65 ± 10.75	345.03 ± 35.58
intestine	7.74 ± 1.49	56.23 ± 4.52	99.25 ± 10.13	207.08 ± 8.21	354.16 ± 36.42
spleen	73.24 ± 2.59	65.44 ± 11.45	159.31 ± 16.37	236.35 ± 21.43	381.71 ± 81.45
kidney	25.35 ± 0.25	237.94 ± 16.52	258.97 ± 9.33	394.97 ± 11.03	712.51 ± 40.27
ova	19.93 ± 4.40	34.65 ± 2.84	85.73 ± 10.72	99.99 ± 8.78	87.49 ± 29.01

Table IV. Brain/Liver (B/L) and Brain/Gill (B/G) Ratios of [^{14}C]Endrin in Insecticide-Resistant (R) and -Susceptible (S) Mosquitofish Exposed to [^{14}C]Endrin for 8 h

exposure time, h	population	B/L			B/G		
		0.6 ppb	10.2 ppb	314 ppb	0.6 ppb	10.2 ppb	314 ppb
0.5	S	0.38	2.64		2.75	2.52	
	R		1.10	0.69		1.67	1.43
2	S	1.90	3.09		2.81	5.40	
	R		0.88	0.44		1.64	1.18
4	S	0.47	1.41		1.46	3.33	
	R		0.71	0.33		1.56	1.57
6	S	0.59	1.60		2.84	2.46	
	R		0.56	0.39		1.06	1.28
8	S	1.44	2.58		4.32	3.39	
	R		0.43	0.19		1.00	1.03

There was much less uptake of radioactivity by the brain, liver, and gill tissues in S fish exposed to 0.6 ppb [^{14}C]endrin as compared to tissues from S fish exposed to 10.2 ppb (Table II). [^{14}C]Endrin distribution patterns in the liver, gall bladder, and intestine were different from that seen at the higher treatment level. In general, the spleen had consistently higher levels of radioactivity than any other tissue examined. Radioactivity remained low in the kidney until 6 and 8 h.

When R fish were exposed to 314 ppb of [^{14}C]endrin, brain levels were lower than those in liver (Table III). There was no consistent distribution pattern of [^{14}C]endrin in liver, gall bladder, and intestine throughout the sampling periods. The kidney of the R fish at the 8-h sampling period had accumulated twice as much endrin as any other tissue at this treatment level.

An interesting comparison may be made between R and S fish endrin levels relative to equitoxic exposures. At these exposure levels (314 ppb for R and 0.6 ppb for S for 8 h), there was 72 times as much [^{14}C]endrin in the brains of R fish than in the brains of S fish. This could be indicative of an insensitivity at the target site (CNS) to the toxic effects of endrin in R fish. A similar suggestion has been made by Scales and Yarbrough (1975) in comparing relative ratios of endrin in tissues isolated from fish exhibiting symptons of poisoning.

The existence of an organ and/or cellular barrier can be demonstrated when brain/liver and brain/gill ratios are compared for the two populations (Table IV). When the brain levels of [^{14}C]endrin are compared to the liver levels, an organ that may serve as a major storage compartment for endrin, the resulting ratios are higher for the S fish than for the R fish (2.58 for S and 0.43 for R, at 8 h). If brain levels of endrin are compared to the gill, a tissue that has little storage capacity, but functions as the main site of endrin entry into the fish's body, the resulting ratios are significantly greater than unity in the S fish (3.4 at 8 h) and unity in the R fish. Similar comparisons of brain to liver and gill show the effectiveness of the cellular or organ barrier at other concentrations. The decreased endrin uptake into the brains of the R fish may be explained as a more effective astrocyte cell system as is believed to be the case in blood-brain barriers (Goldstein et al., 1969) or a membrane alteration analogous to the mitochondria isolated from R fish tissues (Yarbrough and Wells, 1971).

When the exposure level for S fish was raised 17-fold (from 0.6 to 10.2 ppb), gall bladder/liver ratios decreased from 3.2 to 1.7 at 8 h, while gall bladder/intestine ratios remained about the same (4.7–3.5). When the treatment dose for R fish was raised 31-fold, gall bladder/liver ratios remained approximately the same (1.5–1.0), while gall bladder/intestine ratios dropped from 2.0 to 1.0. These ratios indicate that the R fish livers may remain functionally effective in processing and excreting endrin over a wider range of insecticide concentrations. There does not appear to be any significant inhibition to biliary excretion in the S fish because there was no apparent change in the gall bladder/intestine ratios. However, in the R fish gall bladder/intestine ratios dropped, which may indicate more endrin excretion into the intestine at the higher concentration.

In summary, the accumulation and tissue distribution patterns of endrin in the two fish populations investigated were drastically different. This difference is believed to be related to the presence of a barrier to endrin penetration at the gill level as well as the organ and cellular membrane levels in the R fish. Of the barriers, the barrier to endrin penetration into the brain appears to be most effective. In addition to this barrier to penetration, an insensitivity to endrin at the target site has been indicated as a factor in the resistance phenomenon.

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Determination of *s*-Triazine Herbicide Residues in Urine: Analytical Method Development

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Analytical methodology for the separation and characterization of *s*-triazine herbicide residues in urine was developed. In the sample preparation procedure developed, a urine sample at pH 12 was extracted with hexane three times, using sodium chloride as an emulsion inhibitor. The combined hexane extract was dried by passing it through a sodium sulfate column and concentrated by rotary evaporation. The sample was transferred to a graduated centrifuge tube and further concentrated to 0.5 mL under a stream of dry nitrogen. The sample was analyzed by gas chromatography using the Hall electrolytic conductivity detector (HECD) in the nitrogen-specific mode. The method is advantageous in that it is simple and fast, lending itself to use as a routine method by technical personnel.

Analysis of *s*-triazine herbicide residues has been reported using a number of techniques and procedures. Several colorimetric methods were developed for the determination of the *s*-triazines in soils and crops. At that time, colorimetric determination was the recommended method for ten different *s*-triazines (Knuesli, 1964). The detection limit was reported to be 2 μg (about 100 ppb in the sample), but the method is neither selective among the individual triazines, nor is it free from interference. In two previously reported studies of nonradiolabeled *s*-triazines in animals, atrizine (St. John et al., 1965) and simazine (St. John et al., 1965) were assayed in cow urine and milk using the colorimetric procedure.

Procedures using thin-layer chromatography for the identification of residue samples have been described (Lawrence and Laver, 1974; Balinova, 1973; Ebing, 1973; Huss and Adamovic, 1973; Mueller, 1973), with detection limits in two cases reported as 0.02 μg/spot and 0.1 μg/spot 5 ppb in the sample). The attainment of these detection limits required the use of special developing reagents for observing the compounds on the plate.

Gas chromatography has been used for the analysis of the *s*-triazine herbicides in soils, plants, and water (Beynon et al., 1972a,b; Beynon, 1972; Kahn and Purayastha, 1975; Greenhalgh and Kavacicova, 1975; Kahn et al., 1975; Ramsteiner et al., 1974; Purkayastha and Cochrane, 1973; Young and Chu, 1973; Schultz, 1970; Lawrence, 1974a,b; Westlake et al., 1973; McKone et al., 1972).

Usually the preparation of residue samples for chemical analysis involves a straightforward partition to remove the bulk of the sample matrix, an extract cleanup process, and final sample preparation. The extraction procedures reported for the isolation of the *s*-triazine herbicides are similar to those generally employed for other pesticide residues. Some of the procedures for soil samples employ a 2-h methanol–water tumbling (Beynon et al., 1972a; Beynon, 1972), a 2-h water–acetonitrile reflux (Mattson et al., 1970), a 16-h chloroform Goldfisch extraction (Tindle et al., 1968), or a 2-h methanol Soxhlet extraction (Hill and Stobbe, 1974). More recently, an ultrasonic procedure has been described (Hill and Stobbe, 1974) in which two 15-min ultrasonic extractions were found to be comparable to a 24-h Soxhlet extraction for atrazine. The extraction of the *s*-triazine herbicides from plant materials involves maceration of the sample in a food blender with solvent, followed by filtration and partitioning. A number of different solvent systems have been employed for this work and were compared (Lawrence, 1974a). In addition, a low-temperature precipitation method has been described for the cleanup of plant residue samples (Lawrence and McLeod, 1974). All of the reported methods for the extraction of *s*-triazine herbicides from water samples involve a simple partitioning with methylene chloride (McKone et al., 1972; Tindle et al., 1968; Kahn and Purkayastha, 1975; Purkayastha and Cochrane, 1973).

While some investigators have found them unnecessary under certain GC conditions, a large number of column cleanup procedures have been used in the determination of *s*-triazine residues. The methods all utilize the successive elution with solvents of increasing polarity to selectively elute the desired compounds. The supports most successfully used are alumina (Kahn and Purkayastha, 1975; Kahn et al., 1975; Ramsteiner et al., 1974; Purkayastha and Cochrane, 1973; Young and Chu, 1973; Lawrence, 1974a; Schroeder et al., 1972), Florisil (Mestres et al., 1973; Westlake et al., 1973) and sodium bisulfate (Delley et al., 1967). Ion-exchange resins were extensively

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