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DISTRIBUTION AND METABOLISM OF ^{14}C-DDT IN THE EEL (*Anguilla rostrata*) AFTER SIX HOURS OF UPTAKE FROM AMBIENT WATER

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Individual sea-water and fresh-water adapted eels weighing from 62-151 g were placed in 2 liters of the appropriate water containing 0.1% ethanol and 1 part per million (ppm) of ring-labeled ^{14}C-DDT (Amersham/Searle). The water was maintained at 15 C and after 6 hr. the animals were bled and dissected. Aliquots of plasma and tissue were placed in 2 ml of NCS Tissue Solubilizer for assay of total ^{14}C (DDT plus any metabolites). Eighteen ml of toluene scintillation solution (4 g PPO and 50 mg POPOP/liter) were added and samples were counted in a scintillation spectrometer with automatic external standardization. Overall uptake was estimated by homogenizing the remaining carcass, i.e., the body exclusive of fluids and organs sampled directly. Uptake values for individual fish depended somewhat on body weight and averaged 56% of the ^{14}C-DDT initially present in ambient water. Aliquots of the ambient water were also counted at the end of the experiments; the remaining DDT plus metabolites averaged 0.41 ppm (range 0.15 to 0.58). Tissue distribution was very similar in sea-water and fresh-water adapted animals (Table 1). At the end of 6 hr. plasma

Table 1

LEVELS (PPM) OF DDT PLUS METABOLITES IN EELS EXPOSED SIX HOURS TO AMBIENT WATER CONTAINING 1 PPM ^{14}C-DDT INITIALLY*

	Mean ± SE for 6 Fish Adapted to:	
Tissue	Sea-water	Freshwater
Plasma	17.2 ± 3.0	19.6 ± 4.0
Liver	34.8 ± 5.8	24.2 ± 5.8
Gill	22.2 ± 2.2	20.2 ± 2.4
Brain	11.2 ± 1.0	8.8 ± 1.0
Heart	10.2 ± 1.2	8.8 ± 1.0
Gut, mucosa	6.8 ± 0.8	6.0 ± 1.0
Gut, whole	5.6 ± 0.8	3.6 ± 0.4
Gut, serosa	4.0 ± 1.0	2.0 ± 0.4
Spleen	5.0 ± 0.6	4.4 ± 0.6
Stomach	3.0 ± 0.4	1.2 ± 0.2
Bile	7.8 ± 3.8	0.6 ± 0.2
Carcass	0.6 ± 0.2	0.6 ± 0.2

* After 6 hr., levels in ambient water averaged 0.41 ppm for DDT plus any any excreted metabolites.

levels of DDT exceeded ambient water concentrations by 40-50 fold. It seems likely that uptake was largely via the gills and that transfer from gills to plasma occurred readily. Even allowing for trapped blood, the DDT plus metabolite level of most tissues exceeded that of ambient water.

Because of the high level of total radioactivity in liver (about 30 ppm) and because liver is a likely site for metabolism of DDT, we analyzed acetonitrile extracts of liver from present eels using thin layer chromatography as described elsewhere in this Bulletin (Pritchard, Guarino, and Kinter). There were no significant differences between livers from sea- and fresh-water adapted animals and most of the radioactivity (average 82%) was associated with the parent compound, DDT. About 10% of the radioactivity occurred as DDD, while DDE and DDA each accounted for about 4% of the total counts. These results suggest that DDT is metabolized at a moderate rate in the eel (on the order of 18% in 6 hr.) and that the tissue levels reported in Table 1 reflect primarily the parent DDT. In addition, the contrast between 18% metabolites in eel liver and only 5% metabolites in winter flounder liver under equivalent experimental conditions (Pritchard, Guarino, and Kinter) points to a marked species difference between these two teleost fish. Evidently, DDT metabolism and excretion will have to be evaluated fully in each species investigated.

Returning to the distribution data in Table 1, the brain level of about 10 ppm is interesting in light of the hyperactivity and convulsions observed in all eels after 4-6 hr. of exposure to 1 ppm DDT. Moreover, the gill and gut mucosa levels of about 20 and 6 ppm, respectively, provide support for the view, expounded elsewhere in this Bulletin (Janicki and Kinter), that the eventual death of eels exposed to 1 ppm DDT (after 8-10 hr.) results in part from specific impairment of osmoregulation. Under *in vitro* conditions such levels are sufficient to inhibit significant fractions of both the Na^+, K^+, Mg^{2+}-ATPases and the active Na^+ transport in these two osmoregulatory organs.

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RENAL AND HEPATIC EXCRETION OF PHENOL RED AND ITS GLUCURONIDE IN THE DOGFISH, *Squalus acanthias*.

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In 1912 Rowntree and Geraghty (Arch. Int. Med. 9, 284,) introduced phenolsulfonephthalein (PSP, phenol red) as a renal function test. In most mammals the majority of the injected dye is excreted by the renal tubules while some is excreted by the liver into the bile. Although phenol red is metabolized to a glucuronide by the rat (Hart and Schanker, Proc. Soc. Exp. Biol. Med. 123, 433, 1966), the usual clinical assay, which is also used extensively in other research, measures only the free drug. Since we had shown (Adamson and Guarino, Comp. Biochem. Physiol., in press, 1971) that the dogfish also excretes phenol red glucuronide in its bile, it was of interest to compare the hepatic and renal excretions of this compound.

Female dogfish weighing from 3-6 kg were prepared for collection of urine by ligating one end of a 10 cm length of PE 190 tubing to the urinary papillae and the free end to a rubber balloon.