Uptake, Disposition and Effects of Dietary 2,3,7,8-Tetrachlorodibenzo-p-dioxin on the Survival, Growth, Reproduction, Histology, Biochemistry and Hematology of Rainbow Trout

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Introduction

Polyhlorinated dibenzo-p-dioxins (PCDD) and compounds with similar structures, such as polychlorinated dibenzofurans (PCDF) and the non-ortho-substituted PCBs have been found to be toxic to fish (1). These compounds have been implicated in the depressed reproductive potential of salmonid fishes in the North American Great Lakes (2). The effects on these fishes were thought to be primarily on reproductive (3,4) capacity so most of the previous work was conducted either with eggs collected from the wild or in controlled laboratory studies of the effects of TCDD or similar compounds absorbed by or injected into eggs (5). Previously, no controlled laboratory studies of the effects of long-term dietary exposure to TCDD on the adult fish or subsequent effects on the survival and growth of eggs and fry, were available. The objective of this study was to determine the uptake and disposition of dietary TCDD by adult, female rainbow trout and the effects of TCDD on the Histology, Biochemistry and Hematology of the adults and on the size, quality, and hatching success of eggs and fry.

Methods

Adult, female rainbow trout (*Oncorhyncus mykiss*) were fed trout chow that had been impregnated with tritium-labelled 2,3,7,8-TCDD for one year. Exposure was started immediately after the spawning season ended. The four exposure concentrations included a control (0 pg/g wet weight) and three concentrations of TCDD: 1.8, 18 and 90 pg/g in the food. Fish were fed approximately 1.5% of body

weight on a daily basis. The specific activity of the ^{3}H-TCDD was varied proportionately so that fish in the three treatment groups would be exposed to the same radiation over the course of the study. The Specific activities for the low, medium and high doses were 541, 57 and 10.5 Bq/pg, respectively. Activity was determined by extracting the tissues with methylene chloride, followed by liquid scintillation detection.

Fish were held at 12 °C in a flow-through exposure system and the light cycle was changed weekly to match ambient conditions. The volume of the tanks was 1715 l. The flow rate through the tanks was 71.5 l/hr so that there were approximately two turnovers per day. Tanks were cleaned weekly and no ^{3}H-labelled TCDD was ever detectable in the water.

Initially ther were 35 fish exposed to each treatment. Initially the fish weighed an average of approximately 350 g. Four fish were collected from each treatment after 50, 100, 150 and 200 days.

After 200 days the fish were checked for reproductive condition every week; Those fish which were "ripe" were transferred to a holding tank and spawned within two days. Subsamples of eggs were collected for determination of TCDD concentration as well as measurments of egg size and quality. The remaining eggs were fertilized with a composite of milt from five untreated males. Fertilized eggs were transferred to Health-Vertical incubators and maintained at 14 °C. Eggs were checked weekly, to minimize handling stress. The number of eggs which died during each week were enumerated and removed.

Samples were collected for histological examination, blood chemistry and biochemical analyses. Here we will only report on P450IA1 monooxygenase activity in liver. This activity was determined fluorometrically with 7-ethoxy resorufin as the substrate.

Results

Concentrations of TCDD increased as a function of treatment and duration of exposure in all of the tissues studied (Table 1).

Mortality of adult fish occurred in a dose-dependent manner (Fig. 1). There were no significant effects on growth and only subtle effects on reproduction of the surviving adults. Induction of P450IA1 activity in the liver was time and dose-dependent (Fig. 2) and most closely related to the concentration of TCDD in the liver. The NOAEL was between 0.0 and 1.8 pg/g (ppt) in the diet and 0.0 and 0.435 pg/g, wet weight in the muscle. Few significant dose-dependent histopathological or hematological effects were observed.

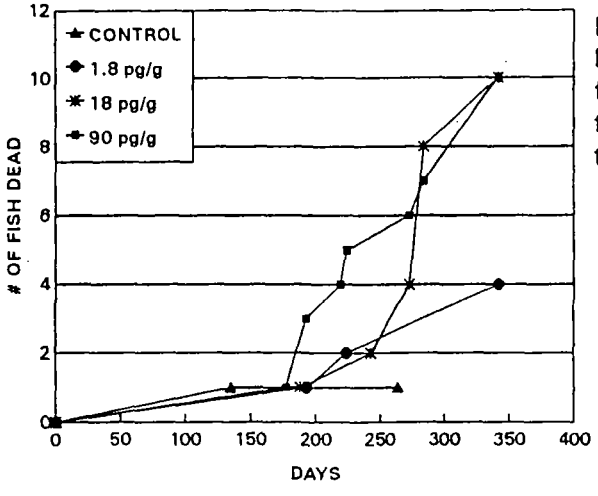

Fig. 1. Mortality of adult Rainbow Trout as a function of time exposed to the four concentrations of TCDD in the diet.

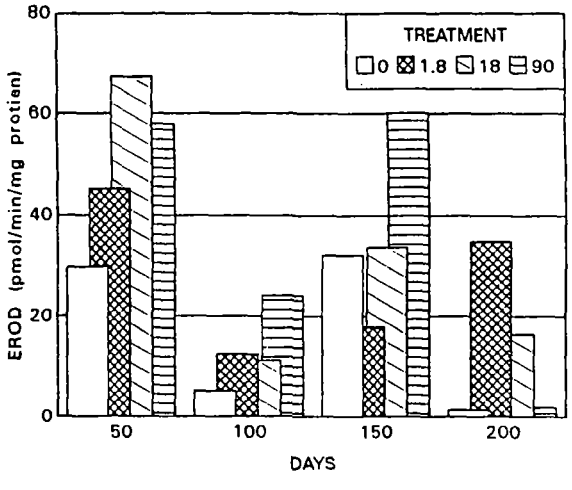

Fig. 2. Induction of EROD activity in liver of exposed fish.

Table 1. Concentrations of TCDD in Tissues.

	TREATMENT pg/g of food (wet wt.)		
TISSUE	1.8	18	90
DAY 50			
ADIPOSE	2.0	8.7	60
LIVER	0.4	1.7	–
OVARY (EGG)	0.8	4.3	15.0
MUSCLE	–	1.4	8.2
DAY 100			
ADIPOSE	0.8	15.4	84
LIVER	0.3	2.9	13
OVARY	0.5	7.9	3.6
MUSCLE	0.2	3.1	21
DAY 150			
ADIPOSE	2.3	17.3	90
LIVER	0.2	1.7	9.9
OVARY	1.1	7.9	35
MUSCLE	0.2	2.7	57
DAY 200			
ADIPOSE	2.0	15.8	92
LIVER	–	2.9	16
OVARY	1.1	5.2	47
MUSCLE	0.4	4.0	8

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