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Oral Toxicity of Chlorinated Dibenzofurans to Juvenile Atlantic Salmon

by
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Chlorinated dibenzofurans, present in some polychlorinated biphenyl (PCB) preparations, are highly toxic compounds, often lethal in rabbits after a single oral dose of 0.5-1.0 mg/kg (VOS et al. 1970). Chlorinated dibenzofurans were not detectable in high trophic-level aquatic animals which accumulated significant quantities of PCB (ZITKO 1972). The tissue levels of these compounds, associated with toxic effects are not known.

The present investigation was undertaken to determine the tissue levels of chlorinated dibenzofurans causing mortality in juvenile Atlantic salmon *(Salmo salar)*.

Dry fish food (trout chow, Purina) was spiked with di-, tri-, tetra-, and octachlorodibenzofuran. The compounds were dissolved in a small volume of benzene, the solution was diluted with pesticide-grade hexane and mixed thoroughly with the food. The solvent was evaporated on a rotatory evaporator at 22°C, and the food was kept in vacuum (27 psi) overnight at room temperature to remove last traces of the solvent. A control batch of food was treated under the same conditions with the solvent only.

Food and fish tissues were analysed as described (ZITKO 1972). The concentration of di-, tri-, tetra-, and octachlorodibenzofuran in the food, calculated and (determined) was 2.7 (2.1), 5.7 (4.4), 2.8 (2.2), and 9.1 (9.7) µg/g, respectively.

Juvenile Atlantic salmon (35 fish) were kept in a 20-liter fiberglass tank in running water (500 ml/min) at 10°C, 16-h light, 8-h dark photoperiod, and fed the spiked food several times a day. A control experiment using the solvent-treated food was carried out under the same conditions. Average length of the fish was 10.9 cm, average weight was 10.7 g.

Fish in the control experiment suffered no mortalities whereas median mortality (LT50) of 120 ± 30 days occurred among fish on the diet containing chlorinated dibenzofurans. The experiment was terminated after 140 days and fish still alive (20%) were sacrificed.

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Muscle and gut of fish that died between the 81st and 136th day of the experiment, and of fish surviving for 140 days were analysed and the results are presented in Table 1.

The levels of PCB and DDE in the control fish were not significantly different from those reported in Table 1. The concentration of lipid in the control fish was 2.85% (whole fish).

TABLE 1

Analyses of fish tissues (data on wet weight basis).

Sample	Number analysed	µg/g PCB* Mean	PCB* SD	DDE Mean	DDE SD	OCDF** Mean	OCDF** SD	Lipid % Mean	Lipid % SD
Muscle									
Dead	5	0.15	0.03	0.04	0.00	0.03	0.01	0.85	0.17
Live	3	0.14	0.02	0.05	0.02	0.01	0.00	0.77	0.06
Gut									
Dead	4	0.52	0.10	0.10	0.01	0.21	0.14	1.21	0.27
Live	3	0.66	0.13	0.20	0.05	0.02	0.04	2.02	0.31

*as Aroclor® 1254
**octachlorodibenzofuran

Di-, tri-, and tetrachlorodibenzofuran were not detectable in any of the samples. The detection limit for these compounds is approximately 0.02 µg/g on wet weight basis (ZITKO et al. 1972). The dead fish contained about 10 times higher concentration of octachlorodibenzofuran in the gut and 3 times higher concentration in the muscle than the fish alive after 140 days.

Octachlorodibenzofuran is less readily absorbed from food than PCB. Juvenile Atlantic salmon fed a diet containing Aroclor® 1254 at a level of 10 µg/g had a PCB concentration of 3.5 µg/g in whole body after 120 days (ZITKO and HUTZINGER 1972).

It is possible that as in the case of chlorinated dibenzodioxins (ROWE et al. 1971), dibenzofurans with intermediate degrees of chlorination are the most toxic compounds. The observed levels of octachlorodibenzofuran may be only a measure of the amount of ingested food, not associated with the toxic effects. It is interesting to note that the lipid level in the gut is higher in the live fish than in the dead fish.

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The presented data indicate that in fish, as well as mammals, chlorinated dibenzofurans are much more toxic than PCB. Nothing is known about the input and possible formation of chlorinated dibenzofurans in the environment. Thus far, we were not able to detect these compounds in Aroclor® preparations.

The results indicate that because of the high toxicity and low residual levels of chlorinated dibenzofurans, a more sensitive analytical method is required. The mechanism and kinetics of the formation of these compounds should be determined and toxicological studies of dibenzofurans with known chlorine substitution patterns should be carried out.

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