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Metabolic Effects of a Polychlorinated Biphenyl (Phenoclor DP6) on Mullets, *Chelon labrosus*

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Contamination of aquatic organisms by polychlorinated biphenyls (PCB) has been widely indicated (Jensen 1984 ; Delval et al. 1986). However, information is still limited concerning the biochemical and physiological effects of such compounds on aquatic vertebrates.

Alterations in reproduction and in liver structure (Jensen et al. 1970) as well as changes in blood glucose (Silbergeld 1974) and tissue lipid compositions (Addison 1982) have indicated that the general metabolism was modified in fish exposed to PCBs.

In previous work carried out with estuarine fish (Chelon labrosus) exposed to Phenoclor DP6 we have investigated the tissue accumulation and distribution (Narbonne 1979 a,b) and indicated the resulting induction of hepatic enzyme activities (Narbonne and Gallis 1979 ; Narbonne et al. 1987). The aim of the present work is to provide information on both appearance and reversibility of some general metabolic alterations occuring in mullets exposed to Phenoclor DP6.

MATERIALS AND METHODS

Mullets (Chelon labrosus) of uniform weight (81 g $\pm$ 6) were caught in the Arcachon bay. They were acclimated 30 days in tanks containing flowing, charcoal - filtered and oxygenated sea water (from the Arcachon bay, salinity 33 % , temperature 18°C) and in starving conditions. Then, seven groups of four animals were distributed into 20-L glass aquaria (one group per aquarium) and fish were fed a commercial fish food "Aqualim" (2% of body weight) every 2 days. Then, food was contaminated by Phenoclor DP6 (48 mg/kg food weight, purchased from Prodelec-Society, France) and added to the experimental aquaria while the control group still received uncontaminated food. Phenoclor DP6 is a commercial PCB mixture containing 6 chlorine atoms per molecule. After 30 days of contamination, fish were given a normal diet for another period of 30 days. Fish were sampled at every time period of 8, 15 and 30 days of each contamination and depuration experiment. Phenoclor DP6 extraction and dosage were carried

out as previously described (Narbonne 1979a).
Concentration of total proteins was evaluated in the tissues by the colorimetric biuret reaction (Schuel and Schuel 1967). Total RNA was measured as described by Fleck and Munro (1962). The glycogen in liver, heart and muscles was assayed by the enzymatic method of Murat and Serfaty (1974). Blood glucose was determined by a colorimetric procedure based on the oxidation-reduction of potassium ferricyanide (Fe+++) to potassium ferrocyanide (Fe++), the decoloration being quantified at 420 nm. Total lipid content of carcass was estimated according to the method of Abraham et al (1964). Results are expressed as percentages from control values. Control values are means $\pm$ SEM (n=4).

RESULTS AND DISCUSSION

All the results concerning the biochemical parameter determinations are summarized in Figure 1 (A-F). Within the experimental period, lipid reserves were not modified as indicated by the stable lipid content (around 2% of wet weight) of fish carcass (Figure 1 F) suggesting that dietary conditions were satisfying. Although no mortality was found, mullet contamination by Phenoclor DP6 induced important alterations affecting glucose and protein metabolism.

Glycohaemia readily increased (+ 222%, P>0.001) within 8 days exposure and remained at high level after 30 days exposure (+ 122%). This biochemical response is not specific to PCB contamination as it has been observed in fish for physiological stresses such as changes in water temperature (Murat and Parent 1974) or for exposure to heavy metals (Narbonne et al. 1975). Concerning organochlorine compounds, Silbergeld (1974) has shown a 63% increase of glycohaemia with Etheostoma nigrum exposed 30 days to 2.3 ppb dieldrin. In our conditions this high glycohaemia decreased to control level (59 $\pm$ 3 mg glucose/100 ml) within 30 days of the depuration period (Figure 1 A). However, Johansson et al (1972) still observed a significant hyperglycohaemia after 38 days in trout previously exposed to Clophen A 50.

Glycogen content increased in liver, heart, white and red muscle (+ 45%, + 28%, + 190%, + 47% from control values respectively) within the exposure to PCB. This period corresponded to maximum concentration of blood glucose. After one week of depuration period control values were recovered in heart and muscles. However the liver glycogen content was strongly increased (3 times than in control animals). Thus maximum values in liver corresponded to minimum values in blood heart and muscles indicating differences in glycogenesis between liver and muscles. Cortisol is known as a main hormone acting for glycogenesis. Camp et al (1974) have observed a 28% decrease in blood cortisol in the catfish (Ictalurus punctatus) exposed 4 h to 8 ppm Aroclor 1254 solubilized in Corexit 7664. Therefore, the observed glycogenesis might not be linked to an increase in blood cortisol.

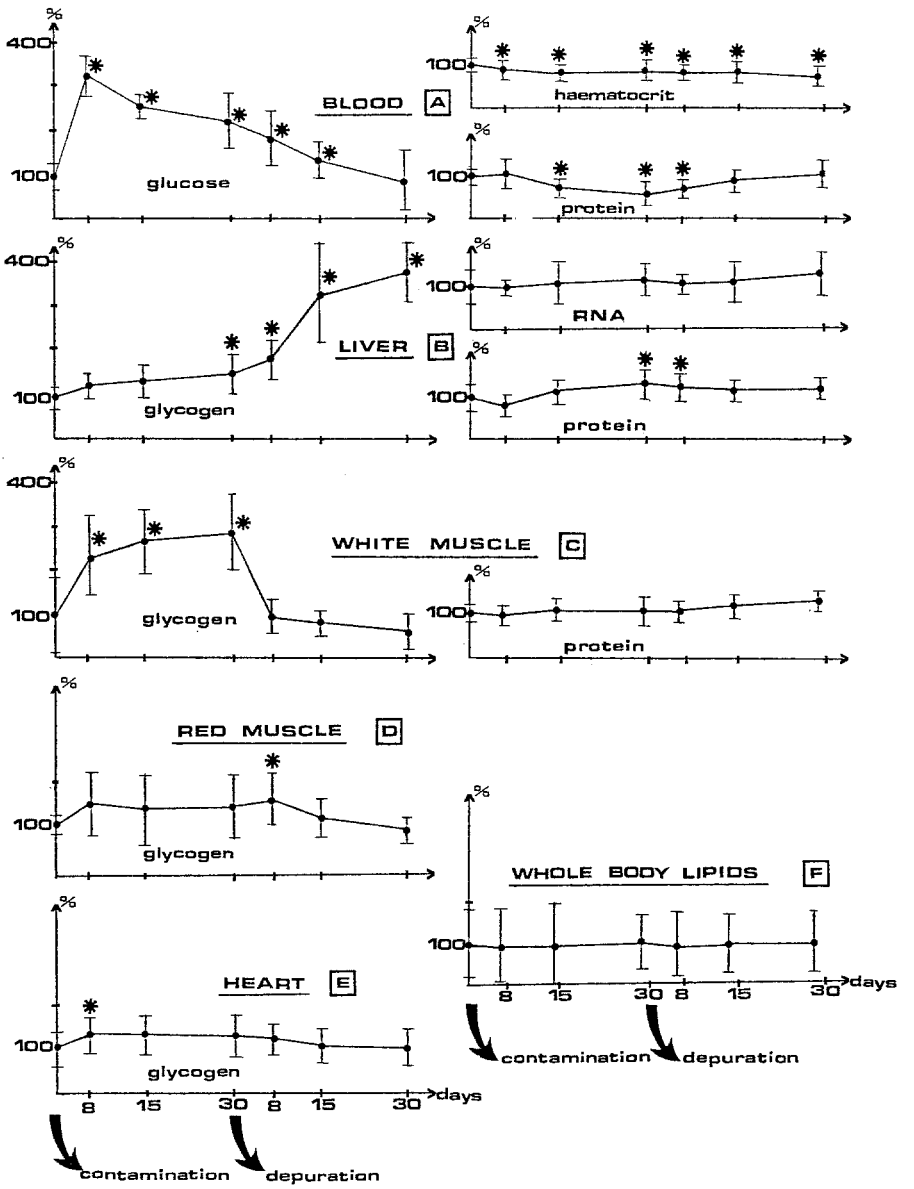

Figure 1. Effect of Phenoclor DP6 on blood [A], liver [B], white muscle [C], red muscle [D], heart [E] and body lipids [F] in fish. Values are percentages from control level, means ± confidence intervals ($P < 0.05$), $n=4$. Level of significance: $P<0.05$ (*) Student t test.

Furthermore, the glycogen storage, resulting from PCB contamination, might involve a decrease in blood cortisol. Then, it is suggested that both hyperglycohaemia and decrease of glycolysis are caused by a poor glucose consumption. According to this hypothesis, Campbell et al (1974) have shown a in vivo decrease in ATPase activities in gill and kidneys of trout exposed to DDT.

As shown in Figure 1 A Phenoclor DP6 contamination caused a decrease of both haematocrit and proteinaemia (- 17% and - 56% from control values respectively). Johansson et al (1972) observed a pronounced anaemia in trout exposed to Clophen A 50 while Camp et al (1974) indicated a 35% decrease in total serum proteins in catfish exposed to Aroclor 1254. Two weeks after the end of PCB exposure normal value of proteinaemia was recovered. As previously observed for glucose metabolism the changes of protein content in blood and in liver were inversely related. Total hepatic RNA did not change, suggesting an increase of hepatic proteosynthesis. Indeed, the inducing effect of polychlorinated biphenyls on hepatic proteins has been largely observed in mammals (Turner and Green 1974 ; Narbonne 1979c) and in fish (Narbonne and Gallis 1979 ; Narbonne et al. 1977).

Results from this study of mullets exposed to PCB show an increase of both glycohaemia and muscular glycogen and a decrease of plasmatic proteins occuring along with an elevation of hepatic protein content. These alterations disappeared within 8 days after intoxication was stopped wherease PCB body burden remained elevated (4.3 mg DP6/kg), suggesting that the PCB kept in lipid reserves, cannot exert any toxic effects on the parameters. However, we observed a delayed effect represented by an increase in hepatic glycogen.

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