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# Effects of Short-Term Sublethal Cadmium Exposures on the Biochemical Composition of Dogfish Liver

The physiology and metabolism of fishes can be disturbed by the presence of pollutants in the water. Amongst them, heavy metals have been appointed as responsible for a wide range of alterations. As far as Cd is concerned, changes in respiration, decrease in both hematocrit and hemoglobin concentration, changes in water-electrolyte status and variations of elemental composition have been described (5, 7, 8). Previous works showed changes in dogfish gill metabolism after sublethal Cd exposures (12). The concentration of 50 mg/l of Cd in water triggered the anaerobic pathways in gill tissue together with a decrease in the respiratory rate. These changes were apparent after two days of exposure but control levels recovered at fourth day. A similar pattern was observed on blood parameters after 25 mg/l Cd exposure (13). Since most of metabolic changes and further compensatory responses can be detected through alterations in liver, the present work analyses the effects of sublethal Cd exposure on liver biochemical composition. The time exposure is longer than previous studies in order to assess whether this central metabolic organ is affected by the metal challenge.

Dogfish, *Scyliorhinus canicula*, keeping and feeding were carried out as in previous works (11). Two metal exposures were tested: 50 mg/l for 4 days (n=16) and 25 mg/l for 10 days (n=10). Other 34 fishes were used as controls. Every 3 days the water was changed (with minimum disturbance to fish) in order to maintain Cd concentrations and prevent its deposition. After killing the fish and excising the liver, the different parameters were determined as follows: Glycogen content by digestion, centrifugation and reaction with anthrone. Protein concentration by a modification of Lowry's method (1). Lipids were extracted by the method of BLIGH and DYER (2) and determinated colorimetrically after reaction with phosphovanilline. One-way analysis of variance followed by a Duncan test were used to check statistical differences between groups. Significance was assumed at 0.05.

Table I shows means of measured parameters. Only the protein content is significantly different between treatments, being the 50 mg/l levels lower than those of control and 25 mg/l groups.

Glycogen depletion in both liver and muscle after metal exposure has been described in trout (6) and catfish (10), being

Table I. *Glycogen, protein and lipid values (means ± SEM), expressed as mg/g fresh tissue, and total caloric amount for the three treatment groups.*
Caloric energy content (CEC) (kcal/g) were calculated from the usual relationiships (i.e. 4.2 kcal/g glycogen, 9.5 kcal/g lipid, 4.9 kcal/g protein). In brackets, there are the percentage contribution to the caloric content from each component.

| Group | Fishes | Glycogen | Protein | Lipid | CEC |
|---|---|---|---|---|---|
| Control | 34 | 0.73±0.11<br>(0.1 %) | 6.54±0.41<br>(1.1 %) | 306.6±26.7<br>(98.8 %) | 2.95 |
| 25 mg/l 10 days | 10 | 1.39±0.34<br>(0.2 %) | 7.26±0.32<br>(1.4 %) | 267.5±48.1<br>(98.4 %) | 2.58 |
| 50 mg/l 4 days | 16 | 0.90±0.25<br>(0.2 %) | 4.99±0.40<br>(1.0 %) | 246.1±32.5<br>(98.8 %) | 2.23 |

attributed to an extra demand for energy or to a hormone and/or enzyme imbalance created by the metal, since a decrease in plasma insulin associated to liver glycogen reduction has been observed (14). The present results agree with those from other authors showing no changes or slight increases in liver glycogen content. These results were explained as a stimulated glycogen synthesis together with the enhancement of gluconeogenesis, as shown by the activity increase of enzymes involved in the transformation of non-carbohydrate metabolites into carbohydrates. In dogfish, an increase in plasma glucose concentration (13) and gill anaerobic metabolism (12) has been observed. These changes could indicate of glycogen depletion during the second and third days of treatment, followed by a recovery or, alternatively, by both carbohydrate synthesis and glycogen depletion taking place during metal exposure.

Exposure to metals has been shown to produce metal accumulation in liver as well as metallothionein (MT) synthesis stimulation (3). However, MT production could not significantly affect the amount of protein in liver. Assuming the relationship between Cd and MT (9), the increase in such proteins would account for 0.2 mg/g. The protein reduction observed in the 25 mg/l group could be attributed to an overall metabolic alteration, especially if the importance of protein metabolism in fish is considered.

The lack of variations in lipid concentration could be due to the high content of lipids in elasmobranch livers, a great part of which does not play a metabolic role and has the mission of guarante buoyancy. If changes are produced in the metabolically active lipids, they are hidden in the inalteration of the whole amount. Although,. a tendency to a decrease with treatment intensity was observed. This feature becomes clearer from the caloric energy content (table I) given by each component. Higher Cd concentration corresponds to lower levels of energetic content. If percentage of lipids without a metabolic function were discarded, these differences would probably increase, thus showing a stronger effect of the metal on the liver energetics. As a whole, the present results indicate that subacute concentrations of Cd, such as the ones used in this work, would not strongly affect the liver biochemical composition, although a tendency to decrease energetic resources is shown after stronger and longer exposures.

Key words: Cadmium, Liver metabolism, Dogfish, Elasmobranchs

Palabras clave: Cadmio, Metabolismo hepático, Pintarroja, Elasmobranquios.

## References


1. Besandoun, E. and Benstein, L.: *Anal. Biochem.*, 70, 241-247, 1976.
2. Bligh, E. G. and Dyer, W. J.: *Can. J. Biochem. Physiol.*, 37, 911-917, 1957.
3. Hidalgo, J. and Flos, R.: *Comp. Biochem. Physiol.*, 83C, 99-103, 1986.
4. Houston, A. H. and Keen, J. E.: *Can. J. Fish. Aquat. Sci.*, 41, 1.829-1.834, 1984.
5. Koyama, J. and Ozaki, H.: *Bull. Jpn. Soc. Sci. Fish.*, 50, 199-203, 1984.
6. Lowe-Jinde, L. and Niimi, A. J.: *Arch. Envir. Contam. Toxicol.*, 13, 759-764, 1984.
7. McCarty, L. S. and Houston, A. H.: *J. Fish. Biol.*, 9, 11-19, 1976.
8. Muramoto, S.: *Envir. Pollut.*, 24A, 125-133, 1981.
9. Olsson, P. E. and Haux, C.: *Aquat. Toxicol.*, 9, 231-242, 1986.
10. Srivastava, D. K.: *Toxicol. Lett.*, 11, 135-139, 1982.
11. Torres, P., Tort, L., Planas, J. and Flos, R.: *Comp. Biochem. Physiol.*, 83C, 89-92, 1986.
12. Tort, L., Torres P. and Hidalgo, J.: *Mar. Pollut. Bull.*, 15, 448-450, 1984.
13. Tort, L. and Torres, P.: *J. Fish. Biol.*, 32, 277-282, 1988.
14. Wagner, G. F. and McKeown, B. A.: *Can. J. Zool.*, 60, 2079-2084, 1982.


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