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Changes in Tissue Lipid and Cholesterol Content in the Catfish *Clarias batrachus* (L.) Exposed to Cadmium Chloride

S. R. Katti and A. G. Sathyanesan

Department of Zoology, Banaras Hindu University, Varanasi-221005, India

Cadmium (Cd) is a potent toxic element which causes chronic toxicosis in exposed industrial workers and miners. In mammals including man, Cd activates or inhibits several enzymes, causes developmental abnormalities, hypertension, anemia, cardiac enlargement, enteropathy, severe bone mineral loss, gonadal atrophy, and kidney damage (Vallee and Ulmer 1972; Friberg et al. 1974; Fox 1982; Kopp et al. 1982). Cd intake is through food, water, air and smoking. In animals Cd is stored in the liver, kidney and muscles. Vegetables grown in soil having Cd or those supplemented with Cd containing fertilizers or urban sewage sludge, are found to contain Cd. Fish exposed to Cd exhibited testicular damage (Sanglang and O'Halloran 1972) and changes in the activity of several enzymes (Jackim et al. 1970; Larsson 1975; Sjobeck and Larsson 1978). Although Cd is known to be present both in freshwaters and sea in minute traces, its biological function has not been well defined. Low levels in food may have biological implications, if the essential nutrients with which it interferes are in marginal amounts (Sandstead 1977). Very little is known about the effect of Cd on the physiology of fishes. In the present study, changes in the lipid and cholesterol contents of the brain, liver and gonad of *C. batrachus* exposed to 50 ppm of Cd chloride for 135 days are reported.

MATERIALS AND METHODS

Over 40 *C. batrachus* used in the present investigation were caught wild, purchased from local fishermen at Varanasi and acclimated to laboratory conditions for 10 days before initiating the experiment. 20 were exposed to 50 ppm of Cd chloride for 135 days. The water containing Cd chloride was

changed every alternate day after feeding. They were sacrificed by decapitation and the tissues were dissected out immediately, weighed and processed for the assays employed. Total lipid estimation was determined according to the method of Folch et al. (1957). Cholesterol was quantified with the aid of Liebermann-Burchard reaction as described by Kabara (1966).

RESULTS AND DISCUSSION

The catfish is a seasonal breeder which spawns during the monsoon months extending from late June to early September. The experiment was initiated on the first of February, when the gonads were in the stage I resting phase. When the experiment was terminated on June 15th, the gonads of the control fish were maturing, stage III or matured stage IV condition. In those exposed to Cd chloride the ovary was in stage I phase with occasional scattered stage II oocytes. The testis resembled that of stage I condition with solid cords of spermatogonia, some of which exhibited necrotic changes. In the controls the testis showed different stages of spermatogenesis with sperm in the lumen. The experimental fish exhibited a decline in the total lipid content of the brain and gonads, which was significant in the former ($P < 0.01$, Table 1, Fig.1). The cholesterol depletion was marked both in the brain and ovary ($P < 0.001$ and $P < 0.05$). However, in contrast the hepatic lipid and cholesterol recorded a significant elevation ($P < 0.05$ and $P < 0.01$, Table 1, Fig.1).

Table 1. Tissue lipid and cholesterol

S.No.	Tissue	Groups	Lipid Mg/g Tissue	Cholesterol Mg/100 g Tissue
1	Brain	Control	66.78±2.74	4425.64±189.23
		Exptl.	49.32±3.37	1151.38±123.68
			$P < 0.01$	$P < 0.001$
2	Liver	Control	58.53±4.29	2656.04±106.09
		Exptl.	70.82±3.08	3403.60±146.59
			$P < 0.05$	$P < 0.01$
3	Ovary	Control	47.24±5.92	843.93±68.62
		Exptl.	39.54±5.24	402.66±78.77
			N.S.	$P < 0.05$
4	Testis*	Control	9.7	397.84
		Exptl.	6.48	286.68

*Value from 5 pooled testes

The biochemical basis of chronic Cd toxicity is little known (Fox 1982). In spite of the extensive studies made on human subjects on Cd toxicity, very few papers published allow conclusions drawn about dose relationships (Piscator 1982). With over 20000 species living in widely varied habitats, fish could form good markers of water pollution. However, our knowledge about the effects of metal pollutants on fish is meagre. As reported in Cd exposed rats (Dutt et al. 1978; Saksena and Lau 1979), and in the brook trouts, alteration in testicular androgen synthesis was noticed (Sanglang et al. 1972,1974). Sanglang et al. (1972) also observed depletion of lipids in the testis of Cd treated brook trout. Reports on the Cd induced changes in the ovary are meagre. In rats (Kar et al. 1959) and Koel (Sarkar et al. 1976) Cd is known to cause ovarian lesions and follicular atresia. In Cd exposed C. batrachus, lipid and cholesterol depletion was evident both in ovary and testis and their growth was also significantly retarded.

In mature rats Cd exerts its neurotoxic effects on the sensory ganglia (Gabbiani 1966; Arvidson 1980) and in the immature ones haemorrhagic lesions and destruction of fibres and cells particularly in the granular layer of the cerebellar cortex were obvious (Gabbiani et al. 1967). Mercury, lead and Cd are reported to interfere with cerebral pyruvate metabolism and simulate symptoms of thiamine deficiency (Review Vallee and Ulmer 1972). Heteropneustes fossilis exposed to 12.5 Mg/l Cd exhibited a decline in glycogen content in brain whereas a smaller dose of 5 to 7.5 Mg/l caused a significant increase (Srivastava 1982). In the present study, Cd induced reduction in levels of brain cholesterol and lipid suggest impairment of some brain metabolism about which little is known.

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