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Effects of Aldicarb on the Blood and Tissues of a Freshwater Fish

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Aldicarb is a commonly used synthetic organic pesticide which ranks 27th in the pollution potential rank order of 82 formulations (Kelso et al. 1978). It is highly soluble and toxic systemic poison. The maximum acceptable toxicant concentration of aldicarb for the fathead minnow, Pimephalus promelas, was found to lie between 78–156 µg/l (Pickering & Gilliam 1982). The 96-h LC50 for this carbamate has been established at 1370 µg/l and 2420 µg/l for the fathead minnow and a cyprinid, Barbus conchonius, respectively (Pickering & Gilliam 1982, Kumar & Pant 1984).

In the recent years, several approaches have been used to monitor deleterious effects of pesticide poisoning in fishes (Eller 1971, Grant & Mehrle 1973, Sastry & Siddiqui 1983, Gill & Pant 1985). A survey of literature, however, reveals a serious lack of information on the adverse effects of aldicarb on the aquatic biota. This study was, therefore, undertaken to identify changes in the hematological and biochemical profile of a freshwater fish, Barbus conchonius, exposed chronically to commercial formulation of aldicarb.

MATERIAL AND METHODS

Specimen of Barbus conchonius (Order Cypriniformes) were hand netted from the local lake and transferred to the laboratory tanks in plastic buckets. They were kept for a week in the PVC 400 L storage tank for acclimatization under natural photoperiod (13L/11D approx.), water temperature (range 10.5-19°C,mean 15°C), and food ad libitum. For experimental exposure to aldicarb, healthy fish, average weight 5 g, were transferred to 35 L glass aquaria in four groups of 24 individual each. Group I and II were exposed for 15 and 30 days to aldicarb at a nominal concentration of 484 µg/l (20% of the 96-h LC50). The remaining III and IV groups served as controls. The commercial preparation (Temik, 99% purity) of aldicarb [2-methyl-2(methylthio) propionaldehyde O-(methylcarbamoyl) oxime] was used to prepare an aquous solution of the desired concentration. During the exposure period, the average measurements for pH, hardness, and dissolved oxygen were 7.4, 359 mg/l (as $CaCO_3$), and 7.8 mg/l, respectively. Experimental tanks were constantly aerated, and the test solution replaced biweekly.

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Fish were fed throughout the exposure period except for two days prior to sacrifice.

Upon completion of the stipulated exposures of 15 and 30 days, fish were sacrtificed without being anaesthetized, and the blood and tissue samples collected. Blood samples were drawn into heparinized tuberculin syringes by heart puncture. Routine hematological procedures were followed to estimate the erythrocyte numbers, hemoglobin (Hb), and hematocrit (Hct). The blood glucose (Nelson 1944), cholesterol (Zlatkis et al. 1953), free fatty acids (FFA) (Novak 1965), liver and ovary cholesterol (Rosenthal et al. 1950), and muscle and liver lipids (Oser 1965) were measured photometrically. Data from control and experimental groups were statistically compared by Student's t-test.

RESULTS AND DISCUSSION

The effects of environmental stressors on the peripheral blood of fishes are well documented in the literature. In the carp, Cyprinus carpio, experimental cobalt poisoning lead to elevated erythrocyte counts and hemoglobin whereas the number of leucocytes declined (Frovola 1960). Helmy et al. (1978) described hemopathological changes in the Kuwait mullets, Liza macrolepis, exposed to graded doses of copper, lead, and mercury for 96 h. In this species, lead at 1.15-18.3 mg/l induced significant polychromasia and 1+ anisocytosis besides causing eosinophilia and lymphopenia. A variety of adverse effects have been demonstrated in other piscine species following exposure to heavy metals and pesticides (Gardner & Yevich 1970, Lowe-Jinde & Niimi 1984, Gill & Pant 1985,1986).

Monitoring of blood parameters, both cellular and non-cellular, may have considerable diagnostic value in assessing early warning signs of pesticide poisoning. In our study, chronic aldicarb exposure caused moderate polycythemia (35.3 and 15.2% increase over control values) together with a rise in Hb content (Table 1). The Hct showed 27.7 and 29.1% increase over the control values after 15 and 30 days, respectively. Alterations in the erythrocyte counts have also been described in other fishes under different forms of stress (O'Connor & Fromm 1975, Helmy et al. 1978). Holmberg et al. (1972) reported an increase in the hemoglobin level in the eel exposed to pentachlorophenol and related it to a hypermetabolic state. The polycythemia observed in B. conchonius may be causally related to an enhanced erythropoiesis. Such a response appears to be more of a compensatory nature rather than a direct effect of aldicarb per se on the blood cell production. Aldicarb is known to cause branchial lesions in this species (Kumar & Pant 1984) which may seriously impair the oxygen uptake. Therefore, possibly it is the hypoxemia that triggers an exodus of erythrocytes from the hemopoietic loci in an attempt to compensate for the reduced oxygen carrying capacity of the blood.

Table 1. Hematological and biochemical status of Barbus conchonius exposed to nominal 20% (484 µg/l) 96-h LC50 concentration of aldicarb for 15 and 30 days.

Variable	Exposure period				
	Control[a]	15 days	% change	30 days	% change
Erythrocytes ($millions/mm^3$)	2.43 ± 0.08[b]	3.29 ± 0.02**	35.3	2.80 ± 0.09*	15.2
Hemoglobin (g/dl)	11.24 ± 0.35	12.42 ± 11.4*	10.4	11.40 ± 0.22	1.4
Hematocrit (%)	35.90 ± 1.37	49.90 ± 0.55**	27.7	46.30 ± 3.02*	29.1
Blood glucose (mg/dl)	98.9 ± 5.46	57.6 ± 8.50**	-41.7	96.9 ± 0.93	-2.03
Blood cholesterol (mg/dl)	269.9 ± 8.11	435.9 ± 55.0*	67.0	336.9 ± 26.7*	29.1
Liver cholesterol (mg%)	175.6 ± 26.0	506.4 ± 93.6*	188.2	192.5 ± 14.6	9.6
Ovary cholesterol (mg%)	140.6 ± 7.15	199.1 ± 5.01**	41.6	154.3 ± 13.3	9.7
Liver lipids (mg/100 mg)	11.31 ± 0.37	43.85 ± 1.88**	287.7	25.20 ± 0.33**	122.8
Muscle lipids (mg/100 mg)	13.88 ± 0.43	10.32 ± 0.51**	-25.6	9.37 ± 0.77**	-32.4
Blood free fatty acids (µEq/ml)	5.20 ± 0.27	9.50 ± 0.65**	82.6	9.02 ± 1.36*	73.4

[a]Pooled values collected at 15 and 30 days; [b]mean ± SE of 8 samples; *$p < 0.05$; **$p < 0.01$

Grant and Mehrle (1973) reported hyperglycemia and elevated glycogen reserves in the rainbow trout fed endrin over an extended period. These biochemical effects of endrin toxicosis were ascribed to inhibition of glycogenolysis or increase in glycogenesis or gluconeogenesis. In the catfish, Heteropneustes fossilis, acute methyl parathion poisoning induced hyperglycemia followed by increase in hepatic glycogen content (Srivastava & Singh 1981). Sastry and Siddiqui (1983) exposed snakehead, Channa punctatus, to 0.2 µg/l endosulfan for upto 60 days and found a decrease in blood glucose and total plasma protein with a concomitant rise in lactic acid and hemoglobin.

The blood glucose level in B. conchonius was significantly lowered after 15 days exposure. The observed hypoglycemia could be due to aldicarb-induced modulation of insulin secretion from the islet beta cells and/or a reduction in the intestinal absorption of glucose in the brush border. Hyperplasia of the pancreatic islets during chronic endrin exposure and intestinal lesions following cadmium poisoning have been described in the trout, Salmo clarki and the catfish, Heteropneustes fossilis, respectively (Eller 1971, Sastry & Gupta 1979).

Result of present investigation suggest a marked influence of aldicarb intoxication on the body lipid profile. A significant hypercholesterolemia was manifested in the experimental fish after both 15 and 30 days exposure to aldicarb. The increase in liver and ovary cholesterol was, however,more pronounced at 15 days although the values at 30 days remained higher, albeit insignificantly, than those in the control fish. Liver rather than the skeletal muscles or the ovary, appears to be the target of deleterious effects of aldicarb as reflected in the total lipids and cholesterol levels. Thus, at 15 days, while the muscle lipid content fell by 25.6%, the increment in liver lipids was 287.7%. Similarly, cholesterol in the ovary registered an increase of 41.6% at 15 days compared to 188.2% in the liver. Free fatty acids in the blood were consistently above control levels both at 15 (82.6%) and 30 days (73.4%) suggesting a sustained mobilization of stored lipids. Fatty infiltration of liver is very common in cases of exposure to chemical stressors, particularly the heavy metals and xenobiotics (Rouiller 1964, Couch 1975). Sastry and Gupta (1979) observed localizationof a considerable amount of lipids in the veins and its deposition in the neighbouring areas of liver of cadmium-exposed Channa punctatus.

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