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Levels of Blood Glucose and Tissue Glycogen in Two Live Fish Exposed to Industrial Effluent

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The pollution of rivers and lakes by the effluents discharged by various industries, has been found to cause heavy fish mortality (GANAPATI & ALIKHUNHI 1950, DAVID 1956, DAVID & RAY 1956, VENKATRAMAN 1966) due to hypoxia, high levels of organic substances, inorganic salts and heavy metals. Recently a decrease in oxygen consumption was found in fish, Labeo rohita, exposed to industrial effluents (unpublished data). The probable physiological changes in fish exposed to industrial effluents have not yet been thoroughly investigated. Therefore, an attempt was made to study the changes in blood glucose and tissue glycogen of fish subjected to the effluents.

MATERIAL AND METHODS

The effluent sample was collected from Sanathnagar Industrial area near Hyderabad, India. There are about 300 major and minor industries manufacturing a variety of products including chemicals, drugs, explosives, food products, machinery and paints, etc., which discharge their effluents into a channel joining Hussain Sugar lake, which is situated in the heart of twin cities of Hyderabad and Secunderabad. The Board for Prevention and Control of Water Pollution, Andhra Pradesh, India, reported the presence of high load of heavy metals like Hg, Zn, Cd, Co, Cr, Pb, Cu and Fe, in the effluents.

Lethal concentration (LC 50) values, determined for both the fish, H. fossilis and C. batrachus which were collected from Hussain Sugar lake, were found to be at 70% concentration of the effluent. A batch of 6 fish of each species was kept in effluent samples in separate tanks at 50% concentration for a period of 15 days. Simultaneously another batch of fish was kept in a tank containing normal tap water (Control) for the same period. After 15 days fishes of both the batches were sacrificed and glycogen levels of liver, kidney, muscles, gills and heart were estimated by the method of KEMP et al. (1954). Blood glucose was estimated by Nelson and Somogyl method as given in HAWK et al. (1954). Blood glucose and tissue glycogen levels were expressed as mg/100 ml of blood and mg/g wet weight of tissue, respectively.

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RESULTS

Some chemical parameters of the effluent used in the present study and normal tap water were analyzed and the data are presented in the Table 1. When compared to normal tap water, the effluent contained nil oxygen, but a several fold increase was noticed in all other parameters.

Table 1

Chemical parameters of the effluents sample collected from Sanatnagar Industrial area near Hyderabad

(Values expressed as mg/l)

	pH	O_2	CO_2	HCO_3^-	Chlorides	Sulphates
Normal tap water	7.9	6.5	6.0	176	24	38.4
Effluent	7.1	Nil	96	570	1000	84.4

Date of collection of the effluent April 19, 1977.

There was 92.2% and 95.3% increase in blood glucose level of H. fossilis and C. batrachus, respectively, after exposure to effluents. In C. batrachus a significant decrease in glycogen level was found in liver, muscles and gills, whereas in heart and kidney there was a slight increase. A significant decrease in glycogen level in H. fossilis was observed only in liver and gills (Table 2). In muscles of H. fossilis there was a 13.2% decrease in glycogen level which is not significant.

DISCUSSION

In the present study, the raise in blood glucose level in both the fish may be due to enhanced glycogen breakdown in liver, probably because of anaerobic stress and toxic substances present in the effluent. Simultaneously more tissue glycogen is utilized resulting in fall of glycogen level. NAKANO and TOULINSON (1967) observed an elevation of blood glucose level in rainbow trout in response to osmotic and physical stress. In H. fossilis, subjected to mercury intoxication, a decrease in glycogen level of liver, muscles and kidney was reported (QAYYUM & SHAFFI 1977). HODSON (1976) found increase in glycogen utilization in muscles of rainbow trout exposed to lethal concentrations of zinc. Some of the substances like nitrates, chlorides and ammonia were shown to oxidize haemoglobin to methaemoglobin thereby inducing anaerobic stress (SMITH & RUSSO 1975, GROTHE & EOTON 1975).

Table 2

Changes in the levels of blood glucose and tissue glycogen in the fish, *Heteropneustes fossilis* and *Clarias batrachus* exposed to industrial effluent

(Values are means of six individual observations)

	Blood glucose (mg/100 ml)	Glycogen (mg/g wet weight)				
		Liver	Gill	Muscle	Heart	Kidney
1. *Heteropneustes fossilis*						
Control	30.8 ± 5.03	70.2 ± 23.37	7.62 ± 0.56	2.72 ± 0.62	35.5 ± 2.09	9.05 ± 2.45
Experimental	59.2 ± 5.88	22.1 ± 3.20	5.24 ± 1.51	2.36 ± 0.67	34.15 ± 1.96	9.09 ± 2.11
% change	± 92.2	- 68.5	- 31.2	- 13.2	- 3.8	+ 0.44
't' test	p < 0.001	p<0.01	p<0.05	NS	NS	NS
2. *Clarias batrachus*						
Control	25.8 ± 2.82	33.8 ± 6.10	3.6 ± 0.45	1.8 ± 0.64	13.52 ± 5.18	3.72 ± 0.82
Experimental	50.4 ± 4.66	13.5 ± 4.19	2.4 ± 0.33	9.92 ± 0.37	15.4 ± 5.17	3.80 ± 0.69
% change	+ 95.3	- 60.15	- 33.3	- 48.8	+ 13.95	+ 2.15
't' test	p 0.001	p<0.01	-<0.02	p<0.05	NS	NS

NS - Not significant

It is also possible that there may be imbalance in pancreatic hormones involved in carbohydrate metabolism, due to damage of pancreas caused by toxic substances in the effluent. In Channa punctatus administration of cobalt chloride and cobalt nitrate was shown to induce hyperglycemia by degranulation and vacuolization of the pancreatic α cells in the initial stages and damage of β cells in later stages (KHANNA & GILL 1975). Further studies are in progress in our laboratory to elucidate the mechanism pertaining to the metabolic impairment.

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