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Research Article

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Effect of cadmium on fresh water teleost, *Heteropneustes fossilis* (Bloch)

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Abstract: *Heteropneustes fossilis*, the fresh water teleost fishes were exposed to sub lethal concentration 6.9 mg/l of Cd for 24, 48, 72, 96, hours. The order of cadmium accumulation rate was found in these tissues were liver>gills> kidney. It was concluded that the fish liver and gills exhibited highest accumulation of heavy metals. The results showed significant fall in all the biochemical constituents in all the tissues except glucose apparently suggest that organism's response to the toxicant stress. The accumulation of cadmium and significant fall in the level of these biochemical constituents in these tissues was found to increase directly. Cadmium blocks the oxidative metabolism in the tissues leading to an altered energy status in the fish as the toxicant effect carbohydrate metabolism in liver, brain and gills. A significant increase in glucose and lactate levels was observed while the glycogen and pyruvate contents were significantly decreased due to the toxic stress.

Keywords: Cadmium, Glucose, Glycogen, *Heteropneustes fossilis*, Lactate, Pyruvate.

Introduction

Cadmium, a non essential heavy metal is highly toxic environmental pollutants. Its exposure leads to various diseases such as cardiovascular, hypertension, chronic kidney disease, lung and prostate cancer. Cadmium in diet is associated with high risk of endometrial, breast and prostate cancer (Julin *et al.*, 2012) as well as osteoporosis (Engström *et al.*, 2012) in humans. It effects the signal transduction of estrogen and MAPK (mitogen-activated protein kinases) signaling pathway at low dose (Ali *et al.*, 2010). Exposures to low levels of cadmium can cause DNA damage and stress in common carp (*Cyprinus carpio* var. color) (Jia *et al.*, 2010). Cadmium, one of the twenty three heavy metal toxicant, may be transported to aquatic ecosystems as a result of both natural (weathering and erosion) and anthropogenic (industrial and agricultural) activities. The rate of cadmium and zinc toxicity in Bull trout and Rainbow trout increases with rise in water temperature from 80 to 120C (Hansen *et al.*, 2002). Cadmium is taken by the fish through the gills and

intestines and transported to other organs via blood and accumulated there or might excreted out. Cadmium accumulation in these organs appears to be related to the presence of cadmium-binding molecules called metallothioneins (Dallinger *et al.*, 1997). Cadmium is an essential trace element that contributes to the surface of more than 300 proteins which play an important role in the growth, reproduction, development and immune system of the fish. The excess amount of cadmium was found to interfere with many protein and carbohydrate metabolism by inhibiting the enzymes involved in the processes. In the present study to evaluate the cadmium toxicity, edible fish *Heteropneustes fossilis* were exposed to the sublethal concentration of cadmium in tissues such as gills, kidney, liver and muscles and its impact on biochemical constituents like glucose, glycogen level in these tissues. Also such exposed when consumed as food lead to the deposition of the heavy metal cadmium in the soft tissues of the human body leading to exposure health effects.

Materials and Methods

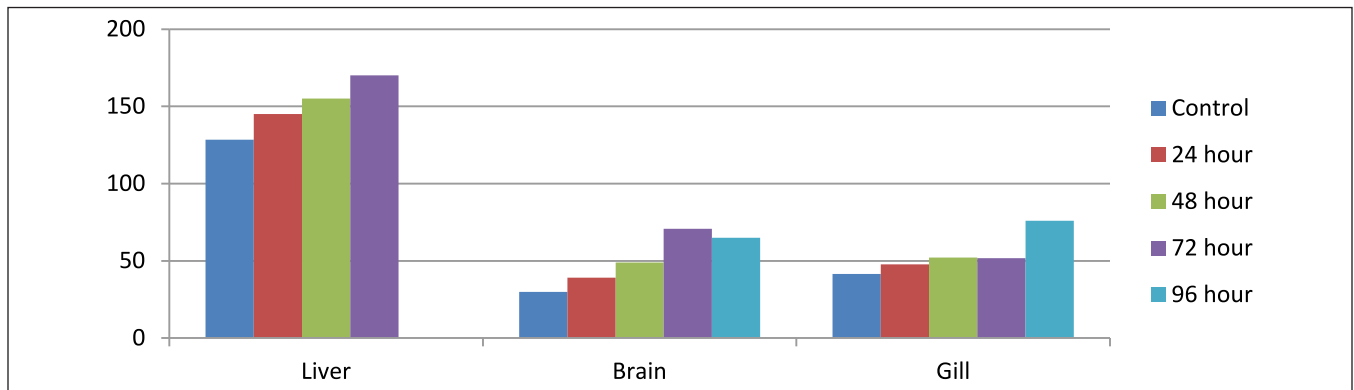
Present study was conducted on adult *Heteropneustes fossilis* (Bloch) (weighing 50±5gm; length 20-22 cm) collected from local resources they were transported in well aerated condition to laboratory, and were treated with 0.1KMNO₄. They were kept in properly aerated glass aquarium & acclimatized in the laboratory condition for a fortnight under natural photo period 11L:13D and temperature 20±5°C (pH 7.1 ± 0.5) before commencement of experiment. All the fish were fed with a formulated diet and natural food. To rectify the deficiencies caused by the artificial diet, natural foods like earthworm and minced goat liver were provided thrice a week. The fishes were fed ad libitum once a day. After

feeding, the remaining food particles were immediately removed from the aquarium.

After acclimatization the fish was exposed to sublethal concentration 6.9 mg/l of cadmium 24, 48, 72, 96 hours. Liver, brain and gill were isolated from the normal and cadmium exposed fish and were used for biochemical estimation. Free glucose was estimated by the method of Mendel *et al.*, (1954) while glycogen by the method of Carroll *et al.*, (1965), lactic acid by the method of Huckabee (1961) and pyruvic acid by the method of Friedman and Hangen (1942).

Results and Discussion**Table 1:**

	Tissue	Control	Exposed to Cadmium (Cd)			
			24 Hours	48 Hours	72 Hours	96 Hours
Glucose (µg /100 mg wet wt.)	Liver	128.32±0.11	145.09±0.64	155.00±0.11	170.01±0.11	178.11±0.10
		-	-11.96	-20.10	-41.06	-38.01
	Brain	29.91±0.42	39.11±0.33	48.88±0.17	70.82±0.46	64.96±0.69
		-	-35.10	-65.95	-46.87	-78.00
	Gill	41.46±0.19	47.64±82	52.19±0.46	51.78±0.38	76.03±0.69
		-	-20.11	-32.06	-47.38	-69.02
Glycogen (µg /100 mg wet wt.)	Liver	551 ± 0.92	424.19±0.09	393.06±0.11	324.90±0.01	285.00±0.82
		-	-23.09	-29.01	-39.92	-46.05
	Brain	132.03±0.83	111.01±0.33	80.96±0.83	70.69±0.15	60.96±0.11
		-	-15.96	-37.69	-46.69	-56.69
	Gill	99.42±0.69	82.42±0.30	67.01±0.74	51.90±0.38	42.06±0.42
		-	-17.87	-32.64	-46.78	-58.56
Pyruvate (µg /100 mg wet wt.)	Liver	58.52±0.54	42.10±0.63	38.96±0.69	19.69±0.69	15.89±0.28
		-	--18.28	-33.33	-48.06	-56.06
	Brain	20.19±0.91	15.69±0.19	10.92±0.38	7.69±0.69	5.01±0.38
		-	-11.19	-22.01	-40.96	-63.78
	Gill	57.42±0.14	46.64±0.1	39.28±0.38	29.96±0.11	22.98±0.11
		-	-12.98	-28.05	-50.01	-56.74
Lactate (µg /100 mg wet wt.)	Liver	0.15±0.29	0.20±0.74	0.38±0.14	0.40±0.74	0.58±0.32
		-	33.69	79.69	87.0.74	101.06
	Brain	0.24±0.11	0.32±0.24	0.43±0.60	0.60±0.11	0.74±0.91
		-	36.78	42.44	69.06	92.05
	Gill	0.14±0.78	0.18±0.04	0.20±0.82	0.29±0.29	0.46±0.11
		-	29.32	50.82	106.69	159.29



Graph 1: Glucose (µg /100 mg wet wt.)

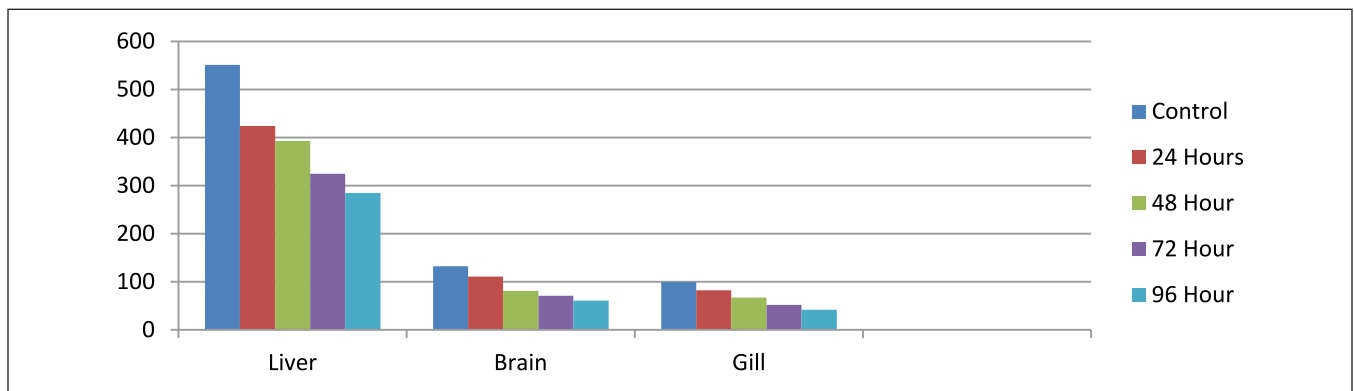
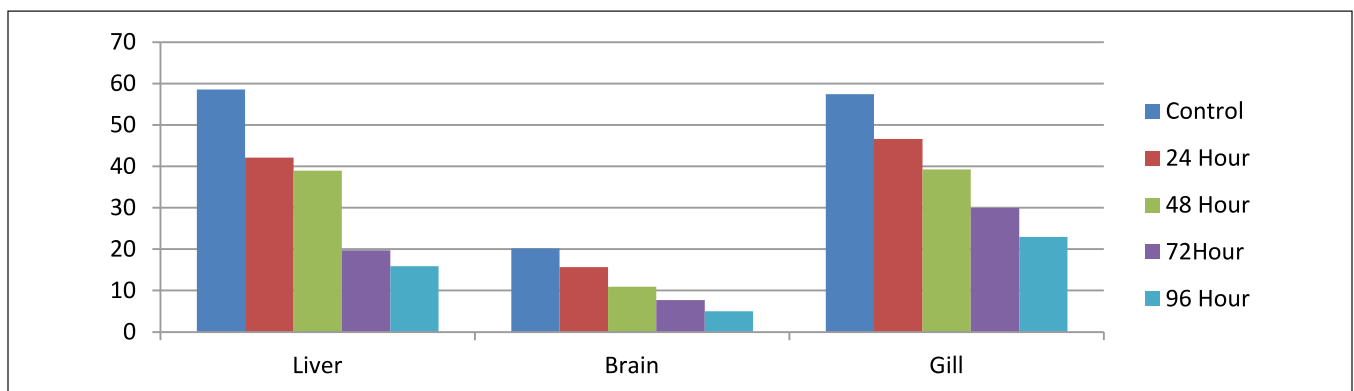
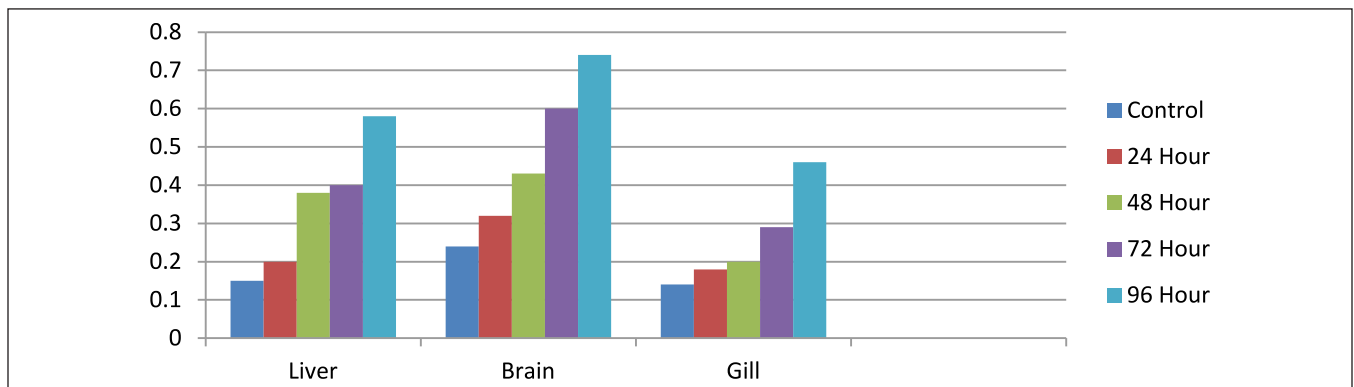


Fig 2: Glycogen (µg /100 mg wet wt.)



Graph 1: Pyruvate (µg /100 mg wet wt.)



Graph 1: Lactate (µg /100 mg wet wt.)

The data presented in Table 1 and Graph 1&2 indicates that the glycogen content decreased while glucose content increased in liver, brain and gill significantly after 24, 48, 72 and 96 hours of exposure to cadmium. It was reported that cadmium reduced glycogen content in the fish, *T. mossambica*. In one study, cadmium concentrations in catfish muscle tissue increased with increasing concentrations in their food (and significantly reduced fish growth) (Ruangsomboon and Wongrat, 2006). These studies taken together with the present data, may suggest the possible onset of an increased rate of glycogenolysis due to the heavy metal toxicity. Further, (Table 1 and Graph 3 & 4) it is clear from the data that cadmium could significantly decrease pyruvate content while markedly elevated lactate levels in all the three tissues studied. As it was suggested that heavy metal could induce hypoxia or anoxic conditions the observed increase in lactate content could be due to enhanced anaerobiosis in these tissues due to toxic stress. Increased glycogen mobilization, enhanced pyruvate conversion to lactate and inhibition of citric acid cycle enzymes clearly suggest increased oxidation of glucose through the anaerobic glycolytic pathway to provide energy for the fish. This is associated with significant reduction in the mitochondrial oxidative metabolism due to the derangement of mitochondrial function directly or indirectly by the pollutants, cadmium.

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