Journal of Advances and Scholarly Researches in Allied Education
Vol. 16, Issue No. 9, June-2019, ISSN 2230-7540

Study on Biochemical Changes in Water and Catla Catla Exposed to the Toxicant Cadmium Chloride

Dr. Akhil Abhishek[1]* Dr. Akhilesh Kumar[2]

[1] Assistant Professor of Zoology, Simtech College, Patna

[2] Associate Professor of Zoology, A. N. College, Patna

Abstract – One of the 23 heavy metal toxicants cadmium (Cd) is commonly utilized for the manufacture, non-corrosive design of Ni-Cd batteries, in the metal & mining industries, & dentistry. Cd is emitted to soil, surface & ground water systems in considerable volumes through industrial effluents. Standard biochemical process in the five tissues, i.e. muscles, gills, liver, cardiac & kidney, healthy fish & fish exposed to 96 hour LC50 (lethals) & sublethals (1/10th lethal for 7 days of exposure) of cadmium c have been determined by the levels of the 5 biochemical components of glucose, glycogen, total proteins & Free aminoacids. Except for glucose, studies indicated that fish grown in aquatic systems close to industrial locations does not have the expected nutritional value. The outcomes showed an important fall in all biochemical elements in all tissues. The high glucose levels seem to indicate the response of the organism to toxic stress. Fish used as food also contributes to heavy metal accumulation in the soft tissues of the body leading to effects of exposure.

Key Words – Toxicity, Heavy Metal, Cadmium Chloride, Biomagnification, Catla catla

----------X----------

INTRODUCTION

Fish farmers face a big problem with infectious diseases in aquaculture, resulting in severe losses. The recent rise in intensive practises in aquaculture has brought considerable attention to the awareness of the different diseases of the fish to be treated or prevented. The prevalence of diseases in fish farms is commonly illustrated by a number of factors related to methods of rearing, environmental conditions & improvements. A. Hydrophila is a gramme negative, mobile rod recorded in freshwater fish species as an opportunist pathogen, & it is known to be geographically widely distributed. A. The signs of relaxed belly, losing skin, deep dorsal ulcers & widespread haemorrhages on the ventral part of fish have been shown by Catla & Cirrhinus mrigala hydrophila. Closely monitored in India due to losses in the economy reported in recent decades are intensive freshwater aquaculture diseases. Cd's toxicity is due to its capacity to generate reactive oxygen species which can serve as signalling molecules for inducing gene expression & apoptosis (Waisberg et al . 2003), deplete endogenous cadmium, such as other toxic metals, can establish conditions leading to arterial & tissue inflammation and cause more Ca2 + to be drawn to the area as a tampon, thus contributing to hardening.

MATERIALS AND METHODS

The catla catla fingerlings used in this research were collected from a Nandivelugu, Guntur Dt, local fish farm (average weight: 6-7 grammes). The laboratory adapted & Andhra Pradesh , India for approximately a week in 50 litre plastic pools. Groundwater utilized in the fish tanks was dissolved with a pH of 7.2±0.1, oxygen 8.0±0.3mg / L & 95.0±5.0mg / L with bicarbonates. After Renewal bioassay LC50 was estimated, & probit analysis method of Finney(1971) was measured. In 5 tissues, i.e. Muscle, Gill, Livers, Heart & Kidneys, normal methods of healthy fish (Control) & those of fish exposed to sublethal & lethal concentrations of Cadmium chloride (Merck) were measured for biochemical constituent agents such as Glucose, Glycogen, Total Proteins & Free Amino acids. A tenth of the lethal level was taken as a sub-lethal dose and for 7 days prior to biochemical analytical slaughter, the fish were exposed to the sub-lethal dose. The Kemp et al.(1954), Lowry et al.(1951), Moore (1954), Pande et al.(1963) separately measure of starch, protein , lipid & free amino acid.

RESULTS AND DISCUSSION

Figure 1 indicates the percentage mortality at various levels of cadmium chloride across various

exposure times. Cadmium chloride LC50 value for fish C. catla was calculated by the simple graphic method (Fig . 2), Probit graphic method (Fig . 3) & unweighted regression method (Finney, 1971) & values from the three methods are 4.57 mg / L, 4.89 mg / L &4.13 mg / L and so on. Thus the average LC50 calculated is 4.533 mg / L. The formula for the dose-mortality regression line was determined to be Y = 2.65X + 3.368.

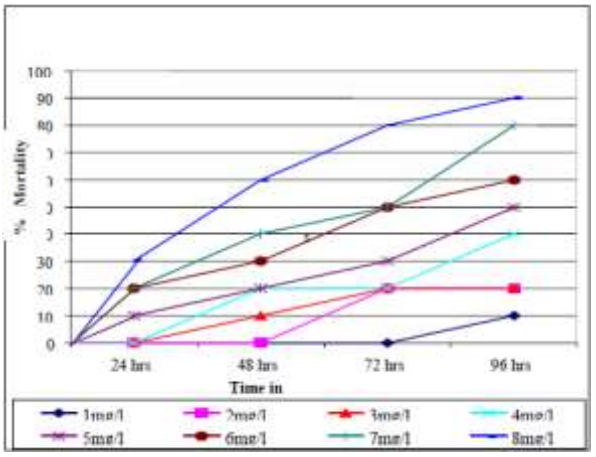

Figure. 1 % Mortality Vs Exposure time

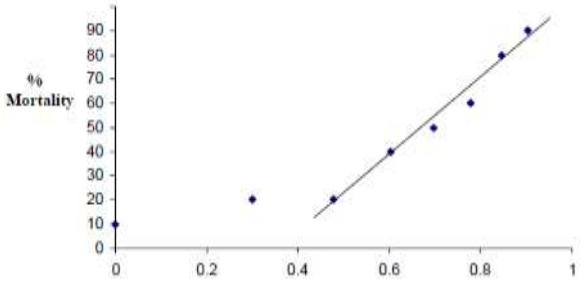

Figure. 2 Simple graphic method

(Log conc. Vs % mortality)

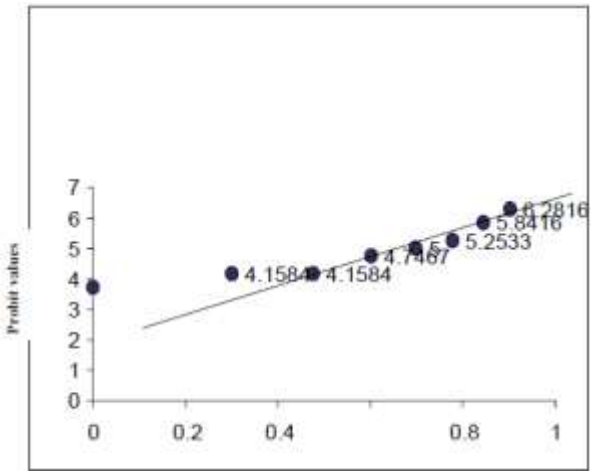

Figure. 3 Probit Graphic Method

Log Concentration

The rate of behaviour & mortality of C. Catla was originate to rely upon exposure period as well as concentrations of the toxicant during our experiments. C. Catla, as some of the other fish and crabs, are very sensitive. These can be seen from the 96-hour-LC50 recorded in Poecil reticulata (30.4 mg / L in a Static Bio-Test System) for Uca rapax (Mehmet Yilmaz et al . , 2004), 43 mg / L for Scorpaena guttata (Brown DA et al . ,1984), Zanders 1996). The metal 's impact is also dependent on the size of the animal, water salinity , temperature & animal form. While the species survive the initial attack of toxins / pollutants as of their defensive adaptations, injuries from the gradual exposure even at small dosages occur later as the resistance of the organism weakens as a outcome of ageing. Furthermore, the state & reaction of the test organism to the quantity of metal entering the body, the retention frequency, & excess rate influence heavy metal's toxic effect.

Figure 4 Behavioral of exposed fish changes. Spinal curling & vertical movement of fish is represented because of a lack of balance. Due to total loss of balance, the fish turned back & died, as shown in the portrait.

Figure. 5 Haemorrhage of dead Cd chloride exposed fish. Hemorrhage was found around the eye orbits if this fish was dissected.

The levels in the control fish & fish exposed to sublethal & lethal doses of cadmium chlorides of numerous biological components in mg / g of a wet tissue are listed in Table 1 & variations in percent increase & decrease are described in Table 2. The analysis shows that the blood glucose levels of muscles, gill, liver, heart , & kidneys increased by 7 days at the end of the 96-hour exposure and sublethal concentration, but not in all tissues. Biomolecules, including glucose, glycogen, full proteins, free amino acids & 5 tissue lipids, have been identified in the control fish: Glucose: L > M > K > H > G; Glycogen: L>M>K>G>H; Total Proteins: M>L>K>H>G; Free aminoacids: L=M=G=H=K; Lipids: M>L>G>K>H. The rise in tissue glucose while decreasing in the exposed fish's tissue glycogen indicates that the reserves of glycogen have been used for stress. Increase in serum glucose levels in fish under stress was documented by Bedii &Kenan (2005), Chowdhury et al . (2004). This can be due to many factors & one of them is the reduction in the particular activity of certain enzymes including phosphofructokinase, lactate dehydrogenase & citrate kinase that increase the ability of glycolysis (Almeida et al., 2001).

Table 1: Catla catla exposed to Cd chloride toxicant variations in biochemical input levels in fish.

Organ	Exposure			Biochemical constituents expressed in mg/g wet weight of the tissue
	Control	Sub-Lethal ($1/10^{th}$ of 96 h – LC_{50})	Lethal (96 h – LC_{50})	
Muscle	9.36±0.54	11.12±0.54**	14.64±1.15***	Glucose
	6.94±0.68	6.34±0.6ns	5.62±0.38**	Glycogen
	36.56±0.67	36.0±0.28ns	34.56±0.67**	Total Proteins
	100.8±14.8	76.8±6.6*	56.8±10.7***	Lipids
	-	-	36.8±14.11	Free amino acids
Gill	2.96±0.54	4.64±0.88**	7.28±0.87***	Glucose
	5.62±0.38	5.34±0.31ns	4.3±0.82*	Glycogen
	8.72±0.44	7.68±0.59*	7.44±0.60**	Total Proteins
	68.8±4.38	54.4±5.36**	47.2±6.57***	Lipids
	-	-	-	Free amino acids
Liver	11.52±0.44	13.52±0.66**	15.12±0.87***	Glucose
	9.18±0.38	7.7±0.78**	6.72±0.82***	Glycogen
	30.0±0.49	23.36±0.60***	19.16±0.67***	Total Proteins
	91.2±6.57	74.4±5.36**	59.2±6.57***	Lipids
	-	-	68.0±12.7	Free amino acids
Heart	6.08±0.44	8.16±0.88**	9.12±0.66***	Glucose
	3.42±0.42	3.1±0.34ns	2.52±0.34*	Glycogen
	14.08±1.0	11.52±0.66**	9.2±1.13***	Total Proteins
	49.6±5.36	35.2±4.38**	27.2±6.57ns	Lipids
	-	-	-	Free amino acids
Kidney	7.76±0.36	9.44±1.00**	10.72±0.36***	Glucose
	6.12±0.49	5.04±0.79*	3.56±0.37***	Glycogen
	20.96±0.78	19.04±0.72ns	11.92±0.59***	Total Proteins
	52.0±8.48	42.4±5.36ns	35.2±4.38**	Lipids
	-	-	78.8±9.85	Free amino acids

Table 2: Differences in the per cent rise in biochemical constituents (↑) or % decrease (↓) in C. Catla exposed in comparison to fish control, to sublethal & lethal cd chloride.

Tissue	Glucose (%↑)		Glycogen (%↓)		Total Proteins (%↓)		Lipids (%↓)	
	Sub-Lethal	Lethal	Sub-Lethal	Lethal	Sub-Lethal	Lethal	Sub-Lethal	Lethal
Muscle	18.8	56.4	8.64	19.02	1.53	5.47	23.8	43.65
Gill	56.75	145.9	4.98	23.48	11.92	14.6	20.93	31.39
Liver	17.36	31.25	16.12	26.79	22.13	36.13	18.42	35.08
Heart	21.64	38.14	9.35	41.83	18.18	34.65	29.03	45.16
Kidney	34.21	50.0	17.64	41.83	9.16	43.12	18.46	32.3

The level of glycogen is the highest in liver since it is the main metabolism organ of carbohydrate and the muscle in animals. Liver glycogen is used to store & export blood glucose hexose units, & muscular glycogen is a readily accessible source of glycolysis hexase units inside the Body. A decrease in the level of glycogen shows its fast deployment to satisfy increased energy requirements for fish exposed to toxicants by the glycolysis or hexosis Monophosphate route. It is supposed to be due to the inhibition of hormones that contribute to glycogen synthesis. Decreases in liver & muscle glycogen levels are supported by reports of earlier workers (Dubale 1981; Sastry 1984). The results are also shown in the following reports: Glycogen stocks have been depleted to K > L > H > M > G in fish exposed to the sublethal dose while the order in fish exposed to the deadly dose of the toxicant has been changed to K > L > H > G > M. This might be due to gills using Glycogen reserves rapidly when revealed to lethal concentration to respond to respiratory strain. The protein-rich muscle forms a mobility-destined and non-metabolistic mechanistic tissue. Liver is also abundant in proteins as a core for different metabolisms. The total protein content has been decreased in all tissues of the exposed fish. When exposes to sublethal dose L > H > G > K > M was shown, the order of reduction in different tissues was observed while lethal exposure changed the order to L > H > G > M. The high levels of the toxicant influence the kidneys & lower levels affect the liver. The reduce in protein as seen in the latest research in most fish tissues could be due to the metabolic use of ketoacid to the pathway of gluconeogenesis for glucose synthesis or to free amino acids for protein synthesis or osmic and ionic control (Schmidt Nielson, 1975). It may also be due or a part of heavy metal-induced apoptosis, to the production of warm shock proteins or destructive free radicals Free amino acids & fish exposed to sub-lethal concentration were not detected in checking tissue. However the fish exposed to lethal concentration, and not in the gills or the heart, in the muscles, liver , & kidney tissues have been detected. The acute effect of the lethal cd concentration on these tissues could be presumed to be this. De smet (2007) description that proteolysis is designed to enhanced the role of proteins in Cd stress energy production.

Lipids also are the way energy such as glycogen can be stored. Lipid concentrations were also reduced in fish's tissue exposed to sub-lethal & lethal cd chloride concentration. Previous researchers documented the impact of cadmium on the lipid content (Dubale 1981; Fabien Pierron 2007). In fish exposed to sublethal intakes the decreased lipid level is as H > M > G > K > L & order is H > M > L > K > G in fish exposed to lethal diet.

CONCLUSION

Acute tests of cd toxicity in the edible carp, Catla catla showed major changes in fish biochemicals such as glucose, glycogen, total protein, lipid & free

amino acids. The fish which were previously exposed to lower concentrations of heavy metals have better resistance to higher concentrations of these heavy metals. This might be due to the adaptive response which is characteristic of vertebrates. Also, the adults are found to be more susceptible to the toxicant than the fingerlings. Unlike organic pollutants, these heavy metals could not biodegraded. Even though some of the micro-organisms can be used for the biosorption, the higher concentrations of these metals are toxic to those micro-organisms even. This process is specific and depends on the cell wall composition of the micro-organism. Additional work wants to be done to identify & utilize the effective strains of microbes for effective removal of heavy metal toxicants to allow for safe utilize for human consumption of crop-fish with a high nutrient value.

REFERENCES

[1]. Almeida JA, Novelli EL, Dal Pai Silva M, Junior RA (2001). Environmental cadmium exposure and metabolic responses of the Nile tilapia, Oreochromis niloticus. Environ Pollut. 114 (2): pp. 169-175.

[2]. Arno Kaschl, Volker Römheld and Yona Chen (2002). Cadmium binding by fractions of dissolved organic matter & humic substances from municipal solid waste compost. J.Environ.Qual. 31: pp. 1885-1892.

[3]. Audry S, Schafer J, Blanc G, Jouanneau JM (2004). Fifty-year sedimentary record of heavy metal pollution (Cd,Zn,Cu, Pb) in the Lot River reservoirs (France). Environ Pollut. 132(3): pp. 413-426.

[4]. Bedii CiCiK and Kenan ENGiN (2005). The effects of Cadmium on levels of glucose in serum and glycogen reserves in the liver and muscle tissues of Cyprinus carpio (L., 1758); Turk.J.Vet.Anim.Sci., (29): pp. 113-117.

[5]. BR Kiran, TR Shashi Shekhar, ET Puttaiah and Y Shivaraj (2006). Trace metal levels in the organs of Finfish Oreochromis mossambicus (Peter) and relevant water of Jannapura lake, India. Journal Environ.Science & Engg. 48(1): pp. 15-20.

[6]. Brown DA, Bay SM, Alfafara JF, Hershelman GP and Rosenthal KD (1984). Detoxification/toxification of cadmium in scorpionfish (Scorpaena guttata): Acute exposure. Aquatic Toxicol. 5(2): pp. 93-107.

[7]. Bryan GW and Langston WJ (1992). Bioavailability, accumulation and effects of heavy metals in sediments with special reference to United Kingdom estuaries: A Review. Environmental Pollution. 76 (2): pp. 89-131.

[8]. Chowdhury MJ, Pane EF, Wood CM (2004). Physiological effects of dietary cadmium acclimation and waterborne cadmium challenge in rainbow trout: respiratory, ionoregulatory, and stress parameters. Comp Biochem Physiol C Toxicol Pharmacol., 139(1-3): pp. 163-173.

[9]. Chrastny V, Komarek M, Tlustos P, Svehla J (2006). Effects of flooding on lead and cadmium speciation in sediments from a drinking water reservoir. Environ Monit Assess., 118(1-3): pp. 113-123.

[10]. De Smet H, Blust R (2001). Stress responsed and changes in protein metabolism in carp Cyprinus carpio during cadmium exposure. Ecotoxicol Environ Saf., 48(3): pp. 255- 262.

[11]. Dubale MS and Punita Shah (1981). Biochemical alterations induced by cadmium in the liver on Channa punctatus. Environmental research. 26 (1): pp. 110-118.

[12]. Dupler D (2001). Heavy metal poisoning. Gale Encyclopedia of Alternative Medicine. Farmington Hills, MI: Gale Group.

[13]. Fabien Pierron, Magalie Baudrimont, Angélique Bossy, Jean-Paul Bourdineaud, Daniel Brèthes, Pierre Elie and Jean-Charles Massabuau (2007). Impairment of lipid storage by cadmium in the European eel (Anguilla anguilla). Aquatic Toxicology. 81(3): pp. 304-311.

[14]. Finney DJ (1971). Probit Analysis, Cambridge University Press. 3rd Edition.

[15]. Forstner U and Prosi F (1979). Heavy metal pollution in freshwater ecosystem. In “Biological aspects of Freshwater Pollution”: In O. Ravera (ed.), Pergamon Press, Oxford) pp. 129-161.

[16]. Jarrup L (2003). Hazards of heavy metal contamination. Br Med Bull, 68: pp. 167-182.

[17]. Kemp A, Adrienne JM, Kits Van Hejningen 1954. A colorimetric method for the determination of glycogen in tissues. The Biochemical Journal (56): pp. 640-648.

[18]. Levesque HM, Moon TW, Campbell PGC and Hontela A (2002). Seasonal variation in carbohydrate and lipid metabolism of

yellow perch (Perca flavescens) chronically exposed to metals in the field. Aquatic Toxicology. 60(3-4): pp. 257-267.

[19]. Lide D (1992). CRC Handbook of Chemistry and Physics. 73rd Edition. Boca Raton FL: CRC Press.

[20]. Lowry OH, Rosenbrough NJ, Farr RL, Randall RJ (1951). Protein measurement with the Folin Phenol reagent. J.Biol.Chem.,(193): pp. 265-275.

[21]. Luo SQ, Plowman MC, Hopfer SM, Sunderman FW Jr 1993. Mg (2+) deprivation enhances and Mg(2+) supplementation diminishes the embryotoxic and teratogenic effects of Ni2+, Co2+, Zn2+ and Cd2+ for frog embryos in the FETAX assay. Ann Clin Lab Sci. 23(2): pp. 121-129.

[22]. Mehmet Yılmaz, Ali Gül and Erhan Karaköse 2004. Investigation of acute toxicity and the effect of cadmium chloride (CdCl2 • H2O) metal salt on behavior of the guppy (Poecilia reticulata). Chemosphere. 56 (4): 375-380.

[23]. Moore S and Stein WH 1954. A modified ninhydrin reagent for the photometric determination of amino acids and related compounds: J.Biol.Chem., (221): 907

[24]. Pande SV, Parvin Khan A, Venkatasubramanium TA 1963. Microdetermination of lipids and serum fatty acids. Analyt. Biochem. 6 (5): pp. 120-125.

[25]. Rashed MN 2001. Cadmium and lead levels in fish (Tilapia nilotica) tissues as biological indicator for lake water pollution. Environ Monit Assess: 68(1): pp. 75-89.

[26]. Roberts JR 1999. Metal toxicity in Children. Training Manual on Pediatric Environmental Health: Putting It into Practice. Emeryville, CA: Children's Environmental Health Network.

[27]. Sastry KV and Subhadra K 1982. Effect of cadmium on some aspects of carbohydrate metabolism in a freshwater catfish, Heteropneustes fossilis. Toxicol. Lett. 14: pp. 45-55.

[28]. Schmidt Nielson B 1975. Osmoregulation: Effect of Salinity and heavy metal. Fed. Proc. 33: pp. 2137-2146.

[29]. Seebaugh DR, Goto D, Wallace WG 2005. Bioenhancement of cadmium transfer along a multi level food chain. Mar Environ Res. 59(5): pp. 473-491.

[30]. Sunderman FW Jr., Plowman MC, Hopfer SM 1991. Embryotoxicity and teratogenicity of cadmium chloride in Xenopus laevis, assayed by the FETAX procedure. Ann. Clin. Lab Sci, 21(6): pp. 381-391.

[31]. Waisberg M, Joseph P, Hale B and Beyersmann D 2003. Target for Toxicity and Death due to Exposure to Cadmium. Chloride. Toxicology. 192(2-3): pp. 95-117.

[32]. Wright DA and Frain JW 1981. The effect of calcium on cadmium toxicity in the freshwater amphipod, Gammarus pulex (L.). Arch. Environ. Contam. Toxicol., 10(3): pp. 321-328.

[33]. Zanders IP and Rojas WE 1996. Salinity effects on cadmium accumulation in various tissues of the tropical fiddler crab Uca rapax. Environmental Pollution, 94(3): pp. 293- 299.

Corresponding Author

Dr. Akhil Abhishek*

Assistant Professor of Zoology, Simtech College, Patna