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Comparative account of impact of heavy metals cadmium and chromium on haematological parameters of *Channa punctatus*

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Abstract

The aim of present investigation was to determine the effect of heavy metal pollutants such as cadmium and chromium on haematological parameters of fresh water fish, *Channa punctatus*. The experimental group of fish was exposed to a sublethal concentrations of cadmium and chromium for a period of 45 days and observations were made on Haemoglobin (Hb), Packed Cell Volume(PCV), Total Erythrocyte Count(TEC), Mean Cell Volume (MCV), Mean Cell Hemoglobin Concentration (MCHC), Total Leucocytes Count (TLC), Serum Glutamate Oxaloacetate Transaminase (SGOT) and Serum Glutamate Pyruvate Transaminase (SGPT). It was found that there was significant decrease in Haemoglobin Content, PCV and TEC in majority of results. This was accompanied by decrease in MCV and MCH and increase in MCHC in both cases of cadmium and chromium exposure. TLC, SGOT increased on exposure to cadmium while a significant decrease was observed in TLC, SGPT and SGOT on exposure to chromium. The study suggested that the presence of toxic heavy metals in aquatic environment has strong influence on the hematological parameters in the fresh water fish.

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Introduction

The heightened concern for reduction of environmental pollutants that has been occurring over the past half century has stimulated active and continuing research on toxicology of heavy metals. Among the various heavy metals, cadmium merits special attention due to its potential hazard to aquatic biota (Mayer *et al.* 1991; Barber and Sharma 1998) as well as to human beings (Groten and Van Bladeron 1994; Vanderpools and Reeves 2001). This heavy metal which is a common aquatic pollutant is known to be highly toxic to most organisms even at low concentration in natural waters (Lovert *et al.* 1972). Cadmium is present as impurity in several products including phosphate fertilizers, detergents, and refined petroleum products and as wastes from many industrial processes like tanning, dyeing, paper pulp, batteries, electroplating *etc*. Cadmium enters water bodies and from there it gets incorporated in the food chain of aquatic biota including fish which eventually ends up on dinner table of human beings. Aquatic organisms take up heavy metals and concentrate them to a higher level than that found in the surroundings. Fish is a high potential diet that is invariably consumed by masses as staple food and suffers metabolically due to heavy metal toxicity. Its productivity and nutritive value is depleted.

Chromium exists primarily in Cr (III) and Cr (VI) oxidation states; the later, hexavalent species, being considered as more toxic in the environment due to its higher solubility and mobility (R. Vinodhini *et al.*, 2009). Chromium is used in metal alloys and pigments for paints, cement, paper, rubber and other materials. Effluents from these industries enter the streams, rivers, lakes *etc*. Chromium often accumulates in aquatic life, adding to the danger of eating fish that may have been exposed to high levels of chromium.

In humans breathing high levels chromium (VI) can cause irritation to the nose, nosebleeds, and ulcers and holes in the nasal septum. Ingesting large amounts of chromium (VI) can cause stomach upsets and ulcers, convulsions, kidney and liver damage, and even death. Skin contact with certain chromium (VI) compounds can cause skin ulcers. Allergic reactions consisting of severe redness and swelling of the skin have been noted. (Irwin R.J., 1997)

The effects of acute cadmium poisoning in humans are very serious. They include severe abdominal pain associated with nausea, vomiting and diarrhoea, headache and vertigo. Chronic symptoms include high blood pressure, kidney damage, destruction of testicular tissue and destruction of red blood cells (Ferard *et al.* 1983).

It is known that physiological and biochemical parameters in fish blood can change when exposed to heavy metals. Several biochemical and physiological parameters in tissue and fish blood could be used as indicators of heavy metal toxicity. Haematological indices are very important parameters for the evaluation of fish physiological status. The present study undertakes the effects of cadmium and chromium exposure on various blood parameters of local fish *Channa punctatus*.

Material and Methods

The Fresh water Murrel, *Channa punctatus* were purchased from fish market and disinfected with 0.1% KMnO4. The fish were then kept in different aquaria for conduction of various experiments. The fish were acclimatized to laboratory conditions in aquaria for a few days. In one aquarium the fish were kept as control specimens given the same food and environment as that of the experimental fish except that they were not given the dose of heavy metal compound. Inorganic salt of the heavy metal Cadmium namely Cadmium nitrate $(CdNO_3)_2\,4H_20$ and an inorganic salt of the heavy metal chromium namely potassium chromate (K_2CrO_4) were selected as the experimental toxicants. To observe the chronic effects of heavy metals, sublethal dose (1/10 concentration of 96 hr

LC_{50}) of the heavy metal compounds were given for 45 days. Fish were fed on pellet diet (Prawn powder-fish powder and minced liver in the ratio of 2:2:1) at the rate of 2% body weight.

Blood from the caudal vessel of control and experimental fish was drawn with help of heparinized needles. Haemoglobin, haematocrit, erythrocyte and leucocyte count were made in whole blood. The activities of glutamate oxaloacetate transaminase and glutamate- pyruvate transaminase were determined in the serum.

Haemoglobin content of the blood was estimated by the Acid haematin method**.** Haematocrit, erythrocyte and leucocytes count were determined by the method given by Dacie and Lewis (1977). The activities of the SGPT and SGOT were estimated by the method of Bergmeyer (1974).

PARAMETERS	CONTROL	CADMIUM	CHROMIUM
Haemoglobin(g/100ml)	11.06 ± 0.04	10.01 ± 0.05***	8.99 ± 0.05***
PCV (%)	31.87 ± 0.06	21.51 ± 0.04***	18.04 ± 0.06***
TEC($\times 10^6$/mm^3)	03.26 ± 0.05	03.06 ± 0.06*	2.98 ± 0.04***
MCV(fl)	97.72 ± 0.03	70.29 ±0.03***	60.52 ± 0.03***
MCH(pg)	33.72 ± 0.02	32.66 ±0.05**	30.21± 0.05***
MCHC(g/dl)	34.51± 0.04	46.49 ± 0.03***	49.89 ± 0.04***
TLC($\times 10^4$/mm^3)	06.91 ± 0.02	8.79 ± 0.04***	5.39 ± 0.03***
SGPT (units/mlserum/hr)	21.6 ± 1.32	20.9 ± 0.84^{NS}	10.6 ± 0.76***
SGOT (units/ml serum/hr)	40.50 ± 0.14	50.16 ± 3.51**	12.08± 0.25***

Values are mean± SD; N=6, NS= Not Significant {Significant *p<0.05 **P< 0.01, ***P<.001}

Table-1: Alteration in haematological parameters in *Channa punctatus* exposed to cadmium and chromium for 45 days

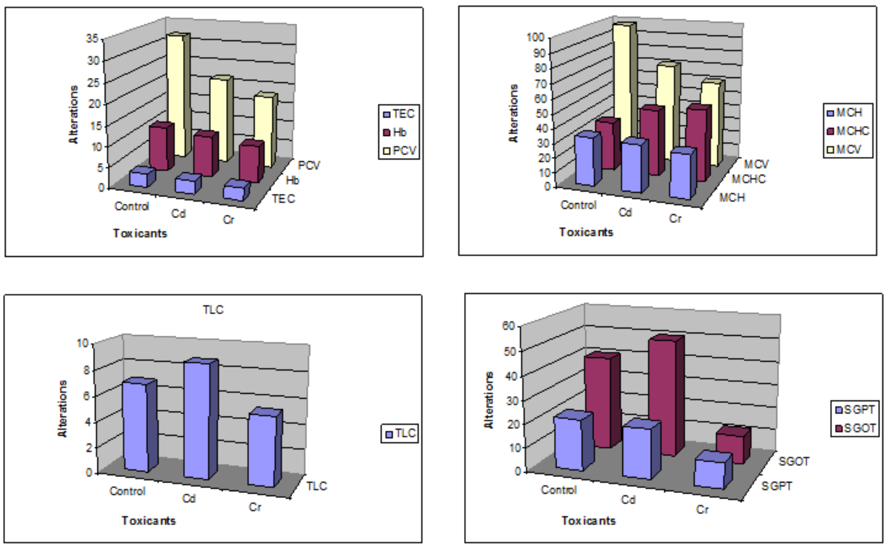

Fig. 1: Haematological alterations produced by the toxicants after 45 day

Observation and Results

Haemoglobin contents decreased on exposure to cadmium for 45 days by 9.49% while decrease was more marked after exposure to chromium by 18.71%. A significant decrease was observed in PCV on exposure to cadmium and chromium by 32.5% and 43.3% respectively. TEC decreased by 6.13% in case of cadmium and 8.58% in case of chromium. A significant decrease was recorded in MCV values on exposure to cadmium (28.06%) and chromium (38.06%). MCH value decreased by 3.14% on exposure to cadmium and 10.40% on exposure to chromium. A significant increase was observed in MCHC values on exposure to cadmium and chromium by 34.71% and 44.56% respectively. TLC increased after exposure to cadmium (27.2%) while decreased after exposure to chromium (21.90%). A significant decrease by 50.92% was recorded in SGPT after exposure to chromium while in cadmium change was observed that was not significant. SGOT value increased by 23.85% on exposure to cadmium while decrease was drastic after exposure to chromium by 70.17%.

Discussion

The present observations on chronic experimentation clearly show significant decrease in Hb content, PCV and TEC in majority of results obtained in both cases of exposure to cadmium and chromium. These results are in agreement with Nasser A. Al-Asgah *et al.* (2015) who found a significant reduction in RBCs, Hb and Hct in comparison with the control in *Oreochromis niloticus* exposed to Cd. Shaheen and Akhtar (2012) also reported significant decline in Hb content and TEC counts of fish *Cyprinus carpio* when exposed to Cr(VI). Reduction in the number of red blood cells may be due to decreased rate of erythropoeisis and/or accelerated destruction of red blood cells (Mc Leay 1973). On the other hand, loss of erythrocytes due to toxicant induced hemorrhages in internal organs cannot be excluded (Johansson-Sjobeck and Larsson 1978). Inadequate haemoglobinization of RBC`s can also be a reason. This is particularly indicated in *Heteropneustes fossilis* exposed to Sewage and potash because haemoglobin deficient red blood corpuscles are observed in the circulation of these individuals (Narain and Srivastava, 1979). M. Javed *et al.* (2012) suggested that heavy metal exposure decreases the TEC count, Hb content due to impaired intestinal absorption of iron.

In the present study decrease in PCV, Hb and TEC is accompanied by decrease in MCV and MCH and increase in MCHC values. Increase in MCHC despite decrease in TEC, Hb content and PCV cannot be attributed to cell shrinkage or swelling but rather to disproportional decrease in the red blood cells and the related values (Wepener *et al.* 1992).

Increase in TLC is observed on exposure to cadmium while decrease is observed after exposure to chromium. An increased response may be the result of direct stimulation of immunological defense due to the presence of foreign substances or may be associated with metal induced tissue damage (Ellis 1981). According to McLeay and Brown (1974) increase in leucocytes under chemical stress in *Onchorhynchus kistush* is, probably for quick removal of cellular debris of necrosed tissue. Metal induced leucocytosis, as an adaptive value to fish has been reported by Goel and sharma (1987). M. Javed *et al.* (2016) also observed leukocytosis in Channa punctatus exposed to thermal power effluents. Other workers have also reported similar observation in *C. punctatus* exposed to Pb (Hymavathi and Rao 2000), *Clarias batrachus* exposed to HgCl2 (Joshi *et al.*

2002). Decrease in TLC on exposure to chromium may be due to fragility of cells and consequent breakdown. Decrease in white blood corpuscles suggests that the fish is losing its capacity to defend microbial or bacterial infection and also autolysis caused by the activity of certain hydrolytic enzymes like phosphatases, lipases, e.t.c. released into plasma under conditions of stress(Wright,1960). The significant decrease in WBC may also be the result of increased secretion of corticosteroid hormones (Ellis 1981). Secretion of these hormones is a non specific response to any environmental stress and is a fundamental mechanism in the increased susceptibility of fish to disease when exposed to a pollutant. Reduction in leucocytes (leucocytopenia) could further be aggravated by necrosis of leucopoietic tissue (Wepener 1992).

Enzymological observations in the present study show elevation of serum glutamate oxaloacetate transaminase (SGOT) activity on exposure to cadmium while a decrease is observed after exposure to chromium. Decrease in the activity of serum glutamate pyruvate transaminase (SGPT) is observed after exposure to chromium. However, on exposure to cadmium, change was observed but that was not significant. Increase in the activity of SGOT exposed to cadmium may be due to chemostress. The blood enzyme pattern is generally believed to indicate the physiological state of the tissue in an organism and is altered after slight injury to cells of an organ in general and liver in particular. In this light when the aforesaid enzyme patterns of exposed and controlled fishes were objectively compared, it becomes evident that heavy metal cadmium cause injury to fish tissues. Structural damage of the liver of *Channa punctatus* exposed to $HgCl_2$ has been reported by Sastry and Gupta (1978). Elevated level of transaminases is also indicative of alteration in the cell membrane properties and therefore permitting rapid leeching of enzymes under hepatotoxic conditions (Slater 1979; Sastry and Sharma 1980). Increase in the activity of SGOT after exposure to cadmium in the present study is supported by findings of Vineeta Shukla and Sastry (1994) in *Channa punctatus* exposed to cadmium. Decrease in the activity of transaminase on exposure to chromium in the present study is evidenced by the findings of Christensen *et al.* (1972) in the blood of brown bullhead (*Iclaluros nebulosus*) on exposure to copper which resulted in decrease in transaminases.

Conclusion

After an analysis of the results obtained in the present study, it can be concluded that anemic state prevails in the fish and the effect of chromium is more pronounced as compared to cadmium.

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