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Hexavalent chromium toxicity on biochemical profile of *Ctenopharyngodon idella*

Sonia Aslam, Ali Muhammad Yousafzai
Islamia College University Peshawar
Corresponding author: Sonia Aslam
Corresponding author email: dua005@yahoo.com

ABSTRACT

This study contemplates the biochemical parameters of grass carp, including serum glutamic-pyruvic transaminase (SGPT), serum glutamic oxaloacetic transaminase (SGOT), lactate dehydrogenase (LDH), creatinine phosphokinases (CPK), glucose, triglycerides, cholesterol and serum total proteins under Cr (VI) toxicity effects for 3 (120 mg/L), 7 (100mg/L), 25 (40mg/L), 40 (20 mg/L) and 60 (10 mg/L) days/concentration. To analyze the biochemical parameters, we followed the biochemical analyzer protocol (Micro lab 300 biochemistry analyzer, China) in the laboratory. Chromium (VI) induced an elevation in the enzymes viz SGPT, SGOT, LDH and CPK throughout the experiments. But SGOT declined after 25 days, LDH declined after 40 and 60 days. CPK declined after 7 days. Glucose decreased after 3 and 60 days. Triglycerides level increased except after 3 days. Cholesterol showed fluctuations. Total proteins decreased after 3 and 25 days as compared to control. Changes in the enzyme activities can result from different types of liver damage, including necrotic hepatocytes. The increasing trend in different parameters is probably due to the activation of the fish's immune system to resist metal stress and liver damage. The decreasing trend reflected retarded and deteriorated fish health.

Key words: enzymes, grass carp, biochemical parameters.

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INTRODUCTION

Heavy metal pollution in the aquatic ecosystem is growing at an alarming rate and is becoming a major problem in many countries around the world. Heavy metal contamination can have detrimental effects on the ecological balance of the environment and on the diversity of aquatic organisms [1]. Some of the heavy metals can cause health problems to fish consumers.

Chromium pollution in the aquatic environment is a serious problem as it has a direct or indirect negative effect on human health and society. Chromium is considered one of the most ubiquitous pollutants in the aquatic environment, but the pure metallic form is naturally absent. There are three oxidation states in the case of chromium, namely Cr (II), Cr (III), Cr (VI). Cr (II) is the most unstable form and (Cr+3) is reported as an essential element in mammals as it takes effective role in glucose, lipid and protein metabolism. Due to poor membrane permeability, non-corrosiveness and very less tendency to biomagnify in the food chain, the toxicity of trivalent chromium is very low [2]. Hexavalent chromium is considered the most toxic form as it easily crosses cell membrane and is therefore reduced to the trivalent form. This trivalent chromium has the ability to combine with several macromolecules and eventually causes toxic and mutagenic changes.

Blood is highly sensitive to environmental changes. Environmental stress factors such as exposure to metals can alter biochemical parameters [3]. Therefore, the measurement of serum biochemical parameters can be useful as a diagnostic tool in toxicology to find the general health and target organs affected by toxic substances.

Exposure to Cr (VI) is considered to be significantly toxic by the scientific community. In recent years, world fish consumption has increased with increasing concern for their nutritional and therapeutic benefits. The content of toxic heavy metals in fish can counteract their beneficial effects; there are several

known adverse effects of heavy metals on human health. This can include serious threats such as renal failure, liver damage, cardiovascular diseases and even death. This study aims to investigate the toxic effects of hexavalent chromium on *Ctenopharyngodon idella* after acute and chronic toxic exposure (03, 07, 25, 40 and 60 days) by evaluating the enzymatic profile including LDH, CPK, SGPT and SGOT and some other biochemical parameters.

MATERIAL AND METHODS

Healthy and active specimens of Grass carp (*Ctenopharyngodon idella*) have been selected as model for this study. Fish of almost equal size of 7-8 inches, weighing 70-80g were taken for the experiment. One group considered the control, consisted of thirty fish and was left aside untreated. Another group, also composed of thirty fish, was selected for the analysis of acute and chronic toxicity of hexavalent chromium in the form of potassium dichromate. All fish were carefully analyzed and fed with commercial feed in both control and experimental aquaria. pH (7.6) and temperature (18.1°C) of the water were kept constant and checked daily.

Blood Collection and storage method

After days of exposure, blood was collected from both the control and experimental group, by piercing the caudal vein with syringe. Blood samples were stored in EDTA tubes and gel tubes for preventing blood coagulation, and then were centrifuged at 3,000 RPM to obtain serum for the analysis of enzymatic profile, including: LDH, CPK, SGPT, and SGOT.

Enzymatic Profile and other biochemical components evaluation

To analyze the enzymatic profile and other biochemical parameters glucose, cholesterol, triglycerides and total proteins, the biochemical analyzer Microlab 300 (manufactured in China) was used. Enzymatic profile and other parameters were analyzed according to the above-mentioned biochemical analyzer proposed protocol.

Statistical Analysis

The results are reported as mean, standard deviation, and standard error of the mean. The SPSS software was used to calculate paired t-test to detect the significant difference (P value) between control and experimental means as shown in table 1.

RESULTS AND DISCUSSION

Changes in biochemical parameters under environmental stress indicate changes in an organism's metabolic and biochemical processes and the measurement of these parameters can be used as a diagnostic tool in fish toxicology to measure fish health and determine the extent of damage to target organs. Comparison between the mean of the control group and the treated group is indicated in table 1. In the control group SGPT mean value was 19, 14.333, 16.333, 21.333 and 20.333 for 3, 7, 25, 40 and 60 days. In chromium treated group SGPT was 22.333, 22, 18, 21.666 and 24 for the 3, 7, 25, 40 and 60 days of exposure periods shown in table 1. The percent increases were 17.54%, 53.49 %, 10.2%, 1.56% and 18.03% compared to control shown in fig. 1.

Parvathi et al., [4] reported that exposure to chromium caused significant increase in serum levels of GOT and GPT. Several researchers have reported that blood enzymes have been greatly increased in fish treated with cadmium, zinc and copper [5]. The increase in blood enzyme activity is due to i) the leakage of these enzymes from liver cells and thus increase their blood levels, ii) increased synthesis or are released into the bloodstream when the hepatic parenchyma cells are damaged [6].

The SGOT mean value in control and chromium treated fish is given in table 1. The SGOT mean value in control group was 21.5, 6.666, 8.333, 11.666 and 10.333 for 3, 7, 25, 40 and 60 days and in chromium treated fish it was 21.666, 11, 7, 12.666 and 14 respectively. The SGOT was increased in 3 days exposure and likewise decreased in 7, 25, 40 days and then again increased in 60 days exposure to chromium. The percent changes were 0.77 %, 65.01%, -15.99%, 8.57% and 35.48% as shown in fig. 2.

Increased level of GOT and GPT in blood serum is an indication of liver damage. Toxicants may have caused cellular necrosis, which eventually led to an increase in the activity of these enzymes in serum. In another research study an elevation in serum GOT due to the extreme cytotoxicity produced by cadmium was investigated [7]. An increase in GOT activity in common carp after exposure to chromium toxicity compared to control was reported by [4]. Low activity may also be due to defective enzymes present in the plasma that are unable to catalyze their reactions [8].

In the present investigation LDH mean value for control group was 593,272.666, 893, 631.666 and 950.666 for 3, 7, 25, 40 and 60 days. While in treated group the mean values were 682.666, 307, 1136.66, 195 and 453 for 3, 7, 25, 40 and 60 days. LDH exhibited an elevation of 15.12%, 12.59%, 27. 28% after 3, 7 and 25 days while declined after 40 days (-69.12%) and 60 days (-52.34%) shown in fig. 3.

This study revealed that the primary cellular component of the liver, which is influenced by the toxicant, appears to be a cellular membrane and such toxicant have increased membrane permeability, resulting in greater extraction of the enzyme. LDH plays role in anaerobic pathway so increased LDH means an increase in anaerobic metabolism due to energy depletion under chromium stress. This study agrees with Yousafzai and Shakoori [9] who stated that the increase in enzyme activity in the liver may be due to the higher level of enzyme synthesis to combat the damage caused by toxic substances. The observed increase in LDH activity may be due to the conversion of accumulated pyruvate into lactate, which is transported through the muscle to hepatopancreas and regenerated glucose and glycogen, to provide energy for the fish.. Low activity may also be due to defective enzymes present in the plasma that are unable to catalyze their routine reactions [8]. Cellular damage is another reason for reduce enzyme synthesis in living organisms. The enhanced enzyme activity also reflects tumors in secretory cells and the presence of stressful environment.

CPK mean value for control was 1956, 394, 4020.666, 62.333, 1868.666 for 3, 7, 25, 40 and 60 days. In chromium treated group the mean value was 6657.667, 76, 4064.33, 344.333 and 4064.333 for 3, 7, 25, 40 and 60 days respectively. CPK was found to be increased 240.37%, 1.08%, 452.4% (significant) and 117.49% after 3, 25, 40 and 60 days treatment while decreased by -80.71% after 7 days exposure as compared to control shown in fig. 4.

The elevated enzyme levels in the blood could be attributable to hepatic damage under pathological conditions [10]. Muscular dystrophy can also cause an increase in CPK activity. Therefore, the reduced activity of these enzymes may be due to reduced enzymatic synthesis or may be due to changes in the permeability of liver cells.

Glucose mean value for control group was 59, 103.666, 75, 70, 82.666 for 3, 7, 25, 40 and 60 days. In chromium exposed group the mean value was 31.666, 122.333, 86.66, 102.333 and 70.666 for 3, 7, 25, 40 and 60 days. Glucose was found to be decreased as -46.23% and -14.51% after 3 and 60 days. However, an abrupt increase of 18%, 15.54% (significant) and 46.19% was noticed after 7, 25 and 40 days exposure as compared to control shown in fig. 5.

The study conducted on *Cyprinus carpio* exposed to sublethal concentration of chromium (III) for 28 days exhibited significant elevation in the blood glucose level [11]. Similar investigation has been reported earlier in the blood of common carp after exposure to a mixture of heavy metals, including chromium for 32 days [4]. Another study reported an increase level of glucose in *C. carpio* by using hexavalent chromium following 8, 16, 24, and 32 days exposure [12]. High blood glucose content as a result of chromium has been attributed to intensive glycogenolysis and the synthesis of glucose from extra hepatic tissue proteins and amino acids as well as the involvement of Cr in glucose metabolism as an insulin co-factor. At the same time, the decreased concentration of glucose reflects the depletion of energy (glycogen) resources and consequently the deterioration of body conditions.

Triglycerides mean value for control group was 132, 415.666, 164.333, 153 and 122.333 for 3, 7, 25, 40 and 60 days as given in table 1. In chromium exposed fish triglycerides mean value was 70.666, 488.666, 211, 197.666, 255.666 for 3, 7, 25, 40 and 60 days. In comparison to control triglycerides declined by -46.46% after 3 days while increasing trend of 17.56%, 28.39%, 29.19% and 108.99% (significant) was observed after 7, 25, 40 and 60 days.

The reduction of the triglyceride level showed that during the exposure lipolysis is the main source of energy [13]. Increased triglyceride levels may be due to the metabolic syndrome, or high energy requirements, caused by intoxication with hexavalent chromium in this study.

Cholesterol mean value for control group was 186.5, 122.666, 163.333, 203.333 and 185 for 3, 7, 25, 40 and 60 days. In chromium treated group cholesterol values were 169, 330.666, 136.33, 235.333 and 161 for 3, 7, 25, 40 and 60 days respectively. Declining trend in cholesterol was noticed as -9.38%, -16.53%, -12.97% after 3, 25 and 60 days exposure. However, cholesterol was increased as 169.56% (significant) and 15.73% after 7 and 40 days.

Cholesterol is an essential structural component of the membranes and a precursor of all steroid hormones can increase due to hepatic failure causing the release of cholesterol in the blood stream. It is well known that heavy metals have hazardous effects on cellular structure, especially on the membranes. For that reason, increase in cholesterol can be the indication of environmental stress [4]. In another study a significant reduction in cholesterol was noticed in fish exposed to different concentrations of Cr (VI) from 25-150 mg/L [14]. This reduction in cholesterol may reflect liver damage, caloric restriction or reduction of serum proteins such as albumin [15].

Total proteins mean value for control were 4.95, 7.033, 3.733, 4.833 and 4.2 for 3, 7, 25, 40 and 60 days. In chromium exposed group mean values were 3.9, 12.566, 3.66, 5.2 and 5.033 for 3, 7, 25, 40 and 60 days

respectively. Total proteins were found to be declined as -21.21% and -1.95% after 3 and 25 days and increase of 78.67%, 7.59% and 19.83% was recorded after 7, 40 and 60 days.

Protein is a main component of the body and its metabolism is almost limited to the liver. The decline in serum protein levels may be due to reduced renal function or reduced protein synthesis due to liver cirrhosis. The decrease in protein content could be due to the increased proteolytic activity and the reduced anabolic activity of the protein as observed by Jenkins et al., [16]. The increase in total protein contents indicates the acceleration of protein synthesis under the influence of toxic substances in response to the stress situation. Similarly another researcher reported an increase in tissue proteins and have attributed with increasing levels, possibly due to the synthesis of proteins to sequester the metals [17].

Table 1. Changes in the various biochemical parameters of *Ctenopharyngodon idella* exposed to sublethal concentration of hexavalent chromium.

S.No	Biochemical profile	Days of exposure	Control group mean±SE	Treated group mean±SE	P-Value
1	SGPT	3	19±2.309	22.333±3.179	0.444
		7	14.333±1.452	22±5.131	0.224
		25	16.333±0.881	18±2.645	0.582
		40	21.333±2.333	21.666±2.185	0.922
		60	20.333±2.333	24±2.886	0.379
2.	SGOT	3	21.5±9.526	21.666±16.230	0.993
		7	6.666±1.452	11±1.527	0.109
		25	8.333±2.027	7±1.527	0.627
		40	11.666±2.027	12.666±1.201	0.693
		60	10.333±1.201	14±0.000	0.093
3.	LDH	3	593±117.202	682.666±85.030	0.569
		7	272.666±99.284	307±229.776	0.410
		25	893±253.184	1136.66±167.799	0.467
		40	631.666±177.771	195±81.835	0.089
		60	950.666±90.598	453±0.0000	0.005**
4.	CPK	3	1956±1008.631	6657.667±454.696	0.013*
		7	394±372.518	76±23.259	0.483
		25	4020.666±761.247	4064.33±1090.958	0.975
		40	62.333±11.095	344.333±42.834	0.017*
		60	1868.666±1800.175	4064.333±100.747	0.347
5.	Sugar	3	59±12.124	31.666±4.409	0.101
		7	103.666±50.174	122.333±7.535	0.732
		25	75±2.886	86.66±2.027	0.030*
		40	70±8.144	102.333±8.647	0.053
		60	82.666±4.333	70.666±6.064	0.183
6.	Triglycerides	3	132±27.135	70.666±2.603	0.088
		7	415.666±165.07	488.666±103.923	0.727
		25	164.333±77.341	211±29.143	0.602
		40	153±17.214	197.666±12.251	0.102
		60	122.333±28.013	255.666±19.341	0.017*
7.	Cholesterol	3	186.5±9.526	169±14.468	0.370
		7	122.666±32.043	330.666±58.473	0.036*
		25	163.333±31.103	136.33±15.059	0.478
		40	203.333±24.319	235.333±16.333	0.336
		60	185±10.969	161±14.433	0.256
8.	Total proteins	3	4.95±0.952	3.9±0.1	0.335
		7	7.033±1.443	12.566±1.865	0.079
		25	3.733±0.338	3.66±0.317	0.893
		40	4.833±0.548	5.2±0.360	0.606
		60	4.2±0.513	5.033±0.088	0.185

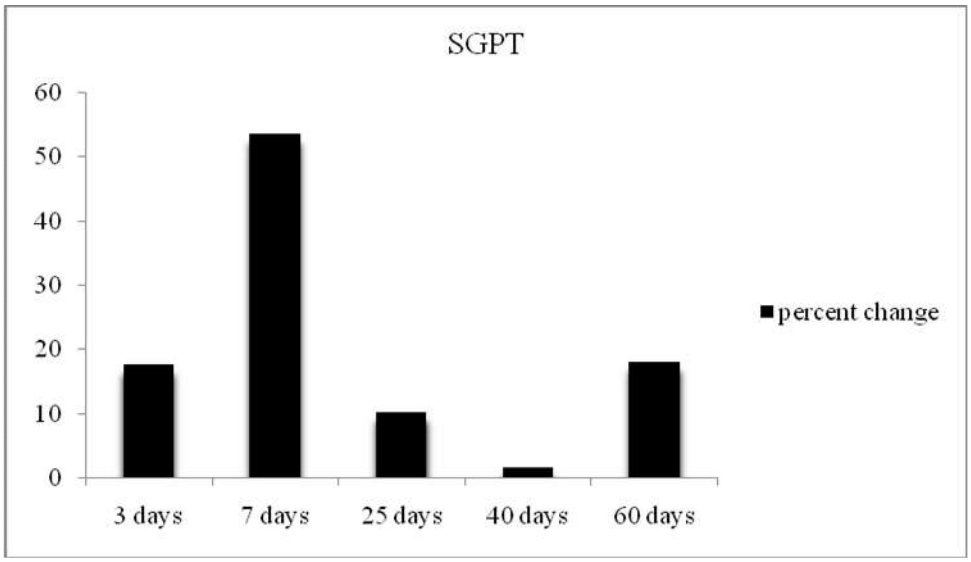

Fig. 1. Effect of hexavalent chromium exposure on SGPT in grass carp.

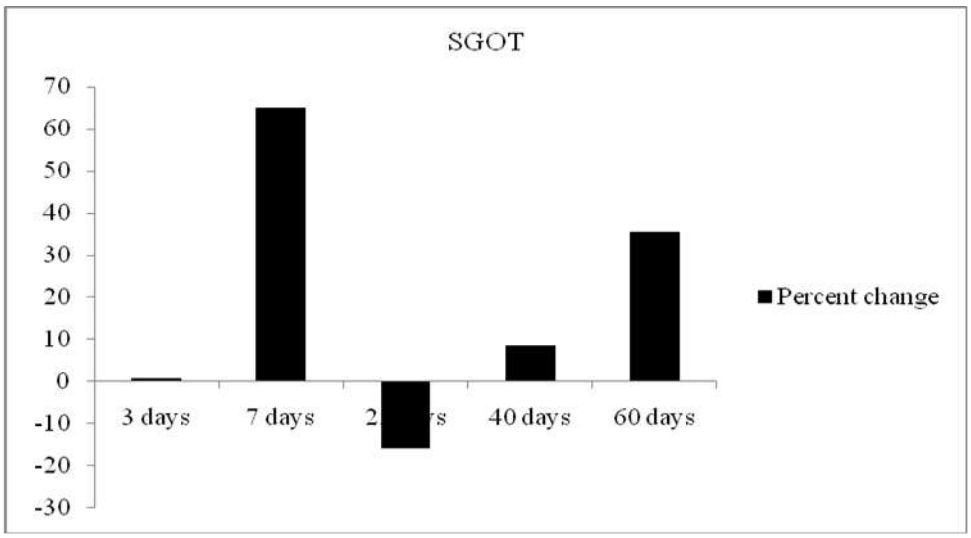

Fig. 2. Effect of hexavalent chromium exposure on SGOT in grass carp.

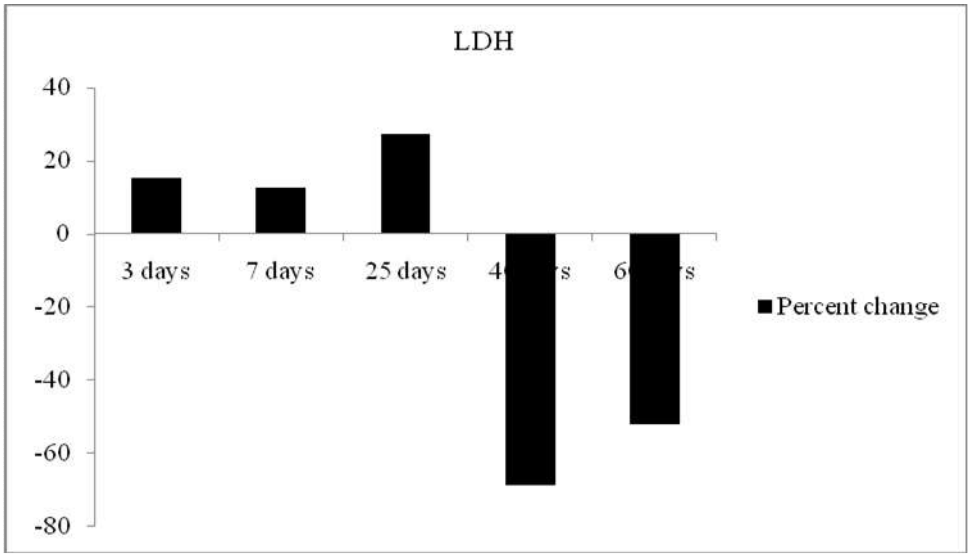

Fig. 3. Effect of hexavalent chromium exposure on LDH in grass carp.

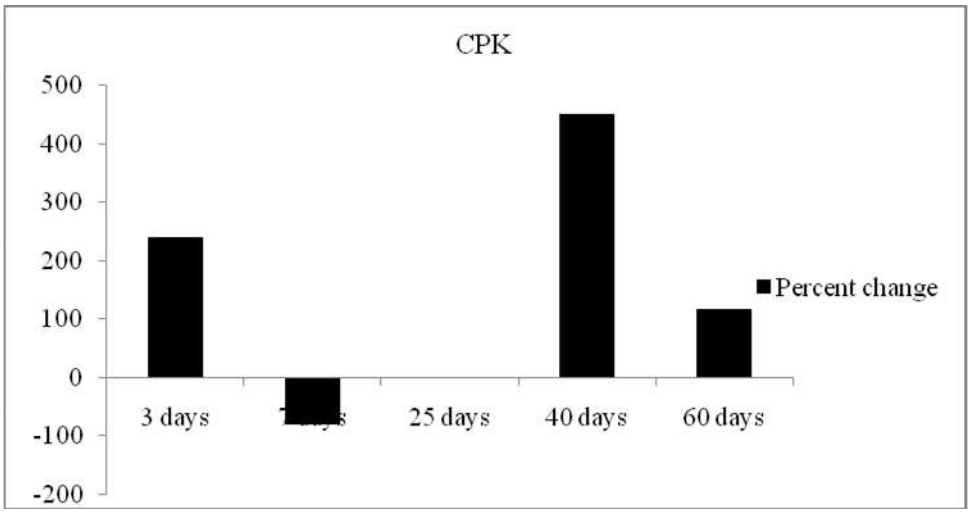

Fig. 4. Effect of hexavalent chromium exposure on CPK in grass carp.

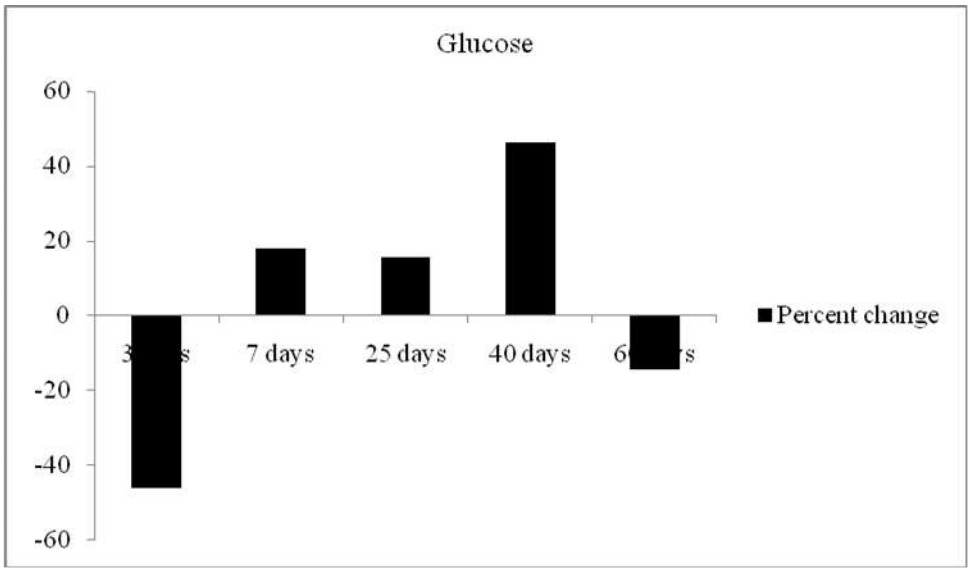

Fig. 5. Effect of hexavalent chromium exposure on glucose in grass carp.

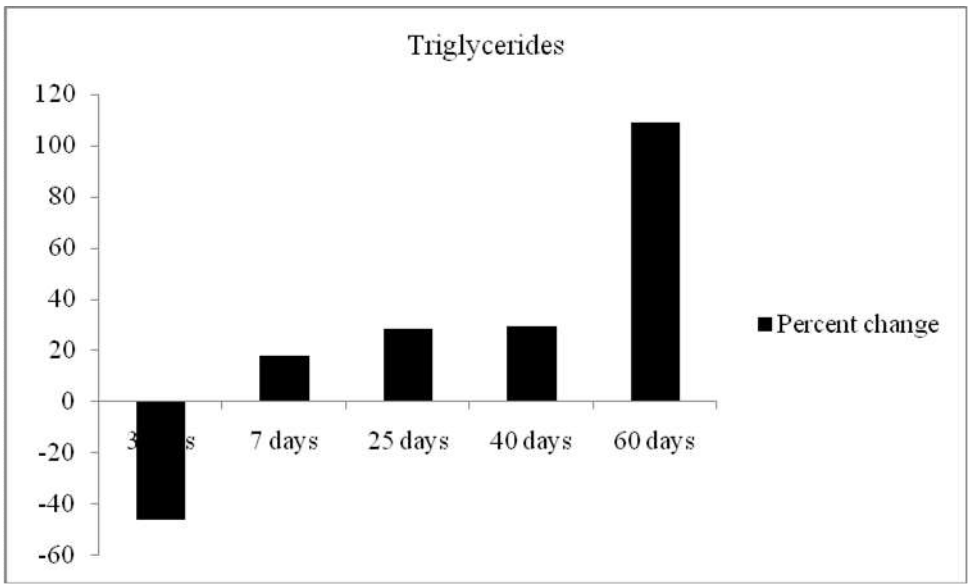

Fig. 6. Effect of hexavalent chromium exposure on triglycerides in grass carp.

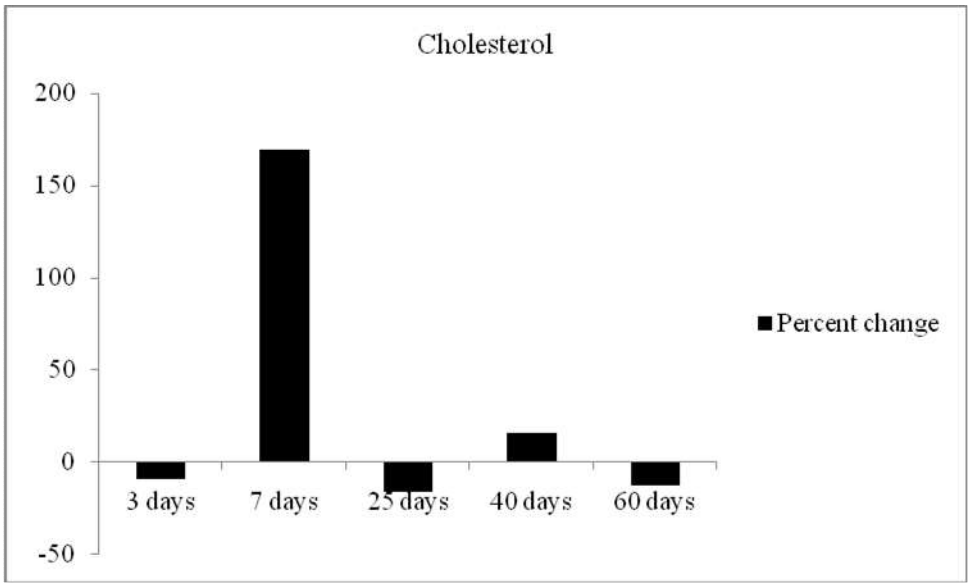

Fig. 7. Effect of hexavalent chromium exposure on cholesterol in grass carp.

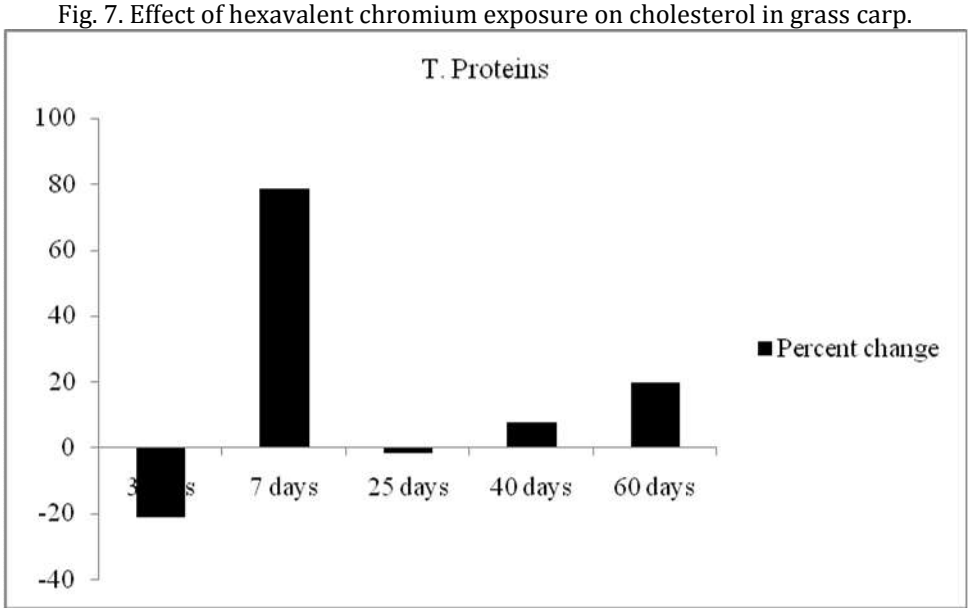

Fig. 8. Effect of hexavalent chromium exposure on total proteins in grass carp.

CONCLUSION

The present study indicated that the dose-ranging investigations of chromium caused hepatotoxic (liver damaging) effects on respective experimental groups. Chromium might have damaged the cell membrane or altered its permeability triggering the selective leakage of the enzymes into the blood. The enhanced enzyme activity also reflects tumors in the secretory cells and the existence of stressful environment. The minimal level of enzyme in the blood shows regenerative capacity of liver as a result of which leaching out of enzymes in the blood serum become least. Low activity may also be due to defective enzymes present in plasma that were incapable to catalyze their routine reactions. Under Cr stress the fish is using energy reserves, causing deterioration of body conditions. Hexavalent chromium is highly toxic to grass carp. Exposure to different chromium concentrations has led to significant biochemical changes that can potentially be harmful to the survival of grass carp.

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