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Full Length Research Paper

Toxicity bioassay of lead acetate and effects of its sub-lethal exposure on growth, haematological parameters and reproduction in *Clarias gariepinus*

Hmoud Fares Alkahemal-Balawi, Zubair Ahmad*, Ali Suliman Al-Akel, Fahad Al-Misned, El-Amin Mohamad Suliman and Khalid Abdullah Al-Ghanim

Department of Zoology, College of Science, King Saud University, PO Box 2455, Riyadh 11451, Saudi Arabia.

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The African catfish, *Clarias gariepinus*, was exposed to different concentrations of lead acetate for 96 h, and the value of LC_{50} for the exposure period was registered as 122 mg/L. The effects of sub-lethal exposure on growth, reproduction and haematological parameters were also investigated in *C. gariepinus*. Lead exposure at all concentrations reduced growth rate, and was inversely related to growth. There were significant effects of lead exposure on erythrocyte count, haemoglobin concentration and haematocrit values. Lead exposure time and dose increased the plasma glutamic-oxaloacetic transaminase (PGOT) and plasma glutamic-pyruvic transaminase (PGPT) activity levels. No significant effect of lead acetate was noticed on fecundity and ovum size. Sperm motility was reduced in all concentrations of lead acetate after 4 weeks of exposure. The present investigation indicates that the lead acetate is less toxic to fish than other metals as the LC_{50} value is comparatively high.

Key words: Lead acetate, acute toxicity, growth, haematological parameters, reproduction.

INTRODUCTION

Lead as an industrial pollutant and immunotoxicant, has the potential to adversely affect human and animal health. It induces a broad range of physiological, biochemical and neurological dysfunctions in human (Nordberg et al., 2007). Exposure to low level of lead during early development was found to produce long-lasting cognitive and neurobehavioural deficit, persistent immune changes, reduced fertility, a delay in sexual maturity, irregular estrus and reduced number of corpora lutea in human and experimental animals (Mobarak, 2008).Recent and notable reports have indicated that lead can cause neurological, gastrointestinal, reproductive,circulatory, immunological, histopathological and histochemical changes in the animals (Park et al., 2006; Berrahal et al., 2007; Reglero et al., 2009; Abdallah et al., 2010; Mobarak and Sharaf 2011).Blood parameters are often measured,when clinical diagnosis of fish physiology is applied to determine the sub-chronic effects of pollutants (Wedemeyer and Yasutake, 1977). The use of haematological variables, such as ion and glucose concentrations, haemoglobin and haematocrit indicate a physiological response to a contaminated environment (Dethloff et al., 2001). The count of red blood cells is quite a stable index and the animal body tries to maintain this count within the limits of certain physiological standards using various physiological mechanisms of compensation (Al-Akel et al., 2010). Studies have shown that when the water quality is affected by toxicants, any physiological changes will be reflected in the values of one or more of the haematological parameters (VanVuren, 1986). Blood cell responses are important indicators of changes in the internal and/or external environment of animals. However, in the fish these parameters are more related to the response of the whole organism, that is, to the effect on fish survival, reproduction and growth. Fish live in very intimate contact with their environment, and are therefore very susceptible to physical and chemical changes which may be reflected in their blood components (Wilson and Taylor, 1993). Blood tissue truly reflects physical and chemical changes occurring in organism and an early diagnosis is also

*Corresponding author. E-mail: zahmed@ksu.edu.sa or zubair.ahmad.ah@gmail.com.

Table 1. Water chemistry of tanks during the experimentation period.

Parameter	Average	± SD
Temperature (ºC)	26.53	0.8
Dissolved oxygen (mg/L)	4.9	0.5
PH	7.3	0.5
Hardness (mg/L)	210	3.20
TDS (ppm)	550	3.27

possible when evaluating haematological data, particularly blood parameters (Luskova, 1997). However, it should be noted that haematological indices are of different sensitivity to various environmental factors and chemicals (Vosylienë, 1999). Previous haematological study of nutritional effects, infectious diseases and pollutants brought the knowledge that erythrocytes are the major and reliable indicators of various sources of stress (O'neal and Weirich, 2001; Ehulka, 2002). This study, therefore, assessed the haematological profile of *Clarias gariepinus* exposed to lead acetate. Moreover, change in plasma glutamic-oxaloacetic transaminase (PGOT) and plasma glutamic-pyruvic transaminase (PGPT) activities also indicate the influence of water pollution on fish (Bucher and Hofer, 1990).

C. gariepinus is an economically important freshwater fish in Saudi Arabia and very little information is available on the effects of lead, particularly through sub-lethal exposure on its metabolic activities. The aim of this study, therefore, was to investigate lead accumulation in *C. gariepinus* through the sub-lethal exposure; effects on haematological parameters in *C. gariepinus* after sub-chronic lead exposure; histopathological alteration in liver, kidney, gills and muscle tissues and to measure the changes in PGOT and PGPT activities. Fish growth and reproduction (egg production and sperm's motility) were also investigated.

MATERIALS AND METHODS

The healthy and active specimens of *C. gariepinus* were obtained from a fish farm run by King Abdul Aziz City for Science and Technology (KACST), Riyadh, Kingdom of Saudi Arabia. The fish were stocked (250 fish per tank) in big tanks (200 × 60 × 90 cm) and acclimated to lab conditions for 1 month. During the period of acclimation, the fish were fed a lead-free diet at a rate of 3% body weight daily (as two meals, 1.5% bw/day) and the water was aerated with mechanical pump. After 1 month of acclimation, fish were randomly transferred to small fish aquaria (75 × 30 × 45 cm). Each aquarium was stocked with 10 fish. There were 8 tanks in three replicates (total 24 tanks) for this experiment. Morphometric characteristics were recorded prior to stocking. Seven groups of fish were exposed to different concentrations (110, 115, 120, 125, 130, 135 and 140 mg/l) of lead acetate. A control set was also run with same number of fish and same amount of water but without lead acetate. Mortality of fish in each concentration was monitored and immediately registered. The method of Finney (1971) was used to calculate LC_{50}.

In another set of experiment, the fish was exposed to three different concentrations (5, 10 and 20% of the LC_{50} which is equivalent to 6.1, 12.2 and 24.4 mg/l, respectively) of lead acetate. A control set was also run for comparison. Each of the four groups of *C. gariepinus* was fed a commercial fish food 3% of the body weight for 6 weeks. The experimental fish were maintained and tested under a 12 h light/dark cycle (06 am to 06 pm/06 pm to 06 am). Water quality was measured after every 3 days during the experimental periods (Table 1).

Biological sampling and analysis

Total weight and total length were recorded for each individual after every 2 weeks. Two fish (from each of the three replicates) were removed from each tank at every sampling time during the whole experimental period. Blood samples of both fish of each group were obtained from the caudal vein of the fish in a heparinized vial. Haematocrit values were determined by a micro-haematocrit centrifuge (5 min, 10,000 g) in glass capillaries, using the micro-haematocrit reader (Hawksley and Sons, England). Haemoglobin concentration of the blood was measured by the cyano-methaemoglobin method using a diagnostics haemoglobin kit (Kit no. 527-A, Merck). The red blood cell (RBC) count was determined in a 1:200 dilution of the blood sample in the Hendrick's solution with hemocytometer (Marienfeld, Germany).

The remaining blood was centrifuged (5000 g for 10 min), and the plasma was frozen until required for analysis. Frozen plasma samples were analyzed for glucose, total protein, calcium and magnesium using their respective kits (Biomerieux, France). Enzyme activities in the plasma were measured with a spectrophotometer. The assays were run in triplicate. PGOT and PGPT were measured using a diagnostic kit (Biomerieux, France). Results were expressed as IU/L. Growth parameters were calculated using the following formula:

Growth rate (GR) = [(Final weight - Initial weight) / Time (days)];
Specific growth rate (SGR) = 100 × [{ln(final weight) - ln(initial weight)} / time (days)];
Hepato-somatic index (HSI) = (liver weight / body weight) × 100;
Condition factor (CF) = (Total weight of fish / (length of fish)^3) × 100.

Reproduction

Three mature females fish randomly selected from each exposed groups were weighed, killed and dissected to remove ovaries. Three sub-samples of known weight (50 mg) were taken from each ovary and counted for total eggs. From the mean of these sub-samples the total numbers of eggs were calculated as:

Absolute fecundity (AF) = [(mean number of eggs × weight of ovary) / weight of sub-sample];

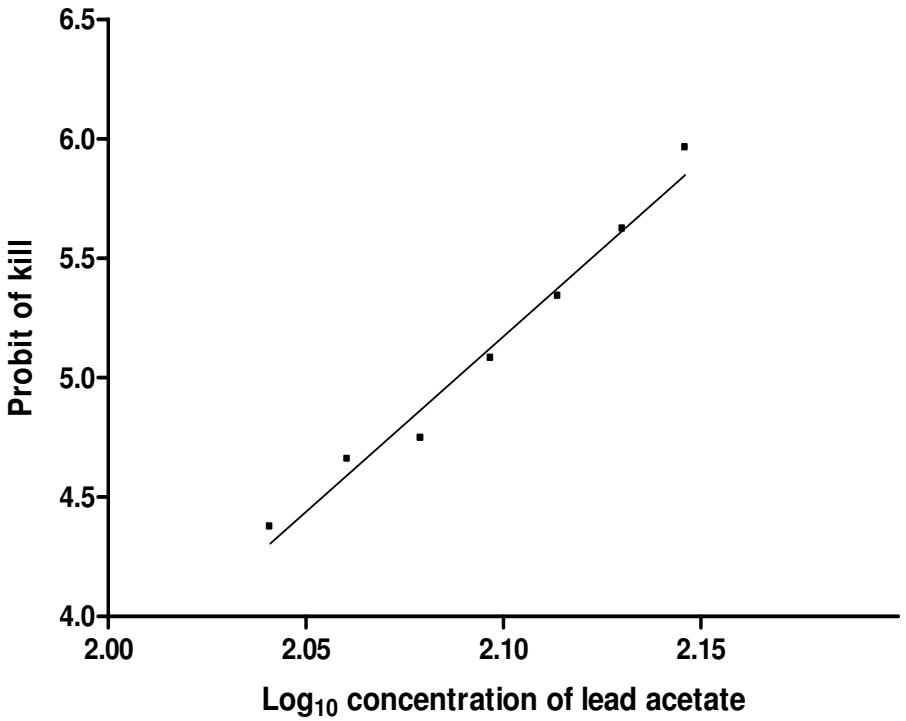

Figure 1. Relationship between probit of kill and $\log_{10}$ concentration of lead acetate to deduceLc_{50}.

Relative fecundity (RF) = [absolute fecundity / body weight (g)];

The mean diameter of long and short axis of ovum was taken as the diameter of oocytes.

Three male fish were selected for the collection of testes. The milt was squeezed out into a Petri dish. A drop of distilled water was quickly added to a drop of milt on a clean slide and the sperm motility was observed under the microscope (×10). The motility was expressed as percentage. The density of spermatozoa was rated on a scale of 1, 2 and 3 (representing low, medium and high density), respectively.

Statistical analysis was done using the analysis of variance (ANOVA) followed by Duncan's multiple range test of mean values were expressed as mean ± SD. The p-values of less than 0.05 were considered statistically significant.

RESULTS

Toxicity

The 96 h LC_{50} value computed as shown in Figure 1 was found to be 122 mg/L.

Growth parameters

Fish mortality was not registered in all groups during the experimental period. The SGR of exposed fish was significantly different from control, and there was a significant inverse relationship between weight gain and exposure concentration at 6.1, 12.2 and 24.4 mg/L lead acetate exposure group, respectively (Table2). The SGR of *C. gariepinus* exposed to lead acetate was 22.6, 30.0 and 35.1%, lower than the control at 6.1, 12.2 and 24.4 mg/L exposures, respectively. SGR in the high-exposure group (24.4 mg/L was significantly lower than control group. Lead exposure produced a gradual decline in the growth rate of the exposed group relative to controls, although, all fish continued to gain weight during the experiment. Food conversion efficiency was affected by lead exposure being lower than the control at all time of exposure, but the effect was more pronounced in the last period (6 week) of treatment (Table 3). No significant differences were observed in HSI and CF in exposed groups (Table 4).

Haematological parameters

Changes in haematological parameters and serum chemistry in the control and exposed group are summarized in Table 5. Although haematocrit, haemoglobin and RBC decreased gradually during the experimental period in all exposed groups, significant differences were observed between control and highest exposed group (Table 5). Concentration of total protein was reduced markedly. The group exposed to the highest lead concentration (24.4 mg/L) displayed a significant decrease after 6 weeks as compared to control.

Table 2. Growth rate, GR and specific growth rate, SGR (in parentheses) of catfish, *C. gariepinus*, exposed to lead acetate.

Concentrations (mg/L)	Times (week)		
	0-2	2-4	4-6
Control	0.46 (1.59)	0.67 (1.93)	0.67 (1.68)
6.1	0.44 (1.46)	0.46 (1.52)	0.49 (1.30)
12.2	0.40 (1.39)	0.47 (1.60)	0.42 (1.16)
24.4	0.37 (1.45)	0.42 (1.50)	0.38 (1.09)

Table 3. Food conversion efficiency (%) of catfish, *C. gariepinus*, exposed to lead acetate.

Concentrations (mg/L)	Times (week)		
	0-2	2-4	4-6
Control	79.52	73.83	73.04
6.1	66.69	56.35	47.54
12.2	52.15	63.94	41.70
24.4	53.52	55.62	42.02

Table 4. The hepato-somatic index (HSI) and condition factor (CF) of *C. gariepinus* exposed to lead acetate.

Time (week)	Concentrations (mg/L)	Hepato -somatic index (HSI)	Condition factor (CF)
2nd	Control	0.68	1.28
	6.1	0.54	1.26
	12.2	0.58	1.26
	24.4	0.55	1.24
4th	Control	0.55	1.33
	6.1	0.52	1.24
	12.2	0.52	1.21
	24.4	0.50	1.25
6th	Control	0.58	1.33
	6.1	0.46	1.24
	12.2	0.47	1.21
	24.4	0.46	1.23

Significant differences were observed in plasma glucose concentrations in the exposed fish in the last period of treatment as compared to the control. The plasma Calcium concentration was unaffected in the exposed groups as compared to the control. There were no significant differences in plasma magnesium (Mg) concentrations observed in the beginning of exposure but a marked decrease was noted in the last phase of the experiment. Changes of PGOT and PGPT activities as a function of exposure time and exposure concentration are shown in Table 5. After 4 weeks of exposure, PGOT concentrations increased significantly in the group treated with 12.2 and 24.4 mg/L exposure and after 6 weeks in all exposed groups. PGPT was significantly elevated after 6 weeks in all exposed groups.

Reproduction

Fish exposed to lead acetate showed changes in reproductive behavior, but these effects were not much prominent. Small variations in the absolute and relative fecundity were registered. Fish exposed to lead acetate produce less number and small sized eggs as compared to unexposed fish. Sperms motility was also affected and reduced markedly (up to 40%) in the fish exposed to different concentration of lead (Table 6) than the control fish. The density of spermatozoa was also decreased in

Table 5. Effects of lead acetate exposure on haematological parameters and biochemical composition of *C. gariepinus.*

Parameter	Concentrations	Exposure time (weeks)		
		2nd	4th	6th
Erythrocytes (Cellx10^6/mm^3)	Control (0.0)	1.32 ± 0.013[a]	1.41 ± 0.075[a]	1.41 ± 0.082[a]
	6.1	1.20 ± 0.035[a]	1.22 ± 0.040[a]	1.41 ± 0.110[a]
	12.2	1.28 ± 0.015[a]	1.16 ± 0.072[a]	1.12 ± 0.072[b]
	24.4	1.10 ± 0.009[b]	1.17 ± 0.110[a]	1.10 ± 0.110[b]
Haematocrit (%)	Control (0.0)	33.06 ± 0.75[a]	34.76 ± 0.54[a]	33.44 ± 1.02[a]
	6.1	32.54 ± 0.61[a]	32.21 ± 0.89[a]	31.23 ± 0.56[a]
	12.2	31.85 ± 0.96[a]	31.35 ± 1.12[b]	30.21 ± 1.05[b]
	24.4	30.45 ± 0.98[b]	30.85 ± 0.99[b]	28.42 ± 1.20[b]
Haemoglobin (g/dl)	Control (0.0)	4.25 ± 0.044[a]	4.35 ± 0.096[a]	4.37 ± 0.032[a]
	6.1	3.34 ± 0.042[a]	2.93 ± 0.087[a]	2.72 ± 0.125[a]
	12.2	3.77 ± 0.131[a]	2.93 ± 0.242[a]	2.57 ± 0.171[b]
	24.4	2.84 ± 0.305[b]	2.96 ± 0.053[b]	2.85 ± 0.159[b]
Total Protein (g/dl)	Control (0.0)	25.62 ± 0.90[a]	27.10 ± 0.55[a]	24.73 ± 0.50[a]
	6.1	26.97 ± 0.88[a]	22.80 ± 0.25[a]	19.00 ± 1.24[a]
	12.2	24.63 ± 1.06[a]	20.59 ± 1.46[a]	15.85 ± 0.51[b]
	24.4	22.94 ± 2.97[a]	22.04 ± 1.22[a]	15.49 ± 0.60[b]
Ca (mg/dl)	Control (0.0)	188.21 ± 4.79[a]	183.74 ± 2.47[a]	184.15 ± 2.11[a]
	6.1	188.45 ± 3.33[a]	180.08 ± 4.13[a]	168.29 ± 5.08[a]
	12.2	182.11 ± 4.30[a]	176.83 ± 7.94[a]	157.32 ± 3.92[b]
	24.4	180.77 ± 5.67[b]	170.73 ± 3.73[a]	153.25 ± 3.18[b]
Mg (mg/dl)	Control (0.0)	35.18 ± 1.59[a]	38.27 ± 1.53[a]	36.32 ± 0.57[a]
	6.1	37.14 ± 1.13[a]	36.49 ± 0.43[a]	35.52 ± 1.33[a]
	12.2	41.11 ± 1.27[a]	38.91 ± 1.81[b]	35.77 ± 2.06[a]
	24.4	33.76 ± 1.32[a]	36.50 ± 1.16[b]	35.87 ± 1.39[b]
SGOT (IU/L)	Control (0.0)	79.56 ± 2.55[a]	86.35 ± 6.84[a]	80.78 ± 3.83[a]
	6.1	78.51 ± 3.68[a]	97.43 ± 13.37[a]	101.73 ± 7.09[a]
	12.2	87.48 ± 2.28[a]	134.73 ± 10.18[b]	116.75 ± 3.54[b]
	24.4	103.89 ± 5.60[a]	127.11 ± 10.29[b]	123.68 ± 5.12[b]
SGPT (IU/L)	Control (0.0)	58.78 ± 3.08[a]	63.03 ± 2.87[a]	61.63 ± 3.01[a]
	6.1	63.96 ± 4.43[a]	67.51 ± 5.91	87.88 ± 5.91[b]
	12.2	71.41 ± 3.29[b]	70.01 ± 1.87[b]	75.37 ± 5.53[b]
	24.4	77.00 ± 2.65[b]	78.40 ± 2.88[b]	90.79 ± 6.05[b]
Glucose (mg/100 ml)	Control (0.0)	344.99 ± 9.42[a]	363.89 ± 17.20[a]	401.52 ± 11. 89[a]
	6.1	489.27 ± 17.30[a]	498.19 ± 9.34[a]	419.16 ± 5.78[a]
	12.2	507.36 ± 9.82[b]	472.65 ± 18.68[b]	420.28 ± 13.62[b]
	24.4	550.85 ± 12.21[b]	490.67 ± 4.11[b]	418.89 ± 8.85[b]

Values in columns with different letters show significant (P<0.05) difference with control.

the fish exposed to lead acetate and pronounced results were registered in higher concentration and last period of investigation.

DISCUSSION

No fish mortality was recorded in all treatments throughout the experimental period. Temperature ranged between 25.4°C and 27.9°C, with a mean of 26.6±0.8°C; dissolved oxygen ranged between 5.0 and 6.5 mg/L with a mean of 5.9±0.5 mg/L, while pH ranged between 7.1 and 8.7 with a mean of 7.6±5.0. These physicochemical parameters were within the limits acceptable for fish culture.

According to Walker et al. (1996), the values obtained by toxicity testing (e.g. LC_{50}) are dependent on the conditions under which tests were performed, so, the interpretation of LC_{50} values needs to be done with caution. Amongst fish species, considerable differences in sensitivity to lead have been reported (Salmerón-Flores et al., 1990). Demayo et al. (1981) had described

Table 6. Variations in reproductive parameters of *C. gariepinus* exposed to different levels of lead acetate.

Time (weeks)	Concentrations (mg/L)	Parameter				
		GSI	Absolute fecundity	Relative fecundity	Motility of sperm (%)	Ovum diameter (mm)
2nd	Control	8.52	9800	239.0	100	0.94
	6.1	7.73	7250	193.3	100	0.83
	12.2	5.54	5280	121.9	100	0.78
	24.4	5.54	5760	218.2	100	0.80
4th	Control	12.12	8664	368.7	100	0.88
	6.1	9.49	8060	294.2	75	0.82
	12.2	7.97	6644	240.7	60	0.79
	24.4	6.63	9728	201.4	65	0.80
6th	Control	11.94	23010	416.0	100	0.89
	6.1	9.72	16270	334.7	60	0.84
	12.2	10.39	8971	300.0	65	0.81
	24.4	11.96	10190	300.0	65	0.82

that lead toxicity is a function of water hardness, species tested and fish age. High water hardness reduces lead toxicity to fish due to a significant inorganic complexation process that controls lead availability to fish (Hodson et al., 1984). Pickering and Henderson (1966) showed that in soft water (20 mg $CaCO_3$/L) the 96 h-LC_{50} for *Pimephales promelas* and *Lepomis macrochirus* was 5.6 and 23.8 mg Pb/L, whereas in hard water (360 mg $CaCO_3$/L) 96 h-LC_{50} was 482 and 442 mg Pb/L, respectively. The short-term lethality test conducted by Martinez et al. (2004) yielded 96 h-LC_{50} as 95 mg Pb/L for juveniles of *Prochilodus lineatus* in water of 82 mg/L hardness ($CaCO_3$), but a higher value (122 mg/L) of LC_{50} for *C. gariepinus* was registered in the present study. Besides the differences related to water hardness and fish age, the differences in toxic potential of lead to different species can be related to its accumulation, biotransformation and excretion. Toxicity also varied with respect to species, size of fish and duration of exposure (Dutta et al., 1995).

The highest values of growth rate, specific growth rate and feed conversion efficiency were however obtained for the control fish which was not exposed to lead acetate. Lead acetate exposure resulted in the reduction of fish growth rate and there was an inverse relationship between growth and lead concentration. Berntssen et al. (1999) reported a significantly reduced growth in juvenile Atlantic salmon when exposed to 500 mg/kg dietary of copper (Cu) for 12 weeks. Gatlin and Wilson (1986) reported that catfish fed a diet with 16 and 32 mg/kg had significantly suppressed the growth and feed efficiency values. Moreover, Lundebye et al. (1999) suggested that decreased growth in Atlantic salmon was due to increased metabolic costs of intestinal cellular changes and Cu excretion. The reduced growth produced by Cu was attributed to metabolic costs associated with metal detoxification (Marr et al., 1996). The aforementioned findings lend a considerable support to the present findings. The decreased growth rate of *C. gariepinus*, registered in the present investigation is probably due to an increased expenditure of energy for sustaining normal metabolism, leaving less energy available for growth. Condition factor (CF) has been used to compare growth conditions of fish. A high condition factor reflects good environmental quality; while a low condition factor reflects poor environmental quality. Hepatosomatic index (HSI) provides an indication on status of energy reserve in an animal. In a poor environment, fish usually have a smaller liver (with less energy reserved in the liver). HSI has been reported to decrease in fish exposed to high concentrations of cadmium and zinc. Reproduction is the most critical stage in the life cycle of a species, which determines its survival. Gonado-Somatic index (GSI) is generally indicative of reproductive success and general water quality. CF, HSI and GSI are not only responsive to pollution; they can be affected by other factors such as temperature and food availability.

Generally, haematological parameters in fish are suitable biomarkers for evaluating the potential risk of pollutants (Roche and Boge, 1996). Cyriac et al. (1989) recognized changes in several haematological variables as indicators of metal exposure. However, the present findings indicate that in the *C. gariepinus*, sub-chronic exposure to lead affects blood parameters and plasma chemistry. The normal blood parameters suggested that there was no much osmotic disturbance and change of oxygen carrying capacity during lead exposure. Adeyemo (2007) reported decreased haemoglobin, RBC count and haematorit values in *C. gariepinus* exposed to lead nitrate. Al-Akel et al. (2010) examined blood chemistry in

Cyprinus carpio exposed to dietary Cu and found no significant change in haematocrit, haemoglobin, RBC and serum minerals. However, waterborne exposure to Cu resulted in significant change in haematological parameters in tilapia (Nussey et al., 1995) and rainbow trout (Dethloff et al., 2001). This difference between dietary and waterborne Cu exposure is possibly due to gill damage from Cu. Generally, metal exposure might result in gill damage, which in turn alters blood parameters (Pelgrom et al., 1995), and waterborne exposure is more toxic than dietary exposure in fish (Handy, 1996). However, Hodson et al. (1978) chronically exposed rainbow trout to waterborne lead and observed that haematocrit was unaffected by metal exposure. GOT and GPT are important diagnostic tools in medicine and are used to detect the adverse effects produced by various pollutants (Nelson and Cox, 2000). Our results showed that lead exposure significantly increased the activity of PGOT and PGPT in *C. gariepinus* in all exposed groups. McKim et al. (1970) suggested that 21 days of exposure to 38.2 and 69.2 mgCu/L in brook trout resulted in increased SGOT and this was due to cellular degradation in the liver. Suttle and Mills (1966) showed that in pigs, the activity of SGOT was markedly elevated when 450 mg Cu/kg was added to food. Moreover, Vaglio and Landriscina (1999) suggested that liver is rich in GOT and GPT, and with tissue damage, resulted in the liberation of large quantities of these enzymes into the blood. Therefore, increase in PGOT and PGPT activities of fish may indicate the presence of liver damage. The high values of activities can be ascribed to the liver necrosis caused by the toxicants and a possible damage to the hepatocytes. Same line of reasoning was given by Zaki et al. (2010) for higher values recorded for these enzymes after the treatment of lead.

Several heavy metals have been reported to stimulate interrenal activity and plasma corticosteroid and glucose levels in fish (Pratap and Wendelaar Bonga, 1990). Hypersecretion of adrenalin and cortisol are considered primary stress responses. These effects trigger a broad suite of biochemical and physiological alterations called secondary stress responses. Metabolic effects include hyperglycemia, depletion of tissue glycogen reserves, catabolism of muscle protein, and altered blood levels of protein, cholesterol, and free fatty acids (Wendelaar Bonga, 1997). *C. gariepinus* exposed to different lead concentrations presented a “classical general adaptation syndrome to stress”, since hyperglycemia was reported. The stress-response magnitude was dose dependent. Fish exposed to 24.4 mg Pb/L showed a larger glucose increase. The response to the lowest lead concentration (6.1 mg/L) might represent adaptation, while that for the highest concentration seems to characterize exhaustion. Adaptation, implying changes in several related physiological processes, permits homeostasis return. Exhaustion, however, may occur if the extent of stress is sufficient and characterized by a depletion of liver glycogen.

Sperm density and motility were reduced after the exposure of lead acetate for 6 weeks. The aforementioned findings get support from the report of Njiwa et al. (2004), where they have recorded reduction in count and activity of sperm after exposure of DDT and Archlor 1254. Lahnsteiner et al. (2005) have also reported the inhibition of maturation and quality of semen and eggs in *Salmo trutta* following exposure of bisphenol during spawning phase. Singh and Singh (2008) have reported the presence of c-HCH, DDT and chlorpyrifos beyond the permissible limit in gonads of some edible catfish and carps during pre-monsoon season captured from the polluted rivers Gomti and Ganga. Generally, the scale and duration of motility was reduced in the edible fishes of polluted environments. Our result is supported further by the observations of Kime and Nash (1999) who have reported that in catfish (*C. gariepinus*), sperm motility was decreased by mercuric chloride at a concentration of 1µg/L. Both cyprinid and salmonid fish were less sensitive (1 mg/L) whereas tributyltin (TBT) was extremely toxic and decreased sperm motility at the concentration less than 1 µg/L. This reduction in scale and duration of sperm motility could therefore be due to the influence of the xenobiotic on the endocrine system. Due to low motility the chance of fertilization is decreased. This may be explained on the basis of the fact that eggs have unique point of penetration, the micropyle, which the spermatozoa have little chance of reaching when their concentration is too low (Billard, 1988) or when their motility is reduced by toxic substances. Rurangwa et al. (1998) have shown that in African catfish, sperm motility has been affected significantly at very low mercury concentration of 0.001 mg/L. A little effect was noticed on the development of the ovary of fish exposed to lead acetate. It is clear from the results that the fecundity of fish was not affected but the sizes of eggs produced were comparatively smaller in size in the fish exposed to lead. Probably the fish exposed to toxicant utilized more energy in normal metabolism for detoxification and excretion of pollutant and spare less amount of energy for growth and reproduction. Hence, availability of inadequate energy results in reduced growth of eggs in the fish exposed to lead acetate.

Conclusion

From the results obtained in the present study it can be concluded that the lead acetate is toxic to *C. gariepinus*. The 96 h LC_{50} value (122 mg/L) recorded for the *C. gariepinus* is high as compared to other heavy metals. Exposure to chronic sub-lethal concentrations of lead acetate resulted in significant haematological and biochemical alterations. The specific growth rate and food conversion efficiency were also reduced after the

exposure of lead. These changes suggest that the treated fish are faced with a serious metabolic crisis. The lower values of RBC count, haemoglobin concentration and haematocrit values in the exposed fish are indicative of damages caused to hemopoitic organs by the lead. The present results clearly indicate that the discharge of the effluents containing lead acetate into the water bodies may be a threat to both aquatic fauna and flora as well as humans.

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