African Journal of Environmental Science and Technology Vol. 5(12), pp. 1017-1026, December 2011
Available online at http://www.academicjournals.org/AJEST
DOI: 10.5897/AJEST10.278
ISSN 1996-0786X ©2011 Academic Journals

Full Length Research Paper

Effect of lead acetate administered orally at different dosage levels in broiler chicks

Muhammad Suleman[1]*, Ayaz Ali Khan[2], Zahid Hussain[3], Muhammad Amir Zia[4], Sohaib Roomi[3] Farooq Rashid[1], Arshad Iqbal [3] and Rafaqat Ishaq[1]

[1]Animal Science Institute, National Agriculture Research Centre, Islamabad.
[2]Department of Biotechnology, University of Malakand, Chakdara, Dir(L) N. W. F. P. Pakistan.
[3]Plant Genomics and Biotechnology PIASA NARC Islamabad.
[4]Crop Sciences Institute, NARC Islamabad.

Accepted 14 February, 2011

The project was conducted to evaluate the effect of lead administered as lead acetate at different dosage levels via drinking water in broiler chicks. Thirty-five healthy chicks were divided into seven groups (five chicks each) and one group was kept as un-medicated control. Groups A, B, C, D, E and F were medicated with lead acetate in a single dose at a rate of 80, 120, 160, 200, 240 and 280 mg/kg of body weight respectively for twenty five days consecutively. Various biochemical parameters, that is, glutamate pyruvate transaminase, creatinine and uric acid were determined by using spectrophotometer. A significant (P<0.05) increase was recorded in GPT, creatinine and uric acid levels in all medicated groups. The GPT, creatinine and uric acid levels were significantly (P<0.05) higher in groups medicated with high doses of 240 and 280 mg/kg b.wt of lead acetate. Analysis of variance showed that the DATA were significant not only from the single factor (dose/days) point of view, but also from their combined effect (dose rate × different days of analysis), which gave significant results with a P value less than 0.05. The mortality rate of 20% was observed for the groups medicated with 120, 160 and 200 mg/kg b.wt, while 60% was observed for the groups medicated with 240 and 280 mg/kg b.wt. Postmortem revealed gross lesions on liver, lungs, kidney and brain at high doses of lead acetate. The lead was also accumulated in different organs, such as, the bone (14.83 ± 0.18 µg/g), brain (2.63 ± 0.16 µg/g) and liver (1.05 ± 0.16 µg/g). These results showed that lead possessed significant capability of bioaccumulation. However, it also revealed that lead toxicity increased as the dose increased and high dose of lead caused both hepatotoxicity and nephrotoxicity in broiler chickens.

Key words: Lead acetate, hepatotoxicity, nephrotoxicity, broiler chicken.

INTRODUCTION

Raising poultry in Pakistan has virtually proven to be a profitable enterprise, as it is the best source of cheap, palatable and nutritious food protein. The shortage of animal protein in Pakistan is estimated at 0.93 million tons on the basis of a human population of 140 million. This shortage of protein can be well managed with increase in the production of poultry meat at a reasonable cost (Alam et al., 2007). The conditions which arouse suspicion of poisoning are illnesses in a number of previously healthy animals, at the same time, and showing the same signs and necropsy findings at the same degree of severity. The appearance of clinical illness, soon after feeding and after a change of ration, medication or spraying, or after a change to new posture, is a common history in many outbreaks of diseases caused by chemical agents. Poisoning is, in most instances, accidental, although it may occasionally be deliberate. The cause of lead poisoning is the accidental ingestion of sources of lead compounds or the ingestion of animal feed (Radostitis et al., 1994).

Heavy metals become toxic when they are not metabolized by the body and accumulated in the soft tissues. Heavy metals may enter the human body through food, water, air, or absorption through the skin when they come in contact with humans in agriculture and in manufacturing,

*Corresponding author. E-mail: somisss@yahoo.com. Tel: +923339498369.

pharmaceutical, industrial, or residential settings (Radostitis et al., 1994). Lead acetate is a chemical compound, that is, a white crystalline substance having slight acetic acid odor with a sweetish taste. Like other lead compounds, it is very toxic. Lead acetate is soluble in water and glycerin. In low concentration, it is the principal active ingredient in progressive types of hair coloring dyes. Lead acetate is also used as a mordant in textile printing and dyeing, as a drier in paints and varnishes, and in preparing other lead compounds (Seacole, 1990). The common sources of lead in animals are lead bearing paints. There is a considerable variation between species in their susceptibility to lead and the chemical composition of the compound containing lead, which may influence its toxicity. The toxicity also varies with the chemical form of the lead. Lead acetate is very soluble and more toxic than insoluble lead oxides, or solid lead sheeting (Radostitis et al., 1994).

Birds can be exposed to lead via air, water, food, lead batteries, paints, pestiside and gasoline. Previous researchers have found trends of decreasing net brain weight with increasing amount of lead in birds (Park et al., 2001). The lesions, including degeneration of liver and kidney, vary in their severity with the tissue levels of lead attained (Radostitis et al., 1994). Serum glutamate pyruvate transaminase (SGPT) is also known as alanineamino transferase (ALT), an enzyme produced in hepatocytes, the major cell type in the liver. ALT is often referred to as a liver function test; however, its level in the blood indicates little about the function of the liver. The level of ALT in the blood is increased in conditions in which hepatocytes are damaged or are dead. As cells are damaged, ALT leaks out into the bloodstream. The ALT level is also increased in cases of liver cell death resulting from other causes, such as shock or drug toxicity (Jaeger and Hedegard, 2002). Exposure to lead acetate can cause adverse effects on the kidneys, ureter or bladder. The kidney is unusually susceptible because of its role in filtering harmful substances from the blood.

Some of the toxicants such as lead and cadmium cause acute injury to the kidney, while others produce chronic changes that can lead to end-stage renal failure or cancer (Amdur and Doull, 1996). Uric acid is made in the liver and excreted by the kidney. Uric acid is a waste product that results from the break down of purine (a nucleic acid). If the liver produces too much uric acid, the patient will have too much of it in the blood (Janis, 2002). Creatinine is a breakdown product of creatine, which is an important molecule of the muscle. Moreover, a serum creatinine test measures the amount of creatinine in the blood. The test is performed to evaluate the kidney's function. If the kidney function is abnormal, creatinine levels will increase in the blood, due to decreased excretion of creatinine in the urine (Nissl and Terra, 2004; Hecht, 2006).

Lead does not remain in tissues for long periods, except in bones where it is deposited in an inert form, but from which it can be liberated at a later date in sufficient quantity to cause chronic lead poisoning (Radostitis et al., 1994).

MATERIALS AND METHODS

Experimental animals

Day old broiler chicks were used as an experimental animal, purchased from the local market. A total of fifty chicks were kept under observation for twenty-one days in the Biopark of Biotechnology Department, University of Malakand with the purpose of selecting three weeks old healthy chicks. Same feed and water was provided to all the chicks during rearing, and it was seen that no antibiotic therapy was done during the experiment. Thirty-five healthy broiler chicks were selected for further experiment.

Heavy metal used

Lead acetate [$(CH_3 COO)_2 Pb.3H_2O$] (Merck Germany) and heparin injection (Rotex Medical Trittau, Germany) were used in this study. Each ml of heparin contains heparin = 5000IU.

EXPERIMENTAL MODEL

Grouping of broiler chicks

After rearing the chicks for twenty-one days, thirty-five chicks were divided into seven groups (A, B, C, D, E, F and G) on the basis of their body weight. Each group comprised five chicks.

Treatment

Groups A, B, C, D, E and F were treated once a day with lead acetate at a dose rate of 80, 120, 160, 200, 240 and 280 mg/kg body weight, respectively. The medication was done via oral route for twenty-five days consecutively. Group G was kept as untreated.

Blood collection

Strict aseptic conditions were used during the blood sampling. The blood samples of 3 ml were collected from the wing vein of chicks for conduction of various biochemical tests. The blood samples were collected at 0 day before medication and at the 5th, 15th and 25th day during medication, while at the 5th day of post medication, GPT, creatinine and uric acid were determined.

Isolation of plasma

The blood samples were centrifuged at 4000 rpm for 5 min. Plasma was separated and was used for the analysis of biochemical tests. Those plasma samples which were kept pending, were stored at -20°C.

Equipment used

Shimadzu UV-visible double beam spectrophotometer 1700 Pharma (Japan) was used for analysis of different biochemical

Table 1. The mean ± SD of GPT (iu/l) of the medicated and control groups of broiler chicks.

Groups	Dose rate (mg/kg- b. wt)	0 day (mg/dl)	5th day (mg/dl)	15th day (mg/dl)	25th (mg/dl)	30th day* (mg/dl)
A	80	1.29 ± 0.008	1.38 ± 0.010	1.57 ± 0.024	1.59 ± 0.033	1.47 ± 0.091
B	120	1.29 ± 0.008	1.51 ± 0.010	1.82 ± 0.048	1.99 ± 0.048	1.88 ± 0.072
C	160	1.28 ± 0.019	2.64 ± 0.017	2.83 ± 0.028	2.94 ± 0.091	2.11 ± 0.039
D	200	1.28 ± 0.017	2.52 ± 0.025	2.65 ± 0.015	2.84 ± 0.036	2.06 ± 0.029
E	240	1.29 ± 0.010	2.35 ± 0.070	2.54 ± 0.080	2.70 ± 0.079	2.03 ± 0.014
F	280	1.29 ± 0.008	2.06 ± 0.036	2.55 ± 0.049	2.59 ± 0.030	2.00 ± 0.024
G	Control	1.29 ± 0.008	1.28 ± 0.052	1.28 ± 0.033	1.26 ± 0.092	1.26 ± 0.025

* (5th day of post medication).

parameters, using plasma. Muffle furnace and atomic absorption spectrophotometer were used for ash preparation and for determination of the concentration of lead acetate in different organs, respectively.

Biochemical analyses

The following biochemical parameters were recorded to evaluate the effect of different dosage levels of lead acetate on liver and kidney function.

Estimation of glutamate pyruvate transaminase (GPT)

The Crescent diagnostic kits (Saudi Arabia) were used for estimation of serum glutamate pyruvate transaminase (SGPT) by following the procedure given in the kit protocol.

Estimation of creatinine

Creatinine was estimated by the Kinetic method, without deproteinisation-Jaffe reaction, using Crescent diagnostics kits prepared by Saudi Arabia.

Estimation of uric acid

Uric acid was estimated by enzymatic colourimetric test using human diagnostic kit (Germany) according to the procedure given in the kit protocol.

Postmortem examination

The post-mortems of dead broiler chicks were performed to study the gross pathological findings of the internal organs (liver, gizzard, heart, kidney and brain).

Lead concentration in tissue

The concentration of lead was determined in various tissues by using the method described by Uyanik et al. *(*2001).

Statistical analysis

The collected data were analyzed by two-way ANOVA for determination of variation among the effects of different dosage levels during different days. The mean, standard deviation (SD) and standard error from the mean (SEM) were also evaluated for each parameter, using Graph Pad Prism (Online software: www.graphpad.com) and SPSS computer programs.

RESULTS

Analysis of biochemical parameters

The study of the following biochemical parameters was undertaken in this research.

1. Glutamate pyruvate transaminase (GPT)
2. Creatinine
3. Uric acid

Glutamate pyruvate transaminase (GPT)

The GPT level of all groups was analyzed at 0 day before treatment, at 5, 15 and 25th day during medication, and at the 30th day of experiment after treatment. The mean ± SD of the obtained values of GPT for all the groups have been shown in Table 2. For the un-medicated control group (G), the maximum value of GPT was 10.62 ± 0.010 IU/l and the minimum was 10.61 ± 0.008 IU /l. The uppermost level of GPT for Group A was 23.56 ± 0.008 at the 25th day of experiment, while the lowest level was 18.33 ± 0.010 at the 5th day of experiment. The maximum level of GPT (48.28 ± 0.010 IU /l) for Group B was recorded at the 25th day of experiment, while the minimum level (35.24 ± 0.013 IU/l) was recorded at the 5th day of the experiment (Table 1). The highest mean ± SD level of GPT for Group C was 65.12 ± 0.010 IU/l at the 25th day of the experiment, while the lowest was 48.42 ± 0.010 at the 5th day of the experiment. Similarly, Group D showed high (58.56 ± 0.006 IU/l) and low level (45.31 ± 0.013 IU/l) levels of GPT at the 25th and 5th day of the experiment, respectively. The GPT level of Group E was high (51.84 ± 0.026 IU/l) at the 25th day and low (43.66 ± 0.006 IU/l) at the 5th day of the experiment. For Group F, the maximum GPT level of 45.06 ± 0.022 IU/l was recorded on day 25 and the minimum GPT level of

Table 2. The mean ± SD of creatinine (mg/dl) of the medicated and control groups of broiler chicks.

Group	Dose rate	0 day (IU/l)	5th day (IU/l)	15th day (IU/l)	25th (IU/l)	30th day* (IU/l)
A	80	10.61 ± 0.013	18.33 ±0.010	20.25 ±0.010	23.56 ± 0.008	22.12 ± 0.014
B	120	10.61 ± 0.010	35.24 ±0.013	38.31 ±0.014	48.28 ± 0.010	44.31 ± 0.012
C	160	10.62 ± 0.010	48.42 ±0.010	58.23 ±0.006	65.12 ± 0.010	59.28 ± 0.010
D	200	10.61 ± 0.005	45.31 ±0.013	48.31 ±0.010	58.56 ± 0.006	49.31 ± 0.013
E	240	10.61 ± 0.005	43.66 ±0.006	44.75 ±0.025	51.84 ± 0.026	48.28 ± 0.013
F	280	10.55 ± 0.126	42.41 ±0.005	43.26 ±0.008	45.06 ± 0.022	43.09 ± 0.008
G	Control	10.61 ± 0.008	10.61 ±0.005	10.61 ±0.006	10.62 ± 0.010	10.62 ± 0.008

* (5th day of post medication).

42.41 ± 0.005 IU/l was recorded on day 5 of the experiment.

The present study showed that lead acetate has an adverse effect on the liver and thus increased the level of GPT. As the dose increased, the GPT level also increased. At a dose rate of 80 mg/kg of b.wt (Group A), the GPT level was low and at a dose rate of 260 mg/kg of b.wt (Group E), the GPT level was high. Group C was highly affected than others, so their dose rate was low as compared to others (for example D, E and F). It was observed that as the administration was stopped, the GPT level tended towards normal as recorded on the 30th day of the experiment for each group. Analysis of variance (ANOVA) of GPT level for all groups has been shown in Table 4. Table 4 showed that there was significant difference ($P<0.05$) in GPT level in different groups and also at different days of analysis. Their combined effect (dose rate × different days of analysis) was significant with a P value less than 0.05.

A graph has been plotted between mean GPT level and different groups, as shown in Figure 1.

Creatinine level

The creatinine level for all non-medicated and medicated groups was recorded at 0 day before medication, at 5, 15 and 25th day during medication, and at the 30th day of the experiment (5th day of post medication). The mean ± SD of the obtained values is shown in Table 4. For Group G, which was kept as the un-medicated control, the minimum creatinine level was 1.26 ± 0.025 mg/dl and the maximum was 1.28 ± 0.033 mg/dl. Group A showed low level of creatinine (1.38 ± 0.010 mg/dl) observed on day 5 and high level (1.59 ± 0.033 mg/dl) of creatinine observed on day 25 of the experiment. Similarly, the minimum level of creatinine (1.51 ± 0.010 mg/dl) for Group B was recorded at the 25th day of the experiment and the maximum (1.99 ± 0.048 mg/dl) was at the 5th day of the experiment (Table 2). The lowest mean ± SD level of creatinine for Group C was 2.64 ± 0.017 mg/dl, while the highest was 2.94 ± 0.091 mg/dl, and they were recorded on the 5th and 25th day of the experiment, respectively. In the same way, Group D showed low level of creatinine (2.52 ± 0.025 mg/dl) on day 5 and high level (2.84 ± 0.036 mg/dl) of creatinine on day 25 of the experiment.

The observed low and high level of creatinine for Group E was 2.35 ± 0.070 and 2.70 ± 0.079 mg/dl at the 5th and 25th day of the experiment, respectively. For Group F, the minimum creatinine level (2.06 ± 0.036 mg/dl) was recorded on day 5 and the maximum (2.59 ± 0.030 mg/dl) was recorded on day 25 of the experiment. Analysis of variance (ANOVA) of the creatinine level for all groups is shown in Table 4. The table showed that there was significant difference ($P<0.05$) in creatinine level in different groups and also at different days of analysis. Their combined effect (dose rate × different days of analysis) was also significant with a P value less than 0.05. A graph has been plotted between the mean creatinine levels of different groups, as shown in Figure 2.

Uric acid

The uric acid level of all groups was analyzed at 0 day before medication, at 5, 15 and 25th day during medication, and at the 30th day of experiment (5th day of post medication). The mean ± SD of the obtained values of uric acid is shown in Table 6. For the un-medicated control group (G), the maximum value of uric acid was 5.60 ± 0.010 µg/dl and the minimum was 5.59 ± 0.022 µg/dl. The uppermost level of uric acid for Group A was 7.12 ± 0.005 µg/dl at the 25th day of the experiment, while the lowest level was 6.02 ± 0.013 µg/dl at the 5th day of the experiment. Similarly, the maximum level of uric acid for Group B was 8.93 ± 0.021 µg/dl and the minimum was 7.12 ± 0.013 µg/dl at the 25th and 5th day of the experiment, respectively. The peak level of uric acid for Group C was recorded on day 25 (11.94 ± 0.015 µg/dl), while the lowest level was found on day 5 of the experiment (10.53 ± 0.371 µg/dl). Group D also showed high level of uric acid on day 25 (11.07 ± 0.024 µg/dl) and low level on day 5 of the experiment (9.67 ± 0.057 µg/dl), as shown in Table 3. However, Group E showed high level of uric acid on the 25th day of the experiment and the obtained value was 10.59 ± 0.013 µg/dl, while the

Table 3. The plasma uric acid level (mean ± SD) of various groups after administration of lead acetate at different dosage levels.

Groups	Dose rate	0 day (µg/dl)	5th day (µg/dl)	15th day (µg/dl)	25th (µg/dl)	30th day* (µg/dl)
A	80	5.58 ± 0.006	6.02 ± 0.013	6.97 ± 0.033	7.12 ±0.005	6.96 ± 0.072
B	120	5.58 ± 0.009	7.12 ± 0.013	7.97 ± 0.029	8.93 ±0.021	8.15 ± 0.021
C	160	5.57 ± 0.010	10.53 ± 0.371	11.31 ± 0.008	11.94 ±0.015	11.00 ± 0.010
D	200	5.58 ± 0.008	9.67 ± 0.057	10.53 ± 0.010	11.07 ±0.024	10.12 ± 0.006
E	240	5.58 ± 0.006	9.16 ± 0.491	10.02 ± 0.033	10.59 ±0.013	10.03 ± 0.049
F	280	5.58 ± 0.010	8.03 ± 0.029	9.27 ± 0.046	10.00 ±0.028	9.52 ± 0.022
G	Control	5.58 ± 0.008	5.59 ± 0.022	5.59 ± 0.013	5.59 ± 0.018	5.60 ± 0.010

* (5th day of post medication).

Table 4. Anova for GPT, creatinine and uric acid at different days for different amounts of medication.

Parameter	Corrected model (F-value)	Intercept (F-value)	Dose rate (F-value)	Days (F-value)	Dose rate* days (F-value)	Sig.*
GPT	10283159.663	1155453036.840	27170568.374	35395863.751	1875856.803	0.00
Creatinine	677.062	251301.869	109.765	3703.531	314.475	0.00
Uric acid	1660.773	803171.494	4579.927	5381.550	310.854	0.00

*Sig = P value. P<0.05 = significant, P>0.05 = Non-significant, *The above observation shows that the DATA are significant not only from the single factor (dose/days) point of view, but also from their combined effect, which gives significant results.

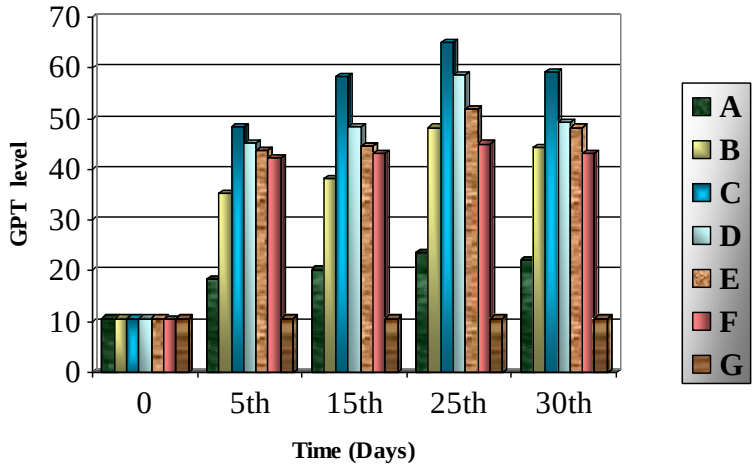

Figure 1. Graph showing the plasma GPT level of the control group, as well as the groups medicated with lead acetate at different dosage levels. A = Lead acetate administered at the dose rate of 80 mg/kg; B = Lead acetate administered at the dose rate of 120 mg/kg; C = Lead acetate administered at the dose rate of 160 mg/kg; D = Lead acetate administered at the dose rate of 200 mg/kg; E = Lead acetate administered at the dose rate of 240 mg/kg; F = Lead acetate administered at the dose rate of 280 mg/kg; G = the un-medicated group.

lowest level of uric acid for the said group was observed on the 5th day of the experiment with a level of 9.16 ± 0.491. For Group F, the maximum uric acid level (10.00 ± 0.028 µg/dl) was recorded on day 25 and the minimum (8.03 ± 0.029 µg/dl) was on day 5 of the experiment (Table 3).

Analysis of variance (ANOVA) of the uric acid level for all groups is shown in Table 4. Table 4 shows that there is a significant difference (P<0.05) in the uric acid level in different groups and also at different days of analysis. Their combined effect (dose rate × different days of analysis) was also significant with a P value less than 0.05. However, a graph has been plotted between the mean uric acid levels of different groups, as shown in

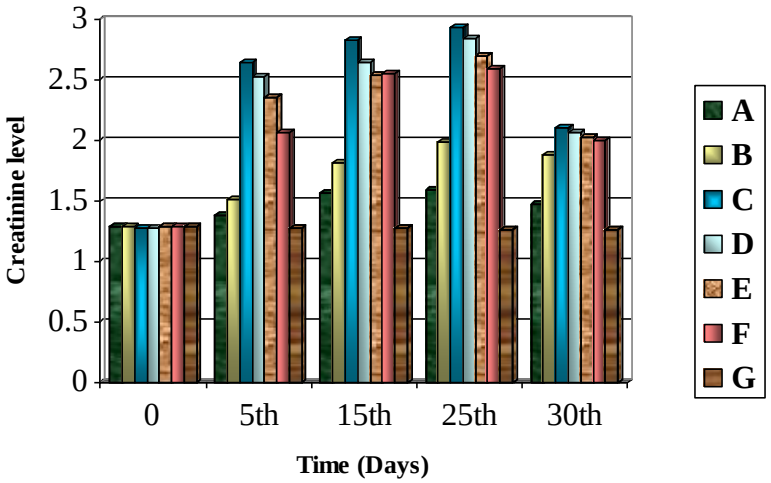

Figure 2. Graphical presentation of creatinine level during different days due to different lead acetate dose administered. A = Lead acetate administered at the dose rate of 80 mg/kg; B = Lead acetate administered at the dose rate of 120 mg/kg; C = Lead acetate administered at the dose rate of 160 mg/kg; D = Lead acetate administered at the dose rate of 200 mg/kg; E = Lead acetate administered at the dose rate of 240 mg/kg; F = Lead acetate administered at the dose rate of 280 mg/kg; G = the un-medicated group.

Table 5. The post-mortem findings of dead chicks during the experiment.

Group	Dose (mg/kg b.wt)	Liver	Lungs	Kidney	Heart	Brain	Gizzard
A	80	Pale	Normal	Swollen	Normal	Normal	Normal
B	120	Pale	Congested	Swollen	Normal	Normal	Normal
C	160	Pale	Congested	Swollen	Swollen	Congested	Normal
D	200	Pale	Congested	Swollen	Swollen	Normal	Normal
E	240	Pale	Congested	Swollen	Swollen	Normal	Normal
F	280	Pale	Congested	Swollen	Swollen and dark in color	Normal	Normal
G	Nil						

Figure 3.

POSTMORTEM FINDINGS

The postmortem of dead chicks were performed to study the effect of lead acetate on liver, kidney, lungs, heart, brain and gizzard (Table 5). One chick from Groups A, B, C and D, and three from E and F died during the entire period of the experiment.

Estimation of mortality rate

The mortality rate for each group was estimated in percentage, using the following formula.

$$\frac{\text{No. of dead chicks}}{\text{Total no of experimental chicks}} \times 100$$

Mortality rate in various groups

The mortility rate was 0% in Groups A and G, 20% in Groups B, C and D and 60% in Groups E and F.

Determination of lead concentration in organs

The concentration of lead in various organs (brain, bone and liver) of the medicated Groups was determined using the atomic absorption spectroscopy at the department of Physics (Centralized Resource Laboratory), University of Peshawar. In the case of the bone, Group C presented the minimum concentration of lead with a mean ± SD value of 5.750 ± 0.957 μg/g, while Group F presented the maximum concentration with a mean ± SD value of 30.75 ± 0.957 μg/g (Table 6). In the case of the brain, the minimum Pb concentration was found in Group B (3.275 ± 0.96 μg/g) and the maximum was found in Group

Table 6. Concentration of lead in various tissues (bone, brain and liver) of medicated groups.

Sample	Group						
	A	B	C	D	E	F	G
Bone	7.250 ± 0.500	12 ± 0.816	5.750 ± 0.957	14.50 ± 1.291	21.00 ± 0.816	30.75 ± 0.957	1.250 ± 0.500
Brain	3.80 ± 0.082	3.275 ± 0.96	2.400 ± 0.294	0.053 ± 0.013	7.675 ± 0.150	1.362 ± 0.038	0.200 ± 0.141
Liver	0.018 ± 0.01	0.680 ± 0.008	1.753 ± 0.010	0.558 ± 0.013	0.950 ± 0.008	2.395 ± 0.013	0.012 ± 0.005

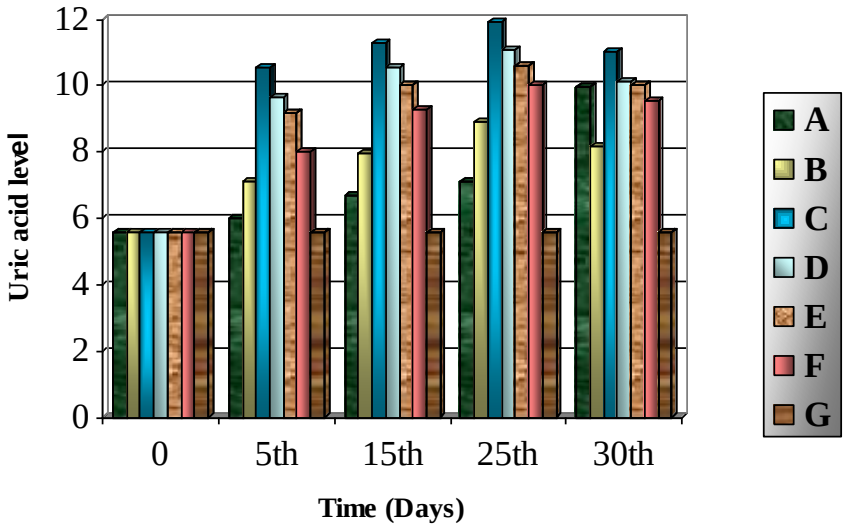

Figure 3. Graph showing the plasma uric acid level of different groups at different days. A = Lead acetate administered at the dose rate of 80 mg/kg; B = Lead acetate administered at the dose rate of 120 mg/kg; C = Lead acetate administered at the dose rate of 160 mg/kg; D = Lead acetate administered at the dose rate of 200 mg/kg; E = Lead acetate administered at the dose rate of 240 mg/kg; F = Lead acetate administered at the dose rate of 280 mg/kg; G = the un-medicated group.

(7.675 ± 0.038 μg/g). After the analysis of the samples of liver, Group A showed the minimum level of lead (0.018 ± 0.01 μg/g), while Group F showed the maximum level of lead (2.395 ± 0.013 μg/g). The control group (G) has a mean ± SD value of 1.250 ± 0.500 μg/g of lead acetate in bones, while it has 0.200 ± 0.141 and 0.012 ± 0.005 μg/g in the brain and liver, respectively. However, a graph has been plotted between the mean concentrations of lead and different medicated and control groups (Figure 4).

From the present results, it has been shown that lead accumulates in bones at high extent than in the brain, while the least quantity was determined in the liver. Therefore, it has been observed that lead has the capability to accumulate in bones and brain.

DISCUSSION

Heavy metals become toxic when they are not metabolized by the body and accumulated in the soft tissues. Heavy metals may enter into the human body through food, water, air, or absorption, or may occur through the skin when they come in contact with humans in agriculture (Roberts, 1999). Like other lead compounds, lead acetate is very toxic. This study was undertaken to investigate the effect of lead acetate on GPT, creatinine and uric acid in broiler chicks. For this purpose, the broiler chicks were reared and intoxicated with lead acetate. Blood samples were collected and analyzed for blood biochemical parameters (GPT, creatinine and uric acid).

Glutamate pyruvate transaminase level (GPT)

The level of GPT is generally increased in situations where there is damage to the liver cell membrane. All types of liver inflammation can cause increase in GPT (Cobot, 2006). Rapid increases in GPT may indicate an acute process, while slow increase may be due to bile duct obstruction. Cell damage causes elevations of serum/plasma GPT due to leakage. The elevation of serum/plasma GPT correlates with the number of cells that is damaged. Falling levels of GPT may indicate

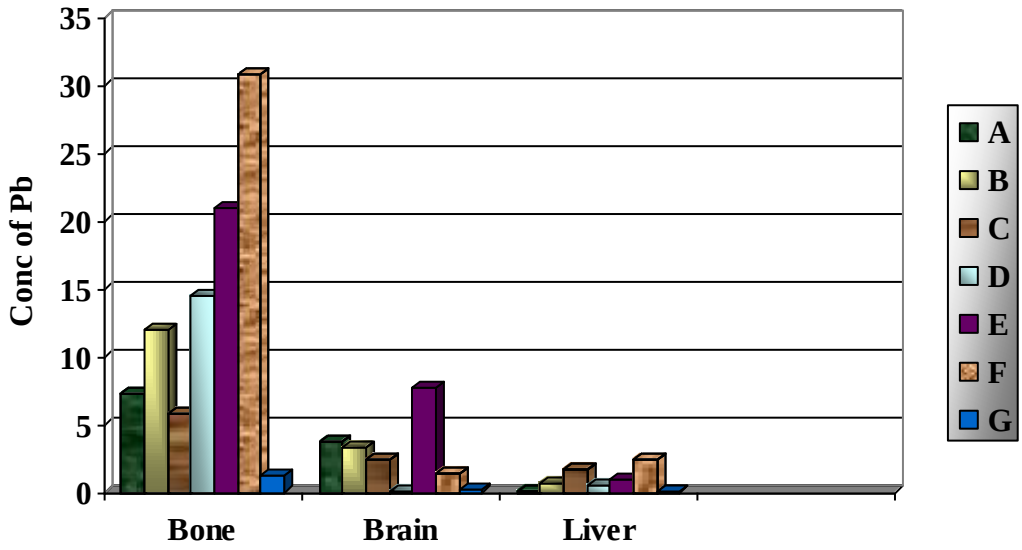

Figure 4. Amount of lead estimated in various tissues of the treated chicks groups. A = Lead acetate administered at the dose rate of 80 mg/kg; B = Lead acetate administered at the dose rate of 120 mg/kg; C = Lead acetate administered at the dose rate of 160 mg/kg; D = Lead acetate administered at the dose rate of 200 mg/kg; E = Lead acetate administered at the dose rate of 240 mg/kg; F= Lead acetate administered at the dose rate of 280 mg/kg; G = control group.

recovery (Fleming, 2006). GPT is more important in the diagnosis of heart and liver damage, which causes heart attack during toxicity or infection (David and Michael, 2000). Plasma GPT in various Groups (A, B, C, D, E, F and G) was determined during and after treatment. The maximum and minimum value of GPT for the un-treated control Group (G) was 10.62 ± 0.010 and 10.61 ± 0.008 IU/l, respectively, which was the lowest when compared with other medicated groups. This has been supported by Tatjana et al. (2005).

In the present study, various dosage levels of lead acetate were administered, which led to the increase in GPT. Al-Wabel et al. (2007) has reported the same results in rats, which have also been treated with lead acetate and it showed that the level of GPT increased from an average of 23.0 to 37.3 IU/L. Same results have also been found by Ashmawy et al. (2005), who mentioned that lead acetate has a toxic effect on liver and increased the level of GPT. Similar results have also been published by Hayashi et al. (2005). They found that, lead acetate increased the serum GPT level. Also, similar results have been mentioned by many researchers (Shalan et al., 2005; Sokkary et al., 2005; Tatjana et al., 2005). Swaran et al. (1987) repeated that lead acetate affected the value of GPT.

Creatinine

Creatinine is the breakdown product of creatine, which is an important part of the muscle. The test is performed to evaluate the kidney function. If the kidney function is abnormal, the creatinine level will increase in the blood, due to decreased excretion of creatinine in the urine (Nissl and Terra, 2004; Hecht, 2006). Serum/plasma creatinine is a more sensitive indicator of renal function than the blood urea nitrogen (June and Juanita, 2004). The presence of the increased level of urea and creatinine concentration in the blood suggests the inability of the kidney to excrete these products (Overu et al., 2004). The creatinine values for all groups showed that kidney had been affected during lead acetate administration. As the dose increased, the creatinine level also increased. In some cases (Group C), the effect was higher as compared to the high dose administrated groups (D, E and F). It was observed that when its administration was stopped, creatinine level tended to decrease. Plasma creatinine in various groups (A, B, C, D, E, F and G) was determined during and after medication. The maximum and minimum values of creatinine for the non-treated control group (G) were 1.28 ± 0.033 and 1.26 ± 0.025 IU/l, respectively, which was the lowest as compared to other treated groups.

The present results have been supported by Khalil-Manesh et al. (1992), who mentioned that lead acetate increased serum creatinine level as compared to the control group, where rats were intoxicated. The normal creatinine level in rabbits, reported by Guignard and Drukker (1999), was also in agreement with the present results. In the present study, various dosage levels of lead acetate were administered, which led to the increase in creatinine as described in the results. Same results have been found by Abd El Rahiem et al. (2007), who mentioned that lead acetate increased the level of creatinine

in lead acetate treated rats. Similar results have been reported by many researchers (Abdel-Razik et al., 2007; Haneef et al., 1998; Khalil-Manesh et al., 1992).

Uric acid

Uric acid is a white, odourless, tasteless crystalline organic compound of carbon, nitrogen, oxygen and hydrogen. It is made in the liver and excreted by the kidney. Uric acid is a waste product that results from the break down of purine, which is a nucleic acid. If the liver produces too much uric acid, the patient will have too much of it in the blood. Uric acid is a weak organic acid that is barely soluble in water and insoluble in alcohol and ether (Encyclopedia, 2006). The uric acid test is used to determine the break down of the body cells. The test is also used to monitor the uric acid level when a patient has taken chemotherapy or radiation treatment (Lab Test Online, 2003). The present study showed that lead acetate affected the level of uric acid. As the dose increased, the uric acid level also increased. In some groups, the effect was higher (that is, Group C) as compared to other high dose medicated groups (that is, D, E and F). After the cessation of lead acetate administration, uric acid tended to decrease. In the present study, uric acid levels in various groups (A, B, C, D, E, F and G) were determined during and after medication. For the un-medicated control group (G), the maximum value of uric acid was 5.60 ± 0.010 µg/dl and the minimum was 5.59 ± 0.022 µg/dl.

In the present study, various dosage levels of lead acetate were administered, which led to the increase in uric acid as described in the results of the study. However, same results have been found by Abd El Rahiem et al. (2007) in rats, and have also been reported by Dioka et al. (2004), who mentioned that exposure of human subjects to lead in petrol increased the concentrations of uric acid (357 ± 123 µ mol/l) as compared to unexposed subjects.

POST-MORTEM FINDINGS

In the un-medicated control (group G), no mortality was recorded. There was no mortality in the groups medicated with lead acetate at the dose rate of 80 mg/kg b.wt (Group A). A total of nine broiler chicks died during the entire experimental period (One from Groups B, C and D, and three from Groups E and F). The mortality rates observed for dosage levels of lead acetate at 120 mg/kg (Group B), 160 mg/kg (Group C) and 200 mg /kg (Group D) were 20% for Groups E and F medicated at the dose rate of 240, while the mortality rates at 280 mg/kg were 60%. So, it has been concluded that as the dose rate increased, the mortality rate also increased, although the postmortem findings of the control group (G) were normal. After performing the post-mortem for the medicated groups, the gross pathological findings were: pale liver, lungs normal at the dosage level of 80 mg/kg but congested at higher doses, swollen kidney, heart normal at low doses but swollen at higher doses, and gizzard normal. Chow et al. (2006) recognized chronic lead exposure as a potential cause of kidney damage.

Similar findings have been reported by other workers (Ashmawy et al., 2005; Sokkary et al., 2005; Francisco et al., 2003).

Concentration of lead in different organs

In the un-medicated control group, the level of lead was at the lowest concentration as compared to the intoxicated groups, described in the results. In the present study, the accumulation pattern of lead in different organs was: bone> brain > liver. Same results have been found by Taggart et al. (2006), who mentioned that lead is accumulated in the bone in greater amount than in the liver. Lopez et al. (2000) have also reported the same results. Similarly, Kalisinska and Szuberla (1996), in males (n = 34) and females (n = 23) of long-tailed duck, have also found same results, by mentioning that lead accumulates more in the brain. Furthermore, Schwarz et al. (1991) have also reported the same results.

Conclusion

From the present study, it has been observed that lead acetate alters the plasma GPT, creatinine and uric acid level in broiler chickens by affecting the liver and kidney function. The toxic signs were much more prominent when high doses (240 and 280 mg/Kg b.wt) were administered. There was no pronounced toxicity present when low doses of lead acetate were used. After postmortem examination, it was found that internal organs, such as liver, lungs, kidney, brain and heart were severely affected at high doses as compared to low doses of lead acetate. However, the presence of lead in different organs indicated that the different organs have the capability of lead bioaccumulation.

REFERENCES

Abd El RA, Maged M, Yassin, Nahed M, Aasi A, Rokaya M (2007). Blood, Serum Glucose and Renal Parameters in Lead-Loaded Albino Rats and Treatment with Some Chelating Agents and Natural Oils. Turk. J. Biol., 31(25): 25-34.

Abdel-Razik H, Farrag A, Mahdy K, Gamal H, Rahman A, Mostafa M 2007). Protective Effect of Nigella sativa Seeds Against Lead-induced Hepatorenal Damage in Male Rats. P. J. Biol. Sci.. 10(17):2809-2816.

Alam SM, Khan MA (2007). Poultry farming in Pakistan. Nuclear Institute of Agriculture Tando Jam, paklistan.http://www.pakistaneconomist.com/issue2000/issue32/i&e3.htm.

Al-Wabel NA, Mousa HM, Omer OH, Abdel-Salam AM (2007). Biological evaluation of symbiotic fermented milk against lead acetate

contamination in rats. J. Food, Agric. Environ., 5 (3,4):169-172.
Amdur CM, Doull J (1996). Health effect of lead acetate. The Basic Science of Poisons, 5th Ed. Pergamon Press, New York.
Ashmawy IM, El-Nahas AF, Salama OM (2005). Protective effect of volatile oil, alcoholic and aqueous extracts of Origanum majorana on lead acetate toxicity in mice. Basic. Clin. Pharmacol. Toxicol., 97(4): 238-43.
Chow KM, Liu ZC, Szeto CC (2006). Lead nephropathy: early leads from descriptive studies. Internal. Med. J. 36:678–681.
Cobot S (2006). The liver tests. http://www./ Weighcontrodoctor. com/healthtopics/healthyliver bo welbook/ p.98.
David JN, Michael MC (2000). Lehninger Principle of Biochemistry 3rd Edi. p. 631.
Dioka CE, Orisakwe OE, Adeniyi FAA, Meludu SC (2004). Liver and Renal Function Tests in Artisans Occupationally Exposed to Lead in Mechanic Village in Nnewi, Nigeria. Int. J. Environ., 21–25.
Encyclopedia (2006). Uric acid. http://www.infoplease.com/ encyclopedia. html.
Fleming S (2006). Liver disease. http://www.canin-epilepsy-guardian-angle.com/diagnostic_testing .html.
Francisco N, R.troya JD, aguera EI (2003). Lead and lead toxicity in domestic and free living birds. Avian Pathol., 32(1): 3-13.
Guinard JP, Drukker A (1999).Why Do Newborn Infants Have a High Plasma Creatinine? Pediatrics, 130(4):49.
Haneef SS, Swarup D, Dwivedi SK, Dash PK (1998). Effects of concurrent exposure to lead and cadmium on renal function in goats. Small Ruminant Res., 28(3):257-261.
Hayashi M, Yamamoto K, Yoshimura M, Kishimoto T, Shitara A (2005). Effects of fasting on distribution and excretion of lead following long-term lead exposure in rats. J. Arch. Environ. Contam. Toxicol. 24(2): 201-205.
Hecht F (2006). Creatinine Blood Test. http://www.medicinenet. com/sript/main/p#1.
Jaeger JJ, Hedegard H (2002). About blood tests. The Danish Hepatitis C web site Denmark.
Janis O (2002). Uric acid Tests. Gale Encyclopedia of Medicine. http:// www. Uric Acid Tests AHealthyMe_com.htm.
June HC, Juanita W (2004). Manual of laboratory tests. p.150.
Kalisinska E, Szuberla U (1996). Heavy metals in the brain of long-tailed duck (Clangula hyemalis) wintering in the Pomeranian Bay, Poland. Biol. Trace Elem. Res. 55(1-2):191-7.
Khalil-Manesh F, Gonick HC Cohen A, Bergamaschi E, Mutti A (1992). Experimental model of lead nephropathy. Environ. Res. 58 (1):35-54.
Lab test online (2003). Uric acid. http://www.labtestoline. org/understanding/analytes /uric_acid/test. html#how.
Lopez Alonso M, Benedito JL, Miranda M, Castillo C, Hernández J, Shore RF (2000). Arsenic, cadmium, lead, copper and zinc in cattle from Galicia, NW Spain. Sci Total Environ. 246(2-3): 237-48.
Nissl J, Terra RP (2004). Creatinine and Creatinine Clearance. Health wise (Medical Review)http:// www.bchealthguid.org/kbase/.
Overu SS, Berepubo NA, Nodu MB (2004). Biochemical blood parameters in semi-adult rabbits experimentally fed crude oil contaminated diets. Afr. J. Biotechnol. 3(6): 343-345.
Park J, Porter N, Franklin EW, College M (2001). The Effects of Lead Acetate on the Neural Development of Chick Embryos.
Radostitis OM, Blood DC, Gay CC (1994). Veterinary medicine. A text book of the diseases of cattle, sheep, pigs, goat and horses. Ed 8th, 31: 1469-1471.
Roberts JR (1999). Metal toxicity in children. In: Training Manual on Pediatric Environmental Health: Putting It into Practice. Emeryville, CA: Children's Environmental Health Network. http://www.cehn.org/cehn/trainingmanual/pdf/manual-full.pdf.
Schwarz T, Busch A, Lenk R (1991). Preliminary studies of the content of lead, cadmium and arsenic in feed, cattle and food of animal origin from different production regions of Saxony. Dtsch. Tierarztl. Wochenschr, 98(10): 369-72.
Seacole M (1990). Properties of lead acetate. http://www.en. wikipedia.org/wiki/Lead_acetate.
Shalan MG, Mostafa MS, Hassouna MM, Nabi EL, Refaie EL (2005). Amelioration of lead toxicity on rat liver with Vitamin C and silymarin supplements. Toxicology, 206(1): 1-15.
Sokkary GH, Rahman A, kamel ES (2005). Melatonin protects against lead-induced hepatic and renal toxicity in male rats. Toxicolology, 213(1-2): 25-33.
Swaran J, Flora S, Sushil K (1987). Effect of combined exposure to lead and ethanol on some biochemical indices in the rat. Biochem. Pharmacol., 36(4): 537-541.
Taggart MA, Figuerola J, Green AJ, Mateo R, Deacon C, Osborn D, Meharg AA (2006). After the Aznalcóllar mine spill: arsenic, zinc, selenium, lead and copper levels in the livers and bones of five waterfowl species. Environ. Res., 100(3):349-61.
Tatjana T, Dozic I, Dragana V, Pejovi J, Marjanovi M (2005). The Influence of chronic lead poisoning on the activity of some serum enzymes in rats. Acta Veterinaria, 55(5-6): 471-482.
Uyanik F, Eren M, Atasever A, Tunoku G, Kolsuz AH (2001). Changes in some Biochemical parameters and organs of broilers exposed to cadmium and effect of zinc on Cadmium induced alterations. Asian J. Animal Sci., 56(4): 317-19.