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# Protective Effect of Vitamin C on Amelioration of Heamatological and Biochemical Toxicity Induced by Lead in Male Rats

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## ABSTRACT

The aim of this study was to investigate the protective role of vitamin C against lead acetate induced in albino rats, considering the hematological and biochemical aspects. Thirty male rats were used and divided into six groups (5 rats per group). The first group was control group, the 2nd and 3rdwerevitamin C groups that received 100 or 200mg vitamin C /kg diet, the 4th and 5th groups were co-administered vitamin C at rate 100 or 200mg/kg diet plus lead (200mg/L), while6th group exposed to lead acetate (200mg/L). Samples were collected at 4, 8 and 12 weeks from start of the experiment. Lead acetate caused a significant increase in serum AST, ALT, ALP activities, in addition to a significant increase in serum cholesterol, triglyceride and urea. The obtained results displayed that treatment with lead significantly decreased serum total protein, albumin, RBCs and hemoglobin compared to the control group. On the other hand, administration of vitamin C with lead reduced the alterations in the previous parameters. Also, the results revealed that treatment with vitamin C alone decreased serum cholesterol and triglyceride and did not show any significant effect on other parameters.

***Key words:*** Treatment of rats with vitamin C with lead acetate alleviates the toxic effect of lead.

## Introduction

Environmental pollution is the presence of a pollutant in environment such as air, water, soil and consequently in food which may be poisonous or toxic and couldcause harm to living things in the polluted environment (Duruibe *et al*., 2007). The excessive amount of pollutants such as heavy metals in animal feed and feed stuffs are often due to human actions, resulting from either agricultural or industrial production or accidental or deliberate misuse (Aboul-Enein *et al*., 2010, Mohamed *et al*., 2009). There are at least 18 elements that characterize one or more inorganic pesticides. Of these elements, eight (barium, cadmium, mercury, thallium, lead, bismuth, antimony and boron) have not been shown to be essential to the growth of animals (El-Beltagi and Mohamed, 2010).Many heavy metals, including Pb, are known to induce over production of reactive oxygen species (ROS) and consequently enhance lipid peroxidation, decrease the saturated fatty acids and increase the unsaturated fatty acid contents of membranes (Afify and El-Beltagi, 2011). Also, it shown enhancing the production of ROS in a variety of cells resulting oxidative stress (Hayes and Laws, 1991).Lead is translocated through the food chain to man and animal, its toxicity depends on the chemical from administrated to animal, the route of administration and the frequency and duration of administration to animals. Lead can affect individuals at any age, but it has a disproportionate effect on children because their behavioral patterns place them at higher risk when expose to lead. Their bodies absorb a larger percentage of the lead that they ingest and they exhibit lead toxicity at lower level of exposure than adults (Baht and Moy, 1977).Vitamin C (ascorbic acid), a water soluble vitamin, derived from dietary sources such as citrus fruits, grape-fruits, berries, cabbage, tomatoes, pepper and leafy vegetables. The therapeutic potential of vitamin C is as a result of its antioxidant effect on free-radicals (Adaramoye *et al*., 2008).

The aim of this study was to determine the effect of vitamin C on hepatotoxic effect caused by lead acetate in rats.

## Material and Methods

*Materials:*

Lead acetate and vitamin C were obtained from Algomhuria pharmaceutical company, Cairo. Lead was reconstituted in distilled water prior daily administration, while vitamin C added to the diet.

*Animals:*

Thirty adult male albino rats were originally purchased from El-Osman farm, Cairo, Egypt, with an average live body weight 135g (ranged from 100-170g).The animals were raised in Animal house belonging to Faculty of Agriculture, Al-Azher University.

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*Experimental design:*

Animals were divided randomly into six equal groups, each contained 5 rats and fed one of the following diets:-

Group 1: Control.
Group 2:100mg Vitamin C mg/ kg diet.
Group 3:200mg Vitamin C mg/ kg diet.
Group 4:100mg Vitamin C mg / kg diet + 200mg lead /L.
Group 5:200mg Vitamin C mg/ kg diet + 200mg lead /L.
Group 6:200mg lead /L.

*Blood samples:*

Blood samples were obtained from rats by withdrawing blood from the orbital venous plexuses using a capillary tube. Samples were collected at 4, 8 and 12weeks from the start of experiment. Blood samples were collected into tubes, the first tube was heparinized for determining leucocytes profile, while the second non-heparinized and taken to centrifuge at 3000 rpm for 15 min to obtain serum. Serum was transferred into ependroff tube and stored at-20°C until subsequent analyses.

*Biochemical assays:*

Samples of red blood cells (RBCs) and hemoglobin were determined using Electronic DIAGON method according to (D-cell60).Serum AST and ALT activities was determined by using a quantitative colorimetric method of Schiele (1982). Alkaline Phosphatase (ALP) was determined using colorimetric method according to Kind and King (1971).Serum total protein was measured using kits depending on the method of Tietz (1995).Serum albumin was determined using colorimetric method according to Gindler and Westgard (1973).While Serum globulin was calculated by subtraction of albumin from total protein. Serum urea was determined by using enzymatic colorimetric method according to Tabacco (1979).Serum cholesterol was determined by using enzymatic colorimetric method according to Trinder (1969).Serum triglyceride was determined by using enzymatic colorimetric method according to Bucolo and David (1973).

*Statistical analysis:*

Data were subjected to analysis of variance using the general linier models procedure of SPSS software program (SPSS, version 11.0). All percentages were first transformed to arcsine and analyzed to approximate normal distribution before ANOVA. Also significant differences among means were determined by Duncan's multiple range tests at 5% level of significance. Data were analyzed by one way method.

**Results and Discussions**

The results presented in tables (1, 2 and 3) indicate that treatment of rats with lead acetate (200mg/L)for 4, 8 and 12 weeks significantly increased serum AST, ALT and ALP activities. While treatment of rats with vitamin C (100 or 200mg/kg diet) alone didn't show any significant effect on serum AST, ALT and ALP activities compared with the control group. Treatment of rats with lead plus vitamin C (100 or 200mg/kg diet) succeeded to decrease the elevation of serum AST, ALT and ALP activities to reach the same normal range of the control group.

The above result showed that exposure of rats to lead acetate significantly increased serum AST, ALT and ALP activities. These effects indicate that lead may cause a destruction of liver cells after accumulation in the tissue. Mongi *et al.* (2012) stated that AST is a mitochondrial enzyme present in large quantities in the heart, liver, skeletal muscles and kidney and its level in serum increase whenever these tissues are acutely destroyed presumably due to release from damaged cells. Meanwhile, the ALT is cytosolic enzyme present in liver, although its absolute amount is less than AST, a greater proportion of ALT present in liver compared with heart and skeletal muscles, thus what increase in serum is there for the AST which more specific for liver damage. Hamadouche *et al.* (2012) found that administration of lead acetate led to a significant rise in total ACP, and ALP activities. They observed that lead exposure produced pronounced hepatic histopathology evidence by histological alternations in liver, including focal necrosis with hepatocyte vacuolization and swelling, pyknotic nuclei, and dilation of central vein and sinusoids.

The result also showed that treatment of rats with vitamin C plus lead successfully prevented the increase in serum AST, ALT and ALP activities and maintained these activities within the normal range of the control group. These results indicate that vitamin C can reduce liver toxicity by lead acetate. Dakshinamuti and Dakshinamuti, (2001) reported that antioxidant vitamins are reported to provide protection against free radical damage in the body through their antioxidant activities.

**Table 1:** Means ±SE for the effect of treatments on serum ALT (U/L) activity.

| Treatments | Week 4 | Week 8 | Week 12 |
|---|---|---|---|
| Control | 25.72±0.46$^{C}$ | 20.73±1.81$^{B}$ | 32.68±0.50$^{B}$ |
| Vitamin C100 | 26.02±1.63$^{C}$ | 22.93±1.33$^{B}$ | 35.38±2.54$^{B}$ |
| Vitamin C200 | 24.20±0.88$^{C}$ | 21.18±1.79$^{B}$ | 34.95±1.17$^{B}$ |
| Vitamin C100+ Lead | 36.20±3.06$^{B}$ | 20.11±3.65$^{B}$ | 39.92±0.54$^{B}$ |
| Vitamin C200+Lead | 37.20±1.19$^{B}$ | 23.33±2.42$^{B}$ | 34.93±1.58$^{B}$ |
| Lead | 42.10±1.49$^{A}$ | 40.10±0.90$^{A}$ | 59.15±6.39$^{A}$ |

*Means in columns with different superscripts are different at (P≤0.05).*

**Table 2:** Means ±SE for the effect of treatments on serum AST (U/L) activity.

| Treatments | Week 4 | Week 8 | Week 12 |
|---|---|---|---|
| Control | 23.25±0.75 $^{B}$ | 16.45±0.40 $^{B}$ | 18.69±0.17 $^{B}$ |
| Vitamin C 100 | 23.52±0.12 $^{B}$ | 16.64±0.80 $^{B}$ | 17.39±0.64 $^{B}$ |
| Vitamin C 200 | 25.85±1.31 $^{B}$ | 15.68±1.19 $^{B}$ | 17.09±1.21 $^{B}$ |
| Vitamin C100 + Lead | 21.50±1.02 $^{B}$ | 19.45±0.48 $^{B}$ | 16.34±0.91 $^{B}$ |
| Vitamin C200 + Lead | 22.95±1.52 $^{B}$ | 18.63±1.81 $^{B}$ | 18.87±1.50 $^{B}$ |
| Lead | 34.23±0.77 $^{A}$ | 38.32±0.23 $^{A}$ | 32.10±0.99 $^{A}$ |

*Means in columns with different superscripts are different at (P≤0.05).*

**Table 3:** Means ±SE for the effect treatments on serum ALP (U/L) activity.

| Treatments | Week 4 | Week 12 |
|---|---|---|
| Control | 19.32±0.51 $^{B}$ | 20.65±0.29 $^{B}$ |
| Vitamin C 100 | 19.72±1.31 $^{B}$ | 20.30±0.89 $^{B}$ |
| Vitamin C 200 | 20.54±1.59 $^{B}$ | 21.52±0.09 $^{B}$ |
| Vitamin C 100 + Lead | 19.34±1.73 $^{B}$ | 18.97±1.60 $^{B}$ |
| Vitamin C 200 + Lead | 20.03±1.37 $^{B}$ | 18.90±0.24 $^{B}$ |
| Lead | 30.35±1.68 $^{A}$ | 35.01±1.91 $^{A}$ |

*Means in columns with different superscripts are different at (P≤0.05).*

The results show that treatment of rats with lead acetate significantly reduced serum total protein and albumin without any significant effect on serum globulin compared with the control group (Tables 4, 5 and 6). While treatment of rats with vitamin C alone (100 or 200mg/kg diet) didn't show any significant effect on serum total protein, albumin and globulin as compared with the control group. Treatment of rats with lead plus vitamin C (100 or 200mg/kg diet) significantly increased serum total protein and albumin as compared to the rats exposed to lead alone to reach the normal range of the control group.

The results show that serum total protein and albumin were markedly decreased by treatment with lead. The decrease in serum total protein may due to the decrease in serum albumin. The decrease in serum albumin may be due to the toxic effect of lead on the liver. Rivarola and Balegon, (1991) reported that the decrease in total proteins and albumin levels in liver could be attributed to changes in protein and free amino acids metabolism and their synthesis in the liver. Osman, (2013) showed that rats treated with lead acetate (3mg/kg) induced a significant decrease in serum total protein and albumin when compared with the control group.

The present results also showed that treatment with vitamin C (100 or 200mg/kg diet) plus lead significantly increased serum total protein and albumin compared to rats exposed to lead alone which reach the normal level of the control group. These results indicate that vitamin C can reduce the liver toxicity by lead. Gursu *et al.* (2004) showed that supplemented of rats with vitamin C (250mg/kg) and folic acid (1mg/kg) increased serum albumin.

**Table 4:** Means ±SE for the effect of treatments on serum total protein (g/dl).

| Treatments | Week 4 | Week 8 | Week 12 |
|---|---|---|---|
| Control | 8.17±0.30 $^{A}$ | 8.37±0.53 $^{A}$ | 8.89±0.79 $^{A}$ |
| Vitamin C 100 | 8.36±0.35 $^{A}$ | 8.34±0.43 $^{A}$ | 8.12±0.56 $^{A}$ |
| Vitamin C 200 | 8.64±0.11 $^{A}$ | 8.96±0.67 $^{A}$ | 8.86±0.53 $^{A}$ |
| Vitamin C100 + Lead | 8.28±0.65 $^{A}$ | 8.58±0.40 $^{A}$ | 8.20±0.29 $^{A}$ |
| Vitamin C200 + Lead | 8.47±0.38 $^{A}$ | 8.37±0.30 $^{A}$ | 8.30±0.28 $^{A}$ |
| Lead | 6.90±0.55 $^{B}$ | 6.65±0.49 $^{B}$ | 6.06±0.50 $^{B}$ |

Means in columns with different superscripts are different at (P≤0.05).

**Table 5:** Means ±SE for the effect of treatments on serum albumin (g/dl).

| Treatments | Week 4 | Week 8 | Week 12 |
|---|---|---|---|
| Control | 4.50±0.20 $^{A}$ | 4.30±0.18 $^{A}$ | 5.27±0.33 $^{A}$ |
| Vitamin C 100 | 4.26±0.26 $^{A}$ | 4.01±0.39 $^{A}$ | 5.16±0.20 $^{A}$ |
| Vitamin C 200 | 4.47±0.48 $^{A}$ | 4.65±0.44 $^{A}$ | 5.71±0.30 $^{A}$ |
| Vitamin C100 + Lead | 4.25±0.38 $^{A}$ | 4.95±0.087 $^{A}$ | 5.58±0.44 $^{A}$ |
| Vitamin C200 + Lead | 4.35±0.19 $^{A}$ | 4.95±0.10 $^{A}$ | 5.14±0.25 $^{A}$ |
| Lead | 3.51±0.43 $^{B}$ | 3.19±0.29 $^{B}$ | 3.18±0.29 $^{B}$ |

*Means in columns with different superscripts are different at (P≤0.05)*

**Table 6:** Means ±S.E for the effect of treatment on serum globulin (g/dl).

| Treatments | Week 4 | Week 8 | Week 12 |
|---|---|---|---|
| Control | 3.67±0.38$^{A}$ | 4.07±0.53 $^{A}$ | 3.62±0.802 $^{A}$ |
| Vitamin C 100 | 4.01±0.39 $^{A}$ | 4.33±1.05 $^{A}$ | 2.96±0.35 $^{A}$ |
| Vitamin C 200 | 4.17±0.51 $^{A}$ | 4.31±0.60 $^{A}$ | 3.15±0.21 $^{A}$ |
| Vitamin C100 + Lead | 3.88±0.23 $^{A}$ | 3.63±0.38 $^{A}$ | 2.62±0.46 $^{A}$ |
| Vitamin C200 + Lead | 4.12±0.40 $^{A}$ | 3.42±0.26 $^{A}$ | 3.16±0.37 $^{A}$ |
| Lead | 3.39±0.52 $^{A}$ | 3.46±0.76 $^{A}$ | 2.88±0.59 $^{A}$ |

*Means in columns with different superscripts are different at (P≤0.05)*

The result presented in Tables (7 & 8) indicates that treatment of rats with lead acetate for 4, 8 and 12 weeks significantly increased serum cholesterol and triglycerides as compared with the control group. Treatment of rats with vitamin C (100 or 200mg/kg diet) alone decreased serum cholesterol and triglycerides as compared with the control group. While treatment of rats with vitamin C (100 or 200mg/kg diet) plus lead succeeded to decrease the elevation of serum cholesterol and triglycerides levels to reach the normal levels of control group. The above result showed that exposure of rats to lead markedly increased serum cholesterol and triglycerides. This effect may due to the increase in lipolysis or to disturbance of lipid metabolism after accumulation of lead in the liver. Badr, (2010) showed that exposure of rats to lead markedly increase plasma triglyceride compared to the control rats. Liu and Lin, (1997) reported that the rise in serum lipid profile may be attributed to increase lipolysis which is mediated by increase of norepinephrine release, that act through interference with the intercellular function of $Ca^{+2}$ in the cytoplasm.

The result also showed that treatment of rats with vitamin C (100 or 200mg/kg diet) plus lead diminished the increase in serum cholesterol and triglycerides to reach the normal levels of the control group. These results may due to that vitamin C decrease the accumulation of lead in liver or/and decrease lipolysis. Eteng *et al.* (2006) showed that oral administration of vitamin C (100 or 200mg/kg) for 30 days produced a significant decrease in total cholesterol compared with the control group. They also revealed that the plausible explanation for the observed effect on serum lipids may be the activation of 7α-hydroxylase enzymes by vitamin C which enhances the conversion of plasma cholesterol to bile acid, resulting in decreasing serum cholesterol level. They also observed that deficiency of vitamin C inhibits 7α-hydroxylase, leading to block of bile acid synthesis and accumulation of cholesterol in a scorbutic Guinea pig. Seyrek *et al.* (2004) showed that vitamin C (500mg/kg) supplement in diet significantly decreased serum concentration of cholesterol and triglycerides in comparison to control animals. Gursu *et al.* (2004) showed that supplement of rats with vitamin C (250 mg/kg) and folic acid (1mg/kg) decreased cholesterol and triglycerides.

**Table 7:** Means ±SE for the effect of treatments on serum cholesterol (mg/dl).

| Treatments | Week 4 | Week 8 | Week 12 |
|---|---|---|---|
| Control | 80.03±13.4 $^{B}$ | 78.90±16.7 $^{B}$ | 79.31±17.6 $^{B}$ |
| Vitamin C 100 | 72.69±12.6 $^{C}$ | 70.52±11.4 $^{C}$ | 73.7±14.5 $^{C}$ |
| Vitamin C 200 | 71.53±11.10 $^{C}$ | 70.06±11.5 $^{C}$ | 70.82±11.7 $^{C}$ |
| Vitamin C100 + Lead | 81.71±17.8 $^{B}$ | 83.11±22.3 $^{B}$ | 81.33±17.2 $^{B}$ |
| Vitamin C200 + Lead | 79.92±14.5 $^{B}$ | 77.26±16.7 $^{B}$ | 80.06±13.7 $^{B}$ |
| Lead | 92.19±22.8 $^{A}$ | 91.02±21.4 $^{A}$ | 92.74±23.3 $^{A}$ |

*Means in columns with different superscripts are different at (P≤0.05)*

**Table 8:** Means ±SE for the effect of treatments on serum triglyceride (mg/dl).

| Treatments | Week 4 | Week 8 | Week 12 |
|---|---|---|---|
| Control | 80.03±13.4 $^{B}$ | 78.90±16.7 $^{B}$ | 79.61±17.6 $^{B}$ |
| Vitamin C 100 | 72.69±12.6 $^{C}$ | 70.52±11.4 $^{C}$ | 73.7±14.5 $^{C}$ |
| Vitamin C 200 | 71.53±11.10 $^{C}$ | 70.06±11.5 $^{C}$ | 70.82±11.7 $^{C}$ |
| Vitamin C100 + Lead | 81.71±17.8 $^{B}$ | 79.11±22.3 $^{B}$ | 81.33±17.2 $^{B}$ |
| Vitamin C200 + Lead | 79.92±14.5 $^{B}$ | 77.26±16.7 $^{B}$ | 80.06±13.7 $^{B}$ |
| Lead | 92.19±22.8 $^{A}$ | 91.02±21.4 $^{A}$ | 92.74±23.3 $^{A}$ |

*Means in columns with different superscripts are different at (P≤0.05).*

Table (9) indicates that treatment of rats with lead acetate for 4, 8 and 12 weeks significantly increased serum urea compared with the control group. While treatment of rats with vitamin C (100 or 200mg/kg diet) didn't show any significant effect on serum urea level compared with the control group. Treatment of rats with vitamin C (100 or 200mg/kg diet) plus lead succeeded to decrease the elevation of serum urea to reach the normal range in the control group.

The result revealed that exposure of rats to lead acetate significantly increased serum urea level. These effects indicate that lead may have a toxic effect on kidney structure and / or function. Hesham and El-Sharkawy (2009) showed significant higher levels of blood urea in lead treated mice compared to the control mice. Mohamed, (2010) showed that treatment of mice with lead acetate significantly increased blood urea. Badr and

Abd-Elhamid, (2011) showed that oral administration of lead acetate or aluminum chloride for 30 or60 days significantly increased plasma urea levels.

The results also show that treatment of rats with lead plus vitamin C (100 or 200mg/kg diet) decreased the elevation of serum urea, due to lead acetate, to reach the normal level of the control group. These results may due to the antioxidant effect of vitamin C. Adeneye and Olagunju, (2009) revealed that vitamin C, as antioxidant agent, may inhibited the chain reaction of acetaminophen-generated free radicals or scavenged the reactive oxygen species before reaching its renal targets. They suggested that vitamin C ameliorating effects likely mediate via inhibition of free radicals generation and/or free radical scavenging activity.

**Table 9:** Means ±SE for the effect of treatments on serum urea (mg/dl).

| Treatments | Week 4 | Week 8 | Week 12 |
|---|---|---|---|
| Control | 34.77±4.50 $^{B}$ | 35.85±8.39 $^{B}$ | 37.53±1.82 $^{B}$ |
| Vitamin C 100 | 37.50±4.64 $^{B}$ | 33.64±8.04 $^{B}$ | 39.79±6.83 $^{B}$ |
| Vitamin C 200 | 41.38±5.25 $^{B}$ | 36.05±9.83 $^{B}$ | 39.00±9.60 $^{B}$ |
| Vitamin C100 + Lead | 41.12±5.38 $^{B}$ | 37.96±2.81 $^{B}$ | 35.17±7.45 $^{B}$ |
| Vitamin C200 + Lead | 39.04±4.86 $^{B}$ | 39.20±4.35 $^{B}$ | 37.63±6.78 $^{B}$ |
| Lead | 66.06±10.80 $^{A}$ | 65.27±5.51 $^{A}$ | 64.88±4.82 $^{A}$ |

*Means in columns with different superscripts are different at (P≤0.05)*

Tables 10 and 11 show that treatment of rats with lead acetate for 4, 8 and 12 weeks significantly decreased hemoglobin and red blood cells (RBCs) compared with the control group. While treatment of rats with vitamin C (100 or 200mg/kg diet) for 4, 8 and 12 weeks didn't show any significant effect on hemoglobin and RBCs. Treatment of rats with lead acetate plus vitamin C (100 or 200mg/kg diet) succeeded to increase blood hemoglobin and RBCs to reach the normal levels of the control group.

These results indicate that exposure of rats to lead acetate significantly decreased blood hemoglobin and RBCs. These results may be due to that lead might decreased hemoglobin synthesis thus interfered with heme biosynthesis. Hans *et al.* (1999) described that lead accumulates in erythrocyte interacts with different stages of hemoglobin synthesis and inhibits ferrochelatase activity. This enzyme catalyzes the incorporation of iron into the pryphyrin ring to form heme. Its inhabitation contributes to the development of anemia thatlead to increase proroporphyrine concentration in the erythrocyte. Ashour, *et al.* (2007) reported that lead may induce anemia by both interfering with heme biosynthesis and diminishing RBC survival.

**Table 10:** Means ±SE for the effect of treatments on RBCs ($\times 10^6$ cell/µl).

| Treatments | Week 4 | Week 8 | Week 12 |
|---|---|---|---|
| Control | 8.88±0.12 $^{A}$ | 9.11±0.66 $^{A}$ | 8.94±0.25 $^{A}$ |
| Vitamin C 100 | 8.83±0.01 $^{A}$ | 9.44±0.18 $^{A}$ | 8.95±0.13 $^{A}$ |
| Vitamin C 200 | 8.97±0.32 $^{A}$ | 9.34±0.39 $^{A}$ | 9.22±0.42 $^{A}$ |
| Vitamin C100 + Lead | 8.95±0.12 $^{A}$ | 9.03±0.52 $^{A}$ | 8.92±0.20 $^{A}$ |
| Vitamin C200 + Lead | 8.99±0.17 $^{A}$ | 9.42±0.48 $^{A}$ | 9.32±0.44 $^{A}$ |
| Lead | 8.11±0.20 $^{B}$ | 8.15±0.21 $^{B}$ | 8.00±0.23 $^{B}$ |

*Means in columns with different superscripts are different at (P≤0.05).*

**Table 11:** Means ±SE for the effect of treatments on hemoglobin (g/dl).

| Treatments | Week 4 | Week 8 | Week 12 |
|---|---|---|---|
| Control | 14.97±0.33 $^{A}$ | 14.63±0.52 $^{A}$ | 14.20±0.46 $^{A}$ |
| Vitamin C 100 | 14.80±0.17 $^{A}$ | 15.05±0.26 $^{A}$ | 14.00±0.91 $^{A}$ |
| Vitamin C 200 | 15.23±0.52 $^{A}$ | 15.10±0.15 $^{A}$ | 15.23±0.32 $^{A}$ |
| Vitamin C100 + Lead | 14.87±0.32 $^{A}$ | 15.10±0.53 $^{A}$ | 14.09±0.06 $^{A}$ |
| Vitamin C200 + Lead | 15.40±0.06 $^{A}$ | 15.43±0.98 $^{A}$ | 15.06±0.14 $^{A}$ |
| Lead | 12.90±0.21 $^{B}$ | 12.63±0.13 $^{B}$ | 12.20±0.23 $^{B}$ |

*Means in columns with different superscripts are different at (P≤0.05).*

The results also show that treatment of rats with lead plus vitamin C significantly stopped the decrease in blood hemoglobin and RBCs that accompanied exposure of rats to lead alone to reach the normal range in the control group. This result indicate that supplementation with vitamin C successfully prevented the harmful effect of lead acetate on blood hemoglobin and RBCs. Jacques-Silva, *et al.* (2001*)*suggested that vitamin C (1m/kg) in mice might have a protective role on organo-diselenide intoxication.

Accordingly, the present results show that vitamin C can ameliorate the toxic effect of lead on rats by maintaining the disturbance in serum, liver and kidney functions and lipid profile either up or down near the normal levels of the normal rats.

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