International Journal of Science and Research (IJSR)
ISSN (Online): 2319-7064
Index Copernicus Value (2013): 6.14 | Impact Factor (2013): 4.438

Protective Effects of Sesame Oil against Lead Acetate Induced Haemato-Biochemical Toxicity in Albino Mice

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Abstract: ***Lead is a heavy metal that has been known for its adverse effects on many body organs and systems. Lead toxicity is associated with a number of morphological, biochemical and physiological changes, including hematological disorders, disturbance of glucose metabolism and kidney dysfunction. The aim of this study was to investigate the possible protective role of sesame oil against lead acetate induced haemato-biochemical toxicity in albino mice from the haematological and biochemical aspects. In this study, thirty two adult male albino mice were used for this study and divided into four groups. The first group was control group, the 2nd was the sesame oil group orally received sesame oil (5 ml/kg body wt.), the 3rd was the experimental and received lead acetate (500 mg /kg diet), the 4th one co-administered lead acetate (500 mg/kg diet) with sesame oil (5 ml/kg body wt.) daily for 30 days. Blood samples were obtained for assessment of haematological (Red blood cell count (RBCs), hemoglobin content (Hb), haematocrit (HCT), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), white blood cell count (WBCs), differential leucocytic count, and platelets count) and serum biochemical (glucose, urea, uric acid, and creatinine) parameters. In lead treated animals, there were severe haemato-biochemical changes. Haematologically, mice that received lead acetate (500 mg/kg diet) daily for 30 days had significantly ($p<0.05$) lower RBCs count, Hb, Ht, MCHC, platelets count, lymphocyte count and basophils count than those in the control mice. On the other hand, MCV, MCH, WBCs, neutrophils, monocytes and eosinophils counts of lead acetate treated mice were significantly ($p<0.05$) elevated as compared to the control mice. Biochemically, the serum glucose, urea, uric acid, and creatinine parameters were increased. Co-administration of sesame oil significantly improved the haematological parameters changes and also the serum glucose, urea, uric acid, and creatinine were significantly declined. It can be concluded that, the lead had adverse effects on haemato-biochemical parameters. Sesame oil showed effective protective action against lead acetate induced haemato-biochemical toxicity in albino mice. So, the populations of high risk to lead should be advised to take sesame oil. Further studies are necessary to elucidate exact mechanism of protection of haemato-biochemical toxicity and potential usefulness of sesame oil as a protective agent against heavy metals toxicity in clinical trials.***

Keywords: Lead acetate, Haematotoxicity, Serum glucose, Kidney function , Sesame oil.

1. Introduction

Heavy metals are trace metals that are at least five times denser than water and are taken into body via inhalation, ingestion and skin absorption. It should be noted that most of the pathological conditions in body arise as a result of the exposure to these injurious substances [1]. Lead is a heavy metal that has been known for its adverse effects on many body organs and systems and thus their functions [2]. Lead is a natural stable element and is bioaccumulative in nature. It is an environmental poison of significance to the grazing livestock and a potential public health hazard, as it is excreted in milk [3]. The exposure to lead possesses the potentials to induce hazardous biological effects in both animals and human beings [4]. It is one of the environmental pollutants of high risk to public health [5]. Lead toxicity is a world wide health problem due to continuous exposure of the population to lead in the environment especially workers in industries [6]. The exposure to lead can occur from a multitude of sources such as soil, air, water and industrial pollutants. It has been used in medicines, paintings, pipes, ammunition and in more recent times in alloys for welding storage materials for chemical reagents [7]. Lead is a protoplasmic poison which can cause damages in many organic bodies. There is little doubt that the nervous system and kidney especially the tubules are affected directly [8]. Lead interferes with a variety of body processes and is toxic to the body systems including the cardiovascular, reproductive, haemato-poietic, gastrointestinal, and nervous systems [9]. It may affect numerous organ systems and is associated with various forms of cancer [10], hypertension, neurodegenerative disease, cognitive impairment [11], a number of morphological, biochemical and physiological changes, including kidney dysfunction [12], disturbance of glucose metabolism and hematological disorders [13 & 14]. The absorbed lead enters the blood stream where over 90 percent of it is bound to the red cells with a biological half life of 25-28 days [15[. Exposure to lead can result in significant effects in multiple organs, with the hematopoietic system being an important target [16]. Lead acetate severely affects the morphology and distribution of various blood cells [4]. Lead can cause damage in the erythrocyte, originating defective cells, preventing them from carrying oxygen and it also produces high blood pressure that increases the risk of

heart attack [17]. The prime targets to lead toxicity are the heme synthesis enzymes, thiol-containing antioxidants and enzymes (superoxide dismutase, catalase, glutathione peroxidase, glucose 6-phosphate dehydrogenase and antioxidant molecules like GSH) and it is observed that the low blood lead levels are also sufficient to inhibit the activity of these enzymes and induce generation of reactive oxygen species and intensification oxidative stress [4]. Toxicities due to lead exposure have been attributed to the ability of lead to induce oxidative stress through the generation of reactive oxygen species [18]. The alterations in hematological changes serve as the earliest indicator of toxic effects on tissue [19].

Sesame (*Sesamum indicum* L.) is one of the most important oil seed crops, having seeds and its edible oil that are highly valued as a traditional healthy food ingredient [20]. Sesame oil comprises approximately 50% of the seed weight, contains large amounts of natural antioxidants, they also contain a good type of monounsaturated and polyunsaturated fatty acids [21] and vitamin E [22]. It has been found to contain considerable amounts of the sesame lignans: sesamin, episesamin, and sesamolin. The lignans present in sesame oil are thought to be responsible for many of its unique chemical and physiological properties, including its antioxidant properties [22]. It is well known for its multiple health benefits, including hypocholesterolemic, anti-hypertensive, anti-carcinogenic, anti-aging, immuno-regulatory, hypoglycemic, anti-thrombotic, hepatoprotective [23-25], anti-bacterial, anti-viral, anti-fungal and anti-inflammatory [26].

Plant seeds and herbs are used for treatments of diseases in the folk medicine. Their use was increased in many fields due to their safetyness and it's low side effects as compared with chemical drugs [27]. Plant extracts and materials of animal origin were observed to protect against lead induced toxicity in experimental animals. The abilities of these extracts to mitigate these toxicities were attributed to the antioxidant properties of principles contained in these extracts[18 & 25]. Antioxidant potential of the sesame oil in the amelioration of metal induced oxidative stress need thorough investigation because these natural antioxidants are components of many edible substances and has the potential for safe future use by humans. The evidence reporting the protective effect of sesame oil against lead induced haemato-biochemical toxicity are hardly found. So, the present work aimed to evaluate effectiveness of sesame oil against the haemato-biochemical alterations of lead induced toxicity in albino mice.

2. Materials and Methods

2.1. Chemicals

Lead acetate and sesame oil were purchased from Sigma Chemical Co., USA. Lead acetate was given in diet as 500 mg/kg diet daily [28] for 30 days. Sesame oil was given orally by gavages at a volume of 5 ml/kg body weight according to the previous study of Hussien *et al.* [29]. The choice of the dose of sesame oil was based on the results of the previous studies, where the antioxidant effect of this agent was confirmed.

2.2. Animals

Thirty two adult male albino mice (*Mus musculus*) weighting 25-30 g were used for this study. The animals were obtained from animal house unit in the Faculty of Pharmacy, Tripoli University, Libya. The animals were housed in plastic cages measuring about (29×15×12) cm, with about four mice per cage. Floors of cages were covered with soft crushed wood shaving; all cages were washed two times per week with 70% alcohol throughout the period of the study. The animals were provided with tape water *ad libitum* and fed with the standard commercial chow. The animals were kept in the animal house of Faculty of Science , Alejelat, Zawia University in an air conditioned room with an optimum temperature of 25±2 °C, humidity (60-70%) and light/dark condition (12/12). The animal procedures were performed in accordance with Guide Lines for Ethical Conduct in the Care and Use of Animals.

2.3. Experimental Design

After one week of acclimation, the animals were randomized and divided into four groups (8 albino mice for each) as follow:

Group I (control group): provided with tape water and fed with normal diet.

Group II (sesame oil group): The animals received sesame oil (5 ml/kg body wt/day) orally by gavage daily for 30 days.

Group III (lead acetate treated group): The animals received 500 mg lead acetate/kg diet daily for 30 days.

Group IV (lead acetate/sesame oil co-administered): The animals received 500 mg lead acetate/kg diet concurrently with sesame oil (5 ml/kg body wt/day) orally by gavage daily for 30 days.

At the end of the experimentation and 24 hours after the last dose, all animals were sacrificed under light ether anesthesia, then rapidly dissected and subjected to the following examinations:

2.4. Blood sampling

Blood samples were drown by cardiac puncture. The first sample was collected in clean dry tube containing the anticoagulant substance EDTA (ethylene diamine tetra acetic acid) and used for the hematological studies. The second sample was collected in clean dry tube and centrifuged at 3000 rpm for 15 minutes then, serum was separated and kept in a deep freezer at -20◦C until biochemical measurements were carried out.

2.4.1. Haematological parameters:

Red, white blood cells and blood platelets counts (RBCs, WBCs & platelets) were done by using the hemocytometer and hemoglobin content (Hb) was determined according to the method of Wong [30]. Hematocrite value (PCV) was estimated by using the heparinized capillary tubes. The mean

corpuscular volume (MCV), the mean corpuscular hemoglobin (MCH) and the mean corpuscular hemoglobin concentration (MCHC) were calculated according to Schalm [31] as the following equations: MCV = PCV / RBC's × 10, MCH = Hb / RBC's × 10 & MCHC = Hb / PCV × 100. Differential leucocytic count was done by using light microscope according to the method described by Dacie and Lewis [32].

2.4.2. Biochemical parameters:

Serum glucose was determined using Trinder method [33]. Serum urea measurement was based upon the cleavage of urea with urease [34]. Serum uric acid was determined [35]. Serum creatinine was measured without protein precipitation [36].

2.5. Statistical Analysis

The values were presented as means ± SD of different groups. Differences between the mean values were estimated using one way ANOVA. The results were considered statistically significant when $p < 0.05$.

3. Results

3.1. Haematological parameters:

Haematological parameters in blood of the different groups are shown in Table 1. Mice that received lead acetate (500 mg/kg diet) daily for 30 days had significantly ($p<0.05$) lower RBCs count, Hb, Ht, MCHC, platelets count, lymphocyte count and basophils count than those in the control mice (Fig. 1, 2, 3, 6,7, 10 &13).

On the other hand, MCV, MCH, WBCs, neutrophils, monocytes and eosinophils counts of lead acetate treated mice were significantly ($p<0.05$) elevated as compared to the control mice (Fig. 4, 5, 8, 9, 11 &12). Co-administration of lead acetate with sesame oil were significantly ($p<0.05$) prevented the changes recorded in blood parameters as compared with control group.

3.2. Biochemical parameters

Table 2 showed the means and standard deviations for serum glucose, urea, creatinine and uric acid concentrations in control group, sesame oil group, lead acetate treated group and albino mice group co-administrated of lead acetate with sesame oil.

The levels of serum glucose, urea, creatinine , and uric acid concentrations were elevated in lead acetate treated animals compared with the control group with statistically significant differences ($p<0.05$). The levels of serum glucose, urea, creatinine, and uric acid concentrations in the co-administration of lead acetate with sesame oil were decreased with statistically significant differences ($p<0.05$), when compared with lead acetate group (Figs. 14,15, 16 & 17).

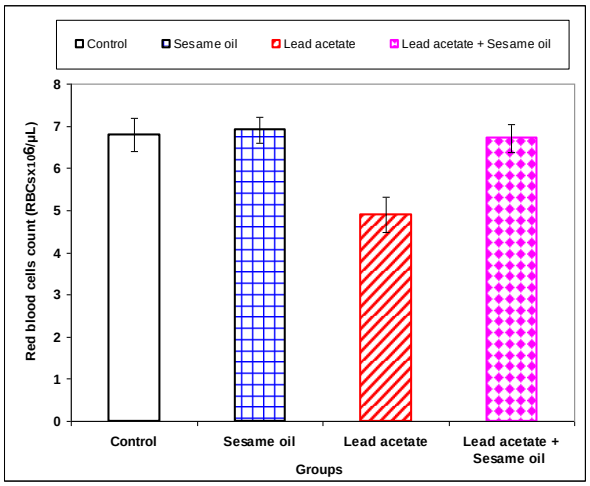

Figure 1: Red blood cells count in different animals groups.

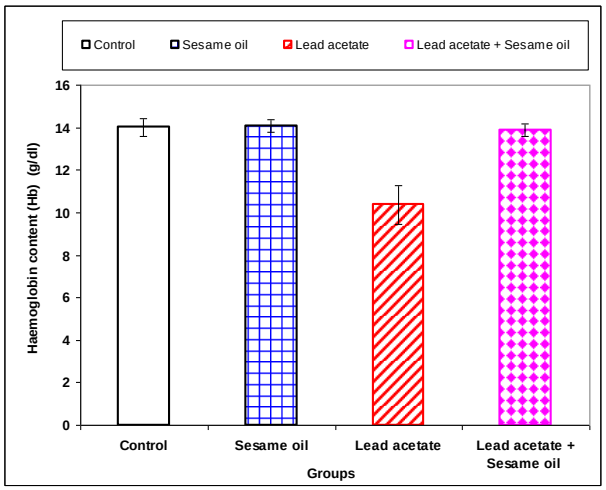

Figure 2: Haemoglobin content in different animals groups.

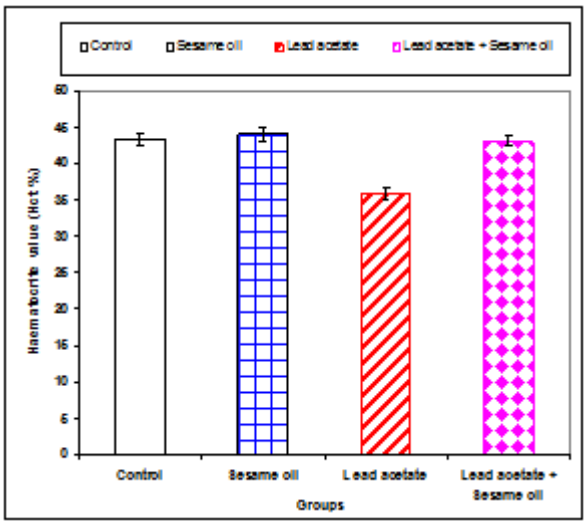

Figure 3: Haematocrite value in different animals groups.

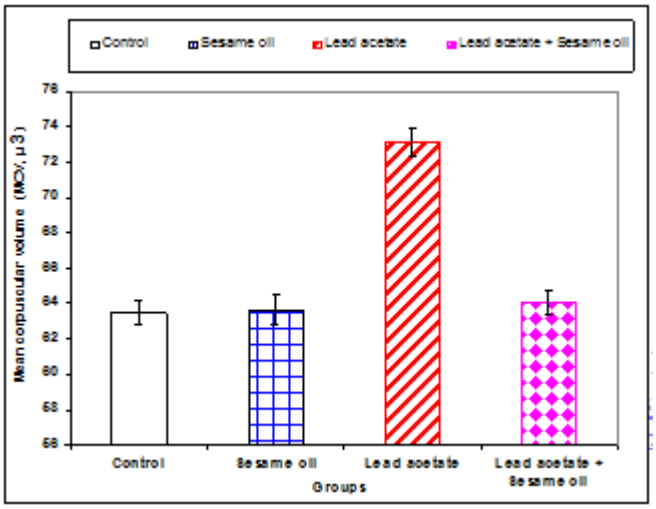

Figure 4: Mean corpuscular volume in different animals groups.

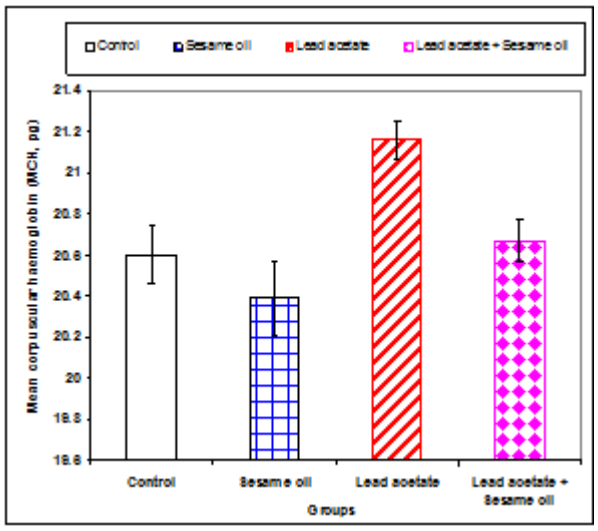

Figure 5: Mean corpuscular haemoglobin in different animals groups.

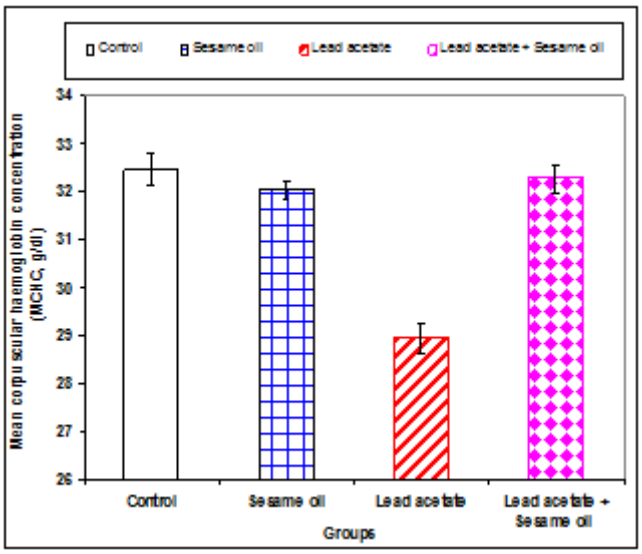

Figure 6: Mean corpuscular haemoglobin concentration in different animals groups.

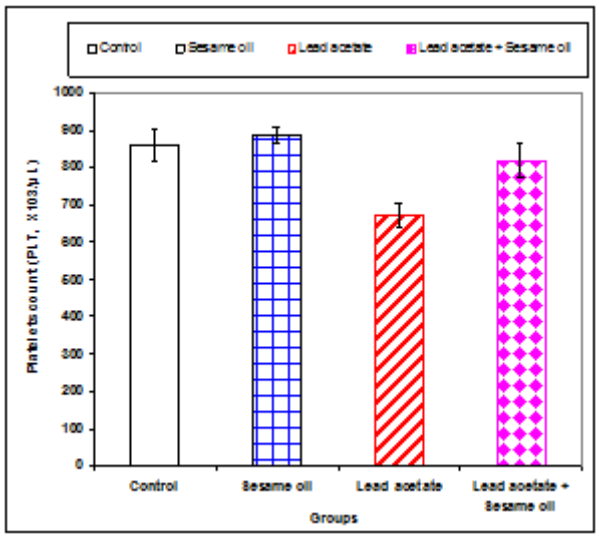

Figure 7: Platelets count in different animals groups.

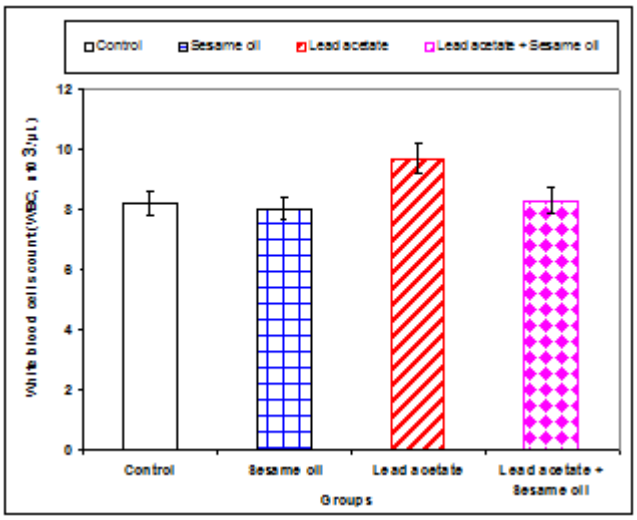

Figure 8: White blood cells count in different animals groups.

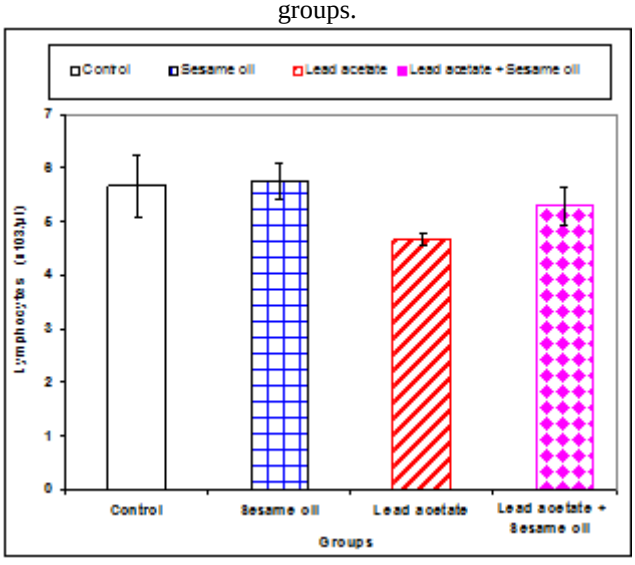

Figure 9: Lymphocytes count in different animals groups.

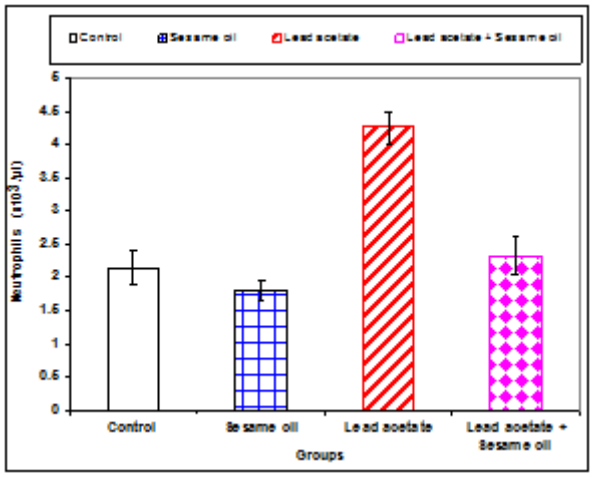

Figure 10: Neutrophils count in different animals groups.

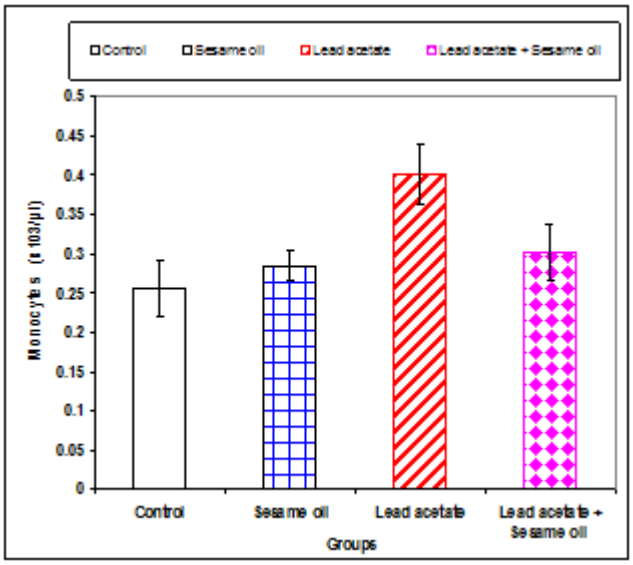

Figure 11: Monocytes count in different animals groups.

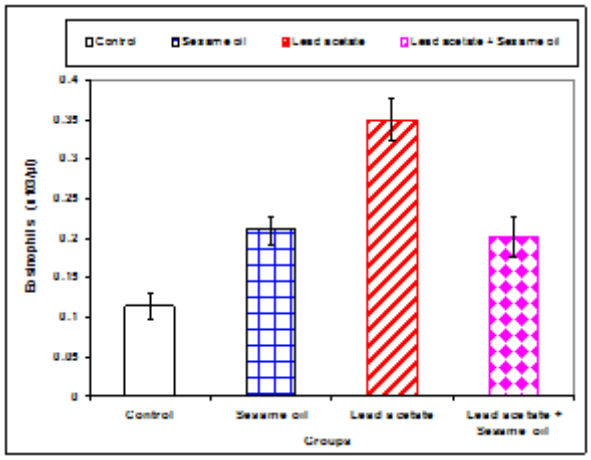

Figure 12: Eosinophils count in different animals groups.

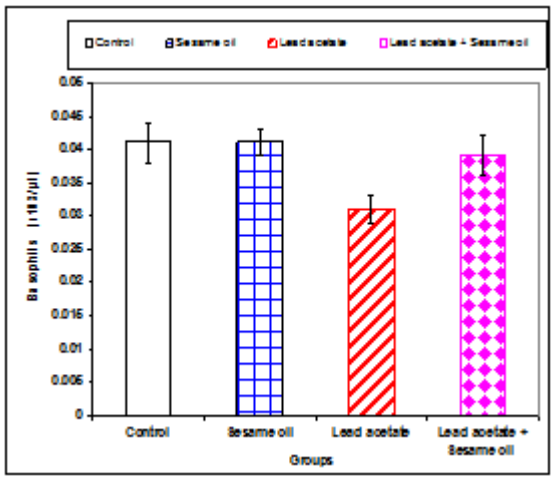

Figure 13: Basophils count in different animals groups.

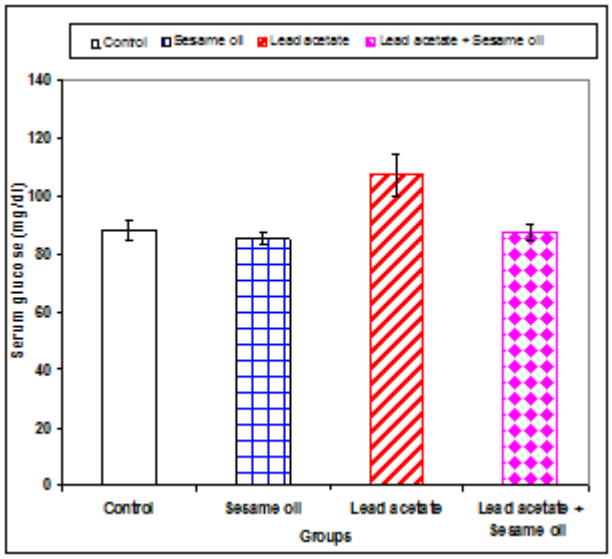

Figure 14: The serum glucose concentration in different animals groups.

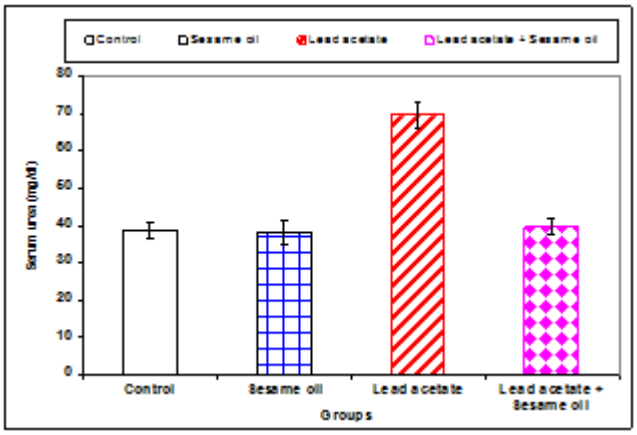

Figure 15: The serum urea concentration in different animals groups. groups.

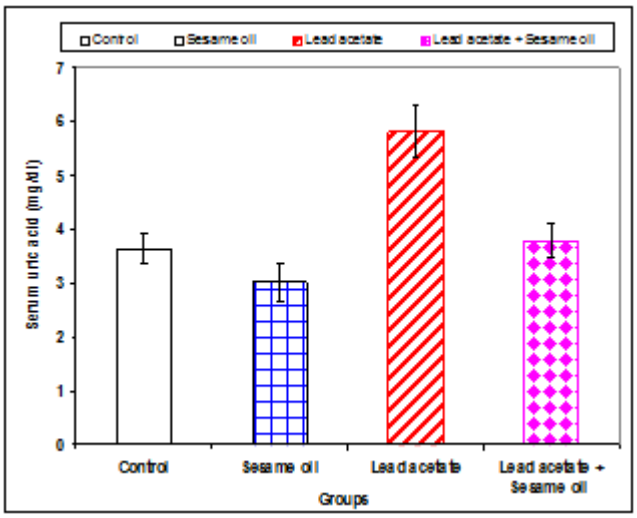

Figure 16 (a): The serum uric acid concentration in different animals groups.

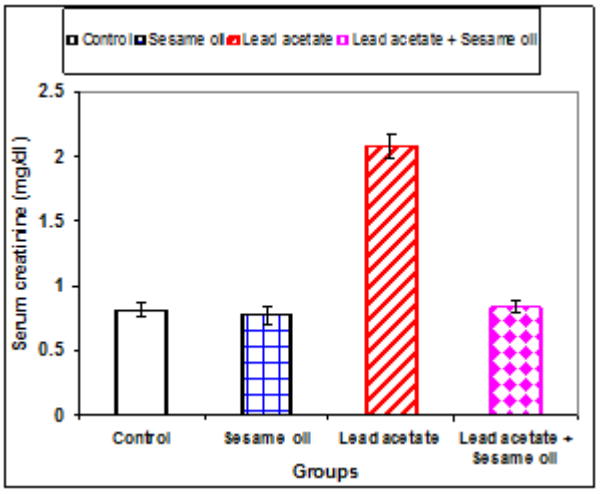

Figure 16 (b): The serum creatinine concentration in different animals groups.

Table 1: Effect of sesame oil on the haematological parameters of lead acetate treated male albino mice in different groups.

Parameters	Groups			
	Control	Sesame oil	Lead acetate	Lead acetate + Sesame oil
	Mean + SD	Mean + SD	Mean + SD	Mean + SD
RBCs ($x10^6$/ µl)	6.80 ±0.39	6.91 ±0.31	4.9 ±0.42[a]	6.71 ±0.34[b]
Hb (g/dl)	14.01 ±0.41	14.09 ±0.31	10.37 ±0.91[a]	13.87 ±0.29[b]
Hct (%)	43.16 ±0.80	43.99 ±0.90	35.82 ±0.89[a]	42.98 ±0.73[b]
MCV (μ^3)	63.47 ±0.65	63.66 ±0.90	73.10 ±0.80[a]	64.05 ±0.71[b]
MCH (pg)	20.6 ±0.14	20.39 ±0.18	21.16 ±0.09[a]	20.67 ±0.10[b]
MCHC (g/dl)	32.46 ±0.33	32.03 ±0.19	28.95 ±0.31[a]	32.27 ±0.28[b]
PLT ($X10^3$/µL)	861 ±43.60	886 ±22.10	670.4 ±32.80[a]	819.8 ±45.70[b]
WBCs ($x10^3$/µl)	8.23 ±0.42	8.03 ±0.35	9.71 ±0.47[a]	8.32 ± 0.41[b]
Lymphocytes ($x10^3$/µl)	5.68 ±0.57	5.76 ±0.36	4.67 ±0.13[a]	5.46 ±0.32[b]
Neutrophils ($x10^3$/µl)	2.14 ±0.26	1.80 ±0.14	4.26 ±0.24[a]	2.32 ±0.28[b]
Monocytes ($x10^3$/µl)	0.255 ±0.037	0.285 ±0.019	0.40 ±0.039[a]	0.301±0.036[b]
Eosinophils ($x10^3$/µl)	0.115 ±0.017	0.21 ± 0.018	0.35 ± 0.027[a]	0.201 ± 0.025[b]
Basophils ($x10^3$/µl)	6.80 ±0.39	6.91 ±0.31	4.9 ±0.42[a]	6.71 ±0.34[b]
Lymphocytes ($x10^3$/µl)	0.041 ± 0.003	0.041 ± 0.002	0.031 ± 0.002a	0.039 ± 0.003b

[a] : Significant differences as compared with control group (P < 0.05) . All data are mean of 8 individuals

[b] : Significant differences as compared with lead acetate treated group (P < 0.05).

Table 2: Effect of sesame oil on the serum glucose, urea, creatinine , and uric acid concentrations of lead acetate treated male albino mice in different groups

Parameters	*Groups*			
	Control	*Sesame oil*	*Lead acetate*	*Lead acetate + Sesame oil*
	Mean + SD	*Mean + SD*	*Mean + SD*	*Mean + SD*
Serum glucose (mg/dl)	88.00±3.38	85.30±2.27	107.15±7.32[a]	87.40±2.68[b]
Serum urea (mg/dl)	38.40 ±2.27	37.90 ±3.29	69.35 ±3.50[a]	39.70 ±2.09[b]
Serum uric acid (mg/dl)	3.65 ±0.28	3.02 ±0.36	5.82 ±0.48[a]	3.79 ±0.31[b]
Serum creatinine (mg/dl)	0.82 ±0.06	0.78 ±0.07	2.08 ±0.09[a]	0.84 ±0.05[b]

[a] : Significant differences as compared with control group ($P < 0.05$) .
[b] : Significant differences as compared with lead acetate treated group ($P < 0.05$).
All data are mean of 8 individuals

4. Discussion

Results of the present study have shown that chronic intoxication with lead acetate induced highly significant (*P*-value < 0.05) reductions in RBCs count Hb, Ht, MCH and MCHC. These results run parallel to those reported by Mylroie *et al*. [37] who recorded a decrease in RBCs count, Hb and Ht in rats treated with (1mg Pb/mL) lead acetate in drinking water for 5 weeks. Also, similar results were recorded by other investigators [2, 38-40]. Also, in rabbits, exposure to lead at different doses in drinking water caused a significant decreases in red blood cell count, hemoglobin concentration and hematocrit value [41].

Recent studies have shown that lead toxicity facilitates conversion of Hb into met-Hb [6]. During Hb oxidation in the presence of lead, H_2O_2 is generated, which may induce lipid peroxidation in the erythrocyte cell membrane [42]. As a result, lead might induce generation of ROS by interacting with oxy-Hb, leading to peroxidative damage of erythrocyte membranes [43].

Lead may interfere in heme biosynthesis apparently characterized by several enzyme blockades and exerts the negative effect on hematopoietic system and give rise to abnormal blood cells [4, 44 & 45]. Toxic materials directly or indirectly damage the membrane structure, ion permeability and cell metabolism of erythrocytes thus may cause morphologically damaged erythrocyte formation [46]. Erythrocyte Na-K-ATPase is somewhat inhibited by lead suggesting a loss of cell membrane integrity, this may account for the shortened lifespan of erythrocytes, lead can also cause damage in the erythrocytes originating defective cells that are eliminated by spleen and their hemolysis [47].

Lead exposure causes hemoglobin oxidation, which can also cause RBC hemolysis. The possible mechanism responsible for this reaction is lead-induced inhibition the activity of aminolevulinic acid dehydratase (ALAD). ALAD is the enzyme most sensitive to lead's toxic effects depressed heme formation [48]. Failure of normal functioning of ALAD to convert two molecules of ALA into prophobilinogen decreases heme formation. This in turn stimulates ALA synthetase, the first enzyme of heme biosynthesis by negative feedback inhibition. As a result of this, there is an increased accumulation of ALA and decreased formation of porphobilinogen [49]. Also, Shah *et al*. [50] reported that lead causes anemia by impairment of heme synthesis and an increased rate of red blood cell destruction. One reason is that the enzyme ferrochelatase that mediates the insertion of ferrous into protoporphyrin is more sensitive to the effects of Pb with co-existing Fe deficiency. Anemia is one of the early manifestations of lead poisoning, it results from reduction of the life span of circulating erythrocyte as well as by inhibition of the body's ability to make haemoglobin by interfering with several enzymatic steps in heme pathway [4 & 38]. Anemia may result when the cell membranes of RBCs become more fragile as the result of damage to their membrane [51]. The reduction of Hb confirmed the decreases in RBCs which may be attributed to the toxicity of lead acetate. It is in agreement with the elevation of serum bilirubin level by lead ingestion, which could be due to the induction of heme oxygenase [52]. The mechanisms for lead-induced immunotoxicity and hematotoxicity include the effects of lead on cell membranes, DNA and antioxidant defense systems of cells [53]. lead treated groups shows abnormal types of RBCs like macrocytes, microcytes showing hypochromasia, anisocytosis and as a certain types of anemia characterized by poikilocytosis is simply observed [4].

WBCs count was found to be markedly increased in mice treated with lead a cetate. These results are in agreement with those of Mazhar *et al.*, [54] who found that WBCs count was increased in the Nile catfish (*Clarias lazera*) caused by mercury as an environmental pollutant. This increase in the total leukocyte count was anticipated to a state of stress after exposure to mercury. Leucoctosis observed in chronic lead poisoning was probably due to an increase in the number of neutrophils [55].

Lymphopenia, neutrophilia, eosinophilia and monocytosis were recorded after chronic intoxication with lead acetate in mice. These results are in agreement with those of Rosenblatt and Marcus [56] who recorded an increase in eosinophils in lead intoxicated patients. Lead caused a significant decrease

in lymphocytes count and significant increase in neutrophils count [2]. Likewise, Abu-El-Zahab *et al.* [57] recorded a decrease in lymphocytes percentage and an increase in neutrophils, and monocytes percentage in mice and rats treated with 1/10 and 1/5 LD_{50} of water hyacinth extract as a result of severe acute stimuli to the haemopoietic system if exposed to toxic materials. But monocytosis occurred for phagocytic the damaged cells resulted from the toxic effect of water- hyacinth extract on the rats organs.

Results of the present study which have shown that chronic intoxication with lead induced significant elevation in serum glucose in lead acetate treated animals compared with control. These results are in concordant with those of Ibrahim *et al.*, [52] who, report that lead acetate causes a significant increase in blood glucose in male albino rat.

The elevations in blood glucose levels may be due to the increases in the rate of glucose transport from the tissues to blood, glycogenolysis and gluconeogenesis or decreased rate removal of glucose from the blood to tissues[52]. The significant change in blood glucose indicates that lead had adverse effects on the pancreas like findings of Ramirez-Cervantes et al. cited by Saka *et al* [58]. These authors found a significant increase in blood glucose levels in subjects with saturnine and therefore directly attributed to the deleterious effects of lead acetate on the pancreas. On the other hand Missoun *et al.* [59] have found that lead acetate induce a decrease in this parameter.

The main function of the kidneys is to excrete the waste products of metabolism and to regulate the body concentration of water and salt [60]. Kidney of mice exposed to lead acetate resulted in serious changes in the histology and function of this organ. In the present investigation, lead acetate treated mice had significant increasing of serum urea, uric acid and creatinine. These results are also in agreement with those of Khalil-Manesh *et al.* [61] and Mohammed [62]. Oral administration of lead acetate in the diet of mice at concentration 0.5% (W/W) for 1 month induced a significant increase in serum urea and creatinine in comparison with the control group [14]. The effect of chronic lead exposure on kidney function in male and female rats was investigated .Lead acetate was administered orally at the rate of 0.3 and 0.6%. The treatment continued for 15, 30, 45, 60 and 90 days, and the results showed an increase of creatinemia and uremia on the 30th day of the experiment in both sexes [63]. Oral administration of 1000 or 2000 ppm lead acetate in albino rats caused significantly increasing in serum urea, uric acid and creatinine [64].

Serum creatinine level is a useful indicator of a regular filtration in the kidney [65]. In the present study, serum creatinine concentration was increased in mice treated with lead acetate as compared to the control group. The present results have been supported by Khalil- Manesh *et al.* [66], who mentioned that lead acetate increased serum creatinine level as compared to the control group, where rats were intoxicated. Same results have been found by Ashour *et al.* [14], who mentioned that lead acetate increased the level of creatinine in lead acetate treated rats. Similar results have been reported by Suleman *et al.* [67] in broiler chicks treated with lead acetate . The presence of the increased level of urea concentration in the blood suggests the inability of the kidney to excrete these products [68]. This dysfunction is confirmed by the increase in blood creatinine which shows a decrease in excretory power of nephrons and even a tendency to renal failure [58].

Saka *et al*. [58] and Aissi *et al.* [69] were found that a highly significant increase in blood urea and uric acid concentration in rats treated with lead acetate. In calves treated with lead acetate (10 mg/kg body weight as 1% solution given in the morning before allowing any feed) daily for 40 days, a significant elevation of serum urea (day 20 onwards) and serum creatinine (day 30 onwards) were noticed which indicate impaired kidney function due to lead toxicity [70]. The increases in blood urea often reflect a nephropathy characterized by glomerular and tubular lesions [68 & 71].
In the present study, serum uric acid was increased in mice treated with lead acetate . However, same results have been found by Ashour *et al.* [14] in rats, and have also been reported by Dioka *et al*. [72] who mentioned that exposure of human subjects to lead in petrol increased the concentrations of uric acid as compared to unexposed subjects. Also, in broiler chicks, lead acetate affected the level of uric acid. As the dose increased, the uric acid level also increased [67]. The increase in blood uric acid apart from the gout that could result is a corollary of saturnine nephropathy [58 & 71]. The elevation of serum uric acid observed in our study is also a marker of oxidative stress linked to a proliferation of pro-oxidative substances such as reactive oxygen species as asserted in Aissi *et al*. [69], and Valko *et al.* [73]. Majority of heavy metals are known to generate free radical system ROS and /or inhibit major antioxidant enzymes in the biological system. This is one of the factors that are attributed to heavy metal toxicity. The damage is more pronounced in organs like liver and kidneys, as they are rich in mitochondria, which is a source of enzymes of antioxidative phosphorylation [74]. It is therefore concluded that oral admistration of EPD (ethanolic extract oF *Phoenix dactylifera* L.) promotes blood's health and annuls lead induced hematotoxicity [2]. Deab [75] found that a significance increases in the PCV and Hb in male rabbits orally treated with sesame oil(1 ml/ kg) daily for 1,3,6 & 8 weeks. Sesame seeds effects which enhances the blood picture represented by the increase in RBC, Hb and PCV, may be related to sesame lignans which have an antioxidant and health promoting activities [76].

Bhuvaneswari and Krishnakumari [60] found that *Sesamum indicum* ameliorates the renal damage in the diabetic rats after the treatment regimen. *Sesamum indicum* extract treatment significantly decreased the levels of blood urea and serum uric acid and creatinine in diabetic rats, which could be due to the prevention of protein and nucleic acid degradation [60]. These results may be attributed to the antioxidant nature of vitamin E present in sesame which acts as protective agent by breaking the chain reactions of both hydroxyl and peroxyl radicals and by regulating the antioxidative defense enzyme system in the kidney tissues [77].

Among the bioactive components in sesame seeds are IP-6 (Phytate; one of the most powerful antioxidants yet found), lignans, pinoresinol, tocopherols, lecithin, myristic acid and linoleate have been identified as the major antioxidantswhich responsible for the resistance of oxidative deterioration of sesame seeds and oil [78]. The potent antioxidant properties of sesame seed extract mainly are attributed to the presence of lignans which are phytoestrogens [79].

5. Conclusion

From the previous discussion, It can be concluded that, the lead had adverse effects on haemato-biochemical parameters. Sesame oil showed effective protective action against lead acetate induced haemato-biochemical toxicity in albino mice. So, the populations of high risk to lead should be advised to take sesame oil. Further studies are necessary to elucidate exact mechanism of protection of haemato-biochemical toxicity and potential usefulness of sesame oil as a protective agent against heavy metals toxicity in clinical trials.

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