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Research Article

THE PROTECTIVE EFFECT OF ARABIC GUM EXTRACT AGAINST LEAD ACETATE TOXICITY ALBINO RATS

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ABSTRACT

Background: Arabic gum is a complex polysaccharide, a mixture of sugars and hemicelluloses composed of Arabic acid nucleus, it is found in nature as slightly acidic calcium, magnesium, potassium or sodium salt and there is different metal ions present in arabic gum molecules. Chemically, it is an arabinogalactan-protein complex composed by weight of 17-34% arabinose, 32-50% galactose, 11-16% rhamnose, 13-19% glucuronic acid and 1.8-2.5% protein. Methodology: lead acetate is a multi-target pollutant, it causes kidney failure and testicular toxicity in rats as it induces oxidative damage and production of reactive oxygen species, Four groups of rats were used in this study, Control, Lead acetate (8 mg/kg body weight intraperitoneally), *Accaciasenegel* (7.5g/kg body weight /day orally) and *Accaciasenegel* (7.5g/kg body weight /day orally) followed by lead acetate (8 mg/kg body weight intraperitoneally) respectively. All groups received the oral treatment by stomach tube once daily for 4 weeks. Lead acetate resulted in an increase in the levels of serum urea, creatinine, uric acid and electrolytes, the testicular catalase activity decreased while the levels of testicular lipid peroxidation & nitric oxide increased. Elevation in the expression of Metallothionein- 1 & 2 genes in the kidney. Severe degeneration of the cortical tubules of kidney, disorganized seminiferous epithelium with depleted number of spermatozoa. *Accaciasenegel* extract administration before lead acetate treatment induced improvement in these changes.

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INTRODUCTION

Arabic gum (AG), is a water-soluble dietary fiber, which is a polysaccharide with branched chains of (1-3) linked β -D-galactopyranosyl units containing α -L-arabinofuranosyl, α -L-rhamnopyranosyl, β -D-glucuronopyranosyl and 4-O-methyl- β -D-glucuronopyranosyl units (Al-Kenanny *et al.*, 2012). AG is rich in Ca^{2+} , K^{+} and Mg^{2+} AG is fabricated from the dried gummy exudates from the stems and branches of the plant *Acacia Senegal*. AG is degraded in the colon by microorganisms into short chain fatty acids (Elshama *et al.*, 2014). It is reported from US Food and Drug Administration that AG according to the AG is one of the safest dietary fibers (Omama, 2013). AG is used in Middle Eastern countries for the treatment of patients with chronic kidney disease and end-stage renal disease. Earlier studies yielded evidence for and against an antioxidant effect of GA as well as protective effects in experimental hepatic-, renal- and cardiac toxicity. Arabic

Gum was reported as a potent superoxide scavenger so that it gives protection in all organs, against the generation of free radicals (Elderbi *et al.*, 2014). AG has been shown to decrease blood pressure, to decrease plasma cholesterol concentrations in rats, to foster dental remineralization, to displays antimicrobial activity and to stimulate intestinal absorption thus counteracting diarrhea.

AG has long been used in traditional medicine in many countries worldwide. AG used as glue and pain-reliever by Egyptians. A wide range of ailments treated by Arabic physicians. Today, it is used widely in the pharmaceutical industry as a demulcent and in the cooking industry to give size and texture to processed food products (Ibrahim *et al.*, 2014). AG produces coloured complexes with certain amines and phenols as it contains peroxidase enzyme and enhances the destruction of many pharmaceutical products including alkaloids and readily oxidizable compounds and also is known

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as a trypsin inhibitor (Ibrahim *et al.*, 2014). AG isn't digested but is fermented in small intestine under the influence of microorganisms in the colon to short chain fatty acids, especially propionic acid. Such degradation products are absorbed in the human colon and subsequently utilized energetically in metabolism (Ibrahim *et al.*, 2014).

Industry is the main cause of environmental poisoning by Lead. The important sources of lead exposure include gasoline additives, lead based paints, ceramic glazes (Bharali, 2013, Khan, *et al.*, 2008 and Ramya & Prasanna, 2013), drinking water system, cosmetics, battery and plastic recycling Industry. Lead entered our body through the skin, gastrointestinal tract or lungs and distributed to three major compartments-blood, soft tissue and bone. Blood lead is in equilibrium with lead in soft tissue (Sujatha *et al.*, 2011). The soft tissues that take up lead like liver, kidneys, brain and muscle. Lead is not metabolized in the body but it is conjugated with glutathione and excreted primarily in the urine. There are many targets for Lead such as gastro intestinal tract, hematopoietic system, cardiovascular system, central and peripheral nervous system, Kidneys, immune system and reproductive system. Lead binds with sulfhydryl proteins, changing the calcium homeostasis and lowers the levels of available sulfhydryl antioxidant reserves in the body. It is known to cause free radical damage in tissues by two mechanisms: Increased generation of reactive oxygen species (ROS), including hydroperoxides, singlet oxygen and hydrogen peroxides, and by causing direct depletion of antioxidant reserves (Shimaa El-Masry *et al.*, 2016). Kidneys play a major role in the excretion of lead from the body and the higher content of lead has been estimated in renal tissue than in liver and brain of the lead intoxicated animals (Bharali, 2013).

Metallothionein (MT) is a low-molecular-weight metalbinding protein and is known to play an important role in the protection against heavy metal toxicity. In addition to, the detoxification of toxic metals such as cadmium (Cd) and mercury (Hg), MT is involved in the maintenance of homeostasis of essential trace elements such as zinc (Zn) and copper (Cu), and in the scavenging of free radicals. MT gene expression is induced by many factors, including metal ions such as Cd, Zn, Cu and Hg, alkylating agents, UV irradiation, and other chemical and physical stressors (Yu J.*et al.*, 2009 and Ghoniem *et al.*, 2012).

MATERIALS AND METHODS

Experimental design

Forty male adult Wistar albino rats *Rattus norvegicus* were divided randomly into four groups with ten animals in each group. Group A, fed standard diet and water ad libitum served as control, Group B rats received lead acetate (Merck, Germany) 8 mg/kg body weight intraperitoneally for four weeks. Group C received (7.5 g/kg/day) of extract of Arabic gum orally for (Amani *et al.*, 2013) 4 weeks, group D received (7.5 g/kg/day) of extract of Arabic gum orally for 7 days before administration of lead acetate, then at the 7th day started receiving lead acetate i.p. for 4 weeks.

Preparation of Plant extract

Crude Arabic Gum obtained from the local market as spheroidal tears were milled and sieved to obtain fine pure powder. This powder is water insoluble, it is dissolved in hot

water then it was filtered, then each rat received (7.5 g/kg/day) orally (Amani *et al.*, 2013).

Blood Collection and Tissue Preparation

At the end of experiment animals were sacrificed by cervical dislocation, serum was separated and stored at -20C until analysis of serum chloride (Schonfeld and Lewellen, 1964), sodium, calcium (Bauer, 1981), phosphorous (Tietz, 2005), urea (Tietz N.W., 1990) creatinine (Bowers, 1980) and uric acid (Fossati, and Prencipe, 1980). Samples from kidney divided into 2 parts; one was taken for RT-PCR analysis, while the other for histopathological analysis. While samples from testis was taken and divided into 2 parts; one for detection of oxidative stress in testis while the other part stored at formalin for histopathological analysis. The testes were quickly isolated, washed with saline, blotted dry on filter paper, and weighed, and 10% of homogenate prepared (% weight per volume [w/v]) 10%w/v) using a homogenizer (Potter-Elvehjem) in ice-cold sodium potassium phosphate buffer (0.01M, pH 7.4) containing 1.15% of KCl. The homogenates were centrifuged at 10,000 ×g for 20mins at 4°C and the supernatant was used for assaying the catalase activity, end product of lipid peroxidation (Ohkawa *et al.*, 1979), MDA and NO level in testis tissues was measured by using the commercial kits supplied by Biodiagnostic, Egypt. The assay is based on the diazotization of sulfanilic acid with nitric oxide at acidic pH and subsequent coupling with N-(1-naphthyl)-ethylenediamine to yield an intensely pink colored product that was measured spectrophotometrically at 540 nm. Sodium nitrite was used as standard. Catalase (CAT) activity was determined according to (Clairborne, 1985) based on decomposition of hydrogen peroxide by catalase enzyme. Specimens of kidney and testis tissues were immediately fixed in 10% formalin, treated with conventional grade of alcohol and xylol, embedded in paraffin, and sectioned at 4–6 μ thickness. The sections were stained with Haematoxylin and Eosin (H&E) stain for studying the histopathological changes of kidney and testis.

Gene expression

Total RNA isolated from testis tissue using total RNA purification kits, then total RNA concentration was determined using Nano drop then cDNA was synthesized from RNA by using Reverse transcriptase Kits. Finally Two-Step RT-PCR was performed by using the amount of change in gene expression was calculated from the obtained cycle threshold (CT) values provided from realtime PCR instrumentation using the 2-ΔΔCT calculation, where ΔCT indicates the CT changes in target genes Mt-1 & Mt-2 in comparison with the reference (house-keeping) gene, which is GAPDH as described by (Livak and Schmittgen, 2001), the primers used sequences of MT-1 with sequence as follow forward primer 5' CAC CGT TGC TCC AGATTCAC-3, reverse primer 5'- GCA GCAGCA CTG TTCGTAC-3 (gene bank accession number RATMETI), MT-2 forward primer 5'- ATC TCC AACTGCCGCTCC-3, reverse primer 5'- TGC ACT TGTCCGAAGCCTCT-3 (gene bank accession number XM_001062488) and GAPDH (internal control) forward primer 5'- CCT TCA TTG ACC TCA ACTACATG-3, reverse primer 5'-CTT CTC C A TGG TGGTGAAGAC-3 (gene bank accession number NM_017008)(Ghoniem, 2012).

Statistical analysis

The data obtained were analyzed by one-way analysis of variance and Student t-test for the significant interrelation between the various groups using (Turner, 2001) GraphPad prism version 5 for windows, GraphPad software (San Diego, CA, USA). Probability levels of more than 0.05 were considered insignificant ($P > 0.05$), while less than 0.05 were considered significant ($P < 0.05$), while less than 0.01 were considered highly significant ($P < 0.01$), less than 0.001 were considered very highly significant ($P < 0.001$).

Table 1 The level of some electrolytes and some Kidney functions of adult male albino rats (*Rattus norvegicus*) treated with Lead acetate, Accaciasenegel & Accaciasenegel- Lead acetate

Groups Parameters	Control (A)	Lead acetate (B)	Arabic gum (C)	Arabic gum + lead acetate (D)
Sodium (mg/dl)	161.4 ± 2.286	187.8 ± 1.881	163.7 ± 2.055	168.38 ± 3.037
Potassium	5.540 ± 0.132	9.024 ± 0.132	5.62 ± 0.1393	6.956 ± 0.245
Chloride (mg/dl)	126.500 ± 1.836	178.6 ± 4.611	126.4 ± 1.72	154.4 ± 3.473
Phosphate (mg/dl)	6.5 ± 0.279	9.28 ± 0.124	7.16 ± 0.4743	8.102 ± 0.110
Calcium (mg/dl)	8.268 ± 0.02871	19.38 ± 0.498	8.364 ± 0.330	12.740 ± 0.476
Creatinine (mg/dl)	0.812 ± 0.04781	2.654 ± 0.04781	0.896 ± 0.06705	1.152 ± 0.02905
Sodium (mg/dl)	35 ± 1.414	69.2 ± 3.967	37.4 ± 1.661	56 ± 2.775
Potassium (mg/dl)	2.62 ± 0.1393	4.362 ± 0.133	2.54 ± 0.1503	3.38 ± 0.128

The results

The mean ±SEM (187.8 ± 1.881) of lead acetate treated group resulted in a highly significant increase in the level of serum Sodium when compared with control group (161.4 ± 2.286), while administration of extract of Arabic gum before lead acetate treatment (168.38 ± 3.037) resulted in a highly significant decline in the level of serum sodium in comparison with data of lead acetate treated group. The mean ±SEM (9.024 ± 0.132) of lead acetate treated group resulted in a highly significant increase in the level of serum Potassium when compared with control group (5.54 ± 0.1327), while mean ±SEM (6.956 ± 0.245) of Arabic gum extract administration before lead acetate resulted in a highly significant decline in the level of serum Potassium when compared with lead acetate treated –group. The mean ±SEM (178.6 ± 4.611) of lead acetate treated group resulted in a highly significant increase in the level of serum Chloride when compared with control group (126.5 ± 1.836), while administration of extract of Arabic gum before lead acetate treatment (154.4 ± 3.473) resulted in a highly significant decline in the level of serum Chloride in comparison with data of lead acetate treated group. The mean ±SEM (9.28 ± 0.124) of lead acetate treated group resulted in a highly significant increase in the level of serum Phosphate when compared with control group (6.5 ± 0.279), while mean ±SEM (7.16 ± 0.474) of Arabic gum extract group did not induce any significant change in the level of serum phosphate when compared with control group. The mean ±SEM of lead acetate treatment (19.38 ± 0.498) revealed a highly significant

increase in the level of serum calcium when compared with control group. On the contrary, the mean ±SEM (12.740 ± 0.476) of Arabic gum extract before administration of lead acetate resulted in a highly significant decline in the level of serum calcium when compared with lead acetate-treated group. The mean ±SEM of lead acetate treatment (2.654 ± 0.04781) revealed a highly significant increase in the level of serum creatinine when compared with control group, while the mean ±SEM (1.152 ± 0.02905) showed that administration of Arabic gum extract before lead acetate administration resulted in a

highly significant decline in the level of serum creatinine when compared with lead acetate-treated group, while the mean ±SEM (0.896 ± 0.0670) showed that administration of Arabic gum extract did not induce any significant change in the level of serum creatinine when compared with control group. The mean ±SEM of lead acetate treatment (69.2 ± 3.967) revealed a highly significant increase in the level of serum urea when compared with control group, while the mean ±SEM (56 ± 2.775) showed that administration of Arabic gum extract before lead acetate administration resulted in a highly significant decline in the level of serum urea when compared with lead acetate-treated group, while the mean ±SEM (37.4 ± 1.661) showed that administration of Arabic gum extract only did not induce any significant change in the level of serum urea when compared with control group. The mean ±SEM of lead acetate treatment (4.362 ± 0.133) revealed a highly significant increase in the level of serum Uric acid when compared with control group, while the mean ±SEM (3.38 ± 0.128) showed that administration of Arabic gum extract before lead acetate administration resulted in a highly significant decline in the level of serum Uric acid when compared with lead acetate-treated group, while the mean ±SEM (2.54 ± 0.150) showed that administration of Arabic gum extract did not induce any significant change in the level of serum Uric acid when compared with control group. The mean ±SEM of lead acetate treatment (112.237 ± 2.313) revealed a highly significant increase in the activity of testis Catalase enzyme when compared with control group, while the mean ±SEM (94.79 ± 2.187) showed that administration of Arabic gum extract before

Table 2 The effect of Lead acetate on some enzyme markers in testis tissue

Groups Parameters	Control (A)	Lead acetate (B)	Arabic gum (C)	Arabic gum + lead acetate (D)
Catalase (U/L)	71.34 ± 1.208	52.237 ± 2.313	71.608 ± 1.312	64.79 ± 2.187
Nitric Oxide (µmol/g tissue)	5.962 ± 0.195	9.276 ± 0.190	6.124 ± 0.195	7.35 ± 0.233
Malondehyde (nmol/g tissue)	4.5 ± 0.136	9.08 ± 0.124	4.06 ± 0.165	6.66 ± 0.209

lead acetate administration resulted in a highly significant decline in the activity of testis Catalase enzyme when compared with lead acetate-treated group, while the mean \pm SEM (71. 608 \pm 1.312) showed that administration of Arabic gum extract did not induce any significant change in the activity of testis catalase activity when compared with control group.

Table 3 The expression level of kidney Metallothioneingenes

Control rats	Control (A)	Lead acetate (B)	Arabic gum (C)	Arabic gum + Lead acetate (D)
Mt-1	0.531 \pm 0.2003	3.974 \pm 0.577	0.666 \pm 0.177	2.179 \pm 0.265
Mt-2	0.826 \pm 0.210	3.634 \pm 0.655	0.831 \pm 0.160	1.929 \pm 0.087

The mean \pm SEM of lead acetate treatment (9.276 \pm 0.190) revealed a highly significant increase in the level of testis Nitric oxide level when compared with control group, while the mean \pm SEM (7.35 \pm 0.233) showed that administration of Arabic gum extract before lead acetate administration resulted in a highly significant decline in the level of testis Nitric oxide when compared with lead acetate-treated group, while the mean \pm SEM (6.124 \pm 0.195) showed that administration of Arabic gum extract did not induce any significant change in the level of testis Nitric oxide when compared with control group.

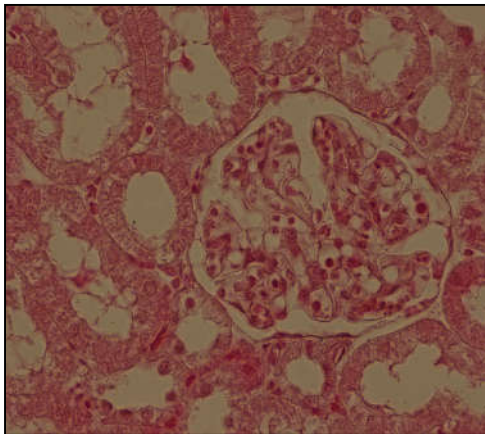


Fig 1 Light photomicrograph of a section in Kidney of cortex of GB X 400, H&E stain.

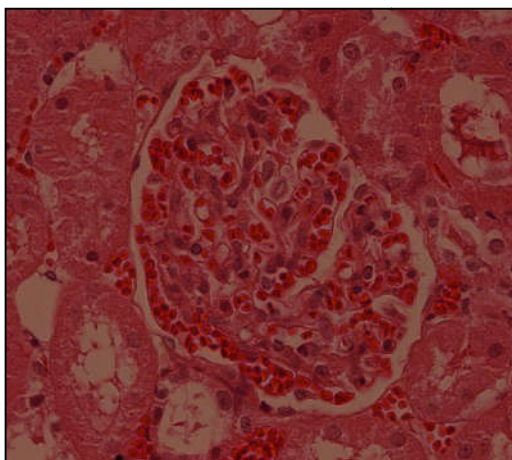


Fig 2 Light photomicrograph of a section in renal showing normal structure of the renal cortex. X 400, H&E stain.

The mean \pm SEM of lead acetate treatment (9.08 \pm 0.124) revealed a highly significant increase in the level of testis Malondehyde level when compared with control group, while the mean \pm SEM (6.66 \pm 0.209) showed that administration of Arabic gum extract before lead acetate administration resulted in a highly significant decline in the level of testis Malondehyde when compared with lead acetate-treated group, while the mean \pm SEM (4.06 \pm 0.165) showed that administration of Arabic gum extract did not induce any significant change in the level of testis Malondehyde when compared with control group.

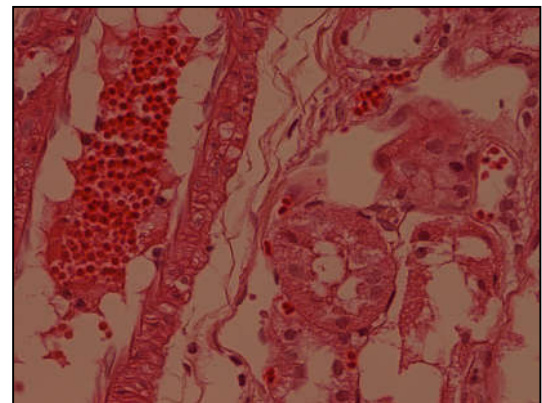


Fig 3 Light photomicrograph of a T.S. i in renal cortex of Group B X 400, H&E stain

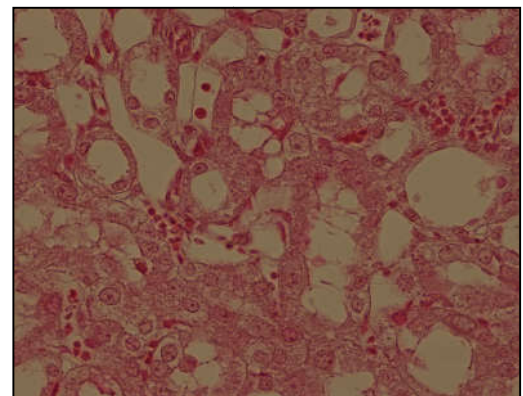


Fig 4 Light photomicrograph of T. s. in renal cortex of group B X 400, stain

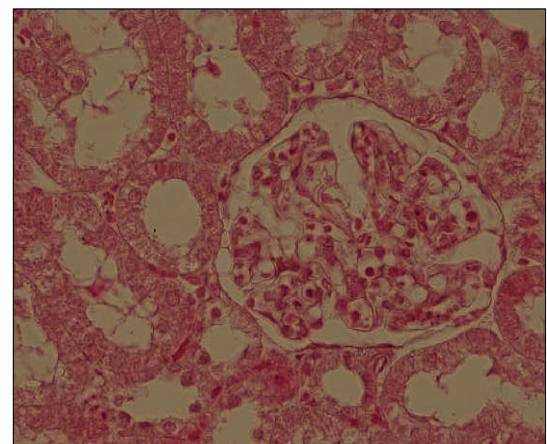


Fig 5 Light photomicrograph of a T.S.in renal cortex of of renal cortex of Group C X400, H&E stain

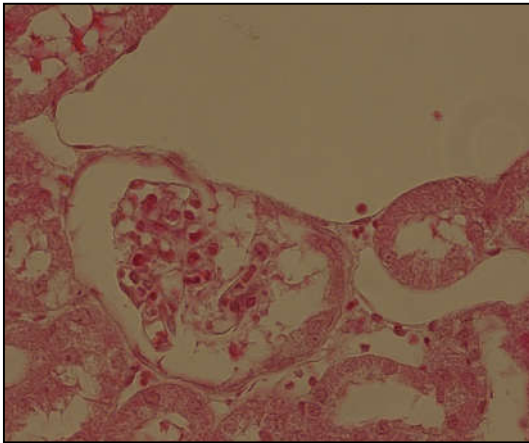


Fig 6 Light photomicrograph of a T.S.in cortex of Group D X400, Hx&E. stain

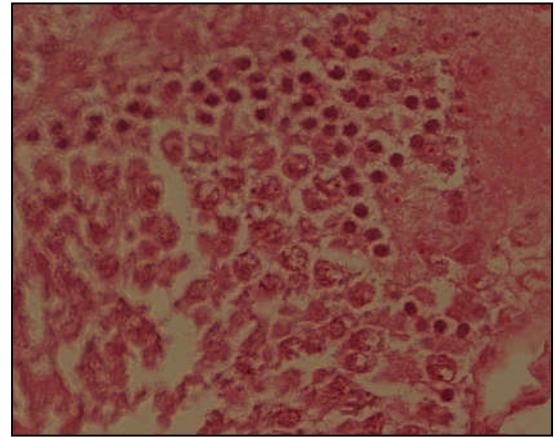


Fig 9 Light micrograph of a section of testis of Group B H&E stain,X200

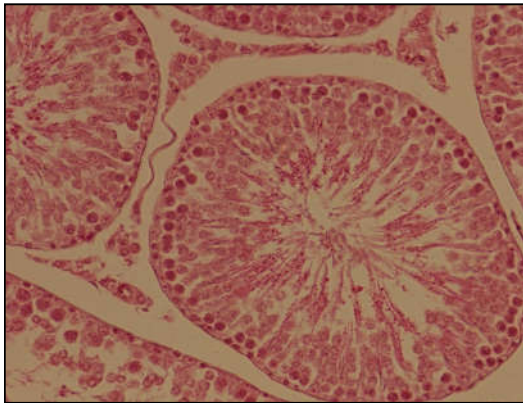


Fig 7 Light micrograph of A section of testis of Group A H&E stain, X200H&E stain, X200.of

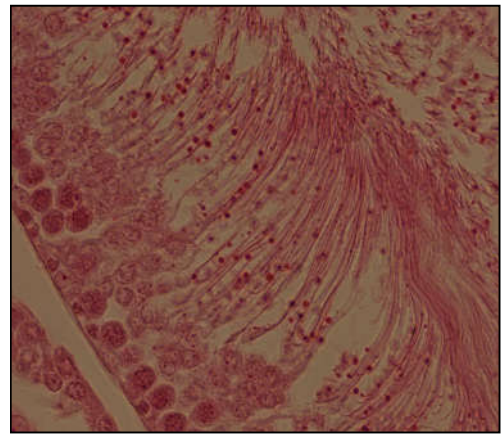


Fig 10 Light micrograph of a section of testis of Group C H&E stain,X200

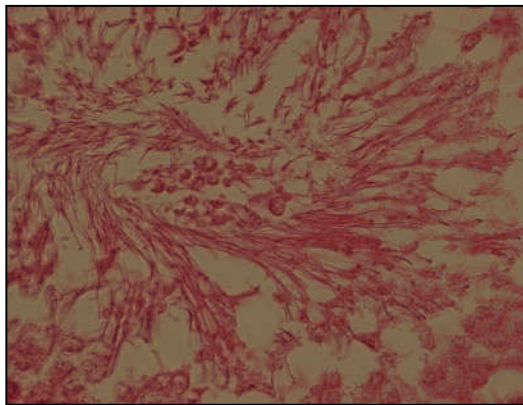


Fig 8 Light micrograph of a section testis of Group B

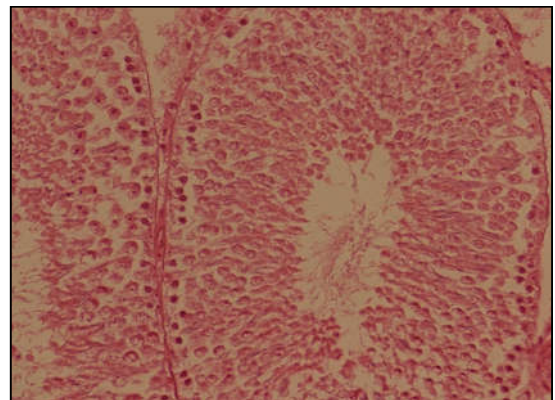


Fig 11 Light micrograph of a section of testis of Group D H&E stain,X200.

The mean \pm SEM of lead acetate treatment ($4.812 \pm 0.0.144$) revealed a highly significant decrease in the level of testis reduced glutathione level when compared with control group, while the mean \pm SEM (6.936 ± 0.070) showed that administration of Arabic gum extract before lead acetate administration resulted in a highly significant improvement in the level of testis reduced glutathione when compared with lead acetate-treated group, while the mean \pm SEM (10.264 ± 0.163) showed that administration of Arabic gum extract did not induce any significant change in the level of testis reduced glutathione when compared with control group.

The mean \pm SEM (3.974 ± 0.577) of lead acetate treatment revealed a significant increase in the level of expression of Mt-1 when compared with control group (0.531 ± 0.2003), while the mean \pm SEM of Arabic gum extract & lead acetate treatment showed a significant decline in expression level of Mt-1 but still higher than that of control group. The mean \pm SEM (3.634 ± 0.655) of lead acetate administration resulted in a significant elevation in the expression level of kidney Mt-2 when compared with control group (0.826 ± 0.210), while the administration of Arabic gum extract before lead acetate treatment resulted in a significant decrease in the level of gene expression when compared with its expression in control and Arabic gum extract -treated groups separately.

Lead acetate treatment resulted in severe degeneration of the cortical tubules. Also the renal corpuscles showed severe congestion of the glomerulus and the peritubular vessels of kidney (fig.2, 3, 4) while administration of extract of Arabic gum alone did not induce any change in the normal structure and pattern of kidney cortex and medulla (fig.5), while the administration of Arabic gum extract before lead acetate treatment shows a slight improvement in the degeneration of kidney (fig.6) when compared with lead acetate- treated kidney (fig.2, 3, 4). The testis of the control group showed the normal structure of testis and normal seminiferous tubule (fig. 7), while lead acetate treatment resulted in disorganized seminiferous epithelium and cells appeared degenerated with pyknotic nuclei (fig.8&9). While administration of extract of Arabic gum before lead acetate treatment showed spermatogonia with pyknotic nuclei and are located away from the basement membrane. The nuclei of other spermatogenic cells show chromatolysis. The number of sperms embedded in Sertoli cells is depleted (fig.11). While the administration of Arabic gum extract did not induce any change in normal structure and pattern of testis and seminiferous tubules (fig.10).

DISCUSSION

Lead (Pb) is a toxic metal that induces a broad range of physiological, biochemical and neurological dysfunctions in humans. The atmospheric Pb pollution due to tetra ethyl lead from gasoline has been improved over the last two decades, humans are still exposed to Pb via contaminated foods, water and through industrial activities and by causing direct depletion of antioxidant reserves (Elgawisha and Abdelrazek, 2014). Lead (Pb) is a well-known multi-organ toxicant and it damages liver and kidney. It is a divalent cation that settles in the proximal tubule of the nephron, leading to nephrotoxicity. The present study declared a significantly elevation in all kidney function tests accompanied by increase in serum sodium and potassium, calcium, chloride and phosphate concentration in lead-treated rats. These results are confirmed with (Odligie *et al.*, 2004; Abd El-Reheem, and Samir, 2007; Ashour, 2007; Zaahkcuk, 2007; Elmenoufy, 2012; Ghoniem, 2012; Ramya and Prasanna, 2013; Taha *et al.*, 2013; Ansar *et al.*, 2016; El Masry, 2016; Salem and Salem, 2016). Lead accumulation in kidney cause damage in renal tubules, decreasing the number of the functional nephrons which weakens the reabsorption process and generate reactive oxygen species which damage the cells leading to apoptosis, these adverse effects on renal function resulted in elevation of nitrogen containing compounds as urea, creatinine and uric acid in the blood. The increased level of blood urea and creatinine concentration in lead-toxicated rats suggests the inability of the kidney to excrete these products causing their increase in blood and decrease their excretion in urine. The increments in uric acid concentrations may be due to degradation of purines or to an increase of uric acid levels by either overproduction or inability of excretion as uric acid is the end product of the catabolism of tissue nucleic acid, i.e. purine and pyrimidine bases metabolism. Chronic exposure to low-levels of lead resulted in electrolyte retention and elevation of sodium and potassium; this is due to lead effects on renal tubular transport mechanisms. Another mechanism of increase sodium and potassium level is the decrease in functioning nephrons that trigger multiple adaptive processes in the hyper functioning remaining nephrons including augmented rates of

electrolyte reabsorption. The proximal tubular cells are particularly vulnerable owing to their high energy demand such as reabsorptive and secretory functions. Lead accumulates in mitochondria and causes both structural and functional alterations. The effects include mitochondrial swelling, inhibition of respiratory functions and energy (ATP) production. Consequently energy dependent processes including tubular transport are impaired (ODIGIE *et al.*, 2004; Abd El-Reheem, and Samir, 2007; ASHOUR 2007; Zaahkcuk, 2007; KHAN, 2008; Elmenoufy, 2012; Ghoniem, 2012; Ramya and Prasanna, 2013; El Masry, 2016; Salem and Salem, 2016).

In the present study lead acetate treatment resulted in an elevation in the level malondehyde and nitric oxide level and a decrease in the activity of catalase in testis tissue these findings are in accordance with those of (Dorostghoal, 2013; Sharma & Bhattacharya, 2015; Ansar *et al.*, 2016) they attributed that to administration of lead, Lead can cross the blood-testis barrier, accumulate in the testis, and damage germinal cells at various levels of differentiation shifted the oxidant/antioxidant profile towards oxidant side as manifested by the marked exhaustion of the enzymatic antioxidants together with the accumulation of lipid peroxidation product in the testicular tissue homogenate. This was in corroboration with earlier reports (Ansar *et al.*, 2016). Lead interrupted redox homeostasis through slow clearance of hydrogen peroxide, stimulation of membrane lipid peroxidation, inhibition of delta-aminolevulinic acid dehydratase and upregulation of production and oxidizing potential of oxidant species (Ansar *et al.*, 2016). The increased oxidative stress was incriminated in playing the main role in the pathogenesis of lead toxicity. Since the membranes of spermatozoa are rich in polyunsaturated fatty acids, they are vulnerable to oxidative stress damage (Ansar *et al.*, 2016). In the present lead acetate resulted in increase in the expression of MT-1& MT-2 genes that is decreased when administration of extract of AG before lead acetate treatment this result is in line with that of that attributed this increase to that Metallothionein (MT) is a low-molecular-weight metal binding protein and is known to play an important role in the protection against heavy metal toxicity. In addition to the detoxification of toxic metals such as cadmium (Cd) and mercury (Hg), MT is involved in the maintenance of homeostasis of essential trace elements such as zinc (Zn) and copper (Cu), and in the scavenging of free radicals. MT gene expression is induced by many factors, including metal ions such as Cd, Zn, Cu and Hg, alkylating agents, UV irradiation, and other chemical and physical stressors. A possible reason for the suppression of MT protein synthesis by Pb is that Pb deprived kidney cells of free Zn ions necessary for the synthesis of Zn-thionein. Pb is known to displace Zn in the active site of δ -aminolevulinic acid dehydratase leading to the depression of the enzyme activity. DNA-binding of Sp127 or GATA-128 *via* Zn-finger motif was also suppressed by Pb treatment (Hasega *et al.*, 2015).

CONCLUSION

Lead acetate is a strong environmental pollutant and multi-target toxin, resulted in elevating serum electrolytes and kidney functions, testis levels of nitric oxide, MDA; while decreasing CAT activity and resulted in increased expression of Mt-1 & Mt-2 genes in kidney, while AG treatment before lead acetate

administration resulted in a modulatory effect for these changes.

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