

Protective and Therapeutic Effects of *Moringa oleifera* Against Toxicity of Lead Chloride

Kadry A. ELbakry¹, Naglaa F. Elarabany¹ and Samaa G. Behery^{1*}

¹Zoology Department, Faculty of Science, Damietta University, New Damietta, Egypt.

Received: 12 May 2017 /Accepted: 24 August 2017

*Corresponding author: samaa.behery@yahoo.com

Abstract

The present study aimed to evaluate the protective and therapeutic effects of *M. oleifera* leaves against toxicity induced by PbCl₂ in albino male rats. Thirty-six albino rats weighing about 182.8 g were divided into 6 groups each of 6 rats. Control group, left without treatment. The PbCl₂ group treated orally with 200 mg PbCl₂ daily for 30 days. The *M. oleifera* group treated orally with 200 mg *M. oleifera* daily for 30 days. PbCl₂ + *M. oleifera* group (200 mg PbCl₂ + 200 mg *M. oleifera*). Therapeutic group (200 mg PbCl₂ followed by 200 mg *M. oleifera*) and protective group (200 mg *M. oleifera* followed by 200 mg PbCl₂). At the end of the experiment, both kidney and liver function tests were assayed as well as catalase and SOD enzymes activity and MDA level. The activity of ALT and the levels of bilirubin, urea, creatinine, cholesterol, ammonia, urea and triglycerides were elevated after lead administration compared with the control and protective groups (p<0.05) but the level of albumin of the same group was decreased compared with the control group. In protective group, the level of MDA was significantly decrease compared with that of the lead group. On the other hand, the activities of SOD and catalase were significantly increase in the protective group as well as other treated groups compared with the control group (p<0.05). In conclusion, treatment with *M. oleifera* leaves can protect liver and kidney against lead toxicity.

Keywords: *M. oleifera*, Lead, Liver, Kidney, Rats.

Introduction

Lead is considered as one of the major environmental pollutants and is amongst four metals that have the most detrimental effects on human (Karrari et al, 2012). It is known to induce a broad range of physiological, biochemical, and behavioural dysfunctions in laboratory animals and humans (Flora et al., 2016), including central and peripheral nervous systems (Bressler et al., 1999), haemopoietic system (Lanphear et al.,

2000), cardiovascular system (Khalil et al.,1993), kidneys (Abdel Moneim et al., 2011), liver (Omotoso et al., 2015) and male (Lancranjan et al., 1975) and female reproductive systems (Ronis, et al., 1998). Lead interferes with bio-systems by alterations in their molecular interactions, signaling processes, and ultimately cellular function (Skerfving and Bergdahl, 2007). Lead has been linked with high incidence of renal dysfunction with attendant glomerular and tubulointerstitial changes, resulting in chronic

kidney disease, hypertension as well as hyperuricaemia (Rastogi, 2008).

Studies have shown that lead exposure to laboratory animals raised lipid peroxidation or lowered antioxidant defense mechanism (Bokara et al., 2008; Adegbesan and Adenuga, 2007). Some researchers further showed that the level of lipid peroxidation has a direct relationship with lead concentration in brain regions (Saxena and Flora, 2006) and in liver of lead exposed rats (Sandhir R, Gill KD, 1995).

Natural plants have drawn much attention for their pharmacological effects in the treatment and prevention of various diseases due to their high biocompatibility, low toxicity, and potential biological activity. Among them, *M. oleifera* is known to be important for the synthesis of useful drugs (Wadhwa, et al., 2013) that can serve medicinal purposes; they also have important agricultural, commercial and economical values.

Almost all the parts of *M. oleifera* are extensively used for the treatment of inflammation, (Mahajan et al., 2008) cardiovascular and liver disease (Omotoso et al., 2015) hematological and renal function (Gupta et al., 2005) and metal intoxications including cadmium (Toppo et al., 2015) and lead (Onah et al., 2016). The present study was conducted to investigate the effects of aqueous leaf extract of *M. oleifera* as hepato-renal protective agent against lead toxicity in rats.

Material and methods

All chemicals were purchased from the Scientific Office in Damietta, Egypt.

Plant Extraction

M. oleifera leaves were obtained from the Agricultural Research Center in Cairo, Egypt.

Preparation of extract

The leaves were dried then crushed into coarse powder. About 200 mg from the powder were soaked in 18 ml distilled water for about 24 hours, then filtered and the desired volume was administered orally.

Experimental animals

Thirty-six albino rats (weighed about 182.81 ± 6.6 g) were obtained from Mansoura University

Lab. The experimental rats were housed in the animal house in Zoology Department, Faculty of Science, Damietta University, New Damietta, Egypt. They were housed in plastic cages under controlled temperature and allowed to adapt for two weeks. Licence for animal handling.

Experimental design

6 groups, six rats per each group assigned as followed:

Control group: left without treatment for 30 days.
PbCl₂ group: treated with 200 mg orally PbCl₂ daily for 30 days.

***M. oleifera* group:** treated with 200 mg orally *M. oleifera* daily for 30 days.

PbCl₂ + *M. oleifera* group: treated with 200 mg orally PbCl₂ + 200 mg orally *M. oleifera* daily at the same time for 30 days.

Therapeutic group: was treated with 200 mg orally PbCl₂ daily for 30 days followed by 200 mg orally *M. oleifera* daily for another 30 days.

Protective group: was treated with 200 mg orally *M. oleifera* daily for 30 days followed by 200 mg orally PbCl₂ daily for another 30 days.

Haematological measurements

At the end of experiment, rats were sacrificed using chloroform and blood samples were collected directly from heart of each rat; one part of blood was collected into heparinized tubes for haematological determinations. The other part was collected without anticoagulant agent to prepare serum. The collected serum was used for liver and kidney functions parameters by using automated analyzer and available commercial kits.

Statistical analysis

The differences between the groups was estimated by the statistical analysis using one-way ANOVA and Tukey tests. A p value less than 0.05 was considered significant.

Results

Physiological studies

a. Haematological Parameters

Table 1 shows the levels of haematological parameters of male albino rats. WBCs was significantly higher in Therapeutic group and

Protective group than groups control, *M. oleifera*, $PbCl_2$ and (*M. oleifera* + $PbCl_2$), ($P < 0.006$). In addition, Monocytes count was significantly higher in Therapeutic group and Protective group than $PbCl_2$, ($P < 0.05$). On the other hand, Lymphocyte was significantly lower in group *M. oleifera* and *M. oleifera* + $PbCl_2$ than groups Therapeutic group and Protective group; ($P < 0.01$).

Table (1): Haematocrit (PCV), haemoglobin (Hb) and haematimetric indices of albino rats treated with $PbCl_2$ only and / or *M. oleifera* for a period of 30 days.

PARAMETERS \ GROUPS	Control (n=6)	<i>M. oleifera</i> (n=6)	$PbCl_2$ (n=6)	<i>M. oleifera</i> + $PbCl_2$ (n=6)	Therapeutic group (n=6)	Protective group (n=6)	P value
Hb (g/dl)	8.3±0.5	8.9±0.4	9.8±0.4	9.2±0.5	9.7±0.8	10±0.9	NS
RBCs ($10^6/\mu l$)	2.4±0.2	2.8±0.2	2.9±0.3	2.5±0.2	3.2±0.3	3.4±0.4	NS
WBCs ($10^3/\mu l$)	2.7±0.3 ^a	2.7±0.3 ^a	3.4±0.1 ^a	3.1±0.3 ^a	5.5±0.5 ^b	4.9±0.5 ^b	<0.006
Lymphocyte (%)	61±11.1 ^{ab}	50.8±7.5 ^b	27.8±3.2 ^b	51.4±10.6 ^{ab}	73±8.5 ^a	75.4±11.2 ^a	<0.01
Monocytes (%)	6±0.7	4.7±0.6 ^{ab}	4.3±0.4 ^b	4±0.4 ^{ab}	4.7±0.3 ^a	3.6±0.24 ^a	<0.05
Eosinophil (%)	2.7±0.3	2.7±0.4	2±0.5	2.7±0.4	4.2±0.5	3±0.8	NS

Values are means ± S.E.M. Values with different superscript letters within each row are significantly different (analysis of variance, $P < 0.05$). NS, non-significant.

b. Liver & Kidney Functions :

Table 2 shows the serum activity of liver and kidney enzymes of male albino rats.

ALT activity was significantly higher in Therapeutic group compared to all other groups ($P < 0.0004$), While Albumin concentration was significantly higher in Therapeutic group than control group, *M. oleifera* and (*M. oleifera* + $PbCl_2$), ($P < 0.02$). Ammonia activity was significantly higher in Therapeutic group than all

other groups except control group, ($P < 0.04$). While Urea was significantly higher in (*M. oleifera* + $PbCl_2$) than all other groups, ($P < 0.04$). On the other hand, Creatinine concentration value was significantly lower in group Protective group than *M. oleifera* and Therapeutic group, ($P < 0.04$).

While Cholesterol concentration value was significantly lower in (*M. oleifera* + $PbCl_2$) than all other groups, ($P < 0.04$).

Table (2): Liver functions and Kidney functions of serum of male albino rats treated with $PbCl_2$ only and / or *M. oleifera* for a period of 30 days.

PARAMETERS \ GROUPS	Control (n=6)	<i>M. oleifera</i> (n=6)	$PbCl_2$ (n=6)	<i>M. oleifera</i> + $PbCl_2$ (n=6)	Therapeutic group (n=6)	Protective group (n=6)	*P value
ALT(U/ml)	41.3±2.2 ^a	54.7±3.2 ^a	52.3±7.3 ^a	46.3±3.2 ^a	89.8±8.5 ^b	58.8±1.7 ^a	<0.0004
AST(U/ml)	22.7±2.5	24±1.8	21±1.7	21.8±1.5	19.8±1.7	21.2±1.4	NS
TP(g/dl)	5.3±1.7	10.3±0.7	4.73±0.55	7.7±0.6	8.4±0.9	8.9±0.3	NS
Albumin (g/dl)	2.6±0.1 ^{ab}	1.6±0.4 ^b	2.34±0.19 ^{ac}	2.5±0.2 ^{ab}	3.8±0.2 ^c	3.1±0.3 ^{ac}	<0.02
Ammonia (g/dl)	114±35.8 ^{ab}	111.8±32.3 ^b	99.2±19.3 ^b	67.5±15.8 ^b	209±57.9 ^a	83.7±27.1 ^b	<0.04
Urea (mg/dl)	92.7±12.4 ^a	176.6±48.9 ^{ab}	366.7±28.3 ^b	546±60.4 ^c	179.8±79.8 ^a	79.1±3.4 ^a	<0.04
Uric Acid (mg/dl)	4.5±1.2	3.9±0.5	4.3±0.9	4.6±0.7	4.3±0.5	4.5±0.7	NS
T. Bilirubin (mg/dl)	2.4±0.5	3.9±0.1	4.7±0.8	3.5±0.6	3.4±0.6	4.5±0.7	NS
Creatinine (g/dl)	2.5±0.3 ^{ab}	2.9±0.3 ^a	2.8±0.3 ^{ab}	2.7±0.3 ^{ab}	3.2±0.3 ^a	2±0.2 ^b	<0.04

Values are means ± S.E.M. Values with different superscript letters within each row are significantly different (analysis of variance, $P < 0.05$). NS, non-significant.

Table (3): Cholesterol, Triglycerides and Glucose of serum of male albino rats treated with PbCl₂ only and / or *M. oleifera* for a period of 30 days.

Groups parameters	Control (n=6)	<i>M. oleifera</i> (n=6)	PbCl ₂ (n=6)	<i>M. oleifera</i> + PbCl ₂ (n=6)	Therapeutic group (n=6)	Protective group (n=6)	*P value
Cholesterol (mg/dl)	294.4±28 ^a	237.1±39.2 ^a	214.1±15.1 ^a	128.1±13.4 ^b	224±44.6 ^a	254.9±44.3 ^a	<0.04
Triglycerides (mg/dl)	91.2±9.8	110.4±15.6	65.9±7.4	84.5±15.3	103.8±25.7	83.3±14.3	NS
Glucose (g/dl)	92.9±9.6	101.7±0.13	95.1±1.8	94.6±6.5	102.5±1.5	89.9±6.6	NS

Values are means ± S.E.M. Values with different superscript letters within each row are significantly different (analysis of variance, P<0.05). NS, non-significant.

c. Antioxidant Enzymes Studies:

Table 4 showed antioxidant enzymes activity of male albino rats. MDA activity was significantly higher in Protective group than groups control, *M. oleifera* and (*M. oleifera* + PbCl₂), (P <0.04).

Also both SOD and CAT activity was significantly higher in *M. oleifera*, PbCl₂, *M. oleifera* + PbCl₂, Therapeutic group and Protective group than control group, (P <0.002 and <0.004, respectively).

Table (4): Lipid peroxidation, superoxide dismutase and catalase activity of male albino rats treated with PbCl₂ only and / or *M. oleifera* for a period of 30 days

GROUPS Parameters	Control (n=6)	<i>M. oleifera</i> (n=6)	PbCl ₂ (n=6)	<i>M. oleifera</i> + PbCl ₂ (n=6)	Therapeutic group (n=6)	Protective group (n=6)	*P value
MDA (nmol/ml)	17.2±5.8 ^{ab}	8.5±2.1 ^b	40±7.7 ^{ac}	12.4±2.8 ^b	34.8±8.5 ^{ac}	31.08±9.85 ^c	<0.04
SOD (g/dl)	92.1±33.8 ^a	229.2±20.8 ^b	260.4±10.4 ^b	218.8±31.3 ^b	239.6±25.1 ^b	262.5±23.4 ^b	<0.002
CAT (U/L)	563.1±54 ^a	988.4±3.6 ^b	1017.4±7.9 ^b	1033±4.6 ^b	932.7±27.1 ^b	938.7±28.8 ^b	<0.004

Values are means ± S.E.M. Values with different superscript letters within each row are significantly different (analysis of variance, P<0.05).

DISCUSSION

The present study evaluates the protective and therapeutic effects of *M. oleifera* on Lead-induced oxidative stress in blood cells, liver and kidneys as the most prominent target organs of the toxicity of these metals.

M. oleifera is a highly valued plant, distributed in many countries of the tropics and subtropics. It has an impressive range of medicinal uses with high nutritional value. Various parts of this plant, such as the leaves, roots, seed, bark, fruit, flowers and immature pods act as cardiac and circulatory stimulants, possess antitumor, antipyretic, antiepileptic, anti-inflammatory, antiulcer, antispasmodic, diuretic, antihypertensive, cholesterol lowering, antioxidant, antidiabetic, hepatoprotective, antibacterial and antifungal activities, and are being employed for the treatment of different ailments in the indigenous system of medicine (Anwar et al., 2007).

Lead is a common environmental toxic heavy metal that has no known biological function in the body and also it is known to induce a broad range of physiological, biochemical and behavioral dysfunctions in laboratory animals and humans, including affecting the central and peripheral nervous system, hematopoietic system, cardiovascular system, kidneys, liver and reproductive system (ATSDR, 2007).

The liver is considered the principal target organ for lead toxicity, the activity of ALT and AST are sensitive indicators of acute hepatic necrosis (Modesto et al., 2013). The liver is a vital organ with a wide range of functions such as detoxification, protein synthesis and production of biochemical necessary for digestion. It is actively involved in many metabolic functions and is the frequent target for a number of toxicants (Flora et al., 2003). Any hepatic damage is associated with distortion of these functions (Wolf, 1999). The absence of consistent hepatoprotective drugs in allopathic medicine, herbs demonstrate therapeutic functions in treatment of several liver damage and disorders (Buraimoh et al., 2011).

Pb hepatotoxicity has been related to the elevation in the levels of serum liver enzymes

aspartate aminotransferase (AST) and alanine aminotransferase (ALT) (Omobowale et al., 2014) and alterations in hepatic cholesterol metabolism (Abdou and Hassan, 2014). This elevated may be due to hepatocellular necrosis, which caused increase in the permeability of the cell membrane resulting in the release of transaminases in the blood stream (Naik, 2010).

Exposure to lead for a short period affects the liver (Sivaprasad et al., 2004). In the present study, there was a significant increase in ALT level in therapeutic group compared to all other groups. Our findings are parallel with those obtained by Omobowale et al., (2014) and Onah et al., (2016) who reported increased liver enzymes after PbCl₂ administration, which is an indication of impaired liver function, hepatocyte or biliary epithelial necrosis, compromise of hepatocyte membrane integrity, and cholestasis (Adaramoye et al., 2008). Also, there is non-significant decrease in AST level of lead injected rats then treated with *M. oleifera*. In addition, no significant elevation observed in total protein level, but Alain et al., (2016) found that total serum protein level decreased significantly in the lead-exposed group compared to control and rats treated with *M. oleifera* or the combination of lead and *M. oleifera* showed no significant variation in total protein level compared to control group. Albumin decreased significantly in *M. oleifera* group and the combination of PbCl₂ and *M. oleifera* group compared to all other groups, which accordance with (Alain et al., (2016) who found that the administration of PbCl₂ to Wistar rats induce a decrease in albumin levels.

The present study showed non-significant increase in total bilirubin and blood glucose level of PbCl₂ injected rat group and *M. oleifera* group or association of both PbCl₂ and *M. oleifera* when compared to control, which accordance with (Alain et al., 2016) who found similar to our findings. But Onah et al., (2016) who stated that total bilirubin levels increased significantly after 6 weeks of PbCl₂ administration compared to their therapeutic group and other groups.

Cholesterol and triglyceride are the two major blood lipids. Cholesterol show significant decrease in *M. oleifera* + PbCl₂ group compared to all other groups, this result is in contrast with (Alain et al., (2016)) and (Hassan and Jassim, 2010) who reported that no significant variation in the serum cholesterol level was noted in the experimental groups compared to the control. On the other hand, Blood triglyceride levels non-significant decrease in protective group, PbCl₂

group and PbCl₂ & *M. oleifera* group compared to control group, also it shows that non-significant increase in therapeutic group and *M. oleifera* group compared to control group. These results in contrast with (Hassan and Jassim, 2010) who showed that the triglyceride levels significantly decreased in PbCl₂ group.

Kidney is a target organ for lead toxicity. The toxic effects of PbCl₂ on the kidney appear to be primarily localized in the kidney tubule and are manifested as excessive urinary excretion of amino acids, glucose and phosphate, natriuresis, kaliuresis and intranuclear bodies inclusion (Jadhav et al., 2007). Lead affects the excretion function of nephrons, the structural and functional unit of the kidneys (Sivaprasad, et al., 2004).

The present study showed that ammonia level was significant increase in therapeutic group compared to all other groups except control group. Also, urea was significantly increased in *M. oleifera* & PbCl₂ group than all other groups. On the other hand, creatinine was significantly decrease in Protective group compared to *M. oleifera* and Therapeutic group. These findings were in agreement with Onah et al., (2016); Alain et al., (2016) and Lakshmana et al. (2013) who reported that *M. oleifera* was found to be an effective herbal medicine in animal models of renal failure.

Also the uric acid level show non-significant change was found between treated groups and control. The current results clearly indicated that treatment with *M. oleifera* shows protective role against renal cytotoxicity-induced by lead toxicity.

The present study, MDA shows significantly increase in Protective group compared to control, *M. oleifera* and *M. oleifera* & PbCl₂ group, these findings were in agreement with (Onah et al., 2016) who found that significant increase in lipid peroxidation index (MDA) of liver after the administration of lead when compared to the control group.

The most important consequence of Pb-induced oxidative stress in liver is lipid peroxidation (Flora et al., 2003; Omobowale et al., 2014) that causes the alteration of membrane integrity and fatty acid composition (Lawton and Donaldson, 1991) and is associated with the increase in malondialdehyde (MDA) level in liver (Liu et al., 2012; Xu et al., 2008). Lead has the ability to bind to enzymes such as superoxide dismutase (SOD) and catalase (CAT) (Patra et al., 2011).

The present study found a significant increase in SOD and CAT levels in all treated groups compared to control group which may be due to

the presence of flavonoids such as quercetin and kaempferol, vitamin A, ascorbic acid, which is a potent antioxidant. Ouedraogo et al (2013); Bharali et al., (2003) and Sreelatha et al., (2009) suggesting therapeutic effect (antioxidant effect) of *M. oleifera*.

In the blood, haematimetric indices (MCH), and Thrombocytes (TC) and Leucocytes (WBCs) showed significant increase on lead exposure, but the administration of *M. oleifera* restored all the parameters back to controls also RBCS, hemoglobin and hematocrit showed no significant changes, in the contrast to Velaga et al., (2014) who observed significant decrease in PbCl₂ group, this may be due to Acute bleeding and blood loss, Allergic reactions, Cancer, Chronic kidney failure or another kidney disorder, Exercise, Heart attack, Coronary artery bypass, Infections, including tuberculosis, Iron deficiency, Vitamin deficiency, Removal of your spleen, Trauma, Burns, Exercise, Hemolytic anemia and Inflammation. In conclusion, the present study revealed hepatorenal protective activities of *M. oleifera* against lead toxicity and throw light on the effects of *M. oleifera* leaves powder have antitoxic properties. It has prevented some lead toxicities effect on many biochemical oxidative stress parameters of blood, kidney, liver and brain in albino rats so the *M. oleifera* extract is recommended to pharmaceutical industries for further research and possible use in the manufacture of drugs.

References

- Abdel Moneim, A.E., Dkhil, M.A. and Al-Quraishy, S. (2011): The protective effect of flaxseed oil on lead acetate-induced renal toxicity in rats. *Journal of Hazardous Materials*. 194:250–255.
- Abdou, H.M. and Hassan, M.A. (2014): Protective role of omega-3 polyunsaturated fatty acid against lead acetate-induced toxicity in liver and kidney of female rats. *Biomed. Res. Int.* 435857.
- Adaramoye, O.A., Osaimoje, D.O., Akinsanya, A.M., Nneji, C.M., Fafunso, M.A. and Ademowo, O.G. (2008): Changes in Antioxidant Status and Biochemical Indices after Acute Administration of Artemether, Artemether-Lumefantrine and Halofantrine in Rats. *Basic and Clinical Pharmacology and Toxicology*. 102:412-418.
- Adegbesan, B.O. and Adenuga, G.A. (2007): Effect of lead exposure on liver lipid peroxidative and antioxidant defense systems of protein-undernourished rats. *Biol Trace Elem Res*. 116:219-25.
- Agency for Toxic Substances and Disease Registry (ATSDR, 2007): Toxicological Profile for Lead. U.S. Department of health and Human Services. Public Health Service, Atlanta, GA.
- Alain, A.K., Casimir D., Sègbo, J., Senou, M., Anago, E., Ahoyo, T.A., Klotoé, J.R., Edoh, P.A. and Loko, J.R. (2016): Attenuation effect of *M. oleifera* leaves powder on Blood Biochemical disturbance induced in lead-exposed rats. *International Research Journal of Biological Sciences*. 5: 14-21.
- Anwar F, Latif S, Ashraf, M. and Gilani, A.H. (2007): *M. oleifera* : a food plant with multipurpose medicinal uses. *J. Agric. Food Chem*. 51: 6558–6563.
- Bharali, R, Tabassum, J. and Azad, M.R.H. (2003): Chemomodulatory effect of *M. oleifera* Lam. on hepatic carcinogen metabolizing enzymes, antioxidant parameters and skin papillomagenesis in mice. *Asian. Pac. J. Cancer. Prev*. 4: 131–139.
- Bokara, K.K., Brown, E., McCormick, R., Yallapragada, P.R., Rajanna, S. and Bettaiya, R. (2008): Lead-induced increase in antioxidant enzymes and lipid peroxidation products in developing rat brain. *Biometal*. 21: 9-16.
- Bressler, J., Kim, K.A., Chakraborti, T. and Goldstein, G. (1999): Molecular mechanisms of lead neurotoxicity. *Neurochem Res*. 24: 595-600.
- Buraimoh, A.A., Bako, I.G. and Ibrahim, F.B. (2011): Hepatoprotective Effect of Ethanolic Leaves Extract of *M. oleifera* on the Histology of Paracetamol Induced Liver Damage in Wistar Rat. *International Journal of Animal and Veterinary Advances*. 3: 10-13.
- Flora, S.J., Pande, M. and Mehta, A. (2003): Beneficial effect of combined administration of some naturally occurring antioxidants (vitamins) and thiol chelators in the treatment of chronic lead intoxication. *Chem. Biol. Interact*. 145: 267-280.
- Flora, S.J.S., Flora, G. and Saxena, G. (2016): Environmental occurrence, health effects and management of lead poisoning” In: Cascas SB, Sordo J, editors. *Lead chemistry, analytical aspects, environmental impacts and health effects*. Netherlands: Elsevier Publication. p. 158-228.
- Gupta, R., Kannan, G.M., Sharma, M. and Flora, S.G. (2005): Therapeutic effect of *M. oleifera* in arsenic induced toxicity in rat. *Envro Toxicol Pharmaco*. 20:456-464.
- Hassan, A.A. and Jassim, H.M. (2010): Effect of treating lactating rats with lead acetate and its interaction with vitamin E or C on neurobehavioral, development and some biochemical parameters in their pups. *Iraqi Journal of Veterinary Sciences*. 24:45-52.
- Jadhav, S.H., Sarkar, S.N., Patil, R.D. and Tripathi, H.C. (2007): Effects of sub chronic exposure via drinking water to a mixture of eight water-contaminating metals: a biochemical and

- histopathological study in male rats. *Arch. Environ. Contam. Toxicol.* 53: 667–677.
- Karrari, P., Mehrpour, O. and Abdollahi, M. (2012): A Systematic Review on Status of Lead Pollution and Toxicity in Iran; Guidance for Preventive Measures. *DARU Journal of Pharmaceutical Sciences.* 20:2.
- Khalil-Manesh, F., Gonick, H.C., weiler, E.W., Prins, B., Weber, M.A. and Purdy, R.E. (1993): Lead-induced hypertension: possible role of endothelial factors. *Am J Hypertens.* 6: 723-9.
- Lakshmana, G., Rajeshkumar, D., Ashok, R.P., Anil, K.M., Kiran, K.M., Divya, V.C., Divya, C. and Ram, S.K.B. (2013): Determination of Nephroprotective Activity of Ethanolic Leaf Extract of *Moringa pterygosperma* on Paracetamol induced Nephrotoxic Rats. *International Journal of Allied Medical Sciences and Clinical Research.* 1: 51-61.
- Lancranjan, I., Popescu, H.I., G.A., vanescu, O., Klepsch, I. and Serbanescu, M. (1975): Reproductive ability of workmen occupationally exposed to lead. *Arch Environ Health.* 30: 396-401.
- Lanphear, B.P., Dietrich, K., Auinger, P. and Cox, C. (2000): Cognitive deficits associated with blood lead concentrations <10µg/dl in adolescents and US children. *Public Health Rep.* 115: 521-9.
- Lawton, L.J. and Donaldson, W.E. (1991): Lead-induced tissue fatty acid alterations and lipid peroxidation. *Biol. Trace Elem. Res.* 28: 83-97.
- Liu, C.M., Ma, J.Q. and Sun, Y.Z. (2012): Puerarin protects the rat liver against oxidative stress-mediated DNA damage and apoptosis induced by lead. *Exp. Toxicol. Pathol.* 64:575-582.
- Mahajan, S.G. and Mehta, A.A. (2008): Effect of *M. oleifera* Lam seed extract on ovalbumin induced airway inflammation in guinea pigs. *Inhalation Toxicology.* 20: 897-909.
- Modesto Dos Santos, V., Da Costa Arruda Jr., Z., De Farias Polcheira, M., Da Silva De Souza, D.W., Rodrigues Oliveira Santos, A.M. and Santos Corrêa Da Costa, M. (2013): Acute hepatitis due to infectious mononucleosis in a 21-year-old man. *Rev. Méd. Chil.* 141: 917–921.
- Naik P. (2010): *Biochemistry.* 3rd ed. Panama. Jaypee Publishers Ltd. pp. 138–141.
- Omobowale, T.O., Oyagbemi, A.A., Akinrinde, A.S., Saba, A.B., Oluwabusola T. Daramola, O.T., Ogunpolu, B.S. and Olopade, J.O. (2014): Failure of recovery from lead induced hepatotoxicity and disruption of erythrocyte antioxidant defence system in Wistar rats. *Environmental toxicology and pharmacology.* 37:1202–1211.
- Omotoso, B.R., Abiodun, A.A., Ijomone, O.M. and Adewole, S.O. (2015): Lead-induced damage on hepatocytes and Hepatic reticular fibres in Rats; Protective Role of Aqueous Extract of *M. oleifera* Leaves (Lam). *Journal of Biosciences and Medicines.* 3: 27-35.
- Onah, C.E., Meludu, S.C., Dioka, C.E., Onuegbu, A.J., Onah, C.F., Ajaghaku, D.L., Nnodim, J.K. and Ejeatuluchukwu, O. (2016): Amelioratory Effect of Methanolic Leaf Extract of *M. oleifera* on Some Liver and Kidney Function and Oxidative Stress Markers in Lead-intoxicated Rats. *European Journal of Medicinal Plants.* 12: 1-
- Ouedraogo M., Lamien-Sanou A. and Ramde N. (2013): Protective effect of *M. oleifera* leaves against gentamicin-induced nephrotoxicity in rabbits. *Exp Toxicol Pathol.* 65: 335-339.
- Patra, R.C., Rautray, A.K. and Swarup, D. (2011): Oxidative stress in lead and cadmium toxicity and its amelioration. *Vet. Med. Int.* 457-327.
- Rastogi, S.K. (2008): Renal effects of environmental and occupational lead exposure. *Indian J. Occup. Environ. Med.* 12: 103–106.
- Ronis M.J.J., Bedger T.M and Shema S.J. (1998): Endocrine mechanism underlying the growth effects of developmental lead exposure in rat. *J Toxicol Environ Health.* 54: 101-20.
- Sandhir R and Gill K.D. (1995): Effect of lead on lipid peroxidation in liver of rats. *Biol Trace Elem Res.* 48:91-7.
- Saxena G. and Flora S.J.S. (2006): Changes in brain biogenic amines and heme-biosynthesis and their response to combined administration of succimer and Centella asiatica in lead poisoned rats. *J Pharm Pharmacol.* 58:547-59.
- Sivaprasad, T.R., Nagaraj, M. and Varalakshmi, P. (2004): Combined Efficacies of Lipoic Acid and 2, 3-Dimercaptosuccinic Acid against Lead-Induced Lipid Peroxidation in Rat Liver. *Journal of Nutritional Biochemistry.* 15: 18-23.
- Skerfving, S. and Bergdahl, I.A. (2007): Lead. In: Nordberg, G.F., Fowler, B.A., Norberg, M. and Friberg, L.T., Eds., *Handbook on the Toxicology of Metals*, 3rd Edition, Academic Press, Amsterdam, 599-643.
- Sreelatha, S. and Padma, P.R. (2009): Antioxidant activity and total phenolic content of *M. oleifera* leaves in two stages of maturity. *Plant Foods Human. Nutr.* 64:303–311.
- Toppo, R., Roy, B.K., Gora, R.H., Baxla, S.L. and Kumar, P. (2015): Hepatoprotective activity of *M. oleifera* against cadmium toxicity in rats. *Veterinary World.* 8:537-540.
- Velaga, M.K., Daughtry, L.K., Jones, A.C., Yallapragada, P.R., Rajanna, S. and Rajanna, B. (2014): Attenuation of Lead-Induced Oxidative Stress in Rat Brain, liver, kidney and blood of male wistar rats by *M. oleifera* seed powder. *J Environ Pathol Toxicol Oncol.* 4: 323-337.
- Wadhwa S., Panwar S., Saini N., Rawat S., Singhal M. (2013): A review on commercial traditional uses

- phytoconstituents and pharmacological activity of *M. oleifera*. *Global J Trad Med.* 2: 1-13.
- Wolf, P.L. (1999): Biochemical Diagnosis of Liver Disease. *Indian Journal of Clinical Biochemistry.* 14: 59-64.
- Xu, J., Lian, L.J., Wu, C., Wang, X.F., Fu, W.Y. and Xu, L.H., (2008): Lead induces oxidative stress, DNA damage and alteration of p53, Bax and Bcl-2 expressions in mice. *Food Chem. Toxicol.* 46: 1488-1494.

الملخص العربي

عنوان البحث: التأثير الوقائي والعلاجي لنبات المورينجا ضد سمية كلوريد الرصاص

قدرى البكرى^١، نجلاء العرباني^١، سماء بحيرى^{١*}

^١قسم علم الحيوان – كلية العلوم – جامعة دمياط

تهدف الدراسة الحالية الي تقييم التأثير الوقائي والعلاجي لأوراق نبات المورينجا أوليفيرا ضد السمية الناتجة عن كلوريد الرصاص في ذكور الفئران البيضاء. تم تقسيم ٣٦ فأر ألباني وزنها حوالي ١٨٢,٨ جم الي ٦ مجموعات كل منها ٦ فئران. المجموعة الضابطة أعطيت النظام الغذائي العادي والماء لمدة شهر. مجموعة كلوريد الرصاص تمت معالجتها ب ٢٠٠ ملجم كلوريد رصاص يوميا لمدة شهر عن طريق الفم. مجموعة المورينجا أوليفيرا تمت معالجتها ب ٢٠٠ ملجم مورينجا اوليفيرا يوميا عن طريق الفم لمدة شهر. كلوريد الرصاص مع المورينجا أوليفيرا تمت معالجتها ب (٢٠٠ ملجم كلوريد الرصاص + ٢٠٠ ملجم مورينجا أوليفيرا) يوميا عن طريق الفم لمدة شهر. المجموعة العلاجية (٢٠٠ ملجم كلوري الرصاص تليها ٢٠٠ ملجم مورينجا أوليفيرا). المجموعة الوقائية (٢٠٠ ملجم مورينجا أوليفيرا تليها ٢٠٠ ملجم كلوريد الرصاص). في نهاية التجربة تم اختبار كل من اختبارات الكلى والكبد وكذلك نشاط أنزيم الكتاليز وSOD وMDA. تمت زيادة نشاط ALT ومستوي البليروبين واليوريا والكريتينين والكوليسترول والامونيا والدهون الثلاثية بعد حقن الرصاص مقارنة بالمجموعة الضابطة والواقية ($P < 0.05$) ولكن تم تقليل مستوى الالبيومين لنفس المجموعة مقارنة مع المجموعة الضابطة. في المجموعة الواقية كان مستوى MDA انخفض مقارنة مع المجموعة الضابطة. أظهرت أنشطة الكتاليز وSOD زيادة معنوية في المجموعة الوقائية وكذلك المجموعة المعالجة مقارنة مع المجموعة الضابطة ($P < 0.05$). في الختام تبين أن العلاج بأوراق نبات المورينجا أوليفيرا يمكن ان تحمي الكبد والكلى ضد سمية كلوريد الرصاص.