Journal of Education for Pure Science- University of Thi-Qar
Vol.9, No.2 (June, 2019)
Website: jceps.utq.edu.iq Email: jceps@eps.utq.edu.iq

DOI: http://doi.org/10.32792/utq.jceps.09.02.21

# Effect of Ginger (Zingiber Officinale) on Serum Blood Enzyme And Lipid Profile of Female (Rattus Norvegicus) Rats Treated with Lead Acetate

*Mahdi M. Thuwaini*[1], *Lateef E. Had*[2]

[1] College of Health and medical techniques, University of South technology, Basrah, Iraq.
[2] College of Veterinary medicine, University of Thi-qar, Thi-Qar, Iraq.

**Received 05/02/2015, Accepted 09/04/2015, Published 02/06/2019**



**Abstract:**

The sub-lethal effects of lead on the liver enzyme (AST,ALT,ALP) and serum lipid profile parameters in the female rats, and the curative capacity of ginger(Zingiber *officinale*) were investigated . Twenty four adult female rats were divided into four equal groups. Group 1 as the control, the animals received the vehicle of distilled water (D.W.). Group 2 received lead acetate (I.P) (10mg/ kg b.w.). Group 3, animals drenched orally (100mg/ kg b.w.) of ginger. Group 4 has been taken (I.P) (10mg/ kg b.w.) of lead acetate in combination with ginger orally/day (100mg/kg b.w.). Weights of all animals at the first and the end of experiment were determined. Rats from all groups were sacrificed at the end of experiment (2 weeks). The study were showed that the weight of the animals was significantly decreased ($p<0.05$) in lead acetate group in comparison to other groups. The results were appeared that lead acetate induced toxicity a significant rise in Triglycerides level, and asignificant lower in glucose level. While did not significant changes in (AST, ALT, ALP and Cholestrol) in spite of there is different in thear means in comparison to control group.

**Key words**: Lead acetate , Ginger Officinale, lipid profile.

## Introduction:

Lead is an environmental contaminant due to it significant role in modern industry (Shalan et al., 2005). Both occupational and environmental exposures remain a serious problem in many developing and industrializing countries (Yücebilgic, et al., 2003). Lead (Pb) is a dangerous heavy metal and harmful even in small amounts. Humans get exposed to Pb through their environment and diet (Gidlow, 2004). The manifestations of Pb poisoning in humans are nonspecific. They may include weight loss, anemia, (Khalil-Manesh, *et al.*, 1994) nephropathy, infertility, liver, testis and heart damages' (Patocka, *et al.*, 2003; Gurer-Orhan, *et al*., 2004). In addition to killing cells via cytotoxicity, lead causes toxic effects by oxidative stress either directly or by indirectly-produced lipid peroxidation. Lead alters lipid metabolism,enhances lipid peroxidation and decreases cell membrane fluidity of developing rats (Gurer and Ercal, 2000; Villeeda-Hernandez, *et al*., 2001).

Ginger, which is the underground stem or rhizome of the plant Zingiber officinale Roscoe, contains polyphenol compounds (6-gingerol and its derivatives), which have a high antioxidant activity (Chen, et al., 1986; Herrman, 1994). It contains a host of compounds, which include acid resins, vitamin C compounds (folic acid, inositol, choline and panthothenic acid), gingerol, sesquiterpene, vitamins B3 and B6, volatile oils, and bio-trace elements (Ca, Mg, P and K). The pungency of ginger is due to gingerol, while its aroma is due to volatile oils, which are bisaboline, zingiberene and zingiberol. The medicinal values of ginger have been intensively reported. Ginger contains Mg, Ca and P, which play important roles in bone formation, and curbing muscle spasm, depression hypertension, convulsion, nausea, gastrointestinal disorders, paralysis, kidney damage, and a host of other biodysfunctions (Lee and Ahn, 1985; Kikuzaki and Nakatani, 1993; Kikuzaki, et al., 1994; Meyer, et al., 1995). There are more than 50 antioxidants isolated from rhizomes of ginger (Masuda, et al., 2004; Kikuzaki and Nakatani, 2006). Among them, 12 compounds exhibited higher antioxidant activity than α-tocopherol. Ginger and its constituents are stated to have antiemetic, antithrombotic, antihepatotoxic, anti-inflammatory stimulant, cholagogue, androgenic and antioxidant effects (Khaki, et al., 2009). Ginger is a strong anti-oxidant substance and may either mitigate or prevent generation of free radicals. It is considered a safe herbal medicine with only few and insignificant adverse/side effects (Ali, et al., 2008). Ginger extracts have been extensively studied for a broad range of biological acti-vities, especially antioxidant activities (Miller, et al., 1993). Antioxidant activities of the bark extracts of Garcinia hombroniana and essential oils of the leaves and stem of Tarchonanthus camphoratus have been reported (Nargis, et al., 2013; Nanyonga, et al., 2013; Ahmed, et al., 2000) found that ginger significantly lowered lipid peroxidation by maintaining the activities of antioxidant enzymes such as superoxide dismutase, catalase and glutathione peroxidase in blood of rats. The present study investigations, aimed to assessment the preventative role of Ginger (Zingiber officinale) against the lead acetate intoxication in rats.

## Materials And Methods:

### Tested plant:

Ginger, which is the underground stem or rhizome of the plant Z. Officinal Roscoe was purchased in a powder form from local market in Al-Shattrah city / Thi-Qar province / Iraq.

### Experimental animals:

Twenty four adult non- pregnant female rats weighing 100-120 gm were obtained from animal house of biology department / college of science/ university of Thi-Qar / Iraq. They were randomly divided into four groups with six rats in each group. The rats were fed a standard diet and free access to water before the start of the experiment. They were housed in stainless steel cages in a temperature- controlled room (25±2ºC) with a 12 h light/ 12 h dark exposure (light sun at 07.00 a.m.). The animals were divided into the below:

**Group one**: received drinking water for 2 weeks.
**Group two:** treated (I.P) with (0.5) ml of 10mg/kg lead acetate, once a day for 2 weeks.
**Group three:** received orally 100mg/kg ginger, once a day for 2 weeks.
**Group four:** received lead acetate similar to that of group 2, in addition, they received ginger similar to that of group 3, for 2 weeks.

At the end of the experimental period, each animal was weighted through a mild anesthesia. Next, each animal was sacrificed and blood samples were collected via heart puncture in plain tubes allowed to clot at room temperature. The serum was separated by centrifugation for chemical analysis which included blood glucose and cholesterol. These biochemical parameters were performed by using of kits.

**Statistical analysis: -** All data was analyzed by applying SPSS test. It was presented as (Mean ± SD).

**Results**

The present study shows that the rats body weight was significantly decreased ($p<0.05$) in lead – treated group in comparison with control group. Ginger did not induce a significant change in the animals' body weight in comparison with control. While lead acetate was given with ginger, there was significance increasing ($p<0.05$) of animals' body weights (table 1). On the other hand, with regard to lipid profile (TG and TC), the study was appeared clearly there is significantly increased ($p<0.05$) in TG concentration in G2, but that decrease was improved in animals treated Ginger plus lead acetate. While there is not significant changes in level of cholesterol between G1 and G2 and G4. However, with respect of glucose level of serum decrease ($p<0.05$) in animals received lead acetate in comparison to control group. While, the study indicated to higher significant ($p<0.05$) in animals received Ginger with lead acetate. Conversely, when Ginger, ginger gave alone, the results showed there was higher significant in glucose level and lower significant in TG level in comparison to G2 (Table 1). But this group did not shows important changes in comparison to G1 and G2 about TC consternation.

On the other hand, the present study was appeared in spit of there is increased in means of serum blood enzymes (AST, ALT and ALP). However, that increment is statically insignificant in comparison to control group. Nevertheless, results were referred to lower significant ($p<0.05$) in those enzymes (AST, ALT and ALP) when the animals were gave lead acetate in combination with Ginger (Group 4) in comparison to lead acetate group.

**Discussion:**

Lead (Pb), a highly toxic heavy metal, is widely distributed in nature. Pb is the most ancient poison known to man. Contamination of soil, water and air has become increasingly widespread through mining, refining and smelting operations (Kojima, et al., 2002).

Well, Exposure to lead can cause a concentration in most of the vital organs as the caused may be by free radicals damage to these organs, where oxidative stress is the most likelihood to be a mechanical responsible. Though, the appointing of acute toxicity is considered as a display step in the assessing of the toxic features of all Compounds. However, for many years, lead has been considered to be toxicant that constitutes the greatest danger (EpA, U.S., 1989; J. Smitherman and Harber, 1991).

The findings of the present study appeared that the ability of lead acetate to causes general disorders in the normal physiological function of the liver. Lead is environmentally omnipresent. The poisoning by common industry and is still the risk of the most common profession (Hurst, and Martin, 2004). It has known toxic effects of lead on the various organs of the body since ancient. Research has shown that all living beings, including humans exposed to lead through contaminated food and water and air as a result of an increase in consumption and fuel and industries, particularly in the areas of mining and smelting of lead crude.

Current study, the effect of lead exposure of rats as explicit on lipid profile (TC, TG), AST, ALT enzymes and glucose were examined. However, the study was appeared the effect of lead acetate on body weight,

whereby the final body weight of intoxicated rats with lead was significantly lower than that of the health normal group. The acquired results are in agreement with the findings in previous study. They found that lead caused decreases in growth rate in rats when fed lead (Seddik, 2010). These results in body weight get which may be caused by the toxic ions could be associated with several factors, one of which is imbalance metabolism produced by impairing zinc status in zinc-dependent enzymes which are necessary for many metabolic processes. Besides, lead caused lower effects on liver and spleen than those on kidney and heart (Nabil, 2012). Conversely, animals drenched orally (100mg/ kg b.w.) of ginger in combination with lead acetate was appeared markedly attenuated lead-induced hepato-toxicity in rats, as indicated by the significant increased in weight of rats after ginger treatment. The result of the above, effects explicitly indicate that ginger may offer protection by stabilizing the cell membrane in lead-induced hepatic damage (Anuradha and Krishnamoorthy, 2011). Hepatic injury following lead exposure is well characterized by changed means levels of serum hepatic marker enzymes which indicate cellular leakage and loss of functional integrity of hepatic membrane architecture. High levels of aminotransaminases (ALT and AST) and ALP are conclusive parameters in detecting liver damage. In present study, Despite the lack of a statistically significant levels in both, but there was a clear change through increased rates, increment in the serum means levels of ALT, AST and ALP was observed in lead-treated rats when compared with the control rats. The present findings are parallel with previous studies which showed increased activities of ALT, AST and ALP in plasma of lead-treated rats (Sivaprasad, et al., 2004; El-Nekeety, et al., 2009: Wang, et al., 2012 and Haleagrahara, et al., 2010). On the other hand, the combination of Ginger with lead acetate significantly ameliorated elevated liver enzymes alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP).Furthermore, The current work revealed an increase in the level of ALT, AST and ALP in lead acetate treated rats in comparison to control and this may be because of the degeneration of hepatocytes by necrosis which causes leakage of these enzymes into blood circulation (Jensen, et al., 2004). Similar hepatotoxicity lesions were also reported ( Banu and Sharma, 2005). Anyhow, alterations in the activities of these enzymes are liver specific and are considered a tool used to study varying cell viability and cell membrane permeability (Dasgupta, et al., 1996).

With regard to glucose concentration, intoxication with lead, the study was showed reduces the rate of glucose metabolism, with consequent reduction of the required energy for many anabolic process, and the profound decrease in serum glucose level which is reported in rats intoxicated with lead, might also be a cause for tissue wasting due to inappropriate availability of energy. The findings of the present study are found consistent with those reported by others (Wright, et al., 1998). However, co administration of lead and Ginger resulted to a elevation mean of blood glucose concentration. Well, Lead-induced oxidative stress in blood and other soft tissues has been proposed to be one of the probable mechanisms of lead-induced toxic impacts [Pande, et al., 2001]. Moreover, hindering of pro-oxidant/antioxidant balance might lead to the tissue injury. It was documented that lead increment the level of lipid peroxidation and altered the antioxidant defense system (Adanaylo and Oteiza, 1999; Upasani and Balaraman, 2001).

On the other hand, with regard to lipid profile (TG and TC), the study was appeared clearly there is significantly increased ($p<0.05$) in TG concentration of animals received lead acetate, but that decrease was improved in animals treated Ginger plus lead acetate. However, the association between lead exposure and high serum lipid levels is biologically conceivable and could be because of either increased synthesis or decreased removal of lipoproteins (Kojima, et al., 2002).

Moreover, Lipid profile such as cholesterol, triglycerides are increased in hepatopathy. In body cholesterol find in two form free cholesterol and estrified form. The lipid content of hepatocytes is regulated by the complete activities of cellular enzymes that catalyze lipid uptake, synthesis, oxidation and export. Fat accumulates within the hepatocytes when the "input" (either uptake or synthesis) of fatty acids to hepatocytes exceeds their "output" (oxidation and export). Different factors, extrahepatic and intrahepatic, can fail both regulator mechanisms and, thereby, promote triglyceride and cholesterol accumulation in the liver of rats with short-term and long-term prehepatic portal hypertension (Seki, et al., 2003). These findings are in accord with (Dina, et al., 2012).

In conclusion, the consequences propose that extract of ginger (*Zingiber officinale*) has powerful antioxidant effect against lead - induced oxidative stress and the extract may be useful remedy agent against lead toxicity. However, there is a need for detailed assessments to determine the active anti-oxidant compounds from this plant extract.

**Table (1):** Effect of lead acetate, ginger and ginger in combination with lead acetate on weight, liver enzymes (AST, ALT, ALk), cholesterol, triglyceride and glucose. Value was expressed as (mean± SD).

| Groups | Weight/g | AST Units\l | ALT Units\l | ALP Units\l | cholesterol mg/dl | triglyceride mg/dl | glucose mg/dl |
|---|---|---|---|---|---|---|---|
| **1-control** | 19.67 ±2.30 | 36.33 ±2.89 | 20.83 ±0.95 | 116.00 ±5.73 | 143.33 ±6.15 | 71.83 ±4.67 | 158.83 ±9.15 |
| **2- lead acetate** | 16.17 ±1.74 | 41.17 ±1.42 | 22.83 ±1.08 | 116.33 ±4.45 | 124.00 ±2.73 | 115.00 ±10.51 | 112.00 ±3.92 |
| **3- Ginger** | 19.17 ±3.50 | 43.67 ±3.59 | 24.50 ±1.41 | 173.67 ±10.01 | 135.33 ±5.43 | 72.50 ±5.90 | 153.50 ±4.90 |
| **4-ginger + lead** | 21.83 ±3.27 | 22.00 ±2.41 | 12.50 ±1.75 | 71.17 ±4.83 | 124.67 ±3.92 | 98.83 ±13.27 | 141.33 ±79 |
| **LSD** | 8.25 | 8 | 3.93 | 19.19 | 27.32 | 8.25 | 14 |

**References:**

1. Adanaylo V., P. Oteiza. 1999; Lead intoxication: antioxidant defense and oxidative stress in rat brain. Toxicology, 135, pp. 77-85.
2. Ahmed RS, Steh V, Banerjee BD. 2000; Influence of dietary ginger (Zingiber officinales Rosc) on antioxidant defense system in rat, comparison with ascorbic acid. Indian J. Exp. Biol. 38:604-606.
3. Ali BH, Blunden G, Tanira MO, Nemmar A. 2008; Some phytochemical, pharmacological and toxicological properties of ginger (Zingiber officinales Roscoe): a review of recent research. Food Chem. Toxicol. 46:409-420.
4. Kojima M, Nemoto K, Murai U, Yoshimura N, Ayabe Y, and Degawa M.2002;Altered gene expression of hepatic lanosterol 14xdemethylase (CYP51) in lead nitrate-treated rats. Arch Toxicol76: 398-403.

5. Anuradha R, Krishna moorthy P. 2011; Antioxidant activity of methanolic extract of Pongamia Pinnata on lead acetate induced hepatic damage in rats. Afr. J. Biochem. Res. 5:348-351.
6. Banu R., R. Sharma. 2005; Protective effect of vitamins C and E on lead induced hepatotoxicity in male Swice mice. Journal of Cell and Tissue Research, 5 (1), pp. 293-298.
7. Chen CH, Kuo M, Wu CH, Ho CH. 1986; Pungent compounds of ginger (Zingiber officinale (L) Rosc) extracted by liquid carbon dioxide. J. Agric. Food Chem. 34:477-480.
8. Dasgupta S, Ghosh S, Das K.1996; Transaminase activities in some metabolically active tissues of nickel treated rats under protein restricted condition. India J. Physiol. Allied Sci. 50:27-33.
9. Dina H.EL Bushuty and Naglaa M.Shanshan. 2012; EFFECT OF NATURAL HERBS OF MARJORAM AND GINGER ON HYPERCHOLESTEROLEMIC RATS. Managing Knowledge and intellectual capital in Higher Education institutions in Egypt and Arab World 11-12 April. Faculty of Specific Education - Mansoura University
10. El-Nekeety AA, El-Kady AA, Soliman MS, Hassan NS, Abdel-Wahhab MA. 2009; Protective effect of Aquilegia vulgaris (L.) against lead acetate-induced oxidative stress in rats. Food Chem. Toxicol. 47:2209-2215.
11. EPA, U.S., 1989; Evaluation of the Potential Carcinogenicity of Lead and Lead Compounds. Office of Health and Environmental Assessment. EP/600/8-89/045A.
12. Gidlow DA. 2004; Lead toxicity. Occup. Med. (Lond). 54:76-81.
13. Gurer H, Ercal N. 2000; Can antioxidants be beneficial in the treatment of lead poisoning? Free Radic. Biol. Med. 29:927-945.
14. Gurer-Orhan H, Sabir HU, Ozgüne H. 2004; Correlation between clinical indicators of lead poisoning and oxidative stress parameters in controls and lead-exposed . Toxicology 195:147-154.
15. Haleagrahara N., Jackie T., Chakravarthi S., Rao M., Kulur A. 2010; Protective effect of Etlingera elatior (torch ginger) extract on lead acetate-induced hepatotoxicity in rats. J. Toxicol. Sci. 35:663-671.
16. Herrman, K.1994; Antioxidativ wirksame Pflanzenphenole sowie Carotinoide als wichtige Inhaltsstoffe von Gewürzen. Gordian 94:113- 117.
17. Hurst, H.E. and Martin, M.D., 2004; Toxicology in Yagiela, J.A. ; Dowd, F.I. Neidle, E.A.Pharmacology and Therapeutic for Dentistry. 5th Edn., Mosby, USA, pp. 829-848.
18. Jensen JE, Stainberg SE, Freese P and Marino E. 2004; Liver function tests. J Digest Disord, 6: 1-3.
19. Khaki A, Fathiazad F, Nouri M, Khaki AA, Chelar C, Ozanci C, Ghafari-Novin M, Hamadeh M. 2009; The effects of ginger on spermatogenesis and sperm parameters of rat. Iranian J. Reprod. Med. 7:7-12.
20. Khalil-Manesh F, Tartaglia-Erler J, Gonick HC. 1994; Experimental model of lead nephropathy. Correlation between renal functional changes and hematological indices of lead toxicity. J. Trace Elem. Electrolytes Health Dis. 8:13-19.
21. Kikuzaki H, Kawasaki Y, Nakatani N. 1994; Structure of Antioxidative Compounds of Ginger, ACS Symp. Ser. 547: 237-243.
22. Kikuzaki H, Nakatani N.1993; Antioxidant Effects of Ginger Constituents, J. Food Sci. 58(6): 1407-1410.
23. Kikuzaki H, Nakatani N. 2006; Antioxidant effects of some ginger constituents. J. Food Sci. 58:1407-1410.
24. Lee IK, Ahn SY (1985). The Antioxidant Activity of Gingerol, Korean J. Food Sci. Technol. 17(2): 55-59.

25. Masuda Y, Kikuzaki H, Hisamoto M, Nikatani N. 2004; Antioxidant properties of gingerol related compounds from ginger. BioFactors 21:293-296.
26. Meyer K, Schwartz J, Crater D, Keyes B. 1995; Zingiber officinale (Ginger) Used to Prevent 8-MOP Associated Nausea, Dermatol. Nurs., 74(4): 242-244
27. Miller JK, Brzezinska-Slebodzinska E, Madsen FC.1993; Oxidative stress, antioxidant and animal function. J. Dairy Sci. 76:2812-2823.
28. Nabil M Ibrahim, Esam A Eweis, and Yasmin E Abdel-Mobdy. 2012; Effect of lead acetate toxicity on experimental male albino rat.Asian Pac J Trop Biomed. 2(1):41-46.
29. Nanyonga SK, Opoku A, Lewu FB, Oyedeji AO, Singh M. 2013; Chemical composition, antioxidant activity and cytotoxicity of the essential oils of the leaves and stem of Tarchonanthus camphoratus. Afr. J. Pharm. Pharmacol. 7:360-367.
30. Nargis J, Melati K. Lai CS, Hasnah O, Wong KC, Vikneswaran M, Khaw KY .2013; Antioxidant, anti-cholinesterase and antibacterial activities of the bark extracts of Garcinia hombroniana. Afr. J. Pharm. Pharmacol. 7:454-459.
31. Pande M., A. Mehta, B. P. Pant, S. J. S. Flora. 2001; Combined administration of a chelating agent and an antioxidant in the prevention and treatment of acute lead intoxication in rats. Environmental Toxicology and Pharmacology. 9 ; pp. 173-84.
32. Patocka J, Cerny K.2003; Inorganic lead toxicity. Acta Medica (Hradec Kralove). 46:65-72.
33. Seki S; T Kitada; H Sakaguchi; K Nakatani; K Wakasa. Histopathology, 2003; 42, 365–371.
34. Seddik L, Bah TM, Aoues A, Brnderdour M, Silmani M. 2010; Dried leaf extract protects against lead-induced neurotoxicity in Wistar rats. Eur J Sci Res. 42(1):139–151.
35. Shalan MG, Mostafa MS, Hassouna MM, Hassab El-Nabi SE, El-Rafaie A .2005; Amelioration of lead toxicity on rat liver with vitamin C and silymarin supplements. Toxicology 206:1-15.
36. Sivaprasad R, Nagaraj M, Varalakshmi P .2004; Combined efficacies of lipoic acid and 2, 3-dimercaptosuccinic acid against lead-induced lipid peroxidation in rat liver. J. Nutr. Biochem. 15:18-23.
37. Smitherman J., D. Harber. A case of mistaken identity. Herbal medicine as a cause of lead toxicity Am. J. Ind. Med., 20 (1991), pp. 795–79.
38. Upasani C. D., R. Balaraman, 2001; Effect of vitamin E, vitamin C and Spiraling on the levels of membrane bound enzymes and lipids in some organs of rats exposed to lead. Indian Journal of. Pharmacology, 33(3), pp. 185- 91.
39. Villeda-Hernandez J, Barroso-Moguel R, Mendez-Armenta M, Nava-Ruiz C, Huerta-Romero R, Rios C. 2001; Enhanced brain regional lipid peroxidation in developing rats exposed to low level lead acetate. Brain Res. Bull. 55:247-251.
40. Wang J, Yang Z, Liu L, Zhanqin Zhao LL, Xuezhong Liu ZL .2012; Protective effect of Naringenin against lead-induced oxidative stress in rats. Biol. Trace Elem. Res. 146:354-359.
41. Wright LS, Kornguth SE, Oberley TD, Siegel FL. 1998; Effects of lead on glutathione s-transferase expression in rat kidney: A dose-response study. Toxicol Sci 46: 254-259.
42. Yücebilgic G, Bilgin R, Tamer L, Tükel S. 2003; Effects of lead on Na+-K+ ATPase and Ca2+ ATPase activities and lipid peroxidation in blood of workers. Int. J. Toxicol. 22:95-97.