

Research Article

Assessment of the Therapeutic Potentials of Selenium and Vitamin E on Ovarian and Renal Tissues of Lead Exposed Rats

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Received: March 8, 2022

Accepted: April 4, 2022

Published: April 18, 2022

Abstract: Background: Lead is a heavy metal that affects the cardiovascular, gastrointestinal, renal, neurological, and reproductive systems when ingested. It is widely distributed in the environment and can persist for lengthy periods of time in biotopes. Selenium (Se) is a vital trace mineral that plays a variety of roles at the cellular level to maintain the health of both animal and humans, thus, making it relevant to a variety of pathophysiological disorders. It has structural and enzymatic functions, and is better known for its catalytic and antioxidant properties. Vitamin E is a fat-soluble vitamin whose major function is to act as an antioxidant, protecting our cells from damage that can lead to diseases like cancer. **Aim:** The study was aimed at evaluating the therapeutic potentials of selenium and vitamin E on ovarian and renal tissues of lead exposed rats. **Materials and methods:** Thirty (30) female wistar rats were weighed and randomly assigned to five groups: A, B, C, D, and E. Each group consisted of six rats which received vehicle and treatment for 30 days. Group A: Control. This group of rats was given rat feed and water *ad libitum*. Group B: lead acetate (Pb). Rats in this group received rat feeds and were gavaged with lead acetate (250mg/kg body weight). Group C: lead acetate (Pb) + Vitamin E. The rats received feeds and were gavaged with lead acetate (250mg/kg body weight) and vitamin E (600mg/kg body weight). Group D: lead acetate (Pb) + Selenium. The rats received rat feeds and were gavaged with lead acetate (250mg/kg body weight) and Selenium (0.5mg/Kg body weight). Group E: lead acetate (Pb) + Vitamin E + Selenium. The rats received rat feeds and were gavaged with lead acetate (250mg/kg body weight) and a combination of vitamin E (600mg/kg body weight) and selenium (0.5mg/Kg body weight). **Results:** The histology result shows normal ovarian tissue architecture in Groups B, C, and E, while in Group D there was proliferation of ovarian stroma. The study reveals that the substance (lead) administered is non-toxic at the dosage, concentration and duration of administration while selenium potentiates the toxic effects of lead in the ovarian tissue. Whereas, lead caused nephrotoxicity of renal tissue in groups B and D while vitamin E only and the combination of vitamin E and selenium shows protective effect. **Conclusion:** The results of the study indicates that lead induces oxidative damage in tissues as evidenced by decreased hormonal levels due to increased activities of beta reductase, a progesterone metabolizing enzyme, but its deleterious effects on ovarian tissue is dose and duration dependent, however, the reverse is the case with the renal tissue. The administration of selenium alone showed no therapeutic effect on both ovarian and renal tissues, but rather induced proliferation of ovarian stroma and tubular necrosis. **Keywords:** Lead acetate, Selenium, Vitamin E, antioxidant.

Introduction

Lead is a heavy metal that can be found in the environment and causes transplacental congenital poisoning. In comparison to other metals, lead has no physiological function in the body and is harmful even in small doses [1, 2]. High levels of lead exposure are more common in underdeveloped countries and can be found in industrial regions. Human exposure to lead is almost inevitable especially in developing countries [3].

Lead becomes poisonous when its concentration in the body blood level is above 5ug/dL or 0.24umol/L. The maximum limit for blood lead for adults is 10ug/dl (10ug/100g) and for children is 5ug/dl [4, 5]. Lead poisoning most commonly occurs via skin contact, oral ingestion, or inhalation. The effects of lead on various systems differ significantly, especially when it comes to gametogenesis and the cyclic pattern of female reproductive function. Toxic metals can disrupt the menstrual cycle, ovulation, and female fertility by causing hormonal changes [6].

Heavy metal exposure, can induce reproductive dysfunction in women; even minor hazardous metal exposure can impact women's reproductive health [7]. Women with blood Pb levels greater than 25 g/L have a 3-fold higher risk of infertility than women with blood Pb levels less than 25 g/L [8, 9].

Apart from the gonads, lead toxicity also impacts negatively on the kidney, liver, lung, brain, blood, and bone marrow. High incidence of renal disease and renal failure has been linked to chronic occupational exposure to lead. Excessive and prolonged exposure to lead can give rise to chronic lead nephropathy which is an irreversible renal disease condition that develops within months or years [10].

Selenium (Se) as a component of selenoproteins plays structural and enzymatic roles and is well known for its catalytic and antioxidant actions [11]. Important cellular processes such as synthesis of deoxynucleotides for deoxyribonucleic acid (DNA), scavenging of harmful signaling peroxides, reduction of oxidized proteins and membranes, redox signaling control, thyroid hormone metabolism, protein folding, and Se transportation and storage rely on selenoproteins, [12, 13]. Studies have shown that selenium plays positive role in female fertility, has favourable impact on pregnancy outcomes and overall ovarian physiology [14].

Despite its potential impact on embryonic gonadal development, Se is also said to preserve female reproductive health against oxidative stress; and a given number of studies have implicated Se shortage in obstetric complications. Se deficiency has been consistently connected with recurrent miscarriage, pre-eclampsia, pre-term birth, and small for gestational age newborns [15, 16].

Research evidence suggests that vitamin E is essential for the prevention of oxidation. As a biochemical molecule Vitamin E also known as α -tocopherol is an exogenous lipid soluble antioxidant that prevents cell membrane from the activities of lipid peroxidation through scavenging of free radicals by the activation of intracellular antioxidant enzymes [15]. Vitamin E requirements are influenced by the intake of dietary polyunsaturated fatty acid (PUFA) [17, 18].

Fatty acids have been identified as significant regulators of biological processes in a variety of living tissues, particularly in reproductive organs. Omega-3 and omega-6 fatty acids, have been identified as critical factors in reproductive systems [19, 20]. Antioxidants are thought to be a crucial influence in avian reproduction because they protect reproductive organs from the harmful effects of reactive oxygen species [17, 21]. However, the role of dietary antioxidants have not been exhaustively evaluated in humans [22].

Vitamin E and selenium have proven to be beneficial in some disease processes by preventing lipid peroxidation and thus protects the body. It is against this background that the researchers wish to assess the effect of vitamin E and selenium on lead induced toxicity on ovarian and renal tissues.

Materials and Methods

Animals

Thirty (30) healthy adult female wistar rats weighing between 130-191g were obtained from the Animal House of the Department of Pharmacology, Niger Delta University (NDU), Wilberforce Island. They were housed in well-aerated laboratory cages in a room under standard conditions, with a temperature range of 25-30°C and a 12-hour light/dark cycle, in the animal house of the Department of Medical Laboratory Science, Niger Delta University, NDU, Amassoma.

The rats were fed with commercial rat feeds provided by the animal house and were given free access to drinking water. They were allowed to acclimatize to the laboratory environment for a period of two weeks (14 days) before commencement of the experimental protocol. The EU directive for animals (2010/63/EU) was strictly adhered to in the handling of animals all through the experimental period.

Purchase of Chemicals

Five hundred (500g) gram of Lead acetate of (99% purity) with LOT # L117921504, DL- α -tocopherol Acetate (99% purity) with LOT#L182801601 and anhydrous Sodium Selenite (98%) with LOT#Lmo125A1708 manufactured by LOBA CHEMIE PVT LTD, Mumbai, India was procured from a reputable chemical and laboratory reagent supplier, Effective Chemical and Laboratory Reagent Ltd, MelfordOkilo Road, opposite Okaka Primary School, Yenagoa, Bayelsa, Nigeria.

Animal Grouping and Experimental Design

The animals were weighed and divided randomly into five groups: Groups A, B, C, D and E. Each group consisted of six rats.

Group A: Control. This group of rats received rat feeds and water *ad libitum*.

Group B: lead acetate (Pb). This group of rats received rat feeds and were gavaged with lead acetate (250mg/kg body weight) daily for 30 days.

Group C: lead acetate (Pb) + Vitamin E. The rats in this group received rat feeds and were gavaged with lead acetate (250mg/kg body weight) and vitamin E (600mg/kg body weight) daily for 30 days.

Group D: lead acetate (Pb) + Selenium. The rats in this group received standard rat feeds and were gavaged with lead acetate (250mg/kg body weight) and Selenium (0.5mg/Kg body weight) daily for 30 days.

Group E: lead acetate (Pb) + Vitamin E + Selenium. The rats in this group received rat feeds and were gavaged with lead acetate (250mg/kg body weight) and a combined therapy of vitamin E (600mg/kg body weight) and selenium (0.5mg/Kg body weight) for 30 days.

Route and Duration of administration

Lead acetate, vitamin E and selenium were administered orally by gastric gavage once daily for 30 days.

Histological studies

At the end of the experimental period, animals were sacrificed using chloroform inhalation method and the ovaries and kidneys of each animal of control and treatment groups were collected and fixed in 10% formal saline solution. Routine tissue processing was done using automatic tissue processor Histokinette (LEICA TP 1020).

The tissues were embedded in paraffin wax using tissue embedder (LEICA EG 1160) and trimmed in a rotary microtome (LEICA RM 2125 RTS) at 20 microns and sectioned at 5 microns thickness.

Tissues sections were attached to slides and subsequently dewaxed in xylene and stained in Haematoxylin and Eosin using the method of Avwioro (2014) [23] for general tissue structure. The stained slides were then examined using light microscope at x100 magnification.

Statistical Analysis

All data were analysed using one-way analysis of variance (ANOVA) and group means were compared using the Dunnett's Multiple Comparison Test using GraphPad Prism software version 5.01. P values of <0.05 were considered statistically significant.

Results

Group A (negative control) shows sections of the ovary and kidney of female rats exposed to rat feeds and water only. The ovarian section shows an ampulla with a lumen and healthy epithelium. Also seen are tertiary, secondary, and primordial follicles with oocytes. The stroma is rich and vascular consistent with normal histology of the ovary. While the renal tissue (kidney) shows glomerulus with normal Bowman capsule and numerous tubules displaying normal epithelium consistent with normal histology of the kidney.

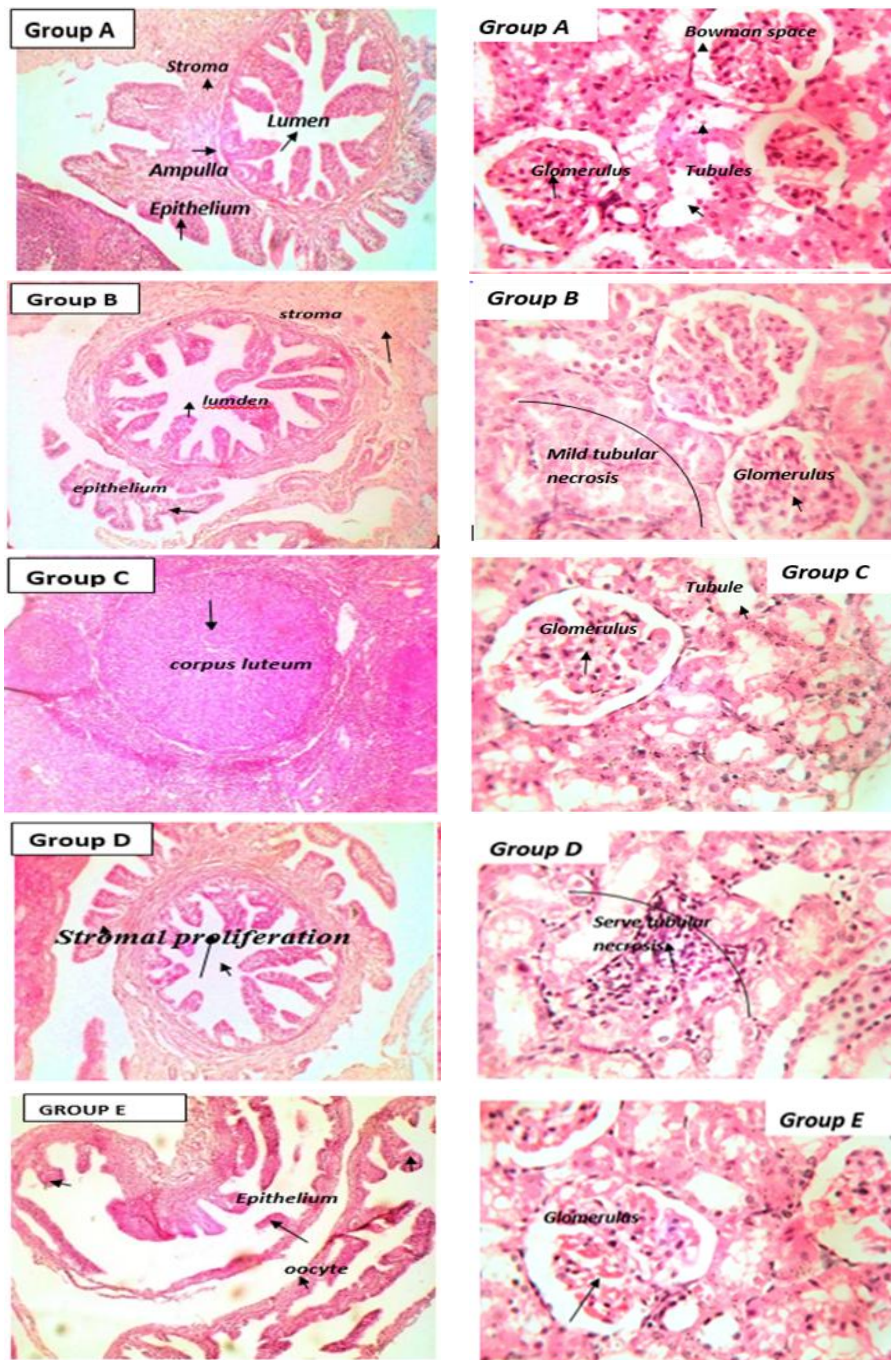
Group B shows sections of the ovary and kidney of female rats exposed to 1.5mls of 250mg/kg body weight of lead for 30 days only. The section shows an ampulla with a lumen and healthy epithelium. Also seen are granulosa cells layer, secondary, and primordial follicles with oocytes. The stroma is rich and vascular consistent with normal histology of the ovary. Substance administered is non-toxic to the ovary at the dosage, and duration. The reverse is the case in the kidney as the photomicrograph (Group B) shows area of mild tubular necrosis.

Group C shows sections of the ovary and kidney of female rats exposed to 1.5mls of 250mg/kg body weight of lead for 30 days and treated with 1.5mls of 600mg/kg of vitamin E only. The ovary shows a rich and vascular stroma consistent with normal histology of the ovary. The kidney tissue also shows normal glomerulus and renal tubules.

Group D shows sections of the ovary and kidney of female rats exposed to 1.5mls of 250mg/kg body weight of lead and treated with 1.2 ml of 0.5mg/kg of selenium. The section shows proliferation of the ovarian stroma. Selenium seems not to protect the ovarian tissue from the toxic effects of lead. In the renal tissue, severe tubular necrosis with numerous inflammatory cells dominated by neutrophils was observed.

Group E shows sections of the ovary and kidney of female rats exposed to 1.5mls 250mg/kg body weight of lead for 30 days and treated with 1.2 ml of 0.5mg/kg of selenium plus 1.5mls of 600mg/kg of vitamin E.

The ovarian section shows an ampulla with a lumen and healthy epithelium. Also seen are granulosa cells, secondary, and primordial follicles with oocytes. The stroma is rich and vascular consistent with normal histology of the ovary. Histomorphology of the renal tissue appears normal compared with control.



Histology of the ovaries

Figure 1. Photomicrographs of haematoxylin and eosin-stained ovarian tissue sections x 400 magnification. Normal histology (A), Rich and vascular stroma consistent with normal histology of the ovary (B), Rich and vascularized stroma-vitamin E group (C), Proliferation of the stroma-Selenium group (D), Normal histology-Vitamin E + Selenium group (E)

Histology of the Kidney

Figure 2. Photomicrographs of haematoxylin and eosin-stained kidney tissue sections x 400 magnification. Glomerulus with normal Bowman capsule and numerous tubules displaying normal epithelium consistent with normal histology of the kidney (A). Mild tubular necrosis (B), Severe tubular necrosis with numerous inflammatory cells dominated by neutrophils (D). Normal histology compared with control (E). Substance administered to group B and D demonstrated nephrotoxicity while treatment in group C and Group E shows protective effect.

Discussion

Lead is one of the heavy metals and a widely dispersed environmental pollutant. Research evidence reveal that renal dysfunction, cognitive impairment, behavioral deficits, high blood pressure are some medical conditions that can arise in humans following lead exposure between 10 and 15 µg/dL [2, 5]. High incidence of renal dysfunction characterized by glomerular and tubulointerstitial changes with eventual chronic renal failure has been linked to prolonged or chronic workplace exposure to lead [24]. Lead have also been found to cause hormonal changes in women and generally affect the menstrual cycle, ovulation, and female fertility [25]. This current study also recorded nephrotoxic effects of lead as evidenced by tubular necrosis of the renal tissue and proliferation of ovarian stroma in animal models.

Chang et al. [8] observed that women with blood lead levels greater than 25 g/L had higher risk of infertility than those with blood Pb levels less than 25 g/L. Our study noticed decrease in reproductive hormonal levels which is consistent with the study by Strivastava et al. [26], which found that prepubertal females exposed maternally to low levels of lead had lower circulating levels of estradiol.

Certain micro-nutrients are known for their public health importance and they include iron, zinc, iodine, selenium, copper, vitamins A, E, C, D, B2 B6 and folate [27]. Selenium is said to exhibit antioxidant, anti-inflammatory, antimutagenic, antitumoral or chemopreventive, antiviral, antibacterial, antifungal and antiparasitic properties [28]. It is also useful in the prevention of dementia and cognitive impairment [29]. Selenium reduces some toxic elements by inhibiting their absorption by forming insoluble compounds [30, 31]. In humans, selenium is essential nutritionally, and has been found to play critical roles in reproduction, thyroid hormone metabolism, DNA synthesis, and protection from oxidative damage [16, 32].

Lately, attention has been focused toward ascertaining the kidney protective potential of selenium. Research evidence claims that Se has the capacity to prevent lead-and cadmium toxicity of the kidney, liver, ovary, and testis; and can prevent oxidative stress, endoplasmic reticulum stress, and as well reduce lipopolysaccharide-induced myocarditis and nephropathy [33, 34]. It is established that, selenium supplements when administered to kidney failure patients resulted in the reduction of oxidative stress. Also, a related study noticed decrease in plasma selenium level in patients with acute renal injury [24]. However, this present study observed that animals treated with selenium following lead administration shows severe tubular necrosis with numerous inflammatory cells dominated by neutrophils and proliferation of ovarian stroma in renal and ovarian tissues respectively. This is indicative of the fact that selenium could not ameliorate lead induced kidney and ovarian tissue toxicity but rather potentiated the toxic effects of lead. There are conflicting reports on the effects of selenium on various tissues, and some studies propose that this mineral can have negative impact on various tissues [35]. Cell damage may be orchestrated by oxidative stress, via mechanisms such as lipid peroxidation and oxidative damage to proteins and DNA. In this study, selenium administration resulted in increased oxidative stress, which potentiated the effects of lead, resulting in renal and ovarian tissue destruction and proliferation. The findings of our study is consistent with previous study by Razavi et al. [35].

Vitamin E is the major lipid-soluble antioxidant in the cell antioxidant system and is exclusively obtained from the diet. Vitamin E protects polyunsaturated fatty acids and other components of cell membranes and low-density lipoproteins from oxidation by free radicals. Antioxidants possess both chelating and ROS scavenging capacity enabling elimination of lead from intracellular sites and blood stream. Vitamin E eliminates free radicals thus preventing cell damage by stabilizing sulfhydryl groups of proteins binding to cell membranes [36, 37]. This study observed that vitamin E exhibited protective effect of both the kidney and ovary in animal models, while selenium and vitamin E co-administration on experimental animals recorded restoration of renal and ovarian tissue architecture. The histomorphology result of this study shows that vitamin E alone and the combined

treatment of vitamin E and selenium is more potent than only selenium which is in absolute agreement with results obtained by previous research which indicates that the use of antioxidants ameliorate the degenerative effects in tissues [35, 38].

Conclusion

The results of this study clearly suggest that lead causes oxidative damage in tissues, as demonstrated by decrease hormonal levels due to increased activities of beta reductase, a progesterone metabolizing enzyme. However, the adverse effects of lead on ovarian and renal tissue are dosage and duration dependent. Selenium treatment alone had no therapeutic effect on ovarian and renal tissue, but rather potentiates lead-induced ovarian stroma proliferation and renal tubular necrosis. While vitamin E exhibited a protective effect of the ovary and kidney, a combination of selenium and vitamin E resulted in restoration of ovarian and renal tissue histomorphology.

Conflicts of interest: There is no conflict of interest of any kind.

References

1. Taupeau C, Poupon J, Nomé F, Lefèvre B. Lead accumulation in the mouse ovary after treatment-induced follicular atresia. *Reprod Toxicol*. 2001;15(4):385-91.
2. Tokar EJ, Benbrahim-Tallaa L, Waalkes MP. Metal ions in human cancer development. *Metal Ions in Toxicology: Effects, Interdepend*. 2015;8:375-401.
3. Andrews JS. Biologic Monitoring and Biomarkers. ATSDR-Hazardous Waste Conference. Atlanta, GA, USA: Agency for Toxic Substances and Disease Registry;2007.
4. Al-Saleh E, Nandakumaran M, Al-Shammari M, Al-Harouny A. Maternal–fetal status of copper, iron, molybdenum, selenium and zinc in patients with gestational diabetes. *J Mat Fet Neonat Med*. 2004;16(1):15-21.
5. Al-Saleh I, Coskun S, Mashhour A, Shinwari N, El-Doush I, Billedo G, Jaroudi K, Al-Shahrani A, Al-Kabra M, Mohamed GE. Exposure to heavy metals (lead, cadmium and mercury) and its effect on the outcome of in-vitro fertilization treatment. *Int J Hyg Environ Health*. 2008;211(5-6):560-79.
6. Sengupta P, Banerjee R, Nath S, Das S, Banerjee S. Metals and female reproductive toxicity. *Human Exper Toxicol*. 2015;34(7):679-97.
7. Bloom MS, Louis GM, Sundaram R, Kostyniak PJ, Jain J. Associations between blood metals and fecundity among women residing in New York State. *Reprod Toxicol*. 2011;31(2):158-63.
8. Chang SH, Cheng BH, Lee SL, Chuang HY, Yang CY, Sung FC, Wu TN. Low blood lead concentration in association with infertility in women. *Environ Res*. 2006;101(3):380-6.
9. Mendola P, Messer LC, Rappazzo K. Science linking environmental contaminant exposures with fertility and reproductive health impacts in the adult female. *Fert Steril*. 2008;89(2):e81-94.
10. Zhou Y, Vaidya VS, Brown RP, Zhang J, Rosenzweig BA, Thompson KL, Miller TJ, Bonventre JV, Goering PL. Comparison of kidney injury molecule-1 and other nephrotoxicity biomarkers in urine and kidney following acute exposure to gentamicin, mercury, and chromium. *Toxicol Sci*. 2008;101(1):159-70.
11. Rayman MP. The importance of selenium to human health. *Lancet*. 2000;356(9225):233-41.
12. Papp LV, Lu J, Holmgren A, Khanna KK. From selenium to selenoproteins: synthesis, identity, and their role in human health. *Antiox Redox Signal*. 2007;9(7):775-806.
13. Zoidis E, Seremelis I, Kontopoulos N, Danezis GP. Selenium-dependent antioxidant enzymes: Actions and properties of selenoproteins. *Antiox*. 2018;7(5):66.

14. Mistry HD, Pipkin FB, Redman CW, Poston L. Selenium in reproductive health. *Am J Obstet Gynecol.* 2012;206(1):21-30.
15. Agarwal A, Aponte-Mellado A, Premkumar BJ, Shaman A, Gupta S. The effects of oxidative stress on female reproduction: a review. *Reprod Biol Endocrinol.* 2012;10(1):1-31.
16. Langley S. A nutrition screening form for female infertility patients. *Can J Diet Pract Res.* 2014;75(4):195-201.
17. Raederstorff D, Wyss A, Calder PC, Weber P, Eggersdorfer M. Vitamin E function and requirements in relation to PUFA. *Brit J Nut.* 2015;114(8):1113-22.
18. Wathes DC, Abayasekara DR, Aitken RJ. Polyunsaturated fatty acids in male and female reproduction. *Biol Reprod.* 2007;77(2):190-201.
19. Blasbalg TL, Hibbeln JR, Ramsden CE, Majchrzak SF, Rawlings RR. Changes in consumption of omega-3 and omega-6 fatty acids in the United States during the 20th century. *Am J Clin Nut.* 2011;93(5):950-62.
20. Fouladi-Nashta AA, Wonnacott KE, Gutierrez CG, Gong JG, Sinclair KD, Garnsworthy PC, Webb R. Oocyte quality in lactating dairy cows fed on high levels of n-3 and n-6 fatty acids. *Reprod.* 2009;138(5):771-81.
21. Fontana R, Torre SD. The deep correlation between energy metabolism and reproduction: a view on the effects of nutrition for women fertility. *Nutri.* 2016;8(2):87.
22. Leroy JL, Sturmev RG, Van Hoeck V, De Bie J, McKeegan PJ, Bols PE. Dietary fat supplementation and the consequences for oocyte and embryo quality: hype or significant benefit for dairy cow reproduction?. *Reprod Domest Anim.* 2014;49(3):353-61.
23. Avwioro OG. Staining. In: *Histochemistry and tissue pathology principles and techniques*, 3rd edition, Claverianum Press Nigeria Limited; 2014:133-168.
24. Iglesias P, Selgas R, Romero S, Díez JJ. Selenium and kidney disease. *J Nephrol.* 2012;26(2):266-72.
25. Krieg Jr EF. The relationships between blood lead levels and serum follicle stimulating hormone and luteinizing hormone in the third National Health and Nutrition Examination Survey. *Environ Res.* 2007;104(3):374-82.
26. Srivastava S, Mehrotra PK, Srivastava SP, Tandon I, Siddiqui MK. Blood lead and zinc in pregnant women and their offspring in intrauterine growth retardation cases. *J Analyt Toxicol.* 2001;25(6):461-5.
27. Ekweagwu E, Agwu AE, Madukwe E. The role of micronutrients in child health: A review of the literature. *Afr J Biotechnol.* 2008;7(21).
28. Labunskyy VM, Hatfield DL, Gladyshev VN. Selenoproteins: molecular pathways and physiological roles. *Physiol Rev.* 2014;94(3):739-77.
29. Song G, Zhang Z, Wen L, Chen C, Shi Q, Zhang Y, Ni J, Liu Q. Selenomethionine ameliorates cognitive decline, reduces tau hyperphosphorylation, and reverses synaptic deficit in the triple transgenic mouse model of Alzheimer's disease. *J Alzh Dis.* 2014;41(1):85-99.
30. Mehdi Y, Hornick JL, Istasse L, Dufrasne I. Selenium in the environment, metabolism and involvement in body functions. *Molec.* 2013;18(3):3292-311.
31. Cardoso BR, Bandeira VS, Jacob-Filho W, Cozzolino SM. Selenium status in elderly: relation to cognitive decline. *J Trace Elem Med Biol.* 2014;28(4):422-6.
32. Ross AC, Caballero BH, Cousins RJ, Tucker KL, Ziegler TR. *Modern nutrition in health and disease.* Wolters Kluwer Health Adis (ESP); 2012 Dec 22.

33. Yu Z, Wang F, Liang N, Wang C, Peng X, Fang J, Cui H, Jameel Mughal M, Lai W. Effect of selenium supplementation on apoptosis and cell cycle blockage of renal cells in broilers fed a diet containing aflatoxin B1. *Biol Trace Elem Res*. 2015;168(1):242-51.
34. Zhang C, Lin J, Ge J, Wang LL, Li N, Sun XT, Cao HB, Li JL. Selenium triggers Nrf2-mediated protection against cadmium-induced chicken hepatocyte autophagy and apoptosis. *Toxicol in Vitro*. 2017;44:349-56.
35. Razavi SM, Seghinsara AM, Abedelahi A, Salimnejad R, Tayefi H. Effect of vitamin E and selenium on oxidative stress and tissue damages induced by electromagnetic fields in immature mice ovarian. *Cres J Med Biol Sci*. 2017;4(3):120-5.
36. Soetan KO, Olaiya CO, Oyewole OE. The importance of mineral elements for humans, domestic animals and plants-A review. *Afr J Food Sci*. 2010;4(5):200-22.
37. Abd El Rahman NA, Abd El Hady AM, Eltahawy NA. Silymarin and vitamin E modulate 950mhz electromagnetic field-induced oxidative stress and hormonal changes in male albino rats. *J Amer Sci*. 2014;10(9):1-8.
38. Asghari A, Montaseri A, Khaki AA. An ultrastructural study of the antioxidant effects of vitamin E and fennel extract on zona pellucida cell changes of rat ovaries under non-ionizing 50hz electromagnetic fields. *Cres J Med Biol Sci*. 2(2):37-41.

Citation: Sylvanus B, Godwin II, Williams T, Ashimiedua UG. Assessment of the Therapeutic Potentials of Selenium and Vitamin E on Ovarian and Renal Tissues of Lead Exposed Rats. *Int J Rec Innov Med Clin Res*. 2022;4(2):1-9.

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