

The histopathological effect of selenium - and vitamin E on liver and testis of Domestic *Lepus lepus* rabbits

Adnan Issa AL badran , Ikbal Abdul Aziz AL rufaei and Hanaa Salman Al sudani

Basrah University, College of science, Department of biology.

Abstract

The study was conducted to investigate the effect of selenium and selenium in addition to vitamin E on liver, and testis of *Lepus Lepus* rabbits after 30 days of treatment . The histological study revealed that selenium treated liver showed obvious necrosis and mild inflammation in contrast with control liver where the hepatic cells arranged around the central vein. While, necrosis was not well visible in rabbit treated with vitamin E and selenium. We can conclude that vitamin E had decreased the hepatocellular damage caused by selenium.

The degeneration in spermatocyte cell .in selenium treated rabbits was very clear, but there was an obvious increase in the number of spermatogonia cell in rabbits testis treated 'with vitamin E and selenium. These results showed that selenium has antigonadal activities "that can be ameliorated by vitamin E administration.

Keyword: Histopathological, selenium, vitamin E, effect

Introduction:

Selenium (se) is a metalloid element, of atomic weight 79; located in group VI of the periodic table, The element is essential for mammals, including humans, as a component of two enzymes glutathione peroxidase and iodothyronine 5'-de-iodinase. In addition, a distinct. Selenium-containing protein, selenoprotein P, has been identified in the plasma⁽¹⁾

Selenium exerts its anticarcinogenic effects by multiple mechanisms. In the physiological dosage range, selenium appears to function as an antimutagenic agent, preventing the malignant transformation of normal cells and the activation of oncogenes.

The protective effects of selenium associated with its presence in the glutathione peroxidases which protect DNA and other cellular components from damage by oxygen radicals⁽²⁾. ⁽³⁾showed that selenium is anormal

component for enzymes, proteins, and .aminoacyl transfer ribonucleic acid

The acute selenium toxicity causes Blind staggers and the features of acute poisoning include vomiting and diarrhoea, mucosal Damage, metabolic acidosis and muscle spasm^(4,5). However, the chronic selenium toxicity causes Alkali disease and the features of chronic selenosis include gastro- intestinal disturbance, loss of hair and nails, infection of nail beds and skin lesion, and neurological impairment^(4,6). The need of vitamin E depends on the supply with selenium, with unsaturated fatty acids and with S- containing amino acids. Selenium is necessary for the synthesis of glutathione peroxidase. The vitamin E is effective in the degradation of fatty acid - Peroxidase compounds in the membranes, the glutathione peroxidase in the soluble proportions of the cells. The vitamin E is important for the detoxication processes in the liver and for the immune defense⁽⁷⁾.

Material and Methods :

Eighteen male adult rabbits *Lepus Lepus* (6 weeks old) (1750 - 1500 gm weight) were housed in iron cages with (1x 0.5 x 0.5) m dimensions. They were separated into 6 rabbits as a control group and 12 rabbits as experimental group. The latter group was divided to group (1) which the animals were inoculated with (10) µg selenium was taken orally and group (2) were the animals had inoculated with (10) µg selenium with addition to (0.4) mg vitamin E was given

orally as small capsules. All the animals were killed under ether anaesthesia after (30) days of treatment. Specimens from liver and testis related to control and experimental groups were prepared for light microscopic study according to⁽⁸⁾. The samples were cut off, washed with normal saline and immediately immersed in 10% formalin fixative. The tissue samples dehydrated in a graded ethanol series (50 %, 70 %, 95 % and absolute alcohol) after that they were cleared with xylene, two changes 15 min in each. All the specimens were infiltrated with melted paraffin for 6-12 hr. Finally these pieces were embedded in paraffin - wax, serially sections of about (5 micron) thickness were cut and stained with hematoxylin-eosin. The sections were dehydrated, cleared, mounted in (D .P .X) and examined microscopically with phase contrast light microscope.

Results:

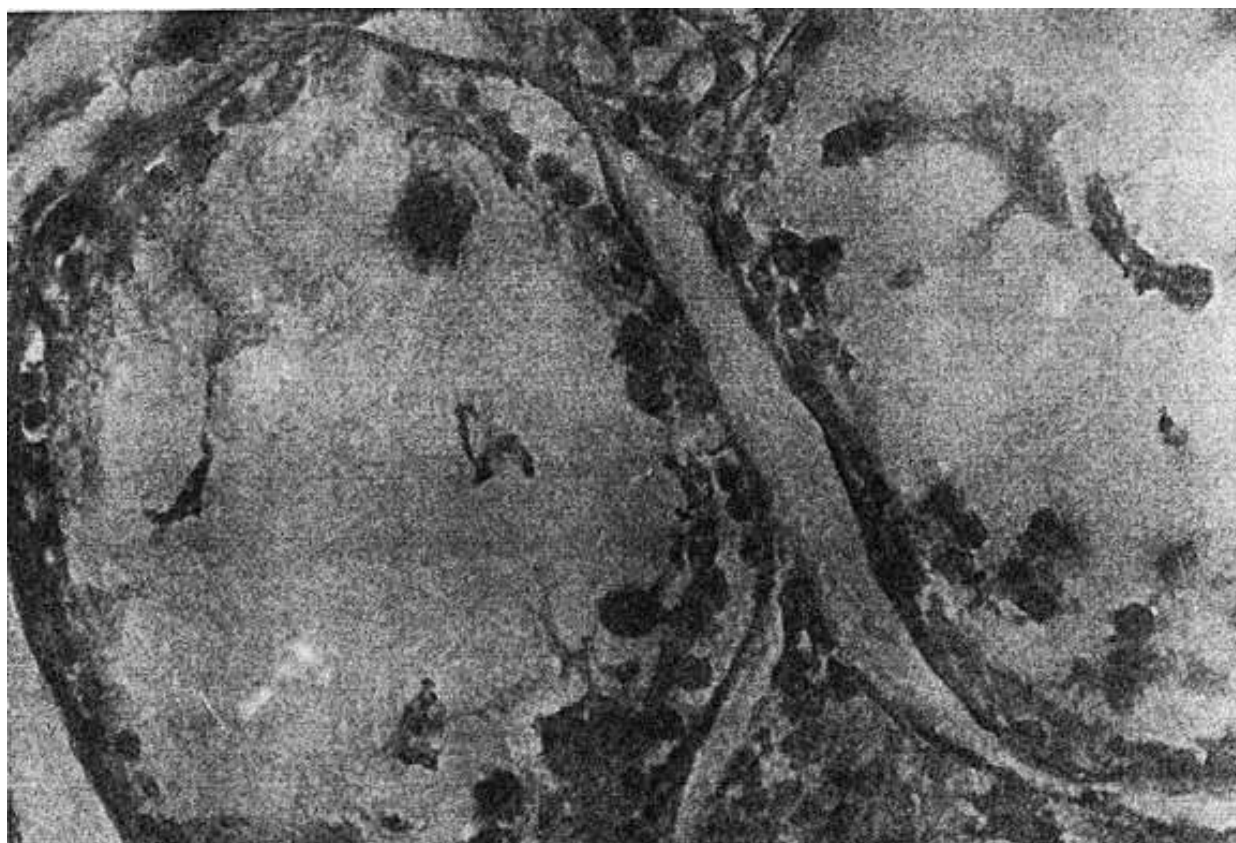
Examination of control sections related to liver showed normal architecture. The hepatic cell arranged as hepatic plates around central vein. (Fig 1).

Light microscope examination of the experimental rabbits related to group (1) treated with selenium showed changes in liver architecture characterized by altered hepatic cells arranged, increased necrosis and mild inflammation. (Fig 2).

Sections from liver related to experimental rabbits related to group (2) treated with (selenium + vitamin. E) Illustrated the hepatic cells were more regularly and the necrosis not well visible but still with significant differences from the control group (Fig 3).

Moreover sections of the test are taken from control rabbits show the spermatogonia cells were arranged peripherally and spermatocytes in different stages of development (Fig 4).

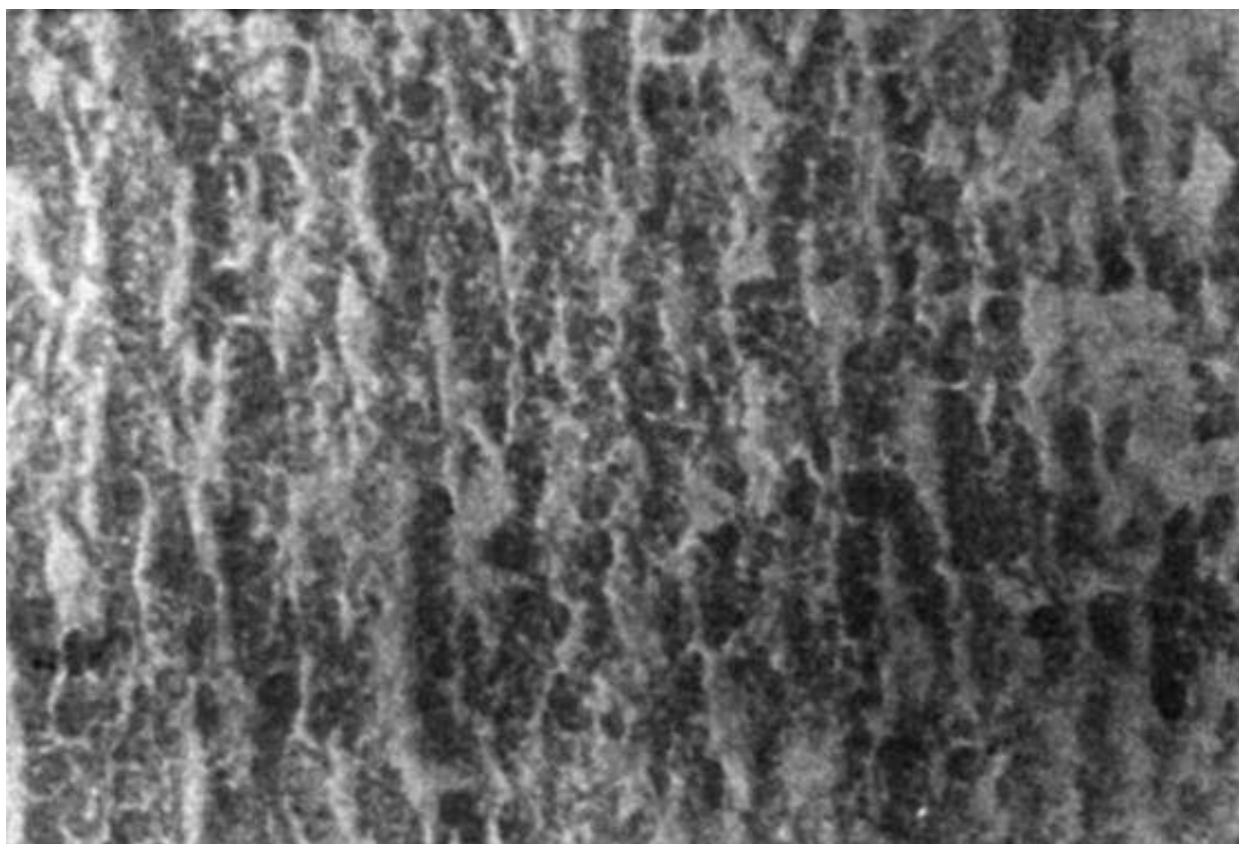
Our results identified the histological changes of testis excised from animals treated with selenium; the seminiferous tubules were enlarged, complete degeneration in spermatocytes and decrease in spermatogonia cells (Fig 5). While sections of testis from rabbits treated with (selenium + vitamin, E) show less changes in histological structure of the seminiferous tubule and the vitamin E causing increase in the spermatogonia cells (Fig 6).



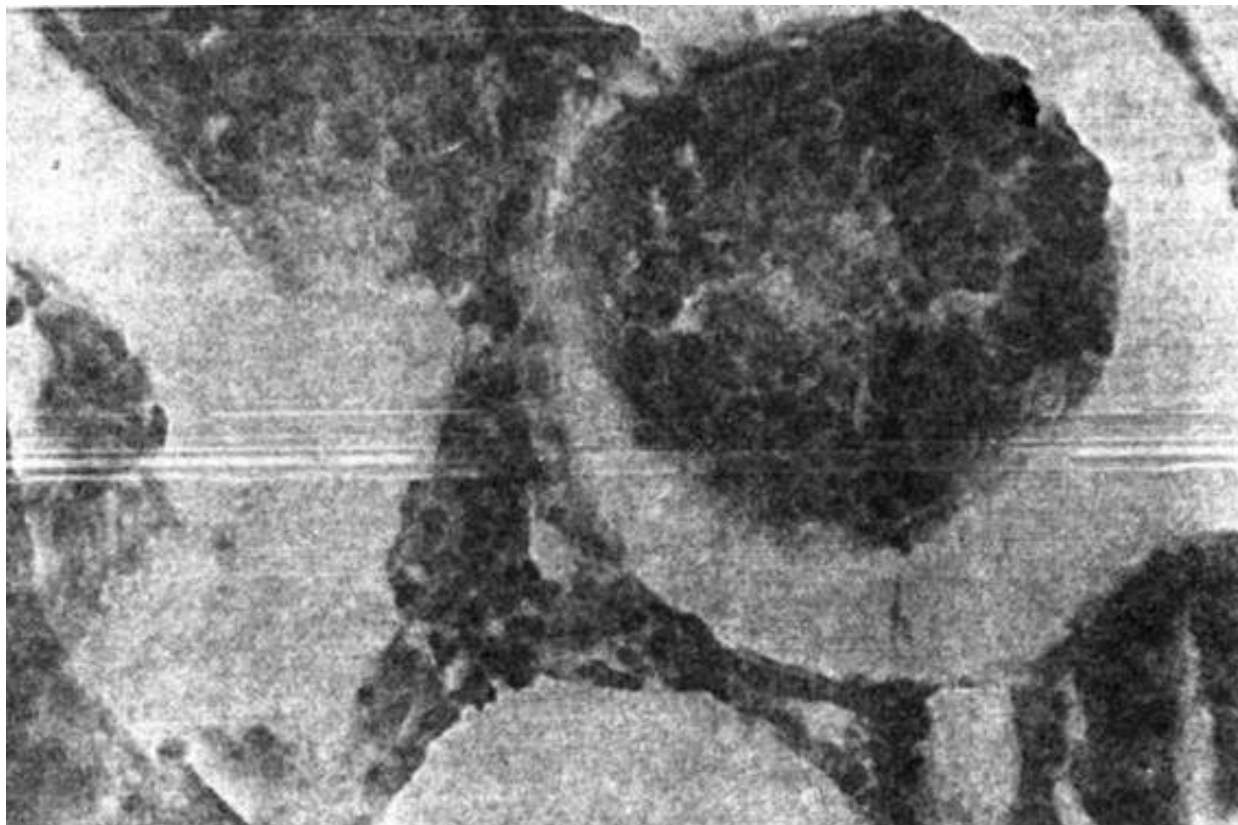
Photograph (1):- section of liver taken from control animal (330 x)



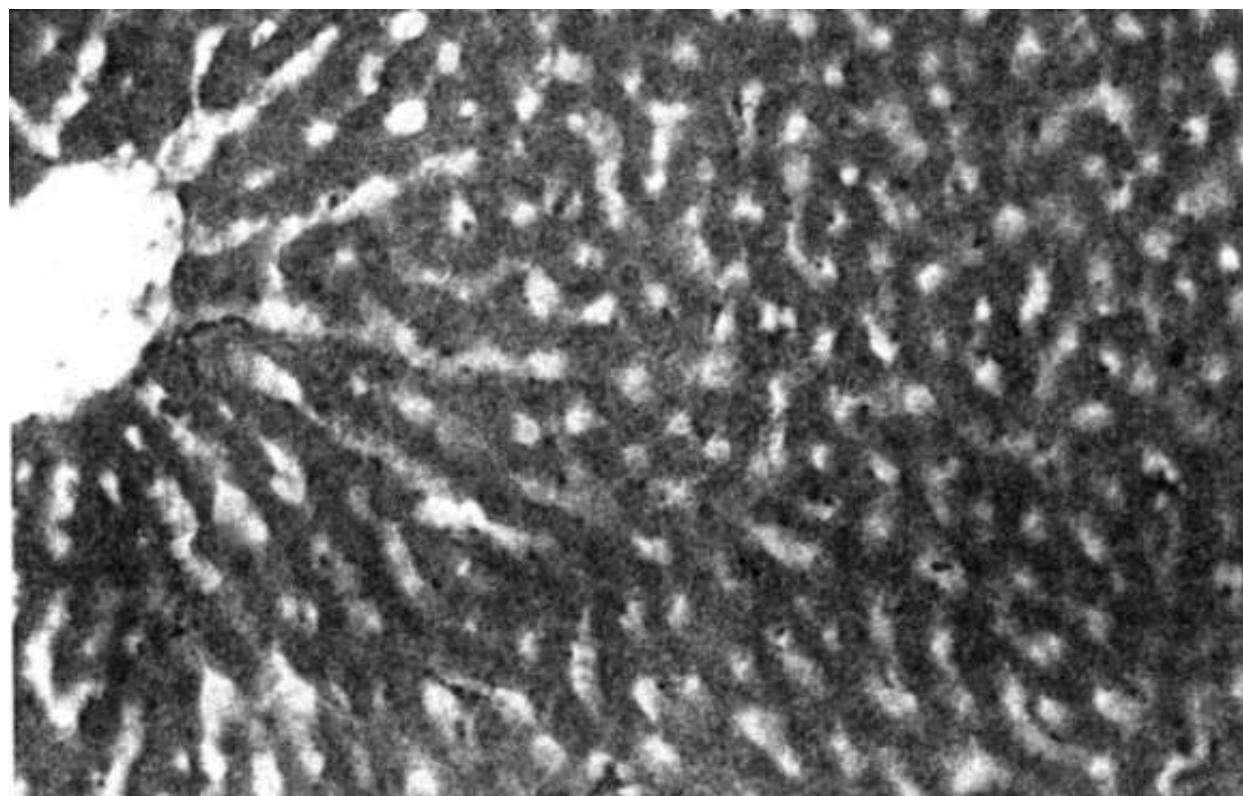
Photograph (2):- section of liver related to selenium treated rabbits (330 x)



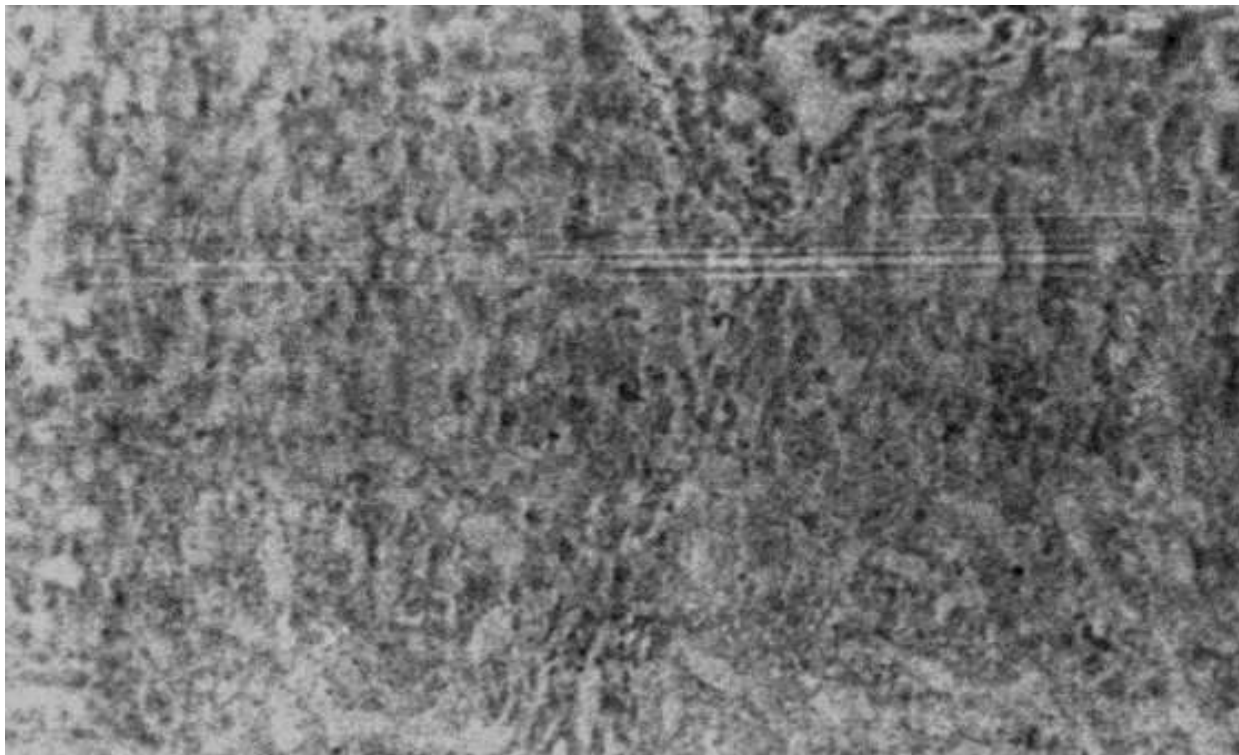
Photograph (3):- section taken from liver of (selenium and vitamin E) treated animals. (330 x)



Photograph (4):- section illustrated the control animal testis (330 x)



Pictograph (5):- showing the effect of selenium on the rabbits testis after (30) days of treatment (660 x)



Photograph (6):- illustrated section of testis treated with selenium and vitamin E (330 x)

Discussion

Livers from reader sunfish which had accumulated approximately 20 ppm selenium in the liver showed central necrosis, reduced quantites of rough endoplasmic reticulum and glycogen particles⁽⁹⁾.

On the other hand, the submicroscopic studies reveald that repeated administration of sodium selenite in rats contributed to chromatin destruction in the hepatocyte nuclei⁽¹⁰⁾. selenium may be like cadmium which caused acute necrosis and damage in rabbits liver⁽¹¹⁾, ⁽¹²⁾found that no hepatocellular damage, portal fibrosis and inflammatory infiltration were found in the selenium and vitamin E group in comparison with cholesterol fed group , it was noticed that this group has the same histological

appearance as the control group. As a result, we can say that combined vitamin E and selenium therapy decreases the hepatocellular damages and these antioxidant agents have protective effects on the hepatocytes cells. Histopathological studies of the testes and cauda epididymis in rats have revealed that se-rich diets cause dose-time-dependent reduction in tubular diameter , epithelial height , number of spermatogenic cells and disintegration of cellular associations in the seminif erous tubules of testes along with reduction in the diameter of cauda epididymal tubules and pseudostratification of the epithelial lining⁽¹³⁾.

⁽¹⁴⁾ found that cyclophosphamide induced oxidative stress in the testis, asignificant diminution in the activities of testicular J5,3g - hydroxysteroid dehydrogenase and 17 g -

hydroxysteroid dehydrogenase with reduction in the plasma level of testosterone and number of spermatogonia and step 7 spermatid stage VII of spermatogenic cycle in mature albino rats, while vitamin E administration resulted as significant restoration of all the above parameter to the control level.

References

- (1)Sheehan, T.M.T. and Halls, D.J. (1999) .Measurement of selenium in clinical specimens. Ann. Clin. Biochem. 36: 301-31.
- (2)Schrauzer, G.N. (2000). Anticarcogenic effects of selenium. Cell. Mol. Life. Sci. 57(13-14):1864-1873.
- (3)Sappington, G.K. (2002).Development of aquatic life criteria for selenium: a regulatory perspective on critical issues and research needs. Aquatic Toxicology 57: 101-113.
- (4)Ruta DA, Hader S. Attempted murder by selenium poisoning. BMJ1989; 299:316-317 .
- (5)Garcia, H.J. Glem, E.Artola, J. And Baumgarer, P.J. (2000) .Bioaccumulation of selenium in the Cienga desanta ciao wetland Sonora Mexico. Envir. Saf 46. (3) 298-304 .
- (6)Davis, J. G.; stefleins T.J.; Engle T.E.; Mallow, K.L. and CoHon , S.E.(2001). Preventing selenium toxicity, Colorado state university cooperative extension 11/100 No 6.10.
- (7)Kolb, E. (1982) recent nutrition biochemical findings on the importance of vitamin E and selenium in man and animal .Z.Gesmate. Lnn. Med. 37(2): 37-42. Selenium in man and animal .Z. Gesmate. Lnn. Med. 37(2): 37-42 .
- (8)Luna, L.G.(1968).Manual of histological staining methods of the armed forces institute of pathology. 3rd ed. Mc Graw-Hill book co. London .
- (9)Sorensen, E.M;Bell,J. S; Harlan, C. W. (1983). Histopathological changes in selenium exposed fish. Am.J.Forensic. Med. Pathol. 4(2): 111-123.
- (10)Chmielnicka, J; Hajdu kiwicz, Z ; Komsta szumsk , E ; Lukaszek , S. (1978). Whole - body retention of mercury and selenium and histopathological and morphological studies of kidneys and liver of rats exposed repeatedly to mercuric chloride and sodium selenite. Arch. Toxicol. 40 (3): 189-199 .
- (11)Dudley, R.E.; Gammal , L. M. & Klassen, C.D. (1985). Cadmium-induced hepatic and renal in Juryin chronically exposed rats: likely role of hepatic cadmium-metallothionein in nephrotoxicity. Toxicol. Appl. Pharmacol. 15(3): 414-421 .
- (12)Suheyyla Gonca, Surveyya ceylan, Melda Yardimo GLu, Hakki Dalcik, Zuhul YamBul. (2000). Histopathological effects of cholesterol and protective effects of

- vitamin E and selenium on the morphology of liver. Turk.J.Med .Sci.30: 551- 555.
- (13)Kaur, R; Kaur, K. (2000). Effects Of dietary selenium on morphology of testis and cauda epididymis in rats. Indian. J.Physiol. Pharmacol. 44(3): 265- 272 .
- (14)Delidas Ghosh, Vjjal Baran Das, Manomohan Misro. (2002). protective role off - tocopherrol-succinate (provitamine E) in cyclophosphamide induced testiculargametogenic and steroidogenic disorder: Acorrelative Approach to oxidative stress. Taylor and Francis Group - Article. 36 (11): 1209 – 1218.

التأثير الامراضي النسيجي لمادتي السيلينيوم وفيتامين E اعلى كبد وخصى الارانب المحلية

عدنان عيسى البدران ، اقبال عبدالعزيز الرفيعي ،

هناك سلمان السوداني

جامعة البصرة - كلية العلوم - قسم علوم الحياة

الخلاصة

أظهرت الدراسة النسيجية لكبد وخصى الأرانب المحلية المعاملة وبالسيلينيوم والسيلينيوم مضاف إليه فيتامين E وحيوانات السيطرة ان السيلينيوم كان له تأثير واضح على نسيج الكبد حيث يحدث النلف في الخلايا الكبدية والتنخر Necrosis مع وجود الارتشاح الدموي Blood infiltration في الخلايا الدفاعية مقارنة مع نسيج الكبد في حيوانات السيطرة حيث تترتب وتنظم الخلايا الكبدية بشكل صفائح كبدية حول الوريد المركزي. Central vein.

اما عند إضافة فيتامين E إلى مادة السيلينيوم وتجريتها للأرانب فيظهر هناك نقصان واضح في تأثير السيلينيوم على حصول التنخر في الخلايا الكبدية وتظهر مقارنة في الشكل لأكباد حيوانات السيطرة .

اما بالنسبة لتأثير اسيلينيوم على الخصى ، فقد حدث التلف الكامل في المراحل المكونة للنطف مع وجود بعض الخلايا المولدة للنطف والتي تترتب محيطياً ضمن النبيب المنوي . أما الزيادة النسبية في عدد الخلايا المولدة للنطف فقد حصلت نتيجة لتأثير فينامين E المضاف إلى مادة السيلينيوم.