

Role of alcoholic extract of Rokat (*Eruca sativa*) leaves on male reproduction of experimentally induced-oxidative stressed rats

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Summary

This study conducted to find out the protective role of ethanolic extract of *Eruca sativa* leaves against the deleterious effect of hydrogen peroxide on some aspect of male reproduction in adult rats. Forty adult male rats were randomly assigned into four equal groups as follows: control group received tap water (untreated); group T₁ were received tap water containing 0.5% H₂O₂; group T₂ were received tap water containing 0.5% H₂O₂ plus administration of 300 mg/kg. B.W. ethanolic extract of *Eruca sativa* leaves and group T₃ administration ethanolic extract of *Eruca sativa* leaves only at the same dose of group T₂. All treatments continued for 60 days. At the end of the experiment, samples of testes and epididymis tissues were taken to prepare histological sections for measurement the diameter of seminiferous tubules, thickness of epithelial cells of seminiferous tubules and histological examination of testes and epididymis. The results in Group T₁ showed a significant decrease in the diameter and thickness of epithelial cells of seminiferous tubules, but these parameters clarified a significant increase in T₂ and T₃ groups as compared with T₁ group. Histological sections of testis and epididymis in group T₁ revealed incomplete spermatogenesis, cell debris, vacuolation of Sertoli cells and view sperms in the lumen of seminiferous tubules and epididymis. Besides, normal obvious histological architecture of seminiferous tubules and epididymis with complete spermatogenesis were shown in sections of testis and epididymis of T₂ and T₃ groups' as compared to T₁. In conclusion, hydrogen peroxide may impair spermatogenesis, furthermore, the results confirm the protective role of *E. sativa* leaves extract against oxidative stress induced by H₂O₂ in rats.

Keywords: Hydrogen peroxide, *Eruca sativa* leaves, Testis, Epididymis, Seminiferous tubules.

Introduction

A considerable amount of literature has been published about the oxidative stress (OS) and reactive oxygen species (ROS) play an important role in the etiology and/or progression of a number of human diseases (1). Under pathological conditions, the uncontrolled production of ROS exceeds the antioxidant capacity of the seminal plasma, resulting in oxidative stress which play an important role in male infertility by causing sperm dysfunction (2 and 3). ROS plays a crucial role in several reproductive development and maturation (4 and 5). When the level of ROS exceeds above the normal, it could damage spermatozoa by inducing lipid peroxidation (LPO) on DNA damage (6 and 7) and are associated with poor sperm function (8). The common causes of male infertility include varicocele, genital tract infection, radiation, chemotherapy, erectile dysfunction, gene mutations and aneuploidy (9 and 10). Rocket (*Eruca sativa*) has a wide spread

medicinal use; it is considered as a medicinal plant with many reported properties, including its strong aphrodisiac effect known since Roman times (11 and 12). Traditionally, it is used as astringent, diuretic, digestive, emollient, tonic, depurative, laxative (13 - 15), antimicrobial (16), antihyperlipidemic, antihyperglycemic and antiophroethiatic (17) and commonly used food additive to improvement rumen degradation (18). Other researchers (15) explained that rocket contain a group of anticancer compounds known as glucosinolates; these compounds exert antioxidant activity (19 - 21). Whereas, (22 and 23) proved that the presence of saponine and alkaloids in rocket extract would cause a significant increase in sperm activity. Some evidence suggested that the ethanolic extract of *Eruca sativa* plant has androgenic activity or stimulate testicular steroid production which increases spermatogenesis in male mice (24). Therefore, the objective of this experiment aimed at investigating the

association of preventive role of ethanolic extract of *Eruca sativa* leaves with oxidative stress induced by H₂O₂ on some aspect of male reproduction in adult rats.

Materials and Methods

The fresh leaves of *Eruca sativa* was purchased from the local market of Baghdad. The fresh leaves of *Eruca sativa* were dried by air, grounded into a fine powder grinder weighing 100 gm then put it in a volumetric conical flask 1000 ml of 70 % Ethyl alcohol was added on the powder, after that the mixture was shaken by using magnetic stirrer apparatus for 24hr, the mixture was filtered and then was filtered again using Whatman (No.1) filter paper. The filtrated mixture was concentrated by using incubator on 40°C for 72hr. The yield equal of 10 gm., the extract was stored in a dark sterile screw bottle at 4°C until used (25).

Forty healthy adult males Wistar rats, weighed 200-275 gm. were used and housed in an animal house/ Department of Physiology and Pharmacology/ College of Veterinary Medicine/ Baghdad University. The animals were kept at 22 -25°C, with 12h light/dark cycle. Animals were allowed freely access to water and pellets along the experimental period. After acclimatization two weeks, rats were randomly divided into four groups (10 rats each) as follows: Control group, animals in this group received tap water (untreated); group T₁ received tap water contain 0.5% H₂O₂ (26); group T₂ received tap water containing 0.5% H₂O₂ and administrated orally 300 mg/kg. B.W. ethanolic extract of *Eruca sativa* leaves and group T₃ received ethanolic extract of *Eruca sativa* leaves only at the same dose of group T₂. All treatments continued for

60 days. At the end of the experiment, animals were sacrificed and samples of testes and epididymis tissues were taken and fixed in neutral formalin solution. Histological sections were prepared at 5 µm and stained with hematoxylin and eosin according to (27) for histopathological examination of testis and epididymis including: measurement of diameter of seminiferous tubule, thickness of epithelial cells of seminiferous tubules and histopathological examination of testes and epididymis has been carried out with image (Java-based image processing program developed at the National Institutes of Health). Analysis of data was carried out using One-Way Analysis of Variance (ANOVA) followed by LSD (28).

Results and Discussion

The mean value of thickness and diameter of seminiferous tubules in testes of four experimental groups were illustrated in (Table, 1). The histological sections of rat testes received 0.5% H₂O₂ (Group T₁) exhibited a significant (P<0.05) decrease in thickness of epithelial cells and diameter of seminiferous tubules as compared to control, T₂ and T₃ groups. Also the increase of space between the seminiferous tubules was observed in T₁. Whereas, in H₂O₂ – *Eruca sativa* treated (T₂) group there was a significant (P<0.05) remarkable elevation in thickness of germ cells and diameter of tubules compared to T₁. Moreover, the values of T₂ tended to normalize that of the control. While, in the group T₃, the appearance of all seminiferous tubules was normal, healthy and there were no differences observed between animal at T₃ and control as compared to each other.

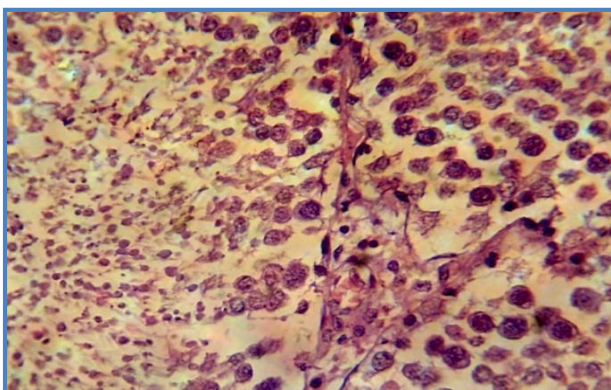
Table, 1: Effect of *Eruca sativa* extract and hydrogen peroxide on histological architecture of seminiferous tubules in adult male rats.

parameter	Control group	Group T ₁ (H ₂ O ₂)	Group T ₂ (H ₂ O ₂ + ES)	Group T ₃ (ES)
Thickness of epithelial Cell of seminiferous tubules (mm)	2.84±0.14 A	1.90±0.17 B	2.51±0.28 A	3.31±0.21 A
Diameter of seminiferous tubules (mm)	8.99±0.34 A	6.25±0.22 B	8.30±0.30 A	10.66±0.41 A

Capital letters denote differences between groups, P<0.05.

Histological sections obtained from rat testis exposed to 0.5% hydrogen peroxide (group T₁) at the end of the experiment revealed decrease in the thickness of basement membrane of seminiferous tubules (Table, 1 and Fig. 1), incomplete spermatogenesis, cell debris, vacuolation of Sertoli cells and sperms in the lumen of seminiferous tubules (Fig. 2) comparing to control group (Fig. 3), while testes of rats treated with 0.5% H₂O₂ plus ethanolic extract of (300 mg/kg B.W) *E. sativa* (T₂) showed complete spermatogenesis with presence of large number of sperms in the lumen of seminiferous tubule and active Leydig cells (Fig. 4). Normal obvious histological architecture of seminiferous tubules and complete spermatogenesis were shown in sections of testis of group T₃ (Fig. 5), as well as, the absence of main differences between seminiferous tubules morphology and thickness of basement membrane in comparison to control (Table, 1 and Fig. 1).

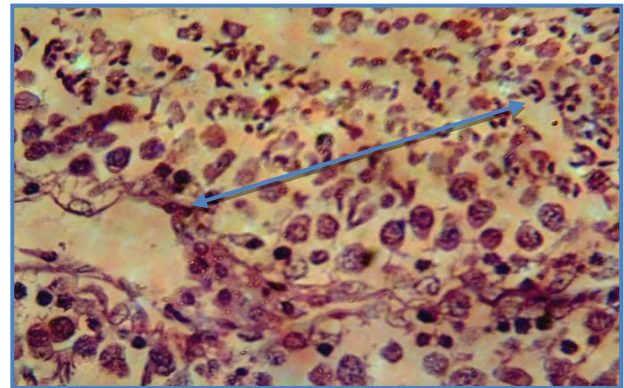
Rats of group T₁ showed few sperms in the lumen of epididymis with round dark nuclei cells (Fig. 6) as compared to control group (Fig. 7). Whereas, the epididymis of group T₂ showed normal structure and the lumen filled with sperms (Fig. 8 and 9) as compared with control group (Fig. 7). Meanwhile, the histopathological sections of epididymis of group T₃ showing normal structure and the lumen compact with sperms (Fig.10) comparing to group T₁ (Fig. 6).



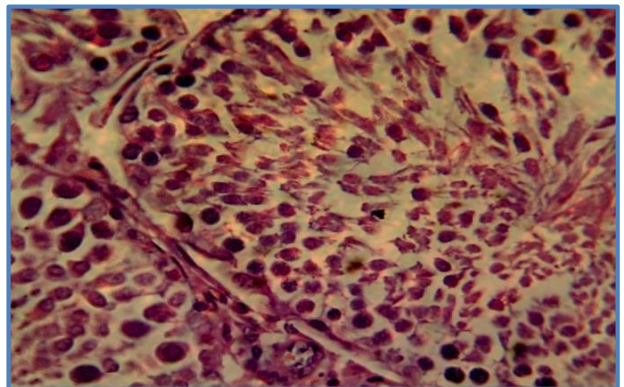
Figure, 1: Cross section of the rat testes group T1 showing decrease in thickness of basement of seminiferous tubules with in complete spermatogenesis (H and E stain 40 x).

Exposure of rats to hydrogen peroxide (group T₁) results in the appearance of thickness of basement membrane of seminiferous tubules (Table, 1) with

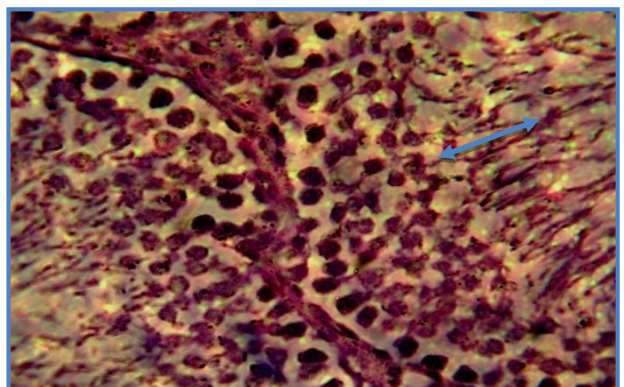
incomplete spermatogenesis and cellular debris (Fig. 2 and 4) as well as few sperms in the lumen of epididymis with round nuclei cells (Fig. 8). It has been proven that H₂O₂ induces oxidative stress in animal models, by forming potent ROS and nitrogen–oxygen species (29 and 30).



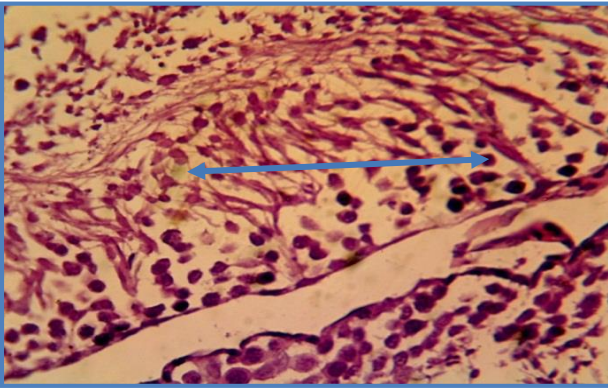
Figure, 2: Cross section of the rat testes of group T1 showing incomplete spermatogenesis with cellular debris in the lumen of seminiferous tubules (H and E stain 40 x).



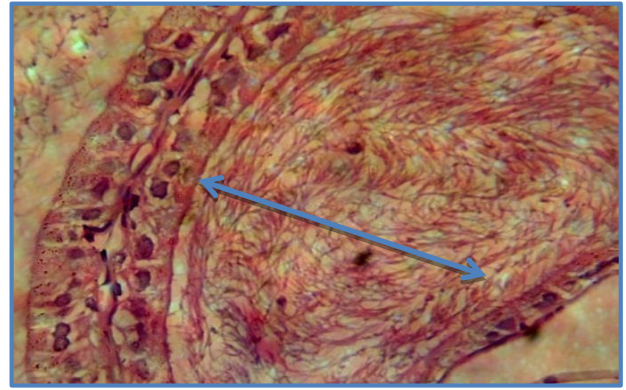
Figure, 3: Cross section of the rat testes control group showing normal structure of seminiferous tubule (H and E stain 40x).



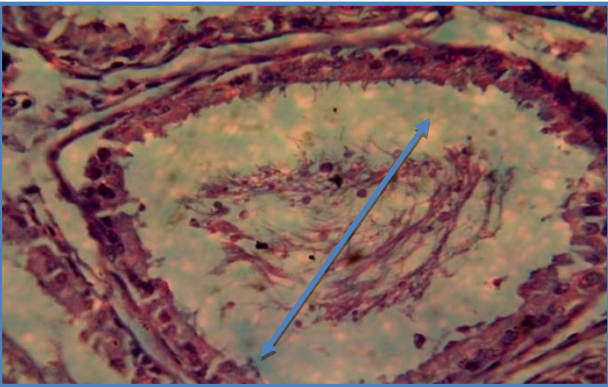
Figure, 4: Cross section of the rat testes of group T2 showing complete spermatogenesis with presence large number of sperm in the lumen of seminiferous tubules (H and E stain 40x).



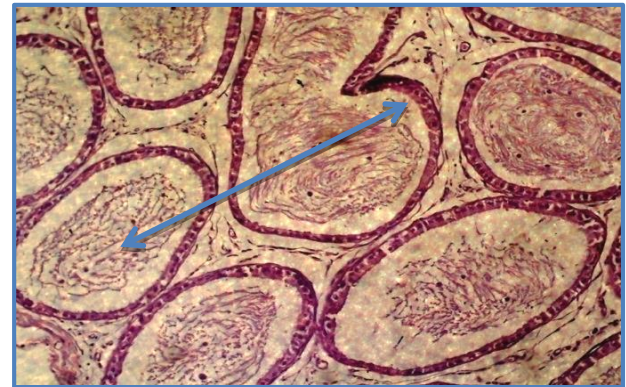
Figure, 5: Cross section of the rat testes of group T3 showing complete spermatogenesis (H and E stain 40x).



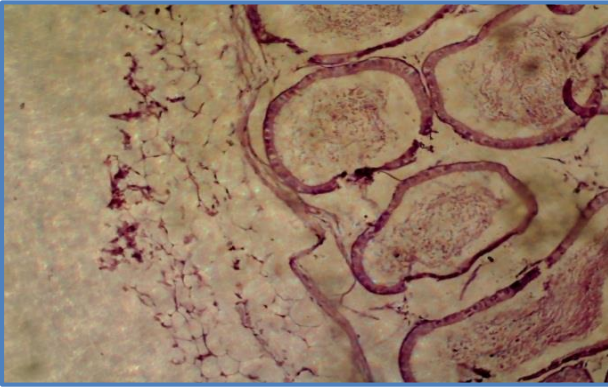
Figure, 9: Cross section of rat epididymis of group T2 showing the lumen was overfilled with sperm (H and E stain 40x).



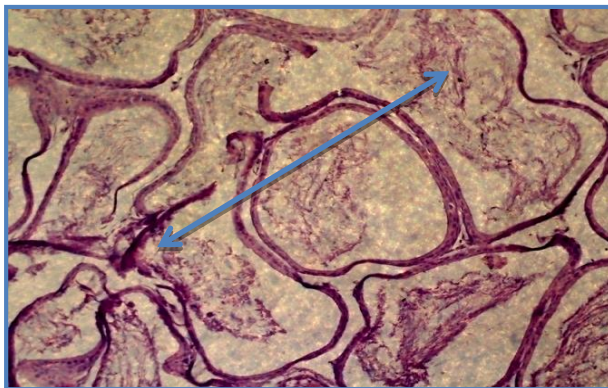
Figure, 6: Cross section of rat epididymis of group T1 showing few sperm in the lumen with round dark nuclei (H and E stain 40x).



Figure, 10: Cross section of rat epididymis of group T3 showing the lumen filled with sperm (H and E stain 40x).



Figure, 7: Cross section of the rat epididymis of control group showing normal structure (H and E stain 10x).



Figure, 8: Cross section of rat epididymis of group T2 showing the lumen was overfilled with sperm (H and E stain 10x).

Oxidative stress augmented production of ROS overwhelms the body's antioxidant defenses (31-33). Therefore, the increase in thickness of basal membrane in seminiferous tubules which accompanied with incomplete spermatogenesis may be due to an increase in production of ROS that damage normal spermatozoa by inducing LPO and DNA damage (6 and 34). Several investigators showed that severe oxidative stress would causes damage to DNA, proteins, and enzymes, including lipid peroxidation enzymes. Such degeneration leads to cell death (35-37) thereby demonstrating the pathological role of ROS on the male reproductive system (38 and 39). Research suggested that the oxidation of the proteins leads to the loss of function or to the degradation in the peroxisomes, whereas the lipid peroxidation affects the biological function of the membrane. However, the most serious damage is detected at DNA level, as this may lead to mutation of genes, inducing translation of defective proteins, in addition to alteration in

gene expression and eruption of apoptosis (40).

Whereas, other studies (37 and 41) explained that the most of the deleterious effects of H₂O₂ on tissues, including lipid peroxidation, depend on the conversion of the compound into OH•, which is catalyzed by iron and copper through the Fenton reaction in cells.

Furthermore, (42 and 43) explained that testicular oxidative stress, will lead to an increase in germ cell apoptosis and subsequent hypospermatogenesis. Such stress conditions can cause changes in the dynamics of testicular microvascular blood flow, endocrine signaling, and germ cell apoptosis. Many investigators have demonstrated that testicular testosterone production is acutely reduced in a number of conditions associated with ROS production and oxidative stress in the testis (42, 44 and 45). It is also true that steroidogenesis itself produces ROS, largely from mitochondrial respiration and the catalytic reactions of the steroidogenic cytochrome P450 enzymes (46-48). The ROS produced by spermatogenesis, if unchecked by intracellular antioxidants, can also damage mitochondrial membranes and contribute to the inhibition of subsequent steroid production (49).

From the data obtained in the current study, oral administration of *Eruca sativa* leaves extract for 60 days to male rats in groups T₂ show an ameliorating the histopathological changes in testis and epididymis which is created by H₂O₂. Meanwhile, testis and epididymis of rats in group T₃ orally administrated *Eruca sativa* extract show the same histological architecture as in the control group which was compatible with (50) who explained that low dose of *Eruca sativa* seed oil caused an increase the diameter of seminiferous tubules, may be due to the high rate of proliferation of haploid cell resulting in stimulation of spermatogenesis and increase sperm count. It has been proven that *Eruca sativa* leaves extract may be capable in improving healing sperm parameter and fertility with increasing the diameter of seminiferous tubules (51). As explained earlier, the presence of bio-active isothiocyanates (ITCs) in ES extract which results from glucosinolate upon myrosinase

hydrolysis (15) have antibacterial properties and induce metabolizing enzymes such as glutathione-s-transferase, NADPH, which play an important role in detoxification and protection against oxidative stress (16 and 52).

The results of the current study ascertained that ethanolic extract of *E. sativa* leaves alleviated the harmful effect of oxidative stress induced by H₂O₂. Thus, it can be suggested that the component of *E. sativa* leaves extract have the potential to improve male reproductive functions attributed to its antioxidant and androgenic properties.

References

1. Dalle-Donne, I.; Scaloni, Giustarini, A.; Cavarra, D.; Tell, E. and Lungarella, G. (2005). Proteins as biomarkers of oxidative stress in diseases: the contribution of redox proteomics, Mass Spectrom Rev., 24: 55-99, 2005.
2. Henkel, R. R. (2011). Leukocytes and oxidative stress: dilemma for sperm function and male fertility. Asian J. Androl., 13: 43-52.
3. Trussell, J. C. (2013). Optimal diagnosis and medical treatment of male infertility. Semen Reprod. Med., 31: 235-6.
4. Grriveau, J. F. and Le Lannou, D. (1997): Reactive oxygen species and human spermatozoa: physiology and pathology. Int., J. Androl., 20: 61-69.
5. Agarwal, A. and Said, T. M. (2003). Role of sperm chromatin abnormalities and DNA damage in male infertility. Hum. Reprod. Update. 9: 331-45.
6. Alvarez, J. G.; Sharma, R. K.; Ollero, M.; Saleh, R. A. and Lopez, M. C. (2002). Increased DNA damage in sperm from leukocytospermic semen samples as determined by the sperm chromatin structure assay. Fertil Steril., 78: 319-329.
7. Saleh, R. A.; Agarwal, A.; Kandirali, E.; Sharma, R. K. and Thomas, A. J. (2002). leukocytospermic is associated with increased reactive oxygen species production by human spermatozoa. Fertil. Steril., 78:1215-1224.
8. Pasqualotto, F. F.; Sharma, R. K.; Nelson, D. R.; Thomas, A. J. and Agarwal, A. (2000). Relationship between oxidative stress, semen characteristic, and clinical diagnosis in men undergoing infertility investigation. Fertil. Steril., 73: 459-464.

9. Venkatesh, S.; Gupta, N. P.; Kumar, R.; Deccaraman, M. and Dada, R. (2009). Correlation of sperm morphology and oxidative stress in infertile men. *Iran J. Reprod. Med.*, 7: 29-34.
10. Hamada, A.; Esteves, S. C. and Agarwal, A. (2011). Unexplained male infertility: potential causes and management. *Hum. Androl.*, 1: 2-16.
11. Padulosi, S. and Pignone, D. (1997). Rocket : Mediterranean crop for the world . International plant genetic resources institute, Rome, Italy.
12. Font, R.; Galan, S.; Ruiz, P.; Villatoro, P. and Delrio, C. (2003). Characterization of the sensorial, morphological and agronomic attributes of a world collection of rocket Brassica, 5th international symposium on brassica and the 16th crucifer genetic work shop.
13. Alam, M.; kaur, G.; Jabbar, Z.; Javed, K. and Athar, M. (2006). *Eruca sativa* seeds possess antioxidant activity and exert a protective effect on chloride mercuric induced renal toxicity *Food Chem. Toxicol.*, 29 :172.
14. Al-Qurainy, F.; Alameri, A. A. and Khan, S. (2010). Rapid profile for the assessment of genotoxicity on a medicinal plant *Eruca sativa*. *J. Med. Plants. Res.*, 4(7): 579-586. 920.
15. Al-Qasoumi, S.; Al-Sohaibani, M.; Al-Howiriny, T.; Al-Yahya, M. and Rafatullah, S. (2009). Rocket "*Eruca sativa*": a salad herb with potential gastric anti-ulcer activity. *World J. Gastroenterol.*, 15(16): 1958-1965.
16. Khoobchandani, M.; Ojeswi, B. K.; Ganesh, N.; Srivastava, M. M. and Gabbanini, S. (2010). Antimicrobial properties and analytical profile of traditional *Eruca sativa* seed oil: Comparison with various aerial and root plant extracts. *Food Chem.*, 120: 217-224.
17. Bukhashi, E.; Maliki, S. A. and Ahmed, S. S. (2007). Estimation of nutritional value and trace elements content of *Carthamus oxycantha*, *Eruca sativa* and *Plantago ovanta*. *Pak. J. Bot.*, 30(4): 1181-1187.
18. Kazemi, M.; Tahmasbi, A. M.; Naserian, A. A.; Valizadeh, R. and Moheghi, A. (2012). Potential nutritive value of some forage species as ruminants feed in Iran. *African J. Biotech.*, 11(57): 12110-12117.
19. Kim, S. J.; Jin, S. and Ishii, G. (2004). Isolation and structural elucidation of 4-(beta-D-glucopyranosylsulfanyl) butyl glucosinolate from leaves of rocket salad (*Eruca sativa* L.) and its antioxidative activity. *Biosci. Biotechnol. Biochem.*, 68: 2444-2450.
20. Yehuda, H.; Khatib, S.; Sussan, I.; Musa, R.; Vaya, J. and Tamir, S. (2009). Potential skin anti-inflammatory effects of 4- methylthiobutylisothiocyanate (MTBI) isolated from rocket (*Eruca sativa*) seeds. *Biofactors*, 35(3): 295-305.
21. Michael, H. N.; Shafik, R. E. and Rasmy, G. E. (2011). Studies on the chemical constituents of fresh leaf of *Eruca sativa* extract and its biological activity as anticancer agent in vitro. *J. Medicinal Plants Res.*, 5(7):1184-1191.
22. Martinez-Sanchez, A.; Liorach, R.; Gil, M. I. and Ferreres, F. (2007). Identification of new flavonoid glycosides and flavonoid profiles to characterize rocket leafy salads. *J. Agric. Food Chem.*, 55(4): 1356-1363.
23. Barillari, J.; Canistro, D.; Paolini, M.; Ferroni, F. and Pedulli, G. F. *et al.*, (2005). Direct antioxidant activity of purified glucoerucin, the dietary secondary metabolite contained in rocket (*Eruca sativa* Mill.) seeds and sprouts. *J. Agric. Food Chem.*, 53: 2475-2482.
24. Homady, M. H.; Hussain, H. H.; Tarawaneh, K. A. and Shakhanbeh, J. M. (2000). Effects of medicinal plant extracts used in Jordan on social aggression as well as testicular and preputial gland structure in male mice. *J. Bio. Sci.*, 3(3): 389-402.
25. Jin, J.; Koroleva, O. A.; Gibson, T.; Swanston, J. and Magan, J. *et al.*, (2009). Analysis of phytochemical composition and chemoprotective capacity of rocket (*Eruca sativa* and *Diplotaxis tenuifolia*) leafy salad following cultivation in different environments. *J. Agric. Food Chem.*, 57(12): 5227-5234.
26. Khudier, K. K. (2000). The role of aqueous extraction of Olive leaves (*Olea europaea* L.) and Garlic (*Allium sativum* L.) in ameliorating the effect of experimentally induced atherosclerosis in rats. Ph.D. Thesis/ College of Veterinary Medicine/ University of Baghdad.

27. Luna, L. G. (1968). Manual of Histology Staining. Methods of Armed Forces. Institute of Pathology. 3rd edition. McGraw-Hill Book Company, New York and London.
28. Snedecor, G.W. and Cochran, W. G. (1973). Statistical Methods. 6th ed. the Iowa state University press., Pp:238-248.
29. Imlay, J. A.; Chin, S. M. and Linn, S. (1988). Toxic DNA damage by hydrogen peroxide through the Fenton reaction *in vivo* and *in vitro*. *Sci.*, 240: 640–642.
30. Sisein, E. A.; Ayakeme, T.; Ebiagbe Ebiere, J. and Adeg, A. A. (2013). Melatonin and vitamin E reduces the effect of H₂O₂ induced oxidative stress in male albino rats *in vitro*. *J. Biol. Food Sci. Res.*, 2(8): 92-96.
31. Cardenas-Rodriguez, N.; Huerta-Gertrudis, B.; Rivera-Espinosa, L.; Montesinos-Correa, H. and Bandala, C. *et al.*, (2013). Role of oxidative stress in refractory epilepsy: Evidence in patients and experimental models. *Int. J. Mol. Sci.*, 14: 1455-1476.
32. Ruttkay-Nedecky, B.; Nejd, L.; Gumulec, J.; Zitka, O. and Masarik, M. *et al.*, (2013). The role of metallothionein in oxidative stress. *Int. J. Mol. Sci.*, 14: 6044-6066.
33. Agarwal, A.; Virk, G.; Ong, C.; Stefan, S. and du Plessis (2014). Effect of oxidative stress on male reproduction. *World J. Mens Health.*, 32(1): 1-17.
34. Aitken, R. J.; Gordon, E.; Harkiss, D.; Twigg, J. P. and Milne, P. (1998). Relative impact of oxidative stress on the functional competence and genomic integrity of human spermatozoa. *Biol. Reprod.*, 59:1037-1046.
35. Ward, J. F.; Blakely, W. and Jone, E. I. (1985). Mammalian cells are not killed by DNA single-strand breaks caused by hydroxyl radicals from hydrogen peroxide. *Radiation Res.*, 103: 383–392.
36. Sharma, R. K. and Agarwal, A. (1996). Role of reactive oxygen species in male infertility. *Urology*, 48: 835–85.
37. Ganie, S. A.; Haq, E.; Hamid, A.; Masood, A. and Zargar, M. A. (2011). Long dose exposure of hydrogen peroxide (H₂O₂) in albino rats and effect of *Podophyllum hexandrum* on oxidative stress. *Eur. Rev. Med. Pharmacol. Sci.*, 15: 906–915.
38. Agarwal, A.; Saleh, R. A. and Bedaiwy, M. R. (2003). Role of reactive oxygen species in the pathophysiology of human reproduction. *Fertil. Steril.*, 79: 829-843.
39. Agarwal, A.; Gupta, S. and Sikka, S. (2006). The role of free radicals and antioxidant in reproduction. *Curr Opin Obstet Gynecol.*, 18(3): 325-32.
40. Sorg, O. (2004). Oxidative stress: A theoretical model or a biological reality? *Biol.*, 327: 649–662.
41. Park, K. J.; Kim, Y. J.; Kim, J.; Kim, S. M. and Bae, J.W. *et al.*, (2012). Protective effects of peroxiredoxin on hydrogen peroxide induced oxidative stress and apoptosis in Cardiomyocytes. *Korean Circ. J.*, 42: 23–32.
42. Turner, T. T. and Lysiak, J. J. (2008). Oxidative stress: a common factor in testicular dysfunction. *J. Androl.*, 29: 488–498.
43. Rahim, S. M.; Taha, E. M.; Mubark, Z. M.; Aziz, S. S. and Simon, K. D. *et al.*, (2013). Protective effect of *Cymbopogon citratus* on hydrogen peroxide-induced oxidative stress in the reproductive system of male rats. *Syst. Biol. Reprod. Med.*, 59(6): 329-336.
44. Ismail, H. and Al-Nahari, H. (2009). Therapeutic and protective role of *Panax ginseng* on pituitary and testicular axis in male rats treated with carbon tetrachloride. *Ale. J. Agric. Res.*, 54(1):1-12.
45. Tijani, A. S.; Ukwenya, V. O.; Sodunke, G. A. and Fakunle, J. B. (2010). Acute administration of co-artesiane® induces oxidative stress in the testes of adult male Wistar rats. *Biosci. Res. Commun.*, 22(5): 259-265.
46. Peltola, V.; Huhtaniemi, I.; Metsa-Ketela and Ahotupa, M. (1996). Induction of lipid peroxidation during steroidogenesis. *Endoc.*, 137: 105–112.
47. Hales, B. D. (2002). Testicular macrophage modulation of Leydig cell steroidogenesis. *J. Reprod. Immunol.*, 57:3–18.
48. Hanukoglu, I. (2006). Antioxidant protective mechanisms against reactive oxygen species (ROS) generated by mitochondrial P450 systems in steroidogenic cells. *Drug. Meta. Rev.*, 38: 171–196.
49. Luo, L.; Chen, H.; Trush, M. A.; Show, M. D. and Anway, M. D. *et al.*, (2006). Aging and the Brown Norway rat Leydig cell antioxidant defense system. *J. Androl.*, 27: 240–247.

50. Salem, M. A. R. and Moustafa, N. A. (2001). Histological and quantitative study of the effect of *Eruca sativa* seed oil on the testis of albino Rat. Egyptian J. Hosp. Med., 2: 148-162.
51. Hussein, Z. F. (2013). Study the effect of *Eruca sativa* leaves extract on male fertility in albino mice, J. Al-Nahrain Uni., 16(1): 143-146.
52. Fahey, J. W. and Talalay, P. (1999). Antioxidant function of sulphoraphane: a potent inducer of phase II detoxication enzymes. Food Chem. Toxicol., 37: 973-979.

دور المستخلص الكحولي لأوراق نبات الجرجير في الجهاز التكاثري لذكور الجرذان المستحدث فيها الكرب التأكسدي تجريبياً

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الخلاصة

هدفت الدراسة إلى التحري عن الدور الوقائي للمستخلص الكحولي لأوراق الجرجير للحد من الأذى المستحدث ببيروكسيد الهيدروجين في كفاءة الجهاز التكاثري لذكور الجرذان البالغة. استعمل 40 من ذكور الجرذان البالغة التي يتراوح معدل أوزانها 200-275غم وقسمت عشوائياً إلى أربعة مجاميع متساوية: المجموعة الأولى والتي أعطيت ماء الحنفية وعدت كمجموعة سيطرة، المجموعة الثانية (T₁) أعطيت ماء الحنفية الحاوي على 0.5% بيروكسيد الهيدروجين، أما المجموعة الثالثة (T₂) فقد أعطيت ماء الحنفية الحاوي على 0.5% بيروكسيد الهيدروجين مع تجريعها 300ملغم/كغم من وزن الجسم من المستخلص الكحولي لأوراق نبات الجرجير، المجموعة الرابعة (T₃) جرعت 300ملغم/كغم من وزن الجسم من المستخلص الكحولي لأوراق نبات الجرجير. وعملت جميع المجاميع لمدة 60 يوماً وفي نهاية التجربة ضُحي بالحيوانات وأخذت عينات من الخصية والبربخ لدراسة التغيرات النسجية فضلاً عن قياس الخلايا المبطنة للنبيبات مع أقطار النبيبات المنوية. أوضحت نتائج القياسات النسجية لمقاطع الخصى والبربخ لذكور الجرذان للمجموعة المعاملة ببيروكسيد الهيدروجين (T₁) انخفاضاً معنوياً في معدل أقطار النبيبات المنوية وسمك الخلايا الطلائية لها، لكن هذه القياسات أظهرت ارتفاعاً معنوياً في سمك الخلايا الطلائية ومعدل اقطار النبيبات المنوية في مجموعتي T₂ و T₃ بالمقارنة مع مجموعة T₁. في حين لم يلاحظ وجود فرق بين مجموعة T₃ مقارنة بمجموعة السيطرة. كما أشارت التغيرات النسجية لمقاطع الخصى والبربخ لذكور الجرذان المعاملة ببيروكسيد الهيدروجين (T₁) إلى قلة عملية تصنيع النطف مع وجود فجوات واسعة بين النبيبات المنوية، ووجود حطام الخلايا ووجود عدد قليل من النطف في تجايف النبيبات المنوية والبربخ، في حين أظهرت المجموعتين T₂ و T₃ ارتفاعاً معنوياً لعملية تصنيع النطف وأمتلاء التجايف بالنطف مقارنة مع المجموعة T₁. تستنتج الدراسة، إن بيروكسيد الهيدروجين يضعف عملية تخليق النطف فضلاً عن ذلك، أكدت النتائج الدور الوقائي للمستخلص الكحولي لأوراق الجرجير ضد الأجهاد التأكسدي لذكور الجرذان.

الكلمات المفتاحية: بيروكسيد الهيدروجين، أوراق الجرجير، الخصى، البربخ، النبيبات المنوية.