Journal of Radiation Research and Applied Sciences

J. Rad. Res. Appl. Sci., Vol. 4, No. 1(B), pp.233-244 (2011)

Preventive Efficacy of Fucoidan in Rats Exposed to γ-Rays

S. S. Tawfik[1] and S. F. Salama[2]

[1]Health Radiation Research Department, [2]Zoology Research Department, Faculty of Science, King Sauod Univ. and Radiation Biology Research Department, Radiation Research Division, Atomic Energy Authority, P. O. Box: 29 Nasr City, Cairo, Egypt.

E-mail: Samosoliman@Yahoo.com

Received: 04/10/2010. *Accepted: 21/11/2010.*

ABSTRACT

Fucoidan (FDN); β-1,3-glucan is a natural polysaccharide extracted from brown seaweeds, with a wide variety of biological properties, especially the anti-inflammatory, anti-coagulation and anti-oxidative effects. The objective of this work was to evaluate the radioprotective effect of FDN. The study was performed on Sprague-Dawley rats that were administered during 10 days FDN (100 mg/ kg body wt), rats exposed to 5 Gy γ-rays, rats administered FDN+ γ-rays comparing with control animals. The haemostatic parameters (protein-C, antithrombin-III and tissue-plasminogen activators activities), the haematological parameters: count of blood elements (total leukocytes, neutrophils, lymphocytes) and bone marrow cells (erythroid, lymphoid and myeloid cells) and biochemical parameters: thiobarbituric acid-reactive substances (TBARS) and activities of superoxide dismutase (SOD) and glutathione peroxidase (GSPx) were evaluated. Results showed that γ-rays provoked marked changes in haemostatic markers, haematological parameters and antioxidative enzymes as well as TBARS level. FDN ameliorated the alteration in all tested parameters and markers induced due to exposure to γ-rays. These data demonstrate that FDN might be viewed as a non-toxic potent agent for preventing γ-rays induced cellular hazards. Conclusion, the results may facilitate the development of a new radio protective agent with low toxicity.

Keywords: Fucoidan, haemostasis, blood cells, antioxidant enzymes, rat, γ-rays.

INTRODUCTION

FDN is a heterogeneous sulphated polysaccharide purified from brown algae such as *Fucus vesiculosus* and has a variety of biological effects including mobilization of hematopoietic progenitor cells in man and animals. Recently, it

has been demonstrated that FDN significantly increased the viability of bone cells, which are the main cellular reservoir for the hematopoietic and immune system[1]. FDN contains substantial percentages of L-fucose and sulphate ester-groups. FDN possesses anti-oxidative effects related to its capacity to interact with scavenger receptors on cell membrane and its ability to inhibit the synthesis and release of reactive oxygen species (ROS), as well as promoting their clearance [2].

To protect the host against the harmful effects of radiation exposure, radio protective agents have been developed over several decades [3]. Recent studies have focused on the development of radio protective agents derived from natural sources and that display minimal side effects on normal cells [4]. Some polysaccharides purified from herbs have been shown to have radio protective and immune-stimulating effects on host immune cells [5].

Although the biological functions of FDN in hematopoietic and immune systems have been studied for many years, its radio protective effects on physiological and biochemical alteration induced in irradiated animals have not been elucidated. In this study, we investigated the preventive efficacy of FDN on physiological and biochemical parameters of irradiated rats.

MATERIALS AND METHODS

Irradiation

Irradiation was performed with ^{137}Cesium, γ-radiation source (γ-Cell^{-40} manufactured by the Atomic Energy of Canada, Ltd.) at NCRRT. The dose rate was 0.462 Gy/ min.

Materials and FDN treatment

All reagents used were of analytical grade purchased from Sigma-Alderch Co., USA. Rats received FDN dosage (100 mg/ kg/ body wt) orally for 10 days before whole-body irradiation according to protocol of Wu *et al.* [6]. FDN dissolved in phosphate-buffered saline.

Animals, experimental design and sampling

Sprague-Dawley rats were purchased from the *Laboratory Animal Centre of the Holding Company for Biological Products and Vaccines*, Cairo, Egypt. Rats were housed under normal standardized conditions with free access to food and tap water. They were handled according to the National Academy of Sciences standards [7]. The animals were randomly divided into 4 groups. Group

1: animals were kept as controls and received 0.5 ml of the vehicle via intra-gastric tubes once daily for 10 days. Group 2: Rats received FDN-dosage dissolved in the vehicle once daily for 10 days. Group 3: Rats exposed to single dose (5 Gy) of whole body γ-rays. Group 4: Rats received FDN-dosage for 10 days and after 24 h exposed to 5 Gy of γ-rays.

All treated rats were sacrificed 24 h after end of the treatment by cervical dislocation. Blood samples were withdrawn from the heart, collected in tubes containing EDTA as anticoagulant. For preparing plasma, it was centrifuged (1000x g for 10 min), collected in test tubes with screw caps and stored at -20°C until analyzed.

Analytical methods

Haemostatic parameters, protein-C and antithrombin-III were determined according to Pabinger [8] and Tollefsen [9] using kits from Quimica Clinica Aplicada, Italy and chromogenix, Sweden, respectively. Tissue plasminogen-activators were determined by ELISA-technique according to Holvoet *et al.*[10]. Haematological parameters, total leukocytes count as well as differential leukocytes (neutrophils and lymphocytes) studied in peripheral blood drawn by cardiac puncture by automated blood counter (coulter model T 450 x, Contronics Co., USA). The bone marrow cells were prepared from femur as described by Goldberg *et al.*[11]. Briefly, femoral bone was exposed under aseptic conditions, cells were washed with medium-199, suspended by a syringe with a needle of various diameter, and washed again 2-3 times with medium-199 (centrifuged at 150 x g, for 10 min). Smears of the cells were drawn on clean slides and fixed with methanol for 10 min and stained with May-Granwald Giemsa. At least, 1000 cells were scored from each animal to determine differential elements (erythroid, lymphoid and myeloid cells). Biochemical parameters, lipid peroxidation in plasma was determined as TBARS as described by Yoshioka *et al.*[12]. Activities of SOD and GSPx [13,14] were determined in rats' fresh blood.

Statistical analysis

All results are expressed as mean± SD. Comparisons among groups (n= 10, both sexes in each group) were performed by one-way analysis of variance (ANOVA) followed by the Tukey's post-test, when indicated. The level of statistical significance was set at $p< 0.05$ [15].

RESULTS

No evident changes in protein-C, antithrombin-III and tissue-plasminogen activators activities were observed in FDN-treated rats in comparison with control group as shown in Table 1.

Table (1): Plasma activity of protein-C (µg/dl), antithrombin-lll (µg/dl) and tissue plasminogen activators (ng/ ml) in different groups of animals.

Groups	Protein-C	Antithrombin-lll	Tissue plasminogen
Control	78.7± 6.66**A**	88.7± 8.58**A**	1.13± 0.11**A**
FDN-treated	78.6± 7.11**A**	87.8± 8.32**A**	1.25± 0.13**A**
Irradiated (5 Gy)	51.4± 5.43**B**	53.3± 4.92**B**	2.85± 0.19**B**
FDN+ Irradiated	65.7± 6.12**C**	63.3± 6.22**C**	1.87± 0.16**C**

Groups not sharing common superscripts (A, B .....) differ significantly at p< 0.05 level.

As shown in Table 1, significant decreases in activities of plasma protein-C and antithrombin-III as well as a significant increase in activity of plasma tissue-plasminogen activators were recorded in the γ-irradiated animal group compared with the corresponding control and FDN-treated groups. In protected animal groups, the recovery of the 3 haemostatic parameters was significant, compared with the control, FDN-treated and irradiated group.

Table (2): Total leukocyte, neutrophils and lymphocytes count (x109/ L) in different groups of animals.

Groups	Total leukocytes	Neutrophils	Lymphocytes
Control	8.6 ± 0.79**A**	3.9± 0.32**A**	4.6± 0.44**A**
FDN-treated	8.7± 0.81**A**	3.9± 0.27**A**	4.6± 0.43**A**
Irradiated (5 Gy)	4.6± 0.57**B**	2.1± 0.31**B**	2.3± 0.19**B**
FDN+ Irradiated	6.3± 0.63**C**	3.0± 0.28**C**	3.1± 0.28**C**

Legends as in Table 1.

Table (2) revealed that total leukocyte count was nearly 2-fold decreased after irradiation in comparison with control group and no significant changes were found in rats treated with FDN. The rate of neutrophil and lymphocytes levels in same rats groups was similar to the changes in total leukocyte count.

The recovery of total leukocytes in peripheral blood following FDN-administration before irradiation was significantly high compared with the irradiated group. Furthermore, significant protection of blood neutrophils and lymphocytes were observed in the FDN+ irradiated group of rats.

Table (3): Bone marrow erythroid, lymphoid and myeloid cells (x103/ femur) in different groups of animals.

Groups	Erythroid	Lymphoid	Myeloid
Control	1.1± 0.09**A**	7.5± 0.64**A**	76.3± 6.72**A**
FDN-treated	1.2± 0.11**A**	7.9± 0.76**A**	77.6± 6.43**A**
Irradiated (5 Gy)	0.5± 0.07**B**	3.6± 0.34**B**	40.3± 4.11**B**
FDN+ Irradiated	0.8± 0.63**C**	5.6± 0.52**C**	53.1± 5.27**C**

Legends as in Table 1.

In Table 3, γ-irradiated group show marked significant decreases in erythroid, lymphoid and myeloid cells count comparing with control and FDN-treated groups. In protected group, the count of the 3 types of cells increased significantly, comparing with their values in irradiated group (Table 3).

Table (4): Lipid peroxidation (TBARS), SOD and GSPx in different groups of animals.

Groups	**TBARS** (n mol/ dl)	**SOD** (U/ ml)	**GSPx** (U/ ml)
Control	8.6 ± 0.79**A**	3.9± 0.32**A**	5.6± 0.53**A**
FDN-treated	8.7± 0.81**A**	3.9± 0.27**A**	5.6± 0.51**A**
Irradiated (5 Gy)	14.6± 1.57**B**	2.1± 0.31**B**	3.3± 0.23**B**
FDN+ Irradiated	10.3± 0.93**C**	3.0± 0.28**C**	4.1± 0.32**C**

Legends as in Table 1.

TBARS level in irradiated-group significantly increased comparing with control and FDN-treated groups, but significantly declined in protected group in comparison with irradiated-group (Table 4).

The blood SOD and GPx activities in irradiated-group significantly decreased, comparing with their activities in control and FDN-treated groups. Administration of FDN before-irradiation significantly enhanced these two enzymes as compared to those of the irradiated-group (Table 4).

DISCUSSION

In animals, naturally occurring carbohydrates such as polysaccharides can be sulphated and acts as molecular signals that regulate growth, development, and survival in the environment [(16)].

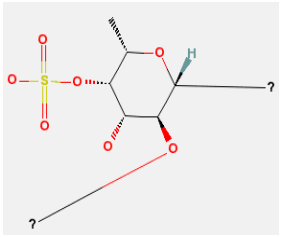
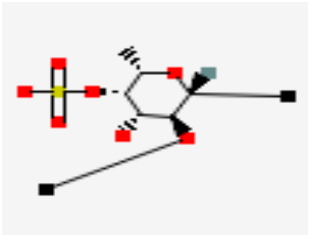


Figure (1): Fucoidan structure.



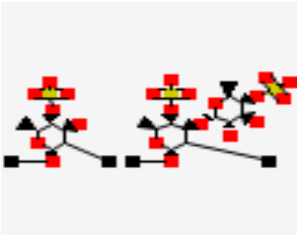


Figure (2): Sulfated fucose.

FDN carries functional groups (carboxymet hyl, phosphate and sulphate groups), these sulfate constituents that occurs in biomolecules, can play a critical role in major physiological functions in animals [17]**.** FDN is made up of β-L-fucose units (Fig. 1) linked by (1 → 4) and (1 → 3) glycosidic bonds and sulphated (Fig. 2) at positions 2 and/or 3 and/or 4 depending on the algal species [18].

FDN possess anti-proliferative and anti-adhesive effects on cells, protect cells from viral infection and is widely used as a health-promoting food component. However, studies on the toxicity of FDN from different brown algae are limited. No significant toxicological changes were induced in rats by FDN at a dose of 600 mg/ kg of body wt/ day. However, with 1200 mg, clotting time was significantly prolonged. No other signs of toxicity were observed [19].

Radiation-induced damage effect to cells by production and activation of haemostatic factors in addition, radiation induced inflammations, which in turn modulate the haemostatic system by induction the coagulation and fibrinolytic systems, altering the balance between pro-coagulant and anticoagulant activities [20].

Protein-C anticoagulant pathway appears to be the major pathway involved in the cross link between inflammation and coagulation [21]. Antithrombin-III is the physiologic inhibitor of thrombin of the clotting cascade, would be anticipated to decrease thrombin-mediated augmentation of the inflammatory events [22]. The endothelium can trigger and control fibrinolysis by the synthesis and release of tissue-type plasminogen activators [23].

The present study showed significant decrements in plasma protein-C and antithrombin-III and a significant increase in plasma tissue-plasminogen activators in irradiated animal group. FDN was shown to give a significant

protection when administered before irradiation. Protective actions of FDN were displayed as the inhibition of γ-radiation induced haemostatic disorder. FDN was able to prolong the clotting time of human plasma and inhibited thrombin activity and adjusted platelet aggregation [24].

In the present study, the recovery efficacy of FDN on total leukocytes count as well as neutrophils and lymphocytes rates in rats' peripheral blood were observed. The direct antioxidant activity of FDN, its ability to protect against radiation-induced cellular damage and enhance the post-irradiation recovery of haematopoiesis, as well as immune modulator activity together could contribute to FDN radio protective efficacy. FDN inhibited leukocyte recruitment in an inflammation model in rats, the relation between their structure and biological activities remain largely unknown [24]. Furthermore, it significantly inhibited neutrophil infiltration into the tissues and increased the number of circulating leukocytes 2 h after administration [25]. In addition, FDN as an immune-regulatory agent could promote the recovery of immunologic function in irradiated rats. The mechanism is associated with the arrest of lymphocyte-apoptosis by FDN [6].

The hematopoietic syndrome death is often sufficient for the organism lethality because of infection due to the impairment of the immune system [26]. Bone marrow is the main cellular source for the hematopoietic and immune systems; it contains large numbers of lymphocytes, granulocytes and stromal cells as precursors and mature cells. Our results suggested that specific populations of erythroid, lymphoid and myeloid cells may selectively survive in response to FDN-treatment following irradiation.

This data attributed to FDN affinity to inhibit radiation-induced apoptosis. Furthermore, FDN altered the production of immune-related cytokines from bone marrow cells (BMCs) and increased the capability of BMCs to induce proliferation of allergenic splenocytes that means, FDN had radio protective effects on BMCs with respect to cell viability and immuno-reactivity [1]. In addition, Wu *et al.*[6] concluded that FDN could inhibit rat splenic-lymphocyte apoptosis induced by radiation, and its mechanism could be associated with its regulation on expressions of Bax-proteins in splenic-lymphocyte.

Irradiation at the dose level of 5 Gy resulted in marked oxidative-stress presented by the significant increase in TBARS level and decreases in SOD and GSPx activities. Recently, Zahran *et al.*[27] and Tawfik *et al.*[28] confirmed these

obtained data. According to present study, treating rats with FDN attenuated the increase in the levels of TBARS caused by radiation-injury and enhanced SOD and GSPx activities.

Mitigation of oxidative stress, or excessive free-radical damage, may be especially relevant. FDN as antioxidant protect the outer membranes of cells, particularly blood and immune cells [(5)].

Recent studies have shown that FDN was able to increase the antioxidant enzymes in rats treated with oxidant agents [(29)]. Furthermore, FDN may represent a new approach for inhibiting the harm caused by excessive free radicals. FDN from *Fucus vesiculosus* had the highest antioxidant activity in relation to the other fractions, with high levels of uronic acid [(30)]. According to the results obtained in the present study, it could be concluded that FDN way offered protection against γ-rays induced cellular hazards.

Acknowledgment

The authors greatly appreciate Prof. Dr. M. S. Soliman, Health Radiation Research Dept., NCRRT, for revising bone marrow examination.

REFERENCES

1. Byon, Y.Y., Kim, M.H., Yoo, E.S., Hwang, K.K., Jee, Y., Shin, T. and Joo, H.G. (2008): Radioprotective effects of fucoidan on bone marrow cells: improvement of the cell survival and immunoreactivity. *J. Vet. Sci.*, 9, 359-365.

2. Cui, Y.Q., Luo, D.Z. and Wang, X.M. (2008): Fucoidan: advances in the study of its anti-inflammatory and anti-oxidative effects. *Yao. Xue. Xue. Bao.*, 43, 1186-1189.

3. Hosseinimehr, S.J. (2007): Trends in the development of radioprotective agents. *Drug Discov. Today*, 12, 794-805.

4. Arora, R., Gupta, D., Chawla, R., Sagar, R., Sharma, A., Kumar, R., Prasad, J., Singh, S., Samanta, N., Sharma, R.K. (2005): Radioprotection by plant products: present status and future prospects. *Phytother. Res.*, 19, 1-22.

5. Kim, H.J., Kim, M.H., Byon, Y.Y., Park, J.W., Jee, Y. and Joo, H.G. (2007): Radioprotective effects of an acidic polysaccharide of Panax ginseng on bone marrow cells. *J. Vet. Sci.*, 8, 39-44.

6. Wu, X., Yang, M., Huang, X., Yan, J. and Luo, Q. (2003): Effect of

fucoidan on splenic lymphocyte apoptosis induced by radiation. *Chin. J. Radiolog. Med. Protect.,* 23, 430-432.

7. National Academy of Sciences standards (1996): Institute of Laboratory Animal Resources (U.S.). Guide for the Care and Use of Laboratory Animals. Washington, D.C.: National Academy Press, 140 p.

8. Pabinger, I. (1986): Clinical relevance of protein C. *Blut.,* 53, 63-75.

9. Tollefsen, D.M. (1990): Laboratory diagnosis of antithrombin and heparin cofactor II deficiency. *Seminars in Thromb. Haemost.,* 16, 162-168.

10. Holvoet, P., Lijnen, H.R. and Collen, D. (1985): A monoclonal antibody specific for Lys-plasminogen. *J. Biol. Chem.,* 260, 12106-12111.

11. Goldberg, E.D., Dygai, A.M and Shakhov, V.P. (1992): Methods for Tissue Culture in Hematology, TGU Publishing House, Tomsk, pp. 256-257.

12. Yoshioka, T., Kawada, K., Shimada, T. and Mori, M. (1979): Lipid peroxidation in material and cord blood and protective enzyme against activated oxygen toxicity in the blood. *Am. J. Obestet. Gynecol.,* 135, 372-376.

13. Minami, M. and Yoshikawa H. (1979): A simplified assay method of superoxide dismutase. *Clin. Chem. Acta,* 92, 337-342.

14. Paglia, D. and Valentine, W. (1967): Studies on the quantitative and qualitative characterization of erythrocyte glutathione peroxidase. *Lab. Clin. Med.,* 70, 158-162.

15. Zar, J. H. (1999): Biostatistical Analysis. Upper Saddle River, N.J.: Prentice Hall, pp, 41-48.

16. Shibuya, N. and Minami, E. (2001): Oligosaccharide signalling for defence responses in plant. *Physiol. Mol. Plant Pathol.,* 59, 223-227.

17. Menard, R., Alban, S., de Ruffray, P., Jamois, F., Franz, G., Fritig, P., Yvin, J. and Kauffmann, S. (2004): β-1,3 glucan sulfate, but not β-1,3 glucan, induces the salicylic acid signaling pathway in tobacco and arabidopsis. *Plant Cell*, 16, 3020-3032.

18. Sezer, A.D., Cevher, E., Hatipoglu, F., Ogurtan, Z., Bas, V. and Akbuğa, J. (2008): The use of fucosphere in the treatment of dermal burns in rabbits. *Eur. J. Pharm. Biopharm.,* 69, 189-198.

19. Gideon, T.P. and Rengasamy, R. (2008): Toxicological evaluation of fucoidan from Cladosiphon okamuranus. *J. Med. Food,* 11, 638-642.

20. Meky, N., Mansour, M., Soliman, M. and Tawfik, E. (2002): Effects of gamma irradiation on some linked processes between coagulation and inflammatory reactions. *Egypt. J. Rad. Sci. Applic.,* 14, 1-12.

21. Iakhiaey, A. and Idell, S. (2006): Activation and degradation of protein C by primary rabbit pleural mesothelial cells. *Lung,* 184, 81-88.

22. Lauterbach, R., Pawlik, D., Radziszewska, R., Wozniak, J. and Rytlewski, K. (2006): Plasma antithrombin III and protein C levels in early recognition of late-onset sepsis in newborns. *Eur. J. Pediatr.,* 166, 585-589.

23. Bozec, A., Formento, P., Ciccolini, J., Fanciullino, R., Padovani, L., Murraciole, X., Fischel, J.L. and Milano, G. (2005): Response of endothelial cells to a dual tyrosine kinase receptor inhibition combined with irradiation. *Mol. Cancer Ther.,* 4, 1962-1971.

24. Cumashi, A., Ushakova, N., Preobrazhenskaya, M. and Nifantiev, N. (2007): A comparative study of the anti-inflammatory, anticoagulant, antiangiogenic, and antiadhesive activities of nine different fucoidans from brown seaweeds. *Glycobiology,* 17, 541-552.

25. Ikegami-Kuzuh1ara, A., Yoshinaka, T., Ohmoto, H., Inoue, Y. and Saito, T. (2001): Therapeutic potential of a novel synthetic selectin blocker, OJ-R9188, in allergic dermatitis. *Brit. J. Pharmacol.,* 134, 1498-1504.

26. Chen, Y., Lin, S., Chiang, W., Wu, K. and Tsai, T. (2006): Pentoxifylline ameliorates proteinuria through suppression of renal monocyte chemoattractant protein-1 in patients with proteinuric primary glomerular diseases. *Kidney Int.,* 69, 1410-1415.

27. Zahran, A.M., Azab, Kh.Sh. and Abbady, M.I. (2006): Modulatory role of allopirinol on xanthine oxidoreductase system. *Egypt. J. Rad. Sci. Applic.,* 19, 373-388.

28. Tawfik, S.S., Abbady, M.I., Zahran, A.M. and Abouelalla, A.M.K. (2006): Therapeutic efficacy attained with thyme essential oil supplementation throughout γ-irradiated rats. *Egypt. J. Rad. Sci. Applic.,* 19, 1-22.

29. Veena, C.K., Josephine, A., Preetha, S.P., Varalakshmi, P. and Sundarapandiyan, R. (2006): Renal peroxidative changes mediated by

oxalate: the protective role of fucoidan. *Life Sci.*, 79, 1789-1795.

30. de Souza, M., Marques, C., Dore, C., da Silva, F., Rocha, A. and Leite, E. (2007): Antioxidant activities of sulfated polysaccharides from brown and red seaweeds. *J. Appl. Phycol.*, 19, 153-160.

مجلة البحوث الإشعاعية والعلوم التطبيقية

مجلد 4 عدد 1(ب) ص ص 233– 244 (2011)

كفاءة الفيوكيدان الوقائية فى الجرذان المعرضة لأشعة جاما

سامح سليمان توفيق ، صفوت فريد سلامة

*قسم البحوث الصحية الإشعاعية ، المركز القومى لبحوث وتكنولوجيا الإشعاع- هيئة الطاقة الذرية , ص. ب. 29 مدينة نصر، القاهرة ، مصر.

*قسم علم الحيوان- كلية العلوم- جامعة الملك سعود و قسم بحوث البيولوجيا الإشعاعية- شعبة البحوث الاشعاعية- المركز القومى لبحوث وتكنولوجيا الاشعاع ، هيئة الطاقة الذرية ص. ب. 29 مدينة نصر، القاهرة، مصر.

يعتبر الفيوكيدان من الكربوهيدرات (السكريات المركبة) ويتم استخلاصه من فطريات البحر البنية، و يختص بميزاته البيولوجية المتعددة و خواصه: كمضاد للالتهاب والتخثر وكمانع للأكسدة.

تم دراسة الكفاءة الوقائية للفيوكيدان (100 ميلليجرام / كجم) بتجريعه لمجموعة من الجرذان لمدة 10 أيام ، وتعريض مجموعة أخرى لجرعة 5 جراى من أشعة جاما ، ومجموعة تجرعت الفيوكيدان ثم عرضت لأشعة جاما ، وذلك بالمقارنة بمجموعة ضابطة من الجرذان.

تم تقدير بعض معايير التجلط الدمو ى: بروتين- س (protein-C) ومضاد البرثرومبين- الثالث (antithrombin-lll) ومنشطات البلازمينوجين النسيجية (Tissue-plasminogen). كما تم تقدير بعض المعايير الدموية: عدد كرات الدم البيضاء الكلية (total leukocytes) ، وبعض أنواع الكرات البيضاء ومنها المتعادلة (neutrophils) والليمفاوية (lymphocytes) ، كما تم تقدير تعداد بعض خلايا النخاع العظم ى ومنها عدد خلايا النخاع العظم ى الحمراء (erythroid) والليمفاوية (lymphoid) والبيضاء (myeloid) ، وكذلك تم تقدير مستو ى حمض الثيوبربتيورك (TBARS) ونشاط إنزيم ى السوبرأكسيد ديسميوتيز (SOD) والجلوتاثيون بيروكسيديز (GSPx) بالدم. أظهرت النتائج حدوث تغييرات إحصائية بمعايير التجلط الدمو ى والمعايير الدموية ومستو ى حمض الثيوبربتيورك ونشاط إنزيمى السوبرأكسيد ديسميوتيز والجلوتاثيون بيروكسيديز المضادان للأكسدة وذلك بسبب تعرض الجرذان لأشعة جاما.

دلت النتائج المتحصل عليها أن وقاية الجرذان بالفيوكيدان آدت الي تحسين مستويات كل المعايير و الدلالات المقدرة التي تبدلت نتيجة التعرض لأشعة جاما. كما تشير النتائج أن عقار الفيوكيدان له تأثير وقائى فعال فى التصدى لتأثير الإشعاع المدمر لخلايا الجسم.

الخلاصة: الفيوكيدان قد يكون عقار جديد فعال يقي من أضرار الإشعاع يتميز بأنه غير سام عند تناوله مثل غيره من واقيات الاشعاع الأخرى.