Role of Omega 3 fatty acids on radiation-induced oxidative and structural damage in different tissues of male albino rats.

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ABSTRACT

Omega-3 fatty acids play a critical role in the development and function of the reproductive and central nervous systems. The aim of this study is to evaluate the effect of omega-3 fatty acids supplementation on lipid peroxidation and antioxidant enzyme levels associated with histopathologic changes induced by gamma irradiation in the testis and brain of male albino rats. Rats were whole body exposed to radiation at a single dose of 3Gy. Omega-3 fatty acids (0.4 gm/kg b wt/day) were given to rats, by gavages, for 15 consecutive days before irradiation and for 15 days after irradiation. Rats were sacrificed one and 15 days post irradiation. Biochemical analysis of testis and cerebral cortex samples showed that irradiation induced a significant increase in xanthine oxidase (XO) activity and lipid peroxidation end product malondialdehyde (MDA) and a decrease in the content of reduced glutathione (GSH) and activity of antioxidant enzymes; glutathione peroxidase (GPX), superoxide dismutase (SOD) and catalase (CAT). Histological examination of testis and cerebral cortex tissues showed spermatogonial degeneration, apoptosis and necrosis in the testis and neurons cell bodies with ill defined and even ruptured cell membrane and damaged blood capillaries in the cerebral cortex. Omega-3 administration has attenuated the toxic effects of radiation by decreasing the levels of MDA, and XO, and increasing the activity of endogenous antioxidant enzymes, which was associated with amelioration of the histological injury markers in both testis and cerebral cortex. It could be postulated that omega-3 fatty acids as a multifunctional dietary supplement could exert a modulatory role in radiation-induced testis and cerebral cortex biochemical and histological changes through its antioxidant properties.

Keywords: Omega-3 fatty acids, gamma rays, antioxidants, testis, cerebral cortex, rats
INTRODUCTION

Interest has grown regarding the role of oxygen toxicity and free radical reactions in association with fertility potential, which can cause oxidative damage to the lipid constituents of the cell membrane. Reactive oxygen species (ROS) have been shown to be involved in fertility due to defective sperm function (1). Control of the cellular redox has been extensively shown to be essential for normal cellular function (2). For example, control of mammalian cell growth pathways are tightly dependent on oxidants generated by normal endogenous metabolism or ROS induced following external stimuli such as radiation, drugs and pathogens causing imbalance in the generation and detoxification of ROS and resulting in the cellular state of oxidative stress (3).

ROS interact with biological molecules, produce toxic free radicals and result in lipid peroxidation and deoxyribonucleic acid (DNA) damage. Lipid peroxidation, in the biological membranes, causes alteration in fluidity, fall in membrane potential and increase in permeability of H\(^0\) and other ions and eventual rupture leading to release of cell organelle contents, such as lysosomal hydrolytic enzymes (4).

Testis is known to be one of the most radiosensitive organs in the body. With the advent of new radiotherapy modalities, there is a considerable improvement in the survival rate of cancer patients and animals. Thus, protection for reproductive potential and hereditary characters in the germ cells of these mammals against radiation damage is recommended (5). In the testis, physiological apoptotic death of selected germ cells plays an important role in limiting the germ cell population (6). Moreover, stress caused by external disturbances such as chemotherapy or irradiation can cause increased testicular apoptosis leading to total germ cell loss. Spermatogonia are especially sensitive to radiation; doses as low as 0.1 Gy are known to cause damage to these cells (7).

Radiation exposure of the adult brain results in variable degrees of cognitive impairment, even though less histological injury is appearing (8). The rate of neurogenesis may be altered by several factors including age, genetic influence, chemicals and radiation (9) & (10). Brain injury is the leading cause of morbidity. Several pathogenic mechanisms including derangements in cerebral blood flow, exitotoxicity, ROS, inflammation and apoptosis, have been described for brain damage (11). Brain tissues are very susceptible to oxidative injury induced by ROS; their antioxidant defence systems are imbalanced in favour of oxidants (12). Oxidative stress has been implicated in the pathogenesis
of ischemic cerebral injury.

Mammalian cell viability is dependent on the supply of essential fatty acids (EFA) linoleic and alpha-linoleic acid. EFA are converted into omega-3 and omega-6 polyunsaturated fatty acids (PUFAs), which are essential constituents of membrane phospholipids and precursors of eicosanoids and docosanoids essential for cell viability. The lake of PUFAs and eicosanoids doesn’t impair the normal viability and life span of male and female fads, but cause sterility (13). It has recently become clear that one of the values of PUFAs can affect various functions of the immune system including inflammatory responses as all PUFAs decreased ROS production (14). Many laboratory studies suggested that n-3 fatty acids, especially long- chain polyunsaturated fatty acids, have antitumor effects (15).

Omega-6 and omega-3 PUFAs play a central role in the normal development and functioning of the brain and central nervous system. Long-chain PUFAs (LC-PUFAs), in particular are involved in numerous neuronal processes, ranging from effects on membrane fluidity to gene expression regulation. Numerous observational studies have shown a link between childhood developmental disorders and omega-6: omega-3 fatty acid imbalances. For instance, neurocognitive disorders such as attention-defect, hyperactivity disorder, dyslexia, dyspraxia and autism spectrum disorders are often associated with a relative lake of omega-3 fatty acids (16).

The objective of this study is to investigate whether (Omega-3) fatty acids could exert protective effect against radiation- induced biochemical and histological disorders in testis and brain.

**MATERIAL AND METHODS**

**Radiation Facility**

Whole body gamma irradiation of rats was performed using a Canadian Gamma cell-40 (137Cs) located at the National Center for Radiation Research and Technology, Cairo, Egypt at a dose rate of 1.5Gy/min. Rats received a dose of 3Gy applied as one shot.

**Omega-3 fatty acids treatment**

Omega-3 fatty acids were purchased from the International Company for Scientific and Medical Import. Animals received a dose of (0.4g/day/kg b.wt), by gavages, for 15 consecutive days before irradiation and 15 consecutive

days after irradiation.

**Experimental design**

All animal experiments were conducted in accordance with the National Institutes of Health (NIH) guide for the care and use of laboratory animals (NIH) publication No.80-23; (revised 1978). Male albino rats (120-150 g) obtained from the Egyptian Holding Company for Biological Products and Vaccines were used as experimental animals. Forty-eight rats were kept under standard conditions along the experimental period and fed on pellet concentrated diet containing all the necessary nutritive elements. Liberal water intake was available. Animals were divided into 6 groups each of 8 animals.

**Control:** Rats were not subjected to any treatment. Omega-3: Rats received omega-3 fatty acids (0.4 g/day/kg b.wt) by gavages for 30 consecutive days according to Songur et al., (2004). Irradiated: rats received a dose of 3Gy as one shot whole body gamma rays. Omega-3 +irradiated group: Rats received oral dose of omega-3 fatty acids (0.4g/day/kg b.wt) for 15 consecutive days before irradiation and 15 consecutive days after irradiation.

**Biochemical analysis**

Rats were sacrificed one and 15 days after irradiation exposure. Testis and cerebral cortex were removed and washed with ice-cold saline and blotted with pieces of filter paper, weighed and homogenized in ice-cold distilled water. The content of GSH was determined according to Beutler et al. (1963). The activity of SOD, CAT and GPX was determined according to Minami and Yoshikawa (1979), Aebi (1984) and Lawrence & Bruk (1976), respectively. The extent of lipid peroxidation was assayed by measurement of MDA according to Yoshioka et al., (1979).

**Statistical Analysis**

Data are expressed as mean ± SE., one way Anova was applied followed by Duncan's test (1955) and differences were considered significant at probability P< 0.05.

**Histological Study**

For light microscopic investigations, testis and cerebral cortex were fixed in buffered formol, processed routinely for paraffin embedding then sectioned at 6 um. Sections were stained with haematoxylin and eosin (HE) and mounted with canada balsam. Sections were examined by Olympus light
microscope (X400) to detect the histological and histopathological changes induced by any of the above mentioned treatments.

**RESULTS**

Animals fed on normal diet and given a daily dose of omega-3 supplement for 30 consecutive days, showed no significant changes in xanthine oxidase (XO) activity, malondialdehyde (MDA) and GSH level in testis and cerebral cortex tissues when compared with control group (tables 1 & 2). The activity of antioxidant enzymes GPX, SOD, and CAT showed approximately normal ranges (tables 3 & 4). Histological study of testis sections showed normal dense fibrous membrane tunica albuginea, seminiferous tubules, normal tubuli recti and normal interstitial cells laying in the loose connective tissue between the seminiferous tubules (Fig 1a &b). The histological observations in cerebral cortex tissue showed normal neuropil background, normal pyramidal and glial cells and normal oligodendrocytes, normal astrocytes and normal purkinjie cells (Fig 2a &b).

Table (1): Effect of Oral Supplementation of Omega-3 fatty acids on Testis XO-activity and MDA content in male rats.

<table>
<thead>
<tr>
<th>Animal groups</th>
<th>XO activity (mU/mg proteins)</th>
<th>MDA content (nmol/g tissue)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st day</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>1.82±0.16</td>
<td>113.92±0.76</td>
</tr>
<tr>
<td>Omega-3</td>
<td>1.70±0.10</td>
<td>110.8±1.62</td>
</tr>
<tr>
<td>Irradiation</td>
<td>3.67±0.08</td>
<td>136.87±1.18</td>
</tr>
<tr>
<td>Omega-3+Irradiation</td>
<td>2.55±0.12ab</td>
<td>119.08±0.73</td>
</tr>
<tr>
<td>15th day</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>1.93±0.10</td>
<td>112.75±1.03</td>
</tr>
<tr>
<td>Omega-3</td>
<td>1.66±0.11b</td>
<td>110.41±0.77</td>
</tr>
<tr>
<td>Irradiation</td>
<td>2.72±0.10a</td>
<td>134.99±0.62</td>
</tr>
<tr>
<td>Omega-3+Irradiation</td>
<td>2.29±0.10ab</td>
<td>118.29±0.73</td>
</tr>
</tbody>
</table>

Values are means ± SE (n=8)

<sup>a</sup>: Significantly different from control group.

<sup>b</sup>: Significantly different from radiation group.

In the present work, whole body gamma-irradiated rats at a single dose of 3 Gy, showed a significant increase (P<0.05) in XO activity and MDA concentration one and 15 days post-irradiation, in testis and cerebral cortex tissues, compared with control group (tables 1&2). A significant decrease in the content of GSH and the activity of antioxidant enzymes GPX, SOD, and CAT tissues were also recorded (tables 3&4). Clear histopathological changes were noticed as amorphoid testicular tissue, ruptured tunica albuginea, atrophied and ill defined seminiferous tubules, disturbed spermatogenesis, disappearance of tubuli, and degenerated interstitial cells (Fig 1c). In the cerebral cortex,
expanding and dilating purkinje cells, ill defined astrocytes and pyramidal cells, degenerated, vacuolated and hemorrhaged neuropil background and vacuolated glial cell, were observed (Fig 2c).

Table (2): Effect of Oral Supplementation of Omega-3 fatty acids on Cerebral Cortex XO-activity and MDA content in male rats.

<table>
<thead>
<tr>
<th>Animal groups</th>
<th>XO activity (mU/mg proteins)</th>
<th>MDA content (nmol/g tissue)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st day</td>
<td></td>
<td></td>
</tr>
<tr>
<td>control</td>
<td>1.32±0.05</td>
<td>22.49±0.43</td>
</tr>
<tr>
<td>Omega-3</td>
<td>1.33±0.03b</td>
<td>22.5±0.16b</td>
</tr>
<tr>
<td>Irradiation</td>
<td>3.85±0.13a</td>
<td>30.63±5.7a</td>
</tr>
<tr>
<td>Omega-3+Irradiation</td>
<td>2.8±0.11ab</td>
<td>25.47±0.42ab</td>
</tr>
</tbody>
</table>

15th day

<table>
<thead>
<tr>
<th>Animal groups</th>
<th>XO activity (mU/mg proteins)</th>
<th>MDA content (nmol/g tissue)</th>
</tr>
</thead>
<tbody>
<tr>
<td>control</td>
<td>1.43±0.07</td>
<td>21.13±0.64</td>
</tr>
<tr>
<td>Omega-3</td>
<td>1.47±0.14b</td>
<td>22.22±0.35b</td>
</tr>
<tr>
<td>Irradiation</td>
<td>3.79±0.11a</td>
<td>27.85±0.54a</td>
</tr>
<tr>
<td>Omega-3+Irradiation</td>
<td>2.46±0.13ab</td>
<td>225.15±0.37ab</td>
</tr>
</tbody>
</table>

Legends as in table 1

Table (3): Effect of Oral Supplementation of Omega-3 fatty acids on Testicular Endogenous Antioxidants in Male Rats.

<table>
<thead>
<tr>
<th>Animal groups</th>
<th>GSH mg/g tissue</th>
<th>GPX Ug/ml/min</th>
<th>SOD U/mg protein</th>
<th>CAT U/mg protein</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st day</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>25.04±0.55</td>
<td>34.99±0.63</td>
<td>11.08±3.1</td>
<td>110.02±0.72</td>
</tr>
<tr>
<td>Omega-3</td>
<td>26.01±0.33b</td>
<td>34.86±0.73b</td>
<td>10.91±0.37b</td>
<td>108.49±1.15b</td>
</tr>
<tr>
<td>Irradiation</td>
<td>15.15±0.34a</td>
<td>24.75±0.48a</td>
<td>7.75±0.59a</td>
<td>91.20±0.64a</td>
</tr>
<tr>
<td>Omega-3+Irrad</td>
<td>20.54±0.46ab</td>
<td>32.61±0.55ab</td>
<td>16.99±0.15ab</td>
<td>102.46±0.57ab</td>
</tr>
</tbody>
</table>

15th day

<table>
<thead>
<tr>
<th>Animal groups</th>
<th>GSH mg/g tissue</th>
<th>GPX Ug/ml/min</th>
<th>SOD U/mg protein</th>
<th>CAT U/mg protein</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>26.66±0.2</td>
<td>35.6±0.47</td>
<td>11.4±0.34</td>
<td>107.89±0.38</td>
</tr>
<tr>
<td>Omega-3</td>
<td>26.25±0.6b</td>
<td>34.79±0.37b</td>
<td>10.63±0.19b</td>
<td>107.60±0.36b</td>
</tr>
<tr>
<td>Irradiation</td>
<td>12.54±0.31a</td>
<td>23.78±0.35a</td>
<td>7.09±0.31a</td>
<td>88.49±0.54a</td>
</tr>
<tr>
<td>Omega-3+Irrad</td>
<td>24.41±0.40ab</td>
<td>32.45±0.45ab</td>
<td>10.8±0.20ab</td>
<td>99.92±0.58ab</td>
</tr>
</tbody>
</table>

Legends as in table 1

Table (4): Effect of Oral Supplementation of Omega-3 fatty acids on Endogenous Antioxidants of Cerebral Cortex in Male Rats.

<table>
<thead>
<tr>
<th>Animal groups</th>
<th>GSH mg/g</th>
<th>GPX Ug/ml/min</th>
<th>SOD U/mg protein</th>
<th>CAT U/mg protein</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st day</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>2.64±0.03</td>
<td>2.70±0.01</td>
<td>7.84±0.12</td>
<td>2.78±0.07</td>
</tr>
<tr>
<td>Omega-3</td>
<td>2.75±0.03b</td>
<td>2.72±0.02b</td>
<td>7.71±0.12b</td>
<td>2.57±0.16b</td>
</tr>
<tr>
<td>irradiation</td>
<td>1.28±0.03a</td>
<td>1.65±0.02a</td>
<td>4.15±0.09a</td>
<td>1.68±0.19a</td>
</tr>
<tr>
<td>Omega-3+Irrad</td>
<td>2.16±0.01b</td>
<td>2.21±0.02ab</td>
<td>6.37±0.18ab</td>
<td>2.56±0.06ab</td>
</tr>
</tbody>
</table>

15th day

<table>
<thead>
<tr>
<th>Animal groups</th>
<th>GSH mg/g</th>
<th>GPX Ug/ml/min</th>
<th>SOD U/mg protein</th>
<th>CAT U/mg protein</th>
</tr>
</thead>
<tbody>
<tr>
<td>control</td>
<td>2.81±0.1</td>
<td>2.73±0.06</td>
<td>7.01±0.09</td>
<td>2.80±0.08</td>
</tr>
<tr>
<td>Omega-3</td>
<td>2.69±0.1b</td>
<td>2.70±0.10b</td>
<td>6.90±0.16b</td>
<td>2.77±0.07b</td>
</tr>
<tr>
<td>Irradiation</td>
<td>1.51±0.08a</td>
<td>1.60±0.07a</td>
<td>4.18±0.11a</td>
<td>1.80±0.08a</td>
</tr>
<tr>
<td>Omega-3+Irrad</td>
<td>2.47±0.1ab</td>
<td>2.27±0.04ab</td>
<td>6.37±0.15ab</td>
<td>2.55±0.11ab</td>
</tr>
</tbody>
</table>

Legends as in table 1

Oral administration of omega-3 supplement to rats for 15 consecutive days before irradiation and 15 days after irradiation led to significant decrease in XO activity and MDA content in testis and cerebral cortex tissues, compared
with irradiated groups (tables 1& 2). Significant increase in GSH content and the activity of antioxidant enzymes GPX, SOD, and CAT were recorded, compared with control and irradiated groups (tables 3&4). Histological observations in the testis and cerebral cortex of rats supplemented with omega-3 prior and after irradiation revealed well-defined shape of tunica albuginea, regenerated seminiferous tubules, nearly normal spermatogenesis and ameliorated interstitial cells (Fig 1. d). In cerebral cortex, regenerated nerve cells, nearly normal pyramidal cells and regenerated oligodendrite cells were observed (Fig 2d).

Fig.(1): Photomicrograph of a section in the testis of rats :  a) control .  b) omega-3 treated: normal seminiferous tubules (s), normal tunica albugenia (t) and normal interstitial cells (i). C) irradiated (1st day): atrophoid and ill-defined seminiferous tubules(S), ruptured tunica albuginea (t), degenerated interstitial cells(i) and disturbances of spermatogonia (p), (day 15th): severe degenerated seminiferous tubules (s), disappearance of spermatogonia (p) and ruptured tunica albuginea(t), degenerated interstitial cells(i).  d) omega-3 treated irradiated: regenerated seminiferous tubules(s), improved tunica albuginea (t) and improved interstitial cells(i) and nearly normal spermatogenesis. (H&E) (X 400)
Fig.(2): Photomicrograph of a section in the cerebral cortex of rats: a) control. b) omega-3 treated: normal pyramidal cells (py), normal astrocytes (a), normal purkinjie cells(pk) and normal background of neuropil (N). c) irradiated (1\textsuperscript{st} day): ruptured pyrmidal cells (py), vacuolated and ruptured purkinjie cells(pk) and not distinguished background. (day 15\textsuperscript{th}): dilated and expanding purkinjie cells(pk), vacuolated and expanding pyramidal cells (py) and haemorhaged background of neuropil (N). d) omega-3 treated-irradiated: well defined pyramidal cells (py), regenerated purkinjie cells and oligodendrocyes (o). (H&E) (X400).
DISCUSSION

In all biological systems, water is the most abundant molecule and radiation splitting of the water molecule, radiolysis of water, is a primary event in the initiation of biological damage (24). The absorption of energy by a water molecule results in the production of free radicals, which are very reactive species that react with organic molecules within the cells (25). Radiation is one of the most widespread sources of environmental stress in living environment. Ionizing radiation is known to induce various histological, physiological and biochemical changes in human and animals.

This study demonstrated that gamma radiation caused biochemical oxidative damage manifested by increasing activity of XO as well as TBARS level (table 1 & 2), and decreasing the endogenous antioxidants GSH, GPX, SOD and CAT in testis and cerebral cortex tissues of rats (tables 3 & 4). Radiation exposure of rats caused histopathological changes in the testis and cerebral cortex. Oral treatment with the antioxidant omega-3 a potent free radical scavenger agent, significantly attenuated the oxidative damage and histopathological changes in the testis and cerebral cortex tissues.

Xanthine oxidoreductase system includes two intra-convertible enzymatic activities XO and XDH (xanthine dehydrogenase). Interest in XO has increased recently since it is a major sources of free radicals in cells. In the present work, gamma-irradiation may have caused the conversion of XDH to XO resulting in an increase in XO-specific activity in both time intervals (26). However, the increase was less severe on the 15th day post-irradiation which may be due to time elapsing post-irradiation (tables 1 & 2).

In the present study, MDA level increased markedly after irradiation exposure (tables 1 & 2). It is well known that intensive stress response results in creation of ROS that cause lipid peroxidation, especially in membranes and can play an important role in tissue injury. The elevated level of MDA might probably result from the interaction of excess OH, resulting from the radiolysis of water upon exposure to ionizing radiation, with polyunsaturated fatty acids in the phospholipids portion of cellular membranes (4).

Brain cells are at a particular risk from free radicals damage because of their high content of iron (a metal that, in its free from, is catalytically involved in production of damaging oxygen free radical species), and because of their relatively deficient antioxidant defence mechanisms (27). Thus, antioxidative
defence is critically important in nervous tissues protection.

In the present work, irradiation of rats resulted in enhancement of XO activity with concomitant decrease in GSH, GPX, SOD and CAT levels in testis and cerebral cortex at the two time intervals (tables 3 & 4). Therefore, inhibition of XO by omega-3 could result in radioprotection evidenced by decreasing XO activity and increasing antioxidant activities.

Any pathological state that leads to increased, production and/or ineffective scavenging of reactive oxygen species may play a crucial role in determining tissue injury when the generation of ROS and other free radicals overwhelms cellular defences, these unstable radicals react with essential molecules within the cell such as lipids, protein and DNA leading to histological changes as well as functional abnormalities (28).

Most of the basic data regarding the effect of radiation on the testis are issued from animal-studies. They demonstrated the extreme radiosensitivity of germ cell liveage but little is known about the reversible or definitive aspects of these radiation induced effects. (29) postulated that the cells killed by irradiation were mainly spermatogonia and spermatocytes, engaged in replicating their DNA at the time of exposure, but all spermatocytes seemed damaged as they gave abnormal descendent cells influencing cells function in the adult rat.

Hasegawa et al., (1997) (30) suggested that the mechanism of radiation induced spermatogonial degeneration is closely related to apoptosis. (31) showed that all spermatogenic cells, especially primary spermatocytes, displayed prominent degeneration in the groups submitted to total body and abdomino-pelvic irradiation that induced identical apoptosis and testicular damage. Irradiation can cause temporary azospermia in mouse and this effect is reversible after eight weeks (32).

Degeneration of vital tissues may be due to increased oxidative stress. ROS contributes to cerebrovascular complications, reduction in cerebral blood flow, disruption of the blood brain barrier and cerebral oedema. All these neurochemical and neurophysiological changes ultimately contribute to the long-term complications associated with radiation exposure, including morphological abnormalities, and increased vulnerability to physiological events (33): Vulnerability of brain to oxidative stress induced by oxygen free radicals seems to be due to the fact that, on one hand, the brain utilizes about one fifth of the total oxygen demand of the body and on the other, it is not
particularly enriched, when compared with other organs, in any of the antioxidant enzymes. Relatively low levels of these enzymes may be responsible in part for the vulnerability of this tissue (34).

It is well documented that dietary antioxidants play an important role in mitigating the damaging effects of oxidative stress on cell. Omega-3 fatty acids, are more frequently found in green leaves. The leaves and seeds of the perilla plant are the richest plant source of alpha-linolenic acid. Fish oil contains little alpha-linolenic acid (EFA) but is rich in the omega-3 derivatives eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). omega-3 was shown to scavenge reactive oxygen (ROS) and free radicals (35). In this context (36) found that free radicals scavenging potential of PUFA by humans supplemented with different fatty acids reduced execration of lipid peroxidation products after omega-3 intake. (37) mentioned that omega-3 reduced the harmful effects of oxidative stress in testis of rats injected with dizocilpine (MK-801). Also, (17) provided strong support for a therapeutic effect of omega-3 EFA in some neuropsychiatric disorder in which reactive oxygen species (ROS) are recently accused to be an important physicopathogenetic factor.

Fatty acids and lipids are important components of balanced diets as the body uses these as a source of energy, as well as their structural role in cellular membranes and as precursor of inter and intracellular singling. Docosahexaenoic acid (DHA) is an important polyunsaturated fatty acid (PUFA) for brain development and function, primarily derived from fish consumption (38). Fish oils are a key source of n-3 PUFAs for consumers of fish, and have been shown to have anticancer, anti-inflammatory and cardiovascular protective effects (39), (40) and (41).

According to the results obtained in the present study, it appeared that pre and post treatment with omega-3 attenuated the radiation induced increase in the activity of XO and MDA and maintained the content of GSH and the activity of GPX, SOD, and CAT. Consequently, omega-3 would protect cellular membrane from radiation induced lipid peroxidation so maintaining the architecture of testis and cerebral cortex.

REFERENCES


52.


دور الأحماض الدهنية أوميغا-3 على تأثير الإشعاع المتسبب في التدمير التأكسدي والتركيبي في الأنسجة المختلفة في ذكور الجرذان البيضاء

رينيه حورجي رزيق - ناهد محمد أبو زيد - أمال غريب أحمد

قسم البحوث الصحية الإشعاعية - قسم البيولوجيا الإشعاعية - المركز القومي لبحوث وتقنية الإشعاع - هيئة الطاقة الذرية - مصر - القاهرة

تتابع الأوميغا-3 دوراً هاماً في دعم ووظيفة الجهاز التناسلي والجهاز العصبي المركزي. الهدف من هذه الدراسة هو تقييم تأثير تناول أوميغا-3 على أكسدة الدهون ومستوى الإنزيمات مضادة للأكسدة والتفاعلات الهستوبيولوجية المتصلة من الإشعاع الجامى في الخصية والمخ.

تعرضت الصور كلية للإشعاع الجامى بجرعة واحدة (3 جرائد) وتناولت أوميغا-3 (4). جم/كم وزن الجسم يومياً) بالقم خلال 15 يوماً قبل تعرض للإشعاع و15 يوماً آخر بعد التعرض للإشعاع.

توضح النتائج البيologiغية في الخصية والتخصص الكرويين في المخ أن التعرض للإشعاع الجامى يسبب زيادة ملحوظة في الزئنزيء أوكسيدوز والدهون فوق المؤكسدة (الألوديداتالهيدروكسيل) ويصاحبه هذا ارتفاع ملحوظ في نشاط الإنزيمات مضادة للأكسدة مثل (ال obsłودين، بروكسيدوز، سير أكسيد ديمويتيز والكاتالابز).

وتوضح الفحوصات الهستوبيولوجية في شرائح الخصية والتصنيف الكرويين بالميوكروسكوب الضوئي للحيوانات المشععة أن التدمير في الخلايا المكونة للحيوانات المنوية بالخصية وهذا يرجع إلى عملية موت الخلايا والتهامها لبعضها ببعض وأيضاً أوضحت الفحوصات الهستوبيولوجية للمخ صعوبة التعرف على جسم الخلية العصبية وكذلك تدمير في غشاء الخلية العصبية وتدوير الوعوي الدموية المغذية للمخ.

تناول الأوميغا-3 قبل وبعد التشيع بعد من التأثيرات الضارة للإشعاع وهذا يتضح من اختزال مستوى الزئنزيء أوكسيدوز والدهون المؤكسدة في مقابل زيادة الإنزيمات مضادة للأكسدة وهذا مرتبط بالتحسين في أدلة إسحاق أنسجة في كل من الخصية والمخ.

يمكن القول أن تناول الأوميغا-3 يعتبر متعدد الوظائف حيث تقوم بالحد من آثار الإشعاع المتسبب في التغييرات البيولوجي و الهستوبيولوجية في كل من الخصية والتخصص الكرويين في المخ من خلال فعاليتها كمادة مضادة للأكسدة.