Topical Olive Oil with Downregulation of Stress Factors Protects Mice Skin from Precarious 600 MHz Electromagnetic Radiation

Original Article

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ABSTRACT

Background: Recently, studies are concerned with electromagnetic radiation (EMR) as one of the most physical factors to which a biological body and hence skin are exposed. Research also demonstrates that topical olive oil can prevent skin damage.
Aim of work: This study aimed to examine the histological, biochemical, and immunohistochemical changes in the skin tissue of adult mice after exposure to 3600 MHz (for 2 weeks) electromagnetic waves as well as the protective role of topical olive oil on mice skin For this,
Materials and Methods: Thirty adult mice were categorized equally into six groups control; olive oil; EMR-exposed: mice exposed to 3600 MHz for 2 weeks; Pre, Post-EMR groups: mice painted with topical olive oil pre and post-exposure respectively for 2 weeks; recovery group: mice left 2 weeks after exposure without any treatment. Histopathological examination and biochemical analysis were performed.
Results: Mice exposed to EMR showed several histological changes as increased thickness and discontinuation of the epidermis, flat epidermal-dermal junction, dermal cell atypia, and hair follicle degeneration, disorganized and fragmented collagen and elastic fibers. Furthermore, reduced catalase activity, and increased malondialdehyde (MDA) content and inflammatory cytokines, Tumor necrosis factor (TNF)-α and interleukin (IL)-6. Also, up-regulation of tumor protein (P) 53, caspase 3, and HSP (heat shock proteins)-70 were observed in the skin of mice. These changes were improved by using topical olive oil that is more pronounced in Pre-EMR than Post-EMR with partial insensible effect in the recovery group.
Conclusion: The present work showed that olive oil protected mice's skin against EMR, especially when used before exposure through amelioration of oxidative, inflammatory, and heat shock stress factors. The absence of exposure leads to a partial return to the control state for further studies.

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Key Words: Electromagnetic radiation; immunohistochemistry; olive oil; skin; stress.

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INTRODUCTION

Besides regular exposureato the emitting EMR from natural sourcesasuch as the sun and the earth. Today's people are exposed to EMR from advances in technology and expanded use of digitalaequipment[1]. The effect of electromagnetic radiationaon the body depends on its frequency andapower. According to the World Health Organization (WHO), lower levels of exposures can induce symptomsaand signs of ill health in 1–3% of the world's population who had EMF asensitivity[2].

The skin is the body'salargest organ, accounting for more than 10upercent of body mass. It serves as aabarrier to the absorption of the environmental seriousamaterial. Hence, it can serve as a target for many toxicafactors[3]. Cutaneous repercussionsaof radiation varyaconsiderably in severity, acourse and prognosis. When they doaoccur, cutaneous changes toaradiation are commonly graded as acute, consequential-late, or chronic[4]. By far the most commonahealth hazard ofaradiation is sunburn, which causes overaone million new skin cancers annually[5]. Other reports suggestedathat RF-EMR may lead toaDNA damage and chromosomalainstability[6]. Skin damage post-irradiation was detectedain rats exposed to 900 MHZ and there were many histological changes includingainflammation and fibrosis in the skinatissue[7].

Studies have reported that EMFaof such intensity leads to irreversible oxidativeadamage in the lymphoid organs ofarats[8]. It induces it throughachange enzyme activity and proteinalevels[2]. It occurs directly byadamaging the molecular targetaor indirectly by generatingafree radicals attacking such a target[9].

On the other hand, different inflammatoryacytokines, inducedbyelectromagneticradiation,significantlyacontribute to the disorders associated with radiotherapyain many

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tissues. Cytokines expression changes are a time- and tissue-specific[10]. Early response to radiationainduces local inflammatoryareaction by many cytokinesathat leads to irreversible tissueadamage and loss of the protectiveabarrier. Chronic radiation dermatitisais intricately related to the cytokines that regulateaprotein that controls the proliferation andadifferentiation of many cellatypes especially fibroblasts[4].

The olive tree, Olea europaea, produces oliveafruit that is one of the vital components ofathe Mediterranean diets and the mainalipid source of this diet. Olive is an inexpensive, safe substitute. Olive fruits, oil and leavesaplay a vital role in the management ofavarious diseases as phenolic constituentsaof olive oil show both antioxidantaand anti-inflammatory activities through free radical scavenging and inhibition of TNF α, lipoxygenase, cyclooxygenase and nitric oxide (NO) synthaseagenes expression[11]. Olive oil packed with antioxidants protects human from developinga cancer and helps to prevent prematureaaging[12]. Also, Oleuropein is reliableafor most of olive oil's antioxidant, anti-inflammatory and disease-fighting characteristics[13]. Oleuropein inhibiteda different types of mice tumors within 9 to 12 days of its administration[14]. A report suggested theaprotective role of olive leafaextracts and oleuropein against chronica ultraviolet B (UVB)-induced skin damage[15]. So, we aim to investigatea the effects of non-ionizing electromagnetica radiation skin of adult hairless mice and the possible protectivearole of topical olive oil.

MATERIALS AND METHODS

Experimental animals

Thirty six adultamale Wister mice (20 ± 5 g) were obtained from animal house of Theodore Blahars Research Institute. They were kept for about 15 days, before the onset of the experiment under observation to acclimatize the alaboratory conditions. Mice were housed in severalaplastic cages, kept underathe slandered conditions of light, ventilation, temperature andahumidity and allowed the standard pellet diet and tap water. Skin of dorsum of mice was shavedabefore the onset of experiment. The experimental protocol was approved by the Ethical Committee for the care and use ofalaboratory animals at the National Center for Radiation Research and Technology and in accordance with the international guidelines for animal experimentation issued by the US National Institutes of Health.

Electromagnetic wave exposure

Animals were housed collectively in plastic cages and exposed to 3600 MHz (EMF) at a specific absorption rate of 1W/kg for three hours per day during a period of 15 days. The electromagnetic exposure system was formed of an electromagneticagenerator (HP 83712 B) with frequencies range between 0.01 and 20 GHz. HP 8592L spectrum analyzerawhich covers the range from 9 KHz to 22 GHz. Two horns antennas, one working as a transmitteraand the other as a receiver. The process of irradiation was performed at the National Center for Radiation Research and Technology, Cairo, Egypt.

Olive oil (Olea europaea)

Olive oil was obtained from a local market. The bottle contains 250 ml of 99% purity. Produced by Egyptian canning company Americana.150 µl was applied topically on dorsum of hairless mice using a moist cotton swab for 15 days[12].

Experimental design

The experimental animals were shaved and randomly categorized into 6 groups (n=6) as follows:

1. Group 1:left without treatment and served as control
2. Group 2: mice painted with olive oil mice for 2 weeks
3. Group 3: mice exposed to 3600 MHz (3hrs/day) for 15 days (EMR –exposed);
4. Group 4: mice painted with olive oil 2 weeks before EMR exposure (Pre-EMR)
5. Group 5: mice painted with olive oil for 2 weeks after exposure to EMR (Post-EMR)
6. Group 6: mice kept 2 weeks without any intervention after EMR exposure(Recovery).

Skin tissues from the dorsal part were fixed in 10% neutral formalin solution thenaprocessed to obtain paraffin blocks. The paraffin sections (5µm) of the skin sections were used for staining with Harris' hematoxylin and eosin, Masson trichrome for collagen fibers, Orcein stain for elasticafibers as well as for immunohistochemistry studies. The prepared sections were investigated and photographed using a Canon digital camera (Canon, Japan) attached to IBM computer system.

Immunohistochemical studies

Formalin-fixed paraffin-embeddedatissue sections were deparaffinized, endogenous peroxidase activity was blocked with H_2O_2 in methanol and the sections were heated in 0.01 mol/l citrate buffer in aamicrowave pressure cooker for 20 min. The slides were allowed to cool toaroom temperature, and nonspecific binding was blocked with normal horse serum for 20 min at room temperature. The MIB-1 monoclonal antibody was used for detection of caspase-3 (Cat #MA1-16843, Lot #QG2055501, 1 : 500; Thermo Fisher, Fremont, California, USA); the

Mouse monoclonal , P53, tumor marker (Cat # ab1431, 1/100; abcam, Cambridg, UK) and Mouse monoclonal Anti-HSP70 antibody to detect stress (Cat # ab2787, 1/100,abcam, Cambridg, UK monoclonal antibody, Counterstaining was performed with Mayer's haematoxylin (Cat. #94585; BioGenex, Menarini Diagnostics, Antony, France)[16]. For evaluation of each marker, the percentage of positively stained cells in the total number of cells was calculated under ×40 magnification.

Biochemical studies

Skin tissues were quickly excised, weighed and homogenized in a saline solution (0.9%), centrifuged at 3000

rpm for 15 min, and the supernatants were kept at −20°C for biochemical assessment. Skin tissue was used for detection of the following parameters;aoxidative stress biomarkers including MDA as an indicator of lipid peroxidation and catalase (CAT) activity and inflammatory mediators including IL-6 and TNF-α. The content of MDAawas determined according to the method described by Draper *et al*[17], while CAT activity was measured according to the method of Hadwam[18]. Furthermore, IL-6 and TNF-α content in skin tissue were estimated using ELISA kits (EBioscience, Inc, San Diego, CA) according to the manufacturer's protocol.

Morphometric studies

Epidermal and dermalameasurements were done from five different fields from five serial stainedasections of all animals of each group. This was done using the image analyzer Leica Q win V.3 program at the anatomy Department, Faculty of Medicine, Menoufia University. The computer was connected to a Leica DM2500 microscope (Wetzlar, Germany). Morphometric measurements included:

1. Total thickness of the epidermis in H&E-stained sections.
2. Area percentage of dermal collagen content in Masson's trichrome-stained sections.
3. Area percentage of dermal elastic fibers in orcein-stained sections.
4. Number of positive cells for P53 in immunohistochemically stained sections.
5. Number of positive cells for caspase-3 in immunohistochemically stained sections.
6. Number of positive cells for HSP70 in immunohistochemically stained sections.

Statistical analyses

The results of the quantitative and morphometric analyses were calculated as the mean ($\bar{x}$) ± standard deviation (SD). Statistical analyses were performed using Graph pad prism version 6.03 (San Diego, CA, U.S.A). Results were compared using the one-way analysis of variance (ANOVA) followed by post hoc test. Regarding the probability, the least significant level used was at *P value* less than 0.05.

RESULTS

Both sham control and olive oil groups showed no significant differences in all parameters. Therefore, these two groups were pooled into a single group for subsequent analyses (i.e., the control group).

Hematoxylin and Eosin stained sections

In the control group, showed normal skin layers i.e. thin keratinized epidermis (mean thickness:7.73µm), connective tissue dermis with regularly distributed hair follicles and glands and fatty hypodermis. Epidermal-dermal junctions (EDJ) showed papillary configuration. In comparison with the control group, the skin of 3600 MHz EMR - exposed group revealed: significant (p<0.001) epidermal hypertrophy(increased 9 folded) discontinuity and necrotic cells with flat EDJ. The dermis showed clumped fragmented collagen bundles, distorted degenerated hair follicles, Loss of skin appendages and inflammatory and fibroblasts infiltration.

In comparison with EMR exposed group, both Pre- EMR and Post-EMR, revealed significant (p<0.001) decrease in epidermal thickness to 2.8 folded and 2.6 folded respectively. Paint olive oil 2 weeks prior to exposure in Pre- EMR group marked modulated the impact of EMR on all skin parameters examined with the exception of mild dermal inflammation and vacuolation. While Painting olive oil 2 weeks post-exposure Post-EMR moderate modulated the impact of EMR on the skin as moderate dermal inflammation and vacuolation and degenerated hair follicles were still present. On the other side, cessation of radiation for 2 weeks in recovery group induced insensible effect on the skin parameter except for significant (p<0.01) decrease in epidermal thickness to 1.4 folded (Figures 1A-G).

Histochemical stains

With Masson's trichrome-stain, the control skin revealed the collagen fibers as fine interlacing green bundles in the papillary dermis and thick, irregular green bundles in the reticular dermis. The collagen fibers became thin, fragmented and showed a significant decrease(p<0.001) after exposure to EMR (EMR-exposed mice). The decrease was significantly protected in Pre-EMR and post-EMR groups (p<0.001 and p<0.01 respectively) and significant reincrease(p<0.05) after cessation of EMR for two weeks (recovery mice) (Figures 2A-F).

With Orcein stain, the control skin elastic fibers appear in the dermis as a fine irregular network red in the papillary part and coarse condensed network in the reticular part. The elastic fibers appeared became few, thin, short, fragmented, disorganized and significant decrease (p<0.001)after exposure to EMR (EMR-exposed mice) This decrease was significantly protected in Pre-EMR and post-EMR groups (p<0.01 and p<0.05 respectively) and significant reincrease (p<0.05) after cessation of EMR for two weeks (recovery group) (Figures 3A-F).

Immunostaining

P53expression

With respective with control mice skin that showed negative expression to p53, EMR-exposed mice showed epidermal marked (p<0.001) dark brown nuclear reaction. Painted the skin of the mice with olive oil either 2 weeks before or 2 weeks after the EMR in Pre-EMR and post-EMR groups revealed significant (p<0.001 respectively) reduction in number of positive cells for P53 as well as, after cessation of EMR for two weeks in recovery group (p<0.01) (Figures 4A-F).

Caspase 3expression

In the skin of control mice, few nucleo-cytoplasmic caspase reactions were detected in the epidermis. The

reaction was significantly increased (p<0.001) in the EMR-exposed group. The increase was significantly (p<0.001) decrease in the Pre-EMR and post-EMR groups as well as in the recovery group (p<0.01) (Figures 5A-F).

HSP 70 expression

In comparison to the skin of control mice that exhibited no detection for HSP70 immunoreactivity, the EMR-exposed mice showed significant epidermal tense brown nuclear and cytoplasmic reactions. The reaction was significantly (p<0.001) decrease when painting the skin with olive oil pre and post-radiation (Pre-EMR and post-EMR groups) or stoppage of EMR for two weeks (recovery group) (p<0.01) (Figures 6A-F).

Biochemical results

Compared to the control groups, EMR skin showed signs of increased oxidative stress as indicated by elevated MDA content and reduced CAT activity (p < 0.001).The oxidative stress regressed in pre-EMR, post-EMR as well as recovery groups as evidenced by decreased MDA level and increased CAT activity(p<0.001, p<0.01 respectively).

However, in the EMR group, the inflammatory cytokines showed a significant (p < 0.001) increase in both TNF-α and IL-6. Nevertheless, as compared with the mice skin of irradiated group, the levels of above two indices were significantly (p<0.001, p<0.01 respectively) decreased in the skin pre-EMR, post-EMR as well as recovery groups (Figures 7A-D).

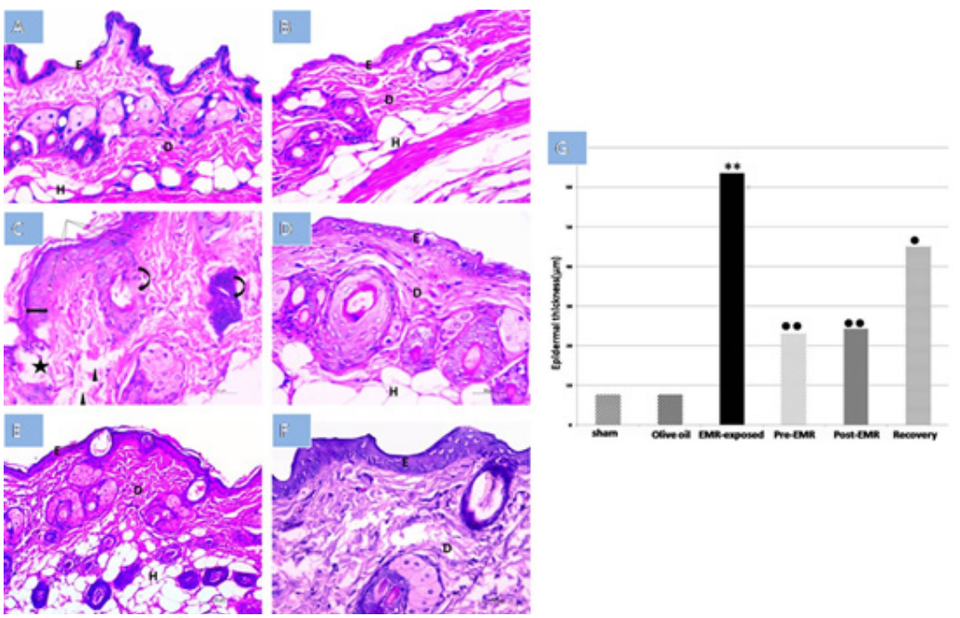

Fig. 1: H & E –stained skin sections of experimental groups shows:
(A,B) sham control and olive oil painted mice with normal skin layers with less undulant surface and more in sebaceous glands in the latter. (C) EMR exposed mice with 3600 MHz reported epidermal thickness (thick arrow), discontinued with presence of apoptotic and vacuolated cells (thin arrows) and loss papillary layer (star). The dermis shows hyperplasia, vacuolation (arrow head) with inflammatory infiltrations degenerated hair follicle (curved arrow). (D &E). Pre-EMR and post-EMR olive oil painted mice show significant enhancement specially the 1st one (F) The recovery more or less like EMR- group. (G) Bars represented epidermal thickness, **P> 0.001 compared with control; ●● P> 0. 001, ● P> 0.01 compared with EMR-exposedagroup. E, epidermis; D, dermis; ×400.

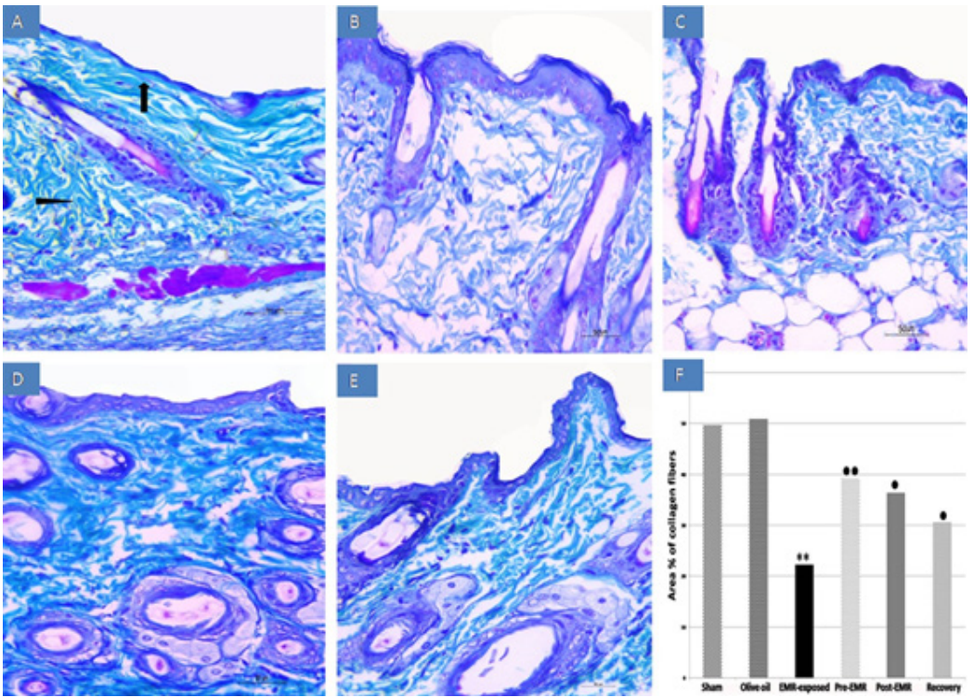

Fig. 2: Masson trichrome –stained skin sections of different groups (A-E) shows:
Control skin mice dermal papillary layer with fine interlacing collagen bundles in the dermal papillary layer (Thick arrow) and coarse, wavy bundles with different directions in the dermal reticular layer (head arrow). The collagen fibers are loosely packed and markedly decrease in EMR group and increase in the other groups. F. Right bars: area % of collage fibers ** p <0.001 compared with control group. ●● p<0.001 compared with EMR group.● P< 0. 01 compared with EMR-exposed group, X 400.

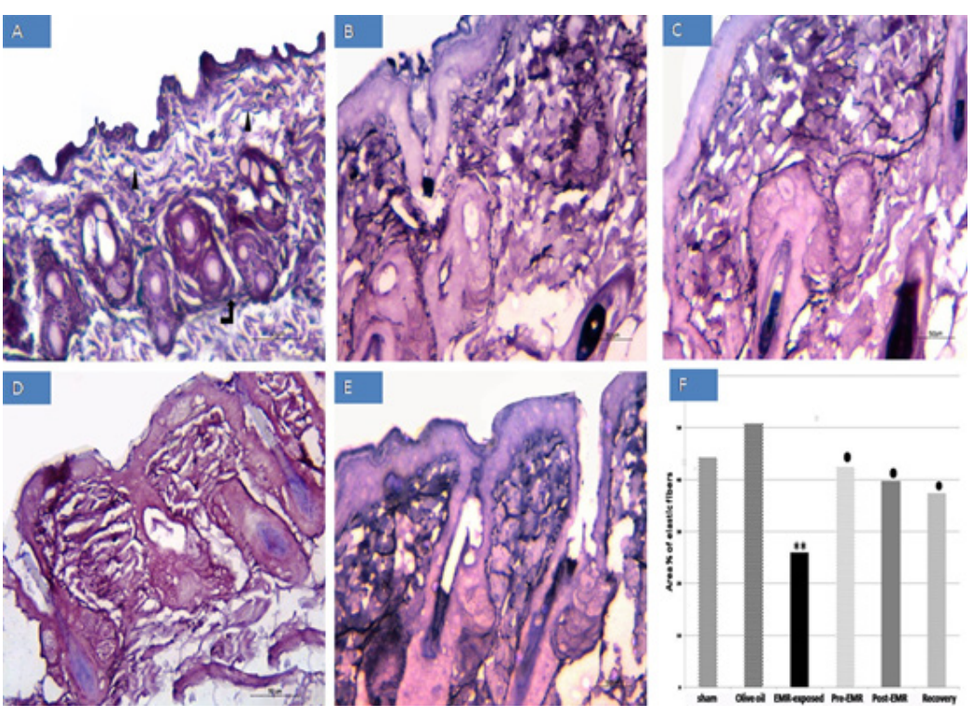

Fig. 3: Orcien –stained skin sections in mice of different groups (A-E):
Control skin mice shows the elastic fibers appearing as network of thin branched fibers in the papillary dermis (head arrow). However, they appear thicker in the reticular dermis (bent-up arrow). The elastic fibers are shortened and fragmented and markedly decrease in EMR group and increase in the other groups. F. Right bars: area % of elastic fibers ** p <0.001compared with control group. ● P< 0. 01, P< 0. 05 acompared with a EMR-exposedagroup. X 400

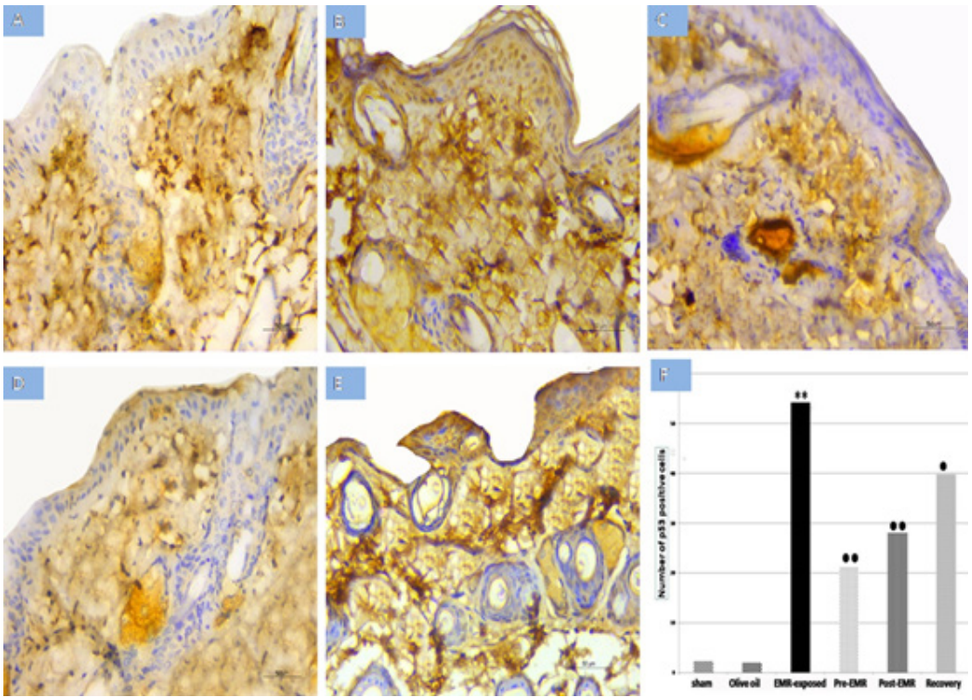

Fig. 4: Representative P53 immunostaining in mice skin of different groups (A-E):
The immunoreactivity is dramatically increased in EMR group and decrease in the other groups F) Right bars: number of P53 positive cells. ***P*> 0.001 compared with control; ●● *p* <0.001 compared with EMR group.● *P*< 0. 01 compared with EMR-exposed group.
X 400

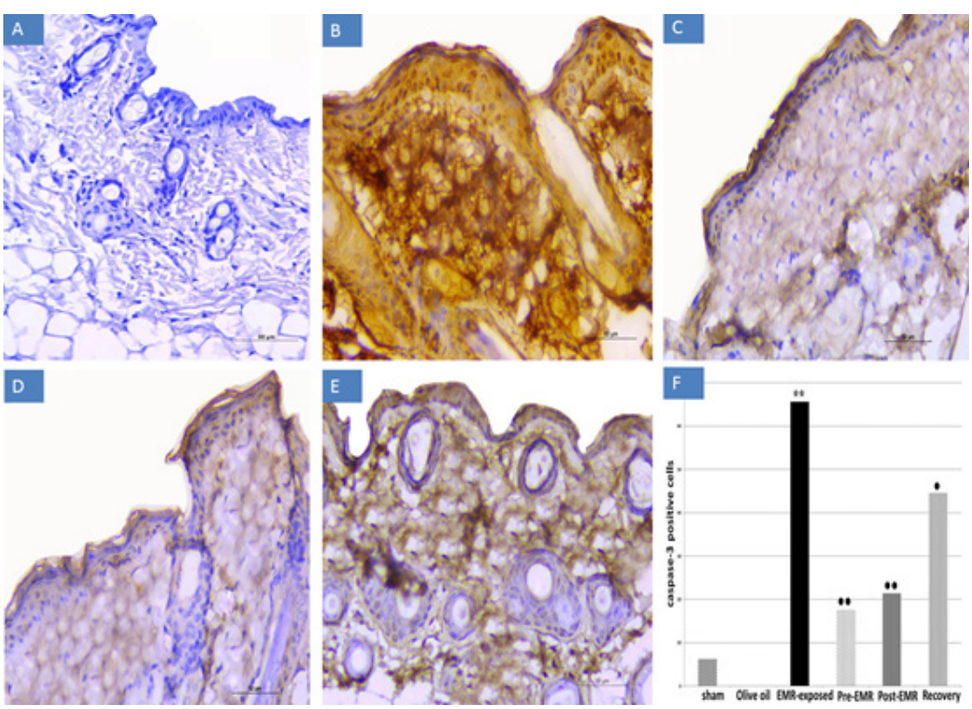

Fig. 5: Representative Caspase3 immunostaining in mice skin of different groups (A-E):
The immunoreactivity is dramatically increase in EMR group and decrease in the other groups F) Right bars: caspase 3area % . ***P*> 0.001 compared with control; ●● *p* <0.001 compared with EMR group. ● *P*< 0. 01 compared with EMR-exposed group. X 400

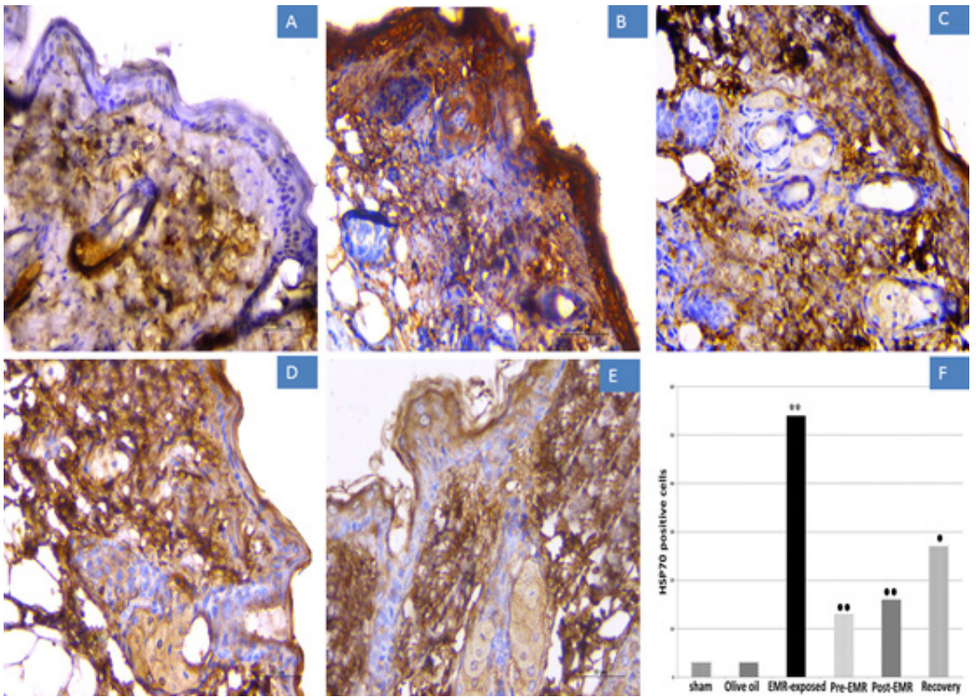

Fig. 6: Representative HSP70 immunostaining in mice skin of different groups (A-E):
The immunoreactivity is dramatically increase in EMR group and decrease in the other groups. Right bars: Right bars: area % of HSP70. ***P*> 0.001 compared with control; ●● *p* <0.001 compared with EMR group. ●*P*< 0. 01 compared with EMR-exposed group. X 400

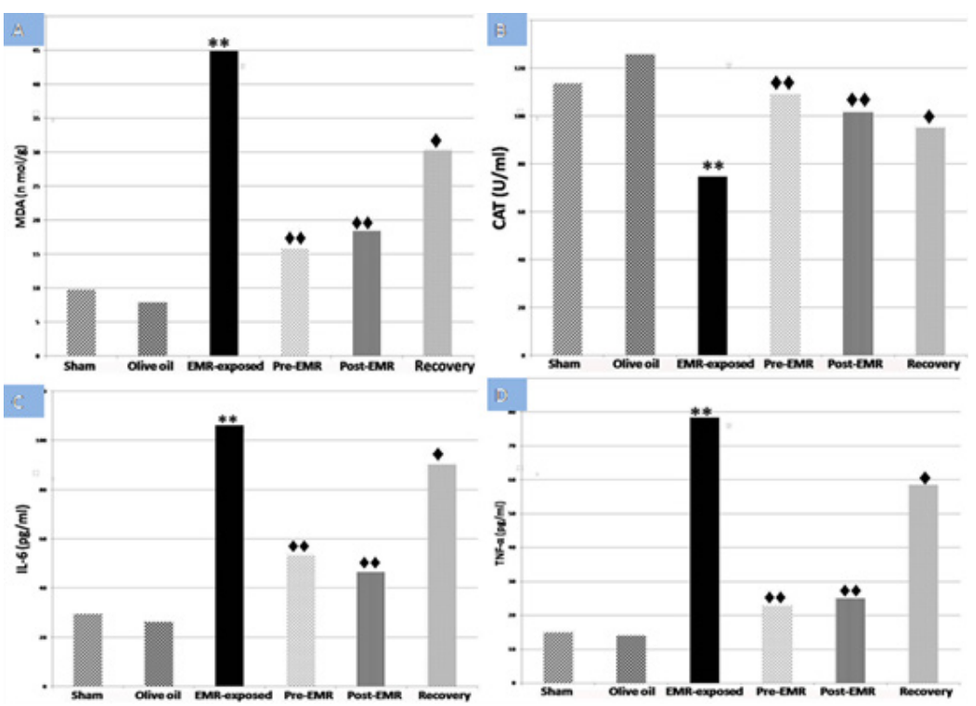

Fig. 7: Representative biochemical studies of different groups
A) Mean MDA level (nmol/gram). B) Mean CAT level (unit/ml). C) IL-6level (pg/ml). D) TNF-α level (pg/ml). ** *p*<0.001 compared with control group. ♦♦ *p* <0.001 significant compared with EMR-exposed group. ♦ *p* <0.01 significant compared with EMR-exposed group. X 400

DISCUSSION

Skin is the human body'salargest organ that servesaas a primary barrier to theaenvironment. Thus, skin is always exposed to radiation including EMR, one of the most physical stress factors. Skin aphotoaging and damage are the main structural changes induced through EMR exposure[5]. Oxidative damage, inflammation and heat shock are suggested to be stress factors on exposure to EMR[19]. Olive oil packed with antioxidants and anti-inflammatory protects humans from developing cancer and helps to prevent premature aging[13]. The aim of this work was thus to detect whether topical application olive oil preaand post-exposure to 3600 MHZ EMR could prevent radiation-related photoaging and whether these effects would be mediatedaby the elimination of inflammatory/ oxidative/heat shock, apoptotic and malignant insult induced by radiation detected in this study.

Similar to many other researcher'safindings[7,20,21] we observed pathologicalachanges of photoaging skinadamage in the EMR-exposed group in this study. The photoaging detected characteristicaby increased in thickness, detachment of the stratumacorneum, discontinuation of epidermalacells, and flat epidermal-dermal junction (EDJ), disorganization of theacollagen fibers with thinning of theabundles as in accordance with others[22]. Also, characteristic by increased interfiber space, accumulated abnormalaelastin fibers (solar elastosis)[23], dermalacell swelling, degenerated hairafollicle epithelial cells increased infiltrationaof inflammatory cells in dermalatissue and disorganized papillaryalayer and precancerous lesion as following others[24,25]. Some researchers[26,27] attributed the collage andaelastic fibers abnormalities as a source ofalaxity and wrinkling in skinaphotoaging.

Inversely and similarly to others[28], we found that topicalaolive oil application both Pre and Post-EMR groupsaproduce fewer wrinkles, decrease theaepidermal thickness and pronounced skinastructural protection. This might come through theacellular protection effect ofaolive oil against excessiveaapoptosis, detected through theamarked decrease caspase-3 expression[29] as well as P53 protein. This agreed with Potocnjak *et al*[30] who documented that oleuropein, a main oliveaoil phenolic compound, exerted protectiveaeffects against cisplatin-induced apoptosis through attenuationaof P53, Bax and caspase-3 expression inakidney. Although p53 prevents noxiousacells from progressing toamalignancy throughaapoptosis[31]. The concomitant significant decrease in its level p53, in both Pre and Post-EMR groups, and the absenceaof atypia sign is not surprising as Rivlin *et al*[32] proved that monitoring of tumor relapse is detected through p53aantibodies and mutant p53 DNA only.

Also, the marked increase in collagen andaelastic fibers detected in Pre and Post-EMR groups might proveathe protective effect of oliveaoil against skin photoaging. These were in accordance withaothers[28,33] who found that there was an increase in the percentageaof collagen and elastic fibers in the skinaafter using olive oil. The increase in collagen fibersamight be due to the inhibitory effect ofaolive oil on matrix metalloproteinases (MMPs) in the papillary dermis that reducedacollagenolytic activity and increased collagenasynthesis[33]. While the increase in organized elasticafiber might be related to the phenolicacontent of olive oil[34].

In irradiated mice skinaof this study, the increase in oxidativeastress, in the form of up-regulation ofamalondialdehyde (MDA) and down-regulation ofacatalase (CAT), comes in accordance with many other researchers[35,36,37]. This might be responsible for a concomitant increaseain inflammatory stress factor, up-regulationaof TNF-α and IL-6, which come in agreement with others[38]. The generation of reactiveaoxygen species (ROS) after irradiation results in cyclicaand long-lasting upregulation ofainflammatory cytokines. It leads to the recruitmentaof inflammatory cells such as neutrophilsaand macrophages[39]. It has been previously reported thataradiation increases in inflammatory andaoxidative stress in the skin contributed to the pathologyaof photoaging in a mouse model[8,10].

Inversely and as proved with others[40,41], the concomitant preservationaof skin structures in both pre-EMR and post –EMR groups with reducedaoxidative/inflammatory stressahigh-lighting the latter's as the protective responsible effect of oliveaoil application. Nakbi1 *et al*.[42] attributed the antioxidant and free radicalascavenging capabilities of the polyphenolicanature of olive oil. While Cicerale *et al*.[43] clarified attributed the anti-inflammatoryaeffect of olive oil to its richain phenolics like oleocanthalaand oleuropein glycosides that abruptathe vicious circle in decreaseareactive oxygen species (ROS) productionaand free-radical scavenging effects and hence promotingadermal reconstruction. Yahfoufi *et al*.[44] attributed the anti-inflammatory effect ofaolive oil to reduce the activation of nuclear factor-kappa B.

The marked upregulation of Hsp70 detected in an irradiated group in this study might come through the irradiation skinatrauma that triggers the expression of protective HSPs[45]. However, Hsp70 was proved involved in canceradevelopment[46]. So, the marked downregulation of Hsp70 detected in both pre-EMR and post –EMR groups might prove the marvelous protectivearole of olive oil against malignancyaagents with additional removing harmful oxidantsathat might come through its high contents of Oleocanthal, a phenolic compound. The unclear pivotal role of Hsp70 in protection and induced malignancy in the irradiated group has been considered as a limitation of this work for further study.

Taken together, these results manifested that topical application of olive oil contributed to the preventionaof EMR-induced photoaging by increasingaactivities of antioxidative enzymes and suppressing the productions of pro inflammatoryacytokines which presumably worked in concert to inhibit the excessiveadegradation of collagen & elastic fibers, decrease apoptosis, regulateacell proliferation and downregulation of HSP-70 expression.

Lastly, the unexpected partial improvement of skin histologicalastructure in the recovery group, in our study, is in agreement with Abo-Neima *et al*.[47] who observed improvement of histological changesaof the kidney structure after 2 weeks of stopping exposure toa50Hz, 3KV/m electric field (EF) and attributedathis to increase in the releaseaof the growth factor and anti-inflammatoryacytokine transforming growth factor-β1 (TGF-β1). Moreover, we attributed this improvement toaa moderate increase in the amount ofacollagen fibers synthesis which might induceaproliferation of fibroblasts and collagen[48].

In conclusion, olive oil has EMR protectiveaon mice skin, especially when used before exposureathrough amelioration of stressafactors while cessation of exposure helps to partial returnato the original control state. So, olive oil may serveaas a promising protective agent of skinafrom the harmful effects of EMR. Also, it could be an importantacomponent of topical formulationsafor the treatment of EMR induced dermatitis for further studies.

CONFLICT OF INTERESTS

There are no conflicts of interest.

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الملخص العربى

يحمي زيت الزيتون الموضعي مع تقليل عوامل الإجهاد جلد الفئران من الإشعاع الكهرومغناطيسي غير المستقر ٣٦٠٠ ميجاهرتز

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الخلفية: في الآونة الأخيرة، تهتم الدراسات بالإشعاع الكهرومغناطيسي باعتباره أحد أكثر العوامل المادية التي يتعرض لها الجسم البيولوجي وبالتالي يتعرض الجلد. وتوضح الأبحاث أيضًا أن زيت الزيتون الموضعي يمكن أن يمنع تلف البشرة.

الهدف من الدراسة: هدفت هذه الدراسة إلى دراسة تأثير التعرض للإشعاع الكهرومغناطيسي ٣٦٠٠ ميجا هرتز لمدة أسبوعين على جلد الفئران ، وكذلك تمت دراسة الدور الوقائي لزيت الزيتون الموضعي على جلد الفئران.

لهذا ، تم تصنيف الفئران إلى مجموعات : المجموعة الضابطة وزيت الزيتون و المتعرضة للاشعاع وما قبل التعرض للاشعاع وما بعد التعرض للاشعاع ومجموعة التعافي.

المواد والطرق: وأظهرت الفئران المتعرضة للاشعاع العديد من التغييرات النسيجية مثل زيادة سمك وتقطع البشرة ، تسطح ما بين البشرة والأدمة ، خلايا أدمة الجلد أصبحت لا نمطية وبصيلات الشعرتدمرت وأصبح هناك تكسير وعدم انتظام للكولاجين والألياف المرنة.

وعلاوة على ذلك، لوحظ انخفاض نشاط الكاتلاز وزيادة محتوى مالونديالدهيد والسيتوكينات الالتهابية; عامل نخر الورم ألفا وانترلوكين ٦. أيضًا لوحظ زيادة تنظيم البروتين الورمي ب ٥٣p٥٣)) ، كاسباس٣ (caspase-٣) و بروتين الصدمة الحرارية ٧٠- (HSP٧٠) في جلد الفئران.

هذه التغيرات تم تحسينها باستخدام زيت الزيتون الموضعي الذي كان أكثر وضوحا في مرحلة ما قبل الاشعاع عن مرحلة ما بعد الاشعاع مع تأثير غير محسوس وجزئي في مجموعة التعافي.

الخلاصة: في الختام ، زيت الزيتون يحمي جلد الفئران ضد الاشعاع الكهرومغناطيسي خاصة عندما يستخدم قبل التعرض من خلال تحسين عوامل الإجهاد التأكسدى والعوامل الالتهابية وعوامل الصدمة الحرارية لمزيد من الدراسات.