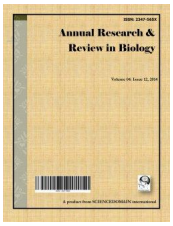

Annual Research & Review in Biology

27(3): 1-11, 2018; Article no.ARRB.42132
ISSN: 2347-565X, NLM ID: 101632869

Photoprotection Comprising Oil Derived from Dromedary Camel Hump Fat

Sabah A. A. Jassim[1*], Atheer A. Aldoori[2], Moutaz A. AbdulMounam[2], Basil R. Faraj[2], Farqad F. AbdulHameed[3] and Richard G. Limoges[1]

[1]Applied Bio Research Inc., Windsor, Ontario, Canada.
[2]College of Veterinary Medicine, University of Baghdad, Baghdad, Iraq.
[3]Epidemiology Division of the Ministry of Science and Technology, Baghdad, Iraq.

Authors' contributions

This work was carried out in collaboration between all authors. Author SAAJ carried out the experiment (design), data interpretation and manuscript writing. Authors AAA, MAA, FFA and BRF conducted the lab work and managed the chemical and histopathological examinations. Author RGL helped in manuscript editing. All authors read and approved the final manuscript.

Article Information

DOI: 10.9734/ARRB/2018/42132
Editor(s):
(1) Dr. Rajeev Kumar, Department of Veterinary Public Health & Epidemiology, Vanbandhu College of Veterinary Science & A.H, Navsari Agricultural University, Navsari, India.
(2) Dr. George Perry, Dean and Professor of Biology, University of Texas at San Antonio, USA.
Reviewers:
(1) Daisy Machado, University of Campinas, Brazil.
(2) Renshan Sun, Third Military Medical University, China.
(3) Stefano Manfredini, University of Ferrara, Italy.
Complete Peer review History: http://www.sciencedomain.org/review-history/25412

Original Research Article

Received 15th May 2018
Accepted 2nd July 2018
Published 5th July 2018

ABSTRACT

Aims: *Camelus dromedarius* (dromedary or one-humped camels) are known to endure harsh conditions including extreme temperatures and high solar ultraviolet (UV) radiation in desert wilderness areas. This remarkable survival in the harsh desert conditions is attributed to distinctive bodily features enabling them to cope with this toxic environment. The present study hypothesized that the oil rendered from camel hump fat, consisting of saturated fatty acids with omega 3, 6, 9 and Vitamin E, has contributed to shield/protect/prevent UVA radiation damage.
Place and Duration of Study: This study was conducted in College of Veterinary Medicine, Baghdad University, Iraq, between June 2011 and July 2012.
Methodology: White BALB/c mice aged about 3 - 4 months weighing 24 - 31 gm were divided into four groups. Mice were shaved and three groups received different treatments of daily exposure to UVA radiation and one group was untreated as a control.

**Corresponding author: E-mail: prof.jassim@yahoo.co.uk;*

Results: Histopathological examinations of mice treated with camel oil prior to or following UVA radiation demonstrated that the camel oil acts as a protective agent, namely, protection of mice skin tissue from radiation-induced apoptosis. The mice treated with oil derived from cows and fat-tailed sheep demonstrated no improvement or worse results than untreated (control) mice. The results suggest that the camel oil protects the mice from UVA radiation injury and also acts as an injury-mitigator when applied following UVA exposure.
Conclusions: The major components in the camel hump fat including saturated fatty acids and noticeable values of omega 3, 6, 9 and Vitamin E have contributed to shield/protect/prevent UVA radiation damage, and may also have unique anti-tumor properties with novel dual radiation-protection and mitigation/healing properties.

Keywords: Camel; camel hump; cancer; fatty acids; omega; radiation; ultraviolet; vitamin E.

1. INTRODUCTION

Camelus 'Camelid' are represented by two domesticated species: the one-humped camel (*Camelus dromedarius*), and the two humped, shorter-legged (*Camelus bactrianus*) camel. While the one-humped camel is found in the hot arid areas of the Middle East, Africa and Eastern Asia, the two-humped camel is found in Central Asia [1,2]. The domesticated one-humped camel, *C. dromedarius*, is one of the most abundant representing 90% of genus *Camelus* in the Arabian Desert [2,3]. They have great tolerance to harsh conditions of high temperatures, solar ultraviolet (UV) radiation, water scarcity and poor vegetation [1]. UV radiation is divided by wavelength into three major types: UVA (315-400 nm), UVB (280-315 nm), and UVC (100-280 nm). Of these, UVA is the most abundant, as it accounts for about 95% of solar UV radiation [4]. UVA penetrates deep into the dermis (5], and has been shown to cause cell proliferation, growth arrest, and apoptosis [4].

Exposure to UV radiation from the sun is a major risk factor for the development of skin cancer and photo-aging skin of humans and animals. However, the dromedary is an exception. The natural habitat for the dromedary is in the desert under direct exposure to burning sunlight and natural UV radiations. These conditions have important health implications for the dromedary, which needs to have special protection against solar UV radiation, enabling them to avoid developing the cancer and tumors associated with genotoxic factors known to cause gross deoxyribonucleic acid **(**DNA) alterations in other organisms. Extrinsic factors such as excessive sun exposure can cause tumors, such as skin malignancies [6] and are believed to play a major causal role in 67% of melanoma cases [7]. Alternatively, intrinsic factors such as genetic errors or mutations may arise from random errors accumulating during DNA replication and chromosome segregation under such harsh conditions. Ionic radiation and sunlight are known producers of Reactive Oxygen Species, one of the causes for DNA lesions and damage to proteins, and lipids [3,4,8,9].

The camel has a unique anatomy; it's the only animal having humps on the torso mainly made up of fat. The fat in a camel is not stored subcutaneously over an extensive area of the animal, as with humans and other animals that store their fat mixed in with muscle tissue or in a layer beneath the skin. In a healthy well-fed dromedary camel, the hump weight can exceed 40 kg, representing (5-13%) of the carcass weight [2]. Camel fat is also present in small proportions in the milk, blood, meat and bones. [2]. Hump and other fat deposits contain mixtures of fatty acids [2] and most of these are esterified as triglycerides or phospholipids and vary according to their anatomical location in the body [2]. Many studies have examined the composition of camel hump fat using thin-layer chromatography, and found the main fatty acid composition of camel hump fat is palmitic acid C16:0 (mole 33.8%), stearic acid C18:0 (mole 25.9%); oleic acid C18:1 (mole 18.1%), along with myristic acid C14:0 (mole 6.3%) [2,10]. In general, camel hump fat contains more saturated fatty acids (74%) than unsaturated fatty acids [2, 10]. This partially accounts for the fact that camel hump fat has a higher melting point than, for example, porcine fat [10]. Furthermore, the component acids of the camel fat of *C. bactrianus* were reported [11]. They found that camel fats are more saturated than the average sheep or ox tallow and reflected mainly in the increased content of stearic acid and decreased amount of oleic acid. Noteworthy the composition of the hump fatty acids is affected by the animal's age. The highest percentage of unsaturated fatty acids and lowest percentage of saturated fatty

acids are found in animals of less than 1 year, whereas the opposite trend applies in animals in the 1-3-year-old age group [2]. The cholesterol in the hump (139 mg/100 g fresh weight) is lower than those in lamb and beef adipose tissues (196 and 206 mg/100g fresh weight), respectively [2].

The camel hump is considered offal from camel; nevertheless, in some African camel producing countries it is considered an important ingredient and commonly used for cooking [2]. Hump fat has been used as a traditional medicine to ease hemorrhoid pains and the hump fat has also been used to remove tapeworm [2]. Other products from the camel including meat, milk, cheese and even urine and dung are considered medicinal in the Middle East and North Africa [2]. For instance, camel milk was reported to cure jaundice, tuberculosis and asthma [2]; oral administration of rennet from camel milk was considered therapeutic for curing impotency in Medieval Persia [2]; and camel dung infusion in water is used to cure earaches and to remove eye cataracts in some parts of Nigeria [2].

The aim of the study is to investigate the photo-protective activity of the oil rendered from the hump fat to determine its impact on the deleterious effects of radiation produced by direct UV light exposure or its ability to treat lesions and tumors induced by damaging radiation. No similar studies have been published on hump fat or oil rendered from the hump fat. A similar rendering process is used to render oil from the fat of each animal involving the application of heat to melt the fat followed by filtration through a simple sieve to remove any solid particles. After cooling, the oil returns to a semi solid state and is applied in similar fashion to an ointment.

2. MATERIALS AND METHODS

2.1 Chemicals

All chemicals used in this study were purchased from Sigma-Aldrich Co (Alcobendas, Madrid, Spain) unless otherwise indicated.

2.2 Animals Studies

All animal studies were conducted in accordance with the requirement of the Institutional Animal Care Committee and have been approved by the Ethics Committee of the College of Veterinary Medicine, University of Baghdad, Iraq.

Both male and female white BALB/c mice aged about 3 - 4 months weighing 24 - 31 gm were randomly divided into four groups of 10 animals and were housed in an animal facility and maintained throughout under standard conditions: 22 ± 2°C, 50 ± 10% relative humidity and 12-h light/12 h dark cycle. The mice were fed a standard diet and watered.

2.3 UV Irradiation

The experimental conditions were designed to simulate severe natural conditions, a solar simulator UV light type (F20 T8 GL 220-240 v UV CE) with a spectral range of 360nm - 370nm corresponding to natural sunlight and environmentally relevant UVA doses were employed. The dorsal shaved mice (2 × 3 cm in length) were clipped to keep animal position and exposed to doses of UVA radiation from about 15 cm from the source of light in a cabinet (75cm long x 50cm width x 75cm high) for 4 h per day for 3 months. During irradiation, the cages were regularly and systematically repositioned below the lights to reduce the variation in radiation intensity (±10%) in different positions and the temperature was stabilized with an electric fan. A digital camera was used to monitor changes resulting from UV exposure and pictures were taken for this purpose. Gross lesions were recorded and specimens of skin 1 x 1 cm were kept in 10% formalin and sent for histopathological examination to study the microscopic changes. Biosafety precautions were followed during the experiment due to the hazardous effect of UV light, such as wearing gloves, cups and masks along with an appropriate hood.

2.4 Preparation of Animal's Oil

Quantities of 2 kg of fresh fat were obtained immediately after slaughtering: an adult male dromedary (from hump), Awassi ram (sheep) (from tail) and a cow 'Friesian Holstein' (body fat). All fats were immediately stored at -80°C. In the experiments, about 500 gm of each fat was defrosted to room temperature. Each type of defrosted fat was then placed in a lab beaker and heated to 60°C for 45 min. The crude melted fat of each animal was cleaned of impurities using a food strainer (25 cm wire mesh sieve). The clear liquid fat from each animal was then collected in a clean sterile container and stored in a fridge at 10°C to hold for use.

2.5 Measurement of Vitamin E in Dromedary Camel's Fat

2.5.1 Preparation of δ-tocopherol standard solutions

Stock solutions of δ-tocopherol standards (10 mg/mL) were prepared in ethanol and kept for one week at -20°C. Spiking solutions of δ-tocopherol standards were prepared on the day of analysis by appropriate dilution of stock solutions. Other tocopherol standards were prepared in the same way. δ-tocopherol was used as internal standard because its chemical structure was like those of the other forms of tocopherol assayed (i.e. α and γ-tocopherol and tocopherol acetate).

2.5.2 Extraction of tocopherols from camel fat

Dromedary camel's fat after defrosting; the specimen was warmed to 38°C and briefly sonicated to ensure sample homogeneity. The fat specimen was extracted using the method described by Korchazhkina et al. [12] in brief as follow: Camel fat (1 gm) was spiked with 10 µL δ-tocopherol standard (7–10 µg). Methanol (1 mL) containing pyrogallol (3%, w/v) then 1 mL aqueous KOH (10%, w/v) were added. Samples were vortex-mixed and placed in a water bath. Saponification was performed at 70°C for 30 min. After the first 15 min of saponification the tubes were briefly vortex-mixed. After cooling of the tubes on ice, samples were acidified to *p*H ~2 with 6 M HCl. Then 4 mL hexane was added. Tubes were vigorously vortex-mixed for 20 s, three times, and kept on ice between mixing. To separate the emulsion the tubes were centrifuged at room temperature at 1300 g for 10 min. The top, organic, layer was carefully transferred to a clean Pyrex container and evaporated, under a gentle stream of nitrogen, on a warm plate at 40°C. The fatty residue was reconstituted in 0.5 mL methanol–propan2-ol (1:1 v/v) with warming to 30°C.

2.5.3 Chromatographic system and conditions

High-performance liquid chromatography **(**HPLC) was performed with a HPLC pump Sykam S 1122 solvent delivery system. Tocopherols were separated on a C18 column Supelco (250 × 4.6 mm, 3.5 nm). The mobile phase was initially a linear gradient of acetonitrile in water (from 95 to 100% in 10 min) then 100% of acetonitrile for 10 min; the flow rate was 1 mL/min. The column temperature was 45°C and the wavelength-range scanned was 275– 350 nm using Sykam S3240 UV multichannel detector. Samples were kept in an autosampler at 30°C and the injection volume was 25 µL using auto injector Sykam S 5200 and oven Sykam S4011 column thermo controller. Peak areas of tocopherols were integrated at 244 nm. Tocopherols were identified by coelution after spiking the extracted samples with solutions of the tocopherols in ethanol. To ensure reproducible retention on the column, after each batch of fat extracts the column was washed with propan-2-ol (1 mL/min for 60 min at 45°C). To evaluate the recovery of the δ-tocopherol spike after extraction, 10 µL spiking solution was diluted with 0.5 mL methanol–propan-2-ol (1:1, v/v) and 25 µL of this solution was injected on the column. Peak areas of δ-tocopherol spikes in camel fat extracts were expressed as percentages of the peak areas of δ-tocopherol solutions prepared as described above. The amounts of native α and γ-tocopherols were calculated on the basis of their peak areas and the percentage recovery. The latter was calculated for each sample using the peak area of δ-tocopherol spike. Regression analysis was performed using Minitab software. The difference was considered significant if $P < 0.05$. Means and standard deviations are reported.

2.6 Animal Treatment

In this research the animals were shaved and then divided into four groups with each group receiving treatments as follows:

1. Group A: Unprotected, daily exposure to UV light for 4 h over 3-month period
2. Group B: Prior to the similar daily exposure to UV light for 4 h over 3 months, each mouse was treated topically on the dorsal skin with about 1 gm of camelid oil to create a thin film of oil on the nude skin. The order of treatments was systematically rotated to ensure comparability of timing of applications, which were always completed within 5 min of the start of irradiation.
3. Group C: After unprotected daily exposure to UV light for 4 h over 3 months, similar to Group A, camelid oil was applied topically each day for the subsequent 3 months.
4. Group D: Control group with neither camelid oil nor UV exposure.

Mid dorsal skin fold thickness was measured regularly during the three-month test period in

all the four groups (A-D). Tumor appearance and growth was mapped and recorded for each mouse for tumors of diameter 1 mm or greater.

2.7 Histopathological Examination

All test animals were sacrificed by inhalation of chloroform (Memmert, Germany) and postmortem examinations were completed. Skin tissue was sampled and processed according to the following procedures. All skin tissue samples were dissected to an equal size of 1 x 1 cm square. Each piece of tissue was immediately fixed in buffered 10% formalin saline (BDH, England). Subsequently, all the pieces were separately labeled and processed together, encased in a Shandon Elliot Histokinette tissue processor (Shandon Southern Products Ltd, Runcorn, UK). Sets of histological sections were stained with hematoxylin stain (Thermo-Shandon, UK) and Eosin stain (Panreac Quimica, Spain). The histological evaluation was performed using an Olympus BX 40 light microscope.

3. RESULTS

3.1 Evaluation of Vitamin E 'δ-Tocopherol' in Camel Oil

Typical chromatograms recorded at λ = 244 nm showing the elution profile of δ-tocopherol in camel hump oil and standard δ-tocopherol are shown in Fig. 1 (A and B). δ-Tocopherol was therefore used in the study as an internal standard for quantification of recovery and levels of other tocopherols. Obviously, the native amount of δ-tocopherol in camel oil is small in comparison with the amount of δ-tocopherol added as the internal standard. The evidence presented herein suggests camel hump oil contains vitamin E by comparing the chromatogram analysis of sample with chromatogram of standard vitamin E, where the retention time for the vitamin E in camel hump (Fig. 1A) asymptotic to the retention time for standard vitamin E (Fig. 1B), as shown in Table 1.

Table 1. The retention time for the vitamin E (δ-tocopherols) in camel hump oil and standard sample

Peak area	Retention time/min	Sample
1551	15.73	Vitamin E from camel hump
10798	15.13	Standard vitamin E

The retention time for the vitamin E (δ-tocopherols) in camel hump oil compared to δ-tocopherols standard sample was satisfactory within the physiological range of concentrations for extraction procedure including saponification, extraction, and HPLC analysis (Table 1). It seems that after saponification the hump oil matrix does not affect the recovery of vitamin E. Levels of the major form of vitamin E, δ-tocopherol, in hump oil sample was variable within the range of 0.003% - 0.00932% (calculated from Fig. 1 and Table 1 with that obtained from chemical analysis in a private lab).

The chemical analysis of camel hump oil (Table 2) conducted in a private lab showed that the saturated fatty acids were 66%, clearly demonstrating that camel hump oil contains a higher proportion of saturated fatty acids than the fats of other animal sources.

Table 2. Fatty acid composition in camel hump fat

Fatty acid composition	Percentage (%)						
	Camel hump fat	Butter fat*	Beef tallow*	Pork lard*	Poultry fat*	Fish oil*	Sheep tail fat†
Saturated fatty acids	66	65	56	44.5	30.5	24	54.8
C-18:1 Oleic acid omega 9	26.4	25	36.5	40	40	14	46.55
C-18:2 Linoleic acid omega 6	1.53	3	2.5	12	21	1.5	2.9
C-18:3 Linolenic acid omega 3	0.581	0.5	0.5	1.0	2.5	1	2.55
Unsaturated fatty acids %	32.6	33	43.5	57	69	73.5	57.7

*[13], †[14]

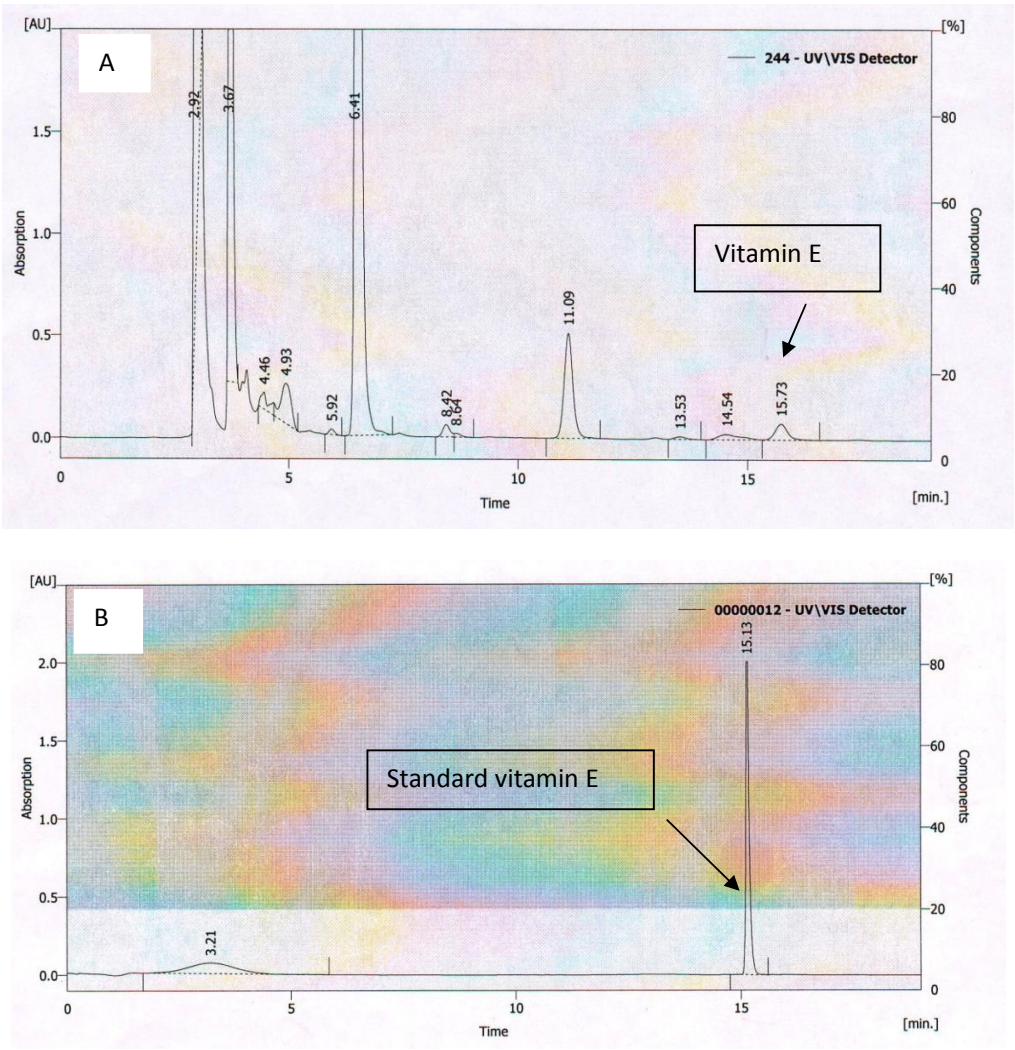

Fig. 1 (A and B). Chromatograms at 244 nm of vitamin E extracted from camel oil using extraction conditions as described. Arrows indicate the elution times for camel hump oil δ-tocopherols (A) and standard δ-tocopherol (B)

A comparison of observations on color, texture, and absorption of each animal's oil were carried-out as follows at room temperature (20 to 25°C):

- Oil derived from dromedary hump fat has a white color with a soft creamy texture that absorbs quickly on skin.
- Oil derived from fat-tailed sheep has a whitish color with a soft creamy texture that absorbs less on the skin than that of camelid fat cream.
- Oil derived from cow fat has a yellowish color with a soft creamy texture that absorbs much less on skin than that of camelid fat cream.

3.2 Histopathological Examinations of Animals Treated with Oil from Camel Hump Fat

Histopathological examination of the skin from the mice and analysis of tissue sections were performed (Fig. 2). Group A (plate 1-6), following three months of radiation, animals had formation of cells with pyknotic nucleus and shrunken eosinophilic cytoplasm, (sunburn cells) in the epidermis of UV irradiated mice. There were also neutrophil and macrophage infiltration in subcutaneous tissue and around hair follicles, edema along and between muscle fibers and fibrin network deposition in adipose tissue.

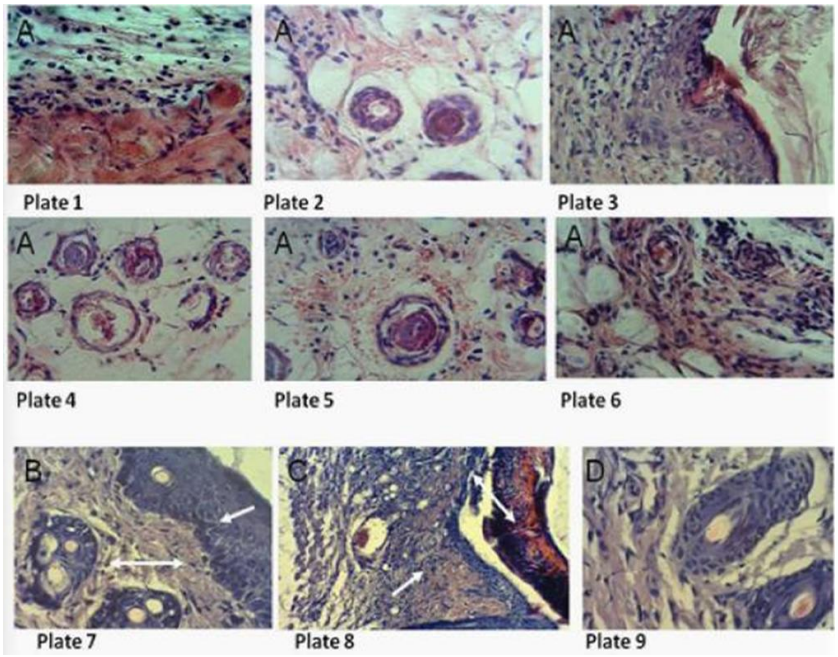

Fig. 2. Histopathological examination sections of mice tissues were collected from each mouse group. Group A following 3 months of radiation, the plate 1-6. Group B (with natural oil application prior to daily exposure) the protected skin of mice in plate 7. Group C (natural oil applied topically daily for 3 months following exposure) plate 8. Group D control untreated on plate 9

The sunburn cell production and focal inflammation were clearly visible. Erosion, necrosis of epidermal layer, focal hyperplasia of epithelial layer with hyper chromatic nuclei neutrophils and mononuclear cell infiltration in the dermis layer were also visible, as well as necrosis of the epithelial layer of hair follicle and sweat gland with neutrophils in their lumen in addition to edema and neutrophil infiltration between sweat gland and hair follicle. Signs of hemorrhage, congestion of blood vessels, edema, neutrophil infiltration, and cellular debris in the lumen of the hair follicle are present. Fibrous connective tissue proliferation and mononuclear cell infiltration in the adipose tissue were also clearly visible. It was clearly evident that the mice in this group have developed tumors (8 mm in diameter, data not shown).

Group B (plate 7, with camelid oil application prior to daily exposure). The protected skin of mice showed moderate mononuclear cell infiltration in the dermis layer and congestion of the blood vessel with normal epidermal layer. There was a normal structure of epidermal layer and limited mononuclear cell infiltration in the dermal layer, and focal aggregation of mononuclear cells around hair follicles, which showed active fibroblast and normal structure of capacious gland.

Group C (plate 8, camelid oil applied topically each day for the 3 months following exposure) showed clearly reduced necrosis and erosion of the epidermis, covered by cellular depression and inflammatory cell infiltration in dermis and subcutaneous layer. There was normal fibrous connective tissue proliferation, mononuclear cell infiltration between adipose tissue and muscular layer. The tumors have shrunk to less than 8 mm in diameter (data not shown).

Group D (plate 9, control), the histological section of skin of normal animal, not exposed to UV radiation, showed tissue with normal hair follicles.

The results of these *in vivo* experiments indicated that camelid oil prevented UV damage to the skin tissue in group B mice. The average skin protection rate of mice treated in group B with camelid oil was indistinguishable from healthy, normal mice in the control group D. The histopathological examinations and daily observations also supported this finding. The camelid oil had a noticeable healing effect on the

skin of group C mice. The cells and tissue appear to be rejuvenated as compared to those from group A. Based on the present results; it appears that camelid oil has the ability to repair UV damage.

3.3 Histopathological Examinations of Animals Treated with oil from Fat-Tailed Sheep and Cows

The results of histopathological examinations sections (were not shown) as follows: With sheep oil application prior to daily exposure, the skin of mice is showed neutrophil infiltration around hair follicles and necrosis of muscle fibers, neutrophil infiltration with edema and congestion of blood vessels, hemorrhage and neutrophil infiltration within muscle fibers, neutrophil infiltration between hair follicles and vacuolation in the hair follicles, neutrophils infiltration in the dermis and in the dilated blood vessels, necrosis, neutrophil infiltration, erosions and sloughing of epidermal layer, destruction in the walls of hair follicles, edema and neutrophil infiltration.

With cow oil application prior to daily exposure the skin of mice showed congested and dilated blood vessels with neutrophils in their lumen, severe congestion of the blood vessels, with neutrophils in their lumen in the subcutaneous tissue, in addition to neutrophil infiltration, severe hypersensitivity of the hair follicles, marked neutrophil and mononuclear cell infiltration in the subcutaneous, proteinous material and neutrophils infiltrations in the necrotic epidermal layer.

The mice that were treated with the oil derived from fat-tailed sheep or cows did not demonstrate any positive effects of treatment or did not survive the initial phase of the experiment and thus results are not reported in greater detail.

4. DISCUSSION

The current study was inspired by the camel *C. dromedarius* having survived extreme conditions in desert areas, such as the Arabian Peninsula desert, Sahara in Northern Africa. The desert sands also reflect a great deal of radiation since the sunshine is mainly positioned directly overhead during the hottest time of day.

Generally, ionic radiation causes damage and/or alterations in the DNA of living cells, resulting in mutation, cancer and cell death [3]. This condition certainly requires extreme adaptations in nature for the camels. Camels bear distinctive fatty deposits known as "humps" on the torso, but not all around the body as in most other animals. Their remarkable survival in the harsh conditions in the desert is due to the distinctive features of the hump, namely, the camel fat [15]. We predict that the hump fat at least partially liquefies into oil during periods of extreme heat allowing the oil to circulate through the internal organs and epidermis of the camel, thereby protecting the animal when it needs it most, however, further investigation is needed to confirm this predication.

The current study, is based on the international patent [15], we have investigated the oil derived from *C. dromedarius* hump fat for its ability to prevent (shield) or treat mice exposed for UVA light for 4 h over 3-month period. The results from the above study showed that the camelid oil application prior to daily exposure (Group B) could protect the skin of mice that showed moderate mononuclear cell infiltration in the dermis layer and congestion of the blood vessels, with a normal epidermal layer and limited mononuclear cell infiltration in the dermal layer. There were no clear lesions in the hair follicle and absence of skin tumors (Fig. 2, plate 7). This is despite that UV radiation is carcinogenic and a principal cause of skin cancers. In comparison, unprotected mice (Group A) showed neutrophil infiltration within adipose tissue and inflammatory cells infiltration around sebaceous gland with calcification, congestion of dilated blood vessels with neutrophils in their lumen and edema in the interstitial tissue, abscess formation in subcutaneous tissue, neutrophil infiltration and fibrin deposition around blood vessels, necrotic areas surrounded by inflammatory cells and fibroblasts (Fig. 2, plate 1-6). In control animals, the histological section of skin of normal animals (Group D), not exposed to UVA radiation, showed tissue with normal epidermal hair follicles and sebaceous gland with no lesion in adipose tissue and muscular layer of the skin (Fig. 2, plate 9).

Clearly, the camel's hump oil was highly effective on mice in treating or preventing skin damage from UVA radiation. This is despite that it has been shown that UVA induces skin carcinogenesis in mice [16]. Exposure to UV's genotoxic potential is linked to its ability to provoke direct DNA damage [3]. Knowing that the transmission of UVA penetrates the basal level of the epidermis, which is of greatest

concern to humans, especially as depletion of the ozone layer causes higher levels of this radiation to reach the planet's surface [17,18].

In contrast, the oil from sheep fat and cow fat did not protect mice's skin from radiation and in some cases the application of cow fat oil resulted in a large tumor. It appears that camel hump fat prevents (blocks\shields) developing of tumors caused by UVA in group B animals. Furthermore, it was observed that tumors caused by UVA radiation in group C were shrunk to negligible size compared to before the treatment with camel hump oil (Data not shown). It appears that camel oil could act as a radio protector, (radio protector: are compounds that are designed to reduce the damage in normal tissue caused by radiation), since when it is administered before exposure it prevented radiation-induced cellular and molecular damage (Group B, Fig. 2 plate 7) and at the same time it could be considered a radiation mitigatory, (mitigators: agents delivered at the time or after irradiation is complete but prior to the manifestation of normal tissue toxicity), since camel oil administered topically after radiation exposure accelerated recovery or repair to the injury and or tumors caused by radiation (Group C, Fig. 2 plate 8). In general, the requirement of topical administration on skin prior to irradiation suggests induction of fatty acids including saturated and unsaturated fats including omega 3,6 and 9 and the vitamin E 'δ-tocopherol' present in camel oil responsible for protection. The naturally occurring δ-tocopherol in camel oil may induce protective anti-radiation activity. The vitamin E 'tocopherol' has been investigated for its radioprotective efficacy using various *in vitro* and *in vivo* models and it was found that tocopherol inhibits radiation-induced apoptosis and DNA damage while enhancing cell proliferation [19,20].

This study surmises that oil derived from camel hump fat may have a unique protective role since fat-containing adipose tissue is suitable for insulation purposes, since fat conducts heat slower than water. Also, blood flow can be very low in adipose tissue, because the fatty deposits do not need oxygen. This further contributes to the insulator function of the hump. Thus, the dromedary camel hump fat may provide an additional insulation from the sun and UV radiations. Therefore, our hypothesis is that since the camel fat may absorb high-energy ultraviolet rays and release the energy as lower-energy rays, thereby preventing the damaging ultraviolet rays from reaching the skin. So, we may surmise, that upon exposure to UVA light, the camel hump fat does not undergo significant chemical change, allowing these ingredients in the camel fat to retain the UVA absorbing potency without significant photo degradation.

It is well known that omega-3 fatty acids can play a role in decreasing skin damage from UV light and the production of cancer cells caused by the UV light. Skin benefits from omega-3 fatty acids in the epidermis, the skin's outer layer.

The current study showed that vitamin E, δ-to copherol present in camel hump fat in the level of 0.003% - 0.00932%. To the best of our knowledge this is first time to report that camel fat contains vitamin E. However, vitamin E was reported in camel milk (129.9 ±26.2 mg l^{-1}) [21]. Vitamin E is well known for its established health benefits, including antioxidant, neuroprotective, anti-inflammatory, cholesterol-lowering properties, diabetes, and cardiovascular and has potential in both the prevention and the treatment of cancer and/or block tumor-induced angiogenesis [22-25]. The current study demonstrated that the camel hump fat is rich in saturated fatty acids with a significant proportion of omega groups 3, 6 and 9. This result is consistent with the previously reported studies [2, 10]. Surprisingly, it was found that the chemical composition, alongside the proportions is the same in both camel fat and butter (Table 2).

From the above, the results indicated that the chemical compositions in the camelid hump fat oil have significant antiradiation properties as demonstrated on lab animals radiated with UVA for 3 months. This antiradiation property was not found with oils from other animals' fats (beef and sheep, Fig. 2). Olive oil was often used as a source of free fatty acid due to its high oleic acid and squalene content, which can help protect skin against free radical-generated damage induced by UV light [26]. Certainly, oil from camel fat (fatty acids composition) needs further investigations to confirm anticlastogenic activity. From the present study and [15], it appears the additional function of the hump is probably to insulate the camel from solar radiation.

5. CONCLUSIONS

The fat in camels is mainly deposited in the hump. Rather, it is stored in the hump on the back of the animal, which is mainly made up of fat. We believe that hump fat has enabled camels to live in the very hot and dry climate

under direct exposure to burning sunlight and natural UV radiations.

The histopathological examinations of the mice treated by oil from fat-tailed sheep and cows tallow concluded that such treatments failed to protect mice from radiation. Whilst, the histopathological examination of the mice treated with camel oil derived from the hump fat could shield UVA radiation and revive cells by repairing damage from UVA, thus exhibiting cancer treatment properties.

Vitamin E, δ-tocopherol was detected in oil from camel fat. To our knowledge, this is also the first study to have shown that camel hump fat contains noticeable levels of vitamin E. The major fatty acids present in camel hump fat are the saturated fatty acids with noticeable prorations of omega 3, 6, 9. We believe that one or more of these fatty acids and vitamin E, δ-tocopherol either in combination, may have a significant effect in shielding against UVA radiation with novel anti-tumor activity.

The therapeutic composition comprising fat derived from a member of the *Camelus* genus needs further studies to include its effectiveness on UV radiation including but not limited to X-ray, microwave, and nuclear radiation. The current, and other [15] studies suggest that one role of a camel's hump is to protect the camel from solar UV radiations in the extreme conditions of the desert.

Furthermore, the results demonstrate that camel oil has dual potentials: (1) radioprotectors, protecting tissues from radiation injury by simple topical administration prior to exposure to UVA-radiation thus improving structural integrity, inhibiting apoptosis, and enhancing cell proliferation in skin tissue in mice; and (2) mitigator-protector when administered on mice after radiation exposure. Therefore, the above results suggest that camel fat or oil may merit further development using appropriate large animal models for the purpose of determining its safe uses and as an effective pharmacologic countermeasure to reduce the therapeutic effect of radiotherapy treatments on cancer patients or for people at risk of acute radiation exposure such as the military, aviation or for those exposed after a nuclear accident. It could be tested as a radiomitigator and for therapeutics in mass-casualty scenarios resulting from nuclear accidents or terrorist attacks involving nuclear radiation or radiological devices.

There is continued interest and need for identification and development of non-toxic and effective radio protective natural products. Although camels have been successfully used in camel racing [1] and recently as an economically productive animal, they are neglected as an animal with the potential for medicinal production and health care products [1]. Recently, Applied Bio Research Inc. in Canada, has produced the first camel ointment, CamOleum to treat burns using camel oil extracted from the camel hump using a process that is protected by international patent [15].

ACKNOWLEDGEMENTS

We would like to thank the Applied Bio Research Inc., Canada for financial support. The support of the Dr. Ibrahim AH Al-Zubaidy at College of Veterinary Medicine, University of Baghdad, Baghdad, Iraq.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Jassim SAA, Naji MA. The desert ship: heritage and science. Biologist (UK). 2001; 48(6):268-72.
2. Kadim IT, Mahgoub O, Faye B, Farouk MM. Camel meat and meat products. Oxford: CABI; 2013.
3. Ataya FS, Fouad D, Malik A, Saeed HM. Molecular cloning and 3D structure modeling of APEX1, DNA base excision repair enzyme from the camel, *Camelus dromedaries*. Int J Mol Sci. 2012;13(7): 8578–96.
4. Sample A, He Y-Y. Autophagy in UV damage response. Photochem Photobiol. 2017;93(4):943-55.
DOI: 10.1111/php.12691
5. Bruls WA, Slaper H, van der Leun JC, Berrens L. Transmission of human epidermis and stratum corneum as a function of thickness in the ultraviolet and visible wavelengths. Photochem Photobiol. 1984;40(4):485-94.
6. Wu S, Powers S, Zhu W, Hannun YA. Substantial contribution of extrinsic risk factors to cancer development. Nature. 2015;529:43–7.
7. Hodis E, Watson IR, Kryukov GV, Arold ST, Imielinski M, Theurillat JP, et al. A

landscape of driver mutations in melanoma. Cell. 2012;150(2):251-63.
8. Ming M, Shea CR, Feng L, Soltani K, He YY. UVA induces lesions resembling seborrheic keratoses in mice with keratinocyte-specific PTEN down regulation. J Invest Dermatol. 2011;131(7): 1583–86.
DOI: 10.1038/jid.2011.33
9. Zhao Y, Zhang CF, Rossiter H, Eckhart L, König U, Karner S, et al. Autophagy is induced by UVA and promotes removal of oxidized phospholipids and protein aggregates in epidermal keratinocytes. J Invest Dermatol. 2013;133(6):1629–37.
DOI: 10.1038/jid.2013.26
10. Haasmann SO. Analytical characterization of camel meat and milk fat. A thesis submitted for the degree of Doctor of Philosophy. University of Brunei: Department of Chemistry; 1998.
11. Gunstone FD, Russell WC. Animal fats. 4. The component acids of crocodile fat. Biochem J. 1954;57(3):462–65
12. Korchazhkina O, Jones E, Czauderna M, Spencer SA, Kowalczyk J. HPLC with UV detection for measurement of vitamin E in human milk. Acta Chromatographica. 2006;16:48–57.
13. Woodgate SL, Veen JTV. Fats and oils – animal based. In: Clark S, Jung S, Lamsal B, editors. Food processing: Principles and applications. Wiley Blackwell. 2nd Edition. 2014;481-97.
14. Mehran M, Filsoof M. Fatty acid composition of sheep tail-fats from five Iranian native breeds. Eur J Lipid Sci Technol. 1976;78(5):187-189.
15. Jassim SAA, Aldoor AA, Limoges RG. Camelid compound(s), composition(s) and method(s). WO 2014135934 A1; 2013.
16. de Laat A, van der Leun JC, de Gruijl FR. Carcinogenesis induced by UVA (365-nm) radiation: the dose-time dependence of tumor formation in hairless mice. Carcinogenesis. 1997;18(5):1013-20.
17. Clancy S. DNA damage and repair: Mechanisms for maintaining DNA integrity. Nature Education. 2008;1(1):103.
18. Jassim SAA, Limoges RG. The impact of changing environmental forces on cyanophage-host interactions in aquatic ecosystems. World J Microbiol Biotechnol. 2013;29(10):1751-62.
DOI: 10.1007/s11274-013-1358-5
19. Singh VK, Beattie LA, Seed TM. Vitamin E: tocopherols and tocotrienols as potential radiation countermeasures. J Radiat Res. 2013;54(6):973-88.
DOI: 10.1093/jrr/rrt048
20. Nukala U, Thakkar S, Krager KJ, Breen PJ, Compadre CM, Aykin-Burns N. Antioxidant tocols as radiation countermeasures (Challenges to be addressed to use tocols as radiation countermeasures in humans). Antioxidants. 2018;7(2):33.
DOI: 10.3390/antiox7020033
21. Kaskous S, Pfaffl M W. Bioactive properties of minor camel milk ingredients-an overview. J Camel Pract Res. 2017;24(1):15-26.
22. Miyazawa T, Tsuzuki T, Nakagawa K, Igarashi M. Antiangiogenic potency of vitamin E. Ann N Y Acad Sci. 2004; 1031:401-4.
23. Nesaretnam K. Multitargeted therapy of cancer by tocotrienols. Cancer Lett. 2008;269:388–95.
24. Aggarwal BB, Sundaram C, Prasad S, Kannappan R. Tocotrienols, the vitamin E of the 21st century: Its potential against cancer and other chronic diseases. Biochem Pharmacol. 2010;80(11):1613–31.
25. Kannappan R, Gupta SC, Kim JH, Aggarwal BB. Tocotrienols fight cancer by targeting multiple cell signaling pathways. Genes Nutr. 2012;7(1):43-52.
DOI: 10.1007/s12263-011-0220-3
26. Ching K. Enzymatic production of feruloylated acylglycerols from olive fatty acid distillates as sunscreen agents. A thesis submitted for the degree of Masters. Universiti Putra Malaysia; 2008.

Peer-review history:
The peer review history for this paper can be accessed here:
http://www.sciencedomain.org/review-history/25412