Restorative effects of camellia oil on the skin-barrier function in a model of DNCB-induced atopic dermatitis

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ABSTRACT

This study aimed to evaluate the therapeutic efficacy of camellia oil on 2,4-dinitrochlorobenzene (DNCB)-induced atopic dermatitis (AD) in mice, as well as its effect on the expression of skin-barrier-related proteins. A mouse model of AD was created via topical application of DNCB; subsequently, the animals were randomly divided into four groups: the blank control (Control), model (Model), moisturizing cream (Moisturizer), and camellia oil (Camellia) groups. The Camellia group received camellia oil, whereas the Moisturizer group was treated with moisturizing cream, as a positive control. Skin lesions, ear and back tissue morphology, and the serum levels of IgE, IL-4, and IFN-γ were analyzed. Compared with the Control group, AD mice exhibited erythema, papules, dryness, peeling, and significantly higher serum IgE and IL-4 levels. Compared with the Model group, treatment with camellia oil and moisturizing cream considerably reduced skin inflammation, ear thickness, and scratching frequency. A histopathological analysis revealed that camellia oil reduced inflammatory-cell infiltration and edema in the AD-affected skin. Furthermore, camellia oil upregulated filaggrin (FLG), thus aiding in skin-barrier repair. These findings suggest that camellia oil significantly improves AD symptoms, enhances FLG expression, and restores the damaged skin barrier in AD mouse models.

Key words: camellia oil; atopic dermatitis; DNCB; skin barrier; filaggrin; loricrin.

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Ethics approval: the experiment was authorized by the Animal Ethics Committee of the Fifth people's Hospital of Hainan Province (approval no. AF-SW-07-1.0). All the experimental processes were operated in accordance with the requirements of experimental animal ethics, and humane care was given to the animals.

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Introduction

Atopic dermatitis (AD) is a chronic inflammatory skin condition that affects individuals of all ages and ethnicities. Its prevalence varies significantly across countries and age groups, with approximately 20% of children worldwide being affected by AD.[1] In Europe, its prevalence among adolescents aged 13-14 years ranges from 1.8% in Lithuania to 17.3% in Hungary.[2] According to the results of the latest epidemiological survey, the global prevalence of AD is 2.62%. More specifically, the prevalence of AD in children (3.96%) is higher than that in adults (1.95%), and the prevalence of AD in women (2.80%) is higher than that in men (2.44%).[3,4] In addition to common skin lesions, AD symptoms include itching,[5] pain, sleep disturbances,[6] and, in severe cases, mental-health conditions, such as depression and anxiety.[7] AD is often considered as the first manifestation of atopic diseases, with many susceptible patients later developing conditions such as asthma, allergic rhinitis, conjunctivitis, or food allergies.[8,9] This multisystem involvement severely impacts the quality of life of those affected by it.

AD is a highly heterogeneous disease with complex causes, including genetic, environmental, and immune factors, which contribute to skin-barrier dysfunction and immune dysregulation. The destruction of the skin barrier in patients with AD facilitates the entry of allergens and infectious pathogens into the skin, thereby triggering immune reactions and inflammation; the immune system of patients with AD exhibits abnormal reactions, abnormal activation and migration of immune cells (such as T cells and dendritic cells), and the release of inflammatory mediators, which jointly promote the occurrence and development of inflammation. Moreover, an obvious family clustering is observed among patients with AD, and specific genetic mutations such the ones in the filaggrin (*FLG*) gene are related to impaired skin-barrier function and immune-system abnormalities. Environmental factors (such as climate change, air pollution, lifestyle, *etc*.) may aggravate the symptoms of AD or trigger the onset of the disease; certain allergens (such as dust mites, pollen, *etc*.) may also induce or aggravate AD.[10] The current treatment strategies for AD are tailored to manage disease severity. Mild cases are managed using non-drug emollients (to relieve dry skin) or topical treatments (such as weak corticosteroids or calcineurin inhibitors).[11,12] In the case of moderate to severe cases, treatments may involve phototherapy or systemic therapies, including biologics, antihistamines, immunosuppressants, or Janus kinase inhibitors. Despite the advances in the treatment of this condition that occurred over the past decade, effective symptom control is not achieved in a significant number of patients using the current therapeutic options.[13,14] Although biologics have revolutionized the treatment of moderate to severe AD, most cases of this condition are mild, and there is less demand for biologics. Topical corticosteroids (TCSs) remain its first-line treatment, although long-term use of these agents can lead to side effects, such as skin atrophy, thinning, and telangiectasia.[15] In addition, the high financial burden of AD treatment highlights the need for affordable and safe solutions to address inflammation, repair the skin barrier, halt disease progression, and prevent recurrence.

Camellia oil, which is derived from the seeds of the *Camellia oleifera* plant that is native to Hainan Island, is rich in unsaturated fatty acids, vitamins, flavonoids, and triterpenoid saponins.[16,17] The camellia plant is widely cultivated in subtropical regions, especially in China, and its oil has long been used for treating various skin conditions, such as diaper dermatitis, burns, oral ulcers, radiation dermatitis, and pressure sores.[18] Traditionally, it has been applied topically to prevent infections and reduce scarring in the skin and mucosal lesions.[19] Research has shown that camellia oil significantly reduces transepidermal water loss and has a strong moisturizing effect.[20] Experimental studies have demonstrated that camellia-oil-based emollients improve skin hydration without causing irritation.[21] The impairment of the skin barrier observed in patients with AD allows the penetration of allergens and pathogens into the skin, thus triggering immune responses and inflammation.[22] Prolonged inflammation disrupts immune homeostasis and hinders wound healing, creating a vicious cycle.[23]

Multiple studies have highlighted the anti-inflammatory, antibacterial, antioxidant, and moisturizing properties of camellia oil.[24-26] Because of its simple extraction process and wide availability, camellia oil presents a promising candidate for the treatment of AD. However, although the existing evidence supports its potential therapeutic benefits, further research using appropriate *in vivo* and *in vitro* models is necessary to confirm its efficacy and elucidate its mechanisms of action.

In summary, the pathogenesis of AD involves skin-barrier dysfunction, immune disorders, and the interaction of genetic and environmental factors. The existing treatments have limitations, including the side effects of drugs and a high financial burden. Therefore, finding new treatments is of great significance for improving the quality of life of patients with AD. As a natural, safe, and inexpensive potential treatment method, camellia oil deserves further research and exploration.

Materials and Methods

Experimental material

2,4-Dinitrochlorobenzene (DNCB, West Asia reagent: 20210303), acetone (production license number: XK13-00144), olive oil (LOT: H29O11P129145), camellia oil (Hainan Wanning), moisturizing cream (Winona, Batch Number: 210928J3S), and an ELISA detection kit (MEIMIAN) were used in this study.

Olive oil and acetone (ratio, 4:1) were mixed to prepare the 10-mL matrix, to which 0.1 g DNCB was added, to obtain the 1% DNCB solution; the same method was used to prepare the 0.1% DNCB solution. The mixed liquid was refrigerated at 4°C and was ready for use.

Experimental animals and groups

Forty BALB/c male SPF mice (age 6 weeks; weight 20-22 g) were purchased from Beijing Sibeifu Biotechnology Co., Ltd. (License No. SCXK (Beijing) 2019-0010). Feeding conditions: the mice were raised in the same cage and placed in opaque plastic cages and kept on a circadian rhythm for 12 h. During the feeding period, the mice were allowed to eat and drink freely. The ambient temperature was 20-26°C and the humidity was 40-70%. Before the experiment, the mice were fed adaptively for 1 week. All experimental processes were operated in accordance with the requirements of experimental animal ethics, and were authorized by the Animal Ethics Committee of the Fifth people's Hospital of Hainan Province (approval no. AF-SW-07-1.0).

Establishment of a DNCB-induced AD mouse model

The villi on the back of the mice were shaved using a shaver, and 100 µL of the 1% DNCB solution was applied to the depilation area of the back and ears of the mice for 3 days. From the 4th day, the mouse skin was externally coated with 100 µL of the 0.1% DNCB solution once every 2 days, for a total of 12 times, until the end of the model-generation (on the 28th day). The modeling process is depicted in Figure 1.

Grouping and administration

Forty mice were randomly coded and divided into four groups: the blank control (Control), model (Model), moisturizing cream (Moisturizer), and camellia oil (Camellia) groups. The mice in the Control group were not treated (with the exception of hair removal). On the 29th day (after modeling), the AD mice in the Camellia group was evenly smeared with 30 mL of Hainan camellia oil twice a day for 4 weeks; whereas the AD mice in Moisturizer group were treated with 3 g of moisturizing cream twice a day for 4 weeks. The lesions were recorded in the model and control groups without treatment. Mice treated with camellia oil were recorded on the first day after treatment. The scratching behavior was observed on the 0th, 7th, 14th, 21st, and 28th day after treatment, and the scratching behavior and back lesions were evaluated. Concomitantly, the thickness of the ear lesions in the mice was measured using an electronic Vernier caliper.

Histopathological observation of the skin lesions

After the establishment of the model, the mice were anesthetized by intraperitoneal injection of 4% chloral hydrate and killed, and the full-thickness skin lesions on their back and ears were cut into sheets of about 0.5×1.5 cm. The skin tissue was fixed with 4% formalin for 48 h, dehydrated with alcohol to prepare paraffin specimens, and continuously cut into 4 mm slices, for preservation. Some slices were dewaxed with xylene and ethanol, then stained with hematoxylin and eosin (H&E) dye solution, and finally dehydrated with alcohol, soaked in xylene, and sealed. The histopathological characteristics of the back and ear skin of the mice in each group were observed under a light microscope.

Evaluation of the scratching behavior in mice

After the beginning of the experiment, the mice in each group were housed separately, and their scratching behavior was observed regularly for 10 min. The scratching behavior of the mice included scratching the ears and head using the front claws, scratching the torso and back using the back claws, and biting all parts of the body using the mouth. The number of scratches performed by the mice was recorded; continuous scratching was counted as 1 time, continuous scratching for more than 3 s was counted as 2 times, and manual intervention was carried out after 3 s to stop the scratching behavior, with a continuous counting of 10 min.

Measurement of the ear thickness

After the beginning of the experiment, the thickness of the mouse ears was measured regularly using an electronic Vernier caliper, while avoiding direct sunlight exposure during the operation. Each site was measured 3 times and the average value was recorded, to ensure the accuracy of the measurements.

Evaluation of the back lesions

The inflammation of the back skin lesion was evaluated according to the standard method of observation of clinical manifestations. According to skin manifestations such as erythema, edema/papule, epidermis exfoliation/scratch, and scales (dry skin), the lesions were divided into four grades: none, mild, moderate, and severe, which were recorded as 0, 1, 2, 3 points, respectively. The scoring criteria used here are reported in Table 1.

ELISA

On the 29th day of modeling, the mice were anesthetized and killed via the neck-severing method. Under aseptic conditions, the eyeballs were dissected and the orbital venous blood was collected. The blood was placed at room temperature for 30 min and centrifuged at 4000 rpm at 4°C for 10 min. The supernatant was stored in a refrigerator at −80°C. The levels of IgE, IL-4, and IFN-γ in the sera of mice were detected using ELISA. Various reagents were prepared according to the kit instructions, standard wells and sample holes were set, 50 μL of the standard at different concentrations were added to the standard hole, and 50 μL of the sample was added to the ELISA plate. Another blank hole was kept free of samples and enzyme-labeled reagents, and the remaining steps were identical. The enzyme-labeled reagent (100 μL) was added to each well and the plate was sealed with sealing foil then placed in an incubator at 37°C to breed for 60 min. The excess reagents were washed out, 50 μL of the chromogenic agent were added to the well, and the reaction was developed for 15 min at 37°C. Finally, 50 μL of terminator was added to each hole, the reaction was terminated, and the absorbance (OD value) was measured in each hole sequentially at a wavelength of 450 nm.

Immunohistochemistry

After the mice were killed, the skin of the ears and back was collected, fixed with 10% neutral formalin, wax embedded and cut into 4 μm paraffin sections that were placed onto glass slides, dewaxed with xylene, and rehydrated with an ethanol series to water. The tissue antigen was retrieved, followed by incubation with 50 μL of a peroxidase blocking solution. Next, 50 μL of the primary antibody solution (anti-filaggrin, Abcam, Cambridge, UK, 1:100; anti-loricrin, Abcam, 1:500) was added and the sections were incubated at 37°C for 45 min. Subsequently, 50 μL of biotinylated goat anti-rabbit IgG (H+L) (Abcam, 1:500) was added to he slices and incubated for 10 min. Then, the slices were incubated with 50 μL of a streptomycin antibiotic–peroxidase solution at room temperature for 10 min, and 100 μL of a freshly prepared DAB solution was added. The slices were washed with tap water, stained with hematoxylin, dehydrated with an alcohol gradient, dried, sealed with neutral gum, and observed under a microscope. The positive staining of the tissue sections was observed under an Olympus DP80 microscope, using a 20× objective to collect images. Five fields of view were randomly selected for each sample for observation and counting, and the percentage of the positive expression areas was measured using the ImageJ software.

Statistical analysis

The SPSS 20.0 software was used for the statistical analysis. The experimental data were measured and all conformed to a normal distribution. All data are expressed as the mean ± standard

Table 1. Evaluation indexes of skin lesions in mice.

Score	Erythema	Edema / papule	Epidermis exfoliation	Scale (dry)
0	No erythema	No edema	No scratches	No scales
1	Faintly visible, pink	Mild edema	Faintly visible, superficial skin	Chaff like desquamation
2	Clearly visible, dark red	Moderate edema	Clearly visible, large number of scratches	Flaky scales
3	Deep red or fiery red	Severe edema	Massive, diffuse scratches	Thicker scales

error ($\bar{x} \pm s$). Comparisons among the experimental groups were performed using one-way analysis of variance (ANOVA), in which those with a uniform variance were tested *via* the least significant difference (LSD) method and those with an uneven variance were tested using Tamhane's method.

Results

Establishment of the AD mouse model

In this experiment, a DNCB sensitization scheme was used to establish an AD-like mouse model.[26] After 28 days of modeling, compared with the normal control group, the skin lesions of the model group were significantly thicker and accompanied by large-area eczema-like changes, such as scabs, erythema, and papules (Figure 2A). The results obtained from additional histopathological sections revealed the presence of hyperkeratosis of the stratum corneum, edema between epidermal cells, and infiltration of endodermal inflammatory cells in the skin lesions of the back and ears of the model mice (Figure 2B).

The acute and chronic stages of AD are accompanied by changes in the levels of various cytokines in the serum. The production of a large amount of serum IgE has been recognized as a typical marker of AD in patients.[27] The serum of mice was obtained on the 29th day of modeling, and the results of the ELISA method showed that the serum contents of IgE and IL-4 in the AD model group were higher than those in the control group (p=0.0117, p=0.0442), whereas the content of IFN-γ in the serum was not significantly different (p=0.9598) (Table 2).

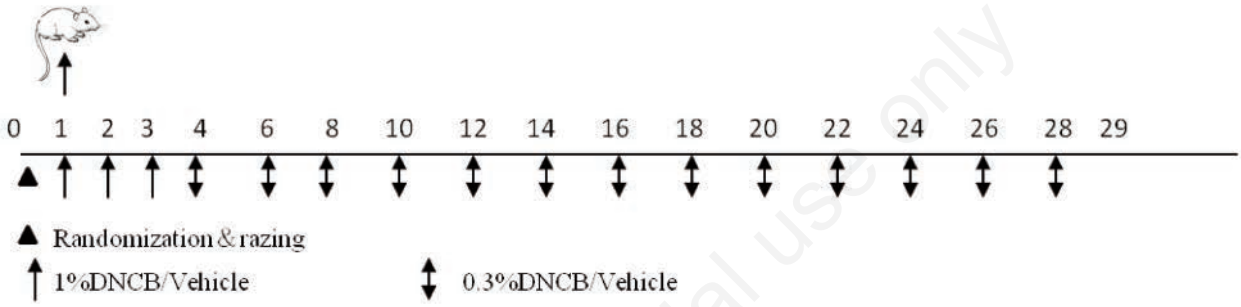

Figure 1. Flow chart of the protocol used for the establishment of an AD mouse model *via* DNCB induction.

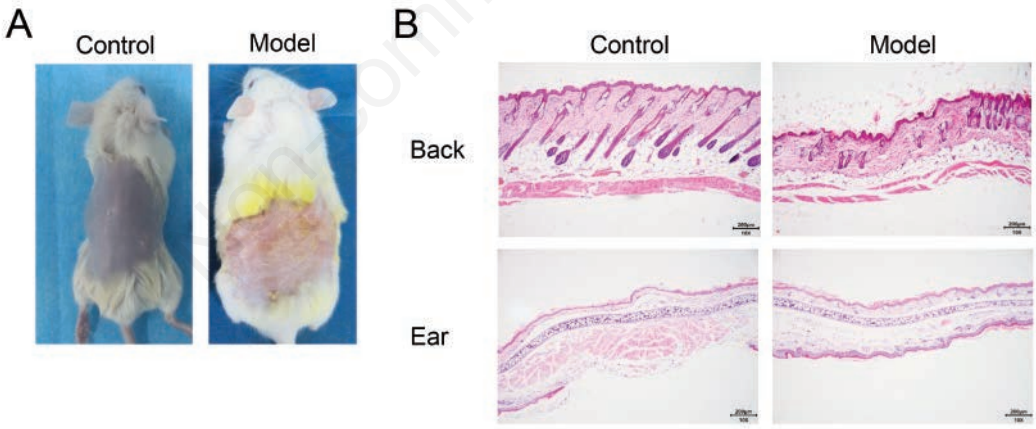

Figure 2. Gross image and pathological observation of the AD mouse model. (**A**) Representative images showing the pathological characteristics of the back of the mice in the model group compared with the normal control group after 28 days of modeling. **B**) H&E staining was performed on the pathological sections of the skin lesions on the backs and ears of the mice, to observe the pathological state of the animals in the model group compared with the control group.

Table 2. Expression levels of serum inflammatory factors in AD mouse models (ng/mL).

Group	IgE	IL-4	IFN-γ
Control group	0.2380±0.0202	3.076±0.3650	12.438±1.9113
Model group	0.2960±0.0415*	3.640±0.8174*	12.1952±2.5531

*p<0.05 compared with Control group.

Camellia oil improves the symptoms of skin lesions in AD mice

After 28 days of treatment, erythema, dryness, scab formation, desquamation, and other skin lesions were still observed on the back of the AD mice in each group; moreover, the symptoms of skin lesions were significantly alleviated after treatment with camellia oil and moisturizing cream (Figure 3A). The results of skin-lesion scoring also revealed that the total lesion score of mice in the model group increased significantly after DNCB sensitization and decreased significantly after treatment with moisturizing cream and camellia oil ($p<0.05$). When treated for 21 days, the therapeutic effect of camellia oil was significantly better than that of moisturizing cream ($p<0.05$) (Figure 3B). After successful modeling, the AD mice exhibited local swelling in the ear and significantly increased scratching times. Furthermore, camellia oil and moisturizing cream significantly reduced AD symptoms in mice, and camellia oil decreased ear thickness and skin itching in mice more significantly ($p<0.05$) (Figure 3 C,D). A histopathological examination of skin lesions in the mice indicated that hyperkeratosis, intercellular edema, and inflammatory-cell infiltration were significantly improved in AD mice treated with moisturizing cream and camellia oil (Figure 3E). Therefore, camellia oil can reduce skin inflammation in AD mice, and its effect is better than that of a moisturizing cream.

Effect of camellia oil on the expression of the FLG protein in skin lesions

Immunohistochemical staining revealed that the FLG and loricrin (LOR) proteins were mainly located in the cytoplasm and nucleus. The FLG protein was strongly expressed in the epidermis of the control group, and was diffusely distributed in the stratum corneum and granular layer; whereas the expression of FLG was significantly decreased in the model group ($p<0.05$). The expression of FLG in the skin lesions of mice in the Moisturizing and

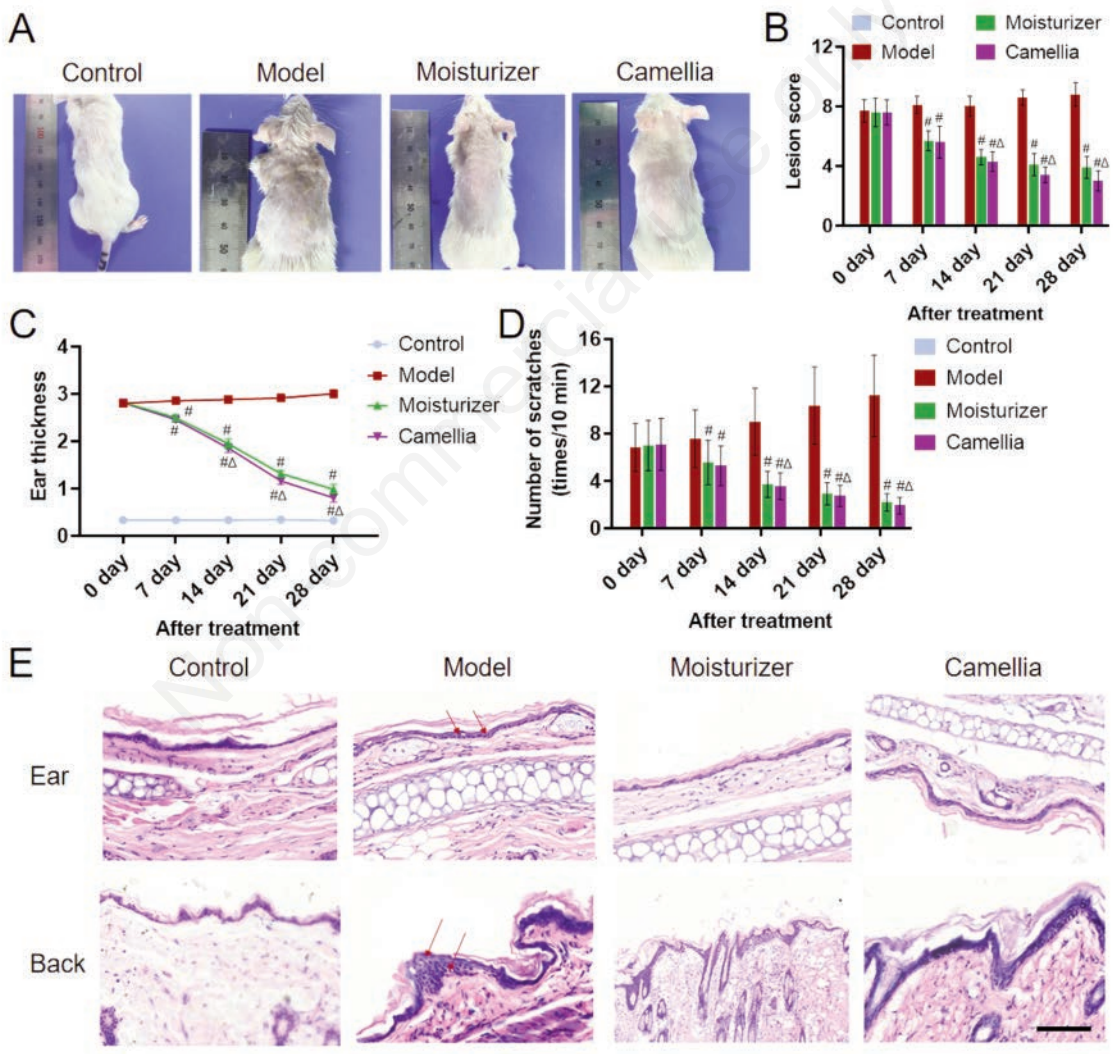

Figure 3. The effect of camellia oil for treating the AD mouse model was observed and analyzed. **A**) Skin lesions on the back of the AD mice in each group were photographed and recorded after 28 days of treatment. **B**) Skin lesions of AD mice in each group were scored at each time period according to the mouse skin lesion evaluation indicators listed in Table 1. **C**) Ear thickness of the mice was measured at each time period using an electronic Vernier caliper, to assess the changes in ear thickness of the AD mice in each group. (**D**) The scratching times of AD mice in each group within 10 min in different time periods were counted. **E**) H&E staining was used to observe the histopathological changes in the ear and back skin lesions of the AD mice in each group. *$p<0.05$ compared with the Control group; $^{\#}p<0.05$ compared with the Model group; $^{\Delta}p<0.05$ compared with the Moisturizing group. Scale bar: 500 μm.

Camellia groups was significantly higher than that detected in the model group, and was almost similar to the normal level (p<0.05) (Figure 4 A,B).

Effect of camellia oil on LOR protein expression in skin lesions

The expression of the LOR protein was also mainly detected in the cytoplasm and nucleus; moreover, expression of the LOR protein was significantly lower in the back skin of the model group vs. that of the control group, whereas the expression of the LOR protein in the skin of the ear exhibited no significant changes (p>0.05). The moisturizing cream and camellia oil treatment slightly upregulated the LOR protein; however, this effect was not significant (p>0.05) (Figure 5 A,B).

Discussion

In the study of skin diseases, the use of various animal models helps expand the knowledge pertaining to new treatments. Currently, the most common human-like AD models used in preclinical research include spontaneous AD mice, transgenic mice, and chemically sensitized mice.[28] AD is a prevalent inflammatory skin condition that is characterized by distinct immune cell changes, which can be divided into acute and chronic stages.[29,30] In human AD, the Th1-related cytokine IFN-γ is predominantly linked to the chronic stage, whereas the Th2-related cytokine IL-4 is associated with the acute phase. A study performed in Stat6VT mice revealed the simultaneous upregulation of the IL-4 and IFN-γ mRNAs from the acute to the chronic phase.[31,32] In the DNCB-induced human-like AD mouse model, a combination of acute (spongy edema) and chronic (thickened epidermis) lesions was observed, suggesting the involvement of IL-4 and IFN-γ in the inflammatory process.[33] DNCB is a well-established compound that is used to induce contact sensitization in human skin by forming haptens with extracellular and intracellular proteins, rendering it a suitable agent for the generation of an AD mouse model.[34] Previous studies have shown that this DNCB-induced model is a cost-effective, simple, and practical tool; therefore, it is the most widely used model in dermatology research.

AD is closely associated with type I allergies, such as allergic asthma and food allergies, which are collectively known as "atopic processes."[35] Exogenous AD, which is characterized by high serum IgE levels, is linked to such allergic reactions.[36] In a humanoid AD mouse model, elevated IgE levels were commonly associated with type I allergies.[37] In our experiment, BALB/c mice exposed to DNCB exhibited an abnormal scratching behavior, local redness, swelling, thickening, scabbing, and desquamation. A histological analysis that was performed using H&E staining revealed the presence of edema and inflammatory-cell infiltration in the epidermis

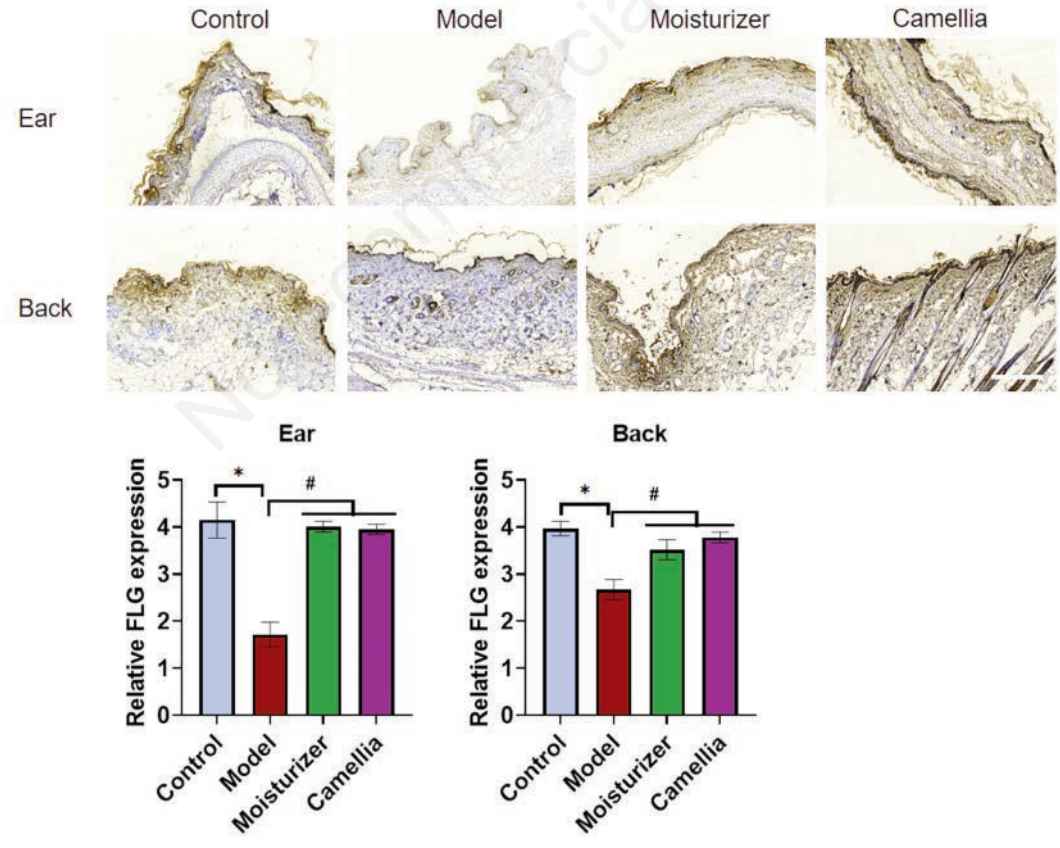

Figure 4. Effect of camellia oil treatment on the expression of the FLG protein in the skin lesions of AD mice. **A**) Expression and localization of the FLG protein in the ear and back skin lesions of AD mice in each group were observed using immunohistochemistry. **B**) Quantitative map of FLG protein expression in the ear and back skin lesions of the AD mice in each group. *p<0.05 compared with the Control group; #p<0.05 compared with the Model group. Scale bar: 500 μm.

of the model group, consistent with previous reports.[38,39] Furthermore, on day 29, the plasma levels of IgE and IL-4 were significantly increased, reflecting the occurrence of the AD cascade and type I allergic reactions. These findings confirm the successful establishment of a human-like AD mouse model in our study. Currently, dermatologists advocate for stage-based, individualized treatment plans for AD, often starting with the application of topical moisturizers to restore the skin barrier function. The selection of effective moisturizers has become a major research focus in clinical treatment. In our experiment, AD mice were treated topically with camellia oil for 4 weeks. The results revealed a reduction in redness, swelling, scabbing, desquamation, and auricle thickening, which was accompanied by significant decreases in the inflammation scores and scratching behavior. Notably, these effects became more pronounced with prolonged treatment. A histological analysis further supported the therapeutic effect of camellia oil, showing reduced inflammation and absence of abnormal skin-appendage proliferation. These results suggest that camellia oil effectively alleviates DNCB-induced AD-like symptoms in mice. A positive control group treated with Winona moisturizing cream, a product with known moisturizing, anti-allergic, and anti-inflammatory properties, was included in the analysis, to highlight the effectiveness of camellia oil. The rationale for this approach was that, although the Winona moisturizing cream also contains moisturizing and anti-inflammatory ingredients, they may not be as comprehensive and efficient as those of camellia oil. Second, because camellia oil is a natural vegetable oil, its ingredients are relatively mild and cause little irritation to the skin. Moreover, there may be a synergistic effect between the multiple bioactive ingredients of the camellia oil, which together exert a stronger therapeutic effect. The significant improvements observed in both groups suggest that camellia oil holds potential as an adjunctive treatment for AD when used in combination with conventional therapies. The pathogenesis of AD remains complex, with environmental factors, genetic predispositions, immune dysregulation, and skin-barrier dysfunction being the central areas ofresearch.[40] The skin barrier is crucial for maintaining skin health, and its impairment is considered a primary mechanism in AD.[41] The physical barrier provided by the stratum corneum, known as the cornified envelope (CE), is primarily composed of involucrin (IVL) and loricrin (LOR), with LOR representing 80% of the CE proteins.[42] Together, LOR and IVL are essential for keratinocyte differentiation and CE stability. Abnormal expression of LOR and IVL compromises CE integrity, thus weakening the skin barrier. FLG, another key structural protein in the stratum corneum, is vital for skin-barrier function.[44] Decreased FLG expression promotes water loss, thereby affecting lipid metabolism and leading to skin dryness and itching.[45,46] Therefore, maintaining FLG and LOR expression is critical for regulating the integrity of the skin barrier.

The immunohistochemical results obtained here revealed high levels of expression of FLG and LOR in the ear and back skin of the control mice, whereas their expression was significantly

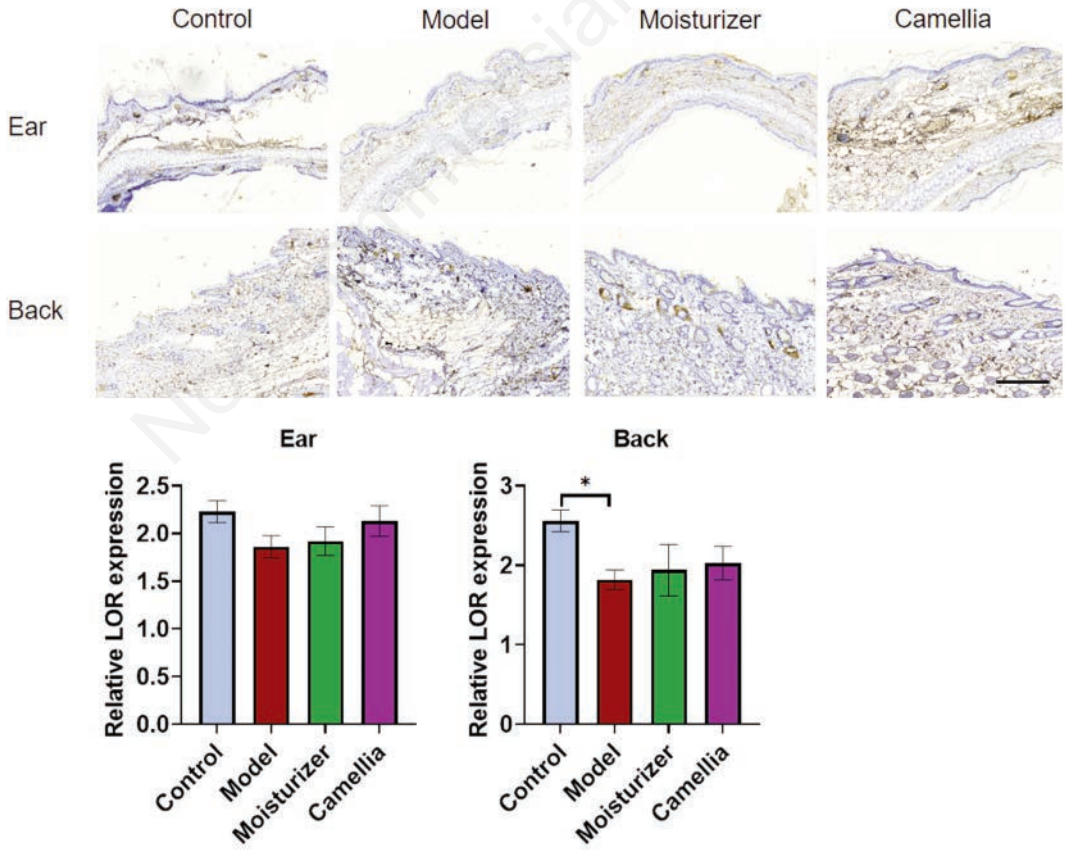

Figure 5. Effect of camellia oil treatment on the expression of the LOR protein in the skin lesions of AD mice. **A**) The expression and localization of the LOR protein in the ear and back skin lesions of the AD mice in each group were observed using immunohistochemistry. **B**) Quantitative map of LOR protein expression in the ear and back skin lesions of the AD mice in each group. *p<0.05 compared with the Control group. Scale bar: 500 μm.

decreased in the AD model mice. This suggests that reduced FLG and LOR levels may mediate the progression of inflammatory responses in AD lesions. In turn, camellia oil treatment significantly upregulated FLG in the ear and back skin, indicating its ability to repair the damaged skin barrier, with its anti-inflammatory effects likely contributing to this improvement. It is known that camellia oil is rich in unsaturated fatty acids, vitamin E, antioxidants, and various sterols, which have significant antioxidant and anti-inflammatory properties.[47] Unsaturated fatty acids can reduce the oxidative stress on cell membranes, stabilize the cell membrane structure, and prevent the excessive release of inflammatory mediators; vitamin E serves as a powerful free-radical scavenger to protect the skin from the oxidative damage caused by external factors, such as ultraviolet light and pollution; and antioxidants and phytosterols regulate the immune response and inhibit the activation of inflammatory cells and the release of inflammatory factors, thereby reducing the skin inflammatory response.[48] It is worth noting that, although camellia oil has shown great potential for skin-barrier repair and anti-inflammation, the elucidation of its specific mechanism of action warrants more in-depth research. The comprehensive analysis of the molecular changes occurring in skin cells after intervention with camellia oil will be the focus of our future research; concomitantly, conducting clinical trials to evaluate the safety and effectiveness of camellia oil in patients with different skin diseases is also an important direction for future research.

In conclusion, this study successfully established a DNCB-induced AD mouse model. Moreover, using visual and histopathological analyses, it demonstrated that camellia oil provided a significant therapeutic benefit in this model. Camellia oil effectively reduced skin symptoms and pruritus in the AD-like mice, while also upregulating FLG and LOR, which strengthened the CE and repaired the damaged skin barrier. These findings offer a solid experimental foundation for further research of AD treatments, and highlight the potential of camellia oil as an effective, safe, and affordable therapeutic option with promising clinical applications.

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