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"Optimized Skincare Formulation for Atopic Dermatitis: Validated Through Comprehensive In Vitro and In Vivo Safety and Efficacy Assessments"

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1. Introduction

Atopic dermatitis (AD), also known as eczema and atopic exzame, is a common chronic, recurrent inflammatory skin disease that affect around 20% of children, and up to 10% of adults in high-income countries [1], and affect increasing population in developing countries [2]. The causes of AD are complex and multifactorial. AD family twin studies reveal approximately 75% genetic heritability [3-5]. AD is characterized by disrupted epidermal barrier functions with basic features include dry skin, acute and chronic eczema-like lesions, and intense pruritus [2]. *Staphylococcus aureus* (*S. aureus*) colonization was reported to be an important trigger of AD [6]. AD skin management aims to improve symptoms should be patent-centred and includes avoidance of individual trigger factors, immunity including anti-inflammation and anti-allergy regulation, and restoration of skin barrier functions [2,7]. While the market still lack of effective daily skin care cosmetics for AD consumers.

In this study, we developed a cream named AUS AD cream for AD consumers, targeting to revlief itch uncomfortableness, control *S. aureus* proliferation, and repair skin barrier. Plant origined ingredients such as ARTEMISIA ANNUA LEAF EXTRACT, PUERARIA LOBATA ROOT EXTRACT, CROCUS SATIVUS FLOWER EXTRACT, and ROSMARINUS OFFICINALIS LEAF EXTRACT were used to suppress *S. aureus*, anti-inflammation, and anti-allergy [8-12]. Other active ingredients such as ceramides, DHA, BIOSACCHARIDE GUM-2, ECTOINE, UCCINOGLYCAN etc. were also incorporated for their beneficial effects on skin barrier [13-15]. The efficacy of the AUS AD cream was evaluated through *in vitro* anti- *S. aureus* assay, *in vitro* skin assay using Desonide cream as reference, and *in vivo* clinical experiments.

2. Materials and Methods

2.1 Experiment samples

The target of the present study named AUS AD cream, which contained major ingredients including AQUA, PETROLATUM, BUTYROSPERMUM PARKII (SHEA) BUTTER, DIMETHICONE, BUTYLENE GLYCOL, BETAINE, SQUALANE, C10-18 TRIGLYCERIDES, SPERMACETYL ALCOHOL, GLYCERYL STEARATE, OLEATE ESTER, ECTOINE, MONTAN ESTER, POLYSORBATE 60, PEG-100 STEARATE, 1,2-HEXANEDIOL, 1,2-PENTANOL, DISODIUM LAURYL ISETHIONATE/VP COPOLYMER,

HYDROXYACETOPHENONE, C18-36 ACID TRIGLYCERIDE, ALLANTOIN, DODECANE-1,6-DIOIC ACID, HYDROGENATED DECENE, 1,3-PROPANEDIOL, and ARTEMISIA ANNUA LEAF EXTRACT, and minor ingredients including C12-18 ACID TRIGLYCERIDE, UCCINOGLYCAN, PUERARIA LOBATA ROOT EXTRACT, CERAMIDE NP, BIOSACCHARIDE GUM-2, PLANTASCOLANOL, CERAMIDE AP, STEARIC ACID, HYLORINATED LECITHIN, HYDROCHOLESTEROL, CROCUS SATIVUS FLOWER EXTRACT, TETRACETYLPLANTOL, FLAVONOID GUM, TOCOPHEROL, CERAMIDE EOP, ROSMARINUS OFFICINALIS LEAF EXTRACT, L-ASCORBYL PALMITATE, and GLYCOSPHINGOLIPIDS.

Other consumables and reagents obtained for this study: Desonide cream that is often used to relieve AD symtoms was included as a reference product (Guangzhou pharmaceutical store, China), *in vitro* EpiKutis® skin (Batch No.: ES241206) and EpiGrowth medium from Guangdong Biocell Biotech Co. Ltd, PolyI: C, lipopolysaccharide (*E.Coli*), WY14643, FLG antiboly, LOR antibody, AQP3 antibody, IVL antibody, and TSLP ELISA kit from Abcam, PBS from Solarbio, dexamethasone from NICPBP, paraformaldehyde from Biosharp, Xylene from SINOPHARM, Hematoxylin and Eosin from Beyotime. [The AUS AD cream and Desonide cream were coded for blind tests to avoid any possible bias]

2.2 *In vitro* antibaterial assay against *S. aureus*

The antibacterial activity against *S. aureus* was assessed using the quantitative suspension method outlined in the QB/T 2738-2023 "Standard Methods for Evaluating the Antibacterial Efficacy of Disinfectants". To begin, a bacterial suspension of S. aureus (ATCC 6538) was prepared to a concentration of 1 x 10^4 colony-forming units (cfu) per milliliter (mL). A mixture was created by combining 0.1 mL of this bacterial suspension with 5.0 mL of the sample solution and thoroughly agitating the mixture. After incubating at room temperature for 6 hours, 0.5 mL of the incubated mixture was transferred into 4.5 mL of sterile phosphate-buffered saline (PBS), mixed for an additional 10 min, and then 1 mL of this solution was spread onto agar plates. Three replicate plates were prepared for each sample. The plates were incubated at 36 ± 1°C for 48 h. Following incubation, the number of bacterial colonies on each plate was counted. Triplicate experiments were conducted for each sample to ensure data reliability. Sterile water was used as a negative control in these assays. *S. aureus* suppression rate was calculated as (Colony $no._{sample}$ - Colony $no._{negative}$)/ Colony $no._{negative}$) ×100%.

2.3 *In vitro* skin assay

EpiKutis® skin tissue samples, each measuring (24 ± 2) mm^2, were cultured in a controlled environment using an in vitro skin tissue culture medium at 37°C with a CO2 concentration of 5% for a period of 5 days. Following this initial culture period, the skin surfaces were treated with cosmetic samples at the same temperature and CO2 concentration for 24 h. Subsequently, the samples were exposed to a combination of PolyI:C and LPS for an additional 24 h. The experimental setup included both negative and positive controls. The negative control consisted of a model treatment with 24 μg/mL PolyI:C and 20 μg/mL LPS. The positive control involved the addition of 50 μM WY14643 to the PolyI:C + LPS treatment. An additional group

treated with PolyI:C + LPS and 0.01% dexamethasone was included as a positive control for the TLSP analysis. Each treatment was replicated three times to ensure the reliability of the results. Following the treatment period, the tissues underwent further processing and analysis as detailed below.

2.4.1 Hematoxylin and Eosin (H&E) staining

Morphological assessments were conducted using Hematoxylin and Eosin (H&E) staining. Tissues were fixed with 4% paraformaldehyde, embedded in paraffin wax, and sectioned. Sections were stained with H&E, photographed under an orthographic microscope (Olympus, BX53), and analyzed using Image-Pro® Plus image processing software.

2.4.2 Immunofluorescence staining

Tissue sections were stained with antibodies against FLG, LOR, AQP3, and IVL. Images were captured using a fluorescence microscope (Olympus, BX43). The integrated optical density (IOD) of the fluorescence signal was quantified with Image-Pro® Plus Image processing software. The improvement rate was calculated as $(Signal_{sample} - Signal_{model})/Signal_{model} \times 100\%$.

2.4.3 ELISA Assay

Tissue culture supernatants were processed to measure TSLP content following the ELISA kit's instructions.

2.5 Human patch Test

The test was conducted following the HRIPT method as outlined in Chapter 2 of the China Safety Technical Specifications for Cosmetics (2015 Edition). Briefly, 30 volunteers (4 males and 26 females), aged 20 to 49 years (average age 30.90 ± 8.15 years), who met the inclusion criteria, participated in the study. A sample of 0.020 - 0.025 g was applied to a 50-mm^2 area on the forearm (one sample per forearm) for 24 hours. After 24 h, the samples were removed, and the skin response was evaluated at 0.5, 24, and 48 h post-treatment. The skin response was recorded according to the specified method.

2.6 Sensory evaluation

Ten volunteers who met the inclusion criteria washed their left inner arm, dried it, and acclimated to an environment of (21.4 ~ 21.6) °C and (43 ~ 45) % humidity for 30 min. Two plots, each 3 cm x 3 cm, were marked on the left inner arm. Each plot was peeled 3 times using 3M tape and treated with 1% histamine at (2.0 ± 0.1) mg/cm^2. The two plots were analyzed using a Skin Thermometer ST500 (Medelink, Canada) to measure temperature and an Antera 3D (Miravex, Ireland) to measure the area of redness. Then, AUS AD cream was applied to one of the plots. After 15 min, the two plots were measured again using the Skin Therometer ST500 and Antera 3D. The improvement rate was calculated as $(Mean_{T15min} - Mean_{T1})/Mean_{T1} \times 100\%$.

Volunteers performed self-assessments of itching sensation before and 15 min after AUS AD cream application. The itch sensation level was rated on a scale of 0 to 5, where 0 indicated

"imperceptible," 1 "barely noticeable," 2 "perceivable," 3 "moderate level," 4 "severe itch," and 5 "pain."

2.7 Statistical analysis

GraphPad Prism software was used to analyze the data. The results were expressed as mean ± SD. Comparisons between groups were performed using two-tailed t-tests and Wilcoxon signed-rank tests, with $p < 0.05$ considered as a significant difference.

3. Results

3.1 Aus AD cream exhibited excellent anti- *S. aureus* activity

The product suppressed 99.99% of *S. aureus* within 6 h treatment, indicating the product can efficiently suppress the proliferation of *S. aureus*, which is a dominant bacterial of AD skin.

3.2 Aus AD cream alleviated the toxicity of PolyI:C+LPS to EpiKutis® skin

H&E staining results (Fig. 1) showed that compared with negative control, which showed no apoptosis cells, PolyI:C+LPS treatment (model control) induced cell apoptosis, and positive, Desonide cream, and AUS AD cream groups showed less apoptosis cells than model control. Counting the number of apoptosis cells showed that the model group contained 9.0 ±1.53 apoptosis cells, compared with which the 0.01% dexamethasone treatment (positive control) reduced the apoptosis cell number by 66.67% to 3.0 ± 1.15 ($p = 0.006$), Desonide cream treatment reduced the apoptosis cell number by 44.44% to 5.0 ± 0.58 ($p = 0.013$), and the AUS AD cream treatment reduced the apoptosis cell number by 66.67% to 3.0 ± 1.00 ($p = 0.006$). These results showed the AUS AD cream alleviated the toxicity of PolyI:C+LPS to *in vitro* skin.

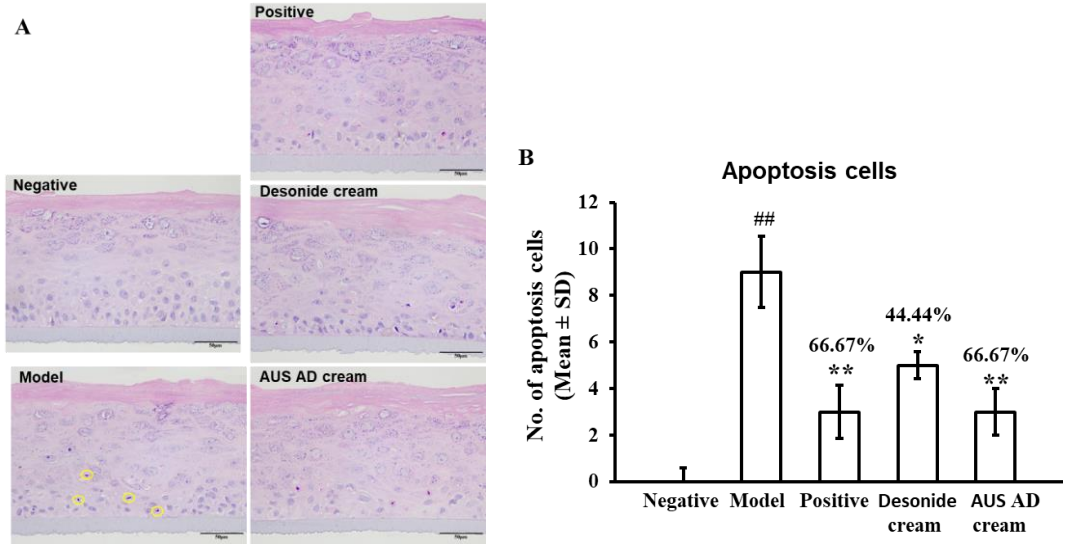


Fig. 1 AUS AD Cream Mitigates the Toxicity of PolyI:C + LPS on EpiKutis® Skin. A) H&E-stained skin sections with yellow circles indicating representative apoptotic cells. B) quantification of apoptotic cells. Model: PolyI:C + LPS (24 μg/mL PolyI:C + 20 μg/mL LPS). Positive: 50 μM WY1464. Scale bar: 50 μm. ## $p < 0.01$ (compared with negative control). * $p < 0.05$ and ** $p < 0.01$ (compared with the model control).

3.3 Aus AD cream exhibited excellent repairing efficacy *in vitro*

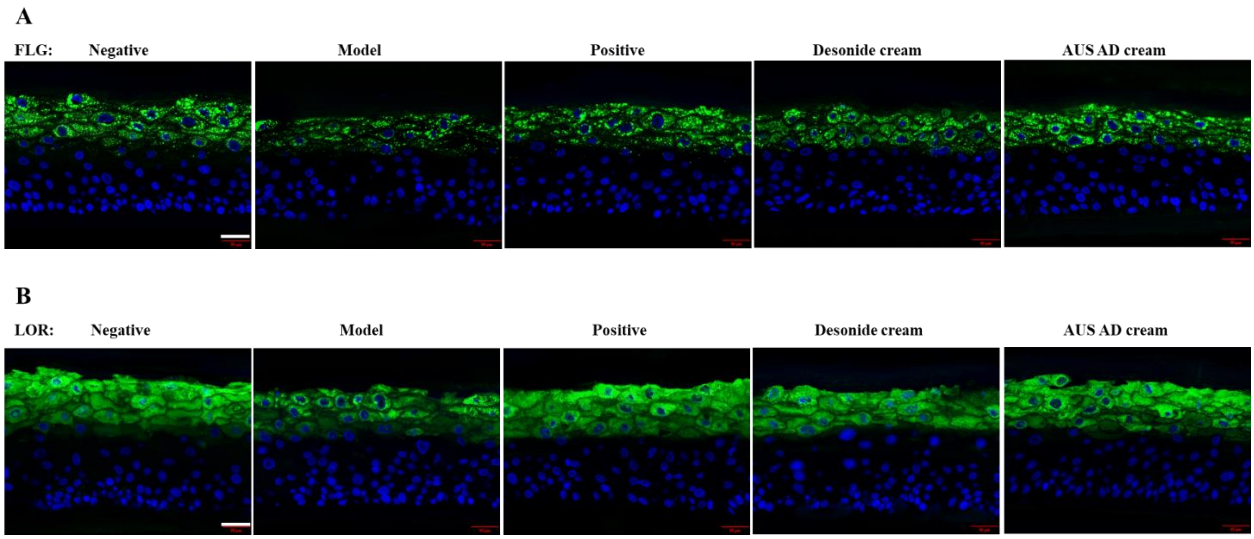
Immunofluorescence analysis, as depicted in representative images (Figs. 2A-C), demonstrated varying fluorescence signals for FLG, LOR, and IVL. The negative control exhibited the strongest fluorescence signal, followed by the AUS AD cream, positive control, Desonide cream, and model control.

The FLG signal intensity (Fig. 2D) was measured as 1.0 ± 0.09 for the negative control, 0.3 ± 0.05 for the model control, 0.7 ± 0.04 for the positive control, 0.5 ± 0.07 for Desonide cream, and 0.81 ± 0.02 for the AUS AD cream group. The model control significantly ($p < 0.001$) reduced FLG signal in the skin. Compared with the model control, the positive control, Desonide cream, and AUS AD cream increased the FLG level by 133.3% ($p < 0.001$), 666.7% ($p = 0.013$), and 170.00% ($p < 0.001$), respectively. The FLG level in the AUS AD cream group was significantly ($p = 0.002$) higher than that of Desonide cream.

The LOR signal intensity (Fig. 2E) was 1.0 ± 0.04 for the negative control, 0.26 ± 0.01 for the model control, 0.72 ± 0.01 for the positive control, 0.51 ± 0.03 for Desonide cream, and 0.84 ± 0.0.5 for the AUS AD cream group. The model control significantly ($p < 0.001$) reduced LOR signal in the skin. Compared with the model control, the positive control, Desonide cream, and AUS AD cream increased the LOR level by 176.29% ($p < 0.001$), 9.15% ($p = 0.001$), and 223.08% ($p < 0.001$), respectively. The LOR level in the AUS AD cream group was significantly ($p = 0.001$) higher than that of Desonide cream.

The IVL signal intensity (Fig. 2F) was 1.0 ± 0.05 for the negative control, 0.30 ± 0.0.1 for the model control, 0.77 ± 0.03 for the positive control, 0.4 ± 0.3 for Desonide cream, and 0.85 ± 0.5 for the AUS AD cream group. The model control significantly ($p < 0.001$) reduced IVL signal in the skin. Compared with the model control, the positive control, Desonide cream, and AUS AD cream increased the IVL level by 156.67% ($p < 0.001$), 80.00% ($p < 0.001$) and 183.3% ($p < 0.001$), respectively. The IVL level in the AUS AD cream group was significantly ($p = 0.001$) higher than that of Desonide cream.

These data suggest that the AUS AD cream efficiently repairs skin damage induced by PolyI:C + LPS treatment, with a superior repairing efficacy compared to Desonide cream.



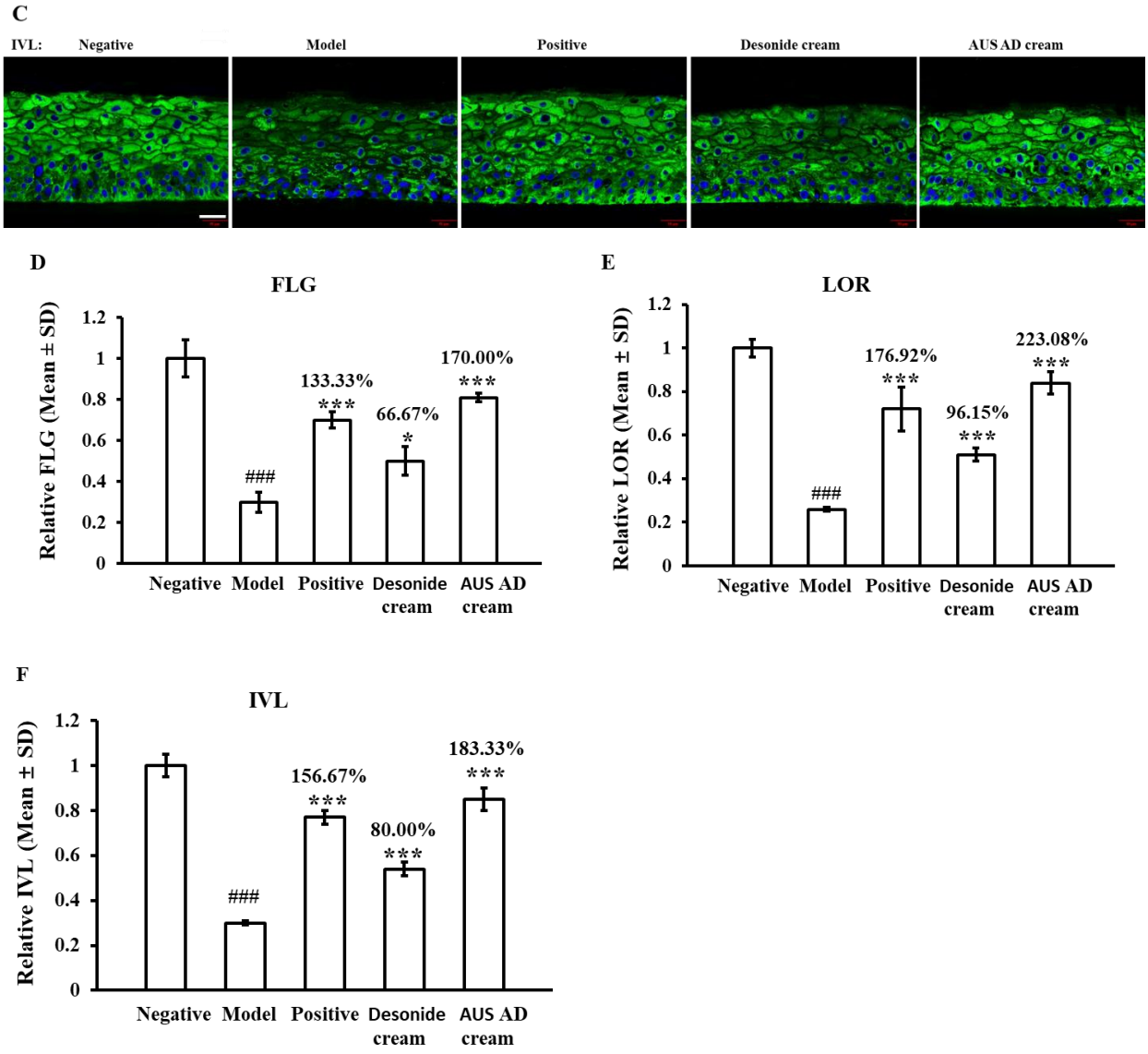


Fig.2 AUS AD cream repaired EpiKutis® skin through increasing FLG, LOR, and IVL levels. A to C are FLG, LOR, and IVL immuno-stained skin sections respectively. D to F are measured signal intensity of FLG, LOR, and IVL respectively. Model: PolyI:C+LPS (24 µg/mL+ 20 µg/mL). Positive: 50 µM WY14643. Scale bar: 50 µm. ### $p < 0.001$ (compared with negative control), * $p < 0.05$ and *** $p < 0.001$ (compared with model control).

3.4 Aus AD cream exhibited hydration efficacy *in vitro*

Immunofluorescence analysis of AQP3 (Fig. 3A) revealed that the negative control exhibited the strongest AQP3 signal, followed by the AUS AD cream, positive control, Desonide cream, and model control. The AQP3 signal intensity analysis (Fig. 3B) demonstrated that compared to the negative control, which was arbitrarily set as 1.0, the model control significantly ($p < 0.001$) reduced the AQP3 signal to 0.4 ± 0.04, indicating that PolyI:C + LPS induced a skin dehydration challenge. In contrast, the positive control (50 µM WY1463), Desonide cream, and AUS AD cream increased the AQP3 level to 0.79 ± 0.05 (by 132.5%, $p < 0.001$), 0.5 ± 0.03 (by 132.5%, $p = 0.002$), and 0.83 ± 0.03 (by 132.5%, $p < 0.001$), respectively. These results

indicate that 50 µM WY1463, Desonide cream, and AUS AD cream could significantly alleviate the dehydration condition caused by PolyI:C + LPS, with the hydration efficacy of AUS AD cream being significantly ($p < 0.001$) superior to that of Desonide cream.

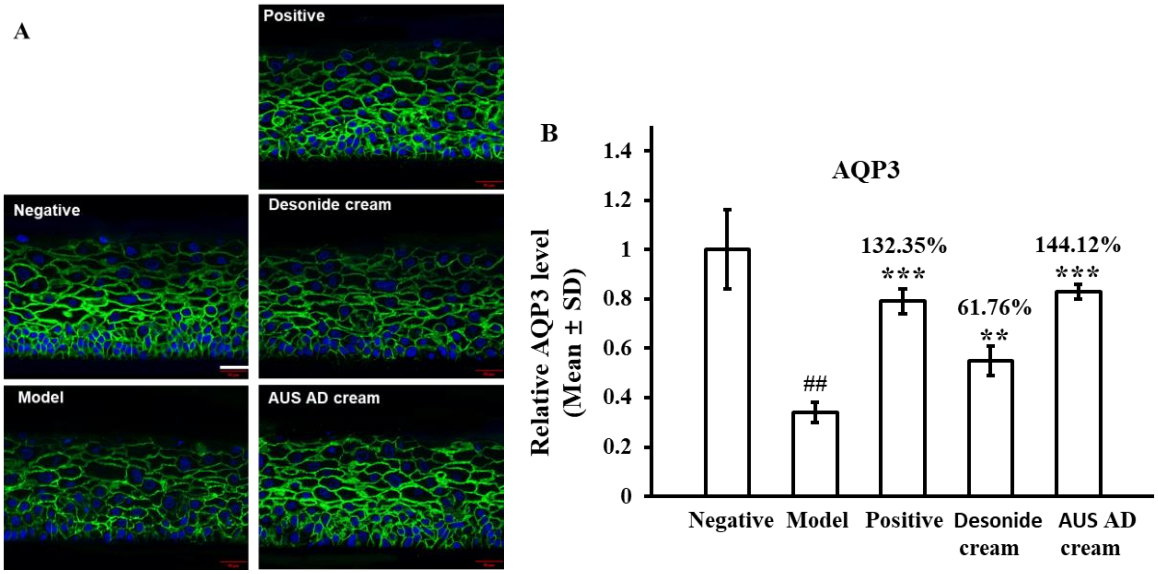


Fig.3 AUS AD cream increased AQ3 level in PolyI:C+LPS treated EpiKutis® skin. A) AQP3 immuno-stained skin sections. B) APQ3 levels. Model: PolyI:C+LPS. Positive: 50 µM WY14643. Scale bar: 50 µm. ## $p < 0.01$ (compared with negative control), ** $p < 0.01$ and *** $p < 0.001$ (compared with model control).

3.5 Aus AD cream exerted strong soothing efficacy *in vitro*

The ELISA analysis results showed that the TSLP was 2.76 ± 0.32 pg/mL in the negative control, 17.86 ± 0.80 pg/mL in the model control, which means PolyI:C+LPS significantly ($p < 0.001$) induced TSLP level. Compared with model control, Desonide cream induced cell apoptosis, and less apoptosis cells identified in positive, 0.01% dexamethasone, Desonide cream, and AUS AD cream decreased the level of TSLP to 5.22 ± 0.49 pg/mL (70.77% decrease, $p < 0.001$), 8.52 ± 0.32 pg/mL (52.30% decrease, $p < 0.001$), and 5.86 ± 0.18 pg/mL (67.19% decrease, $p < 0.001$), respectively. This means 0.01% dexamethasone, Desonide cream, and AUS AD cream could significantly alleviate the irritation by PolyI:C+LPS, and the soothing efficacy of AUS AD cream was significantly ($p < 0.001$) stronger than Desonide cream.

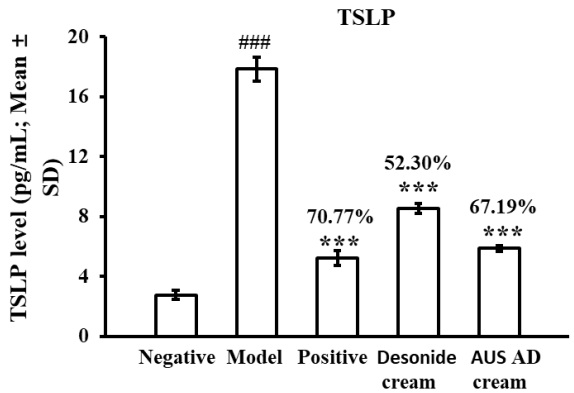


Fig. 4 AUS AD cream decreased TSLP level in the culture medium of PolyI:C+LPS treated EpiKutis® skin. Model: PolyI:C+LPS (24 μg/mL+ 20 μg/mL). Positive: 0.01% dexamethasone. ### $p < 0.001$ (compared with negative control), and *** $p < 0.001$ (compared with model control).

3.6 AUS AD cream was mild and non-irritant to general human skin

The AUS AD cream was subjected to a human patch test to evaluate its potential for mildness and non-irritancy on human skin. The study involved 30 volunteers who applied the cream to their skin. The results demonstrated that none of the participants experienced any adverse reactions to the product, suggesting that the AUS AD cream is both mild and non-irritating to general human skin. This finding supports the safety profile of the cream for potential use in skincare applications.

3.7 AUS AD cream showed excellent soothing efficacy *in vivo*

3.7.1 AUS AD cream effectively soothed irritation caused by histamine

The AUS AD cream demonstrated significant soothing effects on skin irritation induced by a 1% histamine application. Machine-measured temperature data (Table 1) indicated that the temperatures of the two test sites were comparable immediately after the histamine application. However, 15 minutes later, the model control group exhibited a sustained elevated temperature ($p = 0.29$), whereas the AUS AD cream treatment significantly ($p < 0.001$) reduced the temperature by 4.36%. Redness area measurements (Table 2) revealed that the redness of the two sites was similar immediately post-application, but 15 minutes later, the model control group maintained a higher redness area ($p = 0.59$), while the AUS AD cream treatment significantly ($p = 0.025$) reduced the redness by 4.94%. Itchiness rating results (Table 3) indicated that although the itch rating improved significantly ($p = 0.044$) by 33.3%, the improvement in the AUS AD cream group (37.50%, $p = 0.019$) was significantly superior to that of the model control group. These findings underscore the excellent in vivo soothing efficacy of the AUS AD cream.

Table 1. The AUS ASD cream significantly dropped the skin surface temperature afte 1% histamin application. N=10.

Treatment	Temperature (℃; Mean ± SD)		Improvement rate	Improvement difference
	T0	T15min		
Model control	30.66 ± 0.35	30.51 ± 0.28	0.49% ($p = 0.29$)	$p = 0.0073$
AUS AD cream	30.50 ± 0.35	29.17 ± 0.36	4.36% ($p = 0.0006$)	

Table 2. The AUS ASD cream significantly shrinked the skin redness area caused by a 1% histamine application. N=10.

Treatment	Antera 3D redness (Mean ± SD)		Improvement rate	Improvement difference
	T0	T15min		
Model control	20.54 ± 1.82	20.82 ± 1.98	-1.36%($p = 0.59$)	$p = 0.0009$
AUS AD cream	21.26 ± 1.92	20.21 ± 1.95	4.94% ($p = 0.025$)	

Table 3. The AUS ASD cream significantly reduced the skin itchiness rating caused by a 1% histamine application. N=10.

Treatment	Itchiness rating (Mean ± SD)		Improvement rate	Improvement difference
	T0	T15min		
Model control	2.10 ± 0.28	1.40 ± 0.43	33.33% (p = 0.044)	p = 0.0006
AUS AD cream	2.40 ± 0.34	1.50 ± 0.43	37.50% (p = 0.019)	

4. Discussion

This study introduces the development of AUS AD Cream, an innovative formulation tailored for individuals with Atopic Dermatitis (AD). Our approach is multifaceted: (1) incorporating historically mild and non-irritating basic ingredients, (2) utilizing natural plant extracts including *Artemisia annua* leaf, *Crocus sativus* flower, and *Pueraria lobata* root extracts to mitigate allergic reactions and inflammation, (3) employing antimicrobial agents against *S. aureus* with plant extracts like *Artemisia annua*, and (4) incorporating active ingredients including ceramide NP/AP/EOP, DHA, ectoine, biosaccharide gum-2, glycerol, squalane, and petrolatum to bolster skin repair, hydration, and protection.

Our results demonstrate that AUS AD Cream exhibits significant anti-S. aureus efficacy, achieving a remarkable 99.99% reduction in bacterial growth. Moreover, when compared to the pharmaceutical Desonide cream, AUS AD Cream demonstrated superior skin protection, enhanced skin barrier repair (as evidenced by increased FLG, LOR, and IVL, and decreased TSLP), and improved skin hydration (as indicated by elevated AQP3 levels) when tested on EpiKutis® skin challenged with PolyI:C and LPS. Additionally, AUS AD Cream effectively reduced skin temperature, reduced skin redness, and alleviated itchiness induced by 1% histamine. Collectively, these findings highlight the substantial potential of AUS AD Cream as a daily skincare product for AD consumers. Further studies are necessary to explore the skincare efficacy of AUS AD Cream in AD patients.

5. Conclusion

The AUS AD cream showed excellent soothing, hydration, and repairing efficacy *in vitro* and *in vivo*, demonstrating its suitability for fragile and sensitive skin consumers and even AD consumers.

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