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Effects of a Moisturizer Containing Colloidal Oatmeal and Filaggrin Technology on *Staphylococcus Species* In Vitro

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ABSTRACT

The pathophysiology of atopic dermatitis (AD) is multifactorial, with genetic predisposition, environmental exposures, and dysbiosis of the skin microbiome all associated with disease activity. Colonization by *Staphylococcus aureus* (*S. aureus*) is specifically associated with AD, and modifying the skin microbiota through topical skincare products may play a role in AD management. A moisturizer containing colloidal oatmeal, a patented filaggrin technology, niacinamide, and tocopheryl acetate was assessed for its impact on the growth, biofilm formation, and bacterial mix adhesion of *S. aureus* and/or *S. epidermidis* on reconstructed human epidermis (RHE). Compared to control conditions, the bacterial growth and adhesion of *S. aureus* were decreased compared to *S. epidermidis* in the presence of the moisturizer. Additionally, the moisturizer did not significantly induce nor inhibit the formation of *S. aureus* biofilm relative to control. Overall, the moisturizer improved the growth ratio of *Staphylococcus* species, shifting the predominant species from pathogenic *S. aureus* to commensal *S. epidermidis*, which may be clinically beneficial in the management of AD.

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INTRODUCTION

Atopic dermatitis (AD) is a multifactorial disease associated with both genetic risk factors and environmental stimuli. AD is also associated with dysbiosis, notably increased colonization by *Staphylococcus aureus*, and the degree of colonization correlates with disease severity.[1] Topical skincare products may be beneficial in improving bacterial diversity resulting from barrier dysfunction.[2]

A moisturizer specifically designed for AD (Moisturizer 1642) containing colloidal oatmeal, a patented filaggrin technology (RestoradermTechnology™, sodium pyrrolidone carboxylic acid [PCA], and arginine), niacinamide, and tocopheryl acetate was evaluated for its effect on bacterial growth, biofilm formation, and bacterial mix adhesion on reconstructed human epidermis (RHE).

MATERIALS AND METHODS

The growth of two bacteria strains (*S. aureus* and *S. epidermidis*) cultured without hydrocortisone or antibiotics at 37°C with 5% CO_2 was evaluated in the absence (control) and presence of Moisturizer 1642 at 8 concentrations from 0.004% to 0.5% by measuring optical density (OD) at 600 nanometers (nm) kinetically for 24 hours. Bacterial enumeration and adhesion were measured by colony counting of untreated (control) and pre-treated RHE seeded with a bacterial mix of 50% *S. aureus* and 50% *S. epidermidis* (1.67x107 CFU/ml of each strain). Biofilm formation of *S. aureus* was performed according to the Helaly et al protocol[3] and was assessed by the fixation of crystal violet measured by optical density at 590 nm. Experimental data was standardized to control for color interference with OD measurements. All experiments were performed in triplicate.

Statistical analyses were performed using Microsoft Excel and GraphPad PRISM software. The inter-group comparisons were performed using unpaired Student's t-tests.

RESULTS

Bacterial Growth

Individual growth curves for each bacterial strain showed that under control conditions (n=3), *S. aureus* and *S. epidermidis* growth plateaued after approximately 12 and 8 hours of incubation, respectively. Following treatment with Moisturizer 1642 (n=3), no significant inhibition or promotion of either bacterial strain was observed.

Evaluation of bacterial growth following incubation of a bacterial mix found growth of both *S. aureus* and *S. epidermidis* after 6 hours under control conditions (n=3); however, *S. aureus* grew faster than *S. epidermidis* after 24 hours of incubation (1000% versus 372% of the control at baseline [T0]). Following treatment with Moisturizer 1642 (n=3), no difference in bacterial enumeration of *S. aureus* and *S. epidermidis* was observed at T0 (Figure 1A). At 0.05%, 0.15%, and 0.5% concentrations of the moisturizer, the growth of *S. aureus* was inhibited, whereas the growth of *S. epidermidis* was promoted over time compared to the control, thus rebalancing the ratio of bacterial strain growth (Figure 1A and 1B).

Bacterial Adhesion

Under control conditions (n=5), 70.7% of *S. aureus* and 2.1% of *S. epidermidis* within the bacterial mix adhered to the surface of RHE, corresponding to 97% and 3% of adhered bacteria, respectively (Figure 2A). Application of Moisturizer 1642 (n=5) significantly decreased *S. aureus* adhesion by 79% and strongly promoted *S. epidermidis* adhesion to 1988% of the control ($P<0.001$; Figure 2B).

Biofilm Formation

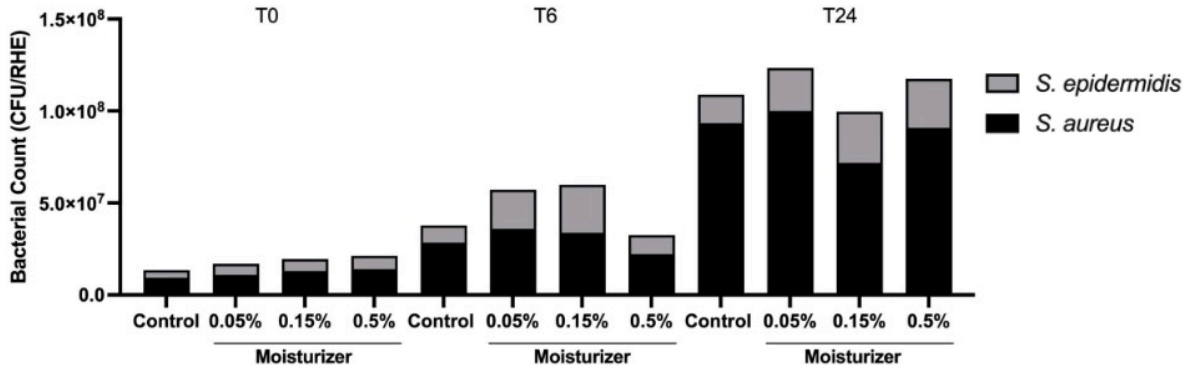
At concentrations 0.004% to 0.125%, the application of Moisturizer 1642 (n=3) resulted in 95-111% biofilm formation compared to control (n=6) following 24 hours of incubation. Thus, the moisturizer did not significantly induce nor inhibit the formation of *S. aureus* biofilm relative to control.

DISCUSSION

In the presence of Moisturizer 1642, the bacterial growth and adhesion of *S. aureus* were decreased to the benefit of *S. epidermidis*. This rebalancing effect could be particularly beneficial in dysbiotic skin disease associated with aberrant *S. aureus* colonization. The ingredients of Moisturizer 1642 likely contributed to these results. Colloidal oatmeal has demonstrated

FIGURE 1. *S. aureus* and *S. epidermidis* (A) bacterial enumeration and (B) ratio versus total bacteria in mix immediately (T0), and after 6 hours (T6) and 24 hours (T24) of incubation in well-plates. Compared to control, treatment with Moisturizer 1642 tended to slow the growth of *S. aureus* and promote the growth of *S. epidermidis*, thus rebalancing the ratio of bacterial species. These effects were greatest at 0.015% of the moisturizer.

(A)



(B)

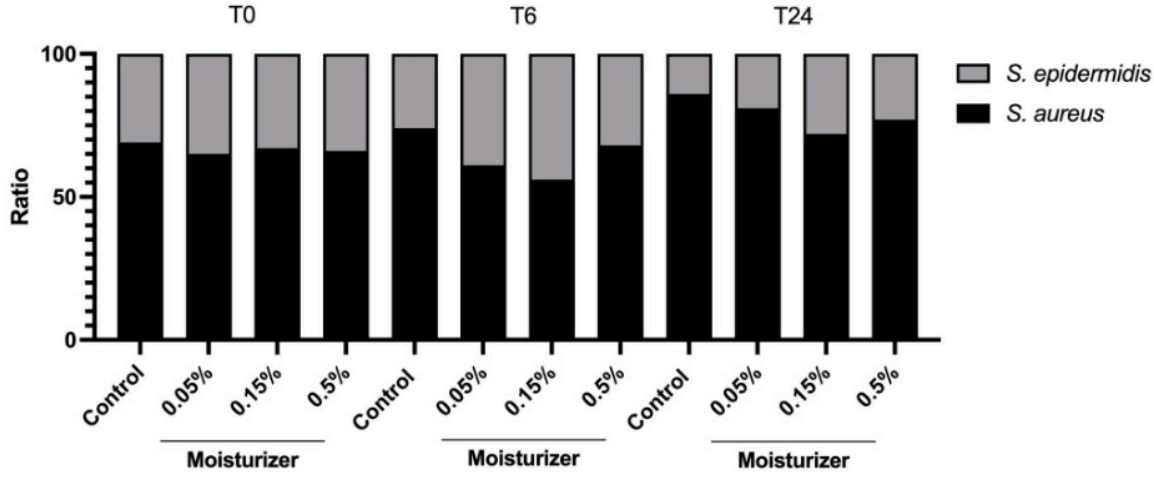
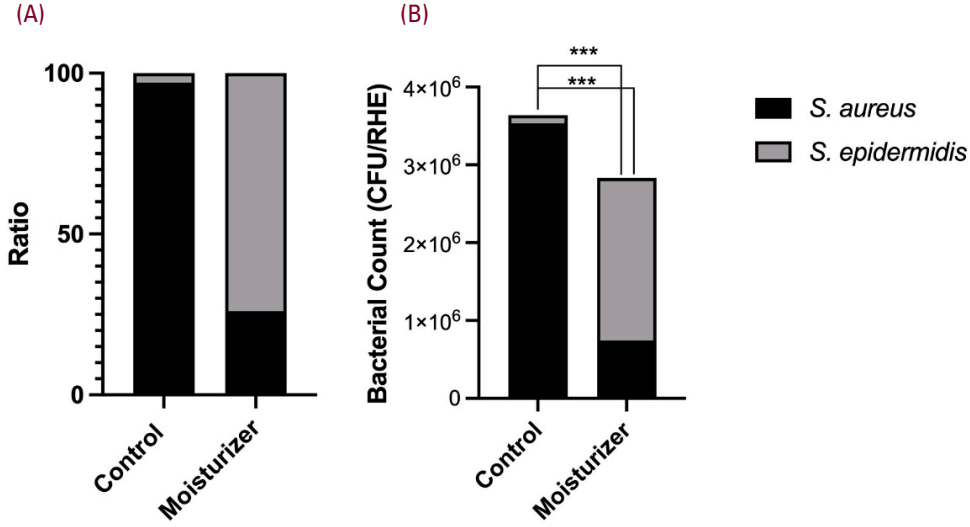


FIGURE 2. (A) Ratio and (B) quantification of *S. aureus* and *S. epidermidis* adhesion on reconstructed human epidermis after application of Moisturizer 1642 compared to control. In control conditions, the majority of bacteria adhered was *S. aureus*, while in the presence of Moisturizer, 1642 *S. epidermidis* predominated.



(n=5; *** P<0.001)

prebiotic properties.[4] In vitro studies by Liu-Walsh et al showed that 1% colloidal oat significantly increases the growth of *S. epidermidis* versus *S. aureus*.[5] Significant upregulation of *S. epidermid* is dltA gene, which confers reduced susceptibility of bacteria to cationic antimicrobial peptides on the skin was also shown, possibly explaining *S. epidermidis* outcompeting *S. aureus*.[5,6] Another key ingredient of Moisturizer 1642 is filaggrin technology containing pyrrolidone carboxylic acid (PCA), a filaggrin breakdown product. Reduced expression of *FLG*, the gene encoding filaggrin, is observed in AD skin through multiple mechanisms. PCA is a main component of epidermal natural moisturizing factor (NMF) which helps maintain hydration and a mildly acidic pH of the stratum corneum.[7] Under mildly acidic conditions created by physiologic concentrations of NMFs, *S. aureus* growth rate and its production of proteins responsible for colonization and immune evasion were decreased. Thus, the presence of PCA in Moisturizer 1642 may have deterred *S. aureus* growth and colonization through acidification of the epidermal environment. The improvement of *Staphylococcal* species growth ratio in the presence of Moisturizer 1642 may be beneficial clinically as *S. aureus* predominance is associated with more severe disease and *S. epidermidis* with less.[8]

This study was limited by its vitro design, as these results may not accurately reflect the dynamic and diverse microbiome of normal and atopic skin in humans. Future clinical studies using this moisturizer, particularly in AD, are warranted.

CONCLUSION

A moisturizer containing ingredients beneficial to AD significantly impacted the growth and adhesion of pathogenic *S. aureus* to the benefit of commensal *S. epidermidis*.

DISCLOSURES

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