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ATOPIC DERMATITIS AND CUTANEOUS MICROBIOME DYSBIOSIS

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Introduction

Atopic dermatitis (AD) is a prevalent, chronic inflammatory skin disease characterized by intense pruritus and recurrent eczematous flares. The pathophysiology of AD is highly complex and multifactorial, arising from a dynamic interplay of epidermal barrier disruption, type 2 immune dysregulation, and profound cutaneous dysbiosis. While healthy skin harbors a diverse and stable commensal microbiota, patients with AD exhibit marked alterations in microbial composition and diversity. Specifically, the disease is tightly linked to the overgrowth of pathogenic bacteria, compromising the skin's first-line chemical and physical defenses. Understanding these age-dependent, host-microbe interactions is vital for developing targeted therapeutic strategies.

Material and Methods

To evaluate the role of the cutaneous microbiome in AD, a narrative literature synthesis was conducted. Comprehensive searches of major electronic databases, including PubMed, Embase, and Web of Science, identified peer-reviewed clinical trials, prospective cohorts, and molecular studies examining skin microflora in pediatric and adult AD patients. Evaluated parameters included microbial alpha-diversity (Shannon-diversity index), taxonomic composition (beta-diversity), relative and absolute abundances of Staphylococcus aureus, and the distribution of coagulase-negative staphylococci (CoNS). Additionally, the analysis investigated correlations between microbial dysbiosis, epidermal barrier biomarkers (filaggrin expression, skin pH, transepidermal water loss), and clinical severity scores (EASI, SCORAD).

Results

Results demonstrate that AD skin is characterized by a significant loss of bacterial diversity and a massive expansion of Staphylococcus aureus. S. aureus density is elevated in lesional and non-lesional AD skin compared to healthy controls, with relative abundances exceeding 40% during acute disease flares. This colonization is facilitated by host barrier defects; loss-of-function mutations in filaggrin (FLG) or Th2/Th22 cytokine-mediated FLG suppression deplete natural moisturizing factors and raise skin pH. A near-neutral pH activates desquamatory serine proteases and upregulates S. aureus clumping factor B, enhancing bacterial adherence to corneocytes. S. aureus subsequently releases virulent phenol-soluble modulins (PSM-alpha), delta-toxin, and enterotoxin B, which trigger keratinocyte alarmin release, mast cell degranulation, and IL-17-dependent inflammation. Conversely, healthy non-lesional skin is dominated by CoNS species like S. epidermidis and S. hominis, which produce antimicrobials that actively suppress S. aureus growth. Notably, targeted systemic treatment with dupilumab (blocking IL-4/IL-13 signaling) restores skin homeostasis, resulting in a significant increase in microbial alpha-diversity and a dramatic reduction in S. aureus abundance.

Conclusion

Cutaneous microbial dysbiosis, driven by a bidirectional feedback loop between skin barrier impairment and Staphylococcus aureus virulence, is a central hallmark of atopic dermatitis pathogenesis. Proactively restoring the symbiotic balance of the skin microbiome represents a vital

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clinical goal. Transitioning from broad-spectrum topical antimicrobials toward targeted anti-staphylococcal therapies, including the transplantation of protective commensal CoNS strains or the application of anti-virulence compounds, is crucial to overcome resistant strains and optimize long-term clinical management.

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