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ATOPIC DERMATITIS ACROSS THE LIFESPAN: PATHOGENESIS AND THERAPEUTIC HORIZONS

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Introduction

Atopic dermatitis (AD) is a highly prevalent, chronic inflammatory skin disease that represents a major global public health and socioeconomic burden, affecting up to twenty percent of children and ten percent of adults worldwide. Pathophysiologically, the disease is characterized by a complex, bimodal lifespan trajectory driven by a multifaceted, dynamic interplay of epidermal barrier dysfunction, innate and adaptive immune dysregulation, and microbial dysbiosis. The clinical presentation undergoes distinct age-dependent transitions, progressing from acute, Th17-skewed extensor lesions in infancy to chronic, Th22- and Th1-skewed flexural plaques in adulthood. Furthermore, early-life skin barrier impairment frequently initiates the "atopic march," sequentially predisposing children to food allergies, asthma, and allergic rhinitis, with longitudinal trajectories displaying significant racial and socioeconomic variations.

Material and Methods

To analyze the clinical, genetic, and therapeutic landscape of AD, a systematic narrative synthesis was conducted. Primary electronic databases, including PubMed, Embase, and Web of Science, were searched utilizing keywords such as "atopic dermatitis," "filaggrin," "microbiome," and "dupilumab." Selection criteria prioritized peer-reviewed randomized controlled trials, prospective longitudinal cohorts, and meta-analyses. Evaluated primary endpoints included molecular markers of skin barrier integrity, such as transepidermal water loss (TEWL) and claudin-1 expression, alongside clinical efficacy outcomes of targeted therapies, measured by Eczema Area and Severity Index (EASI) scores, Children's Dermatology Life Quality Index (CDLQI) scores, and Investigator Global Assessment (IGA) responses.

Results

Genetic and molecular profiling revealed that loss-of-function mutations in the filaggrin (FLG) gene dramatically compromise stratum corneum hydration and elevate skin pH, which activates desquamatory serine proteases and degrades tight junction proteins. Consequently, this barrier disruption facilitates the transepidermal penetration of environmental allergens and golden Staphylococcus aureus, which further drives Th2-mediated inflammation. Longitudinal cohort studies revealed significant demographic disparities: White children exhibit higher baseline TEWL and progress to food allergies, whereas Black children display more intact skin barriers but experience higher asthma risk associated with elevated secondhand smoke and air pollution. In addition to genetic mutations, environmental triggers such as airborne pollutants (specifically formaldehyde and nitrogen dioxide) further compromise barrier homeostasis and escalate transepidermal water loss. Therapeutically, clinical trials and meta-analyses demonstrate that the targeted IL-4Rα antagonist dupilumab, when combined with topical corticosteroids, significantly rescues moderate-to-severe pediatric AD. In the Phase III LIBERTY AD PRE-SCHOOL trial, dupilumab-treated children aged six

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months to five years achieved a forty-six percent EASI-75 response and significant reductions in sleep loss and pruritus compared to placebo.

Conclusion

Atopic dermatitis is a complex, heterogeneous barrier-immune disorder that cannot be managed with a uniform, empirical approach. Identifying patient-specific genetic architecture, epidermal barrier integrity, and systemic cytokine polarization is essential for clinical management. Transitioning from broad-spectrum systemic immunosuppression toward targeted precision therapies, including dupilumab, Janus kinase inhibitors, and commensal microbiome transplants, represents a crucial paradigm shift to optimize long-term dermatological and psychosocial patient outcomes.

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