

TH2-DOMINANT IMMUNE POLARIZATION, INTERLEUKIN-4/INTERLEUKIN-13 AXIS DYSREGULATION, AND IGE-MEDIATED SENSITIZATION IN PEDIATRIC ATOPIC DERMATITIS PATHOGENESIS

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Annotation: *This thesis characterizes the Th2-dominant immunological landscape of pediatric atopic dermatitis, delineating the mechanistic roles of interleukin-4, interleukin-13, and allergen-specific immunoglobulin E in perpetuating cutaneous inflammation and systemic atopic sensitization. Through cytokine multiplex profiling, flow cytometric T-lymphocyte subset analysis, and allergen-specific immunoglobulin E quantification, the research establishes the developmental immunological framework governing disease severity and progression trajectory in the pediatric population.*

Keywords: *pediatric atopic dermatitis, Th2 polarization, interleukin-4, interleukin-13, immunoglobulin E, allergen sensitization, dendritic cells, thymic stromal lymphopoietin, immune dysregulation, atopic march.*

Nowadays, atopic dermatitis in children represents the most prevalent chronic inflammatory skin disorder of early childhood, affecting 15-20% of pediatric populations in industrialized nations and serving as the initiating disorder of the atopic march - a sequential clinical progression through food allergy, allergic rhinitis, and bronchial asthma that affects up to 60% of children with early-onset moderate-to-severe disease. The immunological architecture of pediatric atopic dermatitis is fundamentally distinguished from adult-onset disease by its pronounced Th2 polarization bias, reflecting the physiologically

Th2-skewed immune ontogeny of the neonatal and early childhood periods and the disproportionate susceptibility of the immature adaptive immune system to allergen-driven sensitization through a deficient epidermal barrier interface. Understanding this developmental immunological vulnerability is essential for designing age-appropriate intervention strategies capable of interrupting the atopic march at its earliest clinical manifestation.

The immunological initiation of pediatric atopic dermatitis proceeds through allergen penetration across the structurally deficient neonatal epidermal barrier, where Langerhans cells and dermal plasmacytoid dendritic cells bearing high-affinity immunoglobulin E receptor - Fc epsilon receptor type one - on their surface capture aeroallergen and food allergen molecules and transport them to regional lymph nodes. In the lymph node microenvironment, antigen-presenting dendritic cells expressing the Th2-polarizing cytokines interleukin-4 and thymic stromal lymphopoietin drive naive CD4⁺ T-lymphocyte differentiation toward the Th2 effector phenotype, characterized by high-level secretion of interleukin-4, interleukin-5, interleukin-13, and interleukin-31 and minimal interferon-gamma counter-regulation. Cytokine multiplex analysis of serum samples from 96 children aged 6 months to 10 years with active atopic dermatitis demonstrated markedly elevated interleukin-4 concentrations at a mean of 47.3 picograms per milliliter compared to 8.2 picograms per milliliter in age-matched healthy controls, alongside interleukin-13 levels elevated 5.4-fold above reference range. Interleukin-31, the principal pruritogenic cytokine mediating itch through direct neuronal sensitization of dorsal root ganglion neurons expressing interleukin-31 receptor alpha, correlated positively with Scoring Atopic Dermatitis index values across all age subgroups, confirming its role as both a mechanistic mediator and a quantitative disease activity biomarker.

Flow cytometric T-lymphocyte subset analysis revealed that Th2-positive CRTH2-expressing CD4⁺ lymphocytes constituted 34.7% of

circulating helper T-lymphocytes in moderate-to-severe pediatric patients, significantly exceeding the 11.3% frequency recorded in healthy controls. Total serum immunoglobulin E concentrations demonstrated age-dependent elevation, with the highest values observed in children aged 3-7 years, reflecting peak sensitization acquisition during the critical window of allergen exposure and immune programming. Allergen-specific immunoglobulin E against house dust mite, egg white, and peanut antigens was detectable in 71.8% of the study cohort, confirming the high prevalence of polysensitization concurrent with cutaneous disease activity and its correlation with subsequent respiratory atopic disease development.

Pediatric atopic dermatitis is immunologically defined by profound Th2 polarization, interleukin-4 and interleukin-13 cytokine axis dominance, and allergen-specific immunoglobulin E-mediated sensitization, collectively establishing a developmentally determined immune dysregulation framework that drives cutaneous inflammation and predicts atopic march progression, necessitating early targeted immunological intervention.

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