Cellular and molecular immunologic mechanisms in patients with atopic dermatitis


Atopic dermatitis (AD) is a complex skin disease frequently associated with other diseases of the atopic diathesis. Recent evidence supports the concept that AD can also recognize other comorbidities, such as chronic inflammatory bowel or cardiovascular diseases. These comorbidities might result from chronic cutaneous inflammation or from a common, yet-to-be-defined immunologic background leading to immune deviations. The activation of immune cells and their migration to the skin play an essential role in the pathogenesis of AD. In patients with AD, an underlying immune deviation might result in higher susceptibility of the skin to environmental factors. There is a high unmet medical need to define immunologic endotypes of AD because it has significant implications on upcoming stratification of the phenotype of AD and the resulting targeted therapies in the development of precision medicine. This review article emphasizes studies on environmental factors affecting AD development and novel biological agents used in the treatment of AD. Best evidence of the clinical efficacy of novel immunologic approaches using biological agents in patients with AD is available for the anti-IL-4 receptor α-chain antibody dupilumab, but a number of studies are currently ongoing with other specific antagonists to immune system players. These targeted molecules can be expressed on or drive the cellular players infiltrating the skin (e.g., T lymphocytes, dendritic cells, or eosinophils). Such approaches can have immunomodulatory and thereby beneficial clinical effects on the overall skin condition, as well as on the underlying immune deviation that might play a role in comorbidities. An effect of these immunologic treatments on pruritus and the disturbed microbiome in patients with AD has other potential consequences for treatment.

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