Abstract

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Immune mechanisms of allergen-specific sublingual immunotherapy.

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Abstract

Sublingual immunotherapy has been shown in some clinical studies to modulate allergen-specific antibody responses [with a decrease in the immunoglobulin E/immunoglobulin G4 (IgE/IgG4) ratio] and to reduce the recruitment and activation of proinflammatory cells in target mucosa. Whereas a central paradigm for successful immunotherapy has been to reorient the pattern of allergen-specific T-cell responses in atopic patients from a T helper (Th)2 to Th1 profile, there is currently a growing interest in eliciting regulatory T cells, capable of downregulating both Th1 and Th2 responses through the production of interleukin (IL)-10 and/or transforming growth factor (TGF)-beta. We discuss herein immune mechanisms involved during allergen-specific sublingual immunotherapy (SLIT), in comparison with subcutaneous immunotherapy. During SLIT, the allergen is captured within the oral mucosa by Langerhans-like dendritic cells expressing high-affinity IgE receptors, producing IL-10 and TGF-beta, and upregulating indoleamine dioxygenase (IDO), suggesting that such cells are prone to induce tolerance. The oral mucosa contains limited number of proinflammatory cells, such as mast cells, thereby explaining the well-established safety profile of SLIT. In this context, second-generation vaccines based on recombinant allergens in a native conformation formulated with adjuvants are designed to target Langerhans-like cells in the sublingual mucosa, with the aim to induce allergen-specific regulatory T cells. Importantly, such recombinant vaccines should facilitate the identification of biological markers of SLIT efficacy in humans.

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