Synopsis of Original Research Paper

# New insight on allergen immunotherapy for keeping skin homeostasis

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Allergy is an inappropriate immune response to allergens. Allergy requires initial sensitization with a specific allergen. The subsequent exposure to the same allergen result in patho(physio)logical responses mediated by immunoglobulin E (IgE) and mast cells. We have investigated high-affinity receptor for IgE (FcεRI) signals that control mast cell activation, especially focusing on the complexity of FcεRI activation and of the signaling network in response to different affinity of allergens. What we found is that IgE and FcεRI activate a complex regulatory network by the affinity of allergens that governs the type of allergic disease symptoms. In the mouse model, both high- and low-affinity allegens led to similar levels of immune cells infiltrating the site of inflammation. However, the types of infiltrating cells differed depending on whether the allergens used were high- or low-affinity. Whereas neutrophils were the dominant cell type infiltrating under a high-affinity antigen challenge, monocyte/macrophages were more abundant with the low-affinity antigen challenge. The physiological relevance of the differences in immune cell recruitment is still unclear. In this study, we explore the roles of different immune cell recruitment on physiological relevance on allergic diseases. Thus, we develop novel theories for allergen affinity-dependent immunotherapy for keeping skin homeostasis.