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Specific immunotherapy suppresses Th2 responses via modulating TIM1/TIM4 interaction on dendritic cells.

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Abstract

BACKGROUND: Specific immunotherapy (SIT) is the only curable remedy for allergic disorders currently; however, the underlying mechanism is not fully understood yet. This study aimed to elucidate the mechanism of SIT on suppressing TIM4 (T cell immunoglobulin mucin domain molecule 4) expression in dendritic cells (DCs) and modulating the skewed T helper 2 (Th2) responses in patients with airway allergy.

METHODS: Twenty patients with allergic rhinitis (AR) were treated with SIT for 3 months. Before and after SIT, the expression of TIM4 in peripheral DC and TIM1 in Th2 cells was examined. The role of Fc gamma receptor (FcgammaR) I and II in modulating the expression of TIM4 in DCs was investigated.

RESULTS: The interaction of TIM1/TIM4 played a critical role in sustaining the polarization status of Th2 cells in AR patients. Cross-linking FcgammaRI by antigen/IgG complexes increased the production of TIM4 by dendritic cells via upregulating tumor necrosis factor-alpha in DCs. Exposure to microbial products promoted the expression of FcgammaRI in DCs that further increased the expression of TIM4. Exposure to specific antigens alone upregulated the expression of FcgammaRII in DCs, that suppressed the expression of TIM4.

CONCLUSIONS: We conclude that SIT suppresses the skewed Th2 responses via disrupting the interaction of TIM1/TIM4 in antigen-specific Th2 cells.

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Publication Types, MeSH Terms, Substances

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