Local Immunoglobulin E in nasal polyps: Role and modulation

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Abstract

In the airway, IgE is traditionally regarded as a key mediator in allergic diseases, such as AR and allergic asthma. However, growing evidence demonstrates the importance of local IgE in airway inflammatory diseases, irrespective of the presence of allergy. In this review, we discuss the most recent evidence for IgE in chronic rhinosinusitis with nasal polyps(CRSwNP), including the local IgE's characteristics, the modulation of its synthesis, and function. The levels of local IgE are significantly elevated in polyps independently of IgE serum levels and atopic status. Local IgE is polyclonal and functional, which is correlated with type 2 inflammation. IgE is produced by active B cells and is dependent on the classing switch recombination(CSR). In NPs, this process is triggered by not only allergens but also microbial colonization, especially the superantigen- *Staphylococcus aureus*. The production of local IgE is modulated by lymphocytes(such as Tfh, ILC2s, iTreg), cytokines(such as IL-4, IL-13, IFN-γ, TGF-β, IL-2, IL-21), transcription factors, and B cell intrinsic factor. Due to the central role of IgE in NPs, it is regarded as an ideal target for therapy and has been proved to be clinically successful. Based on this knowledge, we believe that exploring the trigger and regulatory factors for the activation of local B cells and CSR to IgE will provide more valuable information for us to recognize the pathological mechanisms of local IgE and offer the possible option for new [therapeutic](#/javascript:;) targets of NPs.

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