The High Affinity IgE Receptor (FcεRI) as a Target for Anti-allergic Agents

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Prevention of the effector cell activation via high affinity IgE receptor (FcεRI) is thought to be a straightforward strategy for suppressing the allergic reaction. Among the numerous methods to prevent the activation through FcεRI, three versions are described in this article. The first and second ideas involve inhibition of binding between FcεRI and IgE with a soluble form of the FcεRI α chain and a humanized antibody directed against the α chain, respectively. Both of these paths involve suppression the histamine release from human peripheral blood basophils in vitro. They also inhibited the allergic reaction in vivo. The soluble α inhibited the anaphylactic reaction in rodents and the Fab fragments of the humanized anti-FcεRI α chain antibody suppressed the dermal response in rhesus monkeys. The third idea involves repression of FcεRI expression by suppressing the transcription of the genes encoding the subunits of FcεRI. Although no plausible candidate molecule for actualizing this idea can be identified at present, further analyses of the transcriptional regulatory mechanisms in the human FcεRI α and β chain genes will lead to the discovery of novel targets for developing anti-allergic agents.

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