Regulation of murine hypersensitive responses by Fc receptors

http://www.firstlight.cn 2005-10-14

Humoral and cellular immune responses communicate with each other via Fc receptors (FcR) expressed on various hematopoietic cells. Recent studies on several FcR knockout mice demonstrated pivotal roles of an IgG/FcγR system in the regulation of immune responses and the onset of hypersensitivity. The γ subunit of FcR is an essential component of the complex and is required for both receptor assembly and signal transduction. FcR γ chain-deficient mice have lost the functional expression of FcεRI, FcγRI, and FcγRIII and are unable to mount several types of hypersensitive reactions, including the skin Arthus reaction. In contrast, FcγRII-deficient mice exhibit augmented humoral immune responses and IgG-mediated anaphylaxis reactions. Thus, the regulatory system of murine hypersensitive responses involves both positive and negative signaling through FcR. In B cells, FcγRIIb modulates membrane Ig-induced Ca2+ mobilization by inhibiting Ca2+ influx through phosphorylation of its immunoreceptor tyrosine-based inhibition motif and recruitment of cytoplasmic phosphatases. Elucidation of the detailed mechanisms of negative regulatory signaling in the inflammatory effector cells by FcγRIIb as well as several groups of potent inhibitory molecules expressed on such cells should be valuable in the development of novel therapeutic procedures for allergic disorders.

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