1: J Immunol. 2003 Dec 1;171(11):5760-8.

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Human CD25+ regulatory T cells maintain immune tolerance to nickel in healthy, nonallergic individuals.

Cavani A, Nasorri F, Ottaviani C, Sebastiani S, De Pita O, Girolomoni G.

Laboratory of Immunology, Istituto Dermopatico dell'Immacolata, Rome, Italy. cavani@idi.it

We investigated the capacity of CD25(+) T regulatory cells (Treg) to modulate T cell responses to nickel, a common cause of allergic contact dermatitis. CD4(+) T cells isolated from the peripheral blood of six healthy, nonallergic individuals showed a limited capacity to proliferate in response to nickel in vitro, but responsiveness was strongly augmented (mean increment +/- SD, 240 +/- 60%) when cells were depleted of CD25(+) Treg. Although CD25(+) Treg were anergic to nickel, a small percentage up-regulated membrane CTLA-4 upon nickel exposure. CD25 (+) Treg strongly and dose-dependently inhibited nickel-specific activation of CD25(-) T lymphocytes in coculture experiments in a cytokine-independent, but cell-to-cell contact-dependent, manner. Approximately 30% of circulating CD25(+) Treg expressed the cutaneous lymphocyte-associated Ag (CLA), and CLA(+)CD25(+) Treg were more efficient than CLA(-)CD25(+) cells in suppressing nickel responsiveness of CD25(-) T cells. The site of a negative patch test in response to nickel showed an infiltrate of CD4(+)CLA(+) cells and CD25(+) cells, which accounted for approximately 20% of the total T cells isolated from the tissue. Skin-derived T cells suppressed nickel-specific responses of peripheral blood CD25(-) T cells. In addition, 60 +/- 14% of peripheral blood CD25(+) Treg expressed the chemokine receptor CCR7 and strongly inhibited naive T cell activation in response to nickel. Finally, CD25(+) T cells isolated from peripheral blood of nickel-allergic patients showed a limited or absent capacity to suppress metal-specific CD4(+) and CD8(+) T cell responses. The results indicates that in healthy individuals CD25(+) Treg can control the activation of both naive and effector nickel-specific T cells.

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