

Abstract

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Reciprocal TH17 and regulatory T cell differentiation mediated by retinoic acid.

Mucida D, Park Y, Kim G, Turovskaya O, Scott I, Kronenberg M, Cheroutre H.

La Jolla Institute for Allergy and Immunology, 9420 Athena Circle, La Jolla, CA 92037, USA.

BACKGROUND: The cytokine transforming growth factor-beta (TGF-beta) converts naïve T cells into regulatory T (Treg) cells that prevent autoimmunity. However, in the presence of interleukin-6 (IL-6), TGF-beta has also been found to promote the differentiation of naïve T lymphocytes into proinflammatory IL-17 cytokine-producing T helper 17 (T(H)17) cells, which promote autoimmunity and inflammation. This raises the question of how TGF-beta can generate such distinct outcomes.

RESULTS: We identified the vitamin A metabolite retinoic acid as a key regulator of TGF-beta-dependent immune responses, capable of inhibiting the IL-6-driven induction of proinflammatory T(H)17 cells and promoting anti-inflammatory Treg cell differentiation.

CONCLUSION: These findings indicate that a common metabolite can regulate the balance between pro- and anti-inflammatory immunity.

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