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The Cellular source and target of IL-21 in the K/BxN mouse model of rheumatoid arthritis

Haochu Huang
University of Chicago, USA

Abstract

Cytokines are major regulators of immune responses. Particularly, IL-21 is a pluripotent cytokine that regulates B cell and plasma cell differentiation, and is thought to be an autocrine factor for T_{FH} and T_H17 differentiation, two T cell subsets implicated in autoimmunity. The relevant cellular source and target cells of IL-21 in autoimmunity have not been well characterized. We investigated this issue in the K/BxN mice by comparing the ability to induce arthritis of wildtype KRN T cells, IL-21 knockout KRN T cells that cannot produce IL-21, IL-21 receptor knockout KRN T cells or B cells that cannot signal by IL-21, or ROR□t knockout KRN T cells that are defective in Th17 differentiation. Our results showed that IL-21 production by T_{FH} but not T_H17 T cells is critical for arthritis development. However, IL-21 does not act on T cells as an autocrine factor but rather acts on B cells to form germinal centers and produce autoantibodies. These results clarify the roles of IL-21 signaling to T cells or B cells in autoimmune diseases and have implications in developing effective therapies for rheumatoid arthritis and other antibody-mediated autoimmune diseases.

Biography

Haochu Huang has completed his PhD from Johns Hopkins University and postdoctoral studies in the lab of Drs. Christophe Benoist and Diane Mathis at Harvard Medical School. His work focuses on tolerance and regulation of autoreactive T cells and B cells in normal immune system and autoimmune diseases.