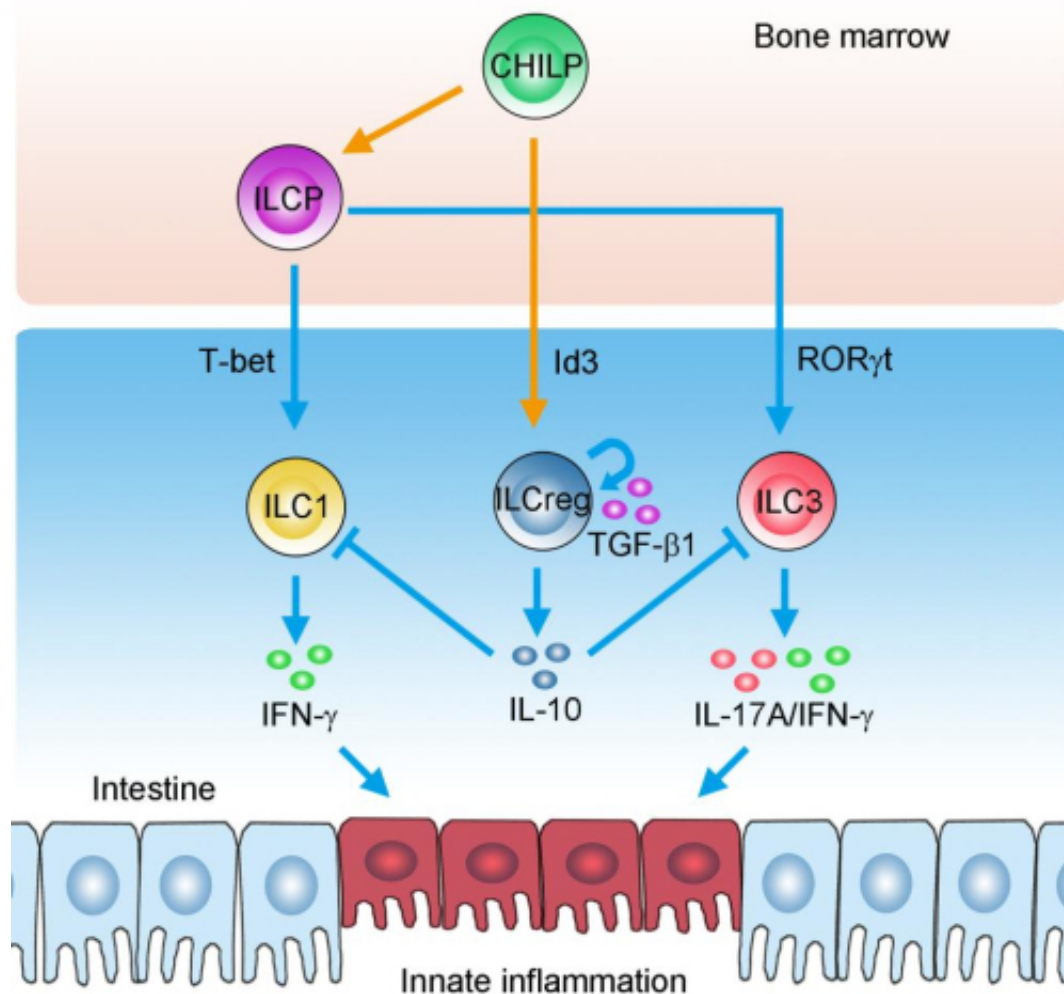


Innate lymphoid cells play an important role in regulation of intestinal inflammation

August 28 2017



ILCregs contribute to the resolution of innate intestinal inflammation by inhibition of ILC1s and ILC3s. Credit: IBP

The intestine contains an extensive and diverse microbial biome, a population that includes potential pathogens and dietary antigens that need to be tolerated. Dysregulation of mucosal responses may cause a loss of tolerance, leading to harmful intestinal inflammation such as human inflammatory bowel disease (IBD).

Innate lymphoid cells (ILCs) are located in mucosal surfaces to potentiate immune responses, sustain mucosal integrity and promote lymphoid organogenesis. Three subsets of ILCs have been defined to date and produce substantial effector cytokines upon harmful stress. These ILCs play critical roles in the regulation of type 1, type 2 and type 3 (or Th17 cell) responses, controlling host protective immunity and intestinal homeostasis.

Researchers from FAN Zusen's group at the Institute of Biophysics (IBP) of the Chinese Academy of Sciences have identified a regulatory subpopulation of ILCs (called ILCregs) that exist in the gut and harbor a unique genetic identity distinct from ILCs or regulatory T [cells](#) (Tregs). During inflammatory stimulation, ILCregs can be induced in the intestine and suppress the activation of ILC1s and ILC3s via secretion of IL-10, leading to protection against innate intestinal inflammation.

In addition, TGF- β 1 is consequently secreted by ILCregs upon intestinal inflammation, and autocrine TGF- β 1 sustains the maintenance and increase of ILCregs. Therefore, ILCregs exert an inhibitory role in the [innate immune response](#), favoring the resolution of [intestinal inflammation](#). Researchers found that ILCregs may be used to develop potential therapies to restore immune tolerance in chronic inflammatory and autoimmune diseases.

More information: Shuo Wang et al, Regulatory Innate Lymphoid Cells Control Innate Intestinal Inflammation, *Cell* (2017). [DOI: 10.1016/j.cell.2017.07.027](#)

Provided by Chinese Academy of Sciences

Citation: Innate lymphoid cells play an important role in regulation of intestinal inflammation (2017, August 28) retrieved 1 January 2023 from

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