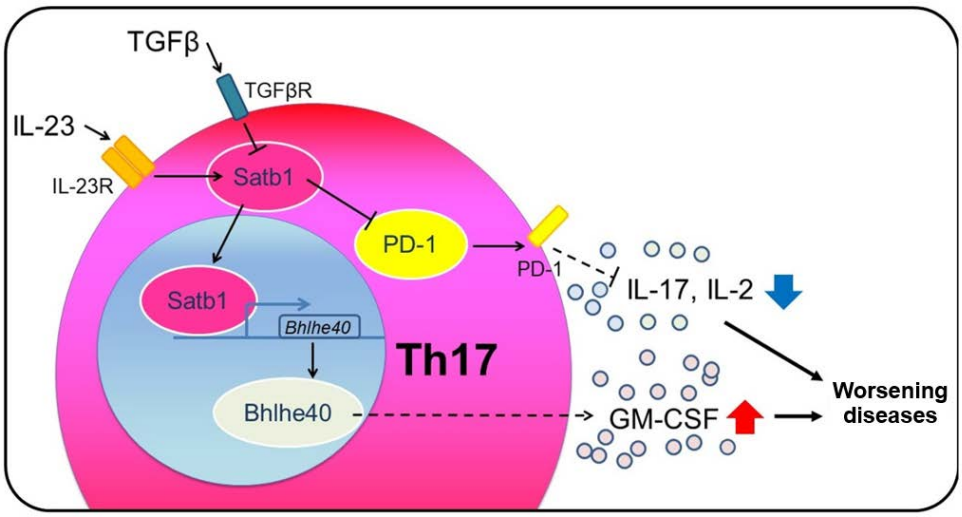
Satb1 regulates the effector program of encephalitogenic tissue Th17 cells in chronic inflammation

Keywords: immunology, cytokine, autoimmunity, acquired immunity, Th17

The genome organizer, special AT-rich sequence-binding protein-1 (Satb1), plays a pivotal role in the regulation of global gene networks in a cell type-dependent manner and is indispensable for the development of multiple cell types, including mature CD4+ T, CD8+ T, and Foxp3+ regulatory T cells in the thymus. However, it remains unknown how the differentiation and effector program of the Th subsets in the periphery are regulated by Satb1. Keiko Yasuda (Osaka University Hospital/Graduate School of Medicine), Keiji Hirota, Shimon Sakaguchi (Experimental Immunology, IFReC) and their research group demonstrate that Satb1 differentially regulates gene expression profiles in non-pathogenic and pathogenic Th17 cells and promotes the pathogenic effector program of encephalitogenic Th17 cells by regulating GM-CSF via Bhlhe40 and inhibiting PD-1 expression. However, Satb1 is dispensable for the differentiation and non-pathogenic functions of Th17 cells. These results indicate that Satb1 regulates the specific gene expression and function of effector Th17 cells in tissue inflammation.



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