MKL-1 is a coactivator for STAT5b, the regulator of Treg cell development and function

Yuan Xiang
Jun Wang
Jia Peng Li
Wei Guo
Feng Huang
Hui Min Zhang
Han Han Li
Zhou Tong Dai
Zi Jian Zhang
Hui Li
Le Yuan Bao
Chao Jiang Gu
Kun Chen
Tong Cun Zhang
Xing Hua Liao

Video Byte

Keywords: MKL-1, STAT5b, Treg, ITP, T cell, regulatory T cell, autoimmunity, autoimmune disease, Cell Communication and Signaling

Posted Date: November 3rd, 2020

DOI: <https://doi.org/10.21203/rs.3.rs-102119/v1>

License: This work is licensed under a Creative Commons Attribution 4.0 International License.

[Read Full License](#)

Abstract

Autoimmune disease happens when the body's immune system reacts to its own cells and tissues. Central to this process are regulatory T cells (Tregs), which control the inflammatory CD4 T cell response. Understanding how to boost Tregs will help researchers develop new therapies for autoimmunity. In a recent study, researchers zeroed in on a broad regulator of cell differentiation, migration, and proliferation – MKL-1. Using molecular techniques, they examined its interaction with STAT5, a transcriptional activator central to Treg development. After overexpressing or silencing MKL-1 and STAT5 in cell lines, they evaluated protein interactions and Treg gene expression. The results showed that MKL-1 acts a coactivator for STAT5b targets in Tregs. MKL-1 was upregulated during Treg differentiation, and overexpressing MKL-1 enhanced the expression of Treg markers. Silencing STAT5b blocked MKL-1 from activating Treg genes, showing its dependence on STAT5b for its function. The study suggests that the pivotal interaction between STAT5 and MKL-1 may be an ideal target for modulating Treg cells, giving hope for new therapies for autoimmune disease.