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

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Video Byte

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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.