

Wnt/beta-catenin and LIF-Stat3 signaling pathways converge on Sp5 to promote mouse embryonic stem cell self-renewal.

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Public Summary:

Leukemia inhibitor factor (LIF)-Stat3 and Wnt/beta-catenin signaling pathways play an important role in promoting mouse embryonic stem cell (mESC) self-renewal. The common downstream targets of these two pathways, however, have never been identified. In this study, we found that the LIF-Stat3 and Wnt/beta-catenin signaling pathways converge on a common gene called Sp5 to promote mESC self-renewal. Forced Sp5 expression can reproduce partial effects of Wnt/beta-catenin signaling but mimics most features of LIF-Stat3 signaling to maintain mESCs in culture. Our results suggest that Sp5 is an important component of the regulatory network governing mESC pluripotency.

Scientific Abstract:

Activation of leukemia inhibitor factor (LIF)-Stat3 or Wnt/beta-catenin signaling promotes mouse embryonic stem cell (mESC) self-renewal. A myriad of downstream targets have been identified in the individual signal pathways, but their common targets remain largely elusive. In this study, we found that the LIF-Stat3 and Wnt/beta-catenin signaling pathways converge on Sp5 to promote mESC self-renewal. Forced Sp5 expression can reproduce partial effects of Wnt/beta-catenin signaling but mimics most features of LIF-Stat3 signaling to maintain undifferentiated mESCs. Moreover, Sp5 is able to convert mouse epiblast stem cells into a naive pluripotent state. Thus, Sp5 is an important component of the regulatory network governing mESC naive pluripotency.

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