SPI1 (PU.1) gene transcription is stimulated by RUNX1:CBFB:KMT2A

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Introduction

Reactome is open-source, open access, manually curated and peer-reviewed pathway database. Pathway annotations are authored by expert biologists, in collaboration with Reactome editorial staff and cross-referenced to many bioinformatics databases. A system of evidence tracking ensures that all assertions are backed up by the primary literature. Reactome is used by clinicians, geneticists, genomics researchers, and molecular biologists to interpret the results of high-throughput experimental studies, by bioinformaticians seeking to develop novel algorithms for mining knowledge from genomic studies, and by systems biologists building predictive models of normal and disease variant pathways.

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Literature references

Fabregat, A., Sidiropoulos, K., Viteri, G., Forner, O., Marin-Garcia, P., Arnau, V. et al. (2017). Reactome pathway analysis: a high-performance in-memory approach. *BMC bioinformatics, 18*, 142. ↗

Sidiropoulos, K., Viteri, G., Sevilla, C., Jupe, S., Webber, M., Orlic-Milacic, M. et al. (2017). Reactome enhanced pathway visualization. *Bioinformatics, 33*, 3461-3467. ↗

Fabregat, A., Jupe, S., Matthews, L., Sidiropoulos, K., Gillespie, M., Garapati, P. et al. (2018). The Reactome Pathway Knowledgebase. *Nucleic Acids Res, 46*, D649-D655. ↗

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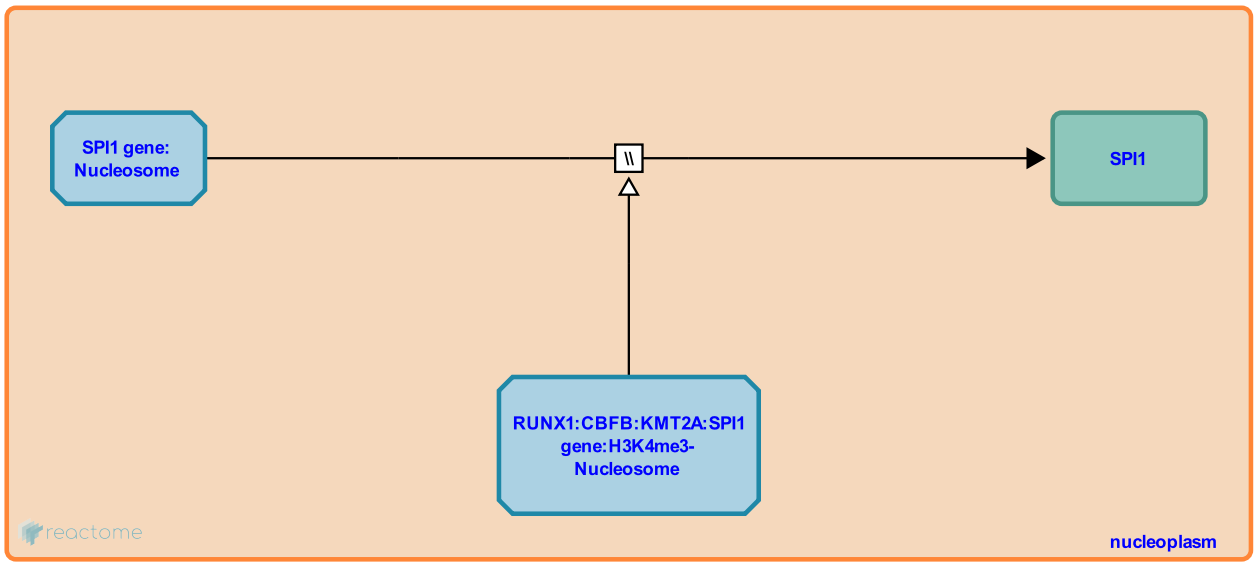
Stable identifier: R-HSA-8865505

Type: omitted

Compartments: nucleoplasm

Inferred from: Spi1 gene transcription is stimulated by RUNX1:Cbfb:KMT2A (Homo sapiens)



The SPI1 (PU.1) transcription factor represses self renewal and proliferation of HSCs (Fukuchi et al. 2008) and is needed for commitment of HSCs to specific hematopoietic lineages (Imperato et al. 2015), for example differentiation of lymphoid cells. SPI1 gene transcription is directly stimulated by the RUNX1:CBFB transcription factor complex, in the presence of the activating histone methyltransferase KMT2A (MLL) (Huang et al. 2011).

Literature references

Menendez, S., Zhang, Y., Xu, H., Liu, Y., Tenen, DG., Nimer, SD. et al. (2011). The ability of MLL to bind RUNX1 and methylate H3K4 at PU.1 regulatory regions is impaired by MDS/AML-associated RUNX1/AML1 mutations. *Blood, 118*, 6544-52. ↗

Editions

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