CEBPA gene transcription is enhanced by RUNX1, SPI1 (PU.1), GATA2, TAL1 (SCL), FLI1, MYB, LEF1, and CEBPA

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11/11/2024

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.

The development of Reactome is supported by grants from the US National Institutes of Health (P41 HG003751), University of Toronto (CFREF Medicine by Design), European Union (EU STRP, EMI-CD), and the European Molecular Biology Laboratory (EBI Industry program).

Literature references

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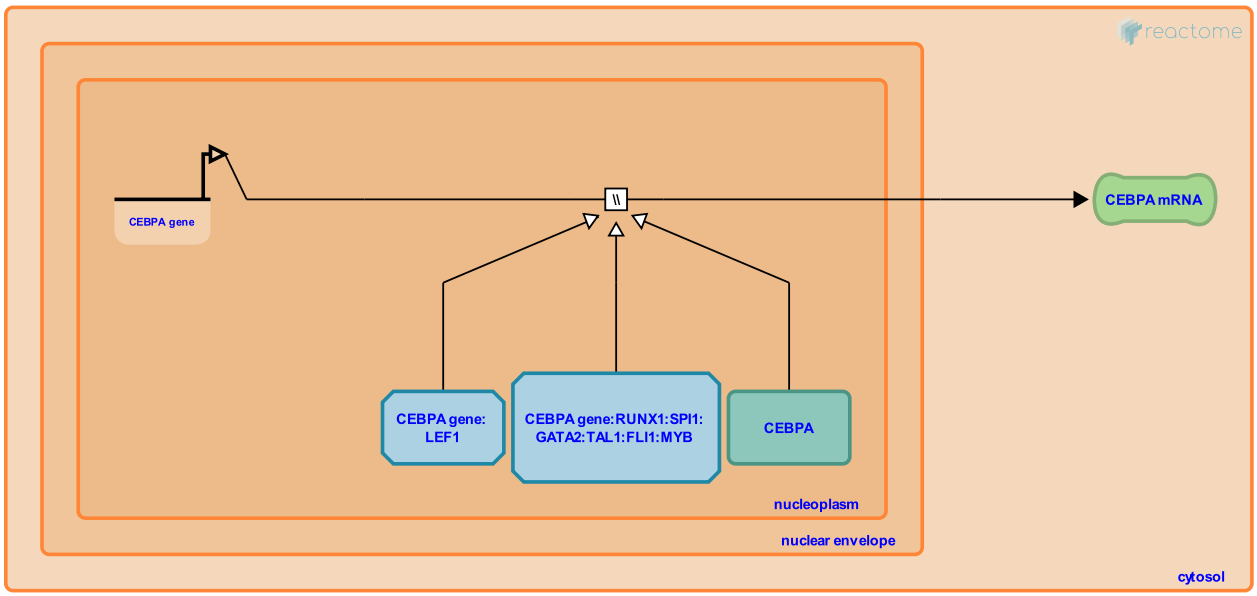
Stable identifier: R-HSA-9616243

Type: omitted

Compartments: nucleoplasm, cytosol

Inferred from: Cebpa transcription is enhanced by Runx1, Spi1 (PU.1), Gata2, Tal1 (Scl), Fli1, Myb, and Cebpa (Mus musculus)



RUNX1, SPI1 (PU.1), GATA2, TAL1 (SCL), MYB, and CEBPA itself all contribute to the level of transcription of CEBPA in hemopoietic progenitor cells and myeloid progenitor cells (inferred from mouse homologs). High levels of CEBPA appear to favor CEBPA:CEBPA homodimers and lead to granulopoiesis; low levels of CEBPA appear to favor CEBPA:AP-1 heterodimers and lead to monopoiesis. LEF1 also directly activates transcription of CEBPA (Skokowa et al. 2006, Skokowa et al. 2012), but appears to act at the transition of granulocyte-macrophage precursors to promyelocytes, a later stage of granulopoiesis.
The relative levels of SPI1 (PU.1) and CEBPA (SPI1 to CEBPA mRNA expression ratio) in granulocytic–macrophage progenitors have been suggested to regulate monocyte versus neutrophil cell-fate choice (Dahl et al. 2003).

Literature references

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Editions

2018-08-10	Authored, Edited	May, B.
2019-03-10	Reviewed	Skokowa, J.