

RUNX proteins bind the p14-ARF promoter at the CDKN2A locus

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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

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Reactome database release: 70

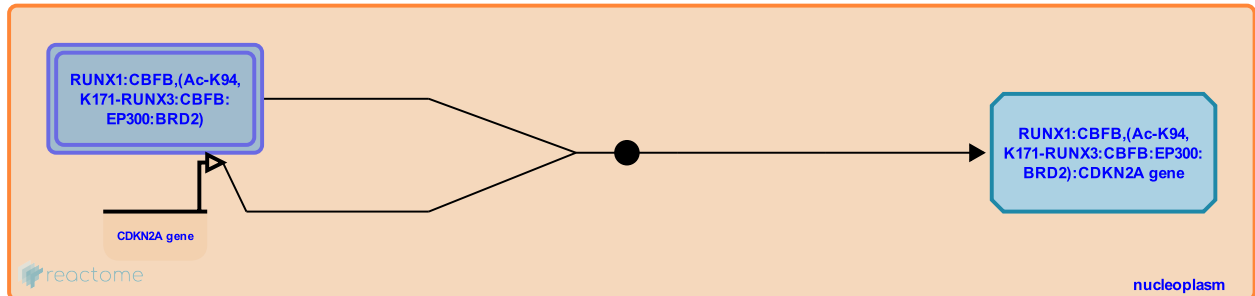
This document contains 1 reaction ([see Table of Contents](#))

RUNX proteins bind the p14-ARF promoter at the CDKN2A locus [↗](#)

Stable identifier: R-HSA-8952081

Type: binding

Compartments: nucleoplasm



The CDKN2A gene promoter that regulates transcription of p14-ARF contains Runx response elements that are known to be recognized by the RUNX1:CBFB complex (Linggi et al. 2002). Based on sequence similarity between RUNX1 and RUNX3 and the fact that the complex of acetylated RUNX3 and BRD2 positively regulate p14-ARF transcription, it is possible that RUNX3 can also bind to the Runx response elements at the p14-ARF promoter (Lee et al. 2013).

Literature references

Lee, YS., Lee, JW., Jang, JW., Chi, XZ., Kim, JH., Li, YH. et al. (2013). Runx3 inactivation is a crucial early event in the development of lung adenocarcinoma. *Cancer Cell*, 24, 603-16. [↗](#)

Linggi, B., Müller-Tidow, C., van de Locht, L., Hu, M., Nip, J., Serve, H. et al. (2002). The t(8;21) fusion protein, AML1 ETO, specifically represses the transcription of the p14(ARF) tumor suppressor in acute myeloid leukemia. *Nat. Med.*, 8, 743-50. [↗](#)

Editions

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