

HOXD1 chromatin is activated

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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: 88

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

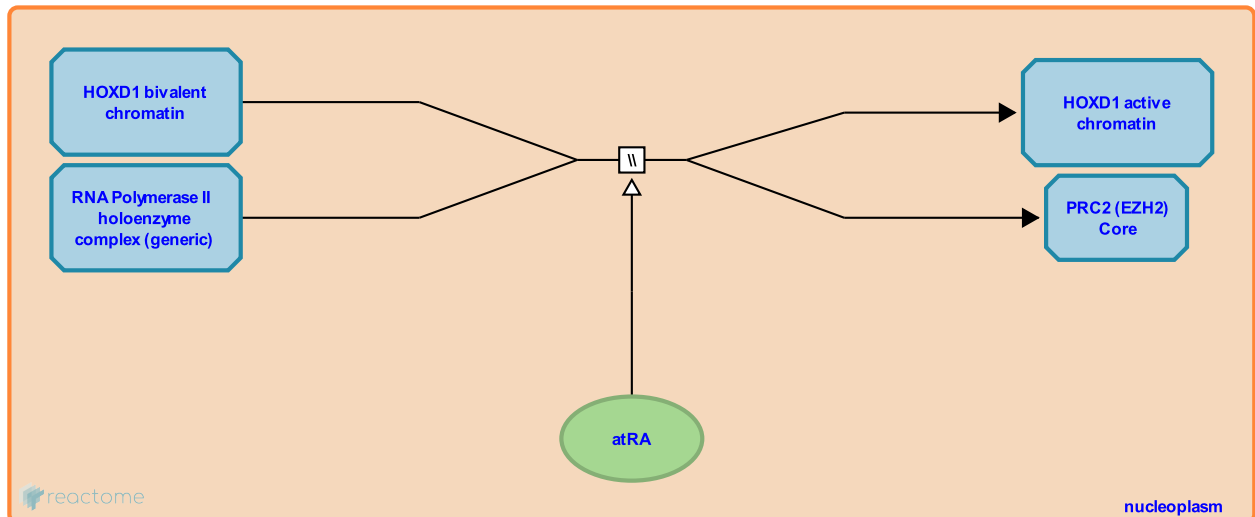
HOXD1 chromatin is activated [↗](#)

Stable identifier: R-HSA-5617445

Type: omitted

Compartments: nucleoplasm

Inferred from: [Hoxd1 chromatin is activated \(Mus musculus\)](#)



As inferred from the *Hoxd1* homolog in mouse embryos, HOXD1 is not expressed in hindbrain. In mouse, expression of *Hoxd1* begins at E8.5 in caudal lateral mesoderm. At E9.5 to E11.5 *Hoxd1* expression is observed in prosomeres p2 and p3 of the diencephalon, dermatomes, urogenital tubercle, and tail bud. Expression is inducible by retinoic acid in neuroblastoma cells, however it is unknown if the induction is direct or indirect (Manohar et al. 1996, Zha et al. 2012). Nerve Growth Factor induces *Hoxd1* expression in nociceptors of mouse embryos. As inferred from human posterior HOXD genes in primary human fibroblasts (Lan et al. 2007), other anterior HOX genes, and mouse *Hoxd1*, the activation of HOXD1 chromatin may be associated with loss of methylation at lysine-27 of histone H3 (H3K27me3) loss of polycomb repressive complex 2 (PRC2), gain of histone acetylation, and gain of methylation at histone H3K4. Like other Hox gene clusters, the *HoxD* cluster in mouse changes position relative to other loci in the nucleus during activation.

Literature references

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Editions

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