CYP24A1 hydroxylates 1,25(OH)2D, inactivating it

D'Eustachio, P., Jassal, B.
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


Reactome database release: 77

This document contains 1 reaction (see Table of Contents)
CYP24A1 hydroxylates 1,25(OH)2D, inactivating it

**Stable identifier:** R-HSA-209765

**Type:** transition

**Compartments:** mitochondrial inner membrane, cytosol

1-alpha, 25-dihydroxyvitamin D (1,25(OH)2D) is biologically inactivated through a series of reactions beginning with 24-hydroxylation and is most likely a mechanism of elimination. 24-Hydroxylation of vitamin D metabolites is largely regulated inversely to 1-hydroxylation, the initial step towards activation. Human cDNA encoding CYP24A1 was isolated in 1993 (Chen et al. 1993). Studies with expressed human CYP24A1 in Sf21 insect cells indicated that the enzyme could catalyze most, if not all, of the steps in the C23 and C24 oxidation pathways of 25(OH)D and 1,25(OH)2D metabolism (Beckman et al. 1996). Sakaki et al observed that the ratio of initial hydroxylation products at C24 to C23 was 4:1, indicating that the C24-oxidation pathway predominates in humans (Sakaki et al. 2000).

**Literature references**


**Editions**

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<td>D'Eustachio, P.</td>
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<td>Edited</td>
<td>Jassal, B.</td>
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