ADH1A,1C,4 oxidise atROL to atRAL in vitro

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06/05/2024

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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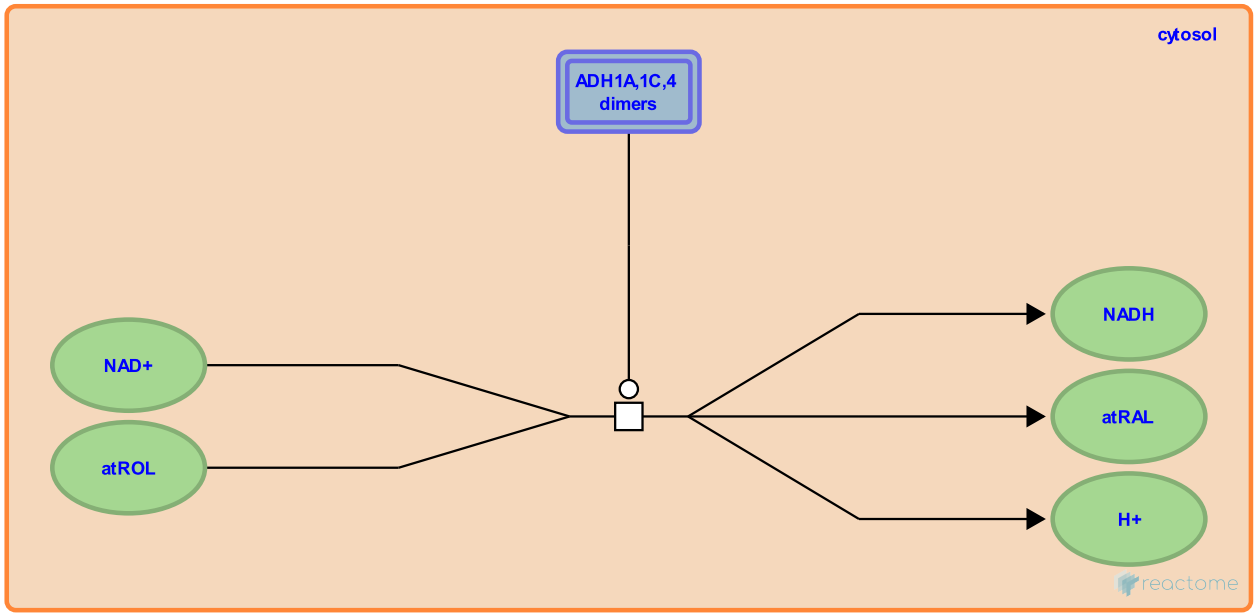
Stable identifier: R-HSA-5362564

Type: transition

Compartments: cytosol



Some alcohol dehydrogenases (ADHs) utilise NAD+ as cofactor to reversibly oxidise all-trans-retinol (atROL) to all-trans-retinal (atRAL), a retinoid aldehyde, in vitro (von Bahr-Lindstrom et al. 1986, Ikuta et al. 1986, von Bahr-Lindstrom et al. 1991, Xie et al. 1997). ADH1A (ADH1) and ADH4 have high activity and ADH1C (ADH3) has low activity with non-physiological amounts of retinol in vitro. ADH1A and ADH1C metabolize toxic amounts of retinol in vivo, but ADH4 does not. Physiological contributions of ADHs to retinol metabolism have not been demonstrated, in contrast to RDHs.

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Editions

2014-04-16	Authored, Edited	Jassal, B.
2014-07-28	Reviewed	Duester, G.
2014-09-01	Reviewed	Napoli, JL.