

# CYP4 ω-oxidises 14(R)-HDHA to MaR-L2

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https://reactome.org

### 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)

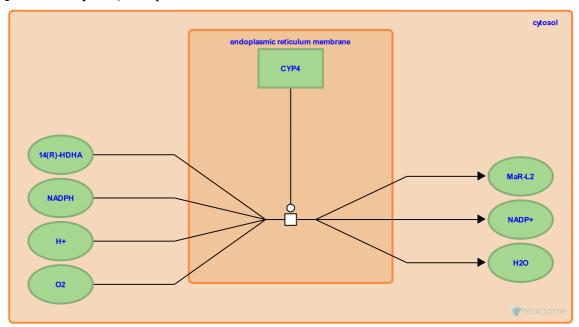
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Stable identifier: R-HSA-9027044

Type: transition

**Compartments:** cytosol, endoplasmic reticulum membrane



Cytochrome P450 (CYP) enzymes are thought to  $\omega$ -hydroxylate (position 22) 14(R)-hydroxy-docosahexaenoic acid (14(R)-HDHA) to 14(R),22-dihydroxy-docosahexaenoic acid, namely maresin-like mediator 2 (MaR-L2) (Hong et al. 2014). CYP inhibition was found to decrease the amount of MaR-L2 formed (Hong et al. 2014). The exact CYP responsible for MaR-L2 formation is unknown but is likely to be a member of the CYP4 family as those enzymes mediate the  $\omega$ -hydroxylation of fatty acids and eicosanoids (Kikuta et al. 2002). Diabetes results in delayed- or non-healing of wounds and is associated with impaired macrophage function (Brem & Tomic-Canic 2007). Leukocytes and platelets play critical roles in wound healing by mechanisms as yet unknown. Maresin-like mediators MaR-L1 and Mar-L2 are produced by leukocytes and platelets and have been shown (in vitro) to restore reparative functions of diabetic macrophages in wounds (Hong et al. 2014).

#### Literature references

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### **Editions**

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