PTGS2 dimer oxidises DHA to 13-HDHA

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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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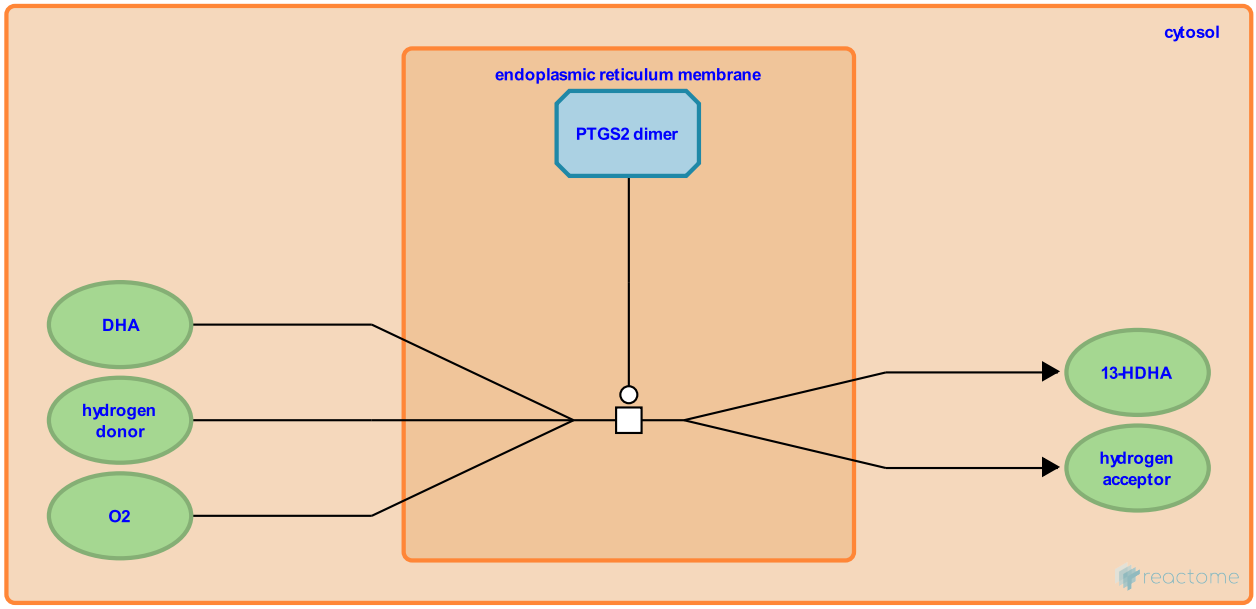
Stable identifier: R-HSA-9027532

Type: transition

Compartments: cytosol, endoplasmic reticulum membrane



In the absence of aspirin, dimeric cyclooxygenase 2 (PTGS2 aka COX2) located on the ER membrane can mediate the oxidation of docosahexaenoic acid (DHA) to 13-hydroxy-docosahexaenoic acid (13-HDHA) in activated macrophages (Serhan et al. 2002, Groeger et al. 2010). PTGS2 is an inducible enzyme expressed at sites of inflammation, infection and cancer where it can generate prostanoids that drive disease pathogenesis. It is the therapeutic target for nonsteroidal antiinflammatory drugs (NSAIDs) such as aspirin. PTGS2 is also constitutively expressed in specific tissues, especially the kidney, gastrointestinal tract, brain and thymus. Constitutive PTGS2 expression is increasingly being recognised to play a major role in homeostatic function in those tissues and is therapeutically important because NSAIDs cause cardiovascular and renal side effects in otherwise healthy individuals (Kirkby et al. 2016).

Literature references

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Editions

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