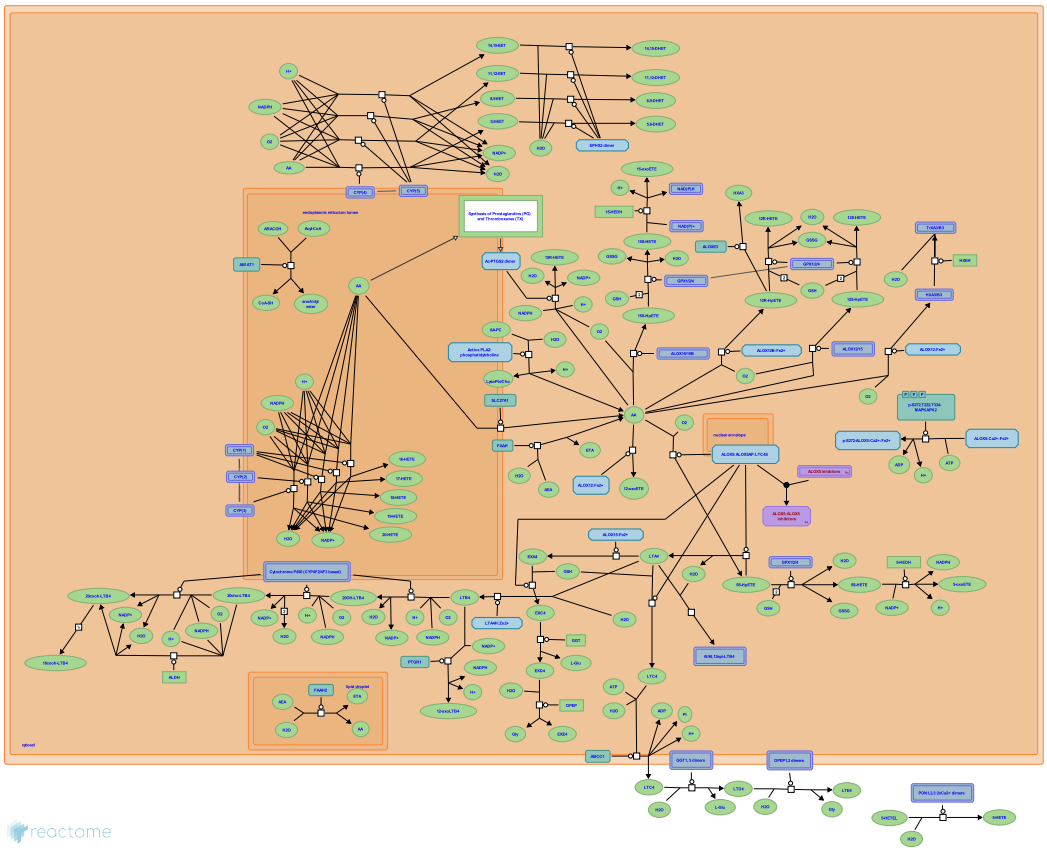
Arachidonate metabolism



D'Eustachio, P., Hill, DP., Jassal, B., Jupe, S., Le Novere, N., Rush, MG., Williams, MG.

European Bioinformatics Institute, New York University Langone Medical Center, Ontario Institute for Cancer Research, Oregon Health and Science University.

This is just an excerpt of a full-length report for this pathway. To access the complete report, please download it at the Reactome Textbook.

14/04/2025

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

Fabregat, A., Fabregat, A., Fabregat, A., Fabregat, A., Fabregat, A., Fabregat, A. et al. (2017). Reactome pathway analysis: a high-performance in-memory approach. *BMC bioinformatics, 18*, 142. ↗

Sidiropoulos, K., Sidiropoulos, K., Sidiropoulos, K., Sidiropoulos, K., Sidiropoulos, K., Sidiropoulos, K. et al. (2017). Reactome enhanced pathway visualization. *Bioinformatics, 33*, 3461-3467. ↗

Fabregat, A., Fabregat, A., Fabregat, A., Fabregat, A., Fabregat, A., Fabregat, A. et al. (2018). The Reactome Pathway Knowledgebase. *Nucleic Acids Res, 46*, D649-D655. ↗

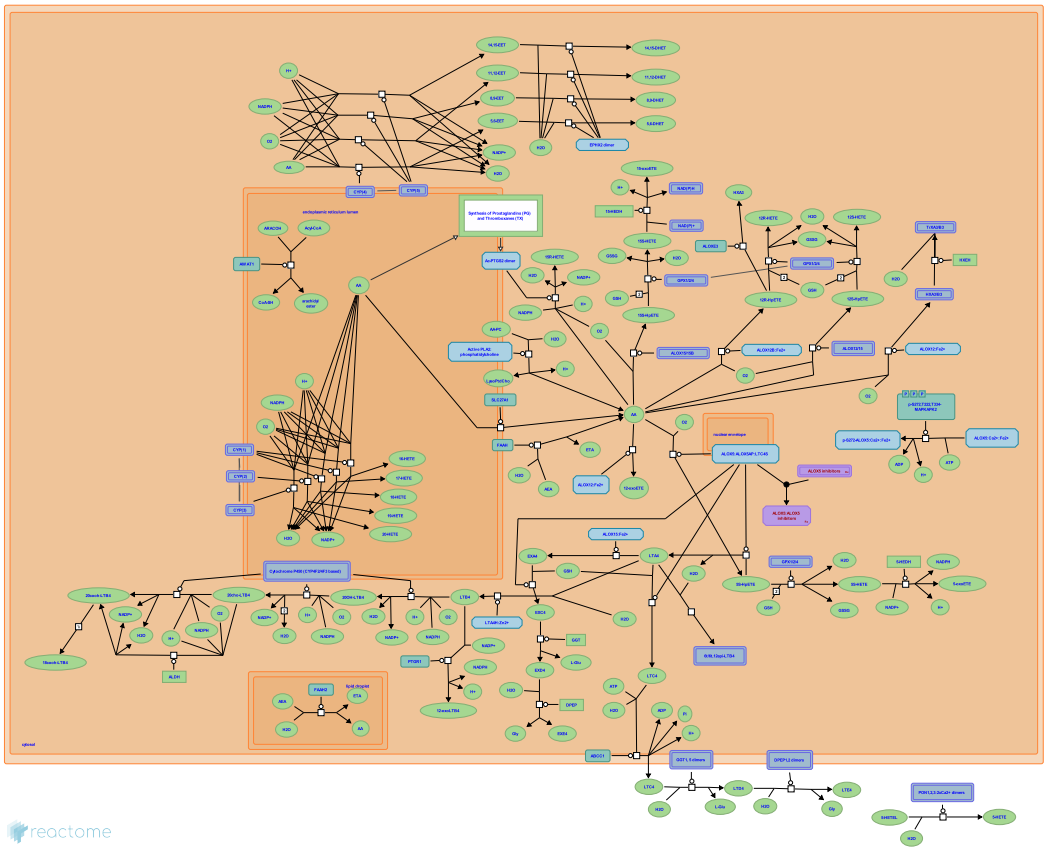
Fabregat, A., Fabregat, A., Fabregat, A., Fabregat, A., Fabregat, A., Fabregat, A. et al. (2018). Reactome graph database: Efficient access to complex pathway data. *PLoS computational biology, 14*, e1005968. ↗

Reactome database release: 92

This document contains 9 pathways and 5 reactions (see Table of Contents)

Arachidonate metabolism ↗

Stable identifier: R-HSA-2142753



Eicosanoids, oxygenated, 20-carbon fatty acids, are autocrine and paracrine signaling molecules that modulate physiological processes including pain, fever, inflammation, blood clot formation, smooth muscle contraction and relaxation, and the release of gastric acid. Eicosanoids are synthesized in humans primarily from arachidonate (all-cis 5,8,11,14-eicosatetraenoate) that is released from membrane phospholipids. Once released, arachidonate is acted on by prostaglandin G/H synthases (PTGS, also known as cyclooxygenases (COX)) to form prostaglandins and thromboxanes, by arachidonate lipoxygenases (ALOX) to form leukotrienes, epoxygenases (cytochrome P450s and epoxide hydrolase) to form epoxides such as 15-eicosatetraenoic acids, and omega-hydrolases (cytochrome P450s) to form hydroxyeicosatetraenoates (Buczynski et al. 2009, Vance & Vance 2008).
Levels of free arachidonate in the cell are normally very low so the rate of synthesis of eicosanoids is determined primarily by the activity of phospholipase A2, which mediates phospholipid cleavage to generate free arachidonate. The enzymes involved in arachidonate metabolism are typically constitutively expressed so the subset of these enzymes expressed by a cell determines the range of eicosanoids it can synthesize.
Eicosanoids are unstable, undergoing conversion to inactive forms with half-times under physiological conditions of seconds or minutes. Many of these reactions appear to be spontaneous.

Literature references

Dumlao, DS., Buczynski, MW., Dennis, EA. (2009). Thematic Review Series: Proteomics. An integrated omics analysis of eicosanoid biology. *J Lipid Res, 50*, 1015-38. ↗

Vance, JE., Vance, DE. (2008). The eicosanoids: cyclooxygenase, lipoxygenase, and epoxygenase pathways, Biochemistry of Lipids, Lipoproteins and Membranes, 5th Edition. *Elsevier Science*, 331-362.

Editions

2012-02-24	Authored, Edited	Williams, MG.
2012-11-10	Reviewed	Rush, MG.

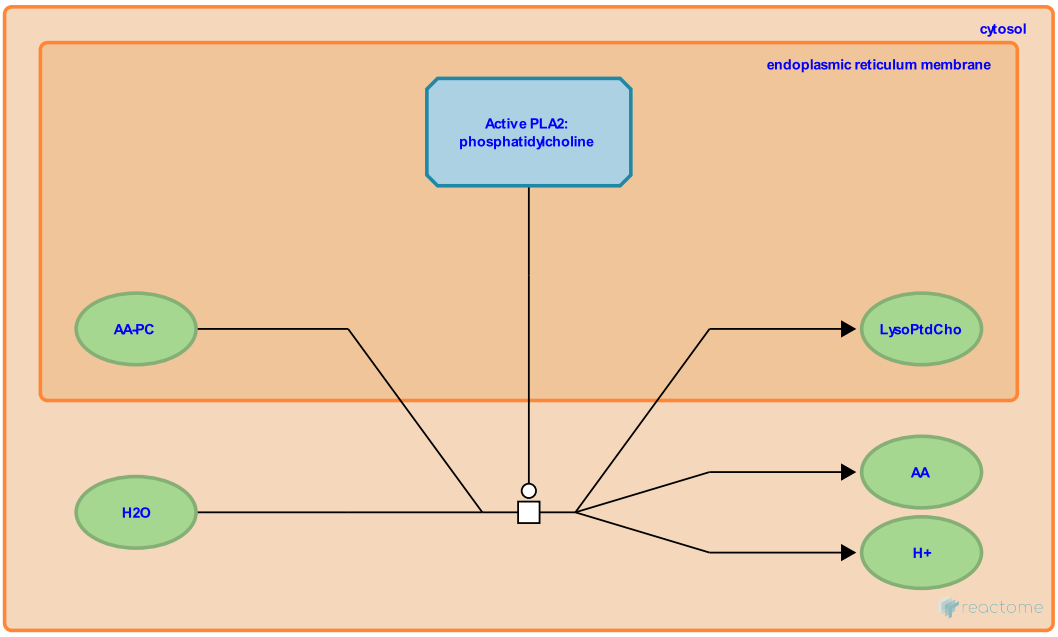
PLA2G4A (cPLA2) hydrolyzes phosphatidylcholine ↗

Location: Arachidonate metabolism

Stable identifier: R-HSA-111883

Type: transition

Compartments: cytosol, endoplasmic reticulum lumen, endoplasmic reticulum membrane



Once associated with the endoplasmic reticulum membrane, PLA2G4A (cPLA2) hydrolyzes phosphatidylcholine to produce arachidonate (AA), a precursor to inflammatory mediators. While several phospholipases can catalyze this reaction in cells overexpressing the enzymes, PLA2G4A is the major enzyme that catalyzes it in vivo (Reed et al. 2011). At the same time, possible physiological roles have been described for soluble phospholipases (sPLA) in the mobilization of arachidonate in some cell types or under some physiological conditions (Murakami et al. 2011).

Followed by: SLC27A1 transports arachidonate across the ER membrane

Literature references

Gelb, MH., Aloulou, A., Adler, D., Leslie, CC., Ghomashchi, F., Boutaud, O. et al. (2011). Functional characterization of mutations in inherited human cPLA? deficiency. *Biochemistry, 50*, 1731-8. ↗

Taketomi, Y., Yamamoto, K., Sato, H., Murakami, M. (2011). Secreted phospholipase A2 revisited. *J. Biochem., 150*, 233-55. ↗

Editions

2004-03-31	Authored	Jassal, B., Le Novere, N.
2008-11-06	Edited	Jassal, B.
2012-11-10	Reviewed	Rush, MG.
2024-08-02	Reviewed	Hill, DP.

SLC27A1 transports arachidonate across the ER membrane ↗

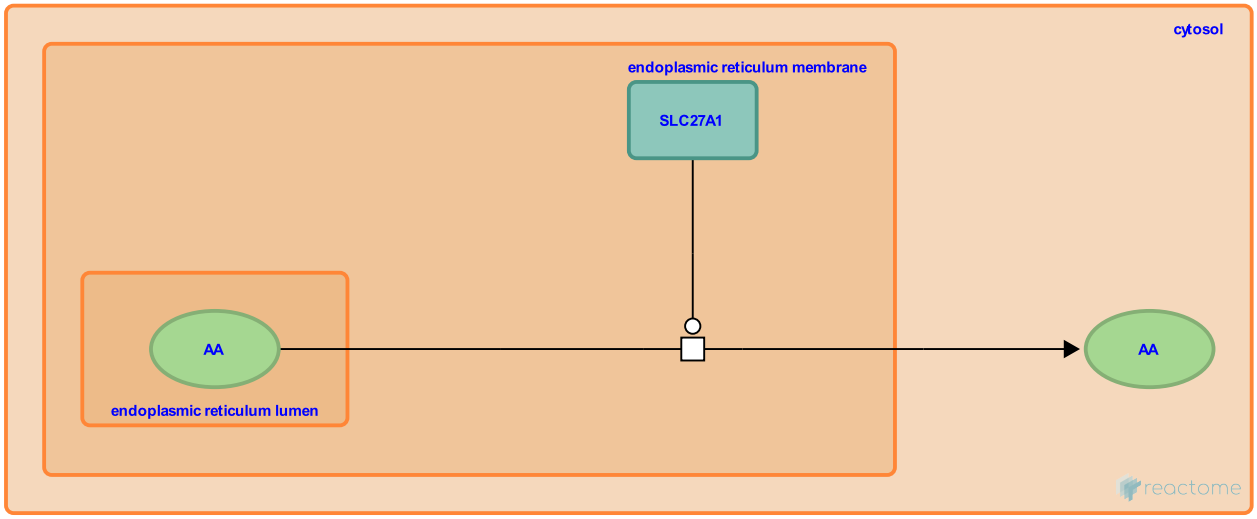
Location: Arachidonate metabolism

Stable identifier: R-HSA-428990

Type: transition

Compartments: endoplasmic reticulum membrane, endoplasmic reticulum lumen, cytosol

Inferred from: Slc27a1 transports arachidonate across the ER membrane (Mus musculus)



Cytosolic arachidonate (AA) released by phospholipases is transported across the ER membrane by SLC27A1, providing input for eicosanoid synthetic reactions in the endoplasmic reticulum lumen as well as the cytosol. Human SLC27A1 has not been characterized experimentally; its subcellular location (Garcia-Martinez et al. 2005) and transporter activity (Shaffer and Lodish 1994) are inferred from the properties of the homologous mouse protein.

Preceded by: PLA2G4A (cPLA2) hydrolyzes phosphatidylcholine

Literature references

Irvine, RF. (1982). How is the level of free arachidonic acid controlled in mammalian cells?. *Biochem J, 204*, 3-16. ↗

Guitart, M., Montell, E., Busquets, S., Moore-Carrasco, R., Marotta, M., Gómez-Foix, AM. et al. (2005). Impact on fatty acid metabolism and differential localization of FATP1 and FAT/CD36 proteins delivered in cultured human muscle cells. *Am J Physiol Cell Physiol, 288*, C1264-72. ↗

Lodish, HF., Schaffer, JE. (1994). Expression cloning and characterization of a novel adipocyte long chain fatty acid transport protein. *Cell, 79*, 427-36. ↗

Editions

2009-07-14	Authored, Edited	Jupe, S.
2012-11-10	Reviewed	Rush, MG.
2024-08-02	Reviewed	Hill, DP.

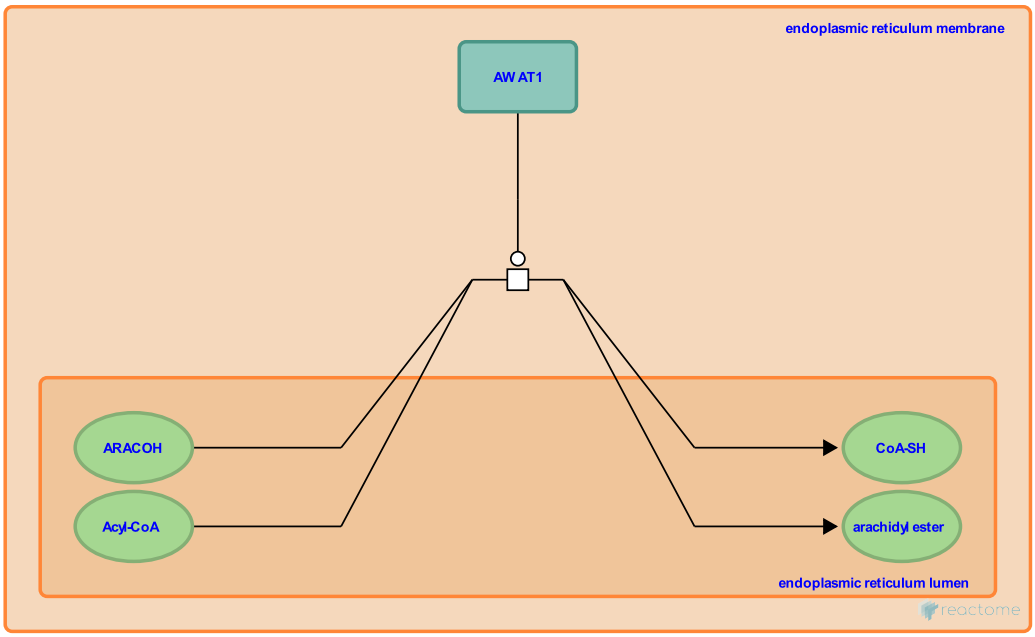
AWAT1 transfers acyl group from acyl-CoA to ARACOH, forming wax esters ↗

Location: Arachidonate metabolism

Stable identifier: R-HSA-5696424

Type: transition

Compartments: endoplasmic reticulum membrane, endoplasmic reticulum lumen



Arachidyl alcohol (ARACOH) is straight-chain fatty alcohol of C20 length used as an emollient in cosmetics. Esterification of alcohols with fatty acids represents the formation of both storage and cytoprotective molecules in the body. Overproduction of these esters is associated with several disease pathologies, including atherosclerosis and obesity. The ER membrane-associated acyl-CoA wax alcohol acyltransferase 1 (AWAT1) mediates the esterification of its preferred substrate ARACOH (Turkish et al. 2005).

Literature references

Bazzi, H., Billheimer, JT., Cromley, D., Turkish, AR., Sturley, SL., Oelkers, P. et al. (2005). Identification of two novel human acyl-CoA wax alcohol acyltransferases: members of the diacylglycerol acyltransferase 2 (DGAT2) gene superfamily. *J. Biol. Chem., 280*, 14755-64. ↗

Editions

2015-05-29	Authored, Edited	Jassal, B.
2015-06-26	Reviewed	D'Eustachio, P.

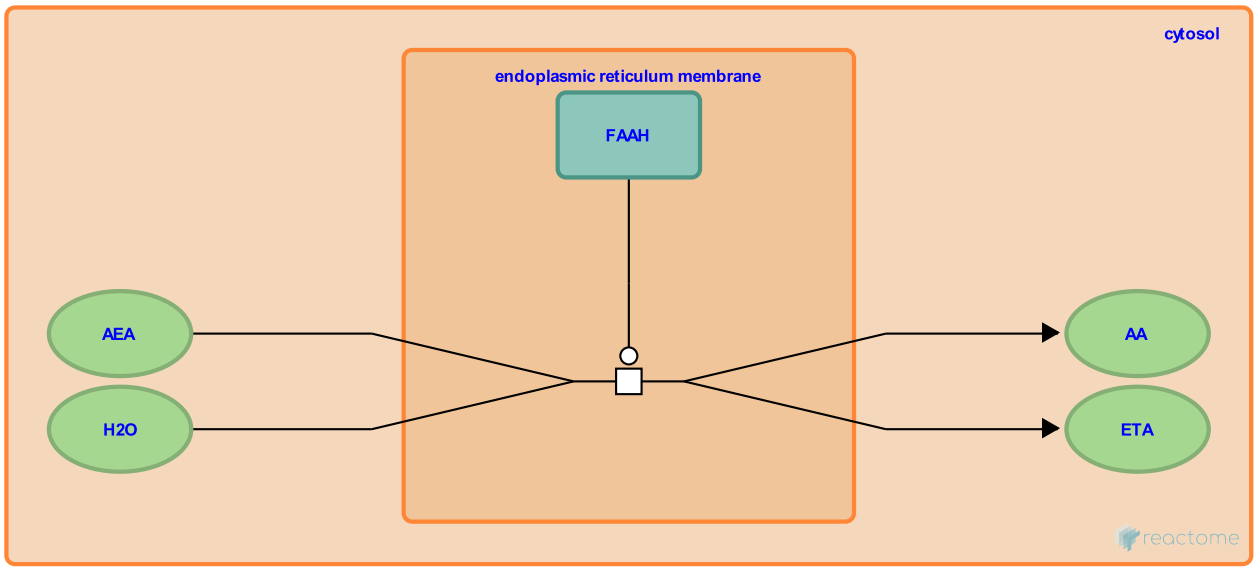
FAAH hydrolyses AEA to AA and ETA ↗

Location: Arachidonate metabolism

Stable identifier: R-HSA-5693742

Type: transition

Compartments: endoplasmic reticulum membrane, cytosol



Fatty acid amides are a class of lipid transmitters that include the endogenous cannabinoid anandamide (AEA) and the sleep-inducing chemical oleamide. The magnitude and duration of their signalling are controlled by enzymatic hydrolysis mediated by fatty-acid amide hydrolases 1 and 2 (FAAH, H2). Hydrolysis of AEA is described here (Wei et al. 2006). FAAH is localised to the ER membrane whereas FAAH2 is localised to lipid droplets (Kaczocha et al. 2010).

Literature references

Cravatt, BF., Wei, BQ., Lander, ES., McKinney, MK., Mikkelsen, TS. (2006). A second fatty acid amide hydrolase with variable distribution among placental mammals. *J. Biol. Chem., 281,* 36569-78. ↗

Deutsch, DG., Glaser, ST., Brown, DA., Chae, J., Kaczocha, M. (2010). Lipid droplets are novel sites of N-acylethanolamine inactivation by fatty acid amide hydrolase-2. *J. Biol. Chem., 285,* 2796-806. ↗

Editions

2015-05-18	Authored, Edited	Jassal, B.
2015-06-26	Reviewed	D'Eustachio, P.

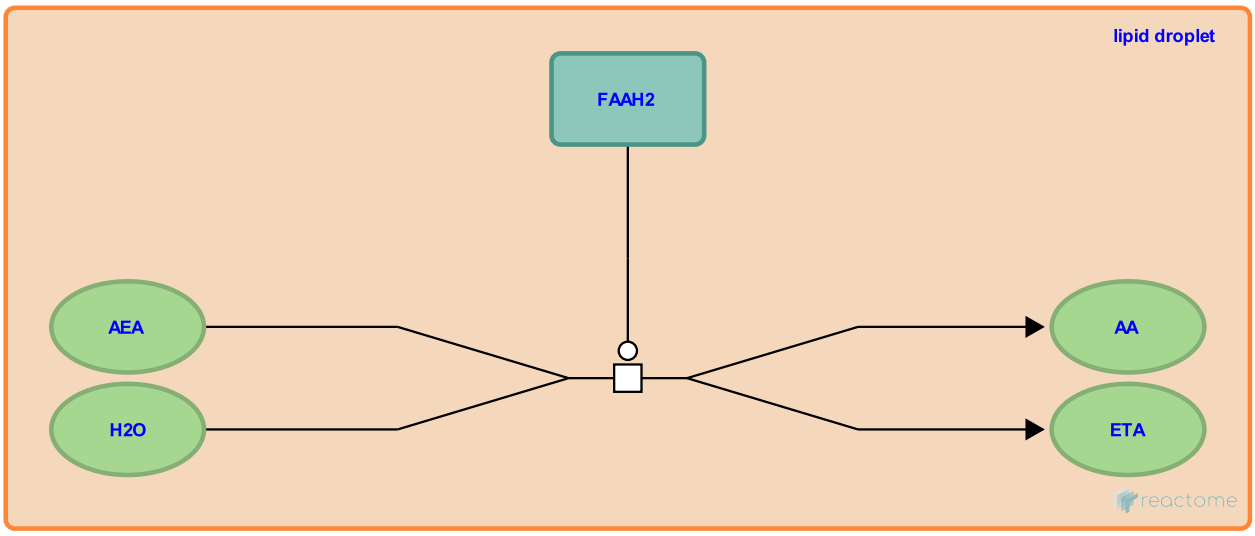
FAAH2 hydrolyses AEA to AA and ETA ↗

Location: Arachidonate metabolism

Stable identifier: R-HSA-5693751

Type: transition

Compartments: lipid droplet



Fatty acid amides are a class of lipid transmitters that include the endogenous cannabinoid anandamide (AEA) and the sleep-inducing chemical oleamide. The magnitude and duration of their signalling are controlled by enzymatic hydrolysis mediated by fatty-acid amide hydrolases 1 and 2 (FAAH, H2). Hydrolysis of AEA is described here (Wei et al. 2006). FAAH is localised to the ER membrane whereas FAAH2 is localised to lipid droplets (Kaczocha et al. 2010).

Literature references

Cravatt, BF., Wei, BQ., Lander, ES., McKinney, MK., Mikkelsen, TS. (2006). A second fatty acid amide hydrolase with variable distribution among placental mammals. *J. Biol. Chem., 281*, 36569-78. ↗

Deutsch, DG., Glaser, ST., Brown, DA., Chae, J., Kaczocha, M. (2010). Lipid droplets are novel sites of N-acylethanolamine inactivation by fatty acid amide hydrolase-2. *J. Biol. Chem., 285*, 2796-806. ↗

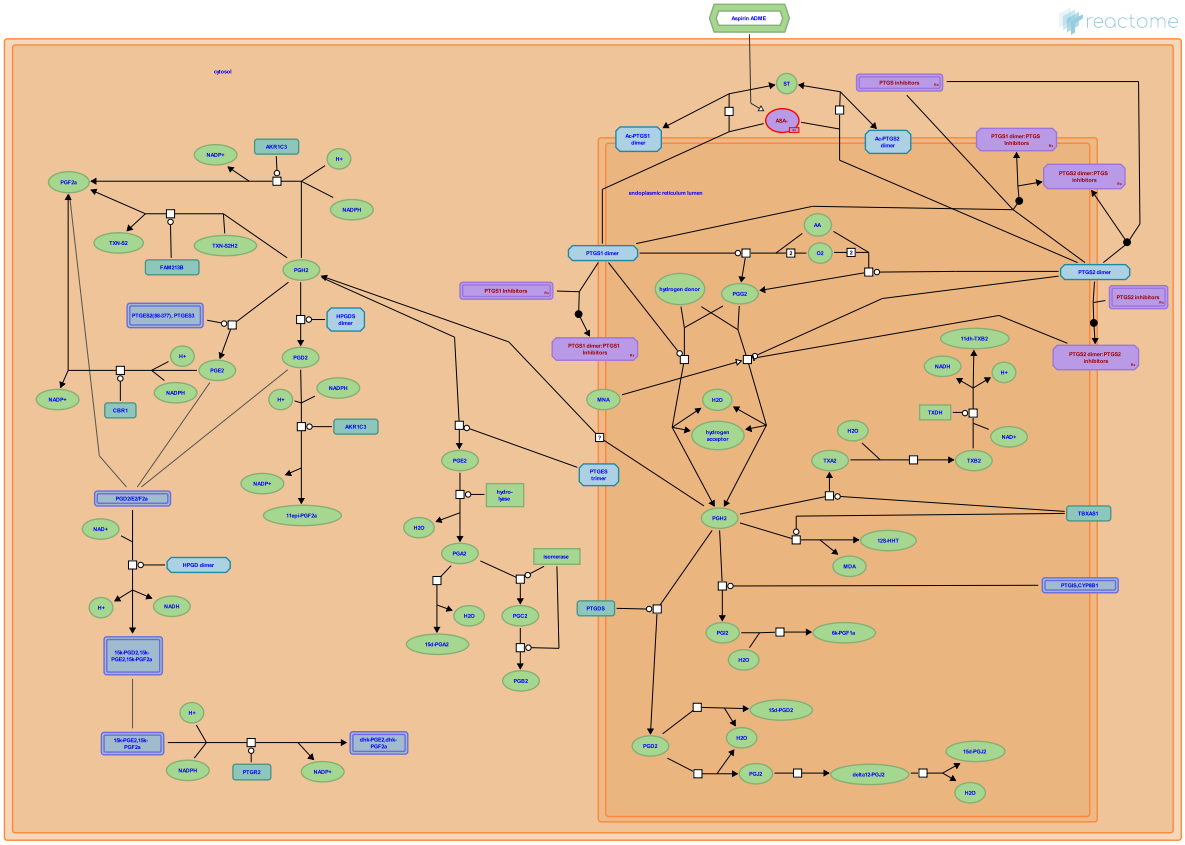
Editions

2015-05-18	Authored, Edited	Jassal, B.
2015-06-26	Reviewed	D'Eustachio, P.

Synthesis of Prostaglandins (PG) and Thromboxanes (TX) ↗

Location: Arachidonate metabolism

Stable identifier: R-HSA-2162123



The bioactive prostaglandin (PG) signalling molecules, including PGA2, PGE2, PGF2a, and PGI2 (prostacyclin) are synthesised from arachidonate and its products by various prostaglandin synthase type enzymes. Prostaglandin H2 (PGH2) is the starting point for the synthesis of Thromboxanes (TXs) (Buczynski et al. 2009, Vance & Vance 2008). PGs and TXs are collectively known as the prostanoids.
Two enzymes, PTGS1 and 2 (COX1 and 2) both catalyze the two-step conversion of arachidonate to PGH2. PTGS1 is constitutively expressed in many cell types while PTGS2 is induced in response to stress and mediates the syntheses of prostaglandins associated with pain, fever, and inflammation. Aspirin irreversibly inactivates both enzymes (though it acts more efficiently on PTGS1), explaining both its antiinflammatory effects and side effects like perturbed gastic acid secretion. Drugs like celecoxib, by specifically inhibiting PTGS2, have a strong anti-inflammatory effect with fewer side effects. These PTGS2-specific drugs, however, probably because of their effects on the balance of prostaglandin synthesis in platelets and endothelial cells, can also promote blood clot formation (Buczynski et al. 2009; Stables & Gilroy 2011).

Literature references

Stables, MJ., Gilroy, DW. (2011). Old and new generation lipid mediators in acute inflammation and resolution. *Prog. Lipid Res., 50*, 35-51. ↗

Vance, JE., Vance, DE. (2008). The eicosanoids: cyclooxygenase, lipoxygenase, and epoxygenase pathways, Biochemistry of Lipids, Lipoproteins and Membranes, 5th Edition. *Elsevier Science*, 331-362.

Dumlao, DS., Buczynski, MW., Dennis, EA. (2009). Thematic Review Series: Proteomics. An integrated omics analysis of eicosanoid biology. *J Lipid Res, 50*, 1015-38. ↗

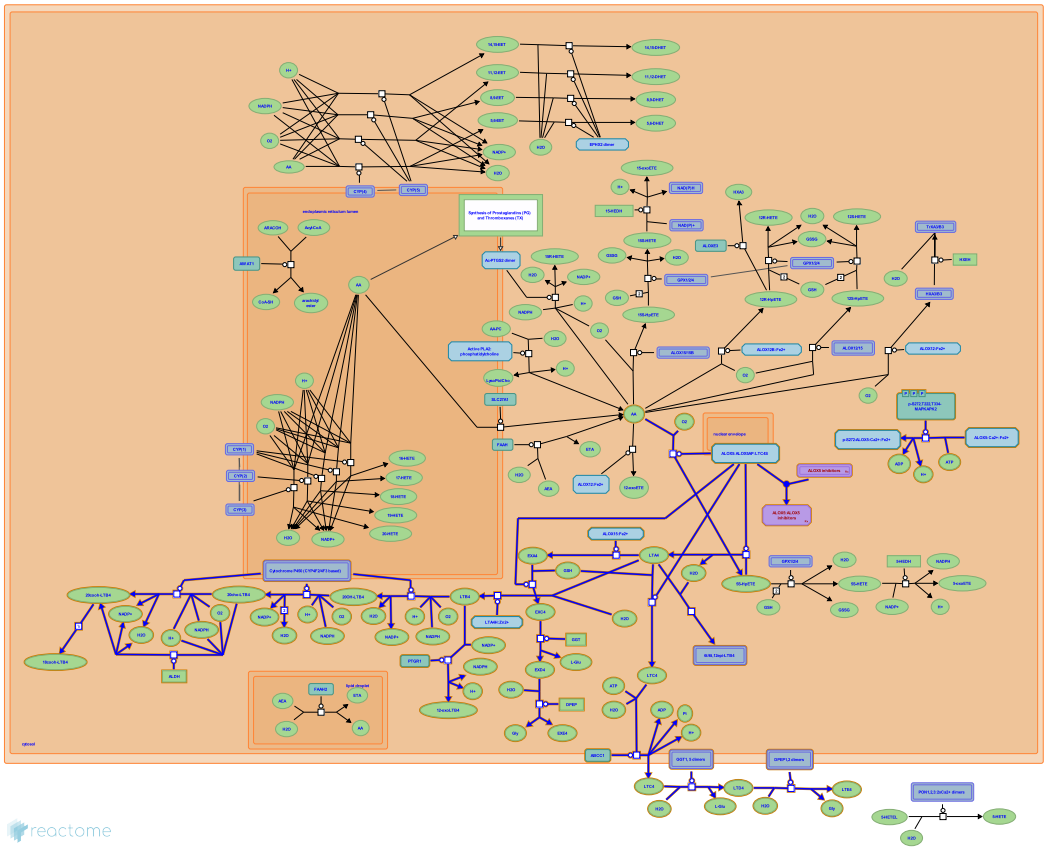
Editions

2012-02-24	Authored, Edited	Williams, MG.
2012-11-10	Reviewed	Rush, MG.

Synthesis of Leukotrienes (LT) and Eoxins (EX) ↗

Location: Arachidonate metabolism

Stable identifier: R-HSA-2142691



Leukotrienes (LTs) are biologically active molecules formed in response to inflammatory stimuli. They cause contraction of bronchial smooth muscles, stimulation of vascular permeability, and attraction and activation of leukocytes. LTs were discovered in 1938 and were termed the "slow release substance" (SRS) until their structures were determined in 1979 and they were then renamed to leukotrienes. LTs are derived from arachidonate through action by arachidonate 5-lipoxygenase (ALOX5). Cysteinyl leukotrienes (LTC4, LTD4, and LTE4) are generated as products derived from leukotriene A4 (LTA4). Eoxins are generated from leukotrienes (LTs) and resemble cysteinyl leukotrienes but have a different three-dimensional structure (Murphy & Gijon 2007, Hammarstrom 1983, MA.Claesson 2009, Vance & Vance 2008, Buczynski et al. 2009).

Literature references

Dumlao, DS., Buczynski, MW., Dennis, EA. (2009). Thematic Review Series: Proteomics. An integrated omics analysis of eicosanoid biology. *J Lipid Res, 50*, 1015-38. ↗

Vance, JE., Vance, DE. (2008). The eicosanoids: cyclooxygenase, lipoxygenase, and epoxygenase pathways, Biochemistry of Lipids, Lipoproteins and Membranes, 5th Edition. *Elsevier Science*, 331-362.

Claesson, HE. (2009). On the biosynthesis and biological role of eoxins and 15-lipoxygenase-1 in airway inflammation and Hodgkin lymphoma. *Prostaglandins Other Lipid Mediat, 89*, 120-5. ↗

Gijon, MA., Murphy, RC. (2007). Biosynthesis and metabolism of leukotrienes. *Biochem J, 405*, 379-95. ↗

Hammarström, S. (1983). Leukotrienes. *Annu Rev Biochem, 52*, 355-77. ↗

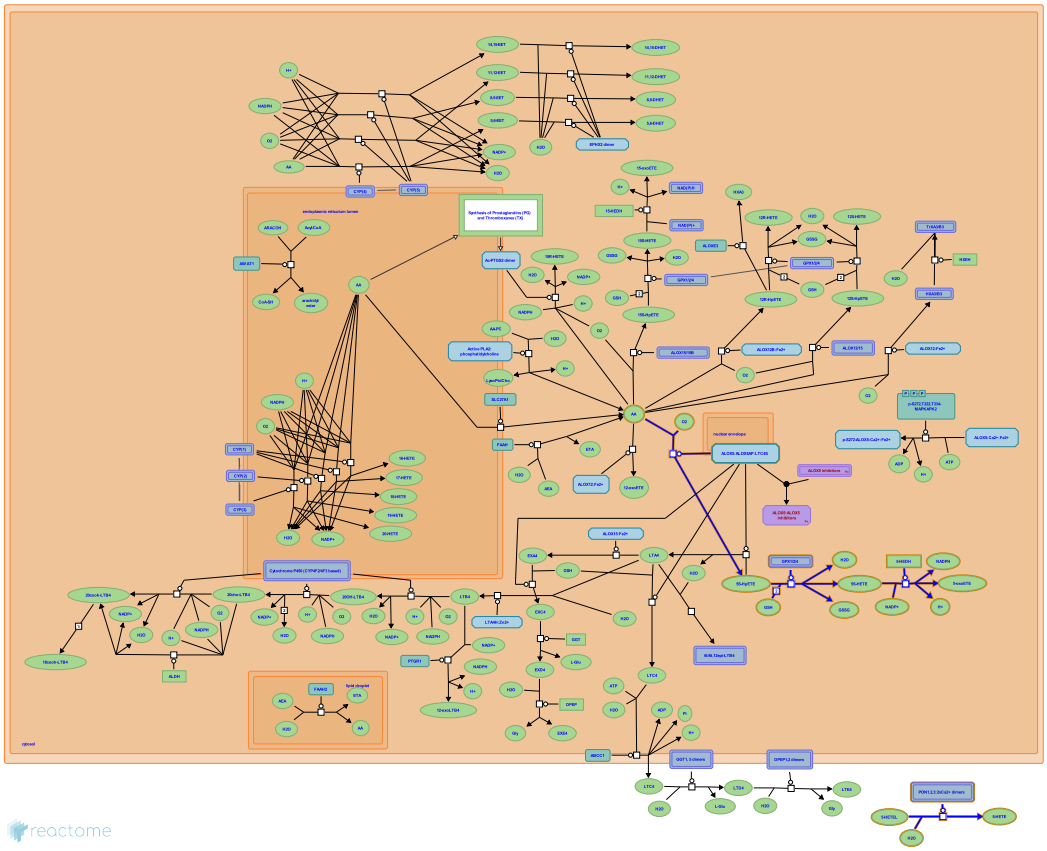
Editions

2012-02-24	Authored, Edited	Williams, MG.
2012-11-10	Reviewed	Rush, MG.
2024-08-02	Reviewed	Hill, DP.

Synthesis of 5-eicosatetraenoic acids ↗

Location: Arachidonate metabolism

Stable identifier: R-HSA-2142688



5-hydroperoxy-eicosatetraenoic acid (5-HpETE), 5-hydroxyeicosatetraenoic acid (5S-HETE) and 5-oxo-eicosatetraenoic acid (5-oxoETE) are formed after the initial step of Arachidonate oxidation by arachidonate 5-lipoxygenase (ALOX5) (Buczynski et al. 2009, Vance & Vance 2008).

Literature references

Vance, JE., Vance, DE. (2008). The eicosanoids: cyclooxygenase, lipoxygenase, and epoxygenase pathways, Biochemistry of Lipids, Lipoproteins and Membranes, 5th Edition. *Elsevier Science,* 331-362.

Dumlao, DS., Buczynski, MW., Dennis, EA. (2009). Thematic Review Series: Proteomics. An integrated omics analysis of eicosanoid biology. *J Lipid Res, 50,* 1015-38. ↗

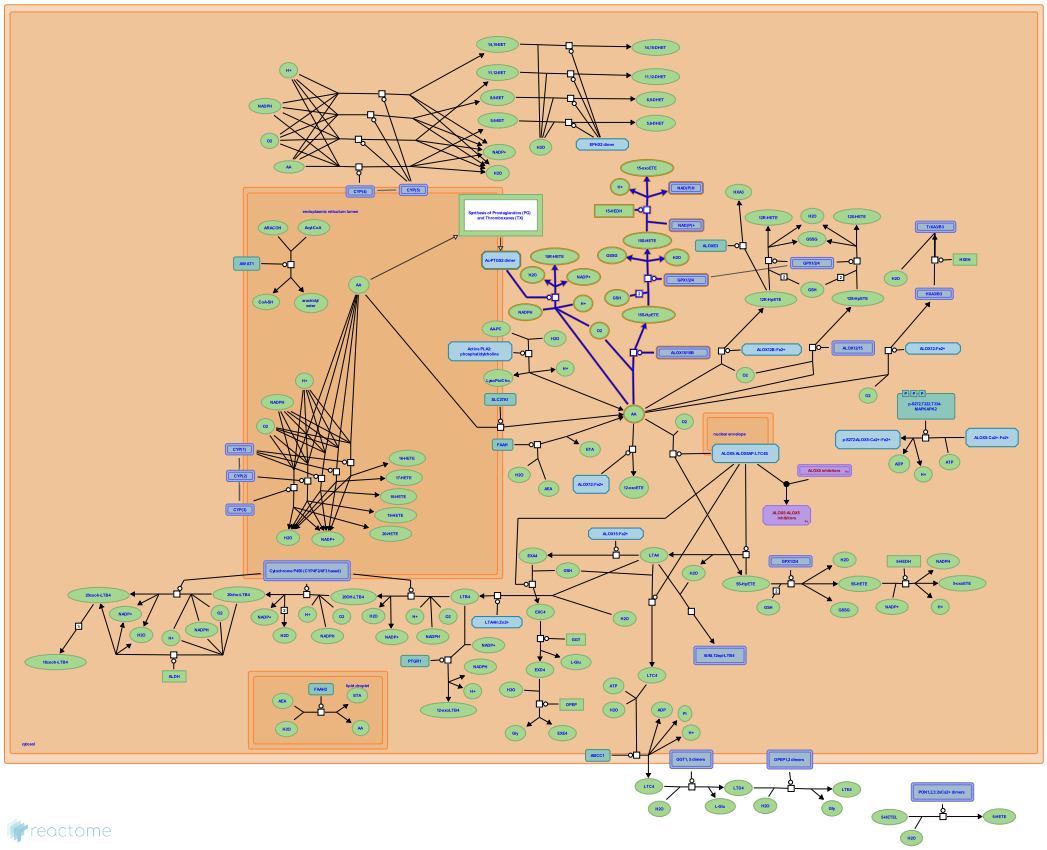
Editions

2012-02-24	Authored, Edited	Williams, MG.
2012-11-10	Reviewed	Rush, MG.

Synthesis of 15-eicosatetraenoic acid derivatives ↗

Location: Arachidonate metabolism

Stable identifier: R-HSA-2142770



The 15-eicosatetraenoates: 15-hydroperoxy-eicosatetraenoate (15-HpETE), 15-hydroxyeicosatetraenoate (15-HETE) and 15-oxo-eicosatetraenoate (15-oxoETE) are formed after the initial step of arachidonate oxidation by the arachidonate 15-lipoxygenases (ALOX15 and ALOX15B) (Buczynski et al. 2009, Vance & Vance 2008).

Literature references

Vance, JE., Vance, DE. (2008). The eicosanoids: cyclooxygenase, lipoxygenase, and epoxygenase pathways, Biochemistry of Lipids, Lipoproteins and Membranes, 5th Edition. *Elsevier Science*, 331-362.

Dumlao, DS., Buczynski, MW., Dennis, EA. (2009). Thematic Review Series: Proteomics. An integrated omics analysis of eicosanoid biology. *J Lipid Res, 50*, 1015-38. ↗

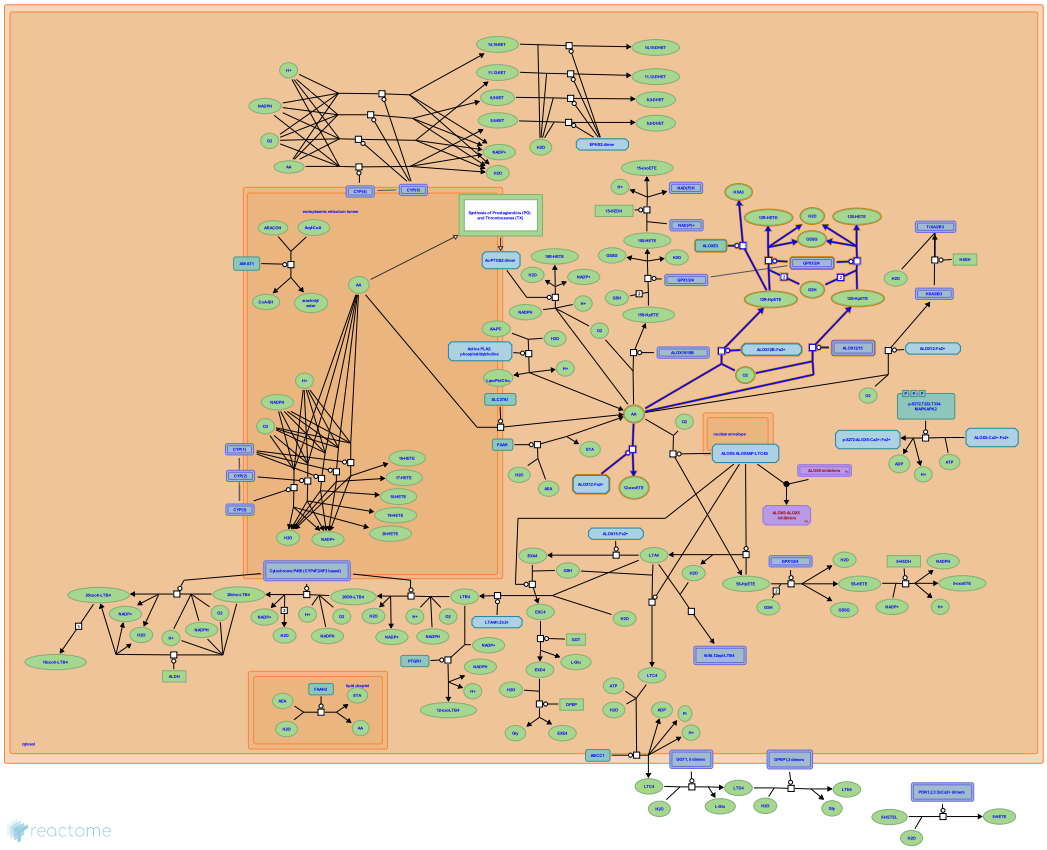
Editions

2012-02-24	Authored, Edited	Williams, MG.
2012-11-10	Reviewed	Rush, MG.

Synthesis of 12-eicosatetraenoic acid derivatives ↗

Location: Arachidonate metabolism

Stable identifier: R-HSA-2142712



The 12-eicosatetraenoic acids: 12-hydroperoxy-eicosatetraenoate (12-HpETE), 12-hydroxyeicosatetraenoate (12-HETE) and 12-oxo-eicosatetraenoate (12-oxoETE) are formed after the initial step of arachidonate oxidation by the arachidonate 12 and 15 lipoxygenases (ALOX12, ALOX12B and ALOX15 respectively). This part of the pathway is bifurcated at the level of 12S-hydroperoxy-eicosatetraenoate (12S-HpETE), which can either be reduced to 12S-hydro-eicosatetraenoate (12S-HETE) or converted to hepoxilins (Buczynski et al. 2009, Vance & Vance 2008).

Literature references

Vance, JE., Vance, DE. (2008). The eicosanoids: cyclooxygenase, lipoxygenase, and epoxygenase pathways, Biochemistry of Lipids, Lipoproteins and Membranes, 5th Edition. *Elsevier Science,* 331-362.

Dumlao, DS., Buczynski, MW., Dennis, EA. (2009). Thematic Review Series: Proteomics. An integrated omics analysis of eicosanoid biology. *J Lipid Res, 50*, 1015-38. ↗

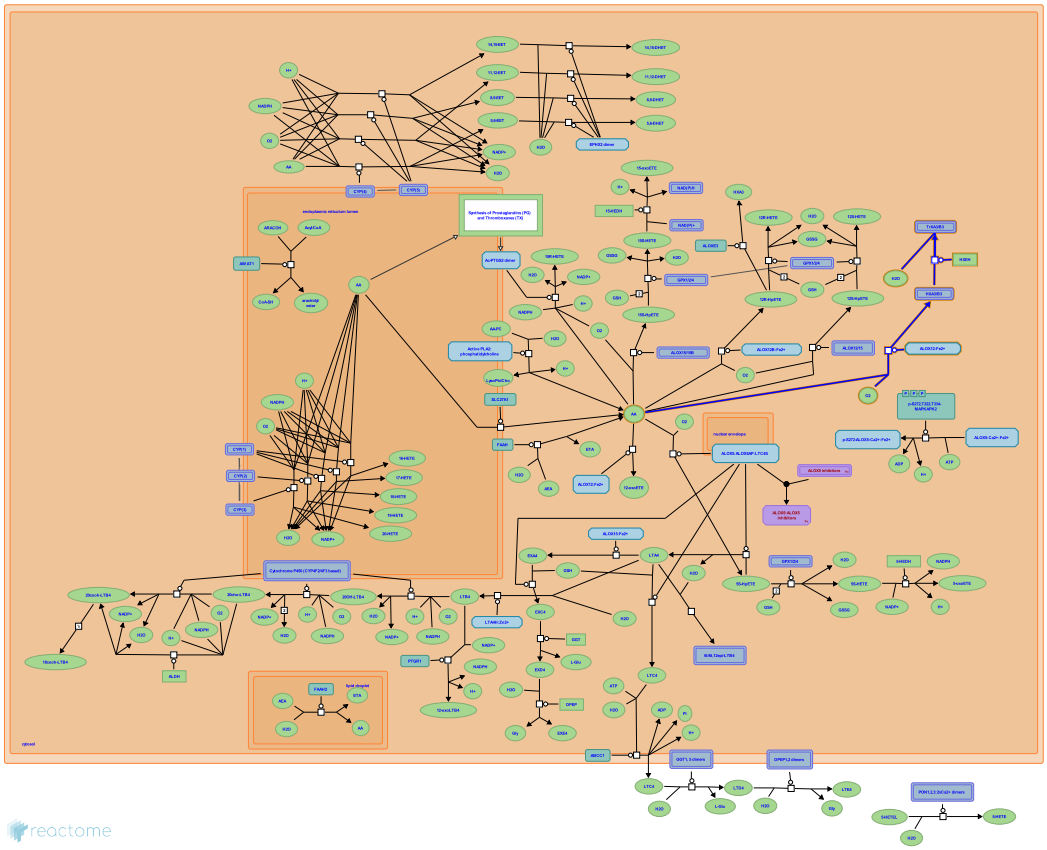
Editions

2012-02-24	Authored, Edited	Williams, MG.
2012-11-10	Reviewed	Rush, MG.

Synthesis of Hepoxilins (HX) and Trioxilins (TrX) ↗

Location: Arachidonate metabolism

Stable identifier: R-HSA-2142696



Hepoxilins are biologically relevant signalling molecules produced by certain arachidonate 12-lipoxygenase (ALOX12s). Hepoxilin A3 (HXA3) and B3 (HXB3) have been identified, both of which incorporate an epoxide across the C-11 and C-12 double bond, as well as an additional hydroxyl moiety. HXA3 has a C-8 hydroxyl, whereas the HXB3 hydroxyl occurs at C-10. The epoxy moiety is labile and can be hydrolyzed either by a hepoxilin specific epoxide hydrolase (HXEH) or in acidic aqueous solution to form the corresponding diol metabolites trioxilin A3 (TrXA3) and B3 (TrXB3) (Buczynski et al. 2009, Vance & Vance 2008).

Literature references

Vance, JE., Vance, DE. (2008). The eicosanoids: cyclooxygenase, lipoxygenase, and epoxygenase pathways, Biochemistry of Lipids, Lipoproteins and Membranes, 5th Edition. *Elsevier Science*, 331-362.

Dumlao, DS., Buczynski, MW., Dennis, EA. (2009). Thematic Review Series: Proteomics. An integrated omics analysis of eicosanoid biology. *J Lipid Res, 50*, 1015-38. ↗

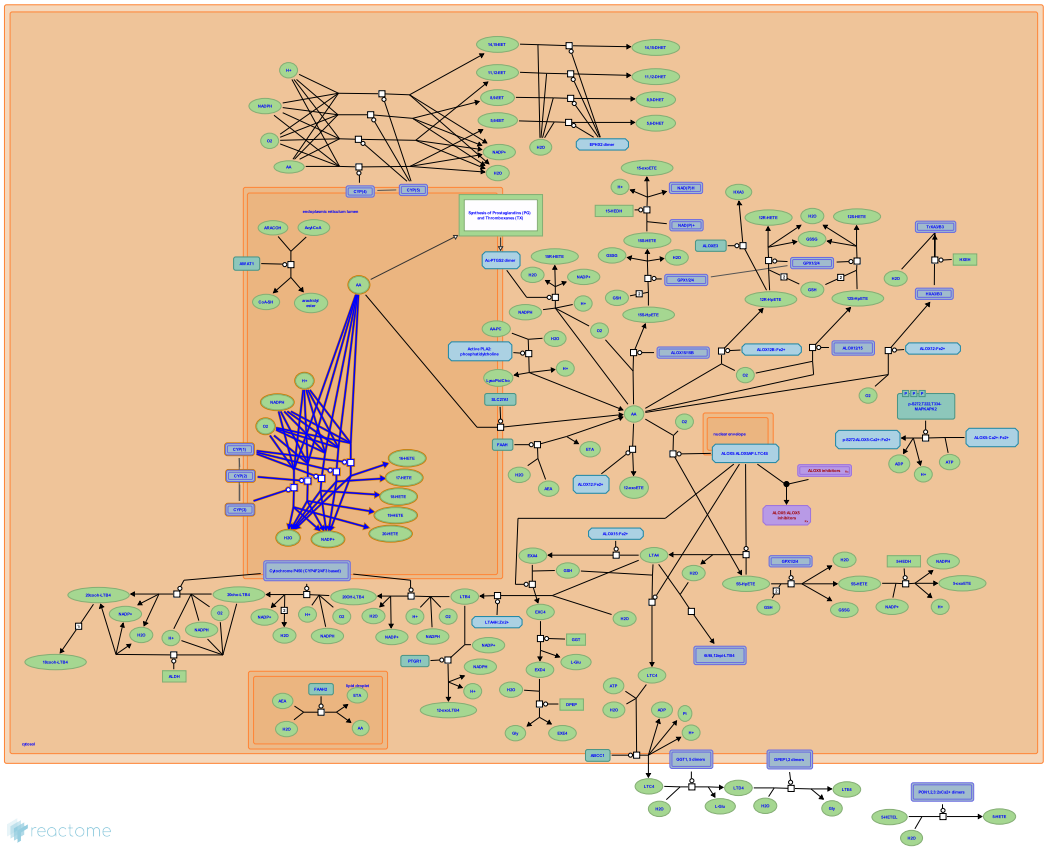
Editions

2012-02-24	Authored, Edited	Williams, MG.
2012-11-10	Reviewed	Rush, MG.

Synthesis of (16-20)-hydroxyeicosatetraenoic acids (HETE) ↗

Location: Arachidonate metabolism

Stable identifier: R-HSA-2142816



Similar to the lipoxygenases, cytochrome P450 (CYP) enzymes catalyse the hydroxylation and epoxygenation of arachidonate. However, whereas lipoxygenases use an active non-heme iron to abstract hydrogen directly from arachidonate, CYPs contain a heme-iron active site that oxidizes its substrate by a different mechanism. They hydroxylate arachidonate between C-5 and C-15 to produce lipoxygenase-like hydroxyeicosatetraenoates (HETEs) and add a hydroxyl moiety to the sp3-hybridized omega-carbons to form a unique class of HETEs. The transfer of oxygen to the unstable arachidonate intermediate terminates the reaction by forming HETE or epoxy-eicosatrienoate (EETs), respectively (Capdevila et al. 2000, Buczynski et al. 2009, Vance & Vance 2008).

Literature references

Falck, JR., Capdevila, JH., Harris, RC. (2000). Cytochrome P450 and arachidonic acid bioactivation. Molecular and functional properties of the arachidonate monooxygenase. *J Lipid Res, 41*, 163-81. ↗

Vance, JE., Vance, DE. (2008). The eicosanoids: cyclooxygenase, lipoxygenase, and epoxygenase pathways, Biochemistry of Lipids, Lipoproteins and Membranes, 5th Edition. *Elsevier Science*, 331-362.

Dumlao, DS., Buczynski, MW., Dennis, EA. (2009). Thematic Review Series: Proteomics. An integrated omics analysis of eicosanoid biology. *J Lipid Res, 50*, 1015-38. ↗

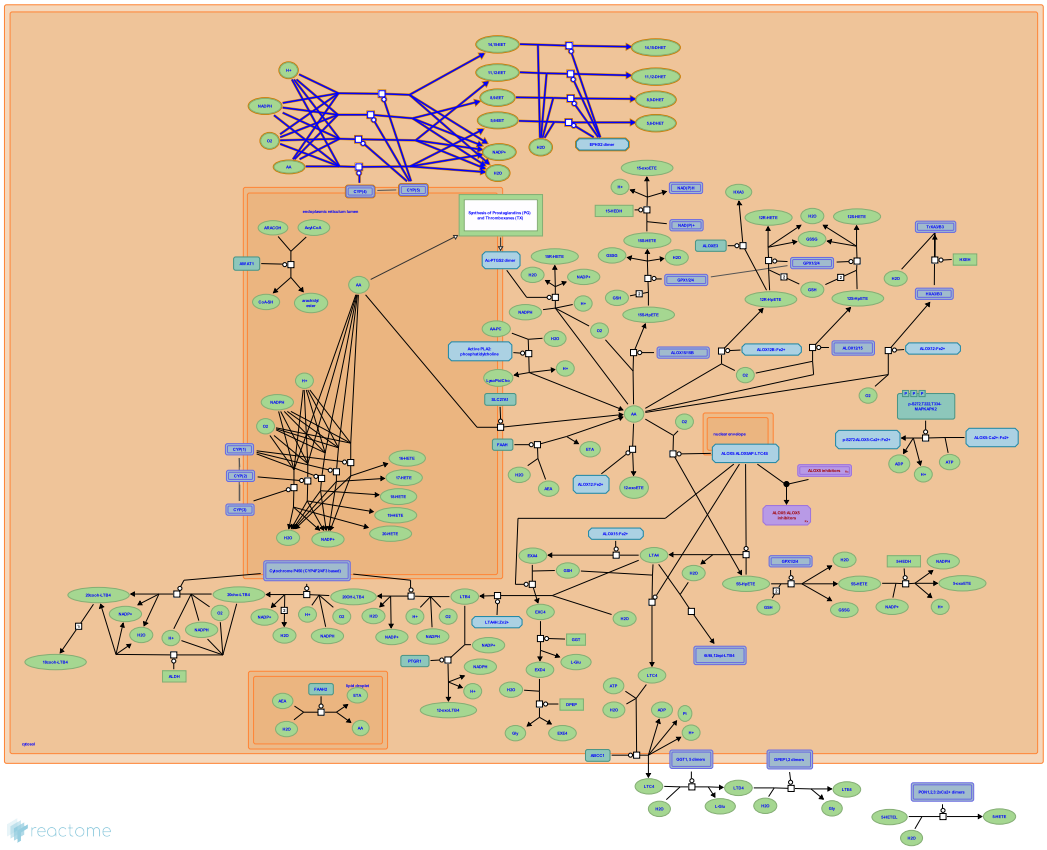
Editions

2012-02-24	Authored, Edited	Williams, MG.
2012-11-10	Reviewed	Rush, MG.
2024-08-02	Reviewed	Hill, DP.

Synthesis of epoxy (EET) and dihydroxyeicosatrienoic acids (DHET)

Location: Arachidonate metabolism

Stable identifier: R-HSA-2142670



The epoxidation of arachidonate by cytochrome P450s (CYPs) results in the formation of unique bioactive lipid mediators termed epoxyeicosatrienoates (EETs). Each double bond has been shown to be susceptible to oxidation, resulting in 5,6-EET, 8,9-EET, 11,12-EET, and 14,15-EET. The majority of the EET biological activities are diminished by the hydrolysis to the corresponding dihydroxyeicosatrienoates (DHET) (Capdevila et al. 2000, Buczynski et al. 2009, Vance & Vance 2008).

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