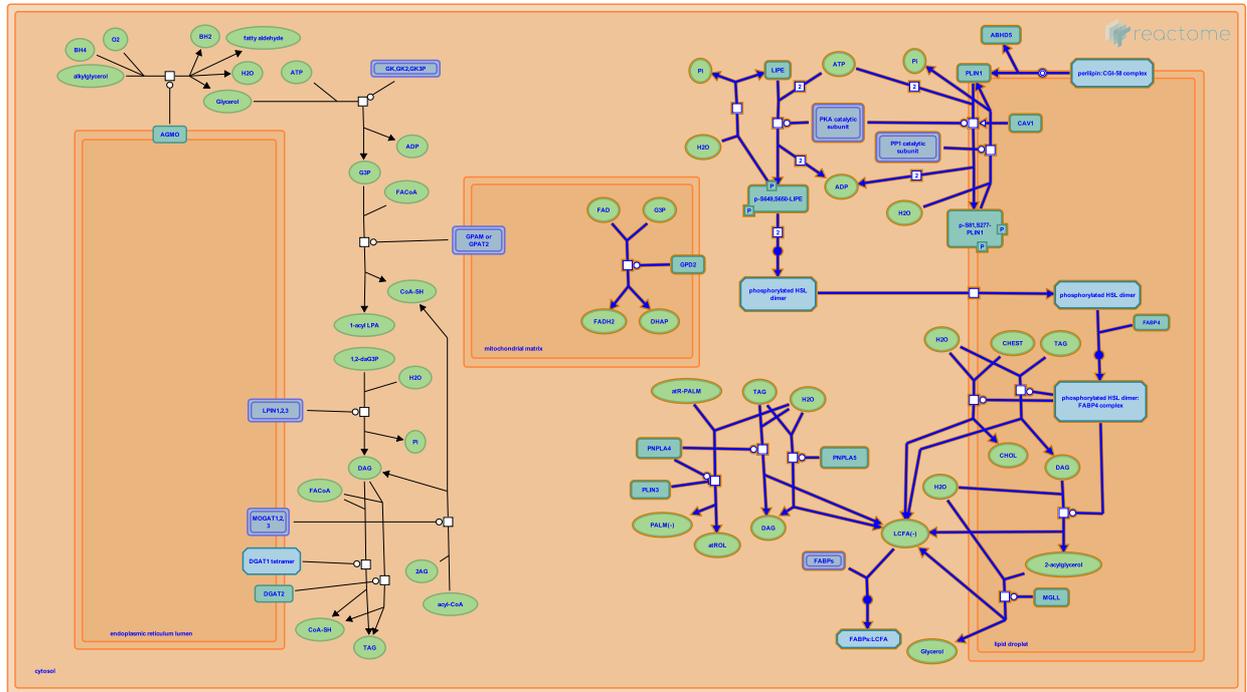


Triglyceride catabolism



D'Eustachio, P., Gillespie, ME., Gopinathrao, G., Jassal, B.

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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

- Fabregat, A., Sidiropoulos, K., Viteri, G., Forner, O., Marin-Garcia, P., Arnau, V. et al. (2017). Reactome pathway analysis: a high-performance in-memory approach. *BMC bioinformatics*, 18, 142. [↗](#)
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- Fabregat, A., Korninger, F., Viteri, G., Sidiropoulos, K., Marin-Garcia, P., Ping, P. et al. (2018). Reactome graph database: Efficient access to complex pathway data. *PLoS computational biology*, 14, e1005968. [↗](#)

Reactome database release: 70

This document contains 1 pathway and 17 reactions ([see Table of Contents](#))

cells and tissues. Most experimental data are derived instead from two rodent model systems: primary adipocytes from rats, and mouse 3T3-L1 cells induced to differentiate into adipocytes.

Literature references

Kraemer, FB., Shen, WJ. (2002). Hormone-sensitive lipase: control of intracellular tri-(di-)acylglycerol and cholesteryl ester hydrolysis. *J Lipid Res*, 43, 1585-94. [↗](#)

Holm, C., Osterlund, T., Laurell, H., Contreras, JA. (2000). Molecular mechanisms regulating hormone-sensitive lipase and lipolysis. *Annu Rev Nutr*, 20, 365-93. [↗](#)

Editions

2005-05-02	Authored	D'Eustachio, P.
2007-02-02	Reviewed	Jassal, B., Gillespie, ME., Gopinathrao, G., D'Eustachio, P.
2019-08-16	Edited	D'Eustachio, P.

hormone-sensitive lipase (HSL) + 2 ATP -> phosphorylated HSL + 2 ADP ↗

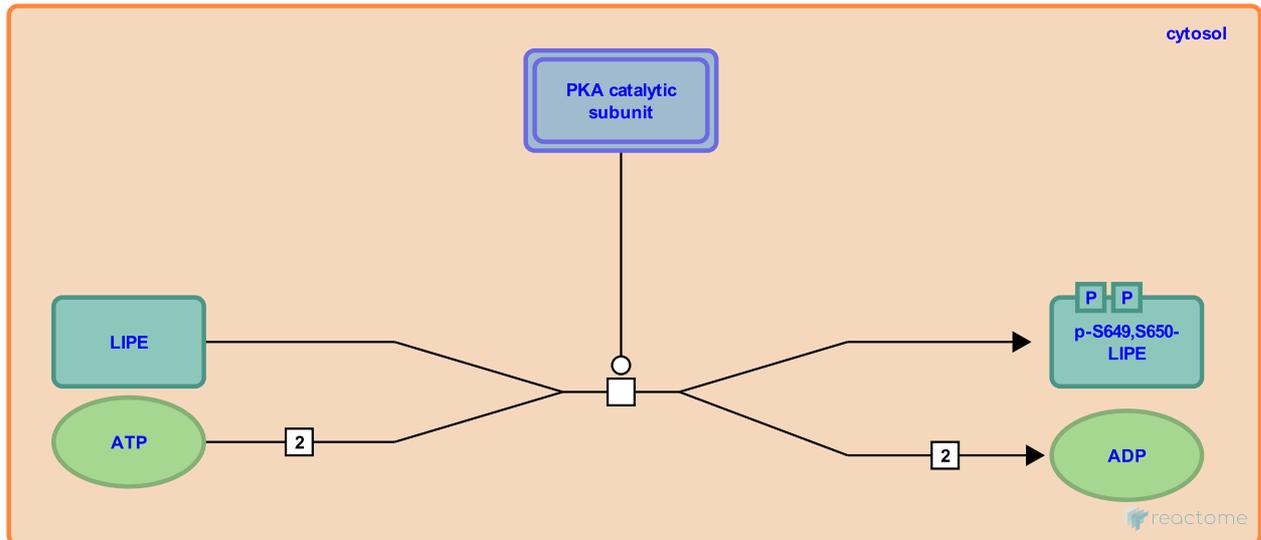
Location: Triglyceride catabolism

Stable identifier: R-HSA-163416

Type: transition

Compartments: cytosol

Inferred from: hormone-sensitive lipase (HSL) + 2 ATP -> phosphorylated HSL + 2 ADP (*Rattus norvegicus*)



Cytosolic rat HSL is phosphorylated on serine residues 659 and 660 by protein kinase A catalytic subunit (Anthonsen et al. 1998; Su et al. 2003). Three isoforms of protein kinase A are known, but with no known differences in substrate specificity or tissue specific expression patterns, so a generic PKA (with all three forms as instances) is annotated as the catalyst of this reaction. Other serine residues in HSL can be phosphorylated both in vitro and in vivo, and while these other phosphorylations appear not to affect triacylglycerol hydrolysis by HSL directly, they may affect the efficiency with which serines 659 and 660 themselves are phosphorylated, or affect the efficiency with which HSL is translocated to cytosolic lipid particles (Holm et al. 2000).

Phosphorylation of human HSL has not been studied in detail, so the human reaction is inferred from the well-studied rat one. By BLAST alignment, human HSL residues 649 and 650 correspond to rat serines 659 and 660.

Followed by: phosphorylated HSL + H₂O -> HSL + orthophosphate, 2 phosphorylated HSL monomers -> phosphorylated HSL dimer

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2 phosphorylated HSL monomers -> phosphorylated HSL dimer ↗

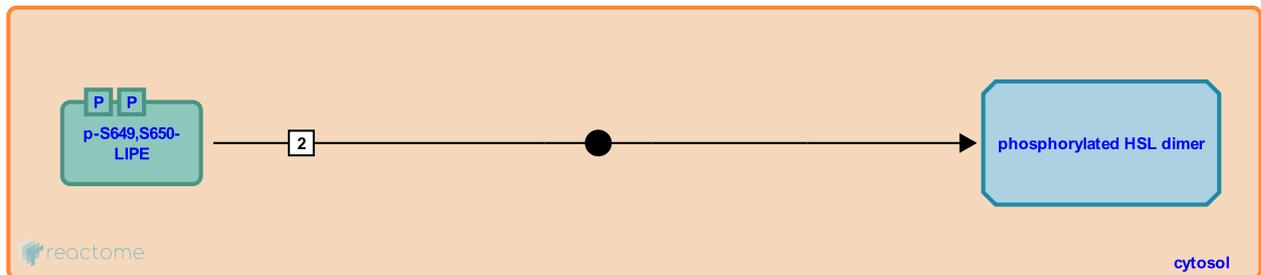
Location: [Triglyceride catabolism](#)

Stable identifier: R-HSA-163602

Type: binding

Compartments: cytosol

Inferred from: [2 phosphorylated HSL monomers -> phosphorylated HSL dimer \(Rattus norvegicus\)](#)



While both monomeric and homodimeric forms of rat HSL protein have been detected, the predominant species, and the one with substantially greater catalytic activity when activated by phosphorylation, is the homodimer so HSL-mediated lipolysis is annotated in Reactome with dimeric phosphorylated enzyme as the catalyst. Phosphorylation appears to be required for dimerization to proceed (Shen et al. 2000).

Dimerization of human HSL has not been studied in detail, so the human reaction is inferred from the well-studied rat one.

Preceded by: [hormone-sensitive lipase \(HSL\) + 2 ATP -> phosphorylated HSL + 2 ADP](#)

Followed by: [Phosphorylated HSL dimer translocates from the cytosol to the lipid particle](#)

Editions

2005-05-02	Authored	D'Eustachio, P.
2019-08-16	Edited	D'Eustachio, P.

perilipin:CGI-58 complex -> perilipin + CGI-58 ↗

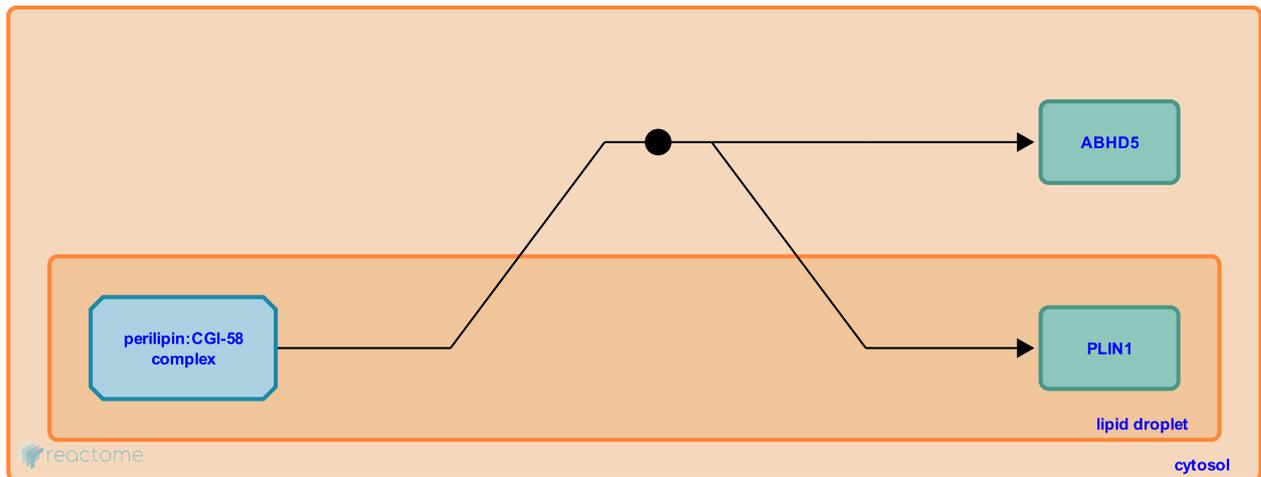
Location: [Triglyceride catabolism](#)

Stable identifier: R-HSA-163539

Type: dissociation

Compartments: cytosol, lipid droplet

Inferred from: [perilipin:CGI-58 complex -> perilipin + CGI-58 \(Mus musculus\)](#)



In unstimulated mouse 3T3-L1 adipocytes, perilipin is localized to the surfaces of cytosolic lipid particles as a complex with CGI-58. Catecholamine stimulation (and by inference glucagon stimulation) is associated with rapid dissociation of the complex and relocalization of the CGI-58 protein away from the lipid particle. The stoichiometry of the complex is unknown. Dissociation of the perilipin:CGI-58 complex appears to precede perilipin phosphorylation, although the molecular link between these two steps is unknown (Subramanian et al. 2004).

The interaction of human CGI-58 and perilipin on the lipid particle surface has not been studied in detail, so the human reaction is inferred from the well-studied mouse one. The observation that humans homozygous for CGI-58 mutations suffer from Chanarin-Dorfman Syndrome, characterized by the abnormal accumulation of triacylglycerol droplets in most tissues (Lefevre et al. 2001), provides indirect evidence that human and mouse CGI-58 proteins have similar functions.

Followed by: [perilipin + 2 ATP -> phosphorylated perilipin + 2 ADP](#)

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perilipin + 2 ATP -> phosphorylated perilipin + 2 ADP ↗

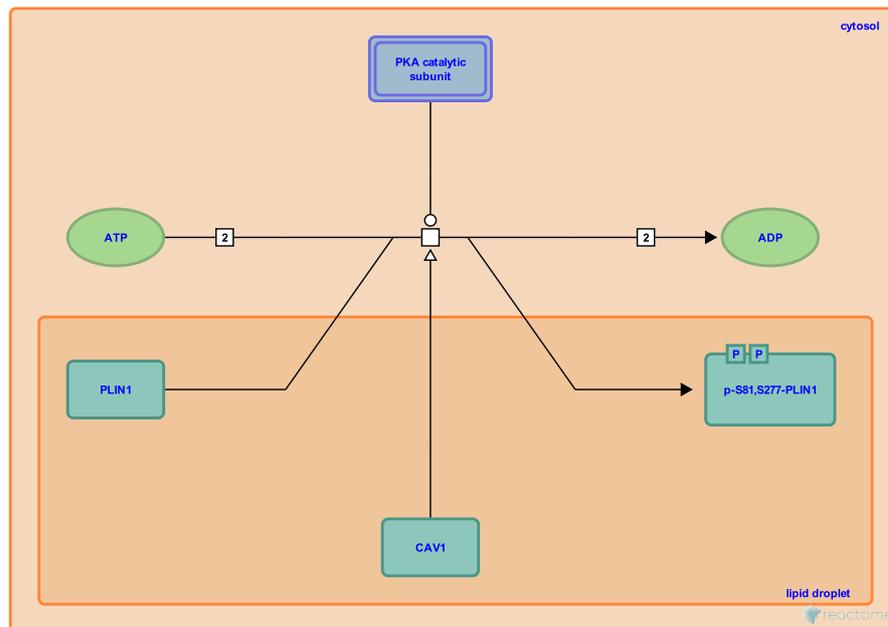
Location: Triglyceride catabolism

Stable identifier: R-HSA-163418

Type: transition

Compartments: cytosol, lipid droplet

Inferred from: perilipin + 3 ATP -> phosphorylated perilipin + 3 ADP (*Rattus norvegicus*), perilipin + 3 ATP -> phosphorylated perilipin + 3 ADP (*Mus musculus*)



Rat perilipin, the major protein at the surfaces of cytosolic lipid particles in adipocytes and steroidogenic cells (Blanchette-Mackie et al. 1995), is phosphorylated by protein kinase A catalytic subunit (Greenberg et al. 1991) on serine residues 81, 223, and 277 (Tansey et al. 2003). All three serine residues and the adjoining sequences that mediate phosphorylation (Cohen 1988) are conserved in mouse perilipin, while only the first and third are conserved in human perilipin. By inference, PKA targets these three mouse and two human serines as well. Phosphorylated perilipin is redistributed on the droplet surfaces (Souza et al. 1998). While two isoforms of rat perilipin protein are found on lipid particles in adipocytes, only the larger isoform appears to regulate lipolysis (Tansey et al 2003). The single human and mouse isoforms of perilipin correspond to the large rat isoform. In mouse 3T3-L1 cells, perilipin phosphorylation requires the presence of caveolin-1 at the surface of the lipid particle (Cohen et al. 2004). This positive regulatory effect of caveolin-1 is inferred for rat and human.

Preceded by: perilipin:CGI-58 complex -> perilipin + CGI-58

Followed by: phosphorylated perilipin + H₂O -> perilipin + orthophosphate, Phosphorylated HSL dimer translocates from the cytosol to the lipid particle

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Phosphorylated HSL dimer translocates from the cytosol to the lipid particle ↗

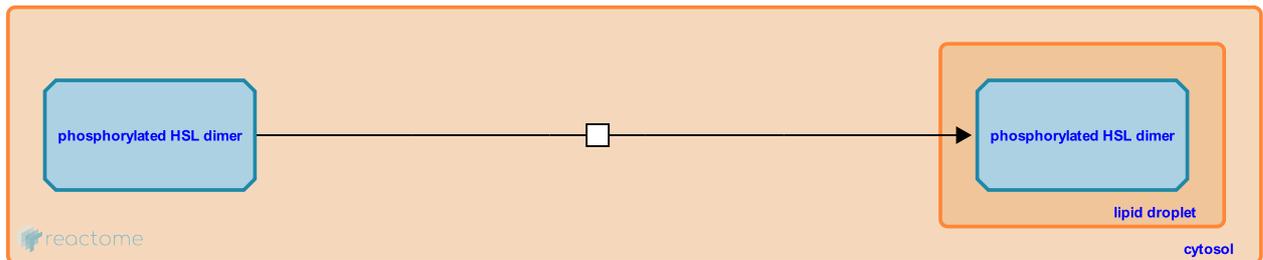
Location: [Triglyceride catabolism](#)

Stable identifier: R-HSA-163554

Type: transition

Compartments: cytosol, lipid droplet

Inferred from: [Phosphorylated HSL dimer translocates from the cytosol to the lipid particle \(Rattus norvegicus\)](#)



In primary adipocytes from young rats and in adipocytes derived from 3T3-L1 cells in vitro, phosphorylated hormone-sensitive lipase translocates from the cytosol to the surfaces of lipid particles following the phosphorylation of perilipin (Clifford et al. 2000; Su et al. 2003; Sztalryd et al. 2003)

The human reaction is inferred from the well-studied rat one.

Preceded by: [2 phosphorylated HSL monomers -> phosphorylated HSL dimer](#), [perilipin + 2 ATP -> phosphorylated perilipin + 2 ADP](#)

Followed by: [phosphorylated HSL dimer + FABP4 -> phosphorylated HSL dimer:FABP4 complex](#)

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phosphorylated HSL dimer + FABP4 -> phosphorylated HSL dimer:FABP4 complex ↗

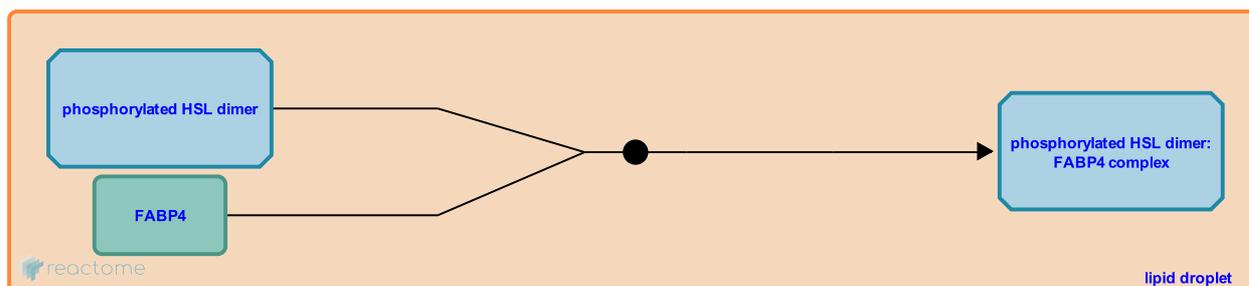
Location: [Triglyceride catabolism](#)

Stable identifier: R-HSA-163549

Type: binding

Compartments: lipid droplet

Inferred from: [phosphorylated HSL dimer + FABPA -> phosphorylated HSL dimer:FABPA complex \(Rattus norvegicus\)](#)



Rat FABPA associates with HSL and increases the rate of triacylglycerol hydrolysis, possibly by sequestering the released fatty acids (Shen et al. 1999; Shen et al. 2001). A similar association of HSL and FABP4 at the lipid droplet surface has been demonstrated in human adipocytes (Smith et al. 2004). The stoichiometry of the fatty acid:FABP complex is unknown. This model implies that HSL-associated FABP loaded with fatty acid should exchange with unloaded, unassociated FABP, allowing HSL to continue to work efficiently while moving newly generated fatty acids away from the lipid particle. To date, there is no evidence for or against such a shuttling process.

Preceded by: [Phosphorylated HSL dimer translocates from the cytosol to the lipid particle](#)

Followed by: [cholesterol ester + H2O -> cholesterol + fatty acid](#), [triacylglycerol + H2O -> diacylglycerol + fatty acid](#)

Literature references

Smith, AJ., Sanders, MA., Thompson, BR., Londos, C., Kraemer, FB., Bernlohr, DA. (2004). Physical association between the adipocyte fatty acid-binding protein and hormone-sensitive lipase: a fluorescence resonance energy transfer analysis. *J Biol Chem*, 279, 52399-405. ↗

Editions

2005-05-02	Authored	D'Eustachio, P.
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cholesterol ester + H₂O -> cholesterol + fatty acid ↗

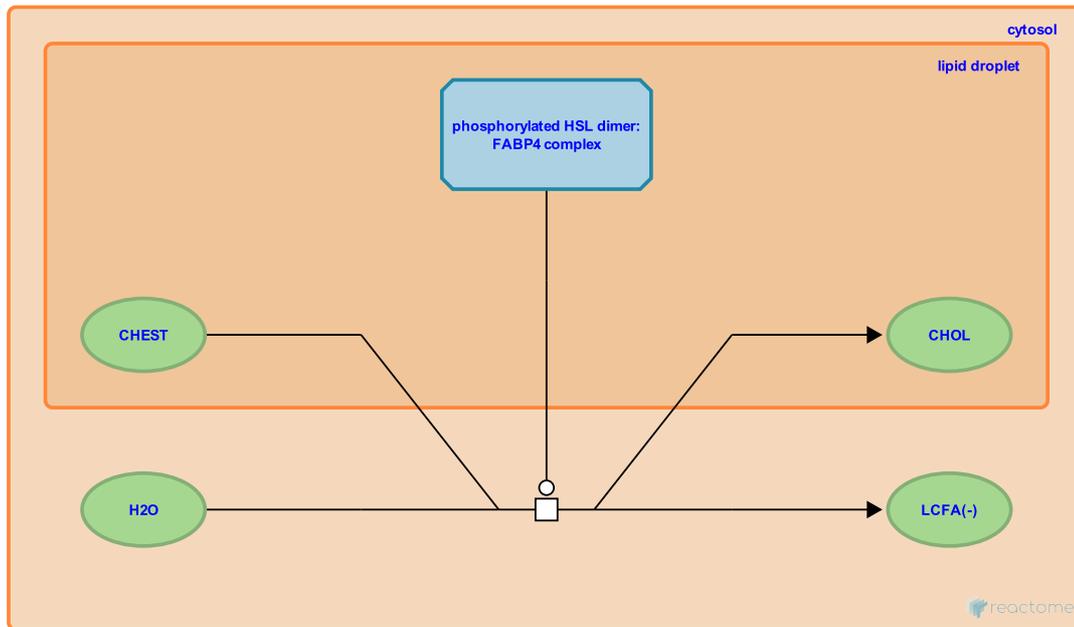
Location: [Triglyceride catabolism](#)

Stable identifier: R-HSA-163432

Type: transition

Compartments: cytosol, lipid droplet

Inferred from: [cholesterol ester + H₂O -> cholesterol + fatty acid \(Rattus norvegicus\)](#)



Activated rat HSL hydrolyzes cholesterol ester to yield cholesterol + fatty acid (Fredrikson et al. 1981). The human reaction has not been studied in detail, and is inferred from the well-characterized rat one.

Preceded by: [phosphorylated HSL dimer + FABP4 -> phosphorylated HSL dimer:FABP4 complex](#)

Followed by: [FABPs bind LCFAs](#)

Editions

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triacylglycerol + H₂O -> diacylglycerol + fatty acid ↗

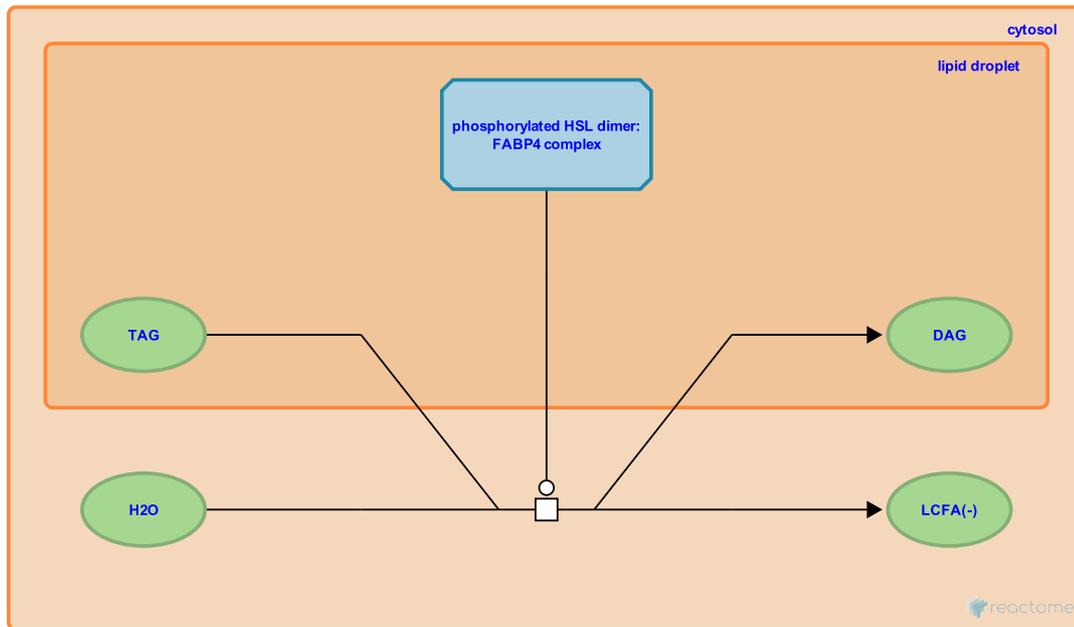
Location: [Triglyceride catabolism](#)

Stable identifier: R-HSA-163551

Type: transition

Compartments: cytosol, lipid droplet

Inferred from: [triacylglycerol + H₂O -> diacylglycerol + fatty acid \(Rattus norvegicus\)](#)



Activated rat HSL at the lipid particle hydrolyzes triacylglycerol to yield diacylglycerol + fatty acid. In vitro, activated partially purified HSL catalyzes this reaction at only about two times the rate measured with non-activated enzyme (Fredrikson et al. 1981). The much greater rate increase caused by HSL phosphorylation in vivo appears to be due to its phosphorylation-dependent translocation to the surface of the lipid particle (Birnbaum 2003).

HSL-mediated triacylglycerol hydrolysis in humans has not been studied in detail, so the human reaction is inferred from the well-studied rat one.

Preceded by: [phosphorylated HSL dimer + FABP4 -> phosphorylated HSL dimer:FABP4 complex](#)

Followed by: [diacylglycerol + H₂O -> 2-acylglycerol + fatty acid](#), [FABPs bind LCFAs](#)

Editions

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diacylglycerol + H₂O -> 2-acylglycerol + fatty acid ↗

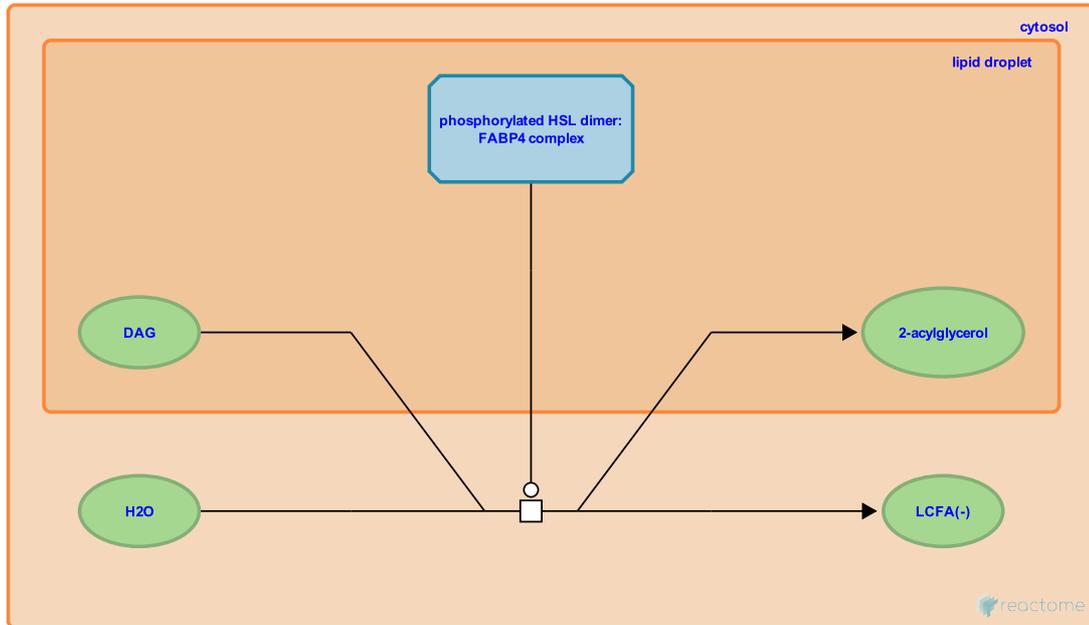
Location: Triglyceride catabolism

Stable identifier: R-HSA-163402

Type: transition

Compartments: cytosol, lipid droplet

Inferred from: diacylglycerol + H₂O -> 2-acylglycerol + fatty acid (Rattus norvegicus)



Rat HSL catalyzes the hydrolysis of diacylglycerol to yield 2-acylglycerol + fatty acid (Fredrikson and Bel-frage 1983). The human event has not been studied in detail and is inferred from the rat one.

Preceded by: triacylglycerol + H₂O -> diacylglycerol + fatty acid

Followed by: 2-acylglycerol + H₂O -> glycerol + fatty acid, FABPs bind LCFAs

Editions

2005-05-02

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2019-08-16

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D'Eustachio, P.

2-acylglycerol + H₂O -> glycerol + fatty acid ↗

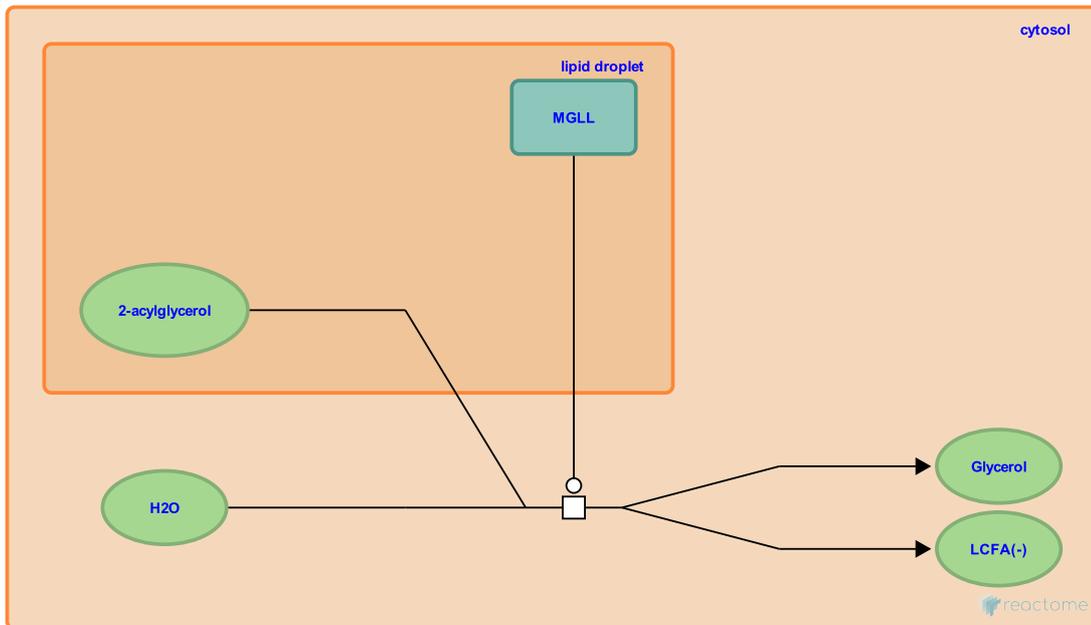
Location: Triglyceride catabolism

Stable identifier: R-HSA-163595

Type: transition

Compartments: cytosol, lipid droplet

Inferred from: 2-acylglycerol + H₂O -> glycerol + fatty acid (Rattus norvegicus)



Rat monoacylglycerol lipase (MGLL) catalyzes the hydrolysis of 2-acylglycerol to yield glycerol + fatty acid (Tornqvist and Belfrage 1976; Fredrikson et al. 1986). Localization of the enzyme to lipid particles is plausible, given its low solubility and its involvement in acylglycerol metabolism, but this localization has not been directly experimentally verified. The human reaction is inferred from the well-studied rat one.

Preceded by: diacylglycerol + H₂O -> 2-acylglycerol + fatty acid

Followed by: FABPs bind LCFAs

Editions

2005-05-02	Authored	D'Eustachio, P.
2019-08-16	Edited	D'Eustachio, P.

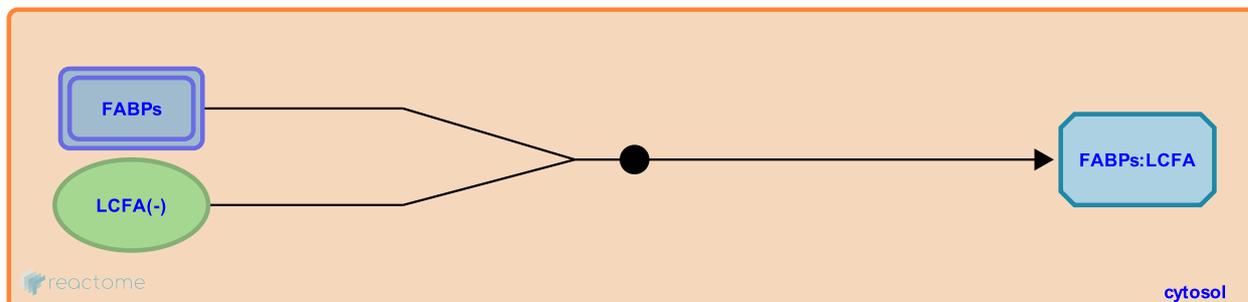
FABPs bind LCFAs ↗

Location: Triglyceride catabolism

Stable identifier: R-HSA-5334794

Type: binding

Compartments: cytosol



Hydrophobic compounds such as long-chain fatty acids (LCFAs) and their acyl-CoA derivatives (LCFA-CoAs) are involved in important functions within a cell such as membrane substrates, energy sources and signalling molecules. The hydrophobic nature of these compounds makes translocation between different compartments of a cell extremely difficult. Fatty acid-binding proteins (FABPs) are able to bind these hydrophobic compounds with high affinity and transport them through the cytosol for delivery to different organelles within the cell. To date, 9 human FABP-coding genes have been identified (Smathers & Petersen 2011).

Preceded by: 2-acylglycerol + H₂O -> glycerol + fatty acid, cholesterol ester + H₂O -> cholesterol + fatty acid, diacylglycerol + H₂O -> 2-acylglycerol + fatty acid, triacylglycerol + H₂O -> diacylglycerol + fatty acid

Literature references

Smathers, RL., Petersen, DR. (2011). The human fatty acid-binding protein family: evolutionary divergences and functions. *Hum. Genomics*, 5, 170-91. ↗

Editions

2014-02-27	Authored, Edited	Jassal, B.
2015-02-11	Reviewed	D'Eustachio, P.

phosphorylated HSL + H₂O -> HSL + orthophosphate ↗

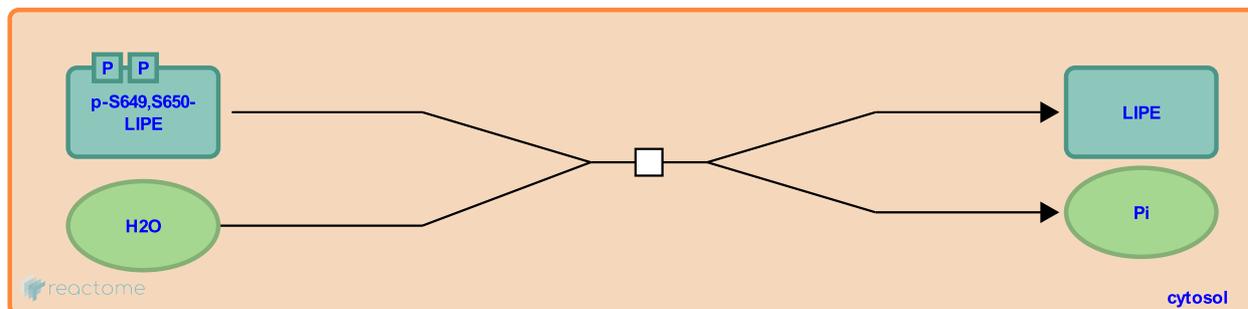
Location: [Triglyceride catabolism](#)

Stable identifier: R-HSA-163489

Type: transition

Compartments: cytosol

Inferred from: [phosphorylated HSL + H₂O -> HSL + orthophosphate \(Rattus norvegicus\)](#)



Rat HSL is inactivated by dephosphorylation. The catalyst of this reaction is unknown. Protein phosphatases 1 and 2A are both abundant in rat adipocytes and both are active on HSL (Olsson and Belfrage 1987; Wood et al. 1993). Whether these enzymes act on phosphate groups attached to serine residues 659 and 660 of HSL is unknown, however (Holm et al. 2000). Although the reaction is annotated as though the phosphatase acts on phosphorylated HSL monomers, this also is unknown: does the HSL:FABP complex dissociate before HSL dephosphorylation (as implied here), or does dephosphorylation of HSL drive dissociation of the complex?

Dephosphorylation of human HSL has not been studied in detail, so the human reaction is inferred from the well-studied rat one.

Preceded by: [hormone-sensitive lipase \(HSL\) + 2 ATP -> phosphorylated HSL + 2 ADP](#)

Editions

2005-05-02	Authored	D'Eustachio, P.
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phosphorylated perilipin + H₂O -> perilipin + orthophosphate ↗

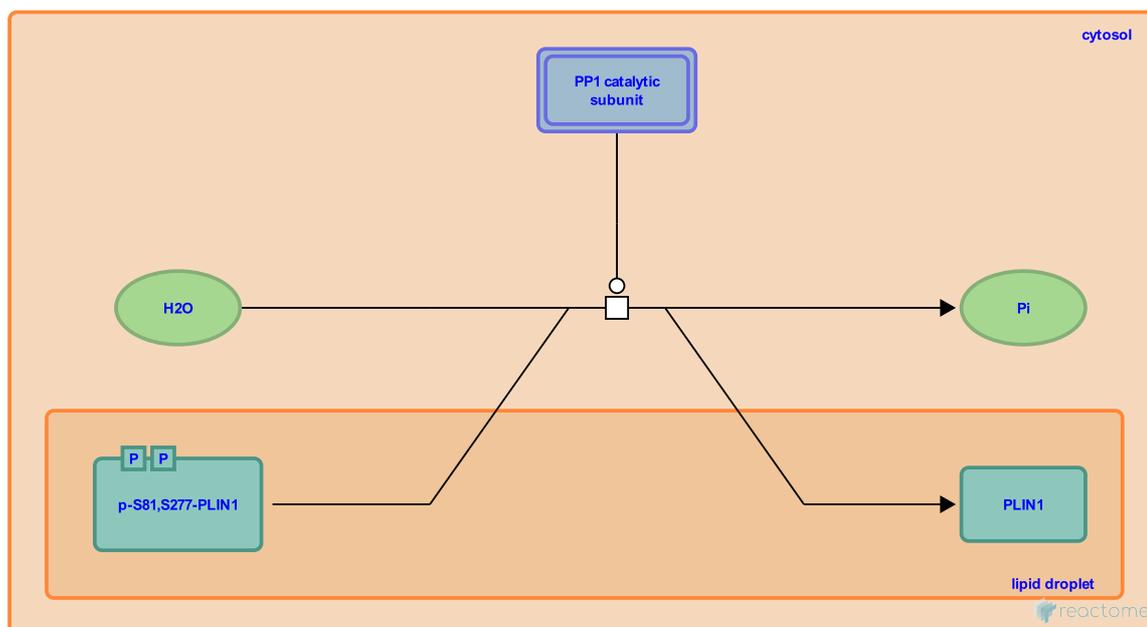
Location: Triglyceride catabolism

Stable identifier: R-HSA-163568

Type: transition

Compartments: cytosol, lipid droplet

Inferred from: phosphorylated perilipin + H₂O -> perilipin + orthophosphate (Rattus norvegicus)



Rat perilipin is dephosphorylated by protein phosphatase 1 (Clifford et al. 1998). All three protein phosphatase 1 isoforms appear competent to carry out this reaction and there are no data to indicate which one preferentially acts on perilipin in vivo. Dephosphorylation of human perilipin has not been studied in detail, so the human reaction is inferred from the well-studied rat one.

Preceded by: perilipin + 2 ATP -> phosphorylated perilipin + 2 ADP

Editions

2005-05-02	Authored	D'Eustachio, P.
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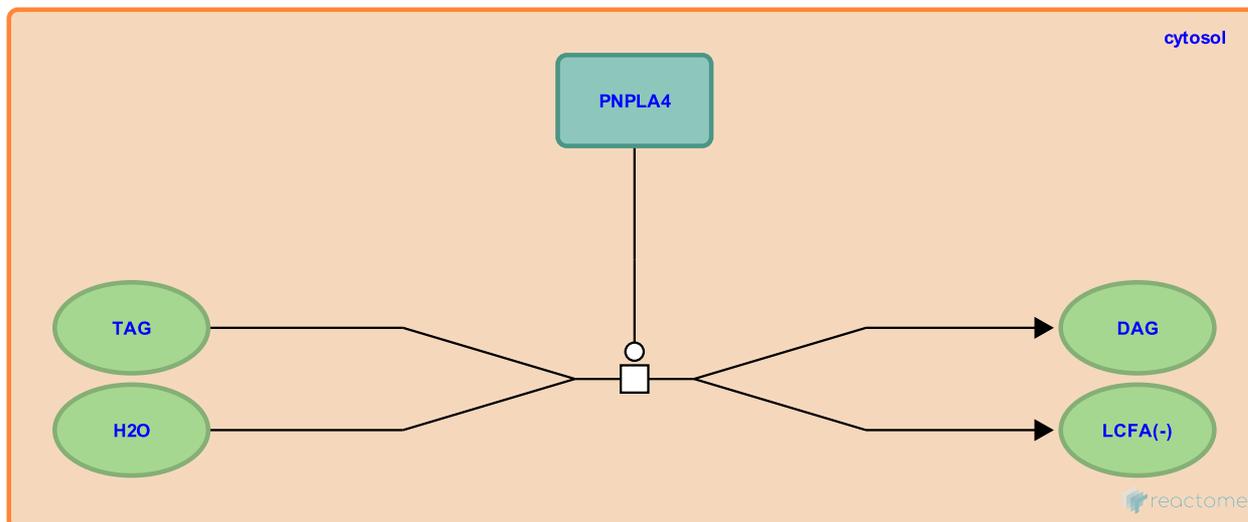
PNPLA4 hydrolyzes TAG ↗

Location: [Triglyceride catabolism](#)

Stable identifier: R-HSA-8848338

Type: transition

Compartments: cytosol, endoplasmic reticulum membrane



PNPLA4 (patatin-like phospholipase domain-containing protein 4, also known as GS2 and iPLA2(eta)) catalyzes the hydrolysis of TAG (triacylglycerol) to DAG (diacylglycerol) and one molecule of LCFA (long chain fatty acid). The enzyme also has transacylase activity not annotated here (Gao and Simon 2007; Jenkins et al. 2004).

Literature references

Gao, JG., Simon, M. (2007). A comparative study of human GS2, its paralogues, and its rat orthologue. *Biochem. Biophys. Res. Commun.*, 360, 501-6. ↗

Jenkins, CM., Mancuso, DJ., Yan, W., Sims, HF., Gibson, B., Gross, RW. (2004). Identification, cloning, expression, and purification of three novel human calcium-independent phospholipase A2 family members possessing triacylglycerol lipase and acylglycerol transacylase activities. *J Biol Chem*, 279, 48968-75. ↗

Editions

2015-12-07	Authored, Edited	D'Eustachio, P.
2016-01-29	Reviewed	Jassal, B.

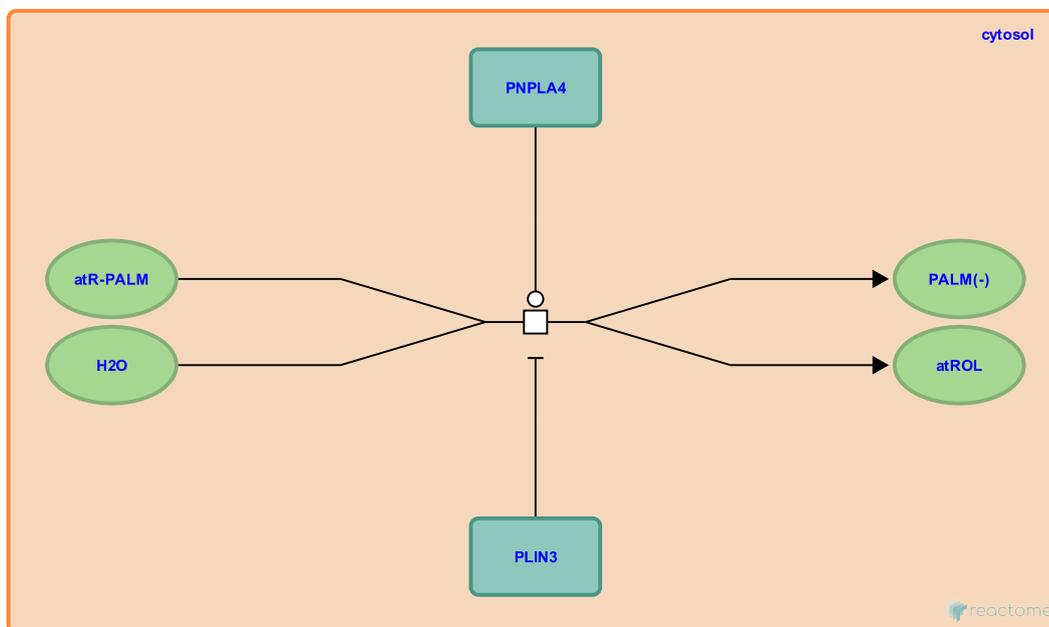
PNPLA4 hydrolyzes retinyl palmitate ↗

Location: [Triglyceride catabolism](#)

Stable identifier: R-HSA-8848355

Type: transition

Compartments: cytosol



PNPLA4 (patatin-like phospholipase domain-containing protein 4, also known as GS2 and iPLA2(eta)) catalyzes the hydrolysis of atR-PALM (all-trans retinyl palmitate) to atROL (all-trans retinol) and a molecule of PALM (palmitate) (Gao and Simon 2006; Gao and Simon 2007). The reaction is inhibited by cytosolic PLIN3 (perilipin 3). The enzyme may also catalyze transacylation reactions to form retinyl esters or promote the activity of other enzymes that do so (Gao et al. 2009).

Literature references

Gao, JG., Simon, M. (2006). Molecular screening for GS2 lipase regulators: inhibition of keratinocyte retinylester hydrolysis by TIP47. *J. Invest. Dermatol.*, 126, 2087-95. ↗

Gao, JG., Simon, M. (2007). A comparative study of human GS2, its paralogues, and its rat orthologue. *Biochem. Biophys. Res. Commun.*, 360, 501-6. ↗

Gao, JG., Shih, A., Gruber, R., Schmuth, M., Simon, M. (2009). GS2 as a retinol transacylase and as a catalytic dyad independent regulator of retinylester accretion. *Mol. Genet. Metab.*, 96, 253-60. ↗

Editions

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2016-01-29	Reviewed	Jassal, B.

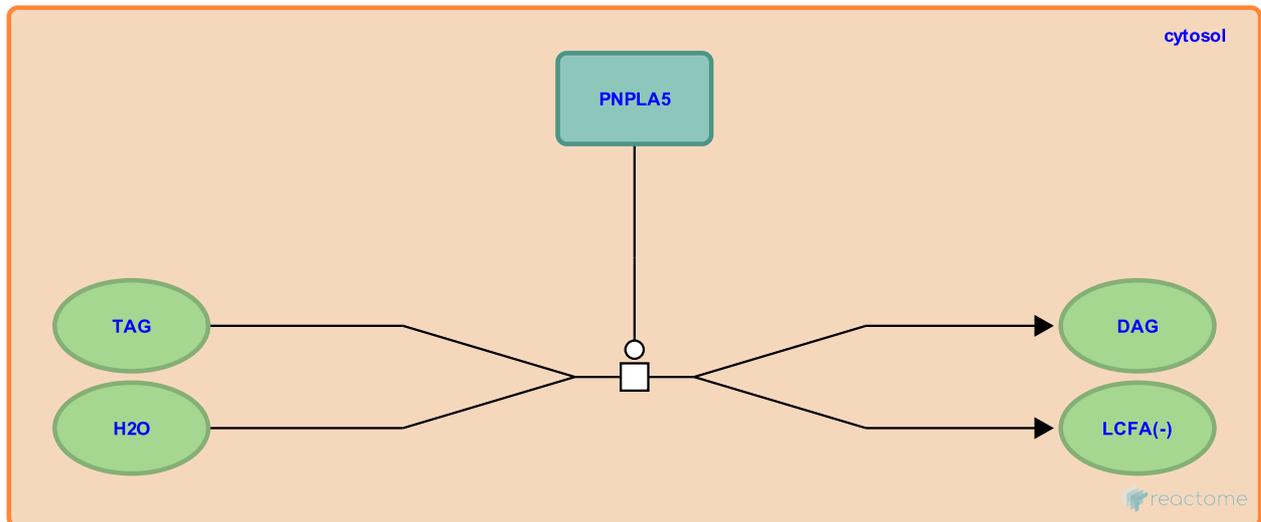
PNPLA5 hydrolyzes TAG ↗

Location: [Triglyceride catabolism](#)

Stable identifier: R-HSA-8848339

Type: transition

Compartments: cytosol, endoplasmic reticulum membrane



PNPLA5 (patatin-like phospholipase domain-containing protein 5, also known as GS2 like) catalyzes the hydrolysis of TAG (triacylglycerol) to DAG (diacylglycerol) and one molecule of LCFA (long chain fatty acid) (Gao and Simon 2007).

Literature references

Gao, JG., Simon, M. (2007). A comparative study of human GS2, its paralogues, and its rat orthologue. *Biochem. Biophys. Res. Commun.*, 360, 501-6. ↗

Editions

2015-12-07	Authored, Edited	D'Eustachio, P.
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Gly-3-P+FAD->DHAP+FADH2 (catalyzed by mitochondrial Gly-Phos dehydrogenase)

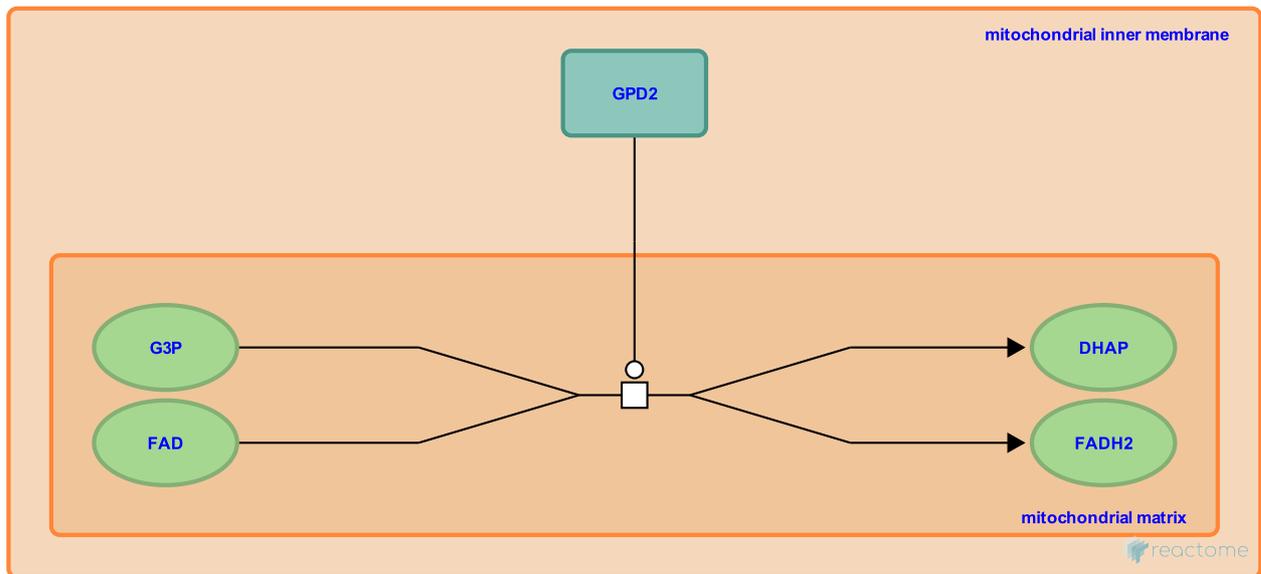


Location: Triglyceride catabolism

Stable identifier: R-HSA-188467

Type: transition

Compartments: mitochondrial matrix, mitochondrial inner membrane



FAD-linked mitochondrial glycerol 3-phosphate dehydrogenase (GPD2, alias: mGPDH) and its NAD-linked cytosolic isoform (GPD1, alias:cGPDH) constitute glycerol phosphate shuttle. GPD2 catalyzes the unidirectional conversion of glycerol-3-phosphate (G-3-P) to dihydroxyacetone phosphate (DHAP) with concomitant reduction of the enzyme-bound FAD. Impaired activity of GPD2 has been suggested to be one of the primary causes of insulin secretory defects in beta-cells and thus it is a candidate gene for type 2 diabetes.

Literature references

Stryer, L. (1995). 21: Oxidative Phosphorylation, *Biochemistry*, 4th ed. *WH Freeman*, 548-549.

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