

# ACSL4 (FACL4) Antibody (Center)

Purified Rabbit Polyclonal Antibody (Pab)

Catalog # AP2536B

## Product Information

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<b>Application</b>	WB, IHC-P, IF, E
<b>Primary Accession</b>	<a href="#">O60488</a>
<b>Reactivity</b>	Human
<b>Host</b>	Rabbit
<b>Clonality</b>	Polyclonal
<b>Isotype</b>	Rabbit IgG
<b>Calculated MW</b>	79188
<b>Antigen Region</b>	236-267

## Additional Information

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<b>Gene ID</b>	2182
<b>Other Names</b>	Long-chain-fatty-acid--CoA ligase 4, Long-chain acyl-CoA synthetase 4, LACS 4, ACSL4, ACS4, FACL4, LACS4
<b>Target/Specificity</b>	This ACSL4 (FACL4) antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 236-267 amino acids from the Central region of human ACSL4 (FACL4).
<b>Dilution</b>	WB~~1:1000 IHC-P~~1:100~500 IF~~1:10~50 E~~Use at an assay dependent concentration.
<b>Format</b>	Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.
<b>Storage</b>	Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
<b>Precautions</b>	ACSL4 (FACL4) Antibody (Center) is for research use only and not for use in diagnostic or therapeutic procedures.

## Protein Information

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<b>Name</b>	ACSL4
<b>Synonyms</b>	ACS4, FACL4, LACS4
<b>Function</b>	Catalyzes the conversion of long-chain fatty acids to their active form acyl-CoA for both synthesis of cellular lipids, and degradation via

beta-oxidation (PubMed:[21242590](#), PubMed:[22633490](#), PubMed:[24269233](#), PubMed:[31061331](#), PubMed:[38720107](#)). Preferentially activates arachidonate and eicosapentaenoate as substrates (PubMed:[21242590](#)). Preferentially activates 8,9-EET > 14,15-EET > 5,6- EET > 11,12-EET (PubMed:[21242590](#)). Modulates glucose-stimulated insulin secretion by regulating the levels of unesterified EETs (By similarity). Modulates prostaglandin E2 secretion (PubMed:[21242590](#)). Acts as an activator of ferroptosis by activating polyunsaturated fatty acids, especially arachidonate and adrenate, to their active form, generating the primary lipid-peroxidation substrates that contribute to ferroptosis (PubMed:[27842070](#), PubMed:[35027735](#), PubMed:[38720107](#)).

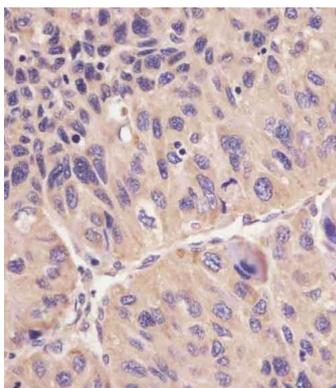
### Cellular Location

Mitochondrion outer membrane; Single-pass type III membrane protein.  
Endoplasmic reticulum membrane; Single-pass type III membrane protein.  
Cell membrane; Single-pass type III membrane protein

## Background

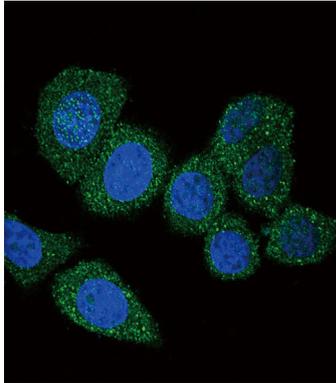
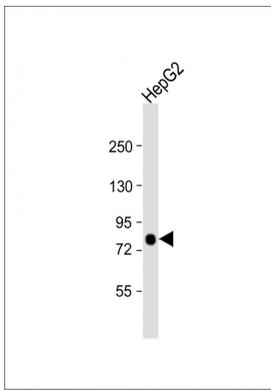
Long chain acyl-CoA synthetase (LACS), or long chain fatty acid-CoA ligase (FACL), converts free long chain fatty acids into fatty acyl-CoA esters, key intermediates in the synthesis of complex lipids. The FACL4 gene encodes a form of LACS and is expressed in several tissues, including brain. FACL4 cDNA from brain encodes a gene product that shows preference for arachidonic acid as a substrate when expressed in mammalian cells.<sup>1</sup> The sequence of the predicted 670-amino acid human protein is 97% identical to that of rat ACS4. FACL4 is highly expressed in adult human brain, especially in the cerebellum and hippocampus, similar to the mouse.<sup>2</sup> A strong cytoplasmic staining was found in the Purkinje and granular cells of the cerebellum and the pyramidal layer of hippocampus, indicating that FACL4 is specifically expressed in neurons and not in glial cells. Two patients with Alport syndrome, elliptocytosis, and mental retardation carried a large deletion of the COL4A5 region that included FACL4.<sup>3</sup> The absence of FACL4 might play a role in the development of mental retardation or other signs associated with Alport syndrome. Two point mutations, 1 missense and 1 splice site change, were reported in the FACL4 gene in 2 families with nonspecific mental retardation.<sup>2</sup> Analysis of enzymatic activity in lymphoblastoid cell lines of affected individuals revealed low levels compared with normal cells, indicating that both mutations are null mutations.

## Images



AP2536B staining hFACL4 in human human hepatocarcinoma sections by Immunohistochemistry (IHC-P - paraformaldehyde-fixed, paraffin-embedded sections). Tissue was fixed with formaldehyde and blocked with 3% BSA for 0.5 hour at room temperature; antigen retrieval was by heat mediation with a citrate buffer (pH6). Samples were incubated with primary antibody (1/25) for 1 hours at 37°C. A undiluted biotinylated goat polyvalent antibody was used as the secondary antibody.

Anti-FACL4 Antibody (E251) at 1:1000 dilution + HepG2 whole cell lysate Lysates/proteins at 20 µg per lane. Secondary Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size : 79 kDa Blocking/Dilution buffer: 5% NFDm/TBST.



Confocal immunofluorescent analysis of ACSL4 (FACL4) Antibody (Center) (Cat#AP2536b) with HeLa cell followed by Alexa Fluor 488-conjugated goat anti-rabbit IgG (green). DAPI was used to stain the cell nuclear (blue).

## Citations

- [The TBX1/miR-193a-3p/TGF- 2 Axis Mediates CHD by Promoting Ferroptosis](#)
- [Increased localization of APP-C99 in mitochondria-associated ER membranes causes mitochondrial dysfunction in Alzheimer disease.](#)
- [Disruption of the Mitochondria-Associated ER Membrane \(MAM\) Plays a Central Role in Palmitic Acid-Induced Insulin Resistance.](#)
- [Highly potent intracellular membrane-associated A \$\beta\$  seeds.](#)
- [Isolation of Endoplasmic Reticulum, Mitochondria, and Mitochondria-Associated Membrane and Detergent Resistant Membrane Fractions from Transfected Cells and from Human Cytomegalovirus-Infected Primary Fibroblasts.](#)
- [Human cytomegalovirus inhibits apoptosis by proteasome-mediated degradation of Bax at endoplasmic reticulum-mitochondrion contacts.](#)
- [Quantitative proteomic analyses of human cytomegalovirus-induced restructuring of endoplasmic reticulum-mitochondrial contacts at late times of infection.](#)
- [Mitochondrial-associated endoplasmic reticulum membranes \(MAM\) form innate immune synapses and are targeted by hepatitis C virus.](#)
- [The human cytomegalovirus protein UL37 exon 1 associates with internal lipid rafts.](#)
- [Intellectual disability, midface hypoplasia, facial hypotonia, and Alport syndrome are associated with a deletion in Xq22.3.](#)
- [Presenilins are enriched in endoplasmic reticulum membranes associated with mitochondria.](#)
- [PACS-2 controls endoplasmic reticulum-mitochondria communication and Bid-mediated apoptosis.](#)

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.