ACSL5 (E-12): sc-398310



The Power to Question

BACKGROUND

Acyl-CoA synthetases, also known as long-chain fatty-acid CoA synthases (FACL) or palmitoyl-CoA ligases, include ACSL1-6, which are all single-pass membrane proteins localizing to the mitochondrion, microsome or peroxisome. ACSL proteins are important for synthesis of cellular lipids and for β -oxidation degradation. Specifically, ACSL proteins catalyze the activation of long-chain fatty acids to acyl-CoAs, which can be metabolized to form CO2, triacylglycerol (TAG), phospholipids (PL) and cholesteryl esters (CE). ACSL5 utilizes a wide range of saturated fatty acids with a preference for C16-C18 unsaturated fatty acids. It is highly expressed in uterus and spleen. A decrease in expression of ACSL5 is correlated with tumorigenesis, including endometrioid adenocarcinomas and colorectal carcinomas. ACSL5 is also useful as a differentiating marker in the gastrointestinal tract.

REFERENCES

- Oikawa, E., et al. 1998. A novel acyl-CoA synthetase, ACS5, expressed in intestinal epithelial cells and proliferating preadipocytes. J. Biochem. 124: 679-685.
- Muoio, D.M., et al. 2000. Acyl-CoAs are functionally channeled in liver: potential role of acyl-CoA synthetase. Am. J. Physiol. Endocrinol. Metab. 279: E1366-E1373.
- Coleman, R.A., et al. 2002. Do long-chain acyl-CoA synthetases regulate fatty acid entry into synthetic versus degradative pathways? J. Nutr. 132: 2123-2126.
- 4. Gassler, N., et al. 2003. Impaired expression of acyl-CoA-synthetase 5 in epithelial tumors of the small intestine. Hum. Pathol. 34: 1048-1052.
- Gassler, N., et al. 2005. Expression of acyl-CoA synthetase 5 in human endometrium and in endometrioid adenocarcinomas. Histopathology 47: 501-507.
- Gassler, N., et al. 2005. Impaired expression of acyl-CoA synthetase 5 in sporadic colorectal adenocarcinomas. J. Pathol. 207: 295-300.

CHROMOSOMAL LOCATION

Genetic locus: ACSL5 (human) mapping to 10q25.2; Acsl5 (mouse) mapping to 19 D2.

SOURCE

ACSL5 (E-12) is a mouse monoclonal antibody specific for an epitope mapping between amino acids 73-104 near the N-terminus of ACSL5 of human origin.

PRODUCT

Each vial contains 200 μ g lgG₁ kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-398310 P, (100 μ g peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% stabilizer protein).

RESEARCH USE

For research use only, not for use in diagnostic procedures.

APPLICATIONS

ACSL5 (E-12) is recommended for detection of short and long isoforms of ACSL5 of mouse, rat and human origin by Western Blotting (starting dilution 1:100, dilution range 1:100-1:1000), immunoprecipitation [1-2 μ g per 100-500 μ g of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for ACSL5 siRNA (h): sc-60621, ACSL5 siRNA (m): sc-60622, ACSL5 shRNA Plasmid (h): sc-60621-SH, ACSL5 shRNA Plasmid (m): sc-60622-SH, ACSL5 shRNA (h) Lentiviral Particles: sc-60621-V and ACSL5 shRNA (m) Lentiviral Particles: sc-60622-V.

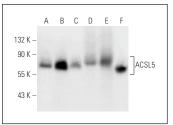
Molecular Weight of ACSL5: 76 kDa.

Positive Controls: HeLa whole cell lysate: sc-2200, Hep G2 cell lysate: sc-2227 or MDA-MB-231 cell lysate: sc-2232.

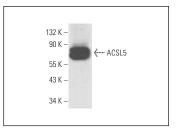
RECOMMENDED SUPPORT REAGENTS

To ensure optimal results, the following support reagents are recommended: 1) Western Blotting: use m-lgG κ BP-HRP: sc-516102 or m-lgG κ BP-HRP (Cruz Marker): sc-516102-CM (dilution range: 1:1000-1:10000), Cruz MarkerTM Molecular Weight Standards: sc-2035, UltraCruz* Blocking Reagent: sc-516214 and Western Blotting Luminol Reagent: sc-2048. 2) Immunoprecipitation: use Protein A/G PLUS-Agarose: sc-2003 (0.5 ml agarose/2.0 ml). 3) Immunofluorescence: use m-lgG κ BP-FITC: sc-516140 or m-lgG κ BP-PE: sc-516141 (dilution range: 1:50-1:200) with UltraCruz* Mounting Medium: sc-24941 or UltraCruz* Hard-set Mounting Medium: sc-359850.

DATA







ACSL5 (E-12): sc-398310. Western blot analysis of ACSL5 expression in K-562 whole cell lysate.

SELECT PRODUCT CITATIONS

 Hou, T., et al. 2022. Cytoplasmic SIRT6-mediated ACSL5 deacetylation impedes nonalcoholic fatty liver disease by facilitating hepatic fatty acid oxidation. Mol. Cell 82: 4099-4115.e9.

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.