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Produktinformation

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Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

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PRODUCT INFORMATION

ACSL3 (human, recombinant; aa 45-720)

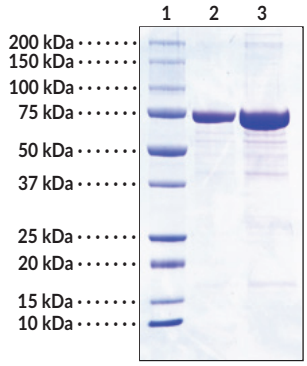
Item No. 39990

Overview and Properties

Synonyms:	ACS3, Fatty Acid CoA Ligase 3, LACS 3, Long-chain Acyl-CoA Synthetase 3, Long-chain Fatty Acid-CoA Ligase 3
Source:	Active recombinant human N-Terminal His-tagged ACSL3 expressed in *E. coli*
Amino Acids:	45-720
Uniprot No.:	P33121
Molecular Weight:	77.19 kDa
Storage:	-80°C (as supplied)
Stability:	≥1 year
Purity:	≥90% estimated by SDS-PAGE
Supplied in:	50 mM Tris pH 8.0, with 150 mM sodium chloride and 10% glycerol
Protein Concentration:	***batch specific*** mg/ml
Activity:	***batch specific*** U/ml
Specific Activity:	***batch specific*** U/mg
Unit Definition:	One unit is defined as the amount of enzyme required to measure the change of free fatty acid concentration in µM per minute at 37 °C.

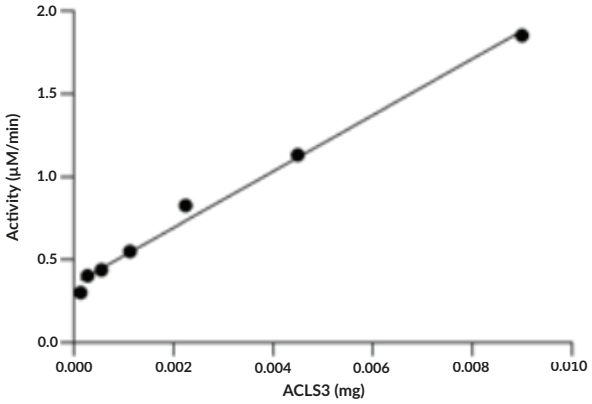
Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Images



Lane 1: MW Markers
Lane 2: ACSL3 (2 µg)
Lane 3: ACSL3 (4 µg)

SDS-Page Analysis of ACSL3. This protein has a calculated molecular weight of 77.19 kDa.



ACLS3 enzyme activity was determined using Cayman's Free Fatty Acid Fluorometric Assay Kit (Item No. 700310) with 100 µm oleic acid as the substrate.

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PRODUCT INFORMATION

Description

Long-chain acyl-CoA synthetase 3 (ACSL3) is an enzyme involved in lipid biosynthesis and fatty acid degradation.[1] It is composed of short N- and C-terminal domains surrounding an AMP-binding domain.[2] It is expressed in the brain and prostate and localizes to the endoplasmic reticulum, mitochondria, and lipid droplets.[1] *ACSL3* is activated by a variety of factors, including peroxisome proliferator-activated receptor δ (PPARδ), octamer-binding transcription factor 1 (Oct1), and the liver X receptor (LXR), among others. ACSL3 converts free monounsaturated long-chain fatty acids into fatty acyl-CoA esters, which are used as substrates for phospholipid and glycerolipid biosynthesis or transported into the mitochondria to be degraded *via* fatty acid β-oxidation for use as an energy source.[1,3,4] ACSL3-dependent conversion of exogenous MUFAs into fatty acyl-CoA esters can induce resistance to ferroptotic cell death by displacing PUFAs and preventing the accumulation of lipid reactive oxygen species (ROS) in the plasma membrane.[3,5] ACSL3 is required for tumor cell survival in non-small cell lung cancer (NSCLC) mouse xenograft models, including those expressing K-Ras containing the glycine-to-aspartate activating mutation at position 12 (K-Ras^{G12D}).[6] It is also highly expressed in tumor tissues isolated from patients with early-stage NSCLC. In contrast, homozygous deletion of ACSL3 is associated with an increased risk of metastasis or recurrence in patients with triple-negative breast cancer (TNBC) receiving adjuvant chemotherapy.[7] Cayman's ACSL3 (human, recombinant) protein can be used for ELISA, enzyme activity assay, and Western blot applications.

References

1. Quan, J., Bode, A.M., and Luo, X. ACSL family: The regulatory mechanisms and therapeutic implications in cancer. *Eur. J. Pharmacol.* **909:174397**, (2021).
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3. Dixon, S.J. and Olzmann, J.A. The cell biology of ferroptosis. *Nat. Rev. Mol. Cell Biol.* (2024).
4. Yang, Y., Zhu, T., Wang, X., *et al.* ACSL3 and ACSL4, distinct roles in ferroptosis and cancers. *Cancers (Basel)* **14(23)**, 5896 (2022).
5. Magtanong, L., Ko, P.-J., To, M., *et al.* Exogenous monounsaturated fatty acids promote a ferroptosis-resistant cell state. *Cell Chem. Biol.* **26(3)**, 420-432 (2019).
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7. Jeong, H.M., Kim, R.N., Kwon, M.J., *et al.* Targeted exome sequencing of Korean triple-negative breast cancer reveals homozygous deletions associated with poor prognosis of adjuvant chemotherapy-treated patients. *Oncotarget* **8(37)**, 61538-61550 (2017).

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