

ACC α (D-5): sc-137104

BACKGROUND

Acetyl-CoA carboxylase (ACC) is a complex multifunctional enzyme system which catalyzes the carboxylation of acetyl-CoA to malonyl-CoA, the rate-limiting step in fatty acid synthesis. Exercise diminishes the activity of acetyl-CoA carboxylase in human muscle. ACC α (ACC1) is the rate-limiting enzyme in the biogenesis of long-chain fatty acids, and ACC β (ACC2) may control mitochondrial fatty acid oxidation. These two isoforms of ACC control the amount of fatty acids in the cells. The catalytic function of ACC α is regulated by phosphorylation (inactive) and dephosphorylation (active) of targeted serine residues and by allosteric transformation by citrate or palmitoyl-CoA, which serve as the enzyme's short-term regulatory mechanism. The gene encoding ACC α maps to human chromosome 17q12 and encodes a form of ACC, which is the major ACC in lipogenic tissues. The catalytic core of ACC β is homologous to that of the ACC α , except for an additional peptide of about 150 amino acids at the N-terminus.

CHROMOSOMAL LOCATION

Genetic locus: ACACA (human) mapping to 17q12.

SOURCE

ACC α (D-5) is a mouse monoclonal antibody raised against amino acids 1-76 mapping at the N-terminus of ACC α of human origin.

PRODUCT

Each vial contains 200 μ g IgG_{2b} kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

ACC α (D-5) is available conjugated to agarose (sc-137104 AC), 500 μ g/0.25 ml agarose in 1 ml, for IP; to HRP (sc-137104 HRP), 200 μ g/ml, for WB, IHC(P) and ELISA; to either phycoerythrin (sc-137104 PE), fluorescein (sc-137104 FITC), Alexa Fluor[®] 488 (sc-137104 AF488), Alexa Fluor[®] 546 (sc-137104 AF546), Alexa Fluor[®] 594 (sc-137104 AF594) or Alexa Fluor[®] 647 (sc-137104 AF647), 200 μ g/ml, for WB (RGB), IF, IHC(P) and FCM; and to either Alexa Fluor[®] 680 (sc-137104 AF680) or Alexa Fluor[®] 790 (sc-137104 AF790), 200 μ g/ml, for Near-Infrared (NIR) WB, IF and FCM.

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APPLICATIONS

ACC α (D-5) is recommended for detection of ACC α of 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 ACC α siRNA (h): sc-40312, ACC α shRNA Plasmid (h): sc-40312-SH and ACC α shRNA (h) Lentiviral Particles: sc-40312-V.

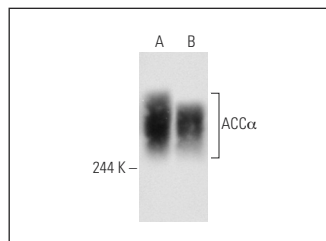
Molecular Weight of ACC α : 265 kDa.

Positive Controls: DU 145 cell lysate: sc-2268 or Jurkat whole cell lysate: sc-2204.

STORAGE

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

DATA



ACC α (D-5): sc-137104. Western blot analysis of ACC α expression in DU 145 (A) and Jurkat (B) whole cell lysates.

SELECT PRODUCT CITATIONS

- Liao, C.C., et al. 2013. Prevention of diet-induced hyperlipidemia and obesity by caffeic acid in C57BL/6 mice through regulation of hepatic lipogenesis gene expression. *J. Agric. Food Chem.* 61: 11082-11088.
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- Huang, L., et al. 2018. Inhibition of protein arginine methyltransferase 5 enhances hepatic mitochondrial biogenesis. *J. Biol. Chem.* 293: 10884-10894.
- Song, H.M., et al. 2018. Carnosic acid protects mice from high-fat diet-induced NAFLD by regulating MARCKS. *Int. J. Mol. Med.* 42: 193-207.
- Balusamy, S.R., et al. 2018. Anti-proliferative activity of *Origanum vulgare* inhibited lipogenesis and induced mitochondrial mediated apoptosis in human stomach cancer cell lines. *Biomed. Pharmacother.* 108: 1835-1844.
- Ferretti, A.C., et al. 2019. Metformin and glucose starvation decrease the migratory ability of hepatocellular carcinoma cells: targeting AMPK activation to control migration. *Sci. Rep.* 9: 2815.
- Li, R., et al. 2019. Cardioprotective roles of Sestrin1 and Sestrin2 against doxorubicin cardiotoxicity. *Am. J. Physiol. Heart Circ. Physiol.* 317: H39-H48.
- Wang, X., et al. 2019. MDG-1, an *Ophiopogon* polysaccharide, restrains process of non-alcoholic fatty liver disease via modulating the gut-liver axis. *Int. J. Biol. Macromol.* 141: 1013-1021.

RESEARCH USE

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