SANTA CRUZ BIOTECHNOLOGY, INC.

CPT1-C (S-17): sc-139482

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUNDBACKGROUND

The mitochondrial β-oxidation of long-chain fatty acids is initiated by the sequential action of CPT (carnitine palmitoyltransferase) I and II, together with carnitine carrier. CPTI catalyzes the first reaction in the transport of long-chain fatty acids from the cytoplasm to mitochondria, a rate-limiting step in β-oxidation. CPT1-C (carnitine palmitoyltransferase 1C), also known as CATL1, CPT1P, CPTIC or CPTI-B, is an 803 amino acid multi-pass membrane protein involved in lipid metabolism. Expressed primarily in testis and brain, CPT1-C belongs to the carnitine/choline acetyltransferase family and catalyzes the conversion of palmitoyl-CoA and L-Carnitine to CoA and L-palmitoylcarnitine. CPT1-C exists as three alternatively spliced isoforms that are encoded by a gene that maps to human chromosome 19q13.33.

REFERENCES

1. Price, N., et al. 2002. A novel brain-expressed protein related to carnitine palmitoyltransferase I. Genomics 80: 433-442.
2. Bonnefont, J.P., et al. 2004. Carnitine palmitoyltransferases 1 and 2: biochemical, molecular and medical aspects. Mol. Aspects Med. 25: 495-520.
3. Wolfgang, M.J., et al. 2006. The role of hypothalamic malonyl-CoA in energy homeostasis. J. Biol. Chem. 281: 37265-37269.
4. Wolfgang, M.J., et al. 2006. The brain-specific carnitine palmitoyltransferase-1c regulates energy homeostasis. Proc. Natl. Acad. Sci. USA 103: 7282-7287.
5. Online Mendelian Inheritance in Man, OMIM™. 2006. Johns Hopkins University, Baltimore, MD. MIM Number: 608846. World Wide Web URL: http://www.ncbi.nlm.nih.gov/omim/
6. Sierra, A.Y., et al. 2008. CPT1c is localized in endoplasmic reticulum of neurons and has carnitine palmitoyltransferase activity. J. Biol. Chem. 283: 6878-6885.

CHROMOSOMAL LOCATION

Genetic locus: Cpt1c (mouse) mapping to 7 B4.

SOURCE

CPT1-C (S-17) is an affinity purified rabbit polyclonal antibody raised against a peptide mapping within an N-terminal cytoplasmic domain of CPT1-C of mouse origin.

PRODUCT

Each vial contains 100 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-139482 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

STORAGE

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

APPLICATIONS

CPT1-C (S-17) is recommended for detection of CPT1-C of mouse and rat origin by Western Blotting (starting dilution 1:100, dilution range 1:50-1:500), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:25, dilution range 1:25-1:250) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000); non cross-reactive with CPTI or CPTI-M.

Suitable for use as control antibody for CPT1-C siRNA (m): sc-142550, CPT1-C shRNA Plasmid (m): sc-142550-SH and CPT1-C shRNA (m) Lentiviral Particles: sc-142550-V.

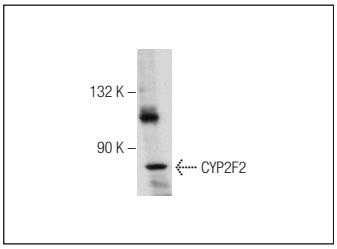
Molecular Weight of CPT1-C isoforms 1/2/3: 91/90/81 kDa.

Positive Controls: mouse liver extract: sc-2256.

RECOMMENDED SECONDARY REAGENTS

To ensure optimal results, the following support (secondary) reagents are recommended: 1) Western Blotting: use goat anti-rabbit IgG-HRP: sc-2004 (dilution range: 1:2000-1:100,000) or Cruz Marker™ compatible goat anti-rabbit IgG-HRP: sc-2030 (dilution range: 1:2000-1:5000), Cruz Marker™ Molecular Weight Standards: sc-2035, TBS Blotto A Blocking Reagent: sc-2333 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 goat anti-rabbit IgG-FITC: sc-2012 (dilution range: 1:100-1:400) or goat anti-rabbit IgG-TR: sc-2780 (dilution range: 1:100-1:400) with UltraCruz™ Mounting Medium: sc-24941.

DATA



CPT1-C (S-17): sc-139482. Western blot analysis of CPT1-C expression in mouse liver tissue extract.

SELECT PRODUCT CITATIONS

1. Abbott, M.J., et al. 2011. AMPKα2 deficiency uncovers time dependency in the regulation of contraction-induced palmitate and glucose uptake in mouse muscle. J. Appl. Physiol. 111: 125-134.
2. Caffin, F., et al. 2013. Altered skeletal muscle mitochondrial biogenesis but improved endurance capacity in trained OPA1-deficient mice. J. Physiol. 591: 6017-6037.
3. Abbott, M.J. and Turcotte, L.P. 2014. AMPK-α2 is involved in exercise training-induced adaptations in Insulin-stimulated metabolism in skeletal muscle following high-fat diet. J. Appl. Physiol. 117: 869-879.

RESEARCH USE

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