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ACSS2 Antibody (Center) Blocking Peptide

Synthetic peptide
Catalog # BP13231c

Specification

ACSS2 Antibody (Center) Blocking Peptide - Product Information

Primary Accession Q9NR19

ACSS2 Antibody (Center) Blocking Peptide - Additional Information

Gene ID 55902

Other Names
Acetyl-coenzyme A synthetase, cytoplasmic, Acetate--CoA ligase, Acetyl-CoA synthetase, ACS, AceCS, Acyl-CoA synthetase short-chain family member 2, Acyl-activating enzyme, ACSS2, ACAS2

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP13231c was selected from the Center region of ACSS2. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

ACSS2 Antibody (Center) Blocking Peptide - Protein Information

Name ACSS2

Synonyms ACAS2

Function
Catalyzes the synthesis of acetyl-CoA from short-chain fatty acids (PubMed:10843999, PubMed:28003429, PubMed:28552616). Acetate is the preferred substrate (PubMed:10843999, PubMed:28003429). Can also utilize propionate with a much lower affinity (By similarity). Nuclear ACSS2 promotes glucose deprivation-induced lysosomal biogenesis and autophagy, tumor cell survival and brain tumorigenesis (PubMed:28552616). Glucose

deprivation results in AMPK-mediated phosphorylation of ACSS2 leading to its translocation to the nucleus where it binds to TFEB and locally produces acetyl-CoA for histone acetylation in the promoter regions of TFEB target genes thereby activating their transcription (PubMed:28552616). The regulation of genes associated with autophagy and lysosomal activity through ACSS2 is important for brain tumorigenesis and tumor survival (PubMed:28552616). Acts as a chromatin-bound transcriptional coactivator that up-regulates histone acetylation and expression of neuronal genes (By similarity). Can be recruited to the loci of memory-related neuronal genes to maintain a local acetyl-CoA pool, providing the substrate for histone acetylation and promoting the expression of specific genes, which is essential for maintaining long-term spatial memory (By similarity).

Cellular Location
Cytoplasm, cytosol. Cytoplasm {ECO:0000250|UniProtKB:Q9QXG4}. Nucleus Note=Glucose deprivation results in its AMPK-dependent phosphorylation and subsequent nuclear translocation (PubMed:28552616). Phosphorylation at Ser-659, leads to exposure of its nuclear localization signal which is required for its interaction with KPNA1 and subsequent translocation to the nucleus (PubMed:28552616). Found in the cytoplasm in undifferentiated neurons and upon differentiation, translocates to nucleus (By similarity). {ECO:0000250|UniProtKB:Q9QXG4, ECO:0000269|PubMed:28552616}

ACSS2 Antibody (Center) Blocking Peptide - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Blocking Peptides

ACSS2 Antibody (Center) Blocking Peptide - Images

ACSS2 Antibody (Center) Blocking Peptide - Background

This gene encodes a cytosolic enzyme that catalyzes theactivation of acetate for use in lipid synthesis and energygeneration. The protein acts as a monomer and produces acetyl-CoAfrom acetate in a reaction that requires ATP. Expression of thisgene is regulated by sterol regulatory element-binding proteins,transcription factors that activate genes required for thesynthesis of cholesterol and unsaturated fatty acids. Alternativesplicing results in multiple transcript variants. [provided byRefSeq].

ACSS2 Antibody (Center) Blocking Peptide - References

Bailey, S.D., et al. Diabetes Care 33(10):2250-2253(2010)Yilmaz, S., et al. Genomics 96(1):57-65(2010)Ban, H.J., et al. BMC Genet. 11, 26 (2010) :Talmud, P.J., et al. Am. J. Hum. Genet. 85(5):628-642(2009)Yun, M., et al. J. Nucl. Med. 50(8):1222-1228(2009)