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BCKDK Antibody (N-term) Blocking Peptide
Synthetic peptide
Catalog # BP7090a

Specification

BCKDK Antibody (N-term) Blocking Peptide - Product Information

Primary Accession O14874

BCKDK Antibody (N-term) Blocking Peptide - Additional Information

Gene ID 10295

Other Names
[3-methyl-2-oxobutanoate dehydrogenase [lipoamide]] kinase, mitochondrial, Branched-chain alpha-ketoacid dehydrogenase kinase, BCKD-kinase, BCKDHKIN, BCKDK

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP7090a was selected from the N-term region of human BCKDK. 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.

BCKDK Antibody (N-term) Blocking Peptide - Protein Information

Name BCKDK {ECO:0000303|PubMed:29779826, ECO:0000312|HGNC:HGNC:16902}

Function
Serine/threonine-protein kinase component of macronutrients metabolism. Forms a functional kinase and phosphatase pair with PPM1K, serving as a metabolic regulatory node that coordinates branched-chain amino acids (BCAAs) with glucose and lipid metabolism via two distinct phosphoprotein targets: mitochondrial BCKDHA subunit of the branched- chain alpha-ketoacid dehydrogenase (BCKDH) complex and cytosolic ACLY, a lipogenic enzyme of Krebs cycle (PubMed:24449431, PubMed:29779826, PubMed:37558654). Phosphorylates and inactivates mitochondrial BCKDH complex a multisubunit complex consisting of three multimeric components each involved in different steps of BCAA catabolism: E1 composed of BCKDHA and BCKDHB, E2 core composed of DBT monomers, and E3 composed of DLD

monomers. Associates with the E2 component of BCKDH complex and phosphorylates BCKDHA on Ser-337, leading to conformational changes that interrupt substrate channeling between E1 and E2 and inactivates the BCKDH complex (PubMed:29779826, PubMed:37558654). Phosphorylates ACLY on Ser-455 in response to changes in cellular carbohydrate abundance such as occurs during fasting to feeding metabolic transition. Refeeding stimulates MLXIPL/ChREBP transcription factor, leading to increased BCKDK to PPM1K expression ratio, phosphorylation and activation of ACLY that ultimately results in the generation of malonyl-CoA and oxaloacetate immediate substrates of de novo lipogenesis and glucogenesis, respectively (PubMed:29779826). Recognizes phosphosites having SxxE/D canonical motif (PubMed:29779826).

Cellular Location
Mitochondrion matrix {ECO:0000250|UniProtKB:Q00972, ECO:0000305|PubMed:24449431}
Note=Detected in the cytosolic compartment of liver cells {ECO:0000250|UniProtKB:Q00972}

Tissue Location
Ubiquitous.

BCKDK Antibody (N-term) Blocking Peptide - Protocols

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

- Blocking Peptides

BCKDK Antibody (N-term) Blocking Peptide - Images

BCKDK Antibody (N-term) Blocking Peptide - Background

The second major step in the catabolism of the branched-chain amino acids, isoleucine, leucine, and valine, is irreversibly catalyzed by the branched-chain alpha-keto acid dehydrogenase complex (BCKD), an inner-mitochondrial enzyme complex composed of 3 catalytic components: a branched-chain alpha-keto acid decarboxylase (E1), a dihydrolipoyl transacylase (E2), and a dihydrolipoamide dehydrogenase (E3). The complex also contains 2 enzymes that regulated the state of activity of the BCKD complex: a kinase (BCKDK), and a phosphorylase. The ubiquitiously expressed kinase contains 1 histidine kinase domain. Maple syrup urine disease (MSUD) is a pathology secondary to an enzyme defect in the catabolic pathway of leucine, isoleucine, and valine. Accumulation of these amino acids and their corresponding keto acids results in encephalopathy and progressive neurodegeneration in infants not treated for MSUD.