

PCK1 Antibody (C-term) Blocking Peptide

Synthetic peptide Catalog # BP8093b

Specification

PCK1 Antibody (C-term) Blocking Peptide - Product Information

Primary Accession

P35558

PCK1 Antibody (C-term) Blocking Peptide - Additional Information

Gene ID 5105

Other Names

Phosphoenolpyruvate carboxykinase, cytosolic [GTP], PEPCK-C, PCK1, PEPCK1

Target/Specificity

The synthetic peptide sequence is selected from aa 606~622 of human PCK1.

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.

PCK1 Antibody (C-term) Blocking Peptide - Protein Information

Name PCK1 {ECO:0000303|PubMed:8490617, ECO:0000312|HGNC:HGNC:8724}

Function

Cytosolic phosphoenolpyruvate carboxykinase that catalyzes the reversible decarboxylation and phosphorylation of oxaloacetate (OAA) and acts as the rate-limiting enzyme in gluconeogenesis (PubMed:30193097, PubMed:24863970, PubMed:26971250, PubMed:28216384). Regulates cataplerosis and anaplerosis, the processes that control the levels of metabolic intermediates in the citric acid cycle (PubMed:30193097, PubMed:24863970, PubMed:26971250, PubMed:26971250, PubMed:24863970, PubMed:<ahref="http://www.uniprot.org/citations/26971250" target="_blank">26971250, PubMed:<ahref="http://www.uniprot.org/citations/28216384" target="_blank">28216384). At low glucose levels, it catalyzes the cataplerotic conversion of oxaloacetate to phosphoenolpyruvate (PEP), the rate-limiting step in the metabolic pathway that produces glucose from lactate and other precursors derived from the citric acid cycle (PubMed:<a



href="http://www.uniprot.org/citations/30193097" target=" blank">30193097). At high glucose levels, it catalyzes the anaplerotic conversion of phosphoenolpyruvate to oxaloacetate (PubMed:30193097). Acts as a regulator of formation and maintenance of memory CD8(+) T-cells: up-regulated in these cells, where it generates phosphoenolpyruvate, via gluconeogenesis (By similarity). The resultant phosphoenolpyruvate flows to glycogen and pentose phosphate pathway, which is essential for memory CD8(+) T-cells homeostasis (By similarity). In addition to the phosphoenolpyruvate carboxykinase activity, also acts as a protein kinase when phosphorylated at Ser-90: phosphorylation at Ser-90 by AKT1 reduces the binding affinity to oxaloacetate and promotes an atypical serine protein kinase activity using GTP as donor (PubMed: 32322062). The protein kinase activity regulates lipogenesis: upon phosphorylation at Ser-90, translocates to the endoplasmic reticulum and catalyzes phosphorylation of INSIG proteins (INSIG1 and INSIG2), thereby disrupting the interaction between INSIG proteins and SCAP and promoting nuclear translocation of SREBP proteins (SREBF1/SREBP1 or SREBF2/SREBP2) and subsequent transcription of downstream lipogenesis- related genes (PubMed:32322062).

Cellular Location

Cytoplasm, cytosol. Endoplasmic reticulum Note=Phosphorylation at Ser-90 promotes translocation to the endoplasmic reticulum.

Tissue Location

Major sites of expression are liver, kidney and adipocytes.

PCK1 Antibody (C-term) Blocking Peptide - Protocols

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

• Blocking Peptides

PCK1 Antibody (C-term) Blocking Peptide - Images

PCK1 Antibody (C-term) Blocking Peptide - Background

This gene is a main control point for the regulation of gluconeogenesis. The cytosolic enzyme encoded by this gene, along with GTP, catalyzes the formation of phosphoenolpyruvate from oxaloacetate, with the release of carbon dioxide and GDP. The expression of this gene can be regulated by insulin, glucocorticoids, glucagon, cAMP, and diet. A mitochondrial isozyme of the encoded protein also has been characterized.

PCK1 Antibody (C-term) Blocking Peptide - References

Dunten, P., et al., J. Mol. Biol. 316(2):257-264 (2002). Strausberg, R.L., et al., Proc. Natl. Acad. Sci. U.S.A. 99(26):16899-16903 (2002). Deloukas, P., et al., Nature 414(6866):865-871 (2001). O'Brien, R.M., et al., Biochim. Biophys. Acta 1264(3):284-288 (1995). Ting, C.N., et al., Genomics 16(3):698-706 (1993).