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PFKL Antibody (C-term L684) Blocking peptide

Synthetic peptide
Catalog # BP8136b

Specification

PFKL Antibody (C-term L684) Blocking peptide - Product Information

Primary Accession P17858

PFKL Antibody (C-term L684) Blocking peptide - Additional Information

Gene ID 5211

Other Names
ATP-dependent 6-phosphofructokinase, liver type {ECO:0000255|HAMAP-Rule:MF_03184}, ATP-PFK {ECO:0000255|HAMAP-Rule:MF_03184}, PFK-L, 27111 {ECO:0000255|HAMAP-Rule:MF_03184}, 6-phosphofructokinase type B, Phosphofructo-1-kinase isozyme B, PFK-B, Phosphohexokinase {ECO:0000255|HAMAP-Rule:MF_03184}, PFKL

Target/Specificity
The synthetic peptide sequence is selected from aa 684~699 of human PFKL.

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.

PFKL Antibody (C-term L684) Blocking peptide - Protein Information

Name PFKL (HGNC:8876)

Function
Catalyzes the phosphorylation of D-fructose 6-phosphate to fructose 1,6-bisphosphate by ATP, the first committing step of glycolysis (PubMed:22923583). Negatively regulates the phagocyte oxidative burst in response to bacterial infection by controlling cellular NADPH biosynthesis and NADPH oxidase-derived reactive oxygen species. Upon macrophage activation, drives the metabolic switch toward glycolysis, thus preventing glucose turnover that produces NADPH via pentose phosphate pathway (By similarity).

Cellular Location
Cytoplasm {ECO:0000255|HAMAP-Rule:MF_03184}.

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PFKL Antibody (C-term L684) Blocking peptide - Protocols

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

- Blocking Peptides

PFKL Antibody (C-term L684) Blocking peptide - Images

PFKL Antibody (C-term L684) Blocking peptide - Background

Phosphofructokinase (PFK), a major regulatory enzyme in all cells of the body, catalyzes the metabolism of sugar, and thereby is pivotal in the production of energy to maintain normal cell function. In human there are three structural loci controlling PFK: M (muscle), L (liver), and P (platelet) type subunits, which are variably expressed in different tissues; human diploid fibroblasts and leukocytes express all three genes. PFK, a tetramer formed by the random association of the products of two separate gene loci to form the five possible tetramers. PFKs of muscle and liver are homotetramers of the M and L subunits, respectively. Red cells have all five isozymes: M4, M3L, M2L2, ML3, and L4. PFK is an allosteric enzyme activated by ADP, AMP, or fructose bisphosphate and inhibited by ATP or citrate. PFK catalyzes the key controlling step of glycolytic pathway. PFK deficiency can present as mild to life-threatening episodic illness. A hallmark sign of this disease is intermittent dark urine, with the color of the urine ranging from orange to dark coffee-brown, which commonly develops following strenuous exercise. The mean red cell PFK is elevated in persons with Down syndrome.

PFKL Antibody (C-term L684) Blocking peptide - References

Strausberg, R.L., et al., Proc. Natl. Acad. Sci. U.S.A. 99(26):16899-16903 (2002).Hattori, M., et al., Nature 405(6784):311-319 (2000).Elson, A., et al., Genomics 7(1):47-56 (1990).Levanon, D., et al., DNA 8(10):733-743 (1989).