10320 Camino Santa Fe, Suite G
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Ketohexokinase (KHK) Antibody (C-term) Blocking peptide

Synthetic peptide
Catalog # BP7069b

Specification

Ketohexokinase (KHK) Antibody (C-term) Blocking peptide - Product Information

Primary Accession P50053

Ketohexokinase (KHK) Antibody (C-term) Blocking peptide - Additional Information

Gene ID 3795

Other Names
Ketohexokinase, Hepatic fructokinase, KHK

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP7069b was selected from the C-term region of human KHK. 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.

Ketohexokinase (KHK) Antibody (C-term) Blocking peptide - Protein Information

Name KHK (HGNC:6315)

Function
Catalyzes the phosphorylation of the ketose sugar fructose to fructose-1-phosphate.

Tissue Location
Most abundant in liver, kidney, gut, spleen and pancreas. Low levels also found in adrenal, muscle, brain and eye

Ketohexokinase (KHK) Antibody (C-term) Blocking peptide - Protocols

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

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- Blocking Peptides

Ketohexokinase (KHK) Antibody (C-term) Blocking peptide - Images

Ketohexokinase (KHK) Antibody (C-term) Blocking peptide - Background

Ketohexokinase (KHK), or fructokinase, catalyzes conversion of fructose to fructose-1-phosphate. Splice variant 1 is the highly active form found in liver, renal cortex, and small intestine, while splice variant 2 is the lower activity form found in most other tissues. KHK, like glucokinase (GCK) and glucokinase regulator (GCKR), is present in both liver and pancreatic islets. The inhibition of GCK by GCKR is blocked by binding of fructose-1-phosphate to GCKR. The chromosomal proximity of the metabolically connected GCKR and KHK genes has a genetic linkage in type 2 diabetes. Fructosuria, or hepatic fructokinase deficiency, is a benign, asymptomatic defect of intermediary metabolism associated with heterozygosity for G50R and A43T mutations in KHK.