

**Ketohexokinase (KHK) Antibody (C-term) Blocking peptide**  
**Synthetic peptide**  
**Catalog # BP7069b****Specification**

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**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](/product/products/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.

- [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.