

**HK2 (Hexokinase II) Antibody (Center) Blocking peptide**  
**Synthetic peptide**  
**Catalog # BP8140f****Specification**

---

**HK2 (Hexokinase II) Antibody (Center) Blocking peptide - Product Information**

Primary Accession [P52789](#)  
Other Accession [NP\\_000180](#)

**HK2 (Hexokinase II) Antibody (Center) Blocking peptide - Additional Information**

**Gene ID** 3099

**Other Names**

Hexokinase-2, Hexokinase type II, HK II, Muscle form hexokinase, HK2

**Target/Specificity**

The synthetic peptide sequence used to generate the antibody [AP8140f](/product/products/AP8140f) was selected from the Center region of human HK2. 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.

**HK2 (Hexokinase II) Antibody (Center) Blocking peptide - Protein Information**

**Name** HK2 ([HGNC:4923](#))

**Function**

Catalyzes the phosphorylation of hexose, such as D-glucose and D-fructose, to hexose 6-phosphate (D-glucose 6-phosphate and D-fructose 6-phosphate, respectively) (PubMed: [23185017](http://www.uniprot.org/citations/23185017), PubMed: [26985301](http://www.uniprot.org/citations/26985301), PubMed: [29298880](http://www.uniprot.org/citations/29298880)). Mediates the initial step of glycolysis by catalyzing phosphorylation of D-glucose to D-glucose 6-phosphate (PubMed: [29298880](http://www.uniprot.org/citations/29298880)). Plays a key role in maintaining the integrity of the outer mitochondrial membrane by preventing the release of apoptogenic molecules from the intermembrane space and subsequent apoptosis (PubMed: [18350175](http://www.uniprot.org/citations/18350175)).

**Cellular Location**

Mitochondrial outer membrane; Peripheral membrane protein. Cytoplasm, cytosol Note=The mitochondrial-binding peptide (MBP) region promotes association with the mitochondrial outer membrane (PubMed:29298880) The interaction with the mitochondrial outer membrane via the mitochondrial-binding peptide (MBP) region promotes higher stability of the protein (PubMed:29298880). Release from the mitochondrial outer membrane into the cytosol induces permeability transition pore (PTP) opening and apoptosis (PubMed:18350175).

**Tissue Location**

Predominant hexokinase isozyme expressed in insulin-responsive tissues such as skeletal muscle

**HK2 (Hexokinase II) Antibody (Center) Blocking peptide - Protocols**

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

- [Blocking Peptides](#)

**HK2 (Hexokinase II) Antibody (Center) Blocking peptide - Images****HK2 (Hexokinase II) Antibody (Center) Blocking peptide - Background**

In vertebrates there are four major glucose-phosphorylating isoenzymes, designated hexokinase I, II, III, and IV. Hexokinase is an allosteric enzyme inhibited by its product GLC-6-P. Hexokinase activity is involved in the first step in several metabolic pathways. HK3 is bound to the outer mitochondrial membrane. Its hydrophobic N-terminal sequence may be involved in membrane binding. It is the predominant hexokinase isozyme expressed in insulin-responsive tissues such as skeletal muscle. The N- and C-terminal halves of this hexokinase show extensive sequence similarity to each other. The catalytic activity is associated with the C-terminus while regulatory function is associated with the N-terminus. Although found in NIDDM patients, genetic variations of HK2 do not contribute to the disease.

**HK2 (Hexokinase II) Antibody (Center) Blocking peptide - References**

Lehto, M., et al., Diabetologia 38(12):1466-1474 (1995).Vidal-Puig, A., et al., Diabetes 44(3):340-346 (1995).Laakso, M., et al., Diabetes 44(3):330-334 (1995).Echwald, S.M., et al., Diabetes 44(3):347-353 (1995).Shinohara, Y., et al., Cancer Lett. 82(1):27-32 (1994).