

# Ketohexokinase (KHK) Antibody (N-term)

Purified Rabbit Polyclonal Antibody (Pab) Catalog # AP7069a

## **Product Information**

Application	WB, E
Primary Accession	<u>P50053</u>
Other Accession	<u>Q02974</u> , <u>P97328</u>
Reactivity	Human, Mouse
Predicted	Rat
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Clone Names	RB05409
Calculated MW	32523
Antigen Region	18-46

## **Additional Information**

Gene ID	3795
Other Names	Ketohexokinase, Hepatic fructokinase, KHK
Target/Specificity	This Ketohexokinase (KHK) antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 18-46 amino acids from the N-terminal region of human Ketohexokinase (KHK).
Dilution	WB~~1:1000 E~~Use at an assay dependent concentration.
Format	Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.
Storage	Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	Ketohexokinase (KHK) Antibody (N-term) is for research use only and not for use in diagnostic or therapeutic procedures.

#### **Protein Information**

Name	KHK ( <u>HGNC:6315</u> )
Function	Catalyzes the phosphorylation of the ketose sugar fructose to fructose-1-phosphate.

# 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.

## References

Strausberg, R.L., et al., Proc. Natl. Acad. Sci. U.S.A. 99(26):16899-16903 (2002). Hayward, B.E., et al., Eur. J. Biochem. 257(1):85-91 (1998). Bonthron, D.T., et al., Hum. Mol. Genet. 3(9):1627-1631 (1994).

#### Images



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