

GCK Monoclonal Antibody

Catalog No: YM0302

Reactivity: Human

Applications: WB;ELISA

Target: GCK

Fields: >>Glycolysis / Gluconeogenesis;>>Galactose metabolism;>>Starch and

sucrose metabolism;>>Amino sugar and nucleotide sugar

metabolism;>>Neomycin, kanamycin and gentamicin biosynthesis;>>Metabolic pathways;>>Carbon metabolism;>>Biosynthesis of nucleotide sugars;>>Insulin signaling pathway;>>Insulin secretion;>>Prolactin signaling pathway;>>Glucagon signaling pathway;>>Type II diabetes mellitus;>>Maturity onset diabetes of the

young;>>Central carbon metabolism in cancer

Gene Name: GCK

Protein Name: Glucokinase

P35557

P52792

Human Gene Id: 2645

Human Swiss Prot

No:

Mouse Swiss Prot

No:

Immunogen: Purified recombinant fragment of human GCK expressed in E. Coli.

Specificity: GCK Monoclonal Antibody detects endogenous levels of GCK protein.

Formulation : Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.

Source: Monoclonal, Mouse

Dilution: WB 1:500 - 1:2000. ELISA: 1:10000. Not yet tested in other applications.

Purification : Affinity purification

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Storage Stability: -15°C to -25°C/1 year(Do not lower than -25°C)

Molecularweight: 52kD

Cell Pathway: Glycolysis / Gluconeogenesis; Galactose metabolism; Starch and sucrose

metabolism; Amino sugar and nucleotide sugar

metabolism;Insulin_Receptor;Type II diabetes mellitus;Maturity onset diabetes of

the yo

P References : 1. Mol Endocrinol. 2009 Dec;23(12):1983-9.

2. Int J Mol Med. 2009 Aug;24(2):233-46.

Background : Hexokinases phosphorylate glucose to produce glucose-6-phosphate, the first

step in most glucose metabolism pathways. Alternative splicing of this gene results in three tissue-specific forms of glucokinase, one found in pancreatic islet beta cells and two found in liver. The protein localizes to the outer membrane of mitochondria. In contrast to other forms of hexokinase, this enzyme is not

inhibited by its product glucose-6-phosphate but remains active while glucose is abundant. Mutations in this gene have been associated with non-insulin

dependent diabetes mellitus (NIDDM), maturity onset diabetes of the young, type 2 (MODY2) and persistent hyperinsulinemic hypoglycemia of infancy (PHHI).

[provided by RefSeq, Apr 2009],

Function: catalytic activity:ATP + D-glucose = ADP + D-glucose

6-phosphate., disease: Defects in GCK are the cause of familial hyperinsulinemic hypoglycemia type 3 (HHF3) [MIM:602485]. HHF is the most common cause of persistent hypoglycemia in infancy. Unless early and aggressive intervention is undertaken, brain damage from recurrent episodes of hypoglycemia may occur.. disease: Defects in GCK are the cause of maturity onset diabetes of the

young type 2 (MODY2) [MIM:125851]; also shortened MODY-2. MODY [MIM:606391] is a form of diabetes mellitus characterized by autosomal dominant

transmission and early age of onset. Mutations in GCK result in mild chronic hyperglycemia due to reduced pancreatic beta cell responsiveness to glucose, decreased net accumulation of hepatic glycogen and increased hepatic gluconeogenesis following meals.,enzyme regulation: The use of alternative

promoters apparently enables

Subcellular Location:

Cytoplasm . Nucleus . Mitochondrion . Under low glucose concentrations, GCK associates with GCKR and the inactive complex is recruited to the hepatocyte

nucleus...

Expression: Lung, Pancreas, Placenta,

Sort : 6503

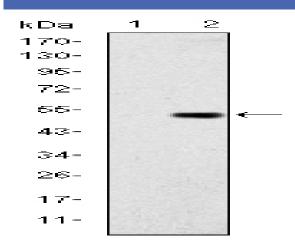
No4: 1



Host: Mouse

Modifications: Unmodified

Products Images



Western Blot analysis using GCK Monoclonal Antibody against HEK293 (1) and GCK-hlgGFc transfected HEK293 (2) cell lysate.

