

## GCK Rabbit pAb

CatalogNo: YT1872

### Key Features

#### Host Species

- Rabbit

#### Reactivity

- Human, Mouse, Rat

#### Applications

- WB, ELISA

#### MW

- 55kD (Observed)

#### Isotype

- IgG

### Recommended Dilution Ratios

**WB 1:500-1:2000**

**ELISA 1:10000**

**Not yet tested in other applications.**

### Storage

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

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

### Basic Information

**Clonality** Polyclonal

### Immunogen Information

**Immunogen** The antiserum was produced against synthesized peptide derived from human GCK. AA range:43-92

**Specificity** GCK Polyclonal Antibody detects endogenous levels of GCK protein.

### Target Information

**Gene name** GCK

**Protein Name** Glucokinase

Organism	Gene ID	UniProt ID
Human	<a href="#">2645;</a>	<a href="#">P35557;</a>
Mouse	<a href="#">103988;</a>	<a href="#">P52792;</a>
Rat	<a href="#">24385;</a>	<a href="#">P17712;</a>

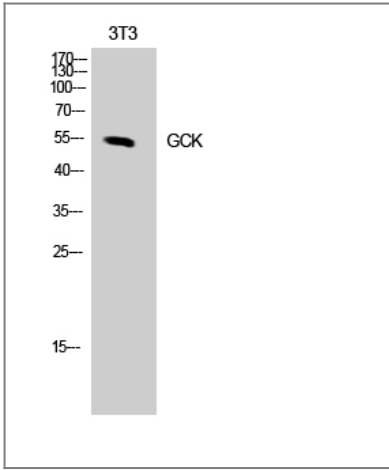
**Cellular Localization** Cytoplasm . Nucleus . Mitochondrion . Under low glucose concentrations, GCK associates with GCKR and the inactive complex is recruited to the hepatocyte nucleus. .

**Tissue specificity** Lung,Pancreas,Placenta,

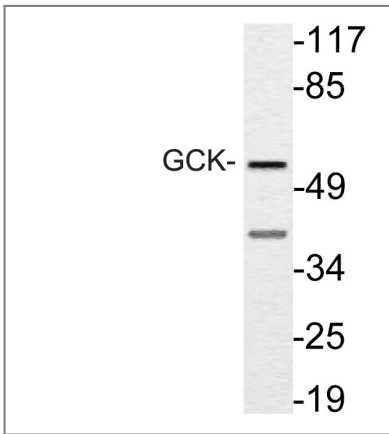
**Function** Catalytic activity:ATP + D-glucose = ADP + D-glucose 6-phosphate.,Disease:Defects in GCK are the cause of familial hyperinsulinemic hypoglycemia type 3 (HHF3) [MIM:602485]. HHF is the most common cause of persistent hypoglycemia in infancy. Unless early and aggressive intervention is undertaken, 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 the type IV hexokinase gene to be regulated by insulin in the liver and glucose in the beta cell. This may constitute an important feedback loop for maintaining glucose homeostasis.,Function:Catalyzes the initial step in utilization of glucose by the beta-cell and liver at physiological glucose concentration. Glucokinase has a high Km for glucose, and so it is effective only when glucose is abundant. The role of GCK is to provide G6P for the synthesis of glycogen. Pancreatic glucokinase plays an important role in modulating insulin secretion. Hepatic glucokinase helps to facilitate the uptake and conversion of glucose by acting as an insulin-sensitive determinant of hepatic glucose usage.,miscellaneous:In vertebrates there are four major glucose-phosphorylating isoenzymes, designated hexokinase I, II, III and IV (glucokinase).,online information:Glucokinase entry,similarity:Belongs to the hexokinase family.,tissue specificity:Pancreas (isoform 1) and liver (isoform 2 and isoform 3).,

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## Validation Data



Western Blot analysis of NIH-3T3 cells using GCK Polyclonal Antibody diluted at 1:1000



Western blot analysis of lysate from NIH/3T3 cells, using GCK antibody.

## Contact information

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**GCK Rabbit pAb**

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