

**GCKR Antibody (N-term)**  
**Purified Rabbit Polyclonal Antibody (Pab)**  
**Catalog # AP8143a****Specification**

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**GCKR Antibody (N-term) - Product Information**

|                   |                        |
|-------------------|------------------------|
| Application       | WB, IHC-P,E            |
| Primary Accession | <a href="#">Q14397</a> |
| Reactivity        | Human                  |
| Host              | Rabbit                 |
| Clonality         | Polyclonal             |
| Isotype           | Rabbit IgG             |
| Antigen Region    | 1-30                   |

**GCKR Antibody (N-term) - Additional Information****Gene ID** 2646**Other Names**

Glucokinase regulatory protein, GKRP, Glucokinase regulator, GCKR

**Target/Specificity**

This GCKR antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 1-30 amino acids from the N-terminal region of human GCKR.

**Dilution**

WB~~1:1000

IHC-P~~1:50~100

**Format**

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

**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**

GCKR Antibody (N-term) is for research use only and not for use in diagnostic or therapeutic procedures.

**GCKR Antibody (N-term) - Protein Information****Name** GCKR {ECO:0000303|PubMed:8589523, ECO:0000312|HGNC:HGNC:4196}

**Function** Regulates glucokinase (GCK) by forming an inactive complex with this enzyme (PubMed:[23621087](#), PubMed:[23733961](#)). Acts by promoting GCK recruitment to the nucleus, possibly to provide a reserve of GCK that can be quickly released in the cytoplasm after a meal

(PubMed:[10456334](#)). The affinity of GCKR for GCK is modulated by fructose metabolites: GCKR with bound fructose 6-phosphate has increased affinity for GCK, while GCKR with bound fructose 1-phosphate has strongly decreased affinity for GCK and does not inhibit GCK activity (PubMed:[23621087](#), PubMed:[23733961](#)).

#### Cellular Location

Cytoplasm. Nucleus. Mitochondrion {ECO:0000250|UniProtKB:Q07071}. Note=Under low glucose concentrations, GCKR associates with GCK and the inactive complex is recruited to the hepatocyte nucleus.

#### Tissue Location

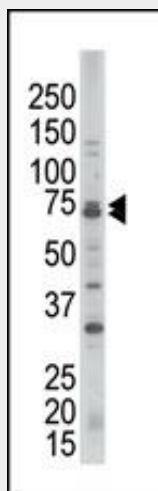
Found in liver and pancreas. Not detected in muscle, brain, heart, thymus, intestine, uterus, adipose tissue, kidney, adrenal, lung or spleen.

### GCKR Antibody (N-term) - Protocols

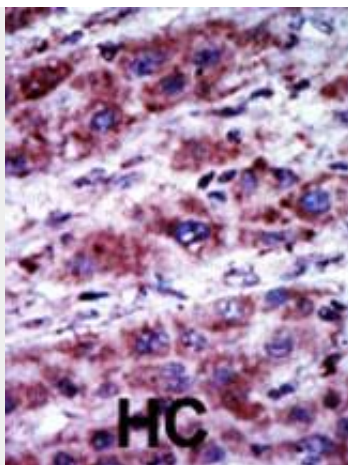
Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

### GCKR Antibody (N-term) - Images



The anti-GCKR Pab (Cat. #AP8143a) is used in Western blot to detect GCKR in A375 cell lysate.



Formalin-fixed and paraffin-embedded human cancer tissue reacted with the primary antibody, which was peroxidase-conjugated to the secondary antibody, followed by DAB staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has not been evaluated. BC = breast carcinoma; HC = hepatocarcinoma.

#### **GCKR Antibody (N-term) - Background**

GCKR belongs to the SIS (Sugar ISomerase) family of proteins. The gene product is a regulatory protein that inhibits glucokinase in liver and pancreatic islet cells by binding non-covalently to form an inactive complex with the enzyme. The GCKR gene is considered a susceptibility gene candidate for a form of maturity-onset diabetes of the young (MODY).

#### **GCKR Antibody (N-term) - References**

- Veiga-da-Cunha, M., et al., Diabetologia 46(5):704-711 (2003).
- Hayward, B.E., et al., Genomics 49(1):137-142 (1998).
- Hayward, B.E., et al., Mamm. Genome 7(6):454-458 (1996).
- Warner, J.P., et al., Mamm. Genome 6(8):532-536 (1995).
- Vaxillaire, M., et al., Diabetes 43(3):389-395 (1994).