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Rabbit Anti-phospho-Kir6.2 (Thr224) antibody

SL12181R

Product Name:	phospho-Kir6.2 (Thr224)
Chinese Name:	磷酸化ATP敏感性钾通道亚基kir6.2抗体
Alias:	Kir6.2 (phospho T224); ATP sensitive inward rectifier potassium channel 11; Beta cell inward rectifier subunit; mBIR; BIR; HHF 2; HHF2; IKATP; Inward rectifier K(+) channel Kir6.2; Inwardly rectifying potassium channel KIR6.2; IRK 11; IRK11; KCNJ11; Kir 6.2; Kir6.2; MGC133230; PHHI; Potassium channel, inwardly rectifying subfamily J member 11; Potassium inwardly rectifying channel J11; TNDM 3; TNDM3.
Organism Species:	Rabbit
Clonality:	Polyclonal
React Species:	Human,Mouse,Rat,Dog,Pig,Cow,Rabbit,Sheep,
Applications:	WB=1:500-2000ELISA=1:500-1000IHC-P=1:400-800IHC-F=1:400-800ICC=1:100-500IF=1:100-500（Paraffin sections need antigen repair） not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Molecular weight:	44kDa
Cellular localization:	The cell membrane
Form:	Lyophilized or Liquid
Concentration:	1mg/ml
immunogen:	KLH conjugated synthesised phosphopeptide derived from human Kir6.2 around the phosphorylation site of Thr224:KT(p-T)SP
Lsotype:	IgG
Purification:	affinity purified by Protein A
Storage Buffer:	0.01M TBS(pH7.4) with 1% BSA, 0.03% Proclin300 and 50% Glycerol.
Storage:	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year when kept at -20°C. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.
PubMed:	PubMed

Product Detail:	Potassium channels are present in most mammalian cells, where they participate in a wide range of physiologic responses. The protein encoded by this gene is an integral membrane protein and inward-rectifier type potassium channel. The encoded protein, which has a greater tendency to allow potassium to flow into a cell rather than out of a cell, is controlled by G-proteins and is found associated with the sulfonylurea receptor SUR. Mutations in this gene are a cause of familial persistent hyperinsulinemic hypoglycemia of infancy (PHHI), an autosomal recessive disorder characterized by unregulated insulin secretion. Defects in this gene may also contribute to autosomal dominant non-insulin-dependent diabetes mellitus type II (NIDDM), transient neonatal diabetes mellitus type 3 (TNDM3), and permanent neonatal diabetes mellitus (PNDM). Multiple alternatively spliced transcript variants that encode different protein isoforms have been described for this gene. [provided by RefSeq] **Function:** ATP-sensitive potassium (K(ATP)) channels are found in endocrine cells, neurons and both smooth and striated muscle, where they play an important role in controlling insulin secretion and vascular tone, and protect neurons under metabolic stress. Kir6.2 is a member of the inward rectifier potassium channel family, which is characterised by a greater tendency to allow potassium flow into the cell rather than out of it. It associates with the sulphonylurea receptor SUR1/ABCC8 to form a subfamily of K(ATP) channels that, when mutated or misregulated, are associated with forms of hyperinsulinemic hypoglycemia, neonatal diabetes, or pre-disposition to type 2 diabetes mellitus. **Subunit:** Interacts with ABCC8/SUR. Interacts with ABCC9/SUR2. **Subcellular Location:** Cell Membrane; Multi-pass membrane protein **DISEASE:** Defects in KCNJ11 are the cause of familial hyperinsulinemic hypoglycemia type 2 (HHF2) [MIM:601820]; also known as persistent hyperinsulinemic hypoglycemia of infancy (PPHI) or congenital hyperinsulinism. HHF is the most common cause of persistent hypoglycemia in infancy and is due to defective negative feedback regulation of insulin secretion by low glucose levels. It causes nesidioblastosis, a diffuse abnormality of the pancreas in which there is extensive, often disorganized formation of new islets. Unless early and aggressive intervention is undertaken, brain damage from recurrent episodes of hypoglycemia may occur. Defects in KCNJ11 are a cause of diabetes mellitus permanent neonatal (PNDM) [MIM:606176]. PNDM is a rare form of diabetes distinct from childhood-onset autoimmune diabetes mellitus type 1. It is characterized by insulin-requiring hyperglycemia that is diagnosed within the first months of life. Permanent neonatal diabetes requires lifelong therapy. Defects in KCNJ11 are the cause of transient neonatal diabetes mellitus type 3 (TNDM3) [MIM:610582]. Neonatal diabetes mellitus, defined as insulin-requiring

hyperglycemia within the first month of life, is a rare entity. In about half of the neonates, diabetes is transient and resolves at a median age of 3 months, whereas the rest have a permanent form of diabetes. In a significant number of patients with transient neonatal diabetes mellitus, diabetes type 2 appears later in life. The onset and severity of TNDM3 is variable with childhood-onset diabetes, gestational diabetes or adult-onset diabetes described.
Note=Defects in KCNJ11 may contribute to non-insulin-dependent diabetes mellitus (NIDDM), also known as diabetes mellitus type 2.

Similarity:
Belongs to the inward rectifier-type potassium channel (TC 1.A.2.1) family. KCNJ11 subfamily.

SWISS:
Q14654

Gene ID:
3767

Database links:

Entrez Gene: 3767Human

Entrez Gene: 16514Mouse

Omim: 600937Human

SwissProt: Q14654Human

SwissProt: Q61743Mouse

Unigene: 248141Human

Unigene: 333863Mouse

Important Note:
This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.