Product name:	KIR6.2 (phospho Thr224) Rabbit Polyclonal Antibody
Cat number:	ABN04923
Conjugate:	Unconjugated
Size:	100µL
Clone:	Polyclonal
Concentration:	1mg/ml
Host:	Rabbit
Isotype:	IgG
Immunogen:	The antiserum was produced against synthesized peptide derived from human Kir6.2 around the phosphorylation site of Thr224. AA range:190-239
Reactivity:	Human,Mouse,Rat
Applications:	WB 1:500-1:2000,IHC 1:100-1:300,ICC/IF 1:200-1:1000,ELISA 1:5000-1:10000
Molecular Weight:	40kDa
Purification:	Affinity purification
Form:	Liquid
Buffer:	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% New type preservative N.
Storage:	Store at 4°C short term. Aliquot and store at -20°C for 12 months. Avoid freeze/thaw cycles.

Background:

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 transdisease:Defects in KCNJ11 are a cause of permanent neonatal diabetes mellitus (PNDM) [MIM:606176]. PNDM is a rare form of diabetes characterized by insulin-requiring hyperglycemia that is diagnosed within the first months of life.,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 hyperinsulinism. HHF2 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.,disease: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.,disease:Defects in KCNJ11 may contribute to non-insulin-dependent diabetes mellitus (NIDDM), also known as diabetes mellitus type 2.,function:This receptor is controlled by G proteins. Inward rectifier potassium channels are characterized by a greater tendency to allow potassium to flow into the cell rather than out of it. Their voltage dependence is regulated by the concentration of extracellular potassium; as external potassium is raised, the voltage range of the channel opening shifts to more positive voltages. The inward rectification is mainly due to the blockage of outward current by internal magnesium. Can be blocked by extracellular barium.,similarity:Belongs to the inward rectifier-type potassium channel family.,subunit:Associates with ABCC8/SUR.,