



KIR6.2 rabbit pAb

Cat#: orb765551 (Manual)

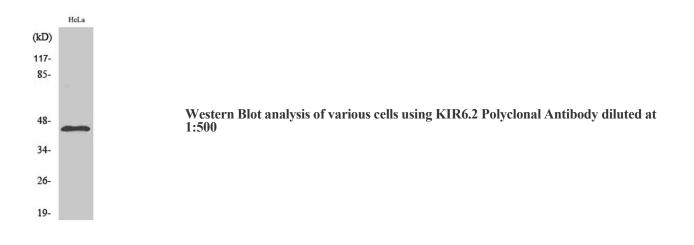
For research use only. Not intended for diagnostic use.

Product Name	KIR6.2 rabbit pAb
Host species	Rabbit
Applications	WB;IHC;IF;ELISA
Species Cross-Reactivity	Human;Mouse;Rat
Recommended dilutions	Western Blot: 1/500 - 1/2000. Immunohistochemistry: 1/100 - 1/300. Immunofluorescence: 1/200 - 1/1000. ELISA: 1/10000. Not yet tested in other applications.
Immunogen	The antiserum was produced against synthesized peptide derived from human Kir6.2. AA range:190-239
Specificity	KIR6.2 Polyclonal Antibody detects endogenous levels of KIR6.2 protein.
Formulation	L: 11 DDG (: 500/ 1 10.50/ DGA 10.020/ 1
rormulation	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide
Storage	Store at -20°C. Avoid repeated freeze-thaw cycles.
	azide
Storage	azide Store at -20°C. Avoid repeated freeze-thaw cycles.
Storage Protein Name	azide Store at -20°C. Avoid repeated freeze-thaw cycles. ATP-sensitive inward rectifier potassium channel 11
Storage Protein Name Gene Name	azide Store at -20°C. Avoid repeated freeze-thaw cycles. ATP-sensitive inward rectifier potassium channel 11 KCNJ11



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Concentration	1 mg/ml
Observed band	40kD
Human Gene ID	3767
Human Swiss-Prot Number	Q14654
Alternative Names	KCNJ11; ATP-sensitive inward rectifier potassium channel 11; IKATP; Inward rectifier K(+) channel Kir6.2; Potassium channel; inwardly rectifying subfamily J member 11
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 trans



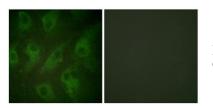


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1 hela 2 mouse-liver 3 mouse-brain Western Blot analysis of NIH-3T3 cells using KIR6.2 Polyclonal Antibody diluted at 1:500

Western Blot analysis of various cells using Antibody diluted at 1:1000. Secondary antibody(catalog#:RS0002) was diluted at 1:20000



Immunofluorescence analysis of HeLa cells, using Kir6.2 Antibody. The picture on the right is blocked with the synthesized peptide.