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KCNJ5 Polyclonal Antibody

Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP59427

Product Information

Application	WB, IHC-P, IHC-F, IF, ICC, E
Primary Accession	P48544
Reactivity	Rat, Pig, Bovine
Host	Rabbit
Clonality	Polyclonal
Calculated MW	47668
Physical State	Liquid
Immunogen	KLH conjugated synthetic peptide derived from human KCNJ5
Epitope Specificity	61-160/419
Isotype	IgG
Purity	affinity purified by Protein A
Buffer	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
SUBCELLULAR LOCATION	Membrane; Multi-pass membrane protein.
SIMILARITY	Belongs to the inward rectifier-type potassium channel (TC 1.A.2.1) family. KCNJ5 subfamily.
SUBUNIT	May associate with GIRK1 and GIRK2 to form a G-protein-activated heteromultimer pore-forming unit. The resulting inward current is much larger (By similarity).
DISEASE	Defects in KCNJ5 are the cause of long QT syndrome type 13 (LQT13) [MIM:613485]. It is a heart disorder characterized by a prolonged QT interval on the ECG and polymorphic ventricular arrhythmias. They cause syncope and sudden death in response to excercise or emotional stress, and can present with a sentinel event of sudden cardiac death in infancy. Defects in KCNJ5 are the cause of familial hyperaldosteronism type 3 (FH3) [MIM:613677]. A form of hyperaldosteronism characterized by hypertension secondary to massive adrenal mineralocorticoid production. Like patients with familial hyperaldosteronism type 1 (glucocorticoid-remediable aldosteronism), patients with FH3 present with childhood hypertension, elevated aldosteronism levels, and high levels of the hybrid steroids 18-oxocortisol and 18-hydroxycortisol. However, hypertension and aldosteronism are not reversed by administration of exogenous glucocorticoids and patients require adrenalectomy to control hypertension. Note=Somatic mutations in KCNJ5 have been found in aldosterone-producing adrenal adenomas and can be responsible for aldosteronism associated with cell autonomous proliferation. These are typically solitary, well circumscribed tumors diagnosed between ages 30 and 70. They come to medical attention due to new or worsening hypertension, often with hypokalemia. KCNJ5 mutations produce increased sodium conductance and cell depolarization, which in adrenal glomerulosa cells produces calcium entry, the signal for aldosterone production and cell proliferation.
Important Note	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
Background Descriptions	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. It may associate with two other G-protein-activated potassium channels to form a heteromultimeric pore-forming complex. [provided by RefSeq, Jul 2008].

Additional Information

Gene ID	3762
Other Names	G protein-activated inward rectifier potassium channel 4, GIRK-4, Cardiac inward rectifier, CIR, Heart KATP channel, Inward rectifier K(+) channel Kir3.4, IRK-4, KATP-1, Potassium channel, inwardly rectifying subfamily J member 5, KCNJ5, GIRK4
Target/Specificity	Islets, exocrine pancreas and heart.
Dilution	WB=1:500-2000,IHC-P=1:100-500,IHC-F=1:100-500,ICC=1:100-500,IF=1:50-200, ELISA=1:5000-10000
Format	0.01M TBS(pH7.4) with 1% BSA, 0.09% (W/V) sodium azide and 50% Glyce
Storage	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. 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.

Protein Information

Name	KCNJ5
Synonyms	GIRK4
Function	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 external barium. This potassium channel is controlled by G proteins.
Cellular Location	Membrane; Multi-pass membrane protein
Tissue Location	Islets, exocrine pancreas and heart. Expressed in the adrenal cortex, particularly the zona glomerulosa

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