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Rabbit Anti-KCNJ5/AP Conjugated antibody

SL9931R-AP

Product Name	Anti-KCNJ5/AP
Chinese Name	碱性磷酸酶（AP）标记的 G 蛋白激活内向钾通道 5 抗体
Alias	inwardly rectifying subfamily J member 5; Cardiac ATP sensitive potassium channel; Cardiac inward rectifier; CIR; G protein activated inward rectifier potassium channel 4; G protein-activated inward rectifier potassium channel 4; GIRK 4; GIRK-4; GIRK4; Heart KATP channel; Inward rectifier K(+) channel Kir3.4; Inward rectifier K+ channel KIR3.4; IRK-4; IRK5_HUMAN; KATP 1; KATP-1; KATP1; KCNJ 5; Kcnj5; KIR 3.4; KIR3.4; Potassium channel; Potassium channel inwardly rectifying subfamily J member 5; Potassium inwardly rectifying channel J5; Potassium inwardly rectifying channel subfamily J member 5.
Research Area	Tumour Cardiovascular Neurobiology Channel protein G protein signal
Immunogen Species	Rabbit
Clonality	Polyclonal
React Species	(predicted:Human,Mouse,Rat,Chicken,Pig,Cow,Horse,Rabbit,Sheep)
Applications	WB=1000-10000,IHC-P=1:100-500,IHC-F=1:100-500,ELISA=1:500-5000 not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Molecular weight	48kDa
Form	Lyophilized or Liquid
Concentration	1mg/ml
immunogen	KLH conjugated synthetic peptide derived from human KCNJ5
Lsotype	IgG
Purification	affinity purified by Protein A
Storage Buffer	1M TBS(pH7.4) with 1% BSA, 3% 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 1M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.
Product Detail	**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. It may associate with two other G-protein-activated potassium channels to form a heteromultimeric pore-forming complex. [provided by RefSeq, Jul 2008].

Function:
This potassium channel 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 external barium.

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).

Subcellular Location:
Membrane; Multi-pass membrane protein.

Tissue Specificity:
Islets, exocrine pancreas and heart.

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.

Similarity:
Belongs to the inward rectifier-type potassium channel (TC 1.A.2.1) family. KCNJ5 subfamily.

Database links:

Entrez Gene: 3762 Human

Entrez Gene: 16521 Mouse

Entrez Gene: 29713 Rat

Omim: 600734 Human

SwissProt: P48544 Human

SwissProt: P48545 Mouse

SwissProt: P48548 Rat

Unigene: 632109 Human

Unigene: 69472 Mouse

Unigene: 10047 Rat

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

Involvement in disease; Defects in KCNJ5 are the cause of long QT syndrome type 13 (LQT13). 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.