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Rabbit Anti-KCNJ5/Biotin Conjugated antibody

SL9931R-Bio

Product Name	Anti-KCNJ5/Biotin
Chinese Name	生物素标记的 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 in rectifier; CIR; G protein activated inward rectifier potassium channel 4; G protein-activated inwa potassium channel 4; GIRK 4; GIRK-4; GIRK4; Heart KATP channel; Inward rectifier K(+) cha Inward rectifier K+ channel KIR3.4; IRK-4; IRK5_HUMAN; KATP 1; KATP-1; KATP1; KCN KIR 3.4; KIR3.4; Potassium channel; Potassium channel inwardly rectifying subfamily J membe Potassium inwardly rectifying channel J5; Potassium inwardly rectifying channel subfamily J m
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,ICC/IF=1:50-200,IF=1:100-500,ELISA= 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 stab temperature for at least one month and for greater than a year when kept at -20°C. When reconst sterile pH 7.4 1M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4
Product	**background:**

Detail

Potassium channels are present in most mammalian cells, where they participate in a wide range physiologic responses. The protein encoded by this gene is an integral membrane protein and inw type potassium channel. The encoded protein, which has a greater tendency to allow potassium t cell rather than out of a cell, is controlled by G-proteins. It may associate with two other G-prote potassium channels to form a heteromultimeric pore-forming complex. [provided by RefSeq, Jul

Function:
This potassium channel is controlled by G proteins. Inward rectifier potassium channels are char a greater tendency to allow potassium to flow into the cell rather than out of it. Their voltage dep regulated by the concentration of extracellular potassium; as external potassium is raised, the volt the channel opening shifts to more positive voltages. The inward rectification is mainly due to the 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-formi 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 he characterized by a prolonged QT interval on the ECG and polymorphic ventricular arrhythmias. syncope and sudden death in response to excercise or emotional stress, and can present with a se of sudden cardiac death in infancy.
Defects in KCNJ5 are the cause of familial hyperaldosteronism type 3 (FH3) [MIM:613677]. A hyperaldosteronism characterized by hypertension secondary to massive adrenal mineralocortico production. Like patients with familial hyperaldosteronism type 1 (glucocorticoid-remediable aldo patients with FH3 present with childhood hypertension, elevated aldosteronism levels, and high hybrid steroids 18-oxocortisol and 18-hydroxycortisol. However, hypertension and aldosteronism reversed by administration of exogenous glucocorticoids and patients require adrenalectomy to c hypertension.
Note=Somatic mutations in KCNJ5 have been found in aldosterone-producing adrenal adenomas responsible for aldosteronism associated with cell autonomous proliferation. These are typically s circumscribed tumors diagnosed between ages 30 and 70. They come to medical attention due to worsening hypertension, often with hypokalemia. KCNJ5 mutations produce increased sodium c and cell depolarization, which in adrenal glomerulosa cells produces calcium entry, the signal for 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 d
applications.

Involvement in disease; Defects in KCNJ5 are the cause of long QT syndrome type 13 (LQT13)
disorder characterized by a prolonged QT interval on the ECG and polymorphic ventricular arrh
They cause syncope and sudden death in response to excercise or emotional stress, and can prese
sentinel event of sudden cardiac death in infancy.