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Kir4.1 Antibody

Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP51735

Product Information

Application	WB
Primary Accession	P78508
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Calculated MW	42508

Additional Information

Gene ID	3766
Other Names	ATP-sensitive inward rectifier potassium channel 10, ATP-dependent inwardly rectifying potassium channel Kir41, Inward rectifier K(+) channel Kir12, Potassium channel, inwardly rectifying subfamily J member 10, KCNJ10
Target/Specificity	KLH-conjugated synthetic peptide encompassing a sequence within the center region of human Kir4.1. The exact sequence is proprietary.
Dilution	WB~~1:1000
Format	0.01M PBS, pH 7.2, 0.09% (W/V) Sodium azide, Glycerol 50%
Storage	Store at -20 °C.Stable for 12 months from date of receipt

Protein Information

Name	KCNJ10 (HGNC:6256)
Function	May be responsible for potassium buffering action of glial cells in the brain (By similarity). Inward rectifier potassium channels are characterized by a greater tendency to allow potassium to flow into the cell rather than out of it (PubMed:8995301). 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 (PubMed:8995301). The inward rectification is mainly due to the blockage of outward current by internal magnesium. Can be blocked by extracellular barium and cesium (PubMed:8995301). In the kidney, together with KCNJ16, mediates basolateral K(+) recycling in distal tubules; this process is critical for Na(+) reabsorption at the tubules (PubMed:24561201).
Cellular Location	Membrane; Multi- pass membrane protein. Basolateral cell membrane. Note=In kidney distal convoluted tubules, located in the basolateral

	membrane where it colocalizes with KCNJ16.
Tissue Location	Expressed in kidney (at protein level) (PubMed:24561201). In the nephron, expressed in the distal convoluted tubule, the connecting tubule, the collecting duct and cortical thick ascending limbs (PubMed:20651251).

Background

May be responsible for potassium buffering action of glial cells in the brain. 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 and cesium (By similarity).

References

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