KCNJ10 Protein, Rat (Cell-Free, His)

Cat. No.:	HY-P702344
Synonyms:	ATP-sensitive inward rectifier potassium channel 10; ATP-sensitive inward rectifier potassium channel KAB-2; BIR10; Brain-specific inwardly rectifying K(+) channel 1; BIRK1; Inward rectifier K(+) channel Kir4.1; Potassium channel, inwardly rectifying subfamily J member 10
Species:	Rat
Source:	E. coli Cell-free
Accession:	P49655 (M1-V379)
Gene ID:	29718
Molecular Weight:	46.6 kDa

PROPERTIES

AA Sequence	MTSVAKVYYS QTTQTESRPL VAPGIRRRRV LTKDGRSNVR MEHIADKRFL YLKDLWTTFI DMQWRYKLLL FSATFAGTWF LFGVVWYLVA VAHGDLLELG PPANHTPCVV QVHTLTGAFL FSLESQTTIG YGFRYISEEC PLAIVLLIAQ LVLTTILEIF ITGTFLAKIA RPKKRAETIR FSQHAVVAYH NGKLCLMIRV ANMRKSLLIG CQVTGKLLQT HQTKEGENIR LNQVNVTFQV DTASDSPFLI LPLTFYHVVD ETSPLKDLPL RSGEGDFELV LILSGTVEST SATCQVRTSY LPEEILWGYE FTPAISLSAS GKYVADFSLF DQVVKVASPG GLRDSTVRYG DPEKLKLEES LREQAEKEGS ALSVRISNV
Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.22 μm filtered solution of Tris/PBS-based buffer, 6% Trehalose, pH 8.0.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 μg/mL in ddH_2O. For long term storage it is recommended to add 5-50% of glycerol (final concentration). Our default final concentration of glycerol is 50%. Customers could use it as reference.
Storage & Stability	Stored at -20°C for 2 years. After reconstitution, it is stable at 4°C for 1 week or -20°C for longer (with carrier protein). It is recommended to freeze aliquots at -20°C or -80°C for extended storage.
Shipping	Room temperature in continental US; may vary elsewhere.

DESCRIPTION

Background	The KCNJ10 protein is implicated in the potassium buffering actions of glial cells in the brain. As an inward rectifier potassium channel, KCNJ10 exhibits a pronounced preference for allowing potassium influx into the cell rather than efflux. Its voltage dependence is finely tuned by extracellular potassium concentrations, resulting in a shift to more positive voltages as external potassium levels rise. The inward rectification mechanism is primarily attributed to the blockade of outward current by internal magnesium and can be impeded by extracellular barium and cesium. In the kidney, KCNJ10 collaborates with KCNJ16 to mediate basolateral K(+) recycling in distal tubules, a process crucial for Na(+) reabsorption. Forming a heterodimer with Kir5.1/KCNJ16, KCNJ10 ensures the necessary interaction for the localization of KCNJ16 to the basolateral membrane in kidney cells. Additionally, KCNJ10 interacts with MAGI1, both independently and possibly as a heterodimer with KCNJ16, potentially facilitating the expression of KCNJ10/KCNJ16 potassium channels at the basolateral membrane in kidney cells. Furthermore, KCNJ10 interacts with PATJ, hinting at its broader involvement in cellular interactions and membrane localization.

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