KCNJ10 Protein, Human (Cell-Free, His)

Cat. No.:	HY-P702341
Synonyms:	ATP-sensitive inward rectifier potassium channel 10; ATP-dependent inwardly rectifying potassium channel Kir4.1; Inward rectifier K(+) channel Kir1.2; Potassium channel, inwardly rectifying subfamily J member 10
Species:	Human
Source:	E. coli Cell-free
Accession:	P78508 (M1-V379)
Gene ID:	3766
Molecular Weight:	48.6 kDa

PROPERTIES

AA Sequence	MTSVAKVYYS QTTQTESRPL MGPGIRRRRV LTKDGRSNVR MEHIADKRFL YLKDLWTTFI DMQWRYKLLL FSATFAGTWF LFGVVWYLVA VAHGDLLELD PPANHTPCVV QVHTLTGAFL FSLESQTTIG YGFRYISEEC PLAIVLLIAQ LVLTTILEIF ITGTFLAKIA RPKKRAETIR FSQHAVVASH NGKPCLMIRV ANMRKSLLIG CQVTGKLLQT HQTKEGENIR LNQVNVTFQV DTASDSPFLI LPLTFYHVVD ETSPLKDLPL RSGEGDFELV LILSGTVEST SATCQVRTSY LPEEILWGYE FTPAISLSAS GKYIADFSLF DQVVKVASPS 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 within the brain. As an inward rectifier potassium channel, it exhibits a greater inclination to permit potassium influx into the cell rather than efflux. The voltage dependence of these channels is modulated by extracellular potassium concentrations, with an upward shift in the voltage range of channel opening in response to elevated external potassium levels. The inward rectification is primarily attributed to the inhibition of outward current by internal magnesium. Furthermore, the KCNJ10 protein can be obstructed by extracellular barium and cesium. In the kidney, it collaborates with KCNJ16 to facilitate basolateral K(+) recycling in distal tubules, a process crucial for sodium (Na(+)) reabsorption. This protein forms a heterodimer with Kir5.1/KCNJ16, a requisite 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 aiding in the expression of KCNJ10/KCNJ16 potassium channels at the basolateral membrane in kidney cells. Furthermore, it interacts with PATJ, suggesting a broader role in cellular interactions and membrane localization.

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