10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

KCNK1 Antibody (C-term) Blocking peptide

Synthetic peptide
Catalog # BP12727b

Specification

KCNK1 Antibody (C-term) Blocking peptide - Product Information

Primary Accession O00180

KCNK1 Antibody (C-term) Blocking peptide - Additional Information

Gene ID 3775

Other Names
Potassium channel subfamily K member 1, Inward rectifying potassium channel protein TWIK-1, Potassium channel KCNO1, KCNK1, HOHO1, KCNO1, TWIK1

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

KCNK1 Antibody (C-term) Blocking peptide - Protein Information

Name KCNK1

Synonyms HOHO1 {ECO:0000303|PubMed:9462864}, KCNO

Function
Ion channel that contributes to passive transmembrane potassium transport and to the regulation of the resting membrane potential in brain astrocytes, but also in kidney and in other tissues (PubMed:15820677, PubMed:21653227). Forms dimeric channels through which potassium ions pass in accordance with their electrochemical gradient. The channel is selective for K(+) ions at physiological potassium concentrations and at neutral pH, but becomes permeable to Na(+) at subphysiological K(+) levels and upon acidification of the extracellular medium (PubMed:21653227, PubMed:22431633). The homodimer has very low potassium channel activity, when expressed in heterologous systems, and can function as weakly inward rectifying potassium channel (PubMed:8605869, PubMed:8978667, PubMed:15820677, PubMed:<a

href="http://www.uniprot.org/citations/21653227" target="_blank">21653227, PubMed:22431633, PubMed:23169818, PubMed:25001086). Channel activity is modulated by activation of serotonin receptors (By similarity). Heterodimeric channels containing KCNK1 and KCNK2 have much higher activity, and may represent the predominant form in astrocytes (By similarity). Heterodimeric channels containing KCNK1 and KCNK3 or KCNK9 have much higher activity (PubMed:23169818). Heterodimeric channels formed by KCNK1 and KCNK9 may contribute to halothane-sensitive currents (PubMed:23169818). Mediates outward rectifying potassium currents in dentate gyrus granule cells and contributes to the regulation of their resting membrane potential (By similarity). Contributes to the regulation of action potential firing in dentate gyrus granule cells and down-regulates their intrinsic excitability (By similarity). In astrocytes, the heterodimer formed by KCNK1 and KCNK2 is required for rapid glutamate release in response to activation of G- protein coupled receptors, such as F2R and CNR1 (By similarity). Required for normal ion and water transport in the kidney (By similarity). Contributes to the regulation of the resting membrane potential of pancreatic beta cells (By similarity). The low channel activity of homodimeric KCNK1 may be due to sumoylation (PubMed:15820677, PubMed:20498050, PubMed:23169818). The low channel activity may be due to rapid internalization from the cell membrane and retention in recycling endosomes (PubMed:19959478).

Cellular Location
Cell membrane; Multi-pass membrane protein {ECO:0000269|PubMed:22282804, ECO:0000269|PubMed:8978667, ECO:0000305} Recycling endosome. Synaptic cell membrane {ECO:0000250|UniProtKB:Q9Z2T2}. Cytoplasmic vesicle {ECO:0000250|UniProtKB:O08581}. Perikaryon {ECO:0000250|UniProtKB:O08581}. Cell projection, dendrite {ECO:0000250|UniProtKB:O08581}. Cell projection {ECO:0000250|UniProtKB:O08581}. Apical cell membrane; Multi-pass membrane protein. Note=The heterodimer with KCNK2 is detected at the astrocyte cell membrane. Not detected at the astrocyte cell membrane when KCNK2 is absent. Detected on neuronal cell bodies, and to a lesser degree on neuronal cell projections. Detected on hippocampus dentate gyrus granule cell bodies and to a lesser degree on proximal dendrites Detected at the apical cell membrane in stria vascularis in the cochlea. Detected at the apical cell membrane of vestibular dark cells situated between the crista and the utricle in the inner ear. Detected at the apical cell membrane in kidney proximal tubule segment S1 and in subapical compartments in segments S1, S2 and S3. Predominantly in cytoplasmic structures in kidney distal convoluted tubules and collecting ducts (By similarity). Detected at the apical cell membrane of bronchial epithelial cells (PubMed:21964404) {ECO:0000250|UniProtKB:O08581, ECO:0000250|UniProtKB:Q9Z2T2, ECO:0000269|PubMed:21964404}

Tissue Location
Detected in bronchial epithelial cells (PubMed:21964404). Detected in heart left atrium and left ventricle (PubMed:17478540). Detected in cardiac myocytes (at protein level) (PubMed:21653227). Widely expressed with high levels in heart, brain and kidney, and lower levels in colon, ovary, placenta, lung and liver (PubMed:8605869, PubMed:9362344). Highly expressed in cerebellum, and detected at lower levels in amygdala, caudate nucleus, brain cortex, hippocampus, putamen, substantia nigra, thalamus, dorsal root ganglion, spinal cord, pituitary, heart, kidney, lung, placenta, pancreas, stomach, small intestine, uterus and prostate (PubMed:11165377) Detected in right and left heart ventricle and atrium, and in heart Purkinje fibers (PubMed:17478540).

KCNK1 Antibody (C-term) Blocking peptide - Protocols

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Provided below are standard protocols that you may find useful for product applications.

- Blocking Peptides

KCNK1 Antibody (C-term) Blocking peptide - Images

KCNK1 Antibody (C-term) Blocking peptide - Background

This gene encodes one of the members of the superfamily ofpotassium channel proteins containing two pore-forming P domains.The product of this gene has not been shown to be a functionalchannel, however, it may require other non-pore-forming proteinsfor activity.

KCNK1 Antibody (C-term) Blocking peptide - References

Feliciangeli, S., et al. J. Biol. Chem. 285(7):4798-4805(2010)McGeachie, M., et al. Circulation 120(24):2448-2454(2009)Zhou, M., et al. J. Neurosci. 29(26):8551-8564(2009)Goldstein, S.A., et al. Pharmacol. Rev. 57(4):527-540(2005)Rajan, S., et al. Cell 121(1):37-47(2005)