

KCNK10 Antibody (C-term)
Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP14248B**Specification**

KCNK10 Antibody (C-term) - Product Information

Application	WB,E
Primary Accession	P57789
Other Accession	NP_066984.1 , NP_612190.1
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	59765
Antigen Region	504-532

KCNK10 Antibody (C-term) - Additional Information**Gene ID** 54207**Other Names**

Potassium channel subfamily K member 10, Outward rectifying potassium channel protein TREK-2, TREK-2 K(+) channel subunit, KCNK10, TREK2

Target/Specificity

This KCNK10 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 504-532 amino acids from the C-terminal region of human KCNK10.

Dilution

WB~~1:1000

E~~Use at an assay dependent concentration.

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

Storage

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

KCNK10 Antibody (C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

KCNK10 Antibody (C-term) - Protein Information

Name KCNK10 {ECO:0000303|PubMed:25766236, ECO:0000312|HGNC:HGNC:6273}

Function K(+) channel that conducts voltage-dependent outward rectifying currents upon membrane depolarization. Voltage sensing is coupled to K(+) electrochemical gradient in an 'ion flux gating' mode where outward but not inward ion flow opens the gate. Converts to voltage-independent 'leak' conductance mode upon stimulation by various stimuli including mechanical membrane stretch, acidic pH, heat and lipids (PubMed:[10880510](#), PubMed:[25766236](#), PubMed:[26919430](#), PubMed:[38605031](#)). Homo- and heterodimerizes to form functional channels with distinct regulatory and gating properties (PubMed:[30573346](#)). In trigeminal ganglia sensory neurons, the heterodimer of KCNK10/TREK-2 and KCNK18/TRESK inhibits neuronal firing and neurogenic inflammation by stabilizing the resting membrane potential at K(+) equilibrium potential as well as by regulating the threshold of action potentials and the spike frequency (By similarity). Permeable to other monovalent ions such as Rb(+) and Cs(+) (PubMed:[26919430](#)).

Cellular Location

Cell membrane {ECO:0000250|UniProtKB:Q8BUW1}; Multi-pass membrane protein

Tissue Location

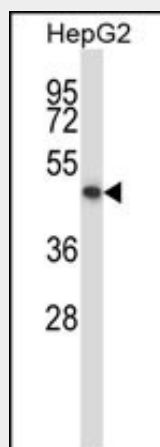
[Isoform A]: Abundantly expressed in pancreas and kidney and to a lower level in brain, testis, colon, and small intestine. In brain, mainly expressed in cerebellum, occipital lobe, putamen, and thalamus. No expression is detected in amygdala and spinal cord. [Isoform C]: Abundantly expressed in brain.

KCNK10 Antibody (C-term) - Protocols

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

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

KCNK10 Antibody (C-term) - Images



KCNK10 Antibody (C-term) (Cat. #AP14248b) western blot analysis in HepG2 cell line lysates (35ug/lane). This demonstrates the KCNK10 antibody detected the KCNK10 protein (arrow).

KCNK10 Antibody (C-term) - Background

The protein encoded by this gene belongs to the family of potassium channel proteins containing two pore-forming P domains. This channel is an open rectifier which primarily passes outward current under physiological K⁺ concentrations, and is stimulated strongly by arachidonic acid and to a lesser degree by membrane stretching, intracellular acidification, and general anaesthetics. Several alternatively spliced transcript variants encoding different isoforms have been identified for this gene. [provided by RefSeq].

KCNK10 Antibody (C-term) - References

Gierten, J., et al. Br. J. Pharmacol. 154(8):1680-1690(2008)
Huang, D., et al. Med. Hypotheses 70(3):618-624(2008)
Goldstein, S.A., et al. Pharmacol. Rev. 57(4):527-540(2005)
Gu, W., et al. J. Physiol. (Lond.) 539 (PT 3), 657-668 (2002) :
Goldstein, S.A., et al. Nat. Rev. Neurosci. 2(3):175-184(2001)