

# 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 <u>NP\_066984.1</u>, <u>NP\_612190.1</u>

Reactivity
Host
Clonality
Polyclonal
Isotype
Calculated MW
Antigen Region

Human
Rabbit
Polyclonal
Rabbit IgG
59765
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: 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**

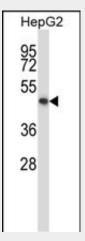
[Isoform A]: Abundantly expressed in pancreas and kidney and to a lower level in brain, testis, colon, and small intestine. In brain, mainly expressed in cerebellum, occipital lobe, putamen, and thalamus. No expression is detected in amygdala and spinal cord. [Isoform C]: Abundantly expressed in brain.

### KCNK10 Antibody (C-term) - Protocols

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

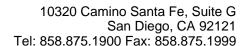
- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- <u>Immunoprecipitation</u>
- Flow Cytomety
- Cell Culture

# 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)