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KCNA4 Blocking Peptide (C-term)

Synthetic peptide
Catalog # BP19983b

Specification

KCNA4 Blocking Peptide (C-term) - Product Information

Primary Accession	P22459
Other Accession	P15385, Q61423, Q05037, NP_002224.1

KCNA4 Blocking Peptide (C-term) - Additional Information

Gene ID 3739

Other Names
Potassium voltage-gated channel subfamily A member 4, HPCN2, Voltage-gated K(+) channel HuKII, Voltage-gated potassium channel HBK4, Voltage-gated potassium channel HK1, Voltage-gated potassium channel subunit Kv14, KCNA4, KCNA4L

Target/Specificity
The synthetic peptide sequence is selected from aa 606-619 of HUMAN KCNA4

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.

KCNA4 Blocking Peptide (C-term) - Protein Information

Name KCNA4

Synonyms KCNA4L

Function
Voltage-gated potassium channel that mediates transmembrane potassium transport in excitable membranes. Forms tetrameric potassium- selective channels through which potassium ions pass in accordance with their electrochemical gradient. The channel alternates between opened and closed conformations in response to the voltage difference across the membrane (PubMed:19912772, PubMed:8495559). Can form functional homotetrameric channels and heterotetrameric channels that contain variable proportions of KCNA1, KCNA2, KCNA4, KCNA5, and possibly other family members as well; channel properties depend on the type of alpha subunits that are part of the channel (PubMed:8495559). Channel

properties are modulated by cytoplasmic beta subunits that regulate the subcellular location of the alpha subunits and promote rapid inactivation. In vivo, membranes probably contain a mixture of heteromeric potassium channel complexes, making it difficult to assign currents observed in intact tissues to any particular potassium channel family member. Homotetrameric KCNA4 forms a potassium channel that opens in response to membrane depolarization, followed by rapid spontaneous channel closure (PubMed:19912772, PubMed:8495559). Likewise, a heterotetrameric channel formed by KCNA1 and KCNA4 shows rapid inactivation (PubMed:17156368).

Cellular Location
Cell membrane; Multi-pass membrane protein Cell projection, axon {ECO:0000250|UniProtKB:P15385}

Tissue Location
Expressed in brain, and at lower levels in the testis, lung, kidney, colon and heart (PubMed:27582084). Detected in heart ventricle.

KCNA4 Blocking Peptide (C-term) - Protocols

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

- Blocking Peptides

KCNA4 Blocking Peptide (C-term) - Images

KCNA4 Blocking Peptide (C-term) - Background

Potassium channels represent the most complex class of voltage-gated ion channels from both functional and structural standpoints. Their diverse functions include regulating neurotransmitter release, heart rate, insulin secretion, neuronal excitability, epithelial electrolyte transport, smooth muscle contraction, and cell volume. Four sequence-related potassium channel genes - shaker, shaw, shab, and shal - have been identified in Drosophila, and each has been shown to have human homolog(s). This gene encodes a member of the potassium channel, voltage-gated, shaker-related subfamily. This member contains six membrane-spanning domains with a shaker-type repeat in the fourth segment. It belongs to the A-type potassium current class, the members of which may be important in the regulation of the fast repolarizing phase of action potentials in heart and thus may influnce the duration of cardiac action potential. The coding region of this gene is intronless, and the gene is clustered with genes KCNA3 and KCNA10 on chromosome 1.

KCNA4 Blocking Peptide (C-term) - References

Schwetz, T.A., et al. Biochim. Biophys. Acta 1798(3):367-375(2010)
Angelova, P.R., et al. Eur. J. Neurosci. 29(10):1943-1950(2009)
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Lee, J.H., et al. Mol. Pharmacol. 73(3):619-626(2008)
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