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KCNAB1 Antibody(N-term) Blocking peptide

Synthetic peptide
Catalog # BP19413a

Specification

KCNAB1 Antibody(N-term) Blocking peptide - Product Information

Primary Accession Q14722

KCNAB1 Antibody(N-term) Blocking peptide - Additional Information

Gene ID 7881

Other Names
Voltage-gated potassium channel subunit beta-1, K(+) channel subunit beta-1, Kv-beta-1, KCNAB1, KCNA1B

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.

KCNAB1 Antibody(N-term) Blocking peptide - Protein Information

Name KCNAB1 (HGNC:6228)

Synonyms KCNA1B

Function
Regulatory subunit of the voltage-gated potassium (Kv) Shaker channels composed of pore-forming and potassium-conducting alpha subunits and of regulatory beta subunits (PubMed:17156368, PubMed:17540341, PubMed:19713757, PubMed:7499366, PubMed:7603988). The beta-1/KCNAB1 cytoplasmic subunit mediates closure of delayed rectifier potassium channels by physically obstructing the pore via its N- terminal domain and increases the speed of channel closure for other family members (PubMed:9763623). Promotes the inactivation of Kv1.1/KCNA1, Kv1.2/KCNA2, Kv1.4/KCNA4, Kv1.5/KCNA5 and Kv1.6/KCNA6 alpha subunit-containing channels (PubMed:12077175, PubMed:12130714, PubMed:15361858, PubMed:<a

href="http://www.uniprot.org/citations/17156368" target="_blank">17156368, PubMed:17540341, PubMed:19713757, PubMed:7499366, PubMed:7603988, PubMed:7649300, PubMed:7890764, PubMed:9763623). Displays nicotinamide adenine dinucleotide phosphate (NADPH)-dependent aldoketoreductase activity by catalyzing the NADPH- dependent reduction of a variety of endogenous aldehydes and ketones (By similarity). The binding of NADPH is required for efficient down- regulation of potassium channel activity (PubMed:17540341). Oxidation of the bound NADPH restrains N-terminal domain from blocking the channel, thereby decreasing N-type inactivation of potassium channel activity (By similarity).

Cellular Location
Cytoplasm. Membrane {ECO:0000250|UniProtKB:P63144}; Peripheral membrane protein; Cytoplasmic side. Cell membrane; Peripheral membrane protein; Cytoplasmic side. Note=Recruited to the cytoplasmic side of the cell membrane via its interaction with pore-forming potassium channel alpha subunits.

Tissue Location
In brain, expression is most prominent in caudate nucleus, hippocampus and thalamus. Significant expression also detected in amygdala and subthalamic nucleus. Also expressed in both healthy and cardiomyopathic heart. Up to four times more abundant in left ventricle than left atrium.

KCNAB1 Antibody(N-term) Blocking peptide - Protocols

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

- Blocking Peptides

KCNAB1 Antibody(N-term) Blocking peptide - Images

KCNAB1 Antibody(N-term) Blocking peptide - Background

Potassium channels represent the most complex class ofvoltage-gated ion channels from both functional and structuralstandpoints. Their diverse functions include regulatingneurotransmitter release, heart rate, insulin secretion, neuronalexcitability, epithelial electrolyte transport, smooth musclecontraction, and cell volume. Four sequence-related potassiumchannel genes - shaker, shaw, shab, and shal - have been identifiedin 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 includes three distinctisoforms which are encoded by three alternatively splicedtranscript variants of this gene. These three isoforms are betasubunits, which form heteromultimeric complex with alpha subunitsand modulate the activity of the pore-forming alpha subunits.

KCNAB1 Antibody(N-term) Blocking peptide - References

Rose, J.E., et al. Mol. Med. 16 (7-8), 247-253 (2010) :Decher, N., et al. EMBO J. 27(23):3164-3174(2008)Cavalleri, G.L., et al. Lancet Neurol 6(11):970-980(2007)Lamesch, P., et al. Genomics 89(3):307-315(2007)Lunetta, K.L., et al. BMC Med. Genet. 8 SUPPL 1, S13 (2007) :