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KCNQ3 Antibody (C-term)

Affinity Purified Rabbit Polyclonal Antibody (Pab)

Catalog # AP14685b

Specification

KCNQ3 Antibody (C-term) - Product Information

Application	**WB,E**
Primary Accession	O43525
Other Accession	NP_004510.1
Reactivity	**Mouse**
Host	**Rabbit**
Clonality	**Polyclonal**
Isotype	**Rabbit IgG**
Calculated MW	**96742**
Antigen Region	**651-679**

KCNQ3 Antibody (C-term) - Additional Information

Gene ID 3786

Other Names
Potassium voltage-gated channel subfamily KQT member 3, KQT-like 3, Potassium channel subunit alpha KvLQT3, Voltage-gated potassium channel subunit Kv73, KCNQ3

Target/Specificity
This KCNQ3 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 651-679 amino acids from the C-terminal region of human KCNQ3.

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
KCNQ3 Antibody (C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

KCNQ3 Antibody (C-term) - Protein Information

Name KCNQ3 (HGNC:6297)

Function Pore-forming subunit of the voltage-gated potassium (Kv) M- channel which is responsible for the M-current, a key controller of neuronal excitability (PubMed:16319223, PubMed:27564677, PubMed:28793216, PubMed:9872318). M-channel is composed of pore-forming subunits KCNQ2 and KCNQ3 assembled as heterotetramers (PubMed:14534157, PubMed:16319223, PubMed:27564677, PubMed:9872318). The native M-current has a slowly activating and deactivating potassium conductance which plays a critical role in determining the subthreshold electrical excitability of neurons as well as the responsiveness to synaptic inputs (PubMed:14534157, PubMed:16319223, PubMed:28793216). M-channel is selectively permeable in vitro to other cations besides potassium, in decreasing order of affinity K(+) > Rb(+) > Cs(+) > Na(+) (PubMed:28793216). M-channel association with SLC5A3/SMIT1 alters channel ion selectivity, increasing Na(+) and Cs(+) permeation relative to K(+) (PubMed:28793216). Suppressed by activation of M1 muscarinic acetylcholine receptors (PubMed:10713961). KCNQ3 also associates with KCNQ5 to form a functional channel in vitro and may also contribute to the M-current in brain (PubMed:11159685).

Cellular Location
Cell membrane; Multi-pass membrane protein

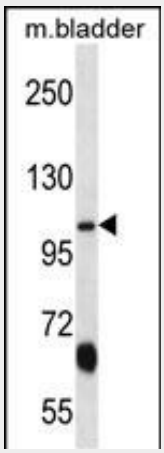
Tissue Location
Predominantly expressed in brain.

KCNQ3 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 Cytomety
- Cell Culture

KCNQ3 Antibody (C-term) - Images



KCNQ3 Antibody (C-term) (Cat. #AP14685b) western blot analysis in mouse bladder tissue lysates (35ug/lane).This demonstrates the KCNQ3 antibody detected the KCNQ3 protein (arrow).

KCNQ3 Antibody (C-term) - Background

The M channel is a slowly activating and deactivating potassium channel that plays a critical role in the regulation of neuronal excitability. The M channel is formed by the association of the protein encoded by this gene and one of two related proteins encoded by the KCNQ2 and KCNQ5 genes, both integral membrane proteins. M channel currents are inhibited by M1 muscarinic acetylcholine receptors and activated by retigabine, a novel anti-convulsant drug. Defects in this gene are a cause of benign familial neonatal convulsions type 2 (BFNC2), also known as epilepsy, benign neonatal type 2 (EBN2).

KCNQ3 Antibody (C-term) - References

Bailey, S.D., et al. Diabetes Care (2010) In press :
Gomez-Posada, J.C., et al. J. Neurosci. 30(27):9316-9323(2010)
Rose, J.E., et al. Mol. Med. 16 (7-8), 247-253 (2010) :
Talmud, P.J., et al. Am. J. Hum. Genet. 85(5):628-642(2009)
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