

### KCNQ3 Antibody (C-term)

Affinity Purified Rabbit Polyclonal Antibody (Pab) Catalog # AP14685b

### Specification

# KCNQ3 Antibody (C-term) - Product Information

Application	WB,E
Primary Accession	<u>043525</u>
Other Accession	<u>NP_004510.1</u>
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 (<u>HGNC:6297</u>)



**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:<u>16319223</u>, PubMed:<u>27564677</u>, PubMed:<u>28793216</u>, PubMed:<u>9872318</u>). M-channel is composed of pore-forming subunits KCNQ2 and KCNQ3 assembled as heterotetramers (PubMed:<u>14534157</u>, PubMed:<u>16319223</u>, PubMed:<u>27564677</u>, PubMed:<u>9872318</u>). 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:<u>14534157</u>, PubMed:<u>16319223</u>, PubMed:<u>16319223</u>, PubMed:<u>28793216</u>). M-channel is selectively permeable in vitro to other cations besides potassium, in decreasing order of affinity K(+) > Rb(+) > Cs(+) > Na(+) (PubMed:<u>28793216</u>). M-channel association with SLC5A3/SMIT1 alters channel ion selectivity, increasing Na(+) and Cs(+) permeation relative to K(+) (PubMed:<u>10713961</u>). KCNQ3 also associates with KCNQ5 to form a functional channel in vitro and may also contribute to the M-current in brain (PubMed:<u>11159685</u>).

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.

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

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KCNQ3 Antibody (C-term) - Images
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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) Hahn, A., et al. Brain Dev. 31(7):515-520(2009)