

**KCNQ1 Antibody (Center) Blocking Peptide**  
**Synthetic peptide**  
**Catalog # BP8944c****Specification**

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**KCNQ1 Antibody (Center) Blocking Peptide - Product Information**Primary Accession [P51787](#)**KCNQ1 Antibody (Center) Blocking Peptide - Additional Information**

Gene ID 3784

**Other Names**

Potassium voltage-gated channel subfamily KQT member 1, IKs producing slow voltage-gated potassium channel subunit alpha KvLQT1, KQT-like 1, Voltage-gated potassium channel subunit Kv71, KCNQ1, KCNA8, KCNA9, KVLQT1

**Target/Specificity**

The synthetic peptide sequence used to generate the antibody [AP8944c](/products/AP8944c) was selected from the Center region of human KCNQ1. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

**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.

**KCNQ1 Antibody (Center) Blocking Peptide - Protein Information**Name KCNQ1 ([HGNC:6294](#))**Function**

Potassium channel that plays an important role in a number of tissues, including heart, inner ear, stomach and colon (PubMed: [10646604](http://www.uniprot.org/citations/10646604), PubMed: [25441029](http://www.uniprot.org/citations/25441029)). Associates with KCNE beta subunits that modulates current kinetics (PubMed: [9312006](http://www.uniprot.org/citations/9312006), PubMed: [9108097](http://www.uniprot.org/citations/9108097), PubMed: [8900283](http://www.uniprot.org/citations/8900283), PubMed: [10646604](http://www.uniprot.org/citations/10646604), PubMed: [11101505](http://www.uniprot.org/citations/11101505), PubMed: [19687231](http://www.uniprot.org/citations/19687231)

target="\_blank">19687231</a>). Induces a voltage-dependent current by rapidly activating and slowly deactivating potassium-selective outward current (PubMed:<a href="http://www.uniprot.org/citations/9312006" target="\_blank">9312006</a>, PubMed:<a href="http://www.uniprot.org/citations/9108097" target="\_blank">9108097</a>, PubMed:<a href="http://www.uniprot.org/citations/8900283" target="\_blank">8900283</a>, PubMed:<a href="http://www.uniprot.org/citations/10646604" target="\_blank">10646604</a>, PubMed:<a href="http://www.uniprot.org/citations/11101505" target="\_blank">11101505</a>, PubMed:<a href="http://www.uniprot.org/citations/25441029" target="\_blank">25441029</a>). Promotes also a delayed voltage activated potassium current showing outward rectification characteristic (By similarity). During beta-adrenergic receptor stimulation participates in cardiac repolarization by associating with KCNE1 to form the I(Ks) cardiac potassium current that increases the amplitude and slows down the activation kinetics of outward potassium current I(Ks) (By similarity) (PubMed:<a href="http://www.uniprot.org/citations/9312006" target="\_blank">9312006</a>, PubMed:<a href="http://www.uniprot.org/citations/9108097" target="\_blank">9108097</a>, PubMed:<a href="http://www.uniprot.org/citations/8900283" target="\_blank">8900283</a>, PubMed:<a href="http://www.uniprot.org/citations/10646604" target="\_blank">10646604</a>, PubMed:<a href="http://www.uniprot.org/citations/11101505" target="\_blank">11101505</a>). Muscarinic agonist oxotremorine-M strongly suppresses KCNQ1/KCNE1 current (PubMed:<a href="http://www.uniprot.org/citations/10713961" target="\_blank">10713961</a>). When associated with KCNE3, forms the potassium channel that is important for cyclic AMP-stimulated intestinal secretion of chloride ions (PubMed:<a href="http://www.uniprot.org/citations/10646604" target="\_blank">10646604</a>). This interaction with KCNE3 is reduced by 17beta-estradiol, resulting in the reduction of currents (By similarity). During conditions of increased substrate load, maintains the driving force for proximal tubular and intestinal sodium ions absorption, gastric acid secretion, and cAMP- induced jejunal chloride ions secretion (By similarity). Allows the provision of potassium ions to the luminal membrane of the secretory canaliculus in the resting state as well as during stimulated acid secretion (By similarity). When associated with KCNE2, forms a heterooligomer complex leading to currents with an apparently instantaneous activation, a rapid deactivation process and a linear current-voltage relationship and decreases the amplitude of the outward current (PubMed:<a href="http://www.uniprot.org/citations/11101505" target="\_blank">11101505</a>). When associated with KCNE4, inhibits voltage-gated potassium channel activity (PubMed:<a href="http://www.uniprot.org/citations/19687231" target="\_blank">19687231</a>). When associated with KCNE5, this complex only conducts current upon strong and continued depolarization (PubMed:<a href="http://www.uniprot.org/citations/12324418" target="\_blank">12324418</a>). Also forms a heterotetramer with KCNQ5; has a voltage-gated potassium channel activity (PubMed:<a href="http://www.uniprot.org/citations/24855057" target="\_blank">24855057</a>). Binds with phosphatidylinositol 4,5- biphosphate (PubMed:<a href="http://www.uniprot.org/citations/25037568" target="\_blank">25037568</a>). KCNQ1-KCNE2 channel associates with Na(+)-coupled myo-inositol symporter in the apical membrane of choroid plexus epithelium and regulates the myo-inositol gradient between blood and cerebrospinal fluid with an impact on neuron excitability.

### Cellular Location

Cell membrane; Multi-pass membrane protein. Cytoplasmic vesicle membrane Early endosome. Membrane raft. Endoplasmic reticulum Basolateral cell membrane. Apical cell membrane {ECO:0000250|UniProtKB:P97414}; Multi-pass membrane protein. Note=Colocalized with KCNE3 at the plasma membrane (PubMed:10646604). Upon 17beta-oestradiol treatment, colocalizes with RAB5A at early endosome (PubMed:23529131). Heterotetramer with KCNQ5 is highly retained at the endoplasmic reticulum and is localized outside of lipid raft microdomains (PubMed:24855057). During the early stages of epithelial cell polarization induced by the calcium switch, it is removed from the plasma membrane to the endoplasmic reticulum, where it is retained, and redistributed to the basolateral cell surface in a PI3K-dependent manner at a later stage (PubMed:21228319). Colocalizes with SLC5A3 at the apical membrane of choroid plexus epithelium {ECO:0000250|UniProtKB:P97414, ECO:0000269|PubMed:10646604, ECO:0000269|PubMed:21228319, ECO:0000269|PubMed:23529131, ECO:0000269|PubMed:24855057}

**Tissue Location**

Abundantly expressed in heart, pancreas, prostate, kidney, small intestine and peripheral blood leukocytes. Less abundant in placenta, lung, spleen, colon, thymus, testis and ovaries

**KCNQ1 Antibody (Center) Blocking Peptide - Protocols**

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

- [Blocking Peptides](#)

**KCNQ1 Antibody (Center) Blocking Peptide - Images****KCNQ1 Antibody (Center) Blocking Peptide - Background**

KCNQ1 is a protein for a voltage-gated potassium channel required for the repolarization phase of the cardiac action potential. The gene product can form heteromultimers with two other potassium channel proteins, KCNE1 and KCNE3.

**KCNQ1 Antibody (Center) Blocking Peptide - References**

Holm,H., et.al., Nat. Genet. 42 (2), 117-122 (2010)Ohshige,T., et. al., Diabetes Care (2010) In press