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KCNQ1 (7E1) Mouse mAb

Catalog # AP53509

Product Information

Application	WB, FC
Primary Accession	P51787
Reactivity	Human
Host	Mouse
Clonality	Monoclonal Antibody
Isotype	IgG2b
Conjugate	Unconjugated
Immunogen	Purified recombinant fragment of human KCNQ1 expressed in E. Coli.
Purification	Ascitic Fluid
Calculated MW	74699

Additional Information

Gene ID	3784
Other Names	LQT; RWS; WRS; LQT1; SQT2; ATFB1; ATFB3; JLNS1; KCNA8; KCNA9; Kv1.9; Kv7.1; KVLQT1; FLJ26167
Dilution	WB~~1:3000 FC~~1:10~50
Format	Ascitic fluid containing 0.03% sodium azide.
Storage	Store at 4°C short term. Aliquot and store at -20°C long term. Avoid freeze/thaw cycles.

Protein Information

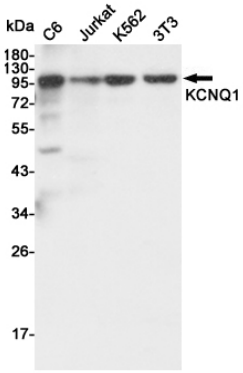
Name	KCNQ1 (HGNC:6294)
Function	Pore-forming subunit of the voltage-gated potassium (Kv) channel involved in the regulation of cardiomyocyte excitability and important in normal development and functions of myocardium, inner ear, stomach and colon (PubMed:10646604, PubMed:25441029). Associates with KCNE beta subunits that modulates current kinetics (PubMed:10646604, PubMed:11101505, PubMed:19687231, PubMed:8900283, PubMed:9108097, PubMed:9312006). Induces a voltage-dependent current by rapidly activating and slowly deactivating potassium-selective outward current (PubMed:10646604, PubMed:11101505, PubMed:25441029, PubMed:8900283, PubMed:9108097, PubMed:9312006). Also promotes 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:10646604, PubMed:11101505, PubMed:8900283, PubMed:9108097, PubMed:9312006). Muscarinic agonist oxotremorine-M strongly suppresses KCNQ1/KCNE1 current (PubMed:10713961). When associated with KCNE3, forms the potassium channel that is important for cyclic AMP-stimulated intestinal secretion of chloride ions (PubMed:10646604). 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:11101505). When associated with KCNE4, inhibits voltage-gated potassium channel activity (PubMed:19687231). When associated with KCNE5, this complex only conducts current upon strong and continued depolarization (PubMed:12324418). Also forms a heterotetramer with KCNQ5; has a voltage-gated potassium channel activity (PubMed:24855057). Binds with phosphatidylinositol 4,5-bisphosphate (PubMed:25037568). 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 (By similarity).
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

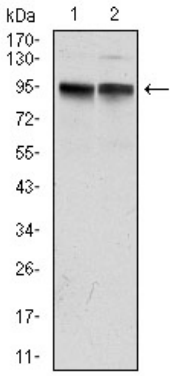
Background

Swiss-Prot Acc.P51787.This gene encodes a voltage-gated potassium channel required for repolarization phase of the cardiac action potential. This protein can form heteromultimers with two other potassium channel proteins, KCNE1 and KCNE3. Mutations in this gene are associated with hereditary long QT syndrome 1 (also known as Romano-Ward syndrome), Jervell and Lange-Nielsen syndrome, and familial atrial fibrillation. This gene exhibits tissue-specific imprinting, with preferential expression from the maternal allele in some tissues, and biallelic expression in others. This gene is located in a region of chromosome 11 amongst other imprinted genes that are associated with Beckwith-Wiedemann syndrome (BWS), and itself has been shown to be disrupted by chromosomal rearrangements in patients with BWS. Alternatively spliced transcript variants have been found for this gene.

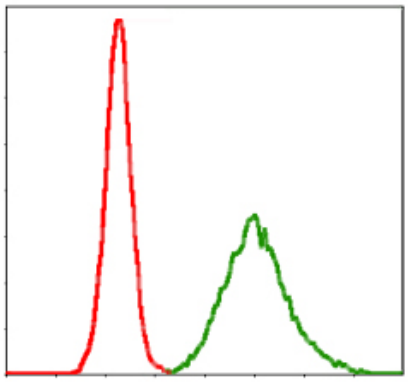
Images



Western blot detection of KCNQ1 in C6,Jurkat,K562 and 3T3 cell lysates using KCNQ1 mouse mAb (1:3000 diluted).Predicted band size:95KDa.Observed band size:95KDa.



Western blot analysis using KCNQ1 mouse mAb against MCF-7 (1) and A431 (2) cell lysate.



Flow cytometric analysis of MCF-7 cells using KCNQ1 mouse mAb (green) and negative control (red).

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.