

Product datasheet for **RC219869L1V**

KCNQ1 (NM_000218) Human Tagged ORF Clone Lentiviral Particle

Product data:

Product Type:	Lentiviral Particles
Product Name:	KCNQ1 (NM_000218) Human Tagged ORF Clone Lentiviral Particle
Symbol:	KCNQ1
Synonyms:	ATFB1; ATFB3; JLNS1; KCNA8; KCNA9; Kv1.9; Kv7.1; KVLQT1; LQT; LQT1; RWS; SQT2; WRS
Mammalian Cell Selection:	None
Vector:	pLenti-C-Myc-DDK (PS100064)
Tag:	Myc-DDK
ACCN:	NM_000218
ORF Size:	2028 bp
ORF Nucleotide Sequence:	The ORF insert of this clone is exactly the same as(RC219869).
OTI Disclaimer:	The molecular sequence of this clone aligns with the gene accession number as a point of reference only. However, individual transcript sequences of the same gene can differ through naturally occurring variations (e.g. polymorphisms), each with its own valid existence. This clone is substantially in agreement with the reference, but a complete review of all prevailing variants is recommended prior to use. More info
OTI Annotation:	This clone was engineered to express the complete ORF with an expression tag. Expression varies depending on the nature of the gene.
RefSeq:	NM_000218.2
RefSeq Size:	3262 bp
RefSeq ORF:	2031 bp
Locus ID:	3784
UniProt ID:	P51787
Cytogenetics:	11p15.5-p15.4
Protein Families:	Druggable Genome, Ion Channels: Potassium, Transmembrane
Protein Pathways:	Vibrio cholerae infection



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MW: 74.7 kDa

Gene Summary: 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. [provided by RefSeq, Aug 2011]