

## Product datasheet for **RC228816L4V**

### Kir6.2 (KCNJ11) (NM\_001166290) Human Tagged ORF Clone Lentiviral Particle

#### Product data:

Product Type:	Lentiviral Particles
Product Name:	Kir6.2 (KCNJ11) (NM_001166290) Human Tagged ORF Clone Lentiviral Particle
Symbol:	KCNJ11
Synonyms:	BIR; HHF2; IKATP; KIR6.2; MODY13; PHHI; PNDM2; TNDM3
Mammalian Cell Selection:	Puromycin
Vector:	pLenti-C-mGFP-P2A-Puro (PS100093)
Tag:	mGFP
ACCN:	NM_001166290
ORF Size:	909 bp
ORF Nucleotide Sequence:	The ORF insert of this clone is exactly the same as(RC228816).
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. <a href="#">More info</a>
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:	<a href="#">NM_001166290.1</a>
RefSeq Size:	2763 bp
RefSeq ORF:	912 bp
Locus ID:	3767
UniProt ID:	<a href="#">Q14654</a>
Cytogenetics:	11p15.1
Protein Families:	Druggable Genome, Ion Channels: Potassium, Transmembrane
Protein Pathways:	Type II diabetes mellitus



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MW: 33.2 kDa

**Gene Summary:** Potassium channels are present in most mammalian cells, where they participate in a wide range of physiologic responses. The protein encoded by this gene is an integral membrane protein and inward-rectifier type potassium channel. The encoded protein, which has a greater tendency to allow potassium to flow into a cell rather than out of a cell, is controlled by G-proteins and is found associated with the sulfonylurea receptor SUR. Mutations in this gene are a cause of familial persistent hyperinsulinemic hypoglycemia of infancy (PHHI), an autosomal recessive disorder characterized by unregulated insulin secretion. Defects in this gene may also contribute to autosomal dominant non-insulin-dependent diabetes mellitus type II (NIDDM), transient neonatal diabetes mellitus type 3 (TNDM3), and permanent neonatal diabetes mellitus (PNDM). Multiple alternatively spliced transcript variants that encode different protein isoforms have been described for this gene. [provided by RefSeq, Oct 2009]