

# Product datasheet for MR227409L3V

# Igf1r (NM\_010513) Mouse Tagged ORF Clone Lentiviral Particle

## **Product data:**

Product Type:	Lentiviral Particles
Product Name:	lgf1r (NM_010513) Mouse Tagged ORF Clone Lentiviral Particle
Symbol:	lgf1r
Synonyms:	A330103N21Rik; CD221; D930020L01; hyft; IGF-1R
Mammalian Cell Selection:	Puromycin
Vector:	pLenti-C-Myc-DDK-P2A-Puro (PS100092)
Tag:	Myc-DDK
ACCN:	NM_010513
ORF Size:	4107 bp
ORF Nucleotide Sequence:	The ORF insert of this clone is exactly the same as(MR227409).
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. <u>More info</u>
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:	<u>NM 010513.2, NP 034643.2</u>
RefSeq Size:	11978 bp
RefSeq ORF:	4110 bp
Locus ID:	16001
UniProt ID:	<u>Q60751</u>
Cytogenetics:	7 37.27 cM



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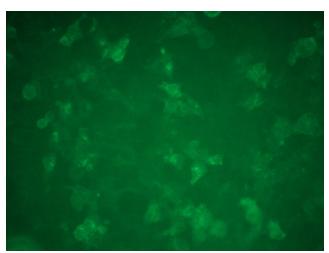
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#### Sourigene Igf1r (NM\_010513) Mouse Tagged ORF Clone Lentiviral Particle – MR227409L3V

Receptor tyrosine kinase which mediates actions of insulin-like growth factor 1 (IGF1). Binds Gene Summary: IGF1 with high affinity and IGF2 and insulin (INS) with a lower affinity. The activated IGF1R is involved in cell growth and survival control. IGF1R is crucial for tumor transformation and survival of malignant cell. Ligand binding activates the receptor kinase, leading to receptor autophosphorylation, and tyrosines phosphorylation of multiple substrates, that function as signaling adapter proteins including, the insulin-receptor substrates (IRS1/2), Shc and 14-3-3 proteins. Phosphorylation of IRSs proteins lead to the activation of two main signaling pathways: the PI3K-AKT/PKB pathway and the Ras-MAPK pathway. The result of activating the MAPK pathway is increased cellular proliferation, whereas activating the PI3K pathway inhibits apoptosis and stimulates protein synthesis. Phosphorylated IRS1 can activate the 85 kDa regulatory subunit of PI3K (PIK3R1), leading to activation of several downstream substrates, including protein AKT/PKB. AKT phosphorylation, in turn, enhances protein synthesis through mTOR activation and triggers the antiapoptotic effects of IGFIR through phosphorylation and inactivation of BAD. In parallel to PI3K-driven signaling, recruitment of Grb2/SOS by phosphorylated IRS1 or Shc leads to recruitment of Ras and activation of the ras-MAPK pathway. In addition to these two main signaling pathways IGF1R signals also through the Janus kinase/signal transducer and activator of transcription pathway (JAK/STAT). Phosphorylation of JAK proteins can lead to phosphorylation/activation of signal transducers and activators of transcription (STAT) proteins. In particular activation of STAT3, may be essential for the transforming activity of IGF1R. The JAK/STAT pathway activates gene transcription and may be responsible for the transforming activity. JNK kinases can also be activated by the IGF1R. IGF1 exerts inhibiting activities on INK activation via phosphorylation and inhibition of MAP3K5/ASK1, which is able to directly associate with the IGF1R (By similarity). When present in a hybrid receptor with INSR, binds IGF1 (By similarity). [UniProtKB/Swiss-Prot Function]

### **Product images:**



[MR227409L3] was used to prepare Lentiviral particles using [TR30037] packaging kit. HEK293T cells were transduced with MR227409L3V particle to overexpress human lgf1r-Myc-DDK fusion protein.

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