

## Product datasheet for **RC203786L1V**

### OLFM2 (NM\_058164) Human Tagged ORF Clone Lentiviral Particle

#### Product data:

Product Type:	Lentiviral Particles
Product Name:	OLFM2 (NM_058164) Human Tagged ORF Clone Lentiviral Particle
Symbol:	OLFM2
Synonyms:	NOE2; NOELIN2; NOELIN2_V1; OlfC
Mammalian Cell Selection:	None
Vector:	pLenti-C-Myc-DDK (PS100064)
Tag:	Myc-DDK
ACCN:	NM_058164
ORF Size:	1362 bp
ORF Nucleotide Sequence:	The ORF insert of this clone is exactly the same as(RC203786).
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_058164.1</a>
RefSeq Size:	1899 bp
RefSeq ORF:	1365 bp
Locus ID:	93145
UniProt ID:	<a href="#">O95897</a>
Cytogenetics:	19p13.2
Domains:	OLF
Protein Families:	Druggable Genome, Secreted Protein



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MW: 51.4 kDa

**Gene Summary:** Involved in transforming growth factor beta (TGF-beta)-induced smooth muscle differentiation. TGF-beta induces expression and translocation of OLFM2 to the nucleus where it binds to SRF, causing its dissociation from the transcriptional repressor HEY2/HERP1 and facilitating binding of SRF to target genes (PubMed:25298399). Plays a role in AMPAR complex organization (By similarity). Is a regulator of vascular smooth-muscle cell (SMC) phenotypic switching, that acts by promoting RUNX2 and inhibiting MYOCD binding to SRF. SMC phenotypic switching is the process through which vascular SMCs undergo transition between a quiescent contractile phenotype and a proliferative synthetic phenotype in response to pathological stimuli. SMC phenotypic plasticity is essential for vascular development and remodeling (By similarity).[UniProtKB/Swiss-Prot Function]