

Product datasheet for **RC208641L1V**

TNFRSF1B (NM_001066) Human Tagged ORF Clone Lentiviral Particle

Product data:

Product Type:	Lentiviral Particles
Product Name:	TNFRSF1B (NM_001066) Human Tagged ORF Clone Lentiviral Particle
Symbol:	TNFRSF1B
Synonyms:	CD120b; p75; p75TNFR; TBPII; TNF-R-II; TNF-R75; TNFBR; TNFR1B; TNFR2; TNFR80
Mammalian Cell Selection:	None
Vector:	pLenti-C-Myc-DDK (PS100064)
Tag:	Myc-DDK
ACCN:	NM_001066
ORF Size:	1383 bp
ORF Nucleotide Sequence:	The ORF insert of this clone is exactly the same as(RC208641).
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_001066.2
RefSeq Size:	3682 bp
RefSeq ORF:	1386 bp
Locus ID:	7133
UniProt ID:	P20333
Cytogenetics:	1p36.22
Domains:	TNFR
Protein Families:	Druggable Genome, Secreted Protein, Transmembrane



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Protein Pathways:	Adipocytokine signaling pathway, Amyotrophic lateral sclerosis (ALS), Cytokine-cytokine receptor interaction
MW:	48.29 kDa
Gene Summary:	<p>The protein encoded by this gene is a member of the TNF-receptor superfamily. This protein and TNF-receptor 1 form a heterocomplex that mediates the recruitment of two anti-apoptotic proteins, c-IAP1 and c-IAP2, which possess E3 ubiquitin ligase activity. The function of IAPs in TNF-receptor signalling is unknown, however, c-IAP1 is thought to potentiate TNF-induced apoptosis by the ubiquitination and degradation of TNF-receptor-associated factor 2, which mediates anti-apoptotic signals. Knockout studies in mice also suggest a role of this protein in protecting neurons from apoptosis by stimulating antioxidative pathways. [provided by RefSeq, Jul 2008]</p>