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Product datasheet for MR206499L3V

Tank (NM_001164072) Mouse Tagged ORF Clone Lentiviral Particle

Product data:

Product Type:	Lentiviral Particles
Product Name:	Tank (NM_001164072) Mouse Tagged ORF Clone Lentiviral Particle
Symbol:	Tank
Synonyms:	C86182; E430026L09Rik; I-TRAF
Mammalian Cell Selection:	Puromycin
Vector:	pLenti-C-Myc-DDK-P2A-Puro (PS100092)
Tag:	Myc-DDK
ACCN:	NM_001164072
ORF Size:	1242 bp
ORF Nucleotide Sequence:	The ORF insert of this clone is exactly the same as(MR206499).
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_001164072.1, NP_001157544.1
RefSeq Size:	2019 bp
RefSeq ORF:	1242 bp
Locus ID:	21353
UniProt ID:	P70347
Cytogenetics:	2 C1.3

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Gene Summary: Adapter protein involved in I-kappa-B-kinase (IKK) regulation which constitutively binds TBK1 and IKBKE playing a role in antiviral innate immunity. Acts as a regulator of TRAF function by maintaining them in a latent state. Blocks TRAF2 binding to LMP1 and inhibits LMP1-mediated NF-kappa-B activation. Negatively regulates NF-kappaB signaling and cell survival upon DNA damage. Plays a role as an adapter to assemble ZC3H12A, USP10 in a deubiquitination complex which plays a negative feedback response to attenuate NF-kappaB activation through the deubiquitination of IKBKG or TRAF6 in response to interleukin-1-beta (IL1B) stimulation or upon DNA damage. Promotes UBP10-induced deubiquitination of TRAF6 in response to DNA damage. May control negatively TRAF2-mediated NF-kappa-B activation signaled by CD40, TNFR1 and TNFR2. Essential for the efficient induction of IRF-dependent transcription following infection with Sendai virus.[UniProtKB/Swiss-Prot Function]