

Product datasheet for **MR207672L4V**

Akt3 (NM_011785) Mouse Tagged ORF Clone Lentiviral Particle

Product data:

Product Type:	Lentiviral Particles
Product Name:	Akt3 (NM_011785) Mouse Tagged ORF Clone Lentiviral Particle
Symbol:	Akt3
Synonyms:	AI851531; D930002M15Rik; Nmf350
Mammalian Cell Selection:	Puromycin
Vector:	pLenti-C-mGFP-P2A-Puro (PS100093)
Tag:	mGFP
ACCN:	NM_011785
ORF Size:	1440 bp
ORF Nucleotide Sequence:	The ORF insert of this clone is exactly the same as(MR207672).
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_011785.2 , NP_035915.2
RefSeq Size:	4735 bp
RefSeq ORF:	1440 bp
Locus ID:	23797
UniProt ID:	Q9WUA6
Cytogenetics:	1 H4



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Gene Summary:

AKT3 is one of 3 closely related serine/threonine-protein kinases (AKT1, AKT2 and AKT3) called the AKT kinase, and which regulate many processes including metabolism, proliferation, cell survival, growth and angiogenesis. This is mediated through serine and/or threonine phosphorylation of a range of downstream substrates. Over 100 substrate candidates have been reported so far, but for most of them, no isoform specificity has been reported. AKT3 is the least studied AKT isoform. It plays an important role in brain development and is crucial for the viability of malignant glioma cells. AKT3 isoform may also be the key molecule in up-regulation and down-regulation of MMP13 via IL13. Required for the coordination of mitochondrial biogenesis with growth factor-induced increases in cellular energy demands. Down-regulation by RNA interference reduces the expression of the phosphorylated form of BAD, resulting in the induction of caspase-dependent apoptosis. [UniProtKB/Swiss-Prot Function]