

Product datasheet for MR216538L4V

OriGene Technologies, Inc.

9620 Medical Center Drive, Ste 200 Rockville, MD 20850, US Phone: +1-888-267-4436 https://www.origene.com techsupport@origene.com EU: info-de@origene.com CN: techsupport@origene.cn

Znrf1 (NM_001168623) Mouse Tagged ORF Clone Lentiviral Particle

Product data:

Product Type: Lentiviral Particles

Product Name: Znrf1 (NM_001168623) Mouse Tagged ORF Clone Lentiviral Particle

Symbol: Znrf1

Synonyms: B830022L21Rik; nin283; Rnf42; Zrfp1

Mammalian Cell

Selection:

Puromycin

Vector: pLenti-C-mGFP-P2A-Puro (PS100093)

Tag: mGFP

ACCN: NM_001168623

ORF Size: 381 bp

ORF Nucleotide

The ORF insert of this clone is exactly the same as(MR216538).

OTI Disclaimer:

Sequence:

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: <u>NM 001168623.1</u>

 RefSeq Size:
 3650 bp

 RefSeq ORF:
 384 bp

 Locus ID:
 170737

 UniProt ID:
 Q91V17

 Cytogenetics:
 8 E1







Gene Summary:

E3 ubiquitin-protein ligase that mediates the ubiquitination of AKT1 and GLUL, thereby playing a role in neuron cells differentiation. Plays a role in the establishment and maintenance of neuronal transmission and plasticity. Regulates Schwann cells differentiation by mediating ubiquitination of GLUL. Promotes Wallerian degeneration, a neurodegeneration disorder, by mediating 'Lys-48'-linked polyubiquitination and subsequent degradation of AKT1 in axons: degradation of AKT1 prevents AKT1-mediated phosphorylation of GSK3B, leading to GSK3B activation and phosphorylation of DPYSL2/CRMP2 followed by destabilization of microtubule assembly in axons.[UniProtKB/Swiss-Prot Function]