10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

MUL1 Blocking Peptide (Center)

Synthetic peptide
Catalog # BP20808c

Specification

MUL1 Blocking Peptide (Center) - Product Information

Primary Accession	Q969V5
Other Accession	Q8VCM5, Q4R7G8

MUL1 Blocking Peptide (Center) - Additional Information

Gene ID 79594

Other Names
Mitochondrial ubiquitin ligase activator of NFKB 1, 632-, E3 SUMO-protein ligase MUL1, E3 ubiquitin-protein ligase MUL1, Growth inhibition and death E3 ligase, Mitochondrial-anchored protein ligase, MAPL, Putative NF-kappa-B-activating protein 266, RING finger protein 218, MUL1, C1orf166, GIDE, MAPL, MULAN, RNF218

Target/Specificity
The synthetic peptide sequence is selected from aa 176-190 of HUMAN MUL1

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

MUL1 Blocking Peptide (Center) - Protein Information

Name MUL1

Synonyms C1orf166, GIDE, MAPL, MULAN, RNF218

Function
Exhibits weak E3 ubiquitin-protein ligase activity (PubMed:18591963, PubMed:19407830, PubMed:22410793). E3 ubiquitin ligases accept ubiquitin from an E2 ubiquitin-conjugating enzyme in the form of a thioester and then directly transfer the ubiquitin to targeted substrates (PubMed:18591963, PubMed:19407830, PubMed:22410793). Can

ubiquitinate AKT1 preferentially at 'Lys-284' involving 'Lys-48'-linked polyubiquitination and seems to be involved in regulation of Akt signaling by targeting phosphorylated Akt to proteasomal degradation (PubMed:22410793). Mediates polyubiquitination of cytoplasmic TP53 at 'Lys-24' which targets TP53 for proteasomal degradation, thus reducing TP53 levels in the cytoplasm and mitochondrion (PubMed:21597459). Proposed to preferentially act as a SUMO E3 ligase at physiological concentrations (PubMed:19407830). Plays a role in the control of mitochondrial morphology by promoting mitochondrial fragmentation, and influences mitochondrial localization (PubMed:19407830, PubMed:18207745, PubMed:18213395). Likely to promote mitochondrial fission through negatively regulating the mitochondrial fusion proteins MFN1 and MFN2, acting in a pathway that is parallel to the PRKN/PINK1 regulatory pathway (PubMed:24898855). May also be involved in the sumoylation of the membrane fission protein DNM1L (PubMed:18207745, PubMed:19407830). Inhibits cell growth (PubMed:18591963, PubMed:22410793). When overexpressed, activates JNK through MAP3K7/TAK1 and induces caspase-dependent apoptosis (PubMed:23399697). Involved in the modulation of innate immune defense against viruses by inhibiting RIGI-dependent antiviral response (PubMed:23399697). Can mediate RIGI sumoylation and disrupt its polyubiquitination (PubMed:23399697).

Cellular Location
Mitochondrion outer membrane; Multi-pass membrane protein. Peroxisome. Note=Transported in mitochondrion- derived vesicles from the mitochondrion to the peroxisome

Tissue Location
Widely expressed with highest levels in the heart, skeletal muscle, placenta, kidney and liver. Barely detectable in colon and thymus.

MUL1 Blocking Peptide (Center) - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Blocking Peptides

MUL1 Blocking Peptide (Center) - Images

MUL1 Blocking Peptide (Center) - Background

Exhibits weak E3 ubiquitin-protein ligase activity. E3 ubiquitin ligases accept ubiquitin from an E2 ubiquitin- conjugating enzyme in the form of a thioester and then directly transfer the ubiquitin to targeted substrates. Can ubiquinate AKT1 preferentially at 'Lys-284' involving 'Lys-48'-linked polyubiquitination and seems to be involved in regulation of Akt signaling by targeting phosphorylated Akt to proteosomal degradation. Proposed to preferentially act as a SUMO E3 ligase at physiological concentrations. Plays a role in the control of mitochondrial morphology. Promotes mitochondrial fragmentation and influences mitochondrial localization. The function may implicate its abilty to sumoylate DNM1L. Inhibits cell growth. When overexpressed, activates JNK through MAP3K7/TAK1 and induces caspase-dependent apoptosis. Involved in the modulation of innate

immune defense against viruses by inhibiting DDX58-dependent antiviral response. Can mediate DDX58 sumoylation and disrupt its polyubiquitination.

MUL1 Blocking Peptide (Center) - References

Zhang B.,et al.Cell Res. 18:900-910(2008).
Matsuda A.,et al.Oncogene 22:3307-3318(2003).
Ota T.,et al.Nat. Genet. 36:40-45(2004).
Bechtel S.,et al.BMC Genomics 8:399-399(2007).
Gregory S.G.,et al.Nature 441:315-321(2006).