

MAD2L2 Blocking Peptide (C-term)

Synthetic peptide Catalog # BP20654c

Specification

MAD2L2 Blocking Peptide (C-term) - Product Information

Primary Accession

09UI95

MAD2L2 Blocking Peptide (C-term) - Additional Information

Gene ID 10459

Other Names

Mitotic spindle assembly checkpoint protein MAD2B, Mitotic arrest deficient 2-like protein 2, MAD2-like protein 2, REV7 homolog, hREV7, MAD2L2, MAD2B, REV7

Target/Specificity

The synthetic peptide sequence is selected from aa 198-211 of HUMAN MAD2L2

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.

MAD2L2 Blocking Peptide (C-term) - Protein Information

Name MAD2L2

Synonyms MAD2B, REV7

Function

Adapter protein able to interact with different proteins and involved in different biological processes (PubMed:11459825, PubMed:11459826, PubMed:17719540, PubMed:17296730, PubMed:19443654, PubMed:29656893, Mediates the interaction between the error-prone DNA polymerase zeta catalytic subunit REV3L and the inserter polymerase REV1, thereby mediating the second polymerase switching in translesion DNA synthesis (PubMed:20164194). Translesion DNA synthesis releases the replication blockade of replicative polymerases, stalled in presence of



DNA lesions (PubMed:20164194). Component of the shieldin complex, which plays an important role in repair of DNA double-stranded breaks (DSBs) (PubMed: 29656893). During G1 and S phase of the cell cycle, the complex functions downstream of TP53BP1 to promote non-homologous end joining (NHEJ) and suppress DNA end resection (PubMed: 29656893). Mediates various NHEJ-dependent processes including immunoglobulin class-switch recombination, and fusion of unprotected telomeres (PubMed:29656893). May also regulate another aspect of cellular response to DNA damage through regulation of the JNK-mediated phosphorylation and activation of the transcriptional activator ELK1 (PubMed:17296730). Inhibits the FZR1- and probably CDC20-mediated activation of the anaphase promoting complex APC thereby regulating progression through the cell cycle (PubMed:11459825, PubMed:17719540). Regulates TCF7L2-mediated gene transcription and may play a role in epithelial-mesenchymal transdifferentiation (PubMed:19443654).

Cellular Location

Nucleus. Cytoplasm, cytoskeleton, spindle. Cytoplasm. Chromosome. Note=Recruited to sites of chromosomal double-stranded breaks during G1 and S phase of the cell cycle

Tissue Location

Ubiquitously expressed.

MAD2L2 Blocking Peptide (C-term) - Protocols

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

• Blocking Peptides

MAD2L2 Blocking Peptide (C-term) - Images

MAD2L2 Blocking Peptide (C-term) - Background

Adapter protein able to interact with different proteins and involved in different biological processes. Mediates the interaction between the error-prone DNA polymerase zeta catalytic subunit REV3L and the inserter polymerase REV1, thereby mediating the second polymerase switching in translesion DNA synthesis. Translesion DNA synthesis releases the replication blockade of replicative polymerases, stalled in presence of DNA lesions. May also regulate another aspect of cellular response to DNA damage through regulation of the JNK-mediated phosphorylation and activation of the transcriptional activator ELK1. Inhibits the FZR1- and probably CDC20-mediated activation of the anaphase promoting complex APC thereby regulating progression through the cell cycle. Regulates TCF7L2-mediated gene transcription and may play a role in epithelial-mesenchymal transdifferentiation.

MAD2L2 Blocking Peptide (C-term) - References

Nelson K.K.,et al.Biochem. J. 343:673-680(1999). Hirota T.,et al.Submitted (DEC-1999) to the EMBL/GenBank/DDBJ databases. Cahill D.P.,et al.Genomics 58:181-187(1999). Murakumo Y.,et al.J. Biol. Chem. 275:4391-4397(2000). Ota T.,et al.Nat. Genet. 36:40-45(2004).