

RBBP8 Antibody (C-term) Blocking peptide

Synthetic peptide Catalog # BP14029b

Specification

RBBP8 Antibody (C-term) Blocking peptide - Product Information

Primary Accession

RBBP8 Antibody (C-term) Blocking peptide - Additional Information

Gene ID 5932

Other Names

DNA endonuclease RBBP8, 31--, CtBP-interacting protein, CtIP, Retinoblastoma-binding protein 8, RBBP-8, Retinoblastoma-interacting protein and myosin-like, RIM, Sporulation in the absence of SPO11 protein 2 homolog, SAE2, RBBP8, CTIP

Q99708

Target/Specificity

The synthetic peptide sequence used to generate the antibody AP14029b was selected from the C-term region of RBBP8. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

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.

RBBP8 Antibody (C-term) Blocking peptide - Protein Information

Name RBBP8

Synonyms CTIP

Function

Endonuclease that cooperates with the MRE11-RAD50-NBN (MRN) complex in DNA-end resection, the first step of double-strand break (DSB) repair through the homologous recombination (HR) pathway (PubMed:17965729, PubMed:19202191, PubMed:19759395, PubMed:20064462, PubMed:26721387). HR is restricted to S and G2 phases of the cell cycle and preferentially repairs DSBs resulting from replication fork collapse (PubMed:<a



href="http://www.uniprot.org/citations/17965729" target=" blank">17965729, PubMed:19202191). Key determinant of DSB repair pathway choice, as it commits cells to HR by preventing classical non-homologous end-joining (NHEI) (PubMed:19202191). Functions downstream of the MRN complex and ATM, promotes ATR activation and its recruitment to DSBs in the S/G2 phase facilitating the generation of ssDNA (PubMed:16581787, PubMed: 17965729, PubMed: 19759395, PubMed:20064462). Component of the BRCA1-RBBP8 complex that regulates CHEK1 activation and controls cell cycle G2/M checkpoints on DNA damage (PubMed: 15485915, PubMed:16818604). During immunoglobulin heavy chain class-switch recombination, promotes microhomology-mediated alternative end joining (A-NHEJ) and plays an essential role in chromosomal translocations (By similarity). Binds preferentially to DNA Y-junctions and to DNA substrates with blocked ends and promotes intermolecular DNA bridging (PubMed: 30601117).

Cellular Location

Nucleus. Chromosome. Note=Associates with sites of DNA damage in S/G2 phase (PubMed:10764811, PubMed:25349192). Ubiquitinated RBBP8 binds to chromatin following DNA damage (PubMed:16818604)

Tissue Location

Expressed in ER-positive breast cancer lines, but tends to be down-regulated ER-negative cells (at protein level)

RBBP8 Antibody (C-term) Blocking peptide - Protocols

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

• Blocking Peptides

RBBP8 Antibody (C-term) Blocking peptide - Images

RBBP8 Antibody (C-term) Blocking peptide - Background

The protein encoded by this gene is a ubiquitouslyexpressed nuclear protein. It is found among several proteins thatbind directly to retinoblastoma protein, which regulates cellproliferation. This protein complexes with transcriptionalco-repressor CTBP. It is also associated with BRCA1 and is thoughtto modulate the functions of BRCA1 in transcriptional regulation, DNA repair, and/or cell cycle checkpoint control. It is suggested that this gene may itself be a tumor suppressor acting in the same pathway as BRCA1. Three transcript variants encoding two differentisoforms have been found for this gene. More transcript variants exist, but their full-length natures have not been determined.

RBBP8 Antibody (C-term) Blocking peptide - References

Kaidi, A., et al. Science 329(5997):1348-1353(2010)Thye, T., et al. Nat. Genet. 42(9):739-741(2010)Notaridou, M., et al. Int. J. Cancer (2010) In press :Yasuno, K., et al. Nat. Genet. 42(5):420-425(2010)Zhao, J., et al. BMC Med. Genet. 11, 96 (2010):