

ATR Antibody (C-term) Blocking Peptide
Synthetic peptide
Catalog # BP8047b**Specification**

ATR Antibody (C-term) Blocking Peptide - Product InformationPrimary Accession [Q13535](#)**ATR Antibody (C-term) Blocking Peptide - Additional Information****Gene ID** 545**Other Names**

Serine/threonine-protein kinase ATR, Ataxia telangiectasia and Rad3-related protein, FRAP-related protein 1, ATR, FRP1

Target/Specificity

The synthetic peptide sequence used to generate the antibody [AP8047b](/product/products/AP8047b) was selected from the C-term region of human ATR. 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.

ATR Antibody (C-term) Blocking Peptide - Protein Information**Name** ATR {ECO:0000303|PubMed:14729973, ECO:0000312|HGNC:HGNC:882}**Function**

Serine/threonine protein kinase which activates checkpoint signaling upon genotoxic stresses such as ionizing radiation (IR), ultraviolet light (UV), or DNA replication stalling, thereby acting as a DNA damage sensor (PubMed:[10597277](http://www.uniprot.org/citations/10597277), PubMed:[10608806](http://www.uniprot.org/citations/10608806), PubMed:[10859164](http://www.uniprot.org/citations/10859164), PubMed:[11721054](http://www.uniprot.org/citations/11721054), PubMed:[12791985](http://www.uniprot.org/citations/12791985), PubMed:[12814551](http://www.uniprot.org/citations/12814551), PubMed:[14657349](http://www.uniprot.org/citations/14657349), PubMed:[14729973](http://www.uniprot.org/citations/14729973), PubMed:[14742437](http://www.uniprot.org/citations/14742437))

target="_blank">14742437, PubMed:15210935, PubMed:15496423, PubMed:16260606, PubMed:21144835, PubMed:27723717, PubMed:27723720, PubMed:33848395, PubMed:9427750, PubMed:9636169, PubMed:21777809, PubMed:25083873, PubMed:30139873, PubMed:37788673, PubMed:37832547). Recognizes the substrate consensus sequence [ST]-Q (PubMed:10597277, PubMed:10608806, PubMed:10859164, PubMed:11721054, PubMed:12791985, PubMed:12814551, PubMed:14657349, PubMed:14729973, PubMed:14742437, PubMed:15210935, PubMed:15496423, PubMed:16260606, PubMed:21144835, PubMed:27723717, PubMed:27723720, PubMed:33848395, PubMed:9427750, PubMed:9636169). Phosphorylates BRCA1, CHEK1, MCM2, RAD17, RPA2, SMC1 and p53/TP53, which collectively inhibit DNA replication and mitosis and promote DNA repair, recombination and apoptosis (PubMed:9925639, PubMed:11114888, PubMed:11418864, PubMed:11865061, PubMed:21777809, PubMed:25083873). Phosphorylates 'Ser-139' of histone variant H2AX at sites of DNA damage, thereby regulating DNA damage response mechanism (PubMed:11673449). Required for FANCD2 ubiquitination (PubMed:15314022). Critical for maintenance of fragile site stability and efficient regulation of centrosome duplication (PubMed:12526805). Acts as a regulator of the S-G2 transition by restricting the activity of CDK1 during S-phase to prevent premature entry into G2 (PubMed:30139873). Acts as a regulator of the nuclear envelope integrity in response to DNA damage and stress (PubMed:25083873, PubMed:37788673, PubMed:37832547). Acts as a mechanical stress sensor at the nuclear envelope: relocalizes to the nuclear envelope in response to mechanical stress and mediates a checkpoint via phosphorylation of CHEK1 (PubMed:25083873). Also

promotes nuclear envelope rupture in response to DNA damage by mediating phosphorylation of LMNA at 'Ser-282', leading to lamin disassembly (PubMed:37832547). Involved in the inflammatory response to genome instability and double-stranded DNA breaks: acts by localizing to micronuclei arising from genome instability and catalyzing phosphorylation of LMNA at 'Ser-395', priming LMNA for subsequent phosphorylation by CDK1 and micronuclei envelope rupture (PubMed:37788673). The rupture of micronuclear envelope triggers the cGAS-STING pathway thereby activating the type I interferon response and innate immunity (PubMed:37788673). Positively regulates the restart of stalled replication forks following activation by the KHDC3L-OOEP scaffold complex (By similarity).

Cellular Location

Nucleus. Chromosome. Nucleus envelope. Note=Depending on the cell type, it can also be found in PML nuclear bodies (PubMed:12814551). Recruited to chromatin during S-phase (PubMed:14871897). Redistributes to discrete nuclear foci upon DNA damage, hypoxia or replication fork stalling (PubMed:27723720). Relocalizes to the nuclear envelope in response to mechanical stress or DNA damage (PubMed:25083873, PubMed:37832547) Also localizes to the micronuclear envelope in response to response to genome instability (PubMed:37788673).

Tissue Location

Ubiquitous, with highest expression in testis.

ATR Antibody (C-term) Blocking Peptide - Protocols

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

- [Blocking Peptides](#)

ATR Antibody (C-term) Blocking Peptide - Images

ATR Antibody (C-term) Blocking Peptide - Background

The protein encoded by this gene belongs to the PI3/PI4-kinase family, and is most closely related to ATM, a protein kinase encoded by the gene mutated in ataxia telangiectasia. This protein and ATM share similarity with *Schizosaccharomyces pombe* rad3, a cell cycle checkpoint gene required for cell cycle arrest and DNA damage repair in response to DNA damage. This kinase has been shown to phosphorylate checkpoint kinase CHK1, checkpoint proteins RAD17, and RAD9, as well as tumor suppressor protein BRCA1. Mutations of this gene are associated with Seckel syndrome. An alternatively spliced transcript variant of this gene has been reported, however, its full length nature is not known. Transcript variants utilizing alternative polyA sites exist.

ATR Antibody (C-term) Blocking Peptide - References

Blume-Jensen P, et al. Nature 2001. 411: 355. Cantrell D, J. Cell Sci. 2001. 114: 1439. Jhng S. Oncogene 2000. 19: 5590. Manning G, et al. Science 2002. 298: 1912. Moller, D, et al. Am. J. Physiol. 1994. 266: C351-C359. Robertson, S. et al. Trends Genet. 2000. 16: 368. Robinson D, et al. Oncogene 2000. 19: 5548. Van der Ven, P, et al. Hum. Molec. Genet. 1993. 2: 1889. Vanhaesebroeck, B, et al. Biochem. J. 2000. 346: 561. Van Weering D, et al. Recent Results Cancer Res. 1998. 154: 271.