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ATM Antibody (C-term) Blocking Peptide

Synthetic peptide
Catalog # BP8046b

Specification

ATM Antibody (C-term) Blocking Peptide - Product Information

Primary Accession Q13315

ATM Antibody (C-term) Blocking Peptide - Additional Information

Gene ID 472

Other Names
Serine-protein kinase ATM, Ataxia telangiectasia mutated, A-T mutated, ATM

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP8046b was selected from the C-term region of human ATM . 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.

ATM Antibody (C-term) Blocking Peptide - Protein Information

Name ATM

Function
Serine/threonine protein kinase which activates checkpoint signaling upon double strand breaks (DSBs), apoptosis and genotoxic stresses such as ionizing ultraviolet A light (UVA), thereby acting as a DNA damage sensor (PubMed:9733514, PubMed:10550055, PubMed:10839545, PubMed:10910365, PubMed:12556884, PubMed:14871926, PubMed:15456891, PubMed:15448695, PubMed:15916964, PubMed:<a href="http://www.uniprot.org/citations/17923702"

target="_blank">17923702). Recognizes the substrate consensus sequence [ST]-Q (PubMed:9733514, PubMed:10550055, PubMed:10839545, PubMed:10910365, PubMed:12556884, PubMed:14871926, PubMed:15456891, PubMed:15448695, PubMed:15916964, PubMed:17923702). Phosphorylates 'Ser-139' of histone variant H2AX at double strand breaks (DSBs), thereby regulating DNA damage response mechanism (By similarity). Also plays a role in pre-B cell allelic exclusion, a process leading to expression of a single immunoglobulin heavy chain allele to enforce clonality and monospecific recognition by the B-cell antigen receptor (BCR) expressed on individual B-lymphocytes. After the introduction of DNA breaks by the RAG complex on one immunoglobulin allele, acts by mediating a repositioning of the second allele to pericentromeric heterochromatin, preventing accessibility to the RAG complex and recombination of the second allele. Also involved in signal transduction and cell cycle control. May function as a tumor suppressor. Necessary for activation of ABL1 and SAPK. Phosphorylates DYRK2, CHEK2, p53/TP53, FBXW7, FANCD2, NFKBIA, BRCA1, CTIP, nibrin (NBN), TERF1, UFL1, RAD9, UBQLN4 and DCLRE1C (PubMed:9843217, PubMed:9733515, PubMed:10550055, PubMed:10766245, PubMed:10839545, PubMed:10910365, PubMed:10802669, PubMed:10973490, PubMed:11375976, PubMed:12086603, PubMed:15456891, PubMed:19965871, PubMed:30612738, PubMed:30886146, PubMed:26774286). May play a role in vesicle and/or protein transport. Could play a role in T-cell development, gonad and neurological function. Plays a role in replication-dependent histone mRNA degradation. Binds DNA ends. Phosphorylation of DYRK2 in nucleus in response to genotoxic stress prevents its MDM2-mediated ubiquitination and subsequent proteasome degradation (PubMed:19965871). Phosphorylates ATF2 which stimulates its function in DNA damage response (PubMed:15916964). Phosphorylates ERCC6 which is essential for its chromatin remodeling activity at DNA double-strand breaks (PubMed:29203878). Phosphorylates TTC5/STRAP at 'Ser-203' in the cytoplasm in response to DNA damage, which promotes TTC5/STRAP nuclear localization (PubMed:15448695). Also involved in pexophagy by mediating phosphorylation of PEX5: translocated to peroxisomes in response to reactive oxygen species (ROS), and catalyzes phosphorylation of PEX5, promoting PEX5 ubiquitination and induction of pexophagy (PubMed:26344566).

Cellular Location

Nucleus. Cytoplasmic vesicle. Cytoplasm, cytoskeleton, microtubule organizing center, centrosome {ECO:0000250|UniProtKB:Q62388}. Peroxisome matrix. Note=Primarily nuclear (PubMed:9050866, PubMed:9150358). Found also in endocytic vesicles in association with

beta-adaptin (PubMed:9707615). Translocated to peroxisomes in response to reactive oxygen species (ROS) by PEX5 (PubMed:26344566)

Tissue Location
Found in pancreas, kidney, skeletal muscle, liver, lung, placenta, brain, heart, spleen, thymus, testis, ovary, small intestine, colon and leukocytes

ATM Antibody (C-term) Blocking Peptide - Protocols

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

- Blocking Peptides

ATM Antibody (C-term) Blocking Peptide - Images

ATM Antibody (C-term) Blocking Peptide - Background

ATM is involved in signal transduction, cell cycle control and DNA repair, and may function as a tumor suppressor. It is necessary for activation of ABL1 and SAPK, and phosphorylates p53, NFKBIA, BRCA1, CTIP, NIBRIN (NBS1), TERF1, and RAD9. This protein has potential roles in vesicle and/or protein transport, T-cell development, gonad and neurological function. ATM is also part of the BRCA1-associated genome surveillance complex. ATM is induced by ionizing radiation. Defects in ATM are the cause of ataxia talangiectasia (AT), also known as Louis-Bar syndrome, a rare recessive disorder characterized by progressive cerebellar ataxia, dilation of the blood vessels in the conjunctiva and eyeballs, immunodeficiency, growth retardation and sexual immaturity. About 30% of AT patients develop lymphomas and leukemias. Defects in ATM also contribute to T-cell acute lymphoblastic leukemia (TALL) and T-prolymphocytic leukemia (TPLL). TPLL is characterized by a high white blood cell count, with a predominance of prolymphocytes, marked splenomegaly, lymphadenopathy, skin lesions and serous effusion. Defects in ATM also contribute to B-cell non-Hodgkin's lymphomas, and to B-cell chronic lymphocytic leukemia, a disease characterized by accumulation of mature CD5+ B lymphocytes, lymphadenopathy, immunodeficiency and bone marrow failure.

ATM Antibody (C-term) Blocking Peptide - References

Suzuki, A., et al., J. Biol. Chem. 278(1):48-53 (2003).Kishi, S., et al., J. Biol. Chem. 276(31):29282-29291 (2001).Schaffner, C., et al., Proc. Natl. Acad. Sci. U.S.A. 97(6):2773-2778 (2000).Gatei, M., et al., Nat. Genet. 25(1):115-119 (2000).Becker-Catania, S.G., et al., Mol. Genet. Metab. 70(2):122-133 (2000).