

ATM Antibody (N-term)

Purified Rabbit Polyclonal Antibody (Pab) Catalog # AP8046a

Specification

ATM Antibody (N-term) - Product Information

Application
Primary Accession
Reactivity
Host
Clonality
Isotype
Antigen Region

WB, IHC-P,E
013315
Human
Rabbit
Polyclonal
Rabbit Ig
5-34

ATM Antibody (N-term) - Additional Information

Gene ID 472

Other Names

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

Target/Specificity

This ATM antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 5~34 amino acids from the N-terminal region of human ATM.

Dilution

WB~~1:500 IHC-P~~1:50~100

Format

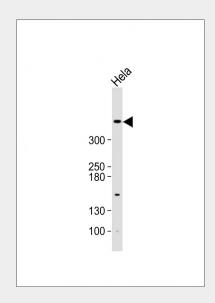
Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.

Storage

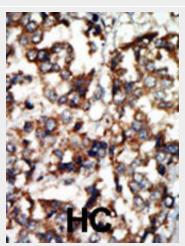
Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

ATM Antibody (N-term) is for research use only and not for use in diagnostic or therapeutic procedures.



Anti-ATM Antibody (C12) at 1:500 dilution + Hela whole cell lysate Lysates/proteins at 20 µg per lane. Secondary Goat Anti-Rabbit lgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size: 351 kDa Blocking/Dilution buffer: 5% NFDM/TBST.



Formalin-fixed and paraffin-embedded human cancer tissue reacted with the primary antibody, which was peroxidase-conjugated to the secondary antibody, followed by DAB staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has



ATM Antibody (N-term) - 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. Recognizes the substrate consensus sequence [ST]- Q. Phosphorylates 'Ser-139' of histone variant H2AX at double strand breaks (DSBs), thereby regulating DNA damage response mechanism. 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, FANCD2, NFKBIA, BRCA1, CTIP, nibrin (NBN), TERF1, UFL1, RAD9, UBQLN4 and DCLRE1C (PubMed:9843217, PubMed:<a href="http://www.uniprot.org/ci

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not been evaluated. BC = breast carcinoma; HC = hepatocarcinoma.

ATM Antibody (N-term) - 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 (N-term) - 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).



target=" blank">10973490, PubMed:11375976, PubMed:12086603, PubMed:15456891, PubMed: tations/19965871" target=" blank">19965871, PubMed:30612738, PubMed:30886146). 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. Phosphorylates ATF2 which stimulates its function in DNA damage response. Phosphorylates ERCC6 which is essential for its chromatin remodeling activity at DNA double-strand breaks (PubMed: 29203878).

Cellular Location

Nucleus. Cytoplasmic vesicle Note=Primarily nuclear. Found also in endocytic vesicles in association with beta-adaptin.

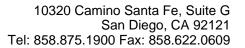
Tissue Location

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

ATM Antibody (N-term) - Protocols

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

- Western Blot
- Blocking Peptides





- Dot Blot
- <u>Immunohistochemistry</u>
- <u>Immunofluorescence</u>
- <u>Immunoprecipitation</u>
- Flow Cytomety
- Cell Culture

ATM Antibody (N-term) - Citations

• <u>Mitochondrial Dysfunctions Regulated Radioresistance through Mitochondria-to-Nucleus Retrograde Signaling Pathway of NF-κB/PI3K/AKT2/mTOR.</u>