abcam

ab36810

Anti-ATM (phospho S1981) antibody [10H11.E12]

Key facts

Isotype	IgG1
Host species	Mouse
Storage buffer	pH: 7.4 Preservative: 0.02% Sodium azide Constituents: PBS, 6.97% L-Arginine
Form	Liquid
Clonality	Monoclonal
Immunogen	The exact immunogen used to generate this antibody is proprietary information.
Clone number	10H11.E12
Purification technique	Affinity purification
Light chain type	kappa
Concentration	1 mg/mL The concentration of this product may be batch-dependent Batch concentration finder →

Reactivity data

IHC-P

Tested

Species	Human
Dilution info	6 µg/mL
Notes	Perform heat-mediated antigen retrieval before commencing with IHC staining protocol.

Flow Cyt (Intra)

Tested

Species	Human
Dilution info	1 µg for 10^5 Cells
Notes	ab170190 - Mouse monoclonal IgG1, is suitable for use as an isotype control with this antibody.

WB

Tested

Species	Human
Dilution info	1/1000
Notes	Abcam recommends using 3% Milk as the blocking agent.

Target data

See full target information ATM pS1981

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:10550055, PubMed:10839545, PubMed:10910365, PubMed:12556884, PubMed:14871926, PubMed:15064416, PubMed:15448695, PubMed:15456891, PubMed:15790808, PubMed:15916964, PubMed:17923702, PubMed:21757780, PubMed:24534091, PubMed:35076389, PubMed:9733514). Recognizes the substrate consensus sequence [ST]-Q (PubMed:10550055, PubMed:10839545, PubMed:10910365, PubMed:12556884, PubMed:14871926, PubMed:15448695, PubMed:15456891, PubMed:15916964, PubMed:17923702, PubMed:24534091, PubMed:9733514). 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, CREBBP/CBP, RBBP8/CTIP, FBXO46, MRE11, nibrin (NBN), RAD50, RAD17, PELI1, TERF1, UFL1, RAD9, UBQLN4 and DCLRE1C (PubMed:10550055, PubMed:10766245, PubMed:10802669, PubMed:10839545, PubMed:10910365, PubMed:10973490, PubMed:11375976, PubMed:12086603, PubMed:15456891,

PubMed:19965871, PubMed:21757780, PubMed:24534091, PubMed:26240375, PubMed:26774286, PubMed:30171069, PubMed:30612738, PubMed:30886146, PubMed:30952868, PubMed:38128537, PubMed:9733515, PubMed:9843217). 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).

Storage

Shipped at conditions	Blue Ice
Appropriate short-term storage conditions	+4°C
Appropriate long-term storage conditions	-20°C
Aliquoting information	Upon delivery aliquot
Storage information	Avoid freeze / thaw cycle

Notes

Want a custom formulation?
This antibody clone is manufactured by Abcam. If you require a custom buffer formulation or conjugation for your experiments, please contact orders@abcam.com

Supplementary info

This supplementary information is collated from multiple sources and compiled automatically.

Activity summary	ATM also known as Ataxia Telangiectasia Mutated is a protein kinase with a molecular weight of approximately 370 kDa. ATM protein primarily resides in the cell nucleus and functions as a critical regulator of the cell cycle. It plays a significant role in the detection of DNA damage and initiation of repair processes. As part of its mechanical functions ATM phosphorylates serine and threonine

	residues on various substrates most notably in response to double-strand breaks in DNA. This activity is important for maintaining genomic stability.
Biological function summary	ATM acts as a coordinator in cellular response to DNA damage highly interacting with multiple components of the DNA repair machinery. It forms a complex with proteins like NBS1 and MRN complex facilitating repair by recruiting and activating other proteins involved in homologous recombination and non-homologous end joining pathways. ATM also modulates p53 activity a primary response factor in cellular stress management linking ATM to control of cell cycle arrest and apoptosis. This positions ATM as an integral part of maintaining cellular integrity in face of genomic insult.
Pathways	ATM integrates neatly within the DNA damage response and cell cycle control pathways. ATM's operative relationship with the MRN complex and its role in the PI3K-related protein kinase family helps initiate appropriate repair processes upon DNA damage detection. Additionally ATM regulates the activity of proteins such as Chk2 which further propagates signals to p53 influencing decisions between cell cycle arrest and apoptosis. These interactions link ATM closely to essential processes like DNA repair and cell survival highlighting its role in genomic maintenance.
Associated diseases and disorders	ATM mutations or dysregulation leads to Ataxia Telangiectasia an autosomal recessive disorder characterized by neurodegeneration immune deficiencies and cancer predisposition. ATM dysfunction also connects to cancer development particularly breast cancer where it transmits signals involving BRCA1 contributing to DNA repair through homologous recombination. Understanding ATM dynamics and related pathways has important implications for developing therapeutic strategies to manage or mitigate effects associated with its dysfunction.

Product promise

Tested

We have tested this species and application combination and it works. It is covered by our product promise.

Expected

We have not tested this specific species and application combination in-house, but expect it will work. It is covered by our product promise.

Predicted

This species and application combination has not been tested, but we predict it will work based on strong homology. However, this combination is not covered by our product promise.

Not recommended

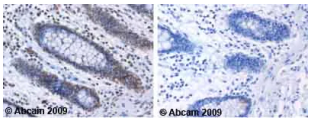
We do not recommend this combination. It is not covered by our product promise.

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In the unlikely event of one of our products not working as expected, you are covered by our product promise.

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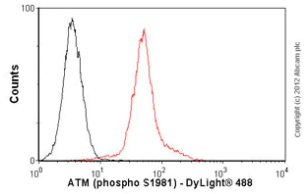
6 product images



Immunohistochemistry (Formalin/PFA-fixed paraffin-embedded sections) - Anti-ATM (phospho S1981) antibody [10H11.E12] (ab36810)

ATM (phospho S1981) Immunohistochemistry (Formalin/PFA-fixed paraffin-embedded sections) staining using mouse Anti-ATM (phospho S1981) antibody

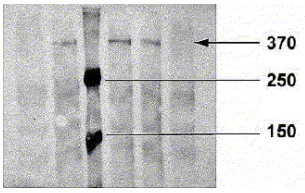
ab36810 staining human colon tissue. Staining is localized to the nucleus.
Left panel: with primary antibody at 4 µg/ml. **Right panel:** Isotype control.
Sections were stained using an automated system DAKO Autostainer Plus at room temperature. Sections were rehydrated and antigen retrieved with the DAKO 3-in-1 antigen retrieval buffer citrate pH 6.0 in a DAKO PT Link. Slides were peroxidase blocked in 3% H_2O_2 in methanol for 10 minutes. They were then blocked with Dako Protein block for 10 minutes (containing casein 0.25% in PBS) then incubated with primary antibody for 20 minutes and detected with Dako Envision Flex amplification kit for 30 minutes. Colorimetric detection was completed with diaminobenzidine for 5 minutes. Slides were counterstained with hematoxylin and coverslipped under DePeX.
Please note that for manual staining we recommend to optimize the primary antibody concentration and incubation time (overnight incubation), and amplification may be required.



Flow Cytometry (Intracellular) - Anti-ATM (phospho S1981) antibody [10H11.E12] (ab36810)

ATM (phospho S1981) Flow Cytometry (Intracellular) staining using mouse Anti-ATM (phospho S1981) antibody

Overlay histogram showing HeLa cells stained with ab36810 (red line). The cells were fixed with 80% methanol (5 min) and then permeabilized with 0.1% PBS-Tween for 20 min. The cells were then incubated in 1x PBS / 10% normal goat serum / 0.3M glycine to block non-specific protein-protein interactions followed by the antibody (ab36810, 1µg/1x10^6 cells) for 30 min at 22°C. The secondary antibody used was DyLight® 488 goat anti-mouse IgG (H+L) (ab96879) at 1/500 dilution for 30 min at 22°C. Isotype control antibody (black line) was mouse IgG1 [ICIGG1] (ab91353, 2µg/1x10^6 cells) used under the same conditions. Acquisition of >5,000 events was performed. This antibody gave a positive signal in HeLa cells fixed with 4% paraformaldehyde (10 min)/permeabilized with 0.1% PBS-Tween for 20 min used under the same conditions.



Western blot - Anti-ATM (phospho S1981) antibody [10H11.E12] (ab36810)

ATM (phospho S1981) Western blot staining using mouse Anti-ATM (phospho S1981) antibody

Lysate loading concentration: 40µg
All lanes:
Western blot - Anti-ATM (phospho S1981) antibody [10H11.E12] (ab36810) at 1/1000 dilution

Lane 1:
Control lane

Lane 2:
Irradiated Human fibroblasts (10 Gy gamma-irradiation)

Lane 3:
Molecular weight marker

Lane 4:
Peroxidated Human fibroblasts (300 µM hydrogen peroxide)

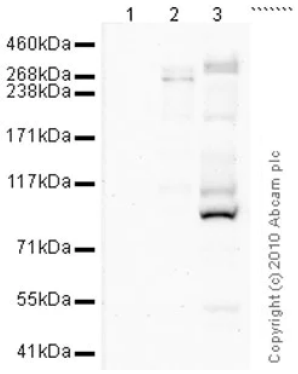
Lane 5:
Peroxidated Human fibroblasts (1 mM hydrogen peroxide)

Lane 6:
Peroxidated Human fibroblasts (10 mM hydrogen peroxide)

Developed using the ECL technique.

Predicted band size: 351 kDa

Observed band size: 370 kDa



Western blot - Anti-ATM (phospho S1981) antibody [10H11.E12] (ab36810)

All lanes:
Western blot - Anti-ATM (phospho S1981) antibody [10H11.E12] (ab36810) at 10 µg/mL

Lane 1:
HeLa (Human epithelial carcinoma cell line) Whole Cell Lysate at 20 µg

Lane 2:
Extract from patient with Ataxia-Telangiectasia whole cell lysate at 20 µg

Lane 3:
Irradiated HeLa Whole Cell Lysate at 20 µg

Secondary

All lanes:
Goat polyclonal to Mouse IgG - H&L - Pre-Adsorbed (HRP) at 1/3000 dilution

Developed using the ECL technique.

Performed under non-reducing conditions.

Predicted band size: 351 kDa

Observed band size: 100 kDa, 110 kDa, 145 kDa, 200 kDa, 370 kDa

Exposure time: 20min

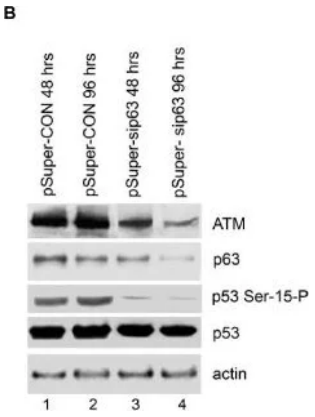


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Western blot - Anti-ATM (phospho S1981) antibody [10H11.E12] (ab36810)

ATM (phospho S1981) Western blot staining using mouse Anti-ATM (phospho S1981) antibody

Western Blotting using Anti-ATM (phospho S1981) antibody [10H11.E12], ab36810. Publication image from Gueven, N. et al., 2010, Mol Cancer, 20663147. Legend direct from paper.

p63 depletion attenuates ATM expression and ATM-dependent phosphorylation. (A) pSUPER-p63 RNAi attenuates ATM mRNA levels. HaCat cells were transfected with 1 µg pSUPER-CON or pSUPER-p63si vectors, and selected for geneticin resistance for 4 days. mRNA was extracted from surviving cells and real-time RT-PCR was used to quantitate changes in ATM mRNA expression. Data is represented as fold-change over empty vector control. (B) pSUPER-p63si inhibits ATM expression and p53 Serine-15 phosphorylation in HaCat cells. pSUPER-CON or pSUPER-p63si transfected HaCat cells were harvested after selection for 48 and 96 hours, and immunoblotted for the indicated proteins. (C) pSUPER-p63 RNAi inhibits HaCat cell proliferation. HaCaT cells were transfected with pSUPER-CON or pSUPER-p63si vectors, selected for geneticin resistance for 14 days, and surviving colonies were giemsa stained and counted. (D) siRNA-mediated p63 depletion attenuates ATM mRNA expression. HaCat cells untreated (NT) or treated with control siRNA or p63-targeted siRNA were harvested after 24 hrs or 48hrs and analysed by real-time RT-PCR for p63 and ATM mRNA expression. Data is normalised to β-actin and represented as fold-change over mRNA level in cells treated with control siRNA in particular time point. The data represent the average of three independent experiments + SD. The symbol asterisk denotes significant difference with p values < 0.05 determined by Mann-Whitney test, from the cells treated with control siRNA.

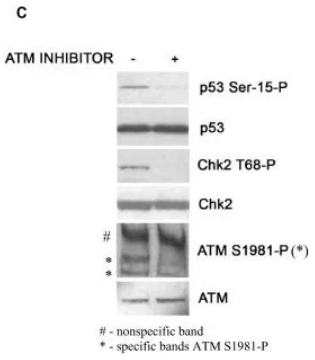


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Western blot - Anti-ATM (phospho S1981) antibody [10H11.E12] (ab36810)

ATM (phospho S1981) Western blot staining using mouse Anti-ATM (phospho S1981) antibody

Western Blotting using Anti-ATM (phospho S1981) antibody [10H11.E12], ab36810. Publication image from Gueven, N. et al., 2010, Mol Cancer, 20663147. Legend direct from paper.

The ATM pathway is active in HaCaT cells. (A) Damage-induced p53 Serine-15 is mediated by ATM. HaCat cells were treated with DMSO carrier, 10 µM KU-55933 (ATM inhibitor) or 1 µM NU7441 (DNA-PK inhibitor) for 2 hours, then with 0.5 µM doxorubicin, and cells were harvested after 2hrs, or cells were treated with 20Jm2 UV radiation and harvested after 6hrs. Lysates were blotted for phosphoSerine-15 p53, phosphoSerine-392 p53, total p53 protein and p63, as indicated. (B) ATM inhibition blocks constitutive p53 Serine-15 phosphorylation. Cells were treated with DMSO carrier, 10 µM KU-55933 (ATM inhibitor) or 1 µM NU7441 (DNA-PK inhibitor) for 24 hrs, and lysates were immunoblotted for phosphoSerine-15 p53 and total p53 protein. (C) ATM inhibition blocks constitutive ATM-dependent phosphorylation. Cells were treated with DMSO carrier or 10 µM KU-55933 for 24 hrs, and lysates were immunoblotted for: phosphoSerine-15 p53, phosphoSerine-392 p53, total p53 protein, phosphoThreonine-68 Chk2, total Chk2 protein, phosphoSerine-1891 ATM, and total ATM protein. (D) siRNA-mediated ATM depletion blocks p53 Serine-15 phosphorylation. HaCat cells were treated with control siRNA or siRNA to ATM for 24, 48 or 96hrs, and cell lysates were blotted for ATM, phosphoSerine-15 p53, and total p53 protein.

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