SANTA CRUZ BIOTECHNOLOGY, INC.

Atm (1B10): sc-135663

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

The phosphatidylinositol kinase (PIK) family members fall into two distinct subgroups. The first subgroup contains proteins such as the PI 3- and PI 4-kinases and the second group comprises the PIK-related kinases. The PIK-related kinases include Atm, DNA-PK_{CS} and FRAP. These proteins have in common a region of homology at their carboxy-termini that is not present in the PI 3- and PI 4-kinases. The Atm gene is mutated in the autosomal recessive disorder ataxia telangiectasia (AT), characterized by cerebellar degeneration (ataxia) and the appearance of dilated blood vessels (telangiec-tases) in the conjunctivae of the eyes. AT cells are hypersensitive to ionizing radiation, impaired in mediating the inhibition of DNA synthesis and display delays in p53 induction.

REFERENCES

1. Hartley, K.O., et al. 1995. DNA-dependent protein kinase catalytic subunit: a relative of phosphatidylinositol 3-kinase and the ataxia telangiectasia gene product. Cell 82: 849-856.
2. Nowak, R. 1995. Discovery of AT gene sparks biomedical research bonanza. Science 268: 1700-1701.

CHROMOSOMAL LOCATION

Genetic locus: ATM (human) mapping to 11q22.3; Atm (mouse) mapping to 9 A5.3.

SOURCE

Atm (1B10) is a mouse monoclonal antibody raised against a recombinant protein corresponding to amino acids 2841-3056 of Atm of human origin.

PRODUCT

Each vial contains 100 µg IgG_{2b} kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide, 0.1% gelatin, 1% glycerol and < 0.1% stabilizer protein.

APPLICATIONS

Atm (1B10) is recommended for detection of Atm of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)] and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for Atm siRNA (h): sc-29761, Atm siRNA (m): sc-29762, Atm shRNA Plasmid (h): sc-29761-SH, Atm shRNA Plasmid (m): sc-29762-SH, Atm shRNA (h) Lentiviral Particles: sc-29761-V and Atm shRNA (m) Lentiviral Particles: sc-29762-V.

Molecular Weight of Atm: 370 kDa.

Positive Controls: HeLa whole cell lysate: sc-2200, Jurkat whole cell lysate: sc-2204 or A-431 whole cell lysate: sc-2201.

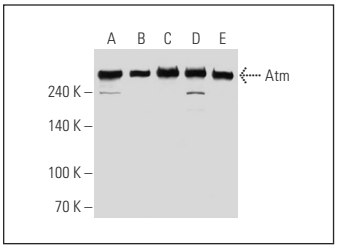
STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

DATA



Atm (1B10): sc-135663. Western blot analysis of Atm expression in HeLa (**A**), Jurkat (**B**), 293T (**C**), Raji (**D**) and A-431 (**E**) whole cell lysates.

SELECT PRODUCT CITATIONS

1. Zhou, Y. and Paull, T.T. 2013. DNA-dependent protein kinase regulates DNA end resection in concert with Mre11-Rad50-Nbs1 (MRN) and ataxia telangiectasia-mutated (Atm). J. Biol. Chem. 288: 37112-37125.
2. Chen, W.T., et al. 2015. Atm regulation of IL-8 links oxidative stress to cancer cell migration and invasion. Elife 4: e07270.
3. Leung, J.W., et al. 2017. ZMYM3 regulates BRCA1 localization at damaged chromatin to promote DNA repair. Genes Dev. 31: 260-274.
4. Hung, P.J., et al. 2018. MRI is a DNA damage response adaptor during classical non-homologous end joining. Mol. Cell 71: 332-342.e8.
5. Xu, S., et al. 2019. Inhibition of protein disulfide isomerase in glioblastoma causes marked downregulation of DNA repair and DNA damage response genes. Theranostics 9: 2282-2298.
6. Shao, X., et al. 2020. A distinct role for recombination repair factors in an early cellular response to transcription-replication conflicts. Nucleic Acids Res. 48: 5467-5484.
7. Chen, D., et al. 2020. Targeting the radiation-induced TR4 nuclear receptor-mediated QKI/circZEB1/miR-141-3p/ZEB1 signaling increases prostate cancer radiosensitivity. Cancer Lett. 495: 100-111.
8. Le, B.V., et al. 2020. TGFβR-SMAD3 signaling induces resistance to PARP inhibitors in the bone marrow microenvironment. Cell Rep. 33: 108221.
9. Ditano, J.P., et al. 2021. Sensitivity of cells to ATR and CHK1 inhibitors requires hyperactivation of CDK2 rather than endogenous replication stress or ATM dysfunction. Sci. Rep. 11: 7077.
10. Marruecos, L., et al. 2021. Single loss of a Trp53 allele triggers an increased oxidative, DNA damage and cytokine inflammatory responses through deregulation of IκBα expression. Cell Death Dis. 12: 359.

CONJUGATES

See **Atm (G-12): sc-377293** for Atm antibody conjugates, including AC, HRP, FITC, PE, and Alexa Fluor® 488, 546, 594, 647, 680 and 790.