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#92356**ATM (11G12) Mouse mAb**

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|                           |                         |                                   |                         |                                      |                               |                               |
|---------------------------|-------------------------|-----------------------------------|-------------------------|--------------------------------------|-------------------------------|-------------------------------|
| <b>Applications:</b><br>W | <b>Reactivity:</b><br>H | <b>Sensitivity:</b><br>Endogenous | <b>MW (kDa):</b><br>350 | <b>Source/Isotype:</b><br>Mouse IgG1 | <b>UniProt ID:</b><br>#Q13315 | <b>Entrez-Gene Id:</b><br>472 |
|---------------------------|-------------------------|-----------------------------------|-------------------------|--------------------------------------|-------------------------------|-------------------------------|

**Product Usage Information****Application**

Western Blotting

**Dilution**

1:1000

**Storage**

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at -20°C. Do not aliquot the antibody.

**Specificity/Sensitivity**

ATM (11G12) Mouse mAb recognizes endogenous levels of total ATM protein.

**Source / Purification**

Monoclonal antibody is produced by immunizing animals with recombinant protein corresponding to amino acids 992-1144 of human ATM protein.

**Background**

ATM (ataxia telangiectasia mutated kinase) is a serine/threonine protein kinase best known for its role in DNA repair signaling in response to DNA double-strand breaks (DSBs). When DSBs occur, the MRE11:RAD50:NBS1 (MRN) sensor complex recruits ATM to sites of DNA damage. ATM then signals to numerous effector proteins, leading to cellular responses including regulation of DNA repair, cell cycle progression, apoptosis, senescence, gene transcription. Along with ATR, DNA-PKcs, SMG1 and mTOR, ATM is a member of the PI3K-like protein kinase (PIKK) family. PIKK family members typically function in response to various types of cellular stress. Substrates of ATM are numerous, and include CHK2, AKT, p53, BRCA1 and DNA-PK (reviewed in 1,3). Inactive ATM exists as a homodimer. In response to DSBs, ATM undergoes autophosphorylation in trans at Ser1981, which leads to dissociation of the complex to become an active monomer (2). Functional DNA repair pathways are important in cellular homeostasis, and defects in these pathways cause genomic instability, which can lead to tumorigenesis (3). Inactivation of ATM results in ataxia telangiectasia (AT), a neurodegenerative disease characterized by predisposition to cancer (4).

**Background References**

- Shiloh, Y. and Ziv, Y. (2013) *Nat Rev Mol Cell Biol* 14, 197-210.
- Bakkenist, C.J. and Kastan, M.B. (2003) *Nature* 421, 499-506.
- Smith, J. et al. (2010) *Adv Cancer Res* 108, 73-112.
- McKinnon, P.J. (2012) *Annu Rev Pathol* 7, 303-21.

**Species Reactivity**

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

**Western Blot Buffer**

**IMPORTANT:** For western blots, incubate membrane with diluted primary antibody in 5% w/v nonfat dry milk, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

**Applications Key**

**W:** Western Blotting

**Cross-Reactivity Key**

**H:** Human

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