Revision 1

Store at -20C

#31068

PhosphoPlus® ATM (Ser1981) Antibody Duet

Cell Signaling TECHNOLOGY®

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For Research Use Only. Not for Use in Diagnostic Procedures.

UniProt ID:	Entrez-Gene Id:
#Q13315	472

Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
ATM (D2E2) Rabbit mAb	2873	100 µl	350 kDa	Rabbit IgG
Phospho-ATM (Ser1981) (D25E5) Rabbit mAb	13050	100 µl	350 kDa	Rabbit IgG

Please visit cellsignal.com for individual component applications, species cross-reactivity, dilutions, protocols, and additional product information.

Description

PhosphoPlus® Duets from Cell Signaling Technology (CST) provide a means to assess protein activation status. Each Duet contains an activation-state and total protein antibody to your target of interest. These antibodies have been selected from CST's product offering based upon superior performance in specified applications.

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.*

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

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

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