Revision 1

Store at -20C

#53217

PhosphoPlus® ATR (Thr1989) Antibody Duet

Cell Signaling TECHNOLOGY®

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Support: 877-678-TECH (8324)

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

UniProt ID:	Entrez-Gene Id:
#Q13535	545

Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
ATR (E1S3S) Rabbit mAb	13934	100 µl	300 kDa	Rabbit IgG
Phospho-ATR (Thr1989) (D5K8W) Rabbit mAb	30632	100 µl	300 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

Ataxia telangiectasia mutated kinase (ATM) and ataxia telangiectasia and Rad3-related kinase (ATR) are PI3 kinase-related kinase (PIKK) family members that phosphorylate multiple substrates on serine or threonine residues that are followed by a glutamine in response to DNA damage or replication blocks (1-3). Despite the essential role of ATR in cell cycle signaling and DNA repair processes, little is known about its activation. ATR was long thought to exist in a constitutively active state in cells, with DNA damage-induced signaling occurring via recruitment of ATR to single stranded DNA and sites of replication stress. Phosphorylation of ATR at serine 428 in response to UV-induced DNA damage has been suggested as a means of activating ATR (4,5). Recent work has shown autophosphorylation of ATR at threonine 1989. Like ATM Ser1981, phosphorylation of ATR Thr1989 occurs in response to DNA damage, indicating that phosphorylation at this site is important in ATR-mediated signaling (6,7).

Background References

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4. Vauzour, D. et al. (2007) *Arch Biochem Biophys* 468, 159-66.
5. Smith, J. et al. (2010) *Adv Cancer Res* 108, 73-112.
6. Nam, E.A. et al. (2011) *J Biol Chem* 286, 28707-14.
7. Liu, S. et al. (2011) *Mol Cell* 43, 192-202.

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