Revision 4

#4859 Store at -20C

# Phospho-Mre11 (Ser676) Antibody

Cell Signaling TECHNOLOGY®

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

| Applications: | Reactivity: | Sensitivity: | MW (kDa): | Source/Isotype: | UniProt ID: | Entrez-Gene Id: |
|---|---|---|---|---|---|---|
| W | H | Endogenous | 81 | Rabbit | #P49959 | 4361 |

## Product Usage Information

| Application | Dilution |
|---|---|
| Western Blotting | 1:1000 |

## Storage

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

## Specificity/Sensitivity

Phospho-Mre11 (Ser676) Antibody detects endogenous levels of Mre11 only when phosphorylated at Ser676.

## Species predicted to react based on 100% sequence homology

Monkey

## Source / Purification

Polyclonal antibodies are produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Ser676 of human Mre11. Antibodies are purified by protein A and peptide affinity chromatography.

## Background

Mre11, originally described in genetic screens from the yeast *Saccharomyces cerevisiae* in which mutants were defective in meiotic recombination (1), is a central part of a multisubunit nuclease composed of Mre11, Rad50 and Nbs1 (MRN) (2,3). The MRN complex plays a critical role in sensing, processing and repairing DNA double strand breaks. Defects lead to genomic instability, telomere shortening, aberrant meiosis and hypersensitivity to DNA damage (4). Hypomorphic mutations of Mre11 are found in ataxia-telangiectasia-like disease (ATLD), with phenotypes similar to mutations in ATM that cause ataxia-telangiectasia (A-T), including a predisposition to malignancy in humans (5). Cellular consequences of ATLD include chromosomal instability and defects in the intra-S phase and G2/M checkpoints in response to DNA damage. The MRN complex may directly activate the ATM checkpoint kinase at DNA breaks (6).

Phospho-Mre11 (Ser676) Antibody is directed to a site that was identified at Cell Signaling Technology (CST) using PhosphoScan®, CST's LC-MS/MS platform for modification site discovery. Phosphorylation at Ser676 was discovered using an ATM/ATR substrate antibody and was shown to be induced by UV treatment (7). Please visit PhosphoSitePlus®, CST's modification site knowledgebase, at www.phosphosite.org for more information.

## Background References

1. Ajimura, M. et al. (1993) *Genetics* 133, 51-66.
2. D'Amours, D. and Jackson, S.P. (2002) *Nat Rev Mol Cell Biol* 3, 317-27.
3. van den Bosch, M. et al. (2003) *EMBO Rep* 4, 844-9.
4. Theunissen, J.W. et al. (2003) *Mol Cell* 12, 1511-23.
5. Stewart, G.S. et al. (1999) *Cell* 99, 577-87.
6. Carson, C.T. et al. (2003) *EMBO J* 22, 6610-20.
7. Stokes, M.P. et al. (2007) *Proc. Natl. Acad. Sci. USA* 104, 19855-19860.

## 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 BSA, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

## Applications Key

**W:** Western Blotting

## Cross-Reactivity Key

**H:** Human

## Trademarks and Patents

## Limited Uses

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