

## Product Information

### Anti-NBS1 (Nibrin), C-terminal

Produced in Rabbit, Affinity Isolated Antibody

Product Number **N 3037**

#### Product Description

Anti-NBS1 (Nibrin), C-terminal is developed in rabbit using as immunogen a synthetic peptide corresponding to amino acids 735-753 of human NBS1(nibrin), conjugated to KLH via an N-terminal added cystein residue. The immunizing peptide differs from the rat and mouse corresponding sequences in two amino acids. The antibody is affinity purified on the immunizing peptide immobilized on agarose.

Anti NBS1 (Nibrin), C-terminal specifically recognizes human NBS1 (Nibrin). Applications include immunoblotting (95 kDa), immunofluorescence, and immunoprecipitation. Staining of the NBS1 band in immunoblotting is specifically inhibited by the immunizing peptide.

The Nijmegen breakage syndrome is caused by a defective response to DNA double-strand breaks (DSB).<sup>1,2</sup> NBS1 (Nibrin) was first isolated as a protein involved in DNA repair through analysis of mutations in patients with this syndrome.<sup>1,2</sup> NBS1 is a 754 amino acid protein containing two domains found in the cell cycle checkpoint proteins, forkhead-associated domain (FHA), and an adjacent breast cancer carboxy-terminal domain (BRCT).<sup>1,3</sup> NBS1 is one of the protein components for the double-strand breaks repair complex NBS1/MRE11/p50. This complex contains five proteins: p95 (NBS1), p200, p400, MRE11 and RAD50, and is important for double-strand break repair, meiotic recombination and telomere maintenance.<sup>2,4,5</sup>

p95/NBS1 (Nibrin) deficiency abrogates the formation of the MRE11/RAD50 ionizing radiation-induced foci, revealing a molecular link between DSB repair and cell cycle checkpoint functions.<sup>2</sup> The phenotypic similarities between ataxia-telangiectasia (AT) and Nijmegen breakage syndrome had suggested that ATM and NBS1 functions in a common signaling pathway. This was confirmed by the finding that in response to ionizing radiation, NBS1 is phosphorylated in Ser<sup>343</sup> in an ATM-dependent manner.<sup>6</sup>

#### Reagent

The antibody is supplied as a solution in 0.01 M phosphate buffered saline, pH 7.4, containing 15 mM sodium azide as a preservative.

Antibody Concentration: ~1.0 mg/mL

#### Precautions and Disclaimer

Due to the sodium azide content, a material safety data sheet (MSDS) for this product has been sent to the attention of the safety officer of your institution. Consult the MSDS for information regarding hazardous and safe handling practices.

#### Storage/Stability

For continuous use, store at 2-8 °C for up to one month. For extended storage, freeze in working aliquots. Repeated freezing and thawing is not recommended. Storage in frost-free freezers is also not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use. Working dilutions should be discarded if not used within 12 hours.

### Product Profile

By immunoblotting, a working antibody concentration of 0.5-1.0 µg/mL is recommended using MCF7 nuclear extracts.

By immunoblotting, a working antibody concentration of 0.25-0.5 µg/mL is recommended using extracts of 293-T cells transfected with recombinant NBS1.

By immunoprecipitation, 2-4 µg of the antibody immunoprecipitates NBS1 from HeLa cell lysates.

By indirect immunofluorescence, a working antibody concentration of 5-10 µg/mL is recommended using 293-T cells transfected with recombinant NBS1, fixed with paraformaldehyde/triton..

Recommendation: For immunoblotting, dilute the antibody in phosphate buffered saline containing 0.5% non-fat dry milk and 0.05 % Tween™ 20.

Note: In order to obtain the best results using various techniques and preparations, we recommend determining the optimal working dilutions by titration.

### References

1. Varon, R., et al., Cell, **93**, 467-476 (1998).
2. Carney, J.P., et al., Cell, **93**, 477-486 (1998).
3. Cerosaletti, K.M., and Concannon, P., J. Biol. Chem., **278**, 21944-21951(2003).
4. Goldberg, M., et al., Nature, **421**, 952-956 (2003).
5. di Fagagna, F., et al., Nature, **426**, 194-198 (2003).
6. Lim, D.-S., et al., Nature, **404**, 613-617 (2000).

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