

BRCA1 Antibody

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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, IP | H | Endogenous | 220 | Rabbit | #P38398 | 672 |

Product Usage Information**Application**

Western Blotting
Immunoprecipitation

Dilution

1:1000
1:100

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

BRCA1 Antibody detects endogenous levels of total BRCA1 protein. Five human isoforms are produced by alternative splicing and alternative initiation. The nuclear isoforms 1, 2, and 4 are detected, whereas the cytoplasmic isoforms 3 and 5 are not. The antibody does not recognize BRCA2.

Source / Purification

Polyclonal antibodies are produced by immunizing animals with a synthetic peptide corresponding to amino acids near the amino terminus of human BRCA1. Antibodies are purified by protein A and peptide affinity chromatography.

Background

The breast cancer susceptibility proteins BRCA1 and BRCA2 are frequently mutated in cases of hereditary breast and ovarian cancers and have roles in multiple processes related to DNA damage, repair, cell cycle progression, transcription, ubiquitination, and apoptosis (1-4). BRCA2 has been shown to be required for localization of Rad51 to sites of double-stranded breaks (DSBs) in DNA, and cells lacking BRCA1 and BRCA2 cannot repair DSBs through the Rad51-dependent process of homologous recombination (HR) (5). Numerous DNA damage-induced phosphorylation sites on BRCA1 have been identified, including Ser988, 1189, 1387, 1423, 1457, 1524, and 1542, and kinases activated in a cell cycle-dependent manner, including Aurora A and CDK2, can also phosphorylate BRCA1 at Ser308 and Ser1497, respectively (6-10). Cell cycle-dependent phosphorylation of BRCA2 at Ser3291 by CDKs has been proposed as a mechanism to switch off HR as cells progress beyond S-phase by blocking the carboxy-terminal Rad51 binding site (11).

Background References

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2. Gayther, S.A. et al. (1999) *Am J Hum Genet* 65, 1021-9.
3. Kerr, P. and Ashworth, A. (2001) *Curr Biol* 11, R668-76.
4. Scully, R. and Livingston, D.M. (2000) *Nature* 408, 429-32.
5. Tutt, A. and Ashworth, A. (2002) *Trends Mol Med* 8, 571-6.
6. Okada, S. and Ouchi, T. (2003) *J Biol Chem* 278, 2015-20.
7. Cortez, D. et al. (1999) *Science* 286, 1162-6.
8. Xu, B. et al. (2002) *Cancer Res* 62, 4588-91.
9. Ouchi, M. et al. (2004) *J Biol Chem* 279, 19643-8.
10. Ruffner, H. et al. (1999) *Mol Cell Biol* 19, 4843-54.
11. Esashi, F. et al. (2005) *Nature* 434, 598-604.

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 **IP:** Immunoprecipitation

Cross-Reactivity Key

H: Human

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