

## BRCA1

**Reactivity:**Human Mouse

**Tested applications:**WB IHC IF

**Recommended Dilution:**WB 1:500 - 1:2000 IHC 1:50 - 1:200 IF 1:20 - 1:50

**Calculated MW:**208kDa

**Observed MW:**Refer to Figures

**Immunogen:**

A synthetic peptide of human BRCA1

**Storage Buffer:**

Store at -20. Avoid freeze / thaw cycles. Buffer: PBS with 0.02% sodium azide, 50% glycerol, pH7.3.

**Concentration:**

def

**Synonym:**

BRCA1;BRCAI;BRCC1;BROVCA1;IRIS;PSCP;RNF53 ;

**Catalog #:**A0212

**Antibody Type:**

Polyclonal Antibody

**Species:**Rabbit

**Gene ID:**672

**Isotype:**IgG

**Swiss Prot:**P38398

**Purity:**Affinity purification

For research use only.

**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 serines 988, 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).

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