SANTA CRUZ BIOTECHNOLOGY, INC.

BARD1 (N-19): sc-7373

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Mutations within the BRCA1 gene, localized to chromosome 17q, are believed to account for approximately 45% of families with increased incidence of both early-onset breast cancer and ovarian cancer. The BRCA1 gene is expressed in numerous tissues, including breast and ovary, and encodes a predicted protein of 1,863 amino acids. This protein contains a RING domain near the N-terminus and appears to encode a tumor suppressor. BARD1 (BRCA1-associated RING domain protein 1) and BAP1 (BRCA1-associated protein 1) have both been shown to bind to the N-terminus of BRCA1 and are potential mediators of tumor suppression. BARD1 contains an N-terminal RING domain and three tandem ankyrin repeats. The C-terminus of BARD1 contains a region with sequence homology to BRCA1, termed the BRCT domain. BAP1 is an ubiquitin hydrolase and has been shown to enhance BRCA1-mediated cell growth suppression.

REFERENCES

1. Hall, J.M., et al. 1990. Linkage of early-onset familial breast cancer to chromosome 17q21. Science 250: 1684-1689.
2. Narod, S.A., et al. 1991. Familial breast-ovarian cancer locus on chromosome 17q12-q23. Lancet 338: 82-83.

CHROMOSOMAL LOCATION

Genetic locus: BARD1 (human) mapping to 2q35.

SOURCE

BARD1 (N-19) is an affinity purified goat polyclonal antibody raised against a peptide mapping at the N-terminus of BARD1 of human origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-7373 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

BARD1 (N-19) is recommended for detection of BARD1 of human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for BARD1 siRNA (h): sc-37311, BARD1 shRNA Plasmid (h): sc-37311-SH and BARD1 shRNA (h) Lentiviral Particles: sc-37311-V.

Molecular Weight of BARD1: 79 kDa.

Positive Controls: MDA-MB-231 nuclear extract, A-431 nuclear extract: sc-2122 or MDA-MB-468 nuclear extract.

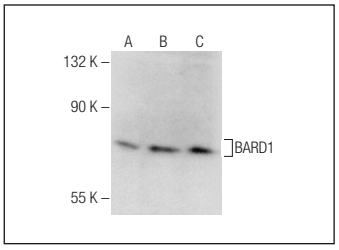
RESEARCH USE

For research use only, not for use in diagnostic procedures.

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

DATA



BARD1 (N-19): sc-7373. Western blot analysis of BARD1 expression in MDA-MB-231 (**A**), MDA-MB-468 (**B**) and A-431 (**C**) nuclear extracts.

SELECT PRODUCT CITATIONS

1. Irminger-Finger, I., et al. 2001. Identification of BARD1 as mediator between proapoptotic stress and p53-dependent apoptosis. Mol. Cell 8: 1255-1266.
2. Jefford, C.E. 2004. Nuclear-cytoplasmic translocation of BARD1 is linked to its apoptotic activity. Oncogene 23: 3509-3520.
3. Feki, A., et al. 2004. BARD1 expression during spermatogenesis is associated with apoptosis and hormonally regulated. Biol. Reprod. 71: 1614-1624.
4. Morris, J.R. and Solomon, E. 2004. BRCA1: BARD1 induces the formation of conjugated ubiquitin structures, dependent on K6 of ubiquitin, in cells during DNA replication and repair. Hum. Mol. Genet. 13: 807-817.
5. Wu, J.Y., et al. 2006. Aberrant expression of BARD1 in breast and ovarian cancers with poor prognosis. Int. J. Cancer 118: 1215-1226.
6. Li, L., et al. 2007. Identification of BARD1 splice-isoforms involved in human trophoblast invasion. Int. J. Biochem. Cell Biol. 39: 1659-1672.
7. Li, L., et al. 2007. Oncogenic BARD1 isoforms expressed in gynecological cancers. Cancer Res. 67: 11876-11885.
8. Marion-Letellier, R., et al. 2008. Comparison of cytokine modulation by natural peroxisome proliferator-activated receptor γ ligands with synthetic ligands in intestinal-like Caco-2 cells and human dendritic cells- potential for dietary modulation of peroxisome proliferator-activated receptor γ in intestinal inflammation. Am. J. Clin. Nutr. 87: 939-948.
9. Wei, Z., et al. 2012. Maternal exposure to di-(2-ethylhexyl)phthalate alters kidney development through the renin-angiotensin system in offspring. Toxicol. Lett. 212: 212-221.