SANTA CRUZ BIOTECHNOLOGY, INC.

PMS2 (C-20): sc-618

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

The finding that mutations in DNA mismatch repair genes are associated with hereditary nonpolyposis colorectal cancer (HNPCC) has resulted in considerable interest in the understanding of the mechanism of DNA mismatch repair. Initially, inherited mutations in the MSH2 and MLH1 homologs of the bacterial DNA mismatch repair genes MutS and MutL were demonstrated at high frequency in HNPCC and were shown to be associated with microsatellite instability. The demonstration that 10 to 45% of pancreatic, gastric, breast, ovarian and small cell lung cancers also display microsatellite instability has been interpreted to suggest that DNA mismatch repair is not restricted to HNPCC tumors but is a common feature in tumor initiation or progression. Two additional homologs of the prokaryotic MutL gene, designated PMS1 and PMS2, have been identified and shown to be mutated in the germline of HNPCC patients.

CHROMOSOMAL LOCATION

Genetic locus: PMS2 (human) mapping to 7p22.1; Pms2 (mouse) mapping to 5 G2.

SOURCE

PMS2 (C-20) is an affinity purified rabbit polyclonal antibody raised against a peptide mapping at the C-terminus of PMS2 of human origin.

PRODUCT

Each vial contains 100 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-618 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

PMS2 (C-20) is recommended for detection of PMS2 of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500), immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

PMS2 (C-20) is also recommended for detection of PMS2 in additional species, including equine, canine, bovine and porcine.

Suitable for use as control antibody for PMS2 siRNA (h): sc-36287, PMS2 siRNA (m): sc-36288, PMS2 shRNA Plasmid (h): sc-36287-SH, PMS2 shRNA Plasmid (m): sc-36288-SH, PMS2 shRNA (h) Lentiviral Particles: sc-36287-V and PMS2 shRNA (m) Lentiviral Particles: sc-36288-V.

Molecular Weight of PMS2: 110 kDa.

Positive Controls: HeLa nuclear extract: sc-2120, Jurkat nuclear extract: sc-2132 or A-431 nuclear extract: sc-2122.

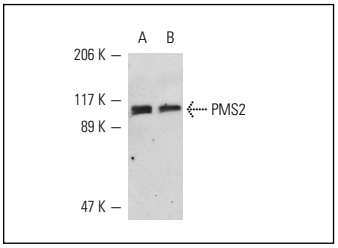
RESEARCH USE

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

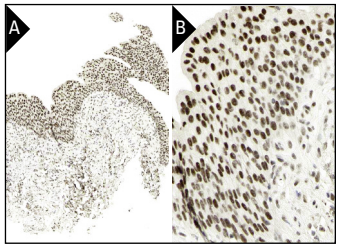
STORAGE

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

DATA



PMS2 (C-20): sc-618. Western blot analysis of PMS2 expression in HeLa (**A**) and A-431 (**B**) nuclear extracts.



PMS2 (C-20): sc-618. Immunoperoxidase staining of formalin-fixed, paraffin-embedded human urinary bladder tissue showing nuclear staining of surface epithelial cells at low (**A**) and high (**B**) magnification. Kindly provided by The Swedish Human Protein Atlas (HPA) program.

SELECT PRODUCT CITATIONS

1. Korabiowska, M., et al. 1997. Decreased expression of MLH1, MSH2, PMS1 and PMS2 in pigmented lesions indicates accumulation of failed DNA repair along with malignant transformation and tumour progression. Oncol. Rep. 4: 653-655.
2. Auclair, J., et al. 2007. Novel biallelic mutations in MSH6 and PMS2 genes: gene conversion as a likely cause of PMS2 gene inactivation. Hum. Mutat. 28: 1084-1090.
3. Cohn, D.E., et al. 2008. Correlation between patient weight and defects in DNA mismatch repair: is this the link between an increased risk of previous cancer in thinner women with endometrial cancer? Int. J. Gynecol. Cancer 18: 136-140.
4. Martin, L., et al. 2009. Recognition of O6MeG lesions by MGMT and mismatch repair proficiency may be a prerequisite for low-dose radiation hypersensitivity. Radiat. Res. 172: 405-413.
5. Resnick, K.E., et al. 2010. Mismatch repair status and outcomes after adjuvant therapy in patients with surgically staged endometrial cancer. Gynecol. Oncol. 117: 234-238.
6. Qi, Y., et al. 2010. Selenium compounds activate ATM-dependent DNA damage response via the mismatch repair protein hMLH1 in colorectal cancer cells. J. Biol. Chem. 285: 33010-33017.
7. Romeo, F., et al. 2011. BRCA1 is required for hMLH1 stabilization following doxorubicin-induced DNA damage. Int. J. Biochem. Cell Biol. 43: 1754-1763.