SANTA CRUZ BIOTECHNOLOGY, INC.

PMS1 (C-20): sc-615

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: PMS1 (human) mapping to 2q32.2.

SOURCE

PMS1 (C-20) is an affinity purified rabbit polyclonal antibody raised against a peptide mapping at the C-terminus of PMS1 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-615 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

PMS1 (C-20) is recommended for detection of PMS1 of 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).

PMS1 (C-20) is also recommended for detection of PMS1 in additional species, including equine and canine.

Suitable for use as control antibody for PMS1 siRNA (h): sc-37409, PMS1 shRNA Plasmid (h): sc-37409-SH and PMS1 shRNA (h) Lentiviral Particles: sc-37409-V.

Molecular Weight of PMS1: 115 kDa.

Positive Controls: HeLa whole cell lysate: sc-2200, T24 cell lysate: sc-2292 or MIA PaCa-2 cell lysate: sc-2285.

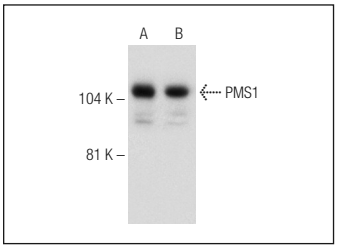
STORAGE

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

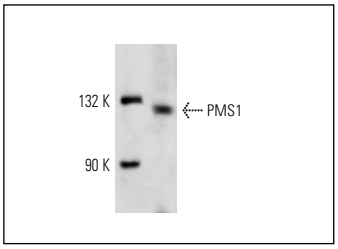
RESEARCH USE

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

DATA



PMS1 (C-20): sc-615. Western blot analysis of PMS1 expression in HeLa (**A**) and MIA PaCa-2 (**B**) whole cell lysates.



PMS1 (C-20): sc-615. Western blot analysis of PMS1 expression in T24 whole cell lysate

SELECT PRODUCT CITATIONS

1. Leung, W.K., et al. 2000. Identification of a second MutL DNA mismatch repair complex (hPMS1 and hMLH1) in human epithelial cells. J. Biol. Chem. 275: 15728-15732.
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3. Domingo, E., et al. 2005. BRAF-V600E is not involved in the colorectal tumorigenesis of HNPCC in patients with functional MLH1 and MSH2 genes. Oncogene 24: 3995-3998.
4. Hampel, H., et al. 2006. Screening for Lynch syndrome (hereditary nonpolyposis colorectal cancer) among endometrial cancer patients. Cancer Res. 66: 7810-7817.
5. Park, I.J., et al. 2007. Clinicopathological characteristics of colorectal cancer with family history: an evaluation of family history as a predictive factor for microsatellite instability. J. Korean Med. Sci. 22: S91-S97.
6. Siehler, S.Y., et al. 2009. Human MutL-complexes monitor homologous recombination independently of mismatch repair. DNA Repair 8: 242-252.
7. Kuo, T.C., et al. 2011. Forced expression of cyclin-dependent kinase 6 confers resistance of pro-B acute lymphocytic leukemia to Gleevec treatment. Mol. Cell. Biol. 31: 2566-2576.
8. Seriola, A., et al. 2011. Huntington's and myotonic dystrophy hESCs: down-regulated trinucleotide repeat instability and mismatch repair machinery expression upon differentiation. Hum. Mol. Genet. 20: 176-185.