Product Name: PMS2 Rabbit Polyclonal Antibody
Catalog #: APRab16313

Summary

Production Name	PMS2 Rabbit Polyclonal Antibody
Description	Rabbit Polyclonal Antibody
Host	Rabbit
Application	WB,IHC,ELISA
Reactivity	Human,Rat,Mouse

Performance

Conjugation	Unconjugated
Modification	Unmodified
Isotype	IgG
Clonality	Polyclonal
Form	Liquid
Storage	Store at 4°C short term. Aliquot and store at -20°C long term. Avoid freeze/thaw cycles.
Buffer	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% New type preservative N.
Purification	Affinity purification

Immunogen

Gene Name	PMS2
Alternative Names	PMS2; PMSL2; Mismatch repair endonuclease PMS2; DNA mismatch repair protein PMS2; PMS1 protein homolog 2
Gene ID	5395.0
SwissProt ID	P54278.The antiserum was produced against synthesized peptide derived from human PMS2. AA range:461-510

Application

Dilution Ratio	WB 1:500 - 1:2000. IHC 1:100 - 1:300. . ELISA: 1:10000. Not yet tested in other applications.
Molecular Weight	85kD

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Background

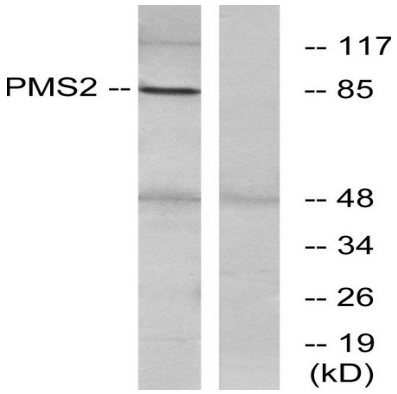
The protein encoded by this gene is a key component of the mismatch repair system that functions to correct DNA mismatches and small insertions and deletions that can occur during DNA replication and homologous recombination. This protein forms heterodimers with the gene product of the mutL homolog 1 (MLH1) gene to form the MutL-alpha heterodimer. The MutL-alpha heterodimer possesses an endonucleolytic activity that is activated following recognition of mismatches and insertion/deletion loops by the MutS-alpha and MutS-beta heterodimers, and is necessary for removal of the mismatched DNA. There is a DQHA(X)2E(X)4E motif found at the C-terminus of the protein encoded by this gene that forms part of the active site of the nuclease. Mutations in this gene have been associated with hereditary nonpolyposis colorectal cancer (HNPCC; also known as Lynch syndrome) and Turcot syndromedisease:Defects in PMS2 are a cause of mismatch repair cancer syndrome (MMRCS) [MIM:276300]; also known as Turcot syndrome and brain tumor-polyposis syndrome 1 (BTPS1). MMRCS is an autosomal dominant disorder characterized by malignant tumors of the brain associated with multiple colorectal adenomas. Skin features include sebaceous cysts, hyperpigmented and cafe au lait spots.,disease:Defects in PMS2 are the cause of hereditary non-polyposis colorectal cancer type 4 (HNPCC4) [MIM:600259]. Mutations in more than one gene locus can be involved alone or in combination in the production of the HNPCC phenotype (also called Lynch syndrome). Most families with clinically recognized HNPCC have mutations in either MLH1 or MSH2 genes. HNPCC is an autosomal, dominantly inherited disease associated with marked increase in cancer susceptibility. It is characterized by a familial predisposition to early onset colorectal carcinoma (CRC) and extra-colonic cancers of the gastrointestinal, urological and female reproductive tracts. HNPCC is reported to be the most common form of inherited colorectal cancer in the Western world, and accounts for 15% of all colon cancers. Cancers in HNPCC originate within benign neoplastic polyps termed adenomas. Clinically, HNPCC is often divided into two subgroups. Type I: hereditary predisposition to colorectal cancer, a young age of onset, and carcinoma observed in the proximal colon. Type II: patients have an increased risk for cancers in certain tissues such as the uterus, ovary, breast, stomach, small intestine, skin, and larynx in addition to the colon. Diagnosis of classical HNPCC is based on the Amsterdam criteria: 3 or more relatives affected by colorectal cancer, one a first degree relative of the other two; 2 or more generation affected; 1 or more colorectal cancers presenting before 50 years of age; exclusion of hereditary polyposis syndromes. The term "suspected HNPCC" or "incomplete HNPCC" can be used to describe families who do not or only partially fulfill the Amsterdam criteria, but in whom a genetic basis for colon cancer is strongly suspected.,function:Component of the post-replicative DNA mismatch repair system (MMR). Heterodimerizes with MLH1 to form MutL alpha. DNA repair is initiated by MutS alpha (MSH2-MSH6) or MutS beta (MSH2-MSH6) binding to a dsDNA mismatch, then MutL alpha is recruited to the heteroduplex. Assembly of the MutL-MutS-heteroduplex ternary complex in presence of RFC and PCNA is sufficient to activate endonuclease activity of PMS2. It introduces single-strand breaks near the mismatch and thus generates new entry points for the exonuclease EXO1 to degrade the strand containing the mismatch. DNA methylation would prevent cleavage and therefore assure that only the newly mutated DNA strand is going to be corrected. MulL alpha (MLH1-PMS2) interacts physically with the clamp loader subunits of DNA polymerase III, suggesting that it may play a role to recruit the DNA polymerase III to the site of the MMR. Also implicated in DNA damage signaling, a process which induces cell cycle arrest

and can lead to apoptosis in case of major DNA damages.,similarity:Belongs to the DNA mismatch repair mutL/hexB family.,subunit:Heterodimer of PMS2 and MLH1 (MutL alpha). Forms a ternary complex with MutS alpha (MSH2-MSH6) or MutS beta (MSH2-MSH3). Part of the BRCA1-associated genome surveillance complex (BASC), which contains BRCA1, MSH2, MSH6, MLH1, ATM, BLM, PMS2 and the RAD50-MRE11-NBS1 protein complex. This association could be a dynamic process changing throughout the cell cycle and within subnuclear domains.,

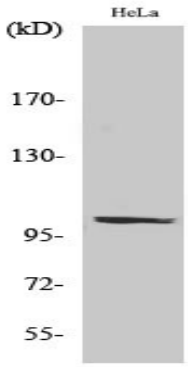
Research Area

Mismatch repair;

Image Data



Western blot analysis of lysates from HeLa cells, using PMS2 Antibody. The lane on the right is blocked with the synthesized peptide.



Western Blot analysis of various cells using PMS2 Polyclonal Antibody cells nucleus extracted by Minute TM Cytoplasmic and Nuclear Fractionation kit （SC-003,Inventbiotech,MN,USA）.

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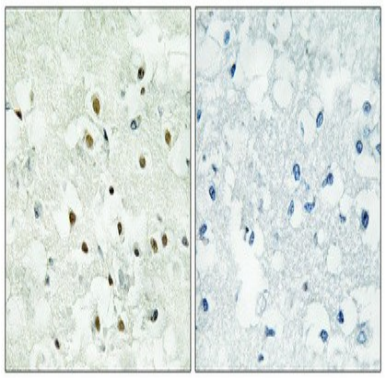

Immunohistochemical analysis of paraffin-embedded Human brain. Antibody was diluted at 1:100（4°,overnight）. High-pressure and temperature Tris-EDTA,pH8.0 was used for antigen retrieval. Negetive contrl （right） obtaned from antibody was pre-absorbed by immunogen peptide.

Note

For research use only.