



## Mouse Monoclonal Antibody **PMS2** conjugated to Sepharose Beads

CatalogNo: **ANT8020-S**

Size 200ul

Storage Store at 4 °C for frequent use

### Description

This Antagene antibody is immobilized via covalent binding of primary amino groups to N-hydroxysuccinimide (NHS)-activated sepharose beads. It is useful for immunoprecipitation assays.

### **PMS2 (ANT0045R) Rabbit mAb**

Formulation: 50% slurry in PBS pH 7.2 with 0.01mg NaN<sub>3</sub> preservative.

#### Host Species

- Rabbit
- Human,
- WB,IHC,IF,IP,ELISA

#### Reactivity

#### Applications

#### MW

- 96kD (Calculated)
- IgG,Kappa
- 110kD (Observed)

#### Isotype

## **Recommended Dilution Ratios**

### **IP**

### **Basic Information**

**Clonality** Monoclonal

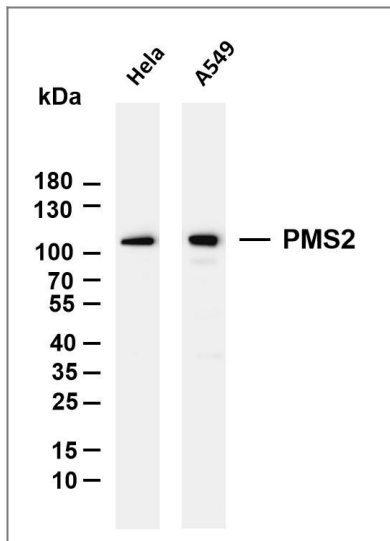
**Clone Number** ANT0045R

Endogenous

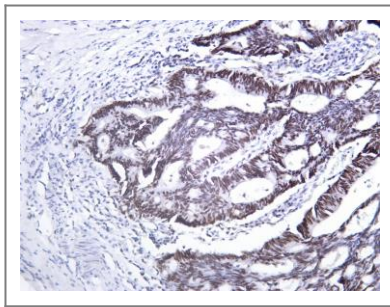
Gene name	PMS2 PMSL2		
Protein Name	Postmeiotic Segregation Increased 2(PMS2)		
Cellular Localization	Organism	Gene ID	UniProt ID
	Human	<a href="#">5395</a> ;	<a href="#">P54278</a> ;
Tissue specificity	Amygdala,Brain,Endometrial tumor,Epithelium,Human endometri		
Function	<p>Disease:Defects in PMS2 are a cause of mismatch repair cancer syndrome (MMRCS) [MIM:276300]; also known as Turcot syndrome and brain tumor-polypsis 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-polypsis 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</p>		

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., Various whole cell lysates were separated by 4-20% SDS-PAGE, and the membrane was blotted with anti-PMS2(ANT0045R) antibody. The HRPconjugated Goat anti-Rabbit IgG(H + L) antibody was used to detect the antibody. Lane 1: HeLa Lane 2: A549 Predicted band size: 96kDa Observed band size: 110kDa

## Validation Data



Human rectal carcinoma tissue was stained with Anti-PMS2 (ANT0045R) rabbit Antibody



For Research use only, not for diagnostics and clinical use  
Contact Antagene Inc Tel 1-866-964-2589 Email: [info@antageneinc.com](mailto:info@antageneinc.com)