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PMS2 (PT0045R) PT® Rabbit mAb

CatalogNo: YM8020 Recombinant R

Key Features

Host SpeciesRabbit

MW • 96kD (Calculated) 110kD (Observed) Human,
 Isotype

Reactivity

IgG,Kappa

Applications
• WB,IHC,IF,IP,ELISA

Recommended Dilution Ratios

IHC 1:200-1000 WB 1:500-5000 IF 1:200-1000 ELISA 1:5000-20000 IP 1:50-200

Storage

Storage*	-15°C to -25°C/1 year(Do not lower than -25°C)
Formulation	PBS, 50% glycerol, 0.05% Proclin 300, 0.05%BSA

Basic Information

Clonality	Monoclonal
Clone Number	PT0045R

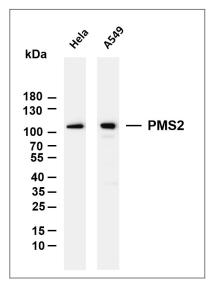
Immunogen Information

Specificity Endogenous

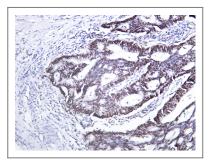
Target Information

Gene name	PMS2 PMSL2				
Protein Name	Postmeiotic Segregation Increased 2(PMS2)				
	0	rganism	Gene ID	UniProt ID	
		Human	<u>5395;</u>	<u>P54278;</u>	
Cellular Localization	Nuclear				
Tissue specificity	Amygdala,Br	rain,Endometrial tumor,E	pithelium,Human endometri		
Function	[MIM:276300 (BTPS1). MM the brain ass cysts, hyperp hereditary no more than of HNPCC phen HNPCC phen HNPCC have inherited dis characterize extra-colonic HNPCC is rep Western wor within benig two subgrou and carcinor cancers in ce and larynx ir criteria: 3 or other two; 2 years of age "incomplete Amsterdam suspected.,F Heterodimer (MSH2-MSH6 recruited to presence of introduces si the exonucle would preve going to be of subunits of E polymerase which induce damages.,sin family.,subun MutS alpha (genome surv BLM, PMS2 a	D]; also known as Turcot RCS is an autosomal dou sociated with multiple co- pigmented and cafe au l on-polyposis colorectal on ne gene locus can be involted otype (also called Lynch mutations in either MLH ease associated with mad d by a familial predispose cancers of the gastroin ported to be the most co- ld, and accounts for 15% n neoplastic polyps term ps. Type I: hereditary pri- na observed in the proxi- ertain tissues such as the naddition to the colon. If more relatives affected or more generation affe- ; exclusion of hereditary HNPCC" can be used to criteria, but in whom a g- unction:Component of the izes with MLH1 to form I b) or MutS beta (MSH2-M the heteroduplex. Assen RFC and PCNA is sufficient ingle-strand breaks near ease EXO1 to degrade the nt cleavage and therefore corrected. MulL alpha (M DNA polymerase III, sugg III to the site of the MMR es cell cycle arrest and co- milarity:Belongs to the D nit:Heterodimer of PMS2 MSH2-MSH6) or MutS before (BASC) and the RAD50-MRE11-N	of mismatch repair cancer sy syndrome and brain tumor-po- minant disorder characterized lorectal adenomas. Skin featu- ait spots.,Disease:Defects in F cancer type 4 (HNPCC4) [MIM: volved alone or in combination syndrome). Most families wit 11 or MSH2 genes. HNPCC is a rked increase in cancer susce ition to early onset colorectal testinal, urological and female mmon form of inherited color 6 of all colon cancers. Cancers ed adenomas. Clinically, HNP edisposition to colorectal cancer and colon. Type II: patients has a uterus, ovary, breast, stoma by colorectal cancer, one a fil cted; 1 or more colorectal can polyposis syndromes. The test describe families who do not enetic basis for colon cancer ne post-replicative DNA mism MutL alpha. DNA repair is initi. SH6) binding to a dsDNA mism futL alpha. DNA repair is initi. SH6) binding to a dsDNA mism futL alpha. DNA repair is initi. SH6) binding to a dsDNA mism futL alpha. DNA repair is initi. SH6) binding to a dsDNA mism futL alpha. DNA repair is initi. SH6) binding to a dsDNA mism futL alpha. DNA repair is initi. SH6) binding to a dsDNA mism futL alpha. DNA repair is initi. SH6) binding to a dsDNA mism futL alpha. DNA repair is initi. SH6) binding to a dsDNA mism futL alpha. DNA repair is initi. SH6) binding to a dsDNA mism futL alpha. DNA repair is initi. SH6) binding to a dsDNA mism futL alpha. DNA repair is initi. SH6) binding to a dsDNA mism futL alpha. DNA repair is initi. SH6) binding to a dsDNA mism futL alpha. DNA repair is initi. SH6) binding to a dsDNA mism futL alpha. DNA repair is initi. SH6) binding to a dsDNA mism futL alpha. DNA repair is initi. SH6) binding to a dsDNA mism futL alpha. DNA repair is initi. SH6) binding to a dsDNA mism futL alpha. DNA repair is initi. SH6) binding to a dsDNA mism futL alpha. So mism	olyposis syndrome 1 I by malignant tumors of ures include sebaceous PMS2 are the cause of 600259]. Mutations in in the production of the ch clinically recognized an autosomal, dominantly eptibility. It is carcinoma (CRC) and e reproductive tracts. rectal cancer in the s in HNPCC originate CC is often divided into cer, a young age of onset, ave an increased risk for ach, small intestine, skin, s based on the Amsterdam rst degree relative of the neers presenting before 50 rm "suspected HNPCC" or or only partially fulfill the is strongly atch repair system (MMR). ated by MutS alpha match, then MutL alpha is duplex ternary complex in activity of PMS2. It rates new entry points for iatch. DNA methylation mutated DNA strand is ly with the clamp loader to recruit the DNA age signaling, a process of major DNA xB ns a ternary complex with BRCA1-associated 12, MSH6, MLH1, ATM, sociation could be a	

Validation Data



Various whole cell lysates were separated by 4-20% SDS-PAGE, and the membrane was blotted with anti-PMS2 antibody. The HRP-conjugated 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



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

Contact information

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Please scan the QR code to access additional product information: PMS2 (PT0045R) PT® Rabbit mAb

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