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Product datasheet

Anti-PMS2 antibody [349.29.5.2] ab34615

1 References 1 Image

Overview

Product name	Anti-PMS2 antibody [349.29.5.2]
Description	Mouse monoclonal [349.29.5.2] to PMS2
Host species	Mouse
Tested applications	**Suitable for:** WB, ELISA, IP
Species reactivity	**Reacts with:** Hamster, Human
Immunogen	Recombinant fragment (Human) -PMS2 corresponding to the first 133 amino acid residues of the protein (N-terminal).
Epitope	The epitope was putatively mapped to amino acids 58-81 of human PMS2.
Positive control	H157 cell lysate

Properties

Form	Liquid
Storage instructions	Shipped at 4°C. Store at +4°C short term (1-2 weeks). Upon delivery aliquot. Store at -20°C or -80°C. Avoid freeze / thaw cycle.
Storage buffer	pH: 7.20 Preservative: 0.01% Sodium azide Constituents: 0.42% Potassium phosphate, 0.87% Sodium chloride
Purity	Ascites
Clonality	Monoclonal
Clone number	349.29.5.2
Myeloma	P3x63-Ag8.653
Isotype	IgG1
Light chain type	kappa

Applications

Our Abpromise guarantee covers the use of **ab34615** in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Application	Abreviews	Notes
WB		1/500 - 1/2000. Predicted molecular weight: 96 kDa.
ELISA		1/5000 - 1/20000.
IP		Use at an assay dependent dilution.

Target

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. MutL 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.

Involvement in 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.
Defects in PMS2 are a cause of mismatch repair cancer syndrome (MMRCS) [MIM:276300]; also known as Turcot syndrome or 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.

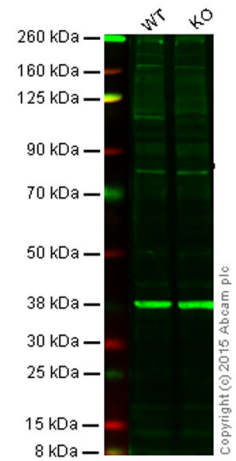
Sequence similarities

Belongs to the DNA mismatch repair mutL/hexB family.

Cellular localization

Nucleus.

Images



Western blot - Anti-PMS2 antibody [349.29.5.2] (ab34615)

Lane 1: Wild-type HAP1 cell lysate (20 µg)
Lane 2: PMS2 knockout HAP1 cell lysate (20 µg)
Lanes 1 - 2: Merged (red and green) signal. Red - loading control, ab181602, observed at 37kDa.

ab34615 was shown not to specifically react with PMS2, when PMS2 knockout samples were used. Wild-type and PMS2 knockout samples were subjected to SDS-PAGE. ab34615 at a dilution of 1/250 and ab181602 (loading control to GAPDH) diluted 1/10000 were incubated overnight at 4°C. Blots were developed with goat anti-rabbit IgG (H + L) and goat anti-mouse IgG (H + L) secondary antibodies at 1/10000 dilution for 1 hour at room temperature before imaging.

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