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MSH2 Mouse mAb

Catalog # AP96610

Product Information

Application	WB, IHC-P, IHC-F, IF
Reactivity	Human
Host	Mouse
Clonality	Monoclonal
Physical State	Liquid
Immunogen	Purified His-tagged MSH2 protein was used to produced this monoclonal antibody.
Isotype	IgG1
Purity	affinity purified by Protein A
Buffer	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
SIMILARITY	Belongs to the DNA mismatch repair MutS family.
SUBUNIT	Heterodimer consisting of MSH2-MSH6 (MutS alpha) or MSH2-MSH3 (MutS beta). Both heterodimer form a ternary complex with MutL alpha (MLH1-PMS1). Interacts with EXO1. 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. Interacts with ATR. Interacts with SLX4/BTBD12; this interaction is direct and links MutS beta to SLX4, a subunit of different structure-specific endonucleases. Interacts with SMARCAD1.
Post-translational modifications	Phosphorylated by PRKCZ, which may prevent MutS alpha degradation by the ubiquitin-proteasome pathway. Phosphorylated upon DNA damage, probably by ATM or ATR.
DISEASE	Hereditary non-polyposis colorectal cancer 1 (HNPCC1) [MIM:120435]: An autosomal dominant disease associated with marked increase in cancer susceptibility. It is characterized by a familial predisposition to early-onset colorectal carcinoma (CRC) and extra-colonic tumors 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. Clinically, HNPCC is often divided into two subgroups. Type I is characterized by hereditary predisposition to colorectal cancer, a young age of onset, and carcinoma observed in the proximal colon. Type II is characterized by 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. Note=The disease is caused by mutations affecting the gene represented in this entry.Muir-Torre syndrome (MRTES) [MIM:158320]: Rare autosomal dominant disorder characterized by sebaceous neoplasms and visceral malignancy. Note=The disease is caused by mutations affecting the gene represented in this

	entry.Endometrial cancer (ENDMC) [MIM:608089]: A malignancy of endometrium, the mucous lining of the uterus. Most endometrial cancers are adenocarcinomas, cancers that begin in cells that make and release mucus and other fluids. Note=Disease susceptibility is associated with variations affecting the gene represented in this entry.
Important Note	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
Background Descriptions	Component of the post-replicative DNA mismatch repair system (MMR). Forms two different heterodimers: MutS alpha (MSH2-MSH6 heterodimer) and MutS beta (MSH2-MSH3 heterodimer) which binds to DNA mismatches thereby initiating DNA repair. When bound, heterodimers bend the DNA helix and shields approximately 20 base pairs. MutS alpha recognizes single base mismatches and dinucleotide insertion-deletion loops (IDL) in the DNA. MutS beta recognizes larger insertion-deletion loops up to 13 nucleotides long. After mismatch binding, MutS alpha or beta forms a ternary complex with the MutL alpha heterodimer, which is thought to be responsible for directing the downstream MMR events, including strand discrimination, excision, and resynthesis. ATP binding and hydrolysis play a pivotal role in mismatch repair functions. The ATPase activity associated with MutS alpha regulates binding similar to a molecular switch: mismatched DNA provokes ADP-->ATP exchange, resulting in a discernible conformational transition that converts MutS alpha into a sliding clamp capable of hydrolysis-independent diffusion along the DNA backbone. This transition is crucial for mismatch repair. MutS alpha may also play a role in DNA homologous recombination repair. In melanocytes may modulate both UV-B-induced cell cycle regulation and apoptosis.

Additional Information

Other Names	COCA1; FCC1; HNPCC; HNPCC1; LCFS2; LYNCH1; MMRCS2; MSH-2; hMSH2; MSH2_HUMAN; MSH2; MutS protein homolog 2; MSH2_MOUSE; MSH2_RAT; mutS homolog 2; mutS (E. coli) homolog 2 (colon cancer, nonpolyposis type 1); mutS homolog 2, colon cancer, nonpolyposis type 1 (E. coli); DNA mismatch repair protein Msh2
Dilution	WB=1:500-2000,IHC-P=1:100-500,IHC-F=1:100-500,IF=1:100-500
Storage	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.
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along the DNA backbone. This transition is crucial for mismatch repair. MutS alpha may also play a role in DNA homologous recombination repair. In melanocytes may modulate both UV-B-induced cell cycle regulation and apoptosis.

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