错配修复蛋白 2 抗体

产品货号： mIR23631

英文名称： MSH2

中文名称： 错配修复蛋白 2 抗体

别　　名： MSH-2; BAT26; DNA mismatch repair protein Msh2; FCC1; COCA1; HNPCC; LCFS2; HNPCC1; BAT26; COCA 1; COCA1; DNA mismatch repair protein Msh2; FCC 1; FCC1; hMSH2; HNPCC 1; HNPCC; HNPCC1; LCFS2; MSH 2; Msh2; MSH2_HUMAN; MutS homolog 2; MutS homolog 2 colon cancer nonpolyposis type 1; MutS protein homolog 2.

研究领域： 肿瘤　细胞生物　表观遗传学

抗体来源： Rabbit

克隆类型： Polyclonal

交叉反应　： Human, Mouse, Rat, Pig, Horse, Rabbit,

产品应用： WB=1:500-2000 IHC-P=1:400-800 IHC-F=1:400-800 ICC=1:100-500 IF=1:100-500 （石蜡切片需做抗原修复）

not yet tested in other applications.

optimal dilutions/concentrations should be determined by the end user.

分 子 量： 104kDa

细胞定位： 细胞核

性　　状： Lyophilized or Liquid

浓　　度： 1mg/ml

免 疫 原： KLH conjugated synthetic peptide derived from human MSH2 :501-600/935

亚　　型： IgG

纯化方法： affinity purified by Protein A

储 存 液： 0.01M TBS(pH7.4) with 1% BSA, 0.03% Proclin300 and 50% Glycerol.

保存条件： Store at -20 ° C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year when kept at -20° C. 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.

PubMed : PubMed

产品介绍 ： 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.

Function:

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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.

Subcellular Location:

Nucleus.

Tissue Specificity:

Ubiquitously expressed.

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.

Similarity:

Belongs to the DNA mismatch repair MutS family.

SWISS:

P43246

Gene ID:

4436

Important Note:

This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.

MSH2 是核苷酸错配修复酶之一，其突变和多种肿瘤的发生有关，主要用于：先天性结肠癌、散发性结肠癌及一些消化系统肿瘤方面的研究。DNA 错配修复系统是指由特异修复 DNA 碱基错配的酶组成，保证 DNA 遗传物质的高保真性。它由 MLH1、MSH2、PMS1、PMS2、MSH6 和 MSH3 基因组成。如果上述基因发生突变或失活，会使细胞错配修复功能缺陷，产生遗传不稳定性，导致肿瘤易感。MSH2 基因突变发生在散发性结肠癌中。

hMSH-2 和 hMLH1 都与肿瘤的发生有关。

产品图片