

## Rabbit Anti-MSH2 antibody

SL0758R

<b>Product Name</b>	MSH2
<b>Chinese Name</b>	错配修复蛋白 2 抗体
<b>Alias</b>	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_HUMAN; MutS homolog 2; MutS homolog 2 colon cancer nonpolyposis type 1; MutS protein homolog 2.
<b>Research Area</b>	Tumour Cell biology Epigenetics
<b>Immunogen Species</b>	Rabbit
<b>Clonality</b>	Polyclonal
<b>React Species</b>	Human, Mouse, (predicted: Cow, ) WB=1:500-2000 (Paraffin sections need antigen repair)
<b>Applications</b>	not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
<b>Theoretical molecular weight</b>	105kDa
<b>Cellular localization</b>	The nucleus
<b>Form</b>	Liquid
<b>Concentration</b>	1mg/ml
<b>immunogen</b>	KLH conjugated synthetic peptide derived from human MSH-2: 101-200/935
<b>Lsotype</b>	IgG
<b>Purification</b>	affinity purified by Protein A
<b>Buffer Solution</b>	1M TBS(pH7.4) with 1% BSA, 3% Proclin300 and 50% Glycerol.
<b>Storage</b>	Shipped at 4°C. Store at -20 °C for one year. Avoid repeated freeze/thaw cycles.
<b>Attention</b>	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
<b>PubMed</b>	<a href="#">PubMed</a>
<b>Product Detail</b>	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:**

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.

**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

**Database links:**

[Entrez Gene: 4436](#) Human

[Entrez Gene: 17685](#) Mouse

[Entrez Gene: 81709](#) Rat

[Omim: 120435](#) Human

[SwissProt: P43246](#) Human

[SwissProt: P43247](#) Mouse

[SwissProt: P54275](#) Rat

[Unigene: 597656](#) Human

[Unigene: 4619](#) Mouse

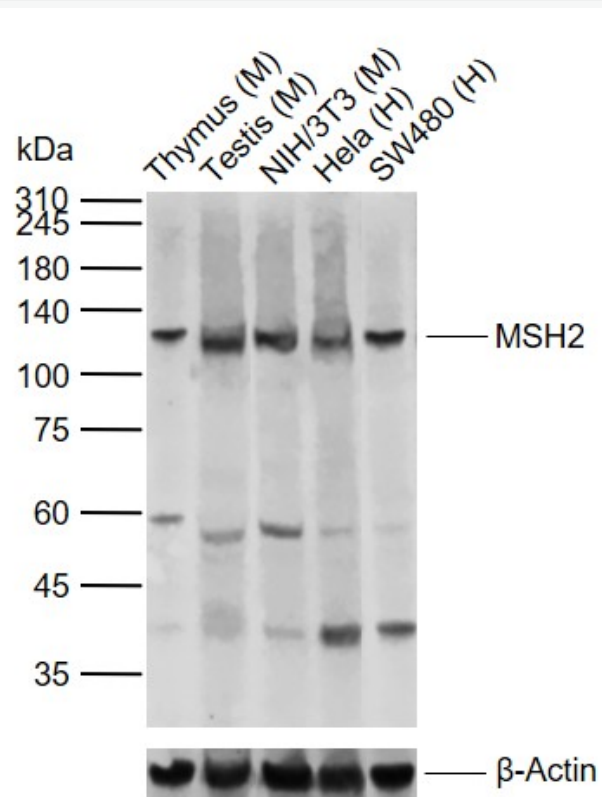
[Unigene: 471165](#) Mouse

[Unigene: 3174](#) Rat

MSH2 是核苷酸错配修复酶之一，其突变和多种 Tumour 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 Tumour 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 Tumour 易感。MSH2 基因突变发生在散发性结肠癌中。

hMSH-2 和 hMLH1 都与 Tumour 的发生有关。

**Product Picture**



Sample:

Lane 1: Mouse Thymus tissue lysates

Lane 2: Mouse Testis tissue lysates

Lane 3: Mouse NIH/3T3 cell lysates

Lane 4: Human Hela cell lysates

Lane 5: Human SW480 cell lysates

Primary: Anti-MSH2 (SL0758R) at 1/1000 dilution

Secondary: IRDye800CW Goat Anti-Rabbit IgG at 1/20000 dilution

Predicted band size: 105 kDa



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Observed band size: 110 kDa