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Immunotag™ MSH2 Polyclonal Antibody

Antibody Specification

Catalog No.	ITT2896
Product Description	Immunotag™ MSH2 Polyclonal Antibody
Size	50 µg, 100 µg
Conjugation	HRP, Biotin, FITC, Alexa Fluor® 350, Alexa Fluor® 405, Alexa Fluor® 488, Alexa Fluor® 555, Alexa Fluor® 594, Alexa Fluor® 647
IMPORTANT NOTE	This product is custom manufactured with a lead time of 3-4 weeks. Once in production, this item cannot be cancelled from an order and is not eligible for return.
Target Protein	MSH2
Clonality	Polyclonal
Storage/Stability	-20°C/1 year
Application	IHC-p,IF,ELISA
Recommended Dilution	Immunohistochemistry: 1/100 - 1/300. Immunofluorescence: 1/200 - 1/1000. ELISA: 1/20000. Not yet tested in other applications.
Concentration	1 mg/ml
Reactive Species	Human,Mouse,Rat
Host Species	Rabbit
Immunogen	Synthesized peptide derived from MSH2, at AA range: 510-590
Specificity	MSH2 Polyclonal Antibody detects endogenous levels of MSH2 protein.
Purification	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen
Form	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Gene Name	MSH2
Accession No.	P43246 P43247 P54275
Alternate Names	MSH2; DNA mismatch repair protein Msh2; hMSH2; MutS protein homolog 2

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Description	mutS homolog 2(MSH2) Homo sapiens This locus is frequently mutated in hereditary nonpolyposis colon cancer (HNPCC). When cloned, it was discovered to be a human homolog of the E. coli mismatch repair gene mutS, consistent with the characteristic alterations in microsatellite sequences (RER+ phenotype) found in HNPCC. Two transcript variants encoding different isoforms have been found for this gene. [provided by RefSeq, Apr 2012],
Cell Pathway/ Category	Mismatch repair,Pathways in cancer,Colorectal cancer,
Protein Expression	Blood,Liver,Muscle,Synovial membrane,Testis,
Subcellular Localization	nuclear chromosome, telomeric region,nucleoplasm,membrane,mismatch repair complex,MutSalpha complex,MutSbeta complex,

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Protein Function	disease:Defects in MSH2 are a cause of Muir-Torre syndrome (MTS) [MIM:158320]. MTS is a rare autosomal dominant disorder characterized by sebaceous neoplasms and visceral malignancy.,disease:Defects in MSH2 are a cause of susceptibility to endometrial cancer [MIM:608089].,disease:Defects in MSH2 are the cause of hereditary non-polyposis colorectal cancer type 1 (HNPCC1) [MIM:120435]. 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. 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. MSH2 mutations may predispose to hematological malignancies and multiple cafe-au-lait spots.,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.,PTM:Phosphorylated by PRKCZ, which may prevent MutS alpha degradation by the ubiquitin-proteasome pathway.,PTM:Phosphorylated upon DNA damage, probably by ATM or ATR.,sequence caution:The frameshift is caused by a single nucleotide deletion which is found in a HNPCC kindred.,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.,tissue specificity:Ubiquitously expressed.,

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