abcam

Product datasheet

Recombinant Human MSH2 protein ab114351

1 Image

Description

Product name	Recombinant Human MSH2 protein
Expression system	Wheat germ
Accession	P43246
Protein length	Full length protein
Animal free	No
Nature	Recombinant
Species	Human
Sequence	MAVQPKETLQLESAAEVGFVRFFQGMPEKPTTTVRLFDR GDFYTAHGEDA LLAAREVFKTQGVIKYMGPAGAKNLQSVVLSKMNFESFV KDLLLVRQYRV EVYKNRAGNKASKENDWYLAYKASPGNLSQFEDILFGNN DMSASIGVVGV KMSAVDGQRQVGVGYVDSIQRKLGLCEFPDNDQFSNLE ALLIQIGPKECV LPGGETAGDMGKLRQIIQRGGILITERKKADFSTKDIYQDLN RLLKGKKG EQMNSAVLPEMENQVAVSSLSAVIKFLELLSDDSNFGQF ELTTFDFSQYM KLDIAAVRALNLFQGSVEDTTGSQSLAALLNKCKTPQGQR LVNQWIKQPL MDKNRIEERLNLVEAFVEDAELRQTLQEDLLRRFPDLNRL AKKFQRQAAN LQDCYRLYQGINQLPNVIQALEKHEGKHQKLLLAVFVTPLT DLRSDFSKF QEMIETTLDMDQVENHEFLVKPSFDPNLSELREIMNDLEK KMQSTLISAA RDLGLDPGKQIKLDSSAQFGYYFRVTCKEEKVLRNNKNFS TVDIQKNGVK FTNSKLTSLNEEYTKNKTEYEEAQDAIVKEIVNISSGYVEPM QTLNDVLA QLDAVVSFAHVSNGAPVPYVRPAILEKGQGRIILKASRHAC VEVQDEIAF IPNDVYFEKDKQMFHIITGPNMGGKSTYIRQTGVIVLMAQIG CFVPCESA

EVSIVDCILARVGAGDSQLKGVSTFMAEMLETASILRSATK
DSLIIIDEL
GRGTSTYDGFGLAWAISEYIATKIGAFCMFATHFHELTALA
NQIPTVNNL
HVTALTTEETLTMLYQVKKGVCDQSFGIHVAELANFPKHVI
ECAKQKALE
LEEFQYIGESQGYDIMEPAAKKCYLEREQGEKIIQEFLSKV
KQMPFTEMS EENITIKLKQLKAEVIAKNNSFVNEIISRIKVTT

Predicted molecular weight 129 kDa including tags

Amino acids 1 to 934

Specifications

Our Abpromise guarantee covers the use of **ab114351** in the following tested applications.

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

Applications ELISA

SDS-PAGE

Western blot

Form Liquid

Preparation and Storage

Stability and Storage Shipped on dry ice. Upon delivery aliquot and store at -80ºC. Avoid freeze / thaw cycles.

pH: 8.00
Constituents: 0.3% Glutathione, 0.79% Tris HCl

General Info

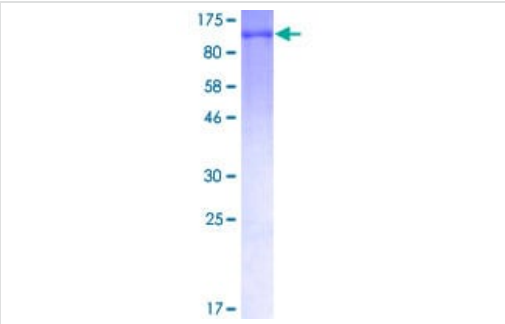
Function Component of the post-replicative DNA mismatch repair system (MMR). Forms two different heterodimers: MutS alpha (MSH2-MSH6 heterodimer) and MutS beta (MSH2-MSH3 heterodimer) which binds to DNA mismatches thereby initiating DNA repair. When bound, heterodimers bend the DNA helix and shields 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.

Tissue specificity Ubiquitously expressed.

Involvement in 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. Defects in MSH2 are a cause of Muir-Torre syndrome (MuToS) [MIM:158320]; also abbreviated MTS. MuToS is a rare autosomal dominant disorder characterized by sebaceous neoplasms and visceral malignancy. Defects in MSH2 are a cause of susceptibility to endometrial cancer (ENDMC) [MIM:608089]. Defects in MSH2 are a cause of hereditary non-polyposis colorectal cancer type 8 (HNPCC8) [MIM:613244]. HNPCC is a 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=HNPCC8 results from heterozygous deletion of 3-prime exons of EPCAM and intergenic regions directly upstream of MSH2, resulting in transcriptional read-through and epigenetic silencing of MSH2 in tissues expressing EPCAM.
Sequence similarities	Belongs to the DNA mismatch repair mutS family.
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.
Cellular localization	Nucleus.

Images



SDS-PAGE - Recombinant Human MSH2 protein (ab114351)

ab114351 analysed on a 12.5% SDS-PAGE gel stained with Coomassie Blue.

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