10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

MSH6 Antibody

Purified Mouse Monoclonal Antibody
Catalog # AO1161a

Product Information

Application	WB, E
Primary Accession	P52701
Reactivity	Human
Host	Mouse
Clonality	Monoclonal
Clone Names	3A10H7
Isotype	IgG1
Calculated MW	152786
Description	Defects in MSH6 are a cause of hereditary non-polyposis colorectal cancer (HNPCC) (Lynch syndrome). 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. MSH6 is central to mismatch DNA repair.
Immunogen	Purified recombinant fragment of MSH6 expressed in E. Coli.
Formulation	Ascitic fluid containing 0.03% sodium azide.

Additional Information

Gene ID	2956
Other Names	DNA mismatch repair protein Msh6, hMSH6, G/T mismatch-binding protein, GTBP, GTMBP, MutS-alpha 160 kDa subunit, p160, MSH6, GTBP
Dilution	WB~~1/500 - 1/2000 E~~N/A
Storage	Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	MSH6 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	MSH6 (HGNC:7329)

Synonyms	GTBP
Function	Component of the post-replicative DNA mismatch repair system (MMR). Heterodimerizes with MSH2 to form MutS alpha, which binds to DNA mismatches thereby initiating DNA repair. When bound, MutS alpha bends the DNA helix and shields approximately 20 base pairs, and recognizes single base mismatches and dinucleotide insertion-deletion loops (IDL) in the DNA. After mismatch binding, 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. Recruited on chromatin in G1 and early S phase via its PWWP domain that specifically binds trimethylated 'Lys-36' of histone H3 (H3K36me3): early recruitment to chromatin to be replicated allowing a quick identification of mismatch repair to initiate the DNA mismatch repair reaction.
Cellular Location	Nucleus. Chromosome. Note=Associates with H3K36me3 via its PWWP domain

References

1. Oncology (Williston Park). 2005 Apr;19(4):455-63. 2. Proc Natl Acad Sci U S A. 2006 Jan 17;103(3):558-63.

Images

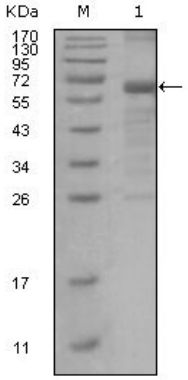

Figure 1: Western blot analysis using MSH6 mouse mAb against truncated MSH6 recombinant protein.

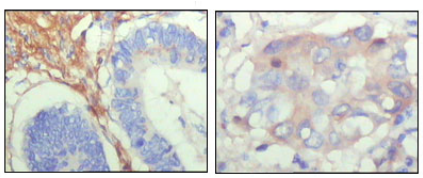

Figure 2: Immunohistochemical analysis of paraffin-embedded human colon cancer (left) and breast cancer (right) showing cytoplasmic localization with DAB staining using FBLN5 mouse mAb.