

Anti-MSH2 Antibody

Mouse monoclonal antibody to MSH2 Catalog # AP61621

Product Information

Application	WB, IHC
Primary Accession	<u>P43246</u>
Reactivity	Human
Host	Mouse
Clonality	Monoclonal
Calculated MW	104743

Additional Information

Gene ID	4436
Other Names	DNA mismatch repair protein Msh2; hMSH2; MutS protein homolog 2
Target/Specificity	KLH-conjugated synthetic peptide encompassing a sequence within human MSH2. The exact sequence is proprietary.
Dilution	WB~~1:1000 IHC~~1:100~500
Format	Mouse IgG. Liquid in PBS containing 50% glycerol, 0.2% BSA and 0.09% (W/V) sodium azide.
Storage	Store at -20 °C.Stable for 12 months from date of receipt

Protein Information

Name	MSH2
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. Recruits DNA helicase MCM9 to chromatin which unwinds the mismatch containing DNA strand (PubMed: <u>26300262</u>). 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.
Cellular Location	Nucleus. Chromosome
Tissue Location	Ubiquitously expressed.

Background

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