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MLH1 Antibody (Center)

Affinity Purified Rabbit Polyclonal Antibody (Pab)

Catalog # AP11686c

Specification

MLH1 Antibody (Center) - Product Information

Application	**FC, WB,E**
Primary Accession	P40692
Other Accession	NP_001161090.1, NP_001161091.1, NP_000240.1, NP_001161089.1
Reactivity	**Human**
Host	**Rabbit**
Clonality	**Polyclonal**
Isotype	**Rabbit IgG**
Antigen Region	**452-480**

MLH1 Antibody (Center) - Additional Information

Gene ID 4292

Other Names
DNA mismatch repair protein Mlh1, MutL protein homolog 1, MLH1, COCA2

Target/Specificity
This MLH1 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 452-480 amino acids from the Central region of human MLH1.

Dilution
FC~~1:10~50
WB~~1:1000
E~~Use at an assay dependent concentration.

Format
Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

Storage
Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions
MLH1 Antibody (Center) is for research use only and not for use in diagnostic or therapeutic procedures.

MLH1 Antibody (Center) - Protein Information

Name MLH1

Synonyms COCA2

Function Heterodimerizes with PMS2 to form MutL alpha, a component of the post-replicative DNA mismatch repair system (MMR). DNA repair is initiated by MutS alpha (MSH2-MSH6) or MutS beta (MSH2-MSH3) binding to a dsDNA mismatch, then MutL alpha is recruited to the heteroduplex. Assembly of the MutL-MutS-heteroduplex ternary complex in presence of RFC and PCNA is sufficient to activate endonuclease activity of PMS2. It introduces single-strand breaks near the mismatch and thus generates new entry points for the exonuclease EXO1 to degrade the strand containing the mismatch. DNA methylation would prevent cleavage and therefore assure that only the newly mutated DNA strand is going to be corrected. MutL alpha (MLH1-PMS2) interacts physically with the clamp loader subunits of DNA polymerase III, suggesting that it may play a role to recruit the DNA polymerase III to the site of the MMR. Also implicated in DNA damage signaling, a process which induces cell cycle arrest and can lead to apoptosis in case of major DNA damages. Heterodimerizes with MLH3 to form MutL gamma which plays a role in meiosis.

Cellular Location
Nucleus. Chromosome. Note=Recruited to chromatin in a MCM9- dependent manner.

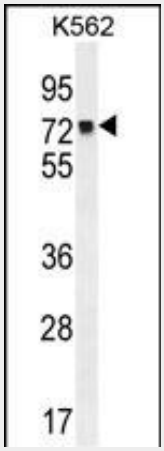
Tissue Location
Colon, lymphocytes, breast, lung, spleen, testis, prostate, thyroid, gall bladder and heart

MLH1 Antibody (Center) - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

MLH1 Antibody (Center) - Images



MLH1 Antibody (Center) (Cat. #AP11686c) western blot analysis in K562 cell line lysates (35ug/lane).This demonstrates the MLH1 antibody detected the MLH1 protein (arrow).

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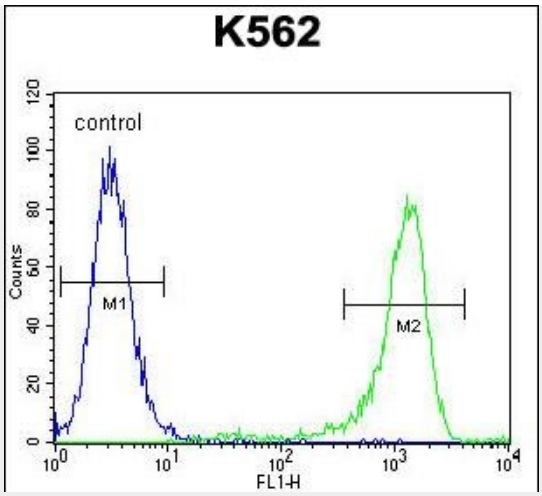

MLH1 Antibody (Center) (Cat. #AP11686c) flow cytometric analysis of K562 cells (right histogram) compared to a negative control cell (left histogram).FITC-conjugated goat-anti-rabbit secondary antibodies were used for the analysis.

MLH1 Antibody (Center) - Background

This gene was identified as a locus frequently mutated in hereditary nonpolyposis colon cancer (HNPCC). It is a human homolog of the E. coli DNA mismatch repair gene mutL, consistent with the characteristic alterations in microsatellite sequences (RER+phenotype) found in HNPCC. Alternative splicing results in multiple transcript variants encoding distinct isoforms. Additional transcript variants have been described, but their full-length natures have not been determined.

MLH1 Antibody (Center) - References

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Borras, E., et al. Cancer Res. 70(19):7379-7391(2010)
Mangoni, M., et al. Int. J. Radiat. Oncol. Biol. Phys. (2010) In press :
Ho-Pun-Cheung, A., et al. Pharmacogenomics J. (2010) In press :