

# MLH1 Antibody

Purified Rabbit Polyclonal Antibody (Pab) Catalog # AP51343

#### **Product Information**

**Application** WB, ICC **Primary Accession** P40692 Reactivity Human Host Rabbit Clonality Polyclonal Calculated MW 84601

### **Additional Information**

4292 Gene ID

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

Target/Specificity KLH-conjugated synthetic peptide encompassing a sequence within the center

region of human MLH1. The exact sequence is proprietary.

**Dilution** WB~~1:1000 ICC~~N/A

**Format** 0.01M PBS, pH 7.2, 0.09% (W/V) Sodium azide, Glycerol 50%

Store at -20 °C. Stable for 12 months from date of receipt **Storage** 

#### **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

## Background

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-MSH6) 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.

#### References

Bronner C.E., et al. Nature 368:258-261(1994). Papadopoulos N., et al. Science 263:1625-1629(1994). Kolodner R.D., et al. Cancer Res. 55:242-248(1995). Han H.-J., et al. Hum. Mol. Genet. 4:237-242(1995). Ota T., et al. Nat. Genet. 36:40-45(2004).

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