Product Name: MLH1 Rabbit Polyclonal Antibody
Catalog #: APRab13947

Summary

Production Name	MLH1 Rabbit Polyclonal Antibody
Description	Rabbit Polyclonal Antibody
Host	Rabbit
Application	WB,IHC-P
Reactivity	Human,Mouse,Rat

Performance

Conjugation	Unconjugated
Modification	Unmodified
Isotype	IgG
Clonality	Polyclonal
Form	Liquid
Storage	Store at 4°C short term. Aliquot and store at -20°C long term. Avoid freeze/thaw cycles.
Buffer	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% New type preservative N.
Purification	Affinity purification

Immunogen

Gene Name	MLH1
Alternative Names	MLH1; COCA2; DNA mismatch repair protein Mlh1; MutL protein homolog 1
Gene ID	4292.0
SwissProt ID	P40692.The antiserum was produced against synthesized peptide derived from human MLH1. AA range:441-490

Application

Dilution Ratio	WB 1:500-2000, IHC-P 1:50-300
Molecular Weight	85kDa

Background

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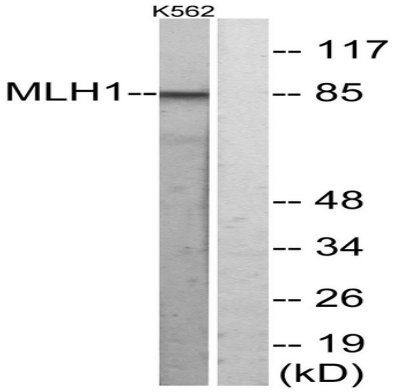
This gene was identified as a locus frequently mutated in hereditary nonpolyposis colon cancer (HNPCC). It is a human homolog of the E. coli DNA mismatch repair gene mutL, consistent with the characteristic alterations in microsatellite sequences (RER+phenotype) found in HNPCC. Alternative splicing results in multiple transcript variants encoding distinct isoforms. Additional transcript variants have been described, but their full-length natures have not been determined. [provided by RefSeq, Nov 2009],disease:Defects in MLH1 are a cause of Muir-Torre syndrome (MTS) [MIM:158320]. MTS is a rare autosomal dominant disorder characterized by sebaceous neoplasms and visceral malignancy.,disease:Defects in MLH1 are a cause of susceptibility to endometrial cancer [MIM:608089].,disease:Defects in MLH1 are a cause of Turcot syndrome [MIM:276300]; also called mismatch repair cancer syndrome (MMRCS). Turcot syndrome is an autosomal dominant disorder characterized by malignant tumors of the brain associated with multiple colorectal adenomas. Skin features include sebaceous cysts, hyperpigmented and cafe au lait spots.,disease:Defects in MLH1 are the cause of hereditary non-polyposis colorectal cancer type 2 (HNPCC2) [MIM:609310]. 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, and accounts for 15% of all colon cancers. 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.,disease:Defects in MLH1 may contribute to lobular carcinoma in situ (LCIS), a non-invasive neoplastic disease of the breast.,disease:Some epigenetic changes can be transmitted unchanged through the germline (termed 'epigenetic inheritance'). Evidence that this mechanism occurs in humans is provided by the identification of individuals in whom 1 allele of the MLH1 gene is epigenetically silenced throughout the soma (implying a germline event). These individuals are affected by HNPCC but does not have identifiable mutations in MLH1, even though it is silenced, which demonstrats that an epimutation can phenocopy a genetic disease.,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-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.,similarity:Belongs to the DNA mismatch repair mutL/hexB family.,subunit:Heterodimer of MLH1 and PMS2 (MutL alpha), MLH1 and PMS1 (MutL beta) or MLH1 and MLH3 (MutL gamma). Forms a ternary complex with MutS alpha (MSH2-MSH6) or MutS beta (MSH2-MSH3). Part of the BRCA1-associated genome surveillance complex (BASC), which contains BRCA1, MSH2, MSH6, MLH1, ATM, BLM, PMS2 and the RAD50-MRE11-NBS1 protein complex. This association could be a dynamic process changing throughout the cell cycle and within subnuclear domains. Interacts with MBD4. Interacts with EXO1.,tissue specificity:Colon, lymphocytes, breast, lung, spleen, testis, prostate, thyroid, gall bladder and heart.,

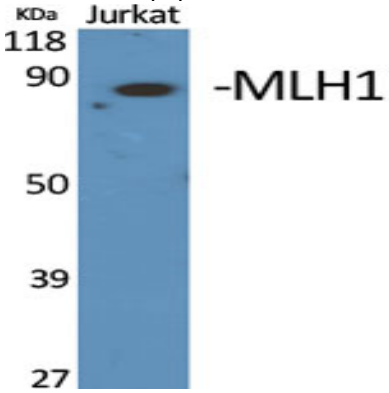
Research Area

Mismatch repair;Pathways in cancer;Colorectal cancer;Endometrial cancer;

Image Data



Western blot analysis of lysates from K562 cells, using MLH1 Antibody. The lane on the right is blocked with the synthesized peptide.



Western Blot analysis of various cells using MLH1 Polyclonal Antibody cells nucleus extracted by Minute TM Cytoplasmic and Nuclear Fractionation kit （SC-003,Inventbiotech,MN,USA）.

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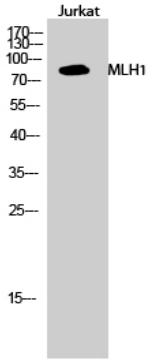

Western Blot analysis of Jurkat cells using MLH1 Polyclonal Antibody cells nucleus extracted by Minute TM Cytoplasmic and Nuclear Fractionation kit （SC-003,Inventbiotech,MN,USA）.

Note

For research use only.