

Anti-MLH1 antibody ab9144

★★★★☆ [2 Abreviews](#) [3 References](#) [1 图像](#)

概述

产品名称	Anti-MLH1抗体
描述	兔多克隆抗体to MLH1
宿主	Rabbit
经测试应用	适用于: IHC-P, WB, IP
种属反应性	与反应: Mouse, Human 预测可用于: Rat, Chicken, Guinea pig, Cow, Dog, Turkey, Pig, Chimpanzee, Rhesus monkey, Gorilla, Orangutan, Xenopus tropicalis, Platypus 
免疫原	Synthetic peptide (Human) - which represented a portion within the last 100 amino acids of the human Homolog 1 of E. coli MutL Protein (GenBank PID 730028), conjugated to KLH.

性能

形式	Liquid
存放说明	Shipped at 4°C. Upon delivery aliquot and store at -20°C. Avoid freeze / thaw cycles.
存储溶液	Preservative: 0.1% Sodium azide Constituents: 0.021% PBS, 1.764% Sodium citrate, 1.815% Tris
纯度	Immunogen affinity purified
纯化说明	Antibodies were affinity purified using the peptide immobilized on solid support. Antibody concentration was determined by extinction coefficient: absorbance at 280 nm of 1.4 equals 1.0 mg of IgG.
克隆	多克隆
同种型	IgG

应用

The Abpromise guarantee **Abpromise™** 承诺保证使用 ab9144 于以下的经测试应用

“应用说明”部分 下显示的仅为推荐的起始稀释度;实际最佳的稀释度/浓度应由使用者检定。

应用	Ab评论	说明
IHC-P		Use at an assay dependent concentration.
WB	★★★★☆ (2)	1/500 - 1/5000. Detects a band of approximately 85 kDa (predicted molecular weight: 84 kDa).
IP		Use at 1-4 - 4 µg/mg of lysate.

靶标

功能

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.

组织特异性

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

疾病相关

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.

Defects in MLH1 are a cause of mismatch repair cancer syndrome (MMRCS) [MIM:276300]; also known as Turcot syndrome or brain tumor-polyposis syndrome 1 (BTPS1). MMRCS 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.

Defects in MLH1 are a cause of Muir-Torre syndrome (MuToS) [MIM:158320]; also abbreviated MTS. MuToS is a rare autosomal dominant disorder characterized by sebaceous neoplasms and visceral malignancy.

Note=Defects in MLH1 may contribute to lobular carcinoma in situ (LCIS), a non-invasive neoplastic disease of the breast.

Defects in MLH1 are a cause of susceptibility to endometrial cancer (ENDMC) [MIM:608089].

Note=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 demonstrates that an epimutation can phenocopy a genetic disease.

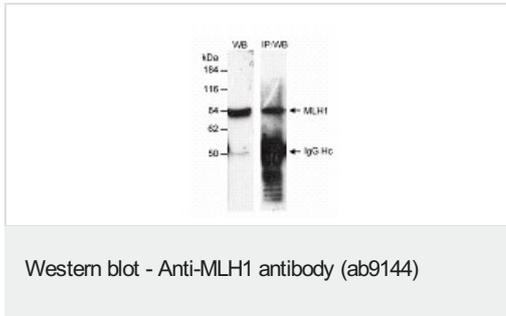
序列相似性

Belongs to the DNA mismatch repair mutL/hexB family.

细胞定位

Nucleus.

图片



Detection of Human MLH1 by Western Blot. *Sample:* Nuclear extract (50 µg for WB; 1 mg for IP) from HeLa cells. *Antibody:* Affinity purified rabbit anti-MLH1 (ab9144) used at 1 µg/ml for WB and 5 µg/mg lysate for IP. *Detection:* Chemiluminescence.

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