


Product datasheet

Anti-MLH1 antibody [EPR3893] ab108622

KO VALIDATED Recombinant RabMAb

★★★★★ 1 Abreviews 5 Images

Overview

Product name	Anti-MLH1 antibody [EPR3893]
Description	Rabbit monoclonal [EPR3893] to MLH1
Host species	Rabbit
Tested applications	Suitable for: WB Unsuitable for: Flow Cyt, IHC-P or IP
Species reactivity	Reacts with: Mouse, Human Predicted to work with: Rat 
Immunogen	Synthetic peptide. This information is proprietary to Abcam and/or its suppliers.
Positive control	WB: HeLa, HAP1, Jurkat, 293, K562 and SH-SY5Y cell lysates.
General notes	<p>To see more of the key markers and tools you need to study the hallmarks of cancer, including genome instability and mutation, please visit the following page.</p> <p>This product is a recombinant monoclonal antibody, which offers several advantages including:</p> <ul style="list-style-type: none">- High batch-to-batch consistency and reproducibility- Improved sensitivity and specificity- Long-term security of supply- Animal-free production <p>For more information see here.</p> <p>Our RabMAb[®] technology is a patented hybridoma-based technology for making rabbit monoclonal antibodies. For details on our patents, please refer to RabMAb[®] patents.</p>

Properties

Form	Liquid
Storage instructions	Shipped at 4°C. Store at -20°C. Stable for 12 months at -20°C.
Storage buffer	pH: 7.20 Preservative: 0.05% Sodium azide Constituents: 0.1% BSA, 40% Glycerol (glycerin, glycerine), 9.85% Tris glycine, 50% Tissue culture supernatant
Purity	Tissue culture supernatant

Clonality	Monoclonal
Clone number	EPR3893
Isotype	IgG

Applications

The Abpromise guarantee Our **Abpromise guarantee** covers the use of ab108622 in the following tested applications. The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Application	Abreviews	Notes
WB	★★★★★ (1)	1/1000 - 1/10000. Detects a band of approximately 85 kDa (predicted molecular weight: 85 kDa).

Application notes Is unsuitable for Flow Cyt, IHC-P or IP.

Target

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.

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

Involvement in 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.

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

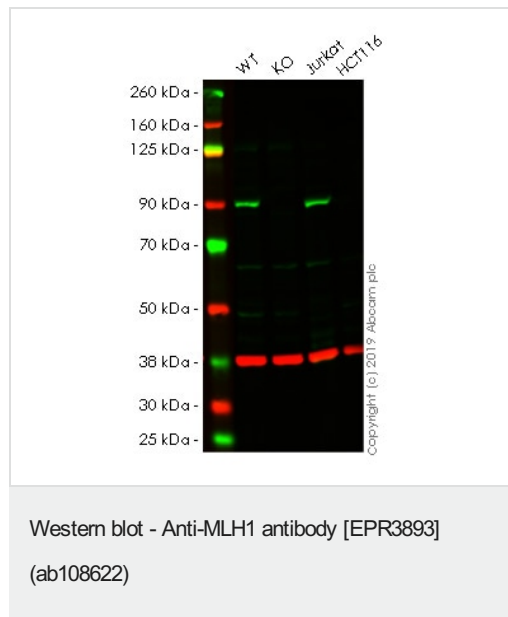
Sequence similarities

Belongs to the DNA mismatch repair mutL/hexB family.

Cellular localization

Nucleus.

Images



All lanes : Anti-MLH1 antibody [EPR3893] (ab108622) at 1/1000 dilution

Lane 1 : Wild-type HeLa cell lysate

Lane 2 : MLH1 knockout HeLa cell lysate

Lane 3 : Jurkat cell lysate

Lane 4 : HCT116 cell lysate

Lysates/proteins at 20 µg per lane.

Performed under reducing conditions.

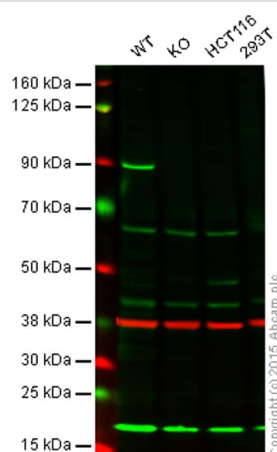
Predicted band size: 85 kDa

Observed band size: 85 kDa

Lanes 1-4: Merged signal (red and green). Green - ab108622 observed at 90 kDa. Red - loading control [ab8245](#) observed at 37 kDa.

ab108622 Anti-MLH1 antibody [EPR3893] was shown to

specifically react with MLH1 in wild-type HeLa cells. Loss of signal was observed when knockout cell line [ab267223](#) (knockout cell lysate [ab257172](#)) was used. Wild-type and MLH1 knockout samples were subjected to SDS-PAGE. ab108622 and Anti-GAPDH antibody [6C5] - Loading Control were incubated overnight at 4°C at 1 in 1000 dilution and 1 in 20000 dilution respectively. Blots were developed with Goat anti-Mouse IgG H&L (IRDye® 800CW) preadsorbed ([ab216772](#)) and Goat Anti-Rabbit IgG H&L (IRDye® 680RD) preadsorbed ([ab216777](#)) secondary antibodies at 1 in 20000 dilution for 1 hour at room temperature before imaging.



Western blot - Anti-MLH1 antibody [EPR3893] (ab108622)

All lanes : Anti-MLH1 antibody [EPR3893] (ab108622) at 1/1000 dilution

Lane 1 : Wild-type HAP1 cell lysate

Lane 2 : MLH1 knockout HAP1 cell lysate

Lane 3 : HCT116 cell lysate

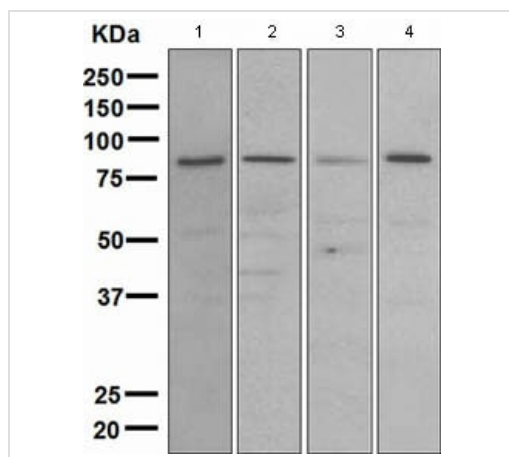
Lane 4 : 293T cell lysate

Lysates/proteins at 20 µg per lane.

Predicted band size: 85 kDa

Lanes 1 - 4: Merged signal (red and green). Green - ab108622 observed at 88 kDa. Red - loading control, [ab8245](#), observed at 37 kDa.

ab108622 was shown to recognize MLH1 in wild-type HAP1 cells along with additional cross-reactive bands. No band was observed when MLH1 knockout samples were examined. Wild-type and MLH1 knockout samples were subjected to SDS-PAGE. ab108622 and [ab8245](#) (loading control to GAPDH) were diluted 1/1000 and 1/2000 and incubated overnight at 4°C. Blots were developed with Goat anti-Rabbit IgG H&L (IRDye® 800CW) preadsorbed [ab216773](#) and Goat anti-Mouse IgG H&L (IRDye® 680RD) preadsorbed [ab216776](#) secondary antibodies at 1/10,000 dilution for 1 hour at room temperature before imaging.



Western blot - Anti-MLH1 antibody [EPR3893]
(ab108622)

All lanes : Anti-MLH1 antibody [EPR3893] (ab108622) at 1/1000 dilution

Lane 1 : 293 cell lysate

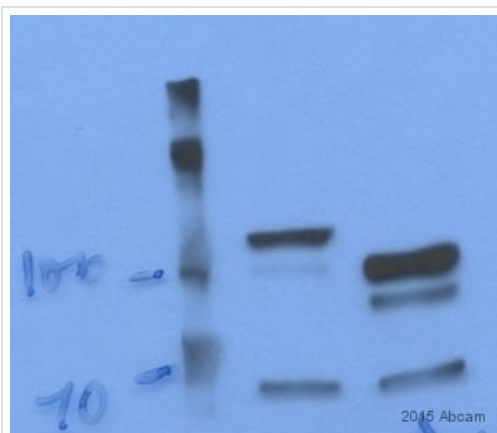
Lane 2 : Jurkat cell lysate

Lane 3 : K562 cell lysate

Lane 4 : SH-SY5Y cell lysate

Lysates/proteins at 10 µg per lane.

Predicted band size: 85 kDa



Western blot - Anti-MLH1 antibody [EPR3893]
(ab108622)

This image is courtesy of an anonymous abreview.

All lanes : Anti-MLH1 antibody [EPR3893] (ab108622) at 1/1000 dilution

Lane 1 : Hela Cells Whole Cell Lysates

Lane 2 : HCT116 Whole Cell Lysates

Lysates/proteins at 30 µg per lane.

Secondary

All lanes : Goat Polyclonal to Rabbit IgG (HRP) at 1/2000 dilution





Developed using the ECL technique.

Performed under reducing conditions.

Predicted band size: 85 kDa

Exposure time: 30 seconds

Why choose a recombinant antibody?

 <p>Research with confidence Consistent and reproducible results</p>	 <p>Long-term and scalable supply Recombinant technology</p>
 <p>Success from the first experiment Confirmed specificity</p>	 <p>Ethical standards compliant Animal-free production</p>

Anti-MLH1 antibody [EPR3893] (ab108622)

Please note: All products are "FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC PROCEDURES"

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