SANTA CRUZ BIOTECHNOLOGY, INC.

MLH1 (A-8): sc-133228

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

DNA-mismatch repair (MMR) is an essential process in maintaining genetic stability. Lack of a functional DNA-mismatch repair pathway is a common characteristic of several different types of human cancers, either due to an MMR gene mutation or promoter-methylation gene silencing. MLH1 is an integral part of the protein complex responsible for mismatch repair expressed in lymphocytes, heart, colon, breast, lung, spleen, testis, prostate, thyroid and gall bladder, and is methylated in several ovarian tumors. Loss of MLH1 protein expression is associated with a mutated phenotype, microsatellite instability and a predisposition to cancer. In hereditary nonpolyposis colorectal cancer (HNPCC), an autosomal dominant inherited cancer syndrome that signifies a high risk of colorectal and various other types of cancer, the MLH1 gene exhibits a pathogenic mutation. Certain cancer cell lines, including leukemia CCRF-CEM, colon HCT 116 and KM12, and ovarian cancers SK-OV-3 and IGROV-1, show complete deficiency of MLH1, while MLH1 is expressed in 60% of melanomas, 70% of noninvasive squamous cell carcinomas and 30% of invasive squamous cell carcinomas.

REFERENCES

1. Taverna, P., et al. 2000. Characterization of MLH1 and MSH2 DNA mismatch repair proteins in cell lines of the NCI anticancer drug screen. Cancer Chemother. Pharmacol. 46: 507-516.
2. Jarvinen, H.J., et al. 2000. Surveillance on mutation carriers of DNA mismatch repair genes. Ann. Chir. Gynaecol. 89: 207-210.
3. Korabiowska, M., et al. 2000. Analysis of the DNA mismatch repair proteins expression in malignant melanomas. Anticancer Res. 20: 4499-4505.
4. Giarnieri, E., et al. 2000. MSH2, MLH1, Fhit, p53, Bcl-2, and Bax expression in invasive and *in situ* squamous cell carcinoma of the uterine cervix. Clin. Cancer Res. 6: 3600-3606.
5. Korabiowska, M., et al. 2001. Relation between DNA ploidy status and the expression of the DNA-mismatch repair genes MLH1 and MSH2 in cytological specimens of melanoma lymph node and liver metastases. Diagn. Cytopathol. 24: 157-162.

CHROMOSOMAL LOCATION

Genetic locus: MLH1 (human) mapping to 3p22.2; Mlh1 (mouse) mapping to 9 F3.

SOURCE

MLH1 (A-8) is a mouse monoclonal antibody raised against amino acids 457-756 mapping at the C-terminus of MLH1 of human origin.

PRODUCT

Each vial contains 200 µg IgG_{2a} kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin. Also available as TransCruz reagent for ChIP application, sc-133228 X, 200 µg/0.1 ml.

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

APPLICATIONS

MLH1 (A-8) is recommended for detection of MLH1 of mouse, rat and human origin by Western Blotting (starting dilution 1:100, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

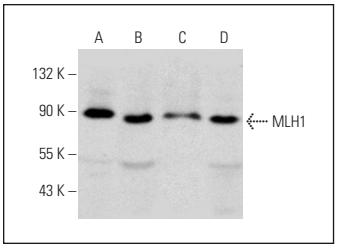
Suitable for use as control antibody for MLH1 siRNA (h): sc-35943, MLH1 siRNA (m): sc-35944, MLH1 shRNA Plasmid (h): sc-35943-SH, MLH1 shRNA Plasmid (m): sc-35944-SH, MLH1 shRNA (h) Lentiviral Particles: sc-35943-V and MLH1 shRNA (m) Lentiviral Particles: sc-35944-V.

MLH1 (A-8) X TransCruz antibody is recommended for ChIP assays.

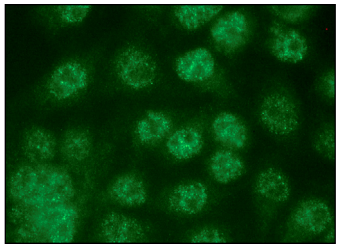
Molecular Weight of MLH1: 85 kDa.

Positive Controls: SK-N-MC cell lysate: sc-2237, Jurkat whole cell lysate: sc-2204 or MCF7 whole cell lysate: sc-2206.

DATA



MLH1 (A-8): sc-133228. Western blot analysis of MLH1 expression in Jurkat (**A**), MCF7 (**B**), SK-N-MC (**C**) and C32 (**D**) whole cell lysates.



MLH1 (A-8): sc-133228. Immunofluorescence staining of methanol-fixed HeLa cells showing nuclear localization.

SELECT PRODUCT CITATIONS

1. D'Arcy, B.M., et al. 2019. Biochemical and structural characterization of two variants of uncertain significance in the PMS2 gene. Hum. Mutat. 40: 458-471.
2. Doukas, S.G., et al. 2020. The effect of NNK, a tobacco smoke carcinogen, on the miRNA and mismatch DNA repair expression profiles in lung and head and neck squamous cancer cells. Cells 9: 1031.
3. D'Arcy, B.M., et al. 2022. PMS2 variant results in loss of ATPase activity without compromising mismatch repair. Mol. Genet. Genomic Med. 10: e1908.
4. Doukas, S.G., et al. 2022. The effect of tobacco smoke N-nitrosamines, NNK and NDEA, and nicotine, on DNA mismatch repair mechanism and miRNA markers, in hypopharyngeal squamous cell carcinoma: an *in vivo* model and clinical evidence. Curr. Oncol. 29: 5531-5549.

RESEARCH USE

For research use only, not for use in diagnostic procedures.