

MLLT10 (AF10) Antibody (Center)

Purified Rabbit Polyclonal Antibody (Pab)

Catalog # AP1906b

Product Information

Application	WB, IF, E
Primary Accession	P55197
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	113320
Antigen Region	294-323

Additional Information

Gene ID	8028
Other Names	Protein AF-10, ALL1-fused gene from chromosome 10 protein, MLLT10, AF10
Target/Specificity	This MLLT10 (AF10) antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 294-323 amino acids from the Central region of human MLLT10 (AF10).
Dilution	WB~~1:1000 IF~~1:10~50 E~~Use at an assay dependent concentration.
Format	Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.
Storage	Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	MLLT10 (AF10) Antibody (Center) is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	MLLT10 (HGNC:16063)
Function	Probably involved in transcriptional regulation. In vitro or as fusion protein with KMT2A/MLL1 has transactivation activity. Binds to cruciform DNA. In cells, binding to unmodified histone H3 regulates DOT1L functions including histone H3 'Lys-79' dimethylation (H3K79me2) and gene activation (PubMed: 26439302).

Cellular Location

Nucleus.

Tissue Location

Expressed abundantly in testis.

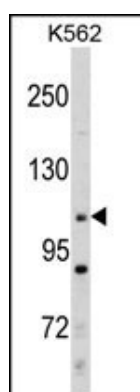
Background

Translocations affecting chromosome 11q23 involve many partner chromosome regions and occur in various leukemias. The 11q23 gene involved in the translocations is MLL. MLLT10 is the partner gene to MLL1 involved in t(10;11)(p12;q23) translocations. In an analysis of two leukemia patients, the in t(10;11)(p12;q23) translocation fuses MLL1, a SET domain containing histone methyltransferase, to the MLLT10 gene. The MLLT10 gene encodes a predicted 1,027-amino acid protein containing an N-terminal zinc finger and a C-terminal leucine zipper domain. The MLLT10 gene is one of the few MLL partner genes to be independently rearranged with a third gene in leukemia, the CALM gene in the t(10;11)(p12;q14) translocation. Chimeric fusion proteins MLL/AF10 and CALM/AF10 consistently retain the leucine zipper motif of MLLT10. The leucine zipper interacts with GAS41, a protein previously identified as the product of an amplified gene in a glioblastoma. GAS41 interacts with integrase interactor-1 (INI1), a component of the SWI/SNF complex, which acts to remodel chromatin and to modulate transcription. Retention of the leucine zipper in the MLL and CALM fusions suggested that a key feature of these chimeric proteins may be their ability to interfere in normal gene regulation through interaction with the adenosine triphosphate-dependent chromatin remodeling complexes.

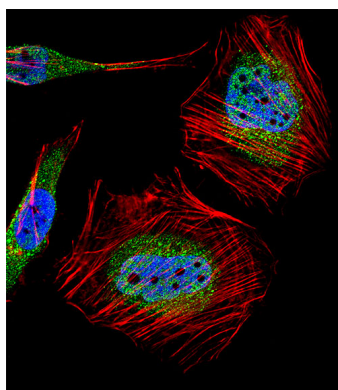
References

- Perrin, L., et al., Mol. Cell. Biol. 23(1):119-130 (2003).
Roll, P., et al., Cancer Genet. Cytogenet. 135(2):187-191 (2002).
Nakamura, T., et al., Mol. Cell 10(5):1119-1128 (2002).
Debernardi, S., et al., Blood 99(1):275-281 (2002).
Cai, Y., et al., Mol. Reprod. Dev. 61(1):126-134 (2002).

Images



Western blot analysis of MLLT10 (Cat. #AP1906b) in K562 cell line lysates (35ug/lane). MLLT10 (arrow) was detected using the purified Pab.(2ug/ml)



Fluorescent confocal image of HeLa cell stained with MLLT10 (AF10) Antibody (Center)(Cat#AP1906b).HeLa cells were fixed with 4% PFA (20 min), permeabilized with Triton X-100 (0.1%, 10 min), then incubated with MLLT10 primary antibody (1:25, 1 h at 37°C). For secondary antibody, Alexa Fluor® 488 conjugated donkey anti-rabbit antibody (green) was used (1:400, 50 min at 37°C).Cytoplasmic actin was counterstained with Alexa Fluor® 555 (red) conjugated Phalloidin (7units/ml, 1 h at 37°C). Nuclei were counterstained with DAPI (blue) (10 µg/ml, 10 min). MLLT10 immunoreactivity is localized to Nucleus significantly and Cytoplasm weakly.

Citations

- [The leukemogenic AF4-MLL fusion protein causes P-TEFb kinase activation and altered epigenetic signatures.](#)

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