10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

AF9 (MLLT3) Antibody (C-term K486)

Purified Rabbit Polyclonal Antibody (Pab)

Catalog # AP6190a

Specification

AF9 (MLLT3) Antibody (C-term K486) - Product Information

Application	**WB, IHC-P,E**
Primary Accession	P42568
Other Accession	A2AM29
Reactivity	**Human**
Predicted	**Mouse**
Host	**Rabbit**
Clonality	**Polyclonal**
Isotype	**Rabbit IgG**
Antigen Region	**471-502**

AF9 (MLLT3) Antibody (C-term K486) - Additional Information

Gene ID 4300

Other Names
Protein AF-9, ALL1-fused gene from chromosome 9 protein, Myeloid/lymphoid or mixed-lineage leukemia translocated to chromosome 3 protein, YEATS domain-containing protein 3, MLLT3, AF9, YEATS3

Target/Specificity
This AF9 (MLLT3) antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 471-502 amino acids from the C-terminal region of human AF9 (MLLT3).

Dilution
WB~~1:1000
IHC-P~~1:50~100
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
AF9 (MLLT3) Antibody (C-term K486) is for research use only and not for use in diagnostic or therapeutic procedures.

AF9 (MLLT3) Antibody (C-term K486) - Protein Information

Name MLLT3 {ECO:0000303|PubMed:16001262, ECO:0000312|HGNC:HGNC:7136}

Function Chromatin reader component of the super elongation complex (SEC), a complex required to increase the catalytic rate of RNA polymerase II transcription by suppressing transient pausing by the polymerase at multiple sites along the DNA (PubMed:20159561, PubMed:20471948, PubMed:25417107, PubMed:27105114, PubMed:27545619). Specifically recognizes and binds acylated histone H3, with a preference for histone H3 that is crotonylated (PubMed:25417107, PubMed:27105114, PubMed:27545619, PubMed:30374167, PubMed:30385749). Crotonylation marks active promoters and enhancers and confers resistance to transcriptional repressors (PubMed:25417107, PubMed:27105114, PubMed:27545619). Recognizes and binds histone H3 crotonylated at 'Lys-9' (H3K9cr), and with slightly lower affinity histone H3 crotonylated at 'Lys-18' (H3K18cr) (PubMed:27105114). Also recognizes and binds histone H3 acetylated and butyrylated at 'Lys-9' (H3K9ac and H3K9bu, respectively), but with lower affinity than crotonylated histone H3 (PubMed:25417107, PubMed:27105114, PubMed:30385749). In the SEC complex, MLLT3 is required to recruit the complex to crotonylated histones (PubMed:27105114, PubMed:27545619). Recruitment of the SEC complex to crotonylated histones promotes recruitment of DOT1L on active chromatin to deposit histone H3 'Lys-79' methylation (H3K79me) (PubMed:25417107). Plays a key role in hematopoietic stem cell (HSC) maintenance by preserving, rather than conferring, HSC stemness (PubMed:31776511). Acts by binding to the transcription start site of active genes in HSCs and sustaining level of H3K79me2, probably by recruiting DOT1L (PubMed:31776511).

Cellular Location
Nucleus {ECO:0000255|PROSITE-ProRule:PRU00376, ECO:0000269|PubMed:27105114}. Chromosome. Note=Colocalizes with acylated histone H3 (PubMed:25417107, PubMed:27105114). Colocalizes with histone H3 crotonylated at 'Lys-18' (H3K18cr) (PubMed:27105114)

Tissue Location
Enriched in undifferentiated hematopoietic stem cells in fetal liver, cord blood and bone marrow

AF9 (MLLT3) Antibody (C-term K486) - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

AF9 (MLLT3) Antibody (C-term K486) - Images

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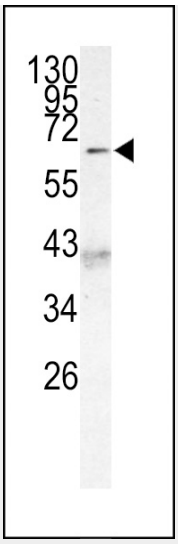
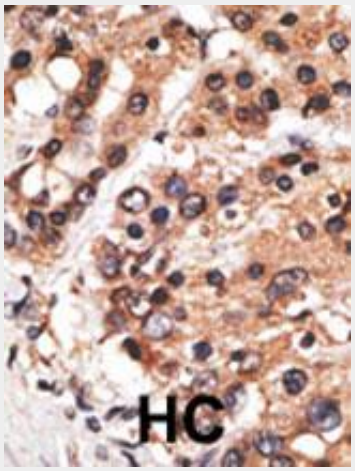


Western blot analysis of AF9 (MLLT3) Antibody (C-term K486) (Cat.#AP6190a) in 293 cell line lysates (35ug/lane). MLLT3(arrow) was detected using the purified Pab.



Formalin-fixed and paraffin-embedded human cancer tissue reacted with the primary antibody, which was peroxidase-conjugated to the secondary antibody, followed by AEC staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has not been evaluated. BC = breast carcinoma; HC = hepatocarcinoma.

AF9 (MLLT3) Antibody (C-term K486) - Background

The human AF9 gene is one of the most common fusion partner genes with the ALL1 gene at 11q23 (also called MLL), resulting in the t(9;11)(p22;q23). The AF9 gene is more than 100 kb, and 2 patient breakpoint cluster regions (BCRs) have been identified; BCR1 is within intron 4, previously called site A, whereas BCR2 or site B spans introns 7 and 8. Several different structural elements have been identified in AF9, including a colocalizing in vivo DNA topo II cleavage site and an in vitro DNase I hypersensitive (DNase 1 HS) site in intron 7 in BCR2. Reversibility experiments demonstrated a religation of the topo II cleavage sites. In addition, 2 scaffold associated regions (SARs) are located centromeric to the topo II and DNase I HS cleavage sites and border breakpoint regions in 2 leukemic cells lines: SAR1 is located in intron 4, whereas SAR2 encompasses parts of exons 5-7. The patient breakpoint regions of AF9 share the same structural elements as the MLL BCR. A DNA breakage and repair model for nonhomologous recombination between MLL and its partner genes, particularly AF9, has been proposed.

AF9 (MLLT3) Antibody (C-term K486) - References

Iida, S., et al., Oncogene 8(11):3085-3092 (1993).
Nakamura, T., et al., Proc. Natl. Acad. Sci. U.S.A. 90(10):4631-4635 (1993).
Strissel, P. L., et al., Hum. Molec. Genet. 9: 1671-1679 (2000).

AF9 (MLLT3) Antibody (C-term K486) - Citations

- The leukemogenic AF4-MLL fusion protein causes P-TEFb kinase activation and altered epigenetic signatures.
- The mixed-lineage leukemia fusion partner AF4 stimulates RNA polymerase II transcriptional elongation and mediates coordinated chromatin remodeling.