

**AF9 (MLLT3) Antibody (Center H197) Blocking peptide**  
Synthetic peptide  
Catalog # BP6190d**Specification**

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**AF9 (MLLT3) Antibody (Center H197) Blocking peptide - Product Information**Primary Accession [P42568](#)**AF9 (MLLT3) Antibody (Center H197) Blocking peptide - 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**

The synthetic peptide sequence used to generate the antibody [AP6190d](/product/products/AP6190d) was selected from the Center region of human MLLT3. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

**Format**

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

**Storage**

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

**Precautions**

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

**AF9 (MLLT3) Antibody (Center H197) Blocking peptide - 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](http://www.uniprot.org/citations/20159561), PubMed: [20471948](http://www.uniprot.org/citations/20471948), PubMed: [25417107](http://www.uniprot.org/citations/25417107), PubMed: [27105114](http://www.uniprot.org/citations/27105114), PubMed: [27545619](http://www.uniprot.org/citations/27545619)). Specifically recognizes and binds acylated histone H3, with a preference for histone H3 that is crotonylated (PubMed: [25417107](http://www.uniprot.org/citations/25417107)),

PubMed: <a href="http://www.uniprot.org/citations/27105114" target="\_blank">27105114</a>, PubMed: <a href="http://www.uniprot.org/citations/27545619" target="\_blank">27545619</a>, PubMed: <a href="http://www.uniprot.org/citations/30374167" target="\_blank">30374167</a>, PubMed: <a href="http://www.uniprot.org/citations/30385749" target="\_blank">30385749</a>). Crotonylation marks active promoters and enhancers and confers resistance to transcriptional repressors (PubMed: <a href="http://www.uniprot.org/citations/25417107" target="\_blank">25417107</a>, PubMed: <a href="http://www.uniprot.org/citations/27105114" target="\_blank">27105114</a>, PubMed: <a href="http://www.uniprot.org/citations/27545619" target="\_blank">27545619</a>). Recognizes and binds histone H3 crotonylated at 'Lys-9' (H3K9cr), and with slightly lower affinity histone H3 crotonylated at 'Lys-18' (H3K18cr) (PubMed: <a href="http://www.uniprot.org/citations/27105114" target="\_blank">27105114</a>). 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: <a href="http://www.uniprot.org/citations/25417107" target="\_blank">25417107</a>, PubMed: <a href="http://www.uniprot.org/citations/27105114" target="\_blank">27105114</a>, PubMed: <a href="http://www.uniprot.org/citations/30385749" target="\_blank">30385749</a>). In the SEC complex, MLLT3 is required to recruit the complex to crotonylated histones (PubMed: <a href="http://www.uniprot.org/citations/27105114" target="\_blank">27105114</a>, PubMed: <a href="http://www.uniprot.org/citations/27545619" target="\_blank">27545619</a>). Recruitment of the SEC complex to crotonylated histones promotes recruitment of DOT1L on active chromatin to deposit histone H3 'Lys-79' methylation (H3K79me) (PubMed: <a href="http://www.uniprot.org/citations/25417107" target="\_blank">25417107</a>). Plays a key role in hematopoietic stem cell (HSC) maintenance by preserving, rather than conferring, HSC stemness (PubMed: <a href="http://www.uniprot.org/citations/31776511" target="\_blank">31776511</a>). Acts by binding to the transcription start site of active genes in HSCs and sustaining level of H3K79me2, probably by recruiting DOT1L (PubMed: <a href="http://www.uniprot.org/citations/31776511" target="\_blank">31776511</a>).

#### 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 (Center H197) Blocking peptide - Protocols

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

- [Blocking Peptides](#)

### AF9 (MLLT3) Antibody (Center H197) Blocking peptide - Images

### AF9 (MLLT3) Antibody (Center H197) Blocking peptide - 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 (Center H197) Blocking peptide - References**

lida, 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).