

AML1 Antibody (Ab-435)
Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP50013

Specification

AML1 Antibody (Ab-435) - Product Information

Application	WB
Primary Accession	O01196
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Calculated MW	49, KDa
Antigen Region	412-440

AML1 Antibody (Ab-435) - Additional Information

Gene ID 861

Other Names

Runt-related transcription factor 1, Acute myeloid leukemia 1 protein, Core-binding factor subunit alpha-2, CBF-alpha-2, Oncogene AML-1, Polyomavirus enhancer-binding protein 2 alpha B subunit, PEA2-alpha B, PEBP2-alpha B, SL3-3 enhancer factor 1 alpha B subunit, SL3/AKV core-binding factor alpha B subunit, RUNX1, AML1, CBFA2

Dilution

WB~~ 1:1000

Format

Rabbit IgG in phosphate buffered saline (without Mg²⁺ and Ca²⁺), pH 7.4, 150mM NaCl, 0.09% (W/V) sodium azide and 50% glycerol.

Storage Conditions

-20°C

AML1 Antibody (Ab-435) - Protein Information

Name RUNX1

Synonyms AML1, CBFA2

Function

Forms the heterodimeric complex core-binding factor (CBF) with CBFβ. RUNX members modulate the transcription of their target genes through recognizing the core consensus binding sequence 5'- TGTGGT-3', or very rarely, 5'-TGCGGT-3', within their regulatory regions via their runt domain, while CBFβ is a non-DNA-binding regulatory subunit that allosterically enhances the sequence-specific DNA-binding capacity of RUNX. The heterodimers bind to the core site of a number of enhancers and promoters, including murine leukemia virus, polyomavirus enhancer, T-cell receptor enhancers, LCK, IL3 and GM-CSF promoters (Probable). Essential for the

development of normal hematopoiesis (PubMed:17431401). Acts synergistically with ELF4 to transactivate the IL-3 promoter and with ELF2 to transactivate the BLK promoter (PubMed:10207087, PubMed:14970218). Inhibits KAT6B-dependent transcriptional activation (By similarity). Involved in lineage commitment of immature T cell precursors. CBF complexes repress ZBTB7B transcription factor during cytotoxic (CD8+) T cell development. They bind to RUNX-binding sequence within the ZBTB7B locus acting as transcriptional silencer and allowing for cytotoxic T cell differentiation. CBF complexes binding to the transcriptional silencer is essential for recruitment of nuclear protein complexes that catalyze epigenetic modifications to establish epigenetic ZBTB7B silencing (By similarity). Controls the energy and suppressive function of regulatory T-cells (Treg) by associating with FOXP3. Activates the expression of IL2 and IFNG and down-regulates the expression of TNFRSF18, IL2RA and CTLA4, in conventional T-cells (PubMed:17377532). Positively regulates the expression of RORC in T-helper 17 cells (By similarity).

Cellular Location

Nucleus.

Tissue Location

Expressed in all tissues examined except brain and heart. Highest levels in thymus, bone marrow and peripheral blood

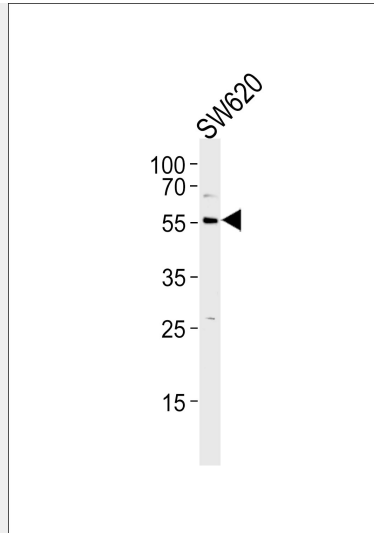
AML1 Antibody (Ab-435) - Protocols

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

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

AML1 Antibody (Ab-435) - Images





Western blot analysis of lysate from SW620 cell line, using AML1 Antibody (Ab-435) (B8287). B8287 was diluted at 1:1000. A goat anti-rabbit IgG H&L(HRP) at 1:5000 dilution was used as the secondary antibody. Lysate at 35ug.

AML1 Antibody (Ab-435) - Background

CBF binds to the core site, 5'-PYGPGGT-3', of a number of enhancers and promoters, including murine leukemia virus, polyomavirus enhancer, T-cell receptor enhancers, LCK, IL-3 and GM-CSF promoters. The alpha subunit binds DNA and appears to have a role in the development of normal hematopoiesis. Isoform AML-1L interferes with the transactivation activity of RUNX1. Acts synergistically with ELF4 to transactivate the IL-3 promoter and with ELF2 to transactivate the mouse BLK promoter. Inhibits KAT6B- dependent transcriptional activation.

AML1 Antibody (Ab-435) - References

Ahn M.-Y., et al. Submitted (SEP-1994) to the EMBL/GenBank/DDBJ databases.
Miyoshi H., et al. Proc. Natl. Acad. Sci. U.S.A. 88:10431-10434(1991).
Sacchi N., et al. Genes Chromosomes Cancer 11:226-236(1994).
Nucifora G., et al. Blood 81:2728-2734(1993).
Levanon D., et al. Genomics 23:425-432(1994).