

AP55801PU-S**Polyclonal Antibody to RUNX1 pSer435 - Aff - Purified****Alternate names:**

1, AML-1, AML1, AML1-EVI-1, AMLCR1, Acute myeloid leukemia 1 protein, CBF-alpha-2, CBFA2, Core-binding factor subunit alpha-2, EVI-1, PEA2-alpha B, PEBP2-alpha B, PEBP2A2, Polyomavirus enhancer-binding protein 2 alpha B subunit, Runt-related transcription factor, SL3-3 enhancer factor 1 alpha B subunit, SL3/AKV core-binding factor alpha B subunit

Quantity:

50 µg

Concentration:

1.0 mg/ml

Background:

CBF binds to the core site, 5'-PYGPYGGT-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 MYST4-dependent transcriptional activation.

Uniprot ID:[Q01196](#)**NCBI:**[NP_001001890.1](#)**GeneID:**[861](#)**Host:**

Rabbit

Immunogen:

Peptide sequence around phosphorylation site of Serine 435 (S-N-S(p)-P-T) derived from Human AML1 (KLH-conjugated)

Format:**State:** Liquid Ig fraction**Purification:** Affinity chromatography using epitope-specific peptide**Buffer System:** Rabbit IgG in phosphate buffered saline (without Mg²⁺ and Ca²⁺), pH 7.4, 150mM NaCl, 0.02% sodium azide and 50% glycerol**Applications:****Western blot:** 1:500~1:1000.

Other applications not tested. Optimal dilutions are dependent on conditions and should be determined by the user.

Molecular Weight:

53 kDa

Specificity:

The antibody detects endogenous levels of AML1 only when phosphorylated at serine 435.

Species Reactivity:**Tested:** Human, Mouse, Rat**Storage:**

Upon receipt, store undiluted (in aliquots) at -20°C.

Avoid repeated freezing and thawing.

Shelf life: one year from despatch.

General Readings:

1. Miyoshi H, Shimizu K, Kozu T, Maseki N, Kaneko Y, Ohki M. t(8;21) breakpoints on chromosome 21 in acute myeloid leukemia are clustered within a limited region of a single gene, AML1. Proc Natl Acad Sci U S A. 1991 Dec 1;88(23):10431-4. PubMed PMID: 1720541.

2. Sacchi N, Nisson PE, Watkins PC, Faustinella F, Wijsman J, Hagemeijer A. AML1 fusion transcripts in t(3;21) positive leukemia: evidence of molecular heterogeneity and usage of splicing sites frequently involved in the generation of normal AML1 transcripts. *Genes Chromosomes Cancer*. 1994 Dec;11(4):226-36. PubMed PMID: 7533526.

3. Nucifora G, Birn DJ, Espinosa R, Erickson P, LeBeau MM, Roulston D, et al. Involvement of the AML1 gene in the t(3;21) in therapy-related leukemia and in chronic myeloid leukemia in blast crisis. *Blood*. 1993 May 15;81(10):2728-34. PubMed PMID: 8490181.

Pictures:

Western blot analysis of extracts from 293 cells (Lane 2), HeLa cells (Lane 3) and HepG2 cells (Lane 4), using AML1 (Phospho-Ser435) Antibody AP55801PU-N. The lane on the left is treated with antigen-specific peptide.

