Product Name: **RUNX1 (phospho Ser435) Rabbit Polyclonal Antibody**
Catalog #: **APRab05398**
For research use only.

Summary

Description	Rabbit polyclonal Antibody
Host	Rabbit
Application	WB,ELISA
Reactivity	Human,Mouse,Rat
Conjugation	Unconjugated
Modification	Phosphorylated
Isotype	IgG
Clonality	Polyclonal
Form	Liquid
Concentration	1mg/ml
Storage	Aliquot and store at -20°C (valid for 12 months). Avoid freeze/thaw cycles.
Shipping	Ice bags
Buffer	Liquid in PBS containing 50% glycerol, 0.5% protective protein and 0.02% New type preservative N.
Purification	Affinity purification

Application

Dilution Ratio	WB 1:500-1:2000,ELISA 1:5000-1:20000
Molecular Weight	53kDa

Antigen Information

Gene Name	RUNX1
Alternative Names	RUNX1; AML1; CBFA2; 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
Gene ID	861.0
SwissProt ID	Q01196
Immunogen	The antiserum was produced against synthesized peptide derived from human AML1 around the phosphorylation site of Ser435. AA range:401-450

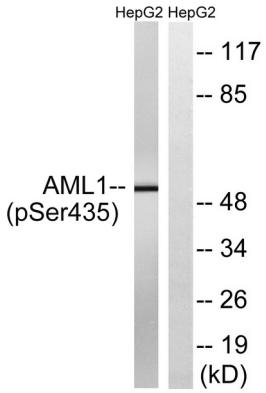
Background

Core binding factor (CBF) is a heterodimeric transcription factor that binds to the core element of many enhancers and promoters. The protein encoded by this gene represents the alpha subunit of CBF and is thought to be involved in the development of normal hematopoiesis. Chromosomal translocations involving this gene are well-documented and have been associated with several types of leukemia. Three transcript variants encoding different isoforms have been found for this gene. [provided by RefSeq, Jul 2008],alternative products:Additional isoforms seem to exist,caution:The fusion of AML1 with EAP in T-MDS induces a change of reading frame in the latter resulting in 17 AA unrelated to those of EAP.,disease:A chromosomal aberration involving RUNX1/AML1 is a cause of chronic myelogenous leukemia (CML). Translocation t(3;21)(q26;q22) with EAP, MSD1 or EVI1.,disease:A chromosomal aberration involving RUNX1/AML1 is a cause of chronic myelomonocytic leukemia. Inversion inv(21)(q21;q22) with USP16.,disease:A chromosomal aberration involving RUNX1/AML1 is a cause of M2 type acute myeloid leukemia (AML-M2). Translocation t(8;21)(q22;q22) with RUNX1T1/MTG8/ETO.,disease:A chromosomal aberration involving RUNX1/AML1 is a cause of therapy-related myelodysplastic syndrome (T-MDS). Translocation t(3;21)(q26;q22) with EAP, MSD1 or EVI1.,disease:A chromosomal aberration involving RUNX1/AML1 is found in childhood acute lymphoblastic leukemia (ALL). Translocation t(12;21)(p13;q22) with TEL. The translocation fuses the 3'-end of TEL to the alternate 5'-exon of AML-1H.,disease:A chromosomal aberration involving RUNX1/AML1 is found in therapy-related myeloid malignancies. Translocation t(16;21)(q24;q22) that forms a RUNX1-CBFA2T3 fusion protein.,disease:Defects in RUNX1 are the cause of familial platelet disorder with associated myeloid malignancy (FPDMM) [MIM:601399]. FPDMM is an autosomal dominant disease characterized by qualitative and quantitative platelet defects, and propensity to develop acute myelogenous leukemia.,domain:A proline/serine/threonine rich region at the C-terminus is necessary for transcriptional activation of target genes.,function: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.,PTM:Methylated.,PTM:Phosphorylated in its C-terminus upon IL-6 treatment. Phosphorylation enhances interaction with MYST3.,similarity:Contains 1 Runt domain.,subunit:Heterodimer with CBFB. RUNX1 binds DNA as a monomer and through the Runt domain. DNA-binding is increased by heterodimerization. Isoform AML-1L can neither bind DNA nor heterodimerize. Interacts with TLE1 and THOC4. Interacts with ELF1, ELF2 and SPI1. Interacts via its Runt domain with the ELF4 N-terminal region. Interaction with ELF2 isoform 2 (NERF-1a) may act to repress RUNX1-mediated transactivation. Interacts with MYST3 and MYST4. Interacts with SUV39H1, leading to abrogate the transactivating and DNA-binding properties of RUNX1.,tissue specificity:Expressed in all tissues examined except brain and heart. Highest levels in thymus, bone marrow and peripheral blood.,

Research Area

Pathways in cancer;Chronic myeloid leukemia;Acute myeloid leukemia;

Image Data



Western blot analysis of lysates from HepG2 cells treated with PMA 125ng/ml 30 ', using AML1 (Phospho-Ser435) Antibody. The lane on the right is blocked with the phospho peptide.