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Product datasheet

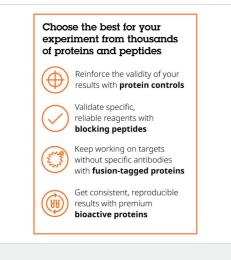
RUNX1 / AML1 peptide ab177141

1 Image

Description	
Product name	RUNX1 / AML1 peptide
Animal free	No
Nature	Synthetic
Specifications	
Our Abpromise guarantee co	vers the use of ab177141 in the following tested applications.
The application notes include re	ecommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.
Applications	Blocking - Blocking peptide for Anti-RUNX1 / AML1 + RUNX3 + RUNX2 antibody [EPR3099] (<u>ab92336</u>)
Form	Liquid
Additional notes	 First try to dissolve a small amount of peptide in either water or buffer. The more charged residues on a peptide, the more soluble it is in aqueous solutions. If the peptide doesn't dissolve try an organic solvent e.g. DMSO, then dilute using water or buffer. Consider that any solvent used must be compatible with your assay. If a peptide does not dissolve and you need to recover it, lyophilise to remove the solvent. Gentle warming and sonication can effectively aid peptide solubilisation. If the solution is cloudy or has gelled the peptide may be in suspension rather than solubilised. Peptides containing cysteine are easily oxidised, so should be prepared in solution just prior to use.
Preparation and Storage	
Stability and Storage	Shipped at 4°C. Store at -20°C.
General Info	
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.
Tissue specificity	Expressed in all tissues examined except brain and heart. Highest levels in thymus, bone marrow and peripheral blood.
Involvement in disease	Note=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. Note=A chromosomal aberration involving RUNX1/AML1 is a cause of therapy-related myelodysplastic syndrome (T-MDS). Translocation t(3;21)(q26;q22) with EAP or MECOM. Note=A chromosomal aberration involving RUNX1/AML1 is a cause of chronic myelogenous leukemia (CML). Translocation t(3;21)(q26;q22) with EAP or MECOM. Note=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. Note=A chromosomal aberration involving RUNX1 is found in acute leukemia. Translocation t(11,21)(q13;q22) that forms a MACROD1-RUNX1 fusion protein. 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. Note=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. Note=A chromosomal aberration involving RUNX1/AML1 is a cause of chronic myelomonocytic leukemia. Inversion inv(21)(q21;q22) with USP16.
Sequence similarities	Contains 1 Runt domain.
Domain	A proline/serine/threonine rich region at the C-terminus is necessary for transcriptional activation of target genes.
Post-translational modifications	Phosphorylated in its C-terminus upon IL-6 treatment. Phosphorylation enhances interaction with MYST3. Methylated.
Cellular localization	Nucleus.

Images



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RUNX1 / AML1 peptide (ab177141)

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