abcam

Product datasheet

RUNX1 / AML1 peptide ab177141

1 Image

Description

Product name	RUNX1 / AML1 peptide
Animal free	No
Nature	Synthetic

Specifications

Our **Abpromise guarantee** covers the use of **ab177141** in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Applications	Blocking - Blocking peptide for Anti-RUNX1 / AML1 + RUNX3 + RUNX2 antibody [EPR3099] (**ab92336**)
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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