

RUNX1 Blocking Peptide (C-term)

Synthetic peptide Catalog # BP21105a

Specification

RUNX1 Blocking Peptide (C-term) - Product Information

Primary Accession Other Accession O6PF39

RUNX1 Blocking Peptide (C-term) - 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

Target/Specificity

The synthetic peptide sequence is selected from aa 421-435 of HUMAN RUNX1

Format

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

RUNX1 Blocking Peptide (C-term) - Protein Information

Name RUNX1

Synonyms AML1, CBFA2

Function

Forms the heterodimeric complex core-binding factor (CBF) with CBFB. 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 CBFB 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, PubMed:14970218, PubMed:14970218 (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 anergy 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

RUNX1 Blocking Peptide (C-term) - Protocols

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

Blocking Peptides

RUNX1 Blocking Peptide (C-term) - Images

RUNX1 Blocking Peptide (C-term) - 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 KAT6B- dependent transcriptional activation.

RUNX1 Blocking Peptide (C-term) - 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).