

## ELF4 Antibody (N-term) Blocking peptide

Synthetic peptide Catalog # BP12761a

### **Specification**

## ELF4 Antibody (N-term) Blocking peptide - Product Information

**Primary Accession** 

Q99607

# ELF4 Antibody (N-term) Blocking peptide - Additional Information

Gene ID 2000

#### **Other Names**

ETS-related transcription factor Elf-4, E74-like factor 4, Myeloid Elf-1-like factor, ELF4 {ECO:0000312|EMBL:CAI428821}

#### **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.

### ELF4 Antibody (N-term) Blocking peptide - Protein Information

Name ELF4 {ECO:0000312|EMBL:CAI42882.1}

#### **Function**

Transcriptional activator that binds to DNA sequences containing the consensus 5'-WGGA-3'. Transactivates promoters of the hematopoietic growth factor genes CSF2, IL3, IL8, and of the bovine lysozyme gene. Acts synergistically with RUNX1 to transactivate the IL3 promoter (By similarity). Transactivates the PRF1 promoter in natural killer (NK) cells and CD8+ T cells (PubMed:<a href="http://www.uniprot.org/citations/34326534" target=" blank">34326534</a>). Plays a role in the development and function of NK and NK T-cells and in innate immunity. Controls the proliferation and homing of CD8+ T-cells via the Kruppel-like factors KLF4 and KLF2 (By similarity). Controls cell senescence in a p53-dependent manner. Can also promote cellular transformation through inhibition of the p16 pathway. Is a transcriptional regulator of inflammation, controlling T-helper 17 (Th17) cells and macrophage inflammatory responses. Required for sustained transcription of anti-inflammatory genes, including IL1RN (PubMed: <a href="http://www.uniprot.org/citations/34326534" target=" blank">34326534</a>, PubMed:<a href="http://www.uniprot.org/citations/35266071" target="blank">35266071</a>). Is a negative regulator of pro- inflammatory cytokines expression including IL17A, IL1B, IL6, TNFA and CXCL1  $(PubMed: <a href="http://www.uniprot.org/citations/34326534" target="\_blank">34326534</a>, PubMed: <a href="http://www.uniprot.org/citations/35266071" target="\_blank">35266071</a>).$ Down-regulates expression of TREM1, a cell surface receptor involved in the amplification of



inflammatory responses (By similarity) (PubMed:<a

href="http://www.uniprot.org/citations/34326534" target="\_blank">34326534</a>, PubMed:<a href="http://www.uniprot.org/citations/35266071" target="\_blank">35266071</a>).

#### **Cellular Location**

Nucleus, PML body. Note=Accumulation into PML nuclear bodies is mediated by PML

#### **Tissue Location**

Abundantly expressed in the placenta and in a variety of myeloid leukemia cell lines. Moderate levels of expression in heart, lung, spleen, thymus, peripheral blood lymphocytes, ovary and colon. Lower levels of expression in Jurkat T-cells and other T-cell lines and no expression in brain.

# ELF4 Antibody (N-term) Blocking peptide - Protocols

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

#### Blocking Peptides

ELF4 Antibody (N-term) Blocking peptide - Images

## ELF4 Antibody (N-term) Blocking peptide - Background

The protein encoded by this gene is a transcriptionalactivator that binds and activates the promoters of the CSF2, IL3,IL8, and PRF1 genes. The encoded protein is involved in naturalkiller cell development and function, innate immunity, and induction of cell cycle arrest in naive CD8+ cells. Two transcriptvariants encoding the same protein have been found for this gene.

### ELF4 Antibody (N-term) Blocking peptide - References

Yamada, T., et al. Nat. Immunol. 10(6):618-626(2009)Sugiyama, N., et al. Mol. Cell Proteomics 6(6):1103-1109(2007)Moore, S.D., et al. Leuk. Res. 30(8):1037-1042(2006)Liu, Y., et al. Mol. Cell. Biol. 26(8):3114-3123(2006)Koga, T., et al. FEBS Lett. 579(13):2811-2816(2005)