

Bi-Phospho-ALK(Y1282/1283) Blocking Peptide Synthetic peptide Catalog # BP3756a

## Specification

# Bi-Phospho-ALK(Y1282/1283) Blocking Peptide - Product Information

Primary Accession Other Accession

#### <u>O9UM73</u> P97793

## **Bi-Phospho-ALK(Y1282/1283) Blocking Peptide - Additional Information**

Gene ID 238

**Other Names** ALK tyrosine kinase receptor, Anaplastic lymphoma kinase, CD246, ALK

Target/Specificity

The synthetic peptide sequence is selected from aa 1274-1287 of HUMAN ALK

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.

# **Bi-Phospho-ALK(Y1282/1283) Blocking Peptide - Protein Information**

Name ALK {ECO:0000303|PubMed:9174053, ECO:0000312|HGNC:HGNC:427}

Function

Neuronal receptor tyrosine kinase that is essentially and transiently expressed in specific regions of the central and peripheral nervous systems and plays an important role in the genesis and differentiation of the nervous system (PubMed:<a

href="http://www.uniprot.org/citations/11121404" target="\_blank">11121404</a>, PubMed:<a href="http://www.uniprot.org/citations/11387242" target="\_blank">11387242</a>, PubMed:<a href="http://www.uniprot.org/citations/16317043" target="\_blank">16317043</a>, PubMed:<a href="http://www.uniprot.org/citations/16317043" target="\_blank">16317043</a>, PubMed:<a href="http://www.uniprot.org/citations/17274988" target="\_blank">10611385</a>, PubMed:<a href="http://www.uniprot.org/citations/30061385" target="\_blank">30061385</a>, PubMed:<a href="http://www.uniprot.org/citations/30061385" target="\_blank">34646012</a>, PubMed:<a href="http://www.uniprot.org/citations/34646012" target="\_blank">34646012</a>, PubMed:<a href="http://www.uniprot.org/citations/34646012" target="\_blank">34646012</a>, PubMed:<a href="http://www.uniprot.org/citations/34646012" target="\_blank">34646012</a>, PubMed:<a href="http://www.uniprot.org/citations/34646012" target="\_blank">34646012</a>, PubMed:<a href="http://www.uniprot.org/citations/34819673" target="\_blank">34819673</a>, Also acts as a key thinness protein involved in the resistance to weight gain: in hypothalamic neurons, controls energy expenditure acting as a negative regulator of white adipose tissue lipolysis and sympathetic tone to fine-tune energy homeostasis (By similarity). Following activation by ALKAL2 ligand at the cell surface, transduces an extracellular signal into an intracellular response



(PubMed:<a href="http://www.uniprot.org/citations/30061385" target=" blank">30061385</a>, PubMed:<a href="http://www.uniprot.org/citations/33411331" target=" blank">33411331</a>, PubMed:<a href="http://www.uniprot.org/citations/34646012" target="\_blank">34646012</a>, PubMed:<a href="http://www.uniprot.org/citations/34819673" target="\_blank">34819673</a>). In contrast, ALKAL1 is not a potent physiological ligand for ALK (PubMed:<a href="http://www.uniprot.org/citations/34646012" target=" blank">34646012</a>). Ligand-binding to the extracellular domain induces tyrosine kinase activation, leading to activation of the mitogen-activated protein kinase (MAPK) pathway (PubMed: <a href="http://www.uniprot.org/citations/34819673" target=" blank">34819673</a>). Phosphorylates almost exclusively at the first tyrosine of the Y-x-x-Y-Y motif (PubMed:<a href="http://www.uniprot.org/citations/15226403" target=" blank">15226403</a>, PubMed:<a href="http://www.uniprot.org/citations/16878150" target=" blank">16878150</a>). Induces tyrosine phosphorylation of CBL, FRS2, IRS1 and SHC1, as well as of the MAP kinases MAPK1/ERK2 and MAPK3/ERK1 (PubMed:<a href="http://www.uniprot.org/citations/15226403" target=" blank">15226403</a>, PubMed:<a href="http://www.uniprot.org/citations/16878150" target=" blank">16878150</a>). ALK activation may also be regulated by pleiotrophin (PTN) and midkine (MDK) (PubMed:<a href="http://www.uniprot.org/citations/11278720" target="\_blank">11278720</a>, PubMed:<a href="http://www.uniprot.org/citations/11809760" target=" blank">11809760</a>, PubMed:<a href="http://www.uniprot.org/citations/12107166" target=" blank">12107166</a>, PubMed:<a href="http://www.uniprot.org/citations/12122009" target=" blank">12122009</a>). PTN-binding induces MAPK pathway activation, which is important for the anti-apoptotic signaling of PTN and regulation of cell proliferation (PubMed:<a href="http://www.uniprot.org/citations/11278720" target=" blank">11278720</a>, PubMed:<a href="http://www.uniprot.org/citations/11809760" target="\_blank">11809760</a>, PubMed:<a href="http://www.uniprot.org/citations/12107166" target="\_blank">12107166</a>, DubMed:<a induces phosphorylation of the ALK target insulin receptor substrate (IRS1), activates mitogen-activated protein kinases (MAPKs) and PI3-kinase, resulting also in cell proliferation induction (PubMed:<a href="http://www.uniprot.org/citations/12122009" target=" blank">12122009</a>). Drives NF-kappa-B activation, probably through IRS1 and the activation of the AKT serine/threonine kinase (PubMed:<a href="http://www.uniprot.org/citations/15226403" target=" blank">15226403</a>, PubMed:<a href="http://www.uniprot.org/citations/16878150" target=" blank">16878150</a>). Recruitment of IRS1 to activated ALK and the activation of NF-kappa-B are essential for the autocrine growth and survival signaling of MDK (PubMed: <a href="http://www.uniprot.org/citations/15226403" target=" blank">15226403</a>, PubMed:<a href="http://www.uniprot.org/citations/16878150" target=" blank">16878150</a>).

## **Cellular Location**

Cell membrane; Single-pass type I membrane protein Note=Membrane attachment is essential for promotion of neuron-like differentiation and cell proliferation arrest through specific activation of the MAP kinase pathway.

#### **Tissue Location**

Expressed in brain and CNS. Also expressed in the small intestine and testis, but not in normal lymphoid cells

## **Bi-Phospho-ALK(Y1282/1283) Blocking Peptide - Protocols**

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

### <u>Blocking Peptides</u>

# Bi-Phospho-ALK(Y1282/1283) Blocking Peptide - Images

# Bi-Phospho-ALK(Y1282/1283) Blocking Peptide - Background

The 2;5 chromosomal translocation is frequently associated



with anaplastic large cell lymphomas (ALCLs). The translocation creates a fusion gene consisting of the ALK (anaplastic lymphoma kinase) gene and the nucleophosmin (NPM) gene: the 3' half of ALK, derived from chromosome 2, is fused to the 5' portion of NPM from chromosome 5. A recent study shows that the product of the NPM-ALK fusion gene is oncogenic. The deduced amino acid sequences reveal that ALK is a novel receptor protein-tyrosine kinase having a putative transmembrane domain and an extracellular domain. These sequences are absent in the product of the transforming NPM-ALK gene. ALK shows the greatest sequence similarity to LTK (leukocyte tyrosine kinase). ALK plays an important role in the development of the brain and exerts its effects on specific neurons in the nervous system.

## **Bi-Phospho-ALK(Y1282/1283) Blocking Peptide - References**

Ardini, E., et al. Cancer Lett. 299(2):81-94(2010) Ohira, M., et al. Cancer Sci. 101(11):2295-2301(2010) Merkel, O., et al. Proc. Natl. Acad. Sci. U.S.A. 107(37):16228-16233(2010) De Brouwer, S., et al. Clin. Cancer Res. 16(17):4353-4362(2010) Bossi, R.T., et al. Biochemistry 49(32):6813-6825(2010)