

## Phospho-ALK(Y1604) Blocking Peptide

Synthetic peptide Catalog # BP3760a

## **Specification**

## Phospho-ALK(Y1604) Blocking Peptide - Product Information

Primary Accession Q9UM73
Other Accession NP 004295.2

# Phospho-ALK(Y1604) 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 1599-1612 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.

## Phospho-ALK(Y1604) 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/17274988" target="\_blank">17274988</a>, PubMed:<a href="http://www.uniprot.org/citations/30061385" target="\_blank">30061385</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>, PubMed:<a



(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>, 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>). MDK-binding 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

## Phospho-ALK(Y1604) Blocking Peptide - Protocols

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

## • Blocking Peptides

Phospho-ALK(Y1604) Blocking Peptide - Images

# Phospho-ALK(Y1604) Blocking Peptide - Background

The 2;5 chromosomal translocation is frequently associated with anaplastic large cell lymphomas





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(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.

# Phospho-ALK(Y1604) 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)