

**ALK Antibody (Center) Blocking Peptide**  
**Synthetic peptide**  
**Catalog # BP7600c****Specification**

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**ALK Antibody (Center) Blocking Peptide - Product Information**Primary Accession [Q9UM73](#)**ALK Antibody (Center) Blocking Peptide - Additional Information****Gene ID** 238**Other Names**

ALK tyrosine kinase receptor, Anaplastic lymphoma kinase, CD246, ALK

**Target/Specificity**

The synthetic peptide sequence used to generate the antibody [AP7600c](/products/AP7600c) was selected from the Center region of human ALK. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

**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.

**ALK Antibody (Center) 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:[11121404](http://www.uniprot.org/citations/11121404), PubMed:[11387242](http://www.uniprot.org/citations/11387242), PubMed:[16317043](http://www.uniprot.org/citations/16317043), PubMed:[17274988](http://www.uniprot.org/citations/17274988), PubMed:[30061385](http://www.uniprot.org/citations/30061385), PubMed:[34646012](http://www.uniprot.org/citations/34646012), PubMed:[34819673](http://www.uniprot.org/citations/34819673)). 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: [30061385](http://www.uniprot.org/citations/30061385), PubMed: [33411331](http://www.uniprot.org/citations/33411331), PubMed: [34646012](http://www.uniprot.org/citations/34646012), PubMed: [34819673](http://www.uniprot.org/citations/34819673)). In contrast, ALKAL1 is not a potent physiological ligand for ALK (PubMed: [34646012](http://www.uniprot.org/citations/34646012)). Ligand-binding to the extracellular domain induces tyrosine kinase activation, leading to activation of the mitogen-activated protein kinase (MAPK) pathway (PubMed: [34819673](http://www.uniprot.org/citations/34819673)). Phosphorylates almost exclusively at the first tyrosine of the Y-x-x-x-Y-Y motif (PubMed: [15226403](http://www.uniprot.org/citations/15226403), PubMed: [16878150](http://www.uniprot.org/citations/16878150)). Induces tyrosine phosphorylation of CBL, FRS2, IRS1 and SHC1, as well as of the MAP kinases MAPK1/ERK2 and MAPK3/ERK1 (PubMed: [15226403](http://www.uniprot.org/citations/15226403), PubMed: [16878150](http://www.uniprot.org/citations/16878150)). ALK activation may also be regulated by pleiotrophin (PTN) and midkine (MDK) (PubMed: [11278720](http://www.uniprot.org/citations/11278720), PubMed: [11809760](http://www.uniprot.org/citations/11809760), PubMed: [12107166](http://www.uniprot.org/citations/12107166), PubMed: [12122009](http://www.uniprot.org/citations/12122009)). PTN-binding induces MAPK pathway activation, which is important for the anti-apoptotic signaling of PTN and regulation of cell proliferation (PubMed: [11278720](http://www.uniprot.org/citations/11278720), PubMed: [11809760](http://www.uniprot.org/citations/11809760), PubMed: [12107166](http://www.uniprot.org/citations/12107166)). 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: [12122009](http://www.uniprot.org/citations/12122009)). Drives NF-kappa-B activation, probably through IRS1 and the activation of the AKT serine/threonine kinase (PubMed: [15226403](http://www.uniprot.org/citations/15226403), PubMed: [16878150](http://www.uniprot.org/citations/16878150)). 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: [15226403](http://www.uniprot.org/citations/15226403), PubMed: [16878150](http://www.uniprot.org/citations/16878150)).

### 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

## ALK Antibody (Center) Blocking Peptide - Protocols

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

- [Blocking Peptides](#)

## ALK Antibody (Center) Blocking Peptide - Images

## ALK Antibody (Center) Blocking Peptide - Background

ALK, a member of the insulin receptor subfamily of Tyr protein kinases, is an orphan receptor. It appears to play an important role in the normal development and function of the nervous system. This Type I membrane protein is expressed in brain and CNS and in the small intestine and testis, but not in normal lymphoid cells. A form of non-Hodgkin's lymphoma is characterized by a chromosomal translocation t(2;5)(p23;q35) that involves NPM1 and ALK. The protein contains 1 LDL-receptor class A domain and 2 putative MAM domains.

#### **ALK Antibody (Center) Blocking Peptide - References**

Morris, S.W., et al., Oncogene 14(18):2175-2188 (1997).Iwahara, T., et al., Oncogene 14(4):439-449 (1997).Morris, S.W., et al., Science 263(5151):1281-1284 (1994).Morris, S.W., et al., Oncogene 15, 2883-2883 (1997).