10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

ALK Blocking Peptide (C-term)

Synthetic peptide
Catalog # BP22071b

Specification

ALK Blocking Peptide (C-term) - Product Information

Primary Accession Q9UM73

ALK Blocking Peptide (C-term) - Additional Information

Gene ID 238

Other Names
ALK tyrosine kinase receptor, 2.7.10.1, Anaplastic lymphoma kinase, CD246, ALK

Target/Specificity
The synthetic peptide sequence is selected from aa 1513-1527 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.

ALK Blocking Peptide (C-term) - 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, PubMed:11387242, PubMed:16317043, PubMed:17274988, PubMed:30061385, PubMed:34646012, PubMed: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,

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

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

- Blocking Peptides

ALK Blocking Peptide (C-term) - Images

ALK Blocking Peptide (C-term) - Background

Neuronal orphan 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. Transduces signals from ligands at the cell surface, through specific activation of the mitogen-activated protein kinase (MAPK) pathway. Phosphorylates almost exclusively at the first tyrosine of the Y-x-x-x-Y-Y motif. Following activation by ligand, ALK induces tyrosine phosphorylation of CBL, FRS2, IRS1 and SHC1, as well as of the MAP kinases MAPK1/ERK2 and MAPK3/ERK1. Acts as a receptor for ligands pleiotrophin (PTN), a secreted growth factor, and midkine (MDK), a PTN-related factor, thus participating in PTN and MDK signal transduction. PTN-binding induces MAPK pathway activation, which is important for the anti-apoptotic signaling of PTN and regulation of cell proliferation. 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. Drives NF-kappa-B activation, probably through IRS1 and the activation of the AKT serine/threonine kinase. Recruitment of IRS1 to activated ALK and the activation of NF-kappa-B are essential for the autocrine growth and survival signaling of MDK.

ALK Blocking Peptide (C-term) - References

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