

ALK Antibody (C-term)
Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP22071b**Specification**

ALK Antibody (C-term) - Product Information

Application	WB,E
Primary Accession	O9UM73
Reactivity	Human
Host	Rabbit
Clonality	polyclonal
Isotype	Rabbit IgG
Calculated MW	176442

ALK Antibody (C-term) - Additional Information**Gene ID** 238**Other Names**

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

Target/Specificity

This ALK antibody is generated from a rabbit immunized with a KLH conjugated synthetic peptide between 1493-1527 amino acids from the C-terminal region of human ALK.

Dilution

WB~~1:2000

E~~Use at an assay dependent concentration.

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

Storage

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

ALK Antibody (C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

ALK Antibody (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

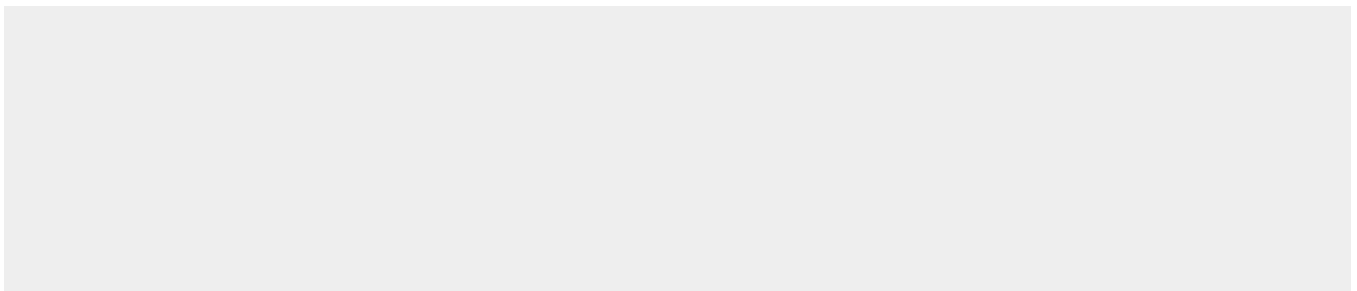
Expressed in brain and CNS. Also expressed in the small intestine and testis, but not in normal lymphoid cells

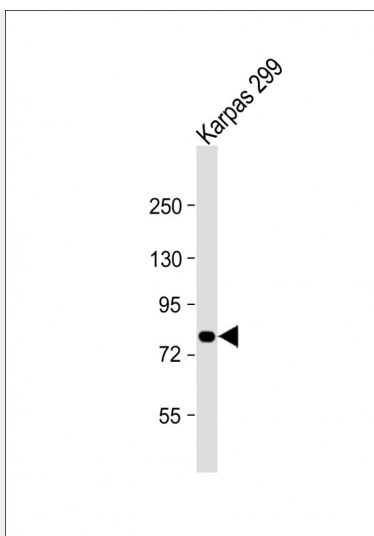
ALK Antibody (C-term) - Protocols

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

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

ALK Antibody (C-term) - Images





Anti-ALK Antibody (C-term) at 1:2000 dilution + Karpas 299 whole cell lysate Lysates/proteins at 20 µg per lane. Secondary Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size : 176 kDa Blocking/Dilution buffer: 5% NFDM/TBST.

ALK Antibody (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 Antibody (C-term) - References

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 Iwahara T.,et al.Oncogene 14:439-449(1997).
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 Hillier L.W.,et al.Nature 434:724-731(2005).