

Anti-ALK antibody

Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP22467a

Product Information

Application	WB, E
Primary Accession	Q9UM73
Reactivity	Human
Host	Rabbit
Clonality	polyclonal
Isotype	Rabbit Ig
Clone Names	R03322
Calculated MW	176442

Additional Information

Gene ID	238
Other Names	ALK tyrosine kinase receptor, 2.7.10.1, Anaplastic lymphoma kinase, CD246, ALK {ECO:0000303 PubMed:9174053, ECO:0000312 HGNC:HGNC:427}
Target/Specificity	This antibody is generated from a rabbit immunized with a KLH conjugated synthetic peptide between amino acids from human.
Dilution	WB~~1:1000 E~~Use at an assay dependent concentration.
Format	Purified polyclonal antibody supplied in PBS with 0.05% (V/V) Proclin 300. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.
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	Anti-ALK antibody is for research use only and not for use in diagnostic or therapeutic procedures.

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](#)). May function as regulator of gastric epithelial differentiation (By similarity).

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

Background

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](#)).

References

Morris S.W., et al. *Oncogene* 14:2175-2188(1997).

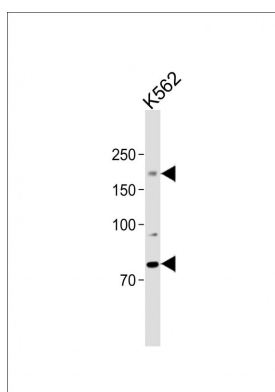
Morris S.W., et al. *Oncogene* 15:2883-2883(1997).

Iwahara T., et al. *Oncogene* 14:439-449(1997).

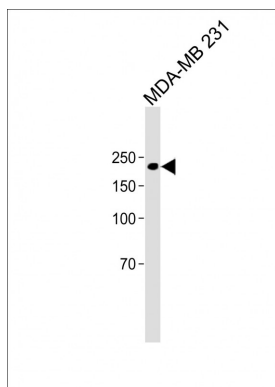
Totoki Y., et al. Submitted (MAR-2005) to the EMBL/GenBank/DBJ databases.

Hillier L.W., et al. *Nature* 434:724-731(2005).

Images



All lanes: Anti-Anti-ALK antibody at 1:1000 dilution + K562 whole cell lysate Lysates/proteins at 20 µg per lane. Secondary: Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated (ASP1615) at 1/15000 dilution. Observed band size: 220, 80 KDa Blocking/Dilution buffer: 5% NFDM/TBST.



All lanes: Anti-Anti-ALK antibody at 1:1000 dilution + MDA-MB 231 whole cell lysate Lysates/proteins at 20 µg per lane. Secondary: Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated (ASP1615) at 1/15000 dilution. Observed band size: 220 KDa Blocking/Dilution buffer: 5% NFDM/TBST.

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.