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Anti-ALK Antibody
Catalog # ABO11049

Specification

Anti-ALK Antibody - Product Information

Application	**WB, IHC-P**
Primary Accession	Q9UM73
Host	**Rabbit**
Reactivity	**Human, Mouse, Rat**
Clonality	**Polyclonal**
Format	**Lyophilized**

Description
Rabbit IgG polyclonal antibody for ALK tyrosine kinase receptor(ALK) detection. Tested with WB, IHC-P in Human;Mouse;Rat.

Reconstitution
Add 0.2ml of distilled water will yield a concentration of 500ug/ml.

Anti-ALK Antibody - Additional Information

Gene ID 238

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

Calculated MW
176442 MW KDa

Application Details
Immunohistochemistry(Paraffin-embedded Section), 0.5-1 µg/ml, Human, Rat, Mouse, By Heat
Western blot, 0.1-0.5 µg/ml, Human, Rat, Mouse

Subcellular Localization
Cell membrane ; Single-pass type I membrane protein . Membrane attachment was crucial for promotion of neuron-like differentiation and cell proliferation arrest through specific activation of the MAP kinase pathway.

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

Protein Name
ALK tyrosine kinase receptor

Contents
Each vial contains 5mg BSA, 0.9mg NaCl, 0.2mg Na2HPO4, 0.05mg Thimerosal, 0.05mg NaN3.

Immunogen
A synthetic peptide corresponding to a sequence at the C-terminus of human ALK(1571-1589aa

LFRLRHFPCGNVNYGYQQQ), identical to the related rat and mouse sequences.

Purification
Immunogen affinity purified.

Cross Reactivity
No cross reactivity with other proteins

Storage **At -20˚C for one year. After r˚Constitution, at 4˚C for one month. It˚Can also be aliquotted and stored frozen at -20˚C for a longer time.Avoid repeated freezing and thawing.**

Sequence Similarities
Belongs to the protein kinase superfamily. Tyr protein kinase family. Insulin receptor subfamily.

Anti-ALK Antibody - 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:<a href="http://www.uniprot.org/citations/12122009"

target="_blank">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

Anti-ALK Antibody - Protocols

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

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

Anti-ALK Antibody - Images

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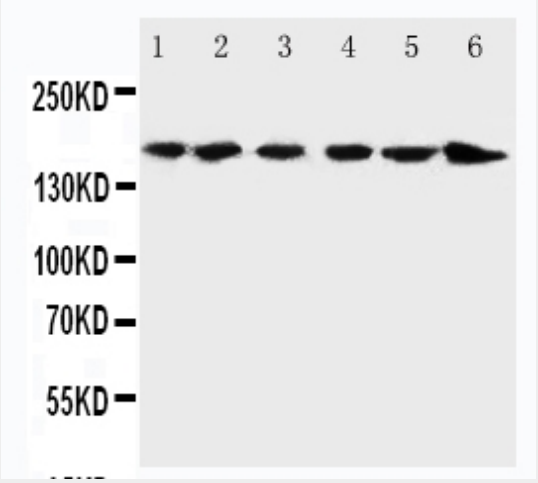
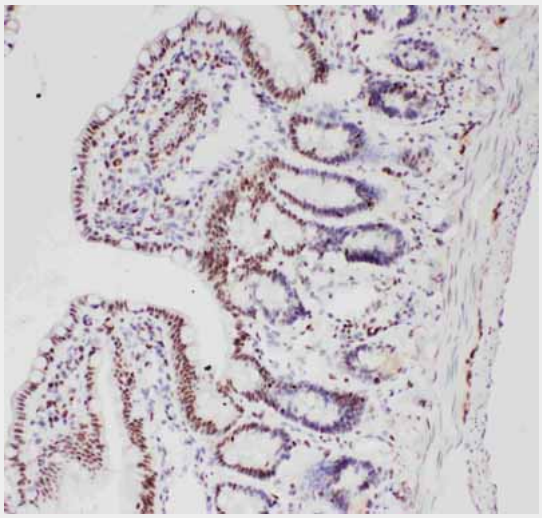
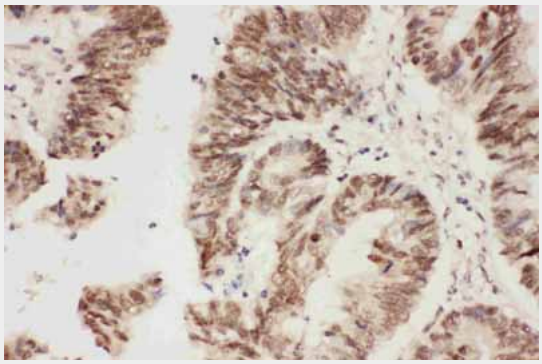


Anti-ALK antibody, ABO11049, Western blottingLane 1: Rat Brain Tissue LysateLane 2: Rat Testis Tissue LysateLane 3: U87 Cell LysateLane 4: HELA Cell LysateLane 5: COLO320 Cell LysateLane 6: JURKAT Cell Lysate



Anti-ALK antibody, ABO11049, IHC(P)IHC(P): Rat Intestine Tissue



Anti-ALK antibody, ABO11049, IHC(P)IHC(P): Human Rectal Cancer Tissue

Anti-ALK Antibody - Background

ALK(Anaplastic lymphoma kinase) also known as ALK tyrosine kinase receptor or CD246(cluster of differentiation 246) is an enzyme that in humans is encoded by the ALK gene. The ALK gene is mapped on 2p23.2-p23.1. Expressed in the small intestine, testis, and brain but not in normal lymphoid cells, ALK shows greatest sequence similarity to the insulin receptor subfamily of kinases.

ALK plays an important role in the development of the brain and exerts its effects on specific neurons in the nervous system. The deduced amino acid sequences reveal that ALK is a novel receptor tyrosine kinase having a putative transmembrane domain and an extracellular domain. In Drosophila, localized Jeb activates Alk and the downstream Ras/mitogen-activated protein kinase cascade to specify a select group of visceral muscle precursors as muscle-patterning pioneers. Functional RNA interference screening on a set of these transcriptional targets revealed that CEBPB and BCL2A1 were absolutely necessary to induce cell transformation and/or to sustain growth and survival of ALK-positive ALCL cells. One particularly informative case presented a high-level gene amplification that was strictly limited to ALK, indicating that this gene may contribute on its own to neuroblastoma development. Mutated ALK proteins were overexpressed, hyperphosphorylated, and showed constitutive kinase activity. The knockdown of ALK expression in ALK-mutated cells, but also in cell lines overexpressing a wildtype ALK, led to a marked decrease of cell proliferation.