

**Anti-ALK (Anaplastic Lymphoma Kinase) / CD246 Antibody**  
**Mouse Monoclonal Antibody**  
**Catalog # AH13245****Specification**

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**Anti-ALK (Anaplastic Lymphoma Kinase) / CD246 Antibody - Product Information**

|                   |                        |
|-------------------|------------------------|
| Application       | ,1,3,4,                |
| Primary Accession | <a href="#">O9UM73</a> |
| Other Accession   | <a href="#">654469</a> |
| Reactivity        | Human                  |
| Host              | Mouse                  |
| Clonality         | Monoclonal             |
| Isotype           | Mouse / IgG1, kappa    |
| Calculated MW     | 176442                 |

**Anti-ALK (Anaplastic Lymphoma Kinase) / CD246 Antibody - Additional Information****Gene ID** 238**Other Names**

ALK Tyrosine Kinase Receptor, ALK/NPM1 fusion gene, Anaplastic lymphoma kinase Ki1, Anaplastic Lymphoma Kinase p80, anaplastic lymphoma receptor tyrosine kinase, CD246, mutant anaplastic lymphoma kinase, NBLST3, Tcrz, TFG/ALK

**Format**

200ug/ml of Ab purified from Bioreactor Concentrate by Protein A/G. Prepared in 10mM PBS with 0.05% BSA & 0.05% azide. Also available WITHOUT BSA & azide at 1.0mg/ml.

**Storage**

Store at 2 to 8°C. Antibody is stable for 24 months.

**Precautions**

Anti-ALK (Anaplastic Lymphoma Kinase) / CD246 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

**Anti-ALK (Anaplastic Lymphoma Kinase) / CD246 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:<a href="http://www.uniprot.org/citations/11121404" target="\_blank">11121404</a>, PubMed:<a href="http://www.uniprot.org/citations/11387242" target="\_blank">11387242</a>, PubMed:<a href="http://www.uniprot.org/citations/16317043" target="\_blank">16317043</a>, PubMed:<a href="http://www.uniprot.org/citations/17274988" target="\_blank">17274988</a>, PubMed:<a href="http://www.uniprot.org/citations/30061385" target="\_blank">30061385</a>, PubMed:<a

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

### 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

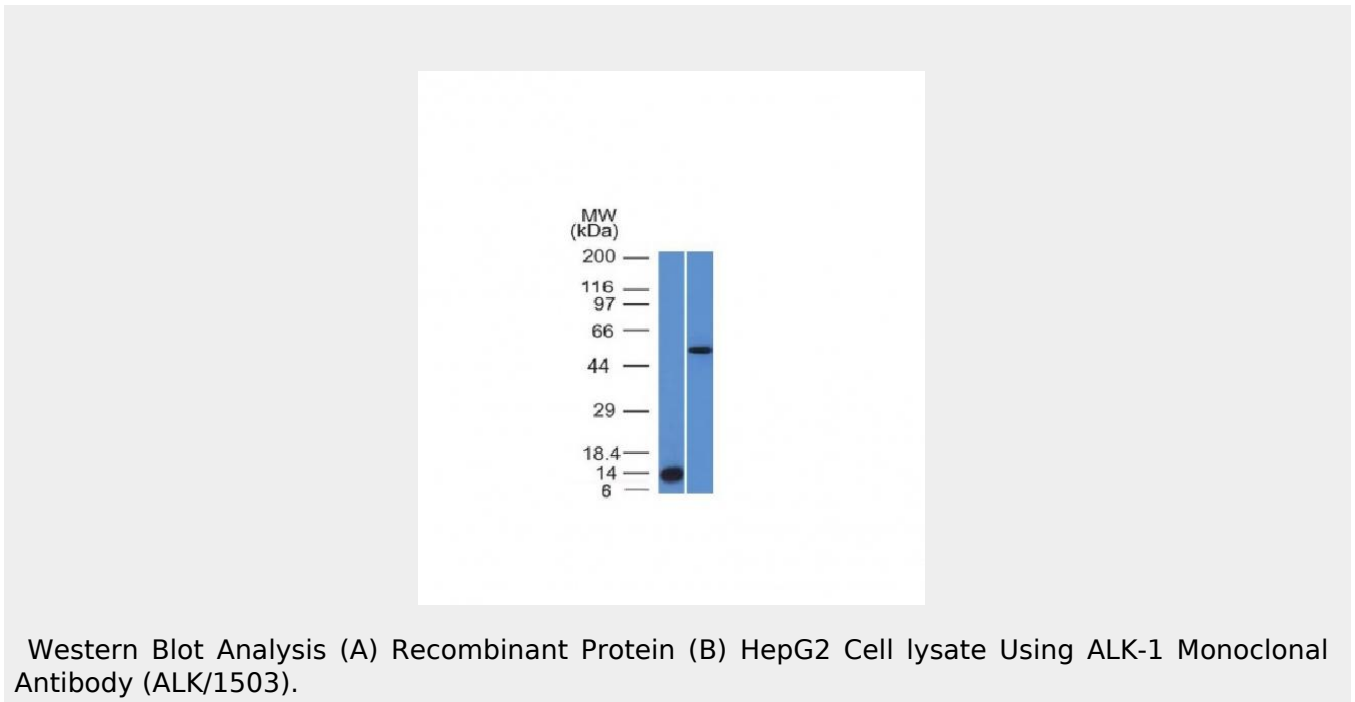
Expressed in brain and CNS. Also expressed in the small intestine and testis, but not in normal lymphoid cells

## Anti-ALK (Anaplastic Lymphoma Kinase) / CD246 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 Cytometry](#)
- [Cell Culture](#)

### Anti-ALK (Anaplastic Lymphoma Kinase) / CD246 Antibody - Images



### Anti-ALK (Anaplastic Lymphoma Kinase) / CD246 Antibody - Background

The wild-type anaplastic lymphoma kinase (ALK) protein is a 200kDa transmembrane receptor tyrosine kinase. Its expression is restricted to a few scattered cells in the nervous system (some glial cells and neurons, and a few endothelial cells and pericytes). The hybrid gene, NPM-ALK, created by the t(2;5)(p23;q35) chromosomal translocation encodes part of the nucleolar phosphoprotein, nucleophosmin (NPM), joined to the entire cytoplasmic portion of the anaplastic lymphoma kinase (ALK) receptor tyrosine kinase. As a consequence, the ALK gene comes under the control of the NPM promoter, which induces a permanent and ubiquitous transcription of the NPM-ALK hybrid gene, resulting in the production of a 80kDa NPM-ALK chimeric protein. This translocation is found in anaplastic large cell lymphomas (ALCL). Reportedly, expression of ALK indicates a better prognosis. Approximately 5%-10% of non-small cell lung carcinomas also express ALK protein producing a cytoplasmic staining pattern. This MAbs also reacts with blood vessels that serves as an internal positive control.