

NLK Antibody (C-term)
Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP7545b**Specification**

NLK Antibody (C-term) - Product Information

Application	WB, IHC-P,E
Primary Accession	Q9UBE8
Other Accession	D3ZSZ3 , O54949 , E1BMN8 , NP_057315
Reactivity	Human
Predicted	Bovine, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Antigen Region	406-436

NLK Antibody (C-term) - Additional Information**Gene ID** 51701**Other Names**

Serine/threonine-protein kinase NLK, Nemo-like kinase, Protein LAK1, NLK, LAK1
{ECO:0000312|EMBL:AAD560131}

Target/Specificity

This NLK antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 406-436 amino acids from the C-terminal region of human NLK.

Dilution

WB~~1:1000
IHC-P~~1:50~100

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

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

NLK Antibody (C-term) - Protein Information**Name** NLK

Synonyms LAK1 {ECO:0000312|EMBL:AAD56013.1}

Function Serine/threonine-protein kinase that regulates a number of transcription factors with key roles in cell fate determination (PubMed:[14960582](#), PubMed:[12482967](#), PubMed:[15004007](#), PubMed:[15764709](#), PubMed:[20061393](#), PubMed:[20874444](#), PubMed:[21454679](#)). Positive effector of the non-canonical Wnt signaling pathway, acting downstream of WNT5A, MAP3K7/TAK1 and HIPK2 (PubMed:[15004007](#), PubMed:[15764709](#)). Negative regulator of the canonical Wnt/beta-catenin signaling pathway (PubMed:[12482967](#)). Binds to and phosphorylates TCF7L2/TCF4 and LEF1, promoting the dissociation of the TCF7L2/LEF1/beta-catenin complex from DNA, as well as the ubiquitination and subsequent proteolysis of LEF1 (PubMed:[21454679](#)). Together these effects inhibit the transcriptional activation of canonical Wnt/beta-catenin target genes (PubMed:[12482967](#), PubMed:[21454679](#)). Negative regulator of the Notch signaling pathway (PubMed:[20118921](#)). Binds to and phosphorylates NOTCH1, thereby preventing the formation of a transcriptionally active ternary complex of NOTCH1, RBPJ/RBPSUH and MAML1 (PubMed:[20118921](#)). Negative regulator of the MYB family of transcription factors (PubMed:[15082531](#)). Phosphorylation of MYB leads to its subsequent proteolysis while phosphorylation of MYBL1 and MYBL2 inhibits their interaction with the coactivator CREBBP (PubMed:[15082531](#)). Other transcription factors may also be inhibited by direct phosphorylation of CREBBP itself (PubMed:[15082531](#)). Acts downstream of IL6 and MAP3K7/TAK1 to phosphorylate STAT3, which is in turn required for activation of NLK by MAP3K7/TAK1 (PubMed:[15004007](#), PubMed:[15764709](#)). Upon IL1B stimulus, cooperates with ATF5 to activate the transactivation activity of C/EBP subfamily members (PubMed:[25512613](#)). Phosphorylates ATF5 but also stabilizes ATF5 protein levels in a kinase-independent manner (PubMed:[25512613](#)). Acts as an inhibitor of the mTORC1 complex in response to osmotic stress by mediating phosphorylation of RPTOR, thereby preventing recruitment of the mTORC1 complex to lysosomes (PubMed:[26588989](#)).

Cellular Location

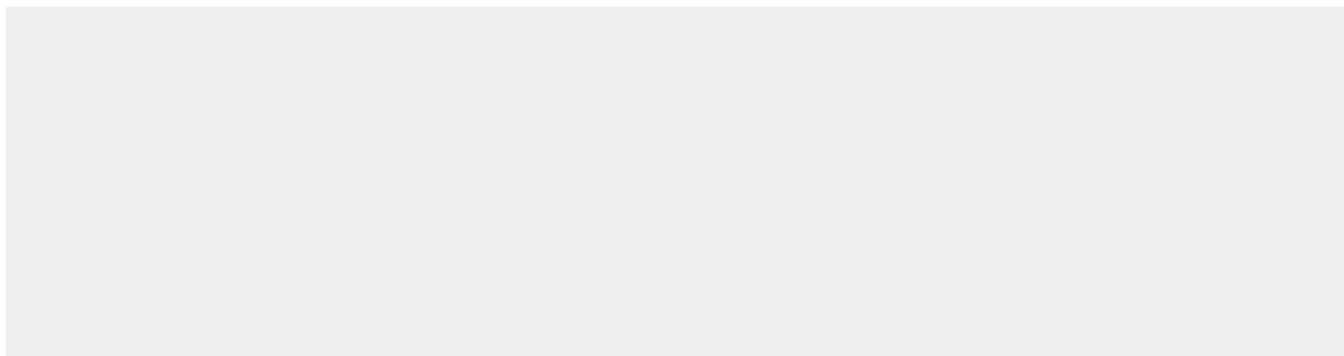
Nucleus {ECO:0000250|UniProtKB:O54949}. Cytoplasm {ECO:0000250|UniProtKB:O54949}.
Note=Predominantly nuclear. A smaller fraction is cytoplasmic.
{ECO:0000250|UniProtKB:O54949}

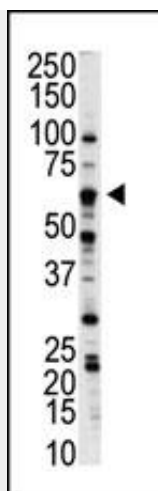
NLK Antibody (C-term) - Protocols

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

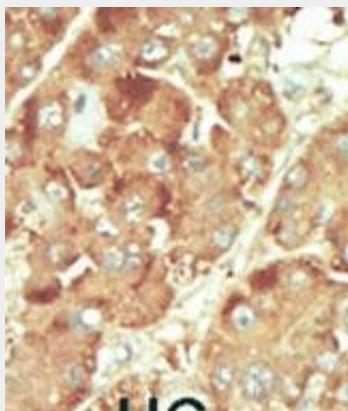
- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

NLK Antibody (C-term) - Images





Western blot analysis of anti-NLK Pab (Cat. #AP7545b) in A375 cell lysate. NLK (arrow) was detected using purified Pab. Secondary HRP-anti-rabbit was used for signal visualization with chemiluminescence.



Formalin-fixed and paraffin-embedded human cancer tissue reacted with the primary antibody, which was peroxidase-conjugated to the secondary antibody, followed by DAB staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has not been evaluated. BC = breast carcinoma; HC = hepatocarcinoma.

NLK Antibody (C-term) - Background

Protein kinases are enzymes that transfer a phosphate group from a phosphate donor, generally the γ phosphate of ATP, onto an acceptor amino acid in a substrate protein. By this basic mechanism, protein kinases mediate most of the signal transduction in eukaryotic cells, regulating cellular metabolism, transcription, cell cycle progression, cytoskeletal rearrangement and cell movement, apoptosis, and differentiation. With more than 500 gene products, the protein kinase family is one of the largest families of proteins in eukaryotes. The family has been classified in 8 major groups based on sequence comparison of their tyrosine (PTK) or serine/threonine (STK) kinase catalytic domains. The STE group (homologs of yeast Sterile 7, 11, 20 kinases) consists of 50 kinases related to the mitogen-activated protein kinase (MAPK) cascade families (Ste7/MAP2K, Ste11/MAP3K, and Ste20/MAP4K). MAP kinase cascades, consisting of a MAPK and one or more upstream regulatory kinases (MAPKKs) have been best characterized in the yeast pheromone response pathway. Pheromones bind to Ste cell surface receptors and activate yeast MAPK pathway.

NLK Antibody (C-term) - References

Yasuda, J., et al., Biochem. Biophys. Res. Commun. 308(2):227-233 (2003).

Ishitani, T., et al., Mol. Cell. Biol. 23(4):1379-1389 (2003).
Ishitani, T., et al., Mol. Cell. Biol. 23(1):131-139 (2003).
Kehrer-Sawatzki, H., et al., Gene 251(1):63-71 (2000).
Brott, B.K., et al., Proc. Natl. Acad. Sci. U.S.A. 95(3):963-968 (1998).