

**M MIKl Antibody (C-term)**  
**Affinity Purified Rabbit Polyclonal Antibody (Pab)**  
**Catalog # AP14272B**

**Specification**

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**M MIKl Antibody (C-term) - Product Information**

Application	IHC-P, WB,E
Primary Accession	<a href="#">Q9D2Y4</a>
Other Accession	<a href="#">NP_083281.1</a>
Reactivity	Mouse
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Antigen Region	444-472

**M MIKl Antibody (C-term) - Additional Information**

**Gene ID** 74568

**Other Names**

Mixed lineage kinase domain-like protein, MIKl {ECO:0000312|EMBL:AAH237551, ECO:0000312|MGI:MGI:1921818}

**Target/Specificity**

This Mouse MIKl antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 444-472 amino acids from the C-terminal region of mouse MIKl.

**Dilution**

IHC-P~~1~400

WB~~1:2000

**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**

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

**M MIKl Antibody (C-term) - Protein Information**

**Name** MIKl {ECO:0000303|PubMed:23835476, ECO:0000312|MGI:MGI:1921818}

**Function** Pseudokinase that plays a key role in TNF-induced necroptosis, a programmed cell

death process (PubMed:[23835476](#), PubMed:[24012422](#), PubMed:[24019532](#), PubMed:[27321907](#), PubMed:[32200799](#), PubMed:[32296175](#)). Does not have protein kinase activity (PubMed:[24012422](#)). Activated following phosphorylation by RIPK3, leading to homotrimerization, localization to the plasma membrane and execution of programmed necrosis characterized by calcium influx and plasma membrane damage (PubMed:[23835476](#), PubMed:[24012422](#), PubMed:[24019532](#), PubMed:[27321907](#)). In addition to TNF-induced necroptosis, necroptosis can also take place in the nucleus in response to orthomyxoviruses infection: following ZBP1 activation, which senses double-stranded Z-RNA structures, nuclear RIPK3 catalyzes phosphorylation and activation of MLKL, promoting disruption of the nuclear envelope and leakage of cellular DNA into the cytosol (PubMed:[32200799](#), PubMed:[32296175](#)). Binds to highly phosphorylated inositol phosphates such as inositolhexakisphosphate (InsP6) which is essential for its necroptotic function (By similarity).

#### Cellular Location

Cytoplasm. Cell membrane. Nucleus. Note=Localizes to the cytoplasm and translocates to the plasma membrane on necroptosis induction (By similarity). Localizes to the nucleus in response to orthomyxoviruses infection (PubMed:32200799). {ECO:0000250|UniProtKB:Q8NB16, ECO:0000269|PubMed:32200799}

#### Tissue Location

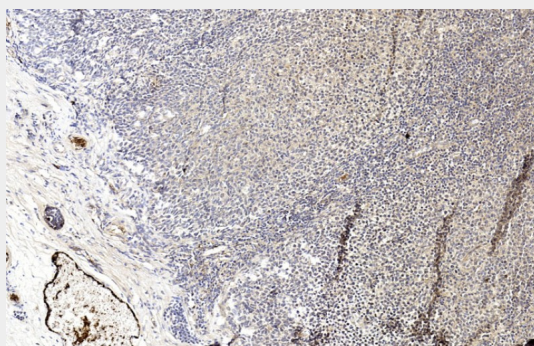
Highly expressed in thymus, colon, intestine, liver, spleen and lung. Expressed at much lower level in skeletal muscle, heart and kidney. Not detected in brain

### M MIK1 Antibody (C-term) - Protocols

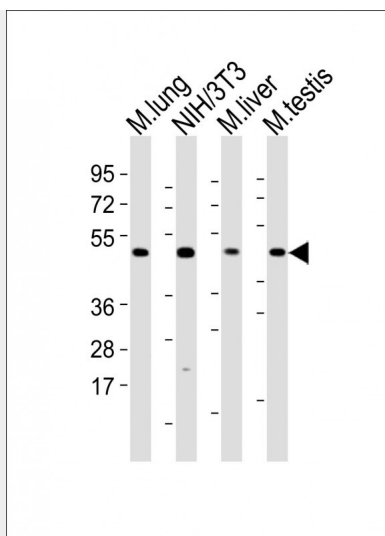
Provided below are standard protocols that you may find useful for product applications.

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

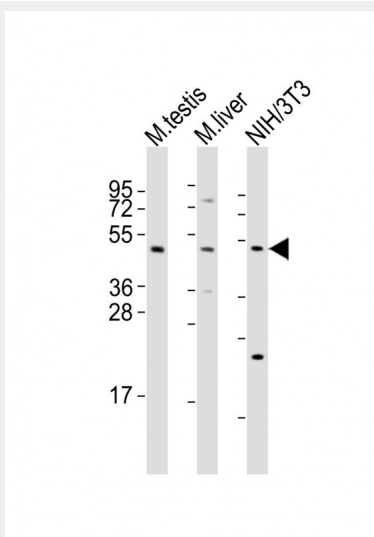
### M MIK1 Antibody (C-term) - Images



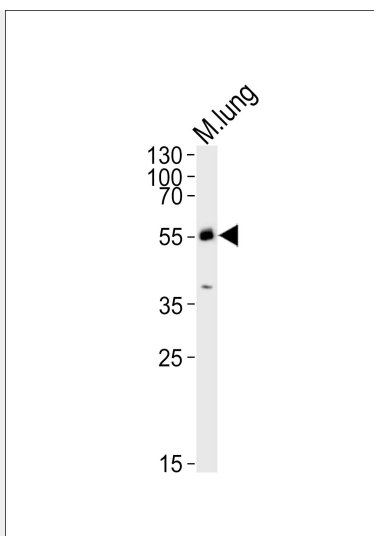
Immunohistochemical analysis of paraffin-embedded Human tonsil section using M MIK1 antibody (Cat#AP14272b). AP14272b was diluted at 1~400 dilution. A undiluted biotinylated goat polyclonal antibody was used as the secondary, followed by DAB staining.



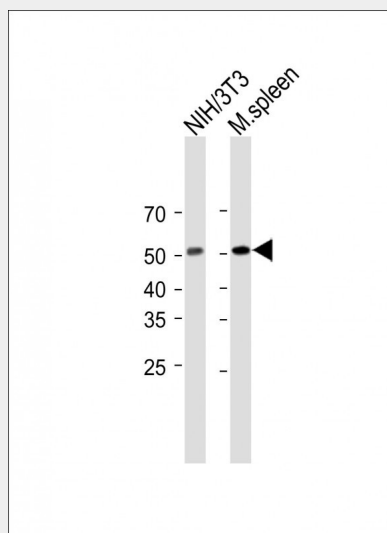
All lanes : Anti-MIK1 Antibody (C-term) at 1:2000 dilution Lane 1: mouse lung lysates Lane 2: NIH/3T3 whole cell lysates Lane 3: mouse liver lysates Lane 4: mouse testis whole cell lysates Lysates/proteins at 20 µg per lane. Secondary Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated at 1/10000 dilution Predicted band size : 54 kDa Blocking/Dilution buffer: 5% NFDM/TBST.



All lanes : Anti-MIK1 Antibody (C-term) at 1:2000 dilution Lane 1: mouse testis lysates Lane 2: mouse liver lysates Lane 3: NIH/3T3 whole cell lysates Lysates/proteins at 20 µg per lane. Secondary Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated at 1/10000 dilution Predicted band size : 54 kDa Blocking/Dilution buffer: 5% NFDM/TBST.



Western blot analysis of lysate from mouse lung tissue lysate, using Mlkl Antibody (C-term)(Cat. #AP14272b). AP14272b was diluted at 1:1000. A goat anti-rabbit IgG H&L(HRP) at 1:10000 dilution was used as the secondary antibody. Lysate at 20ug.



All lanes: Anti-M Mlkl Antibody (C-term) at 1:2000 dilution Lane 1: NIH/3T3 whole cell lysate Lane 2: mouse spleen 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: 53KDa Blocking/Dilution buffer: 5% NFDM/TBST.

### **M Mlkl Antibody (C-term) - Background**

The protein kinase domain is predicted to be catalytically inactive. Molecular function: protein binding. There are two isoforms.

### **M Mlkl Antibody (C-term) - References**

Bisson, N., et al. Cell Cycle 7(7):909-916(2008)

#### **M Mlkl Antibody (C-term) - Citations**

- [β-Cyclodextrin/dialdehyde glucan-coated keratin nanoparticles for oral delivery of insulin](#)
- [Inflammatory cell death, PANoptosis, screen identifies host factors in coronavirus innate immune response as therapeutic targets](#)
- [-GlcNAcylation of RIPK1 rescues red blood cells from necroptosis](#)

- [Extracellular vesicles mediate antibody-resistant transmission of SARS-CoV-2](#)
- [Hyperphosphorylated tau mediates neuronal death by inducing necroptosis and inflammation in Alzheimer's disease](#)
- [ZBP1-dependent inflammatory cell death, PANoptosis, and cytokine storm disrupt IFN therapeutic efficacy during coronavirus infection](#)
- [Salt-inducible kinases inhibitor HG-9-91-01 targets RIPK3 kinase activity to alleviate necroptosis-mediated inflammatory injury](#)
- [Sustained ErbB Activation Causes Demyelination and Hypomyelination by Driving Necroptosis of Mature Oligodendrocytes and Apoptosis of Oligodendrocyte Precursor Cells](#)
- [Activation of mTORC1 and c-Jun by Prohibitin1 loss in Schwann cells may link mitochondrial dysfunction to demyelination](#)
- [AIM2 forms a complex with pyrin and ZBP1 to drive PANoptosis and host defence](#)
- [Down-regulation of pro-necroptotic molecules blunts necroptosis during myogenesis](#)
- [A phosphorylation of RIPK3 kinase initiates an intracellular apoptotic pathway that promotes prostaglandin-induced corpus luteum regression](#)
- [Synergism of TNF- \$\alpha\$  and IFN- \$\gamma\$  Triggers Inflammatory Cell Death, Tissue Damage, and Mortality in SARS-CoV-2 Infection and Cytokine Shock Syndromes](#)
- [ZBP1 promotes fungi-induced inflammasome activation and pyroptosis, apoptosis, and necroptosis \(PANoptosis\)](#)
- [TNF-mediated alveolar macrophage necroptosis drives disease pathogenesis during Respiratory Syncytial Virus infection](#)
- [Discovery of a Potent RIPK3 Inhibitor for the Amelioration of Necroptosis-Associated Inflammatory Injury](#)
- [Myofiber necroptosis promotes muscle stem cell proliferation via releasing Tenascin-C during regeneration](#)
- [Casein kinase 1G2 suppresses necroptosis-promoted testis aging by inhibiting receptor-interacting kinase 3](#)
- [De novo necroptosis creates an inflammatory environment mediating tumor susceptibility to immune checkpoint inhibitors](#)
- [COVID-19 cytokines and the hyperactive immune response: Synergism of TNF- \$\alpha\$  and IFN- \$\gamma\$  in triggering inflammation, tissue damage, and death](#)
- [XJB-5-131 inhibited ferroptosis in tubular epithelial cells after ischemia-reperfusion injury](#)
- [SARM1 acts downstream of neuroinflammatory and necroptotic signaling to induce axon degeneration](#)
- [Beclin 1 functions as a negative modulator of MLKL oligomerisation by integrating into the necrosome complex](#)
- [Crucial Roles of the RIP Homotypic Interaction Motifs of RIPK3 in RIPK1-Dependent Cell Death and Lymphoproliferative Disease](#)
- [Innate immune priming in the absence of TAK1 drives RIPK1 kinase activity-independent pyroptosis, apoptosis, necroptosis, and inflammatory disease](#)
- [Ubiquitination of RIPK1 suppresses programmed cell death by regulating RIPK1 kinase activation during embryogenesis](#)
- [Shifting the balance of autophagy and proteasome activation reduces proteotoxic cell death: a novel therapeutic approach for restoring photoreceptor homeostasis](#)
- [Flotillin-mediated endocytosis and ALIX-syntenin-1-mediated exocytosis protect the cell membrane from damage caused by necroptosis](#)
- [Oncolysis with DTT-205 and DTT-304 generates immunological memory in cured animals](#)
- [Kinase domain dimerization drives RIPK3-dependent necroptosis](#)
- [HECTD3 mediates TRAF3 polyubiquitination and type I interferon induction during bacterial infection](#)
- [Pretreatment of Huaqiqi Huang extractum protects against cisplatin-induced nephrotoxicity](#)
- [RIP kinase 1-dependent endothelial necroptosis underlies systemic inflammatory response syndrome](#)
- [Phenytolol inhibits necroptosis](#)
- [Generation and Use of Chimeric RIP Kinase Molecules to Study Necroptosis](#)

- [Embryonic Lethality and Host Immunity of RelA-Deficient Mice Are Mediated by Both Apoptosis and Necroptosis.](#)
- [RIPK1-RIPK3-MLKL-dependent necrosis promotes the aging of mouse male reproductive system.](#)
- [Nucleotide-binding oligomerization domain \(NOD\) signaling defects and cell death susceptibility cannot be uncoupled in X-linked inhibitor of apoptosis \(XIAP\)-driven inflammatory disease.](#)
- [Regulation of NKT cell-mediated immune responses to tumours and liver inflammation by mitochondrial PGAM5-Drp1 signalling.](#)
- [Necroptosis is preceded by nuclear translocation of the signaling proteins that induce it.](#)
- [Characterization of RIPK3-mediated phosphorylation of the activation loop of MLKL during necroptosis.](#)
- [RIP1 suppresses innate immune necrotic as well as apoptotic cell death during mammalian parturition.](#)
- [Toll-like receptor 3-mediated necrosis via TRIF, RIP3, and MLKL.](#)