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M Mlkl Antibody (C-term)

Affinity Purified Rabbit Polyclonal Antibody (Pab)

Catalog # AP14272B

Specification

M Mlkl Antibody (C-term) - Product Information

Application	**WB, IHC-P,E**
Primary Accession	Q9D2Y4
Other Accession	NP_083281.1
Reactivity	**Mouse**
Host	**Rabbit**
Clonality	**Polyclonal**
Isotype	**Rabbit IgG**
Antigen Region	**444-472**

M Mlkl Antibody (C-term) - Additional Information

Gene ID 74568

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

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

Dilution
WB~~1:2000
IHC-P~~1~400
E~~Use at an assay dependent concentration.

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

M Mlkl Antibody (C-term) - Protein Information

Name Mlkl {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}

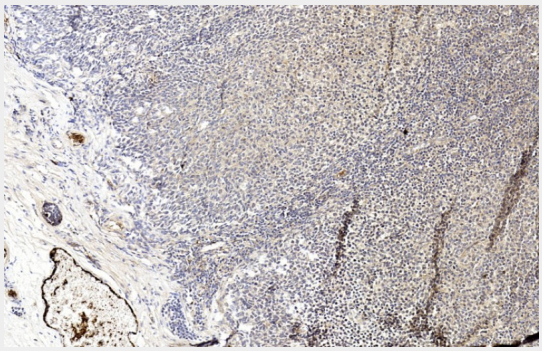
Tissue Location
Highly expressed in thymus, colon, intestine, liver, spleen and lung. Expressed at much lower level in skeletal muscle, heart and kidney. Not detected in brain

M Mlkl 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 Cytomety
- Cell Culture

M Mlkl Antibody (C-term) - Images



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

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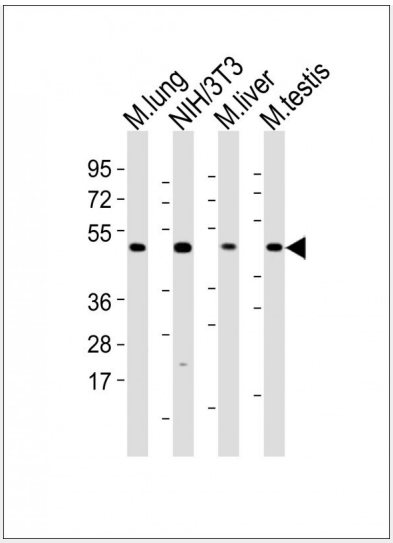
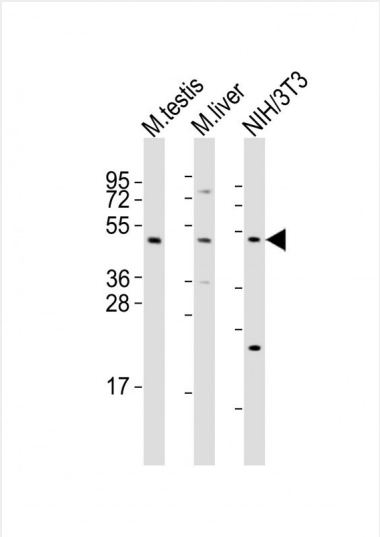


All lanes : Anti-Mlkl Antibody (C-term) at 1:2000 dilution Lane 1: mouse lung lysates Lane 2: NIH/3T3 whole cell lysates Lane 3: mouse liver lysates Lane 4: mouse testis whole cell lysates Lysates/proteins at 20 µg per lane. Secondary Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated at 1/10000 dilution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



All lanes : Anti-Mlkl 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.

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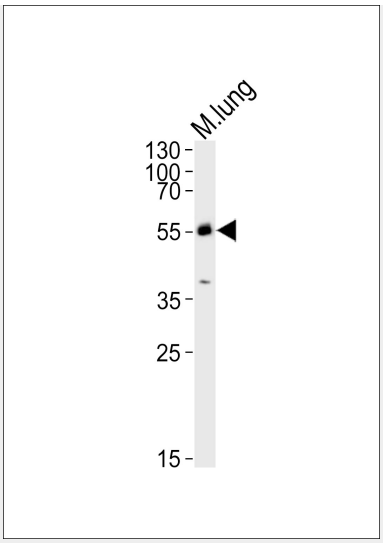
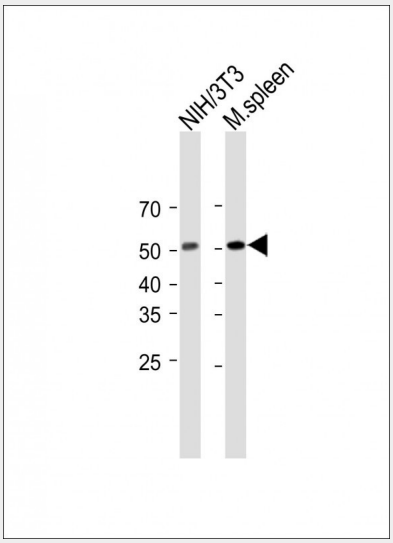


Western blot analysis of lysate from mouse lung tissue lysate, using Mlkl Antibody (C-term)(Cat. #AP14272b). AP14272b was diluted at 1:1000. A goat anti-rabbit IgG H&L(HRP) at 1:10000 dilution was used as the secondary antibody. Lysate at 20ug.



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-α and IFN-γ 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-α and IFN-γ 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 Huaiqihuang extractum protects against cisplatin-induced nephrotoxicity.
- RIP kinase 1-dependent endothelial necroptosis underlies systemic inflammatory response syndrome.
- Phenytoin 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.