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M Mlkl Antibody (C-term)

Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP14272B

Product Information

Application	WB, IHC-P, E
Primary Accession	Q9D2Y4
Other Accession	NP_083281.1
Reactivity	Mouse
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	54317
Antigen Region	444-472

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~~1:1000 IHC-P~~1~400 E~~Use at an assay dependent concentration.
Format	Purified polyclonal antibody supplied in PBS with 0.05% (V/V) Proclin 300. 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.

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

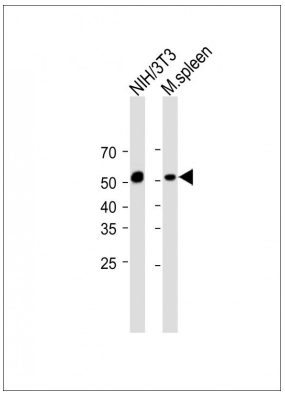
Background

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

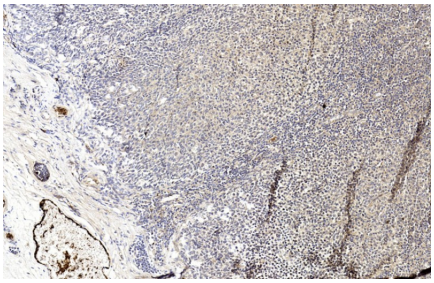
References

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

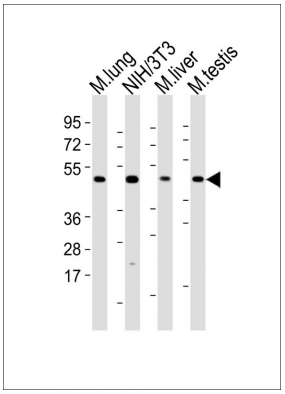
Images



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.



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.



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.

Citations

- Extracellular vesicles mediate antibody-resistant transmission of SARS-CoV-2
- 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
- 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.