

RIP3 Antibody

Catalog # ASC10108

Specification

RIP3 Antibody - Product Information

Application
Primary Accession
Other Accession
Reactivity
Host
Clonality
Isotype

Calculated MW Application Notes WB, IHC, IF Q9QZL0

AAF03133, 6063101

Mouse, Rat Rabbit Polyclonal

lgG

57 kDa KDa

RIP3 antibody can be used for detection of RIP3 by Western blot at 1 μ g/mL. An approximately 57 kDa band can be detected. Antibody can also be used for immunohistochemistry starting at 5 μ g/mL. For immunofluorescence start at 20 μ g/mL.

RIP3 Antibody - Additional Information

Gene ID 56532

Other Names

RIP3 Antibody: Rip3, AW107945, 2610528K09Rik, Rip3, RIP-like protein kinase 3, RIP-3, receptor-interacting serine-threonine kinase 3

Target/Specificity

Ripk3;

Reconstitution & Storage

Antibody can be stored at 4°C up to one year. Antibodies should not be exposed to prolonged high temperatures.

Precautions

RIP3 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

RIP3 Antibody - Protein Information

Name Ripk3 {ECO:0000303|PubMed:27321907, ECO:0000312|MGI:MGI:2154952}

Function

Serine/threonine-protein kinase that activates necroptosis and apoptosis, two parallel forms of cell death (PubMed:<a href="http://www.uniprot.org/citations/27321907"

target="_blank">27321907, PubMed:27746097, PubMed:<a href="http://www.uniprot.org/citations/27917412"

target="_blank">27917412, PubMed:<a href="http://www.uniprot.org/citations/28607035"

target="_blank">28607035, PubMed:<a href="http://www.uniprot.org/citations/32200799"



target=" blank">32200799, PubMed:32296175). Necroptosis, a programmed cell death process in response to death-inducing TNF-alpha family members, is triggered by RIPK3 following activation by ZBP1 (PubMed:19590578, PubMed:22423968, PubMed:24012422, PubMed:24019532, PubMed:24557836, PubMed:27746097, PubMed: 27819681, PubMed:27819682, PubMed:24095729, PubMed: 32200799, PubMed: 27321907, PubMed:32296175). Activated RIPK3 forms a necrosis- inducing complex and mediates phosphorylation of MLKL, promoting MLKL localization to the plasma membrane and execution of programmed necrosis characterized by calcium influx and plasma membrane damage (PubMed: 24813849, PubMed:24813850, 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). Also regulates apoptosis: apoptosis depends on RIPK1, FADD and CASP8, and is independent of MLKL and RIPK3 kinase activity (PubMed: 27321907). Phosphorylates RIPK1: RIPK1 and RIPK3 undergo reciprocal auto- and trans-phosphorylation (By similarity). In some cell types, also able to restrict viral replication by promoting cell death-independent responses (PubMed: 30635240). In response to flavivirus infection in neurons, promotes a cell death-independent pathway that restricts viral replication: together with ZBP1, promotes a death-independent transcriptional program that modifies the cellular metabolism via up-regulation expression of the enzyme ACOD1/IRG1 and production of the metabolite itaconate (PubMed: 30635240). Itaconate inhibits the activity of succinate dehydrogenase, generating a metabolic state in neurons that suppresses replication of viral genomes (PubMed:30635240). RIPK3 binds to and enhances the activity of three metabolic enzymes: GLUL, GLUD1, and PYGL (By similarity). These metabolic enzymes may eventually stimulate the tricarboxylic acid cycle and oxidative phosphorylation, which could result in enhanced ROS production (By similarity).

Cellular Location

Cytoplasm, cytosol. Nucleus. Note=Mainly cytoplasmic (PubMed:32200799, PubMed:32296175). Present in the nucleus in response to influenza A virus (IAV) infection (PubMed:32200799).

Tissue Location

Expressed in embryo and in adult spleen, liver, testis, heart, brain and lung.

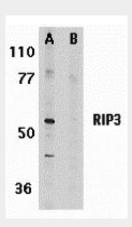
RIP3 Antibody - Protocols

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

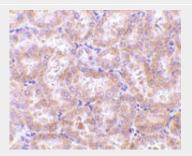


- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- <u>Immunoprecipitation</u>
- Flow Cytomety
- Cell Culture

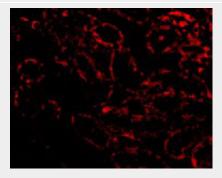
RIP3 Antibody - Images



Western blot analysis of RIP3 in mouse 3T3 whole cell lysate in the absence (A) or presence (B) of blocking peptide with RIP3 antibody at $1 \mu g/mL$.



Immunohistochemistry of RIP3 in rat kidney tissue with RIP3 antibody at 5 µg/mL.



Immunofluorescence of RIP3 in Rat Kidney cells with RIP3 antibody at 20 μg/mL.

RIP3 Antibody - Background

RIP3 Antibody: Certain serine/threonine protein kinases, such as ASK1, RIP, DAP, and ZIP kinases, are mediators of apoptosis. Receptor interacting proteins including RIP and RIP2/RICK mediate apoptosis induced by TNFR1 and Fas, two prototype members in the death receptor family. A novel



member in the RIP kinase family was recently identified and designated RIP3. RIP3 contains N-terminal kinase domain but, unlike RIP or RIP2, lacks the C-terminal death or CARD domain. RIP3 binds to RIP and TNFR1, mediates TNFR1 induced apoptosis, and attenuates RIP and TNFR1 induced NF-kB activation. Overexpression of RIP3 induces apoptosis and NF-kB activation. The messenger RNA of RIP3 is expressed in a subset of adult tissues.

RIP3 Antibody - References

Yu PW, Huang BC, Shen M, Quast J, Chan E, Xu X, Nolan GP, Payan DG, Luo Y. Identification of RIP3, a RIP-like kinase that activates apoptosis and NFkB. Curr Biol. 1999;9(10):539-42.

Sun X, Lee J, Navas T, Baldwin DT, Stewart TA, Dixit VM. RIP3, a novel apoptosis-inducing kinase. J Biol Chem. 1999;274(24):16871-5.

Pazdernik NJ, Donner DB, Goebl MG, Harrington MA. Mouse receptor interacting protein 3 does not contain a caspase-recruiting or a death domain but induces apoptosis and activates NF-κB. Mol Cell Bio. 1999; 19(10):6500-8 (WD0102)

RIP3 Antibody - Citations

- TSC2 Deficiency Unmasks a Novel Necrosis Pathway That Is Suppressed by the RIP1/RIP3/MLKL Signaling Cascade.
- <u>Differential contribution of complement receptor C5aR in myeloid and non-myeloid cells in chronic ethanol-induced liver injury in mice.</u>