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RIP3 Antibody

Catalog # ASC10108

Product Information

Application	WB, IF, E, IHC-P
Primary Accession	Q9QZL0
Other Accession	AAF03133, 6063101
Reactivity	Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Calculated MW	53322
Conjugate	Unconjugated
Application Notes	RIP3 antibody can be used for detection of RIP3 by Western blot at 1 μg/mL. An approximately 57 kDa band can be detected. Antibody can also be used for immunohistochemistry starting at 5 μg/mL. For immunofluorescence start at 20 μg/mL.

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	RIP3 antibody can be stored at 4℃ for three months and -20℃, stable for up to one year. As with all antibodies care should be taken to avoid repeated freeze thaw cycles. 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.

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:27321907, PubMed:27746097, PubMed:27917412, PubMed:28607035, PubMed:32200799, PubMed:32296175). Necroptosis, a programmed cell death process in response to death-inducing TNF family members, is triggered by RIPK3 following activation by ZBP1 (PubMed:19590578, PubMed:22423968, PubMed:24012422, PubMed:24019532, PubMed:24095729, PubMed:24557836, PubMed:27321907, PubMed:27746097, PubMed:27819681, PubMed:27819682, PubMed:32200799,

	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.

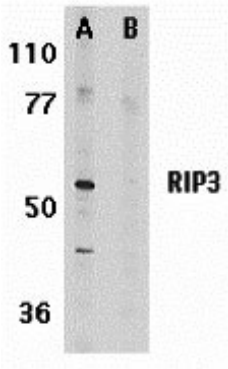
Background

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-κB activation. Overexpression of RIP3 induces apoptosis and NF-κB activation. The messenger RNA of RIP3 is expressed in a subset of adult tissues.

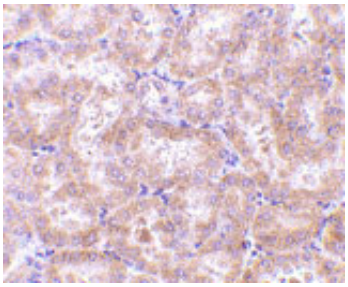
References

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 NFκB. 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)

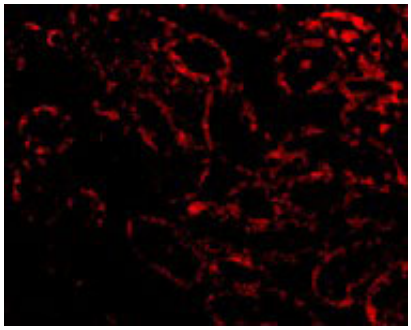
Images



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 µg/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.

Citations

- TSC2 Deficiency Unmasks a Novel Necrosis Pathway That Is Suppressed by the RIP1/RIP3/MLKL Signaling Cascade.

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.