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RIPK3 Antibody (C-term)

Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP7819B

Product Information

Application	WB, IHC-P, E
Primary Accession	Q9Y572
Other Accession	NP_006862
Reactivity	Human, Mouse
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	56887
Antigen Region	489-518

Additional Information

Gene ID	11035
Other Names	Receptor-interacting serine/threonine-protein kinase 3, RIP-like protein kinase 3, Receptor-interacting protein 3, RIP-3, RIPK3, RIP3
Target/Specificity	This RIPK3 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 489-518 amino acids from the C-terminal region of human RIPK3.
Dilution	WB~~1:1000 IHC-P~~1:100~500 E~~Use at an assay dependent concentration.
Format	Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.
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	RIPK3 Antibody (C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	RIPK3 (HGNC:10021)
Function	Serine/threonine-protein kinase that activates necroptosis and apoptosis, two parallel forms of cell death (PubMed:19524512, PubMed:19524513, PubMed:22265413, PubMed:22265414, PubMed:22421439, PubMed:29883609, PubMed:32657447). Necroptosis, a programmed cell

	death process in response to death-inducing TNF family members, is triggered by RIPK3 following activation by ZBP1 (PubMed:19524512, PubMed:19524513, PubMed:22265413, PubMed:22265414, PubMed:22421439, PubMed:29883609, PubMed:32298652). 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:19524512, PubMed:19524513, PubMed:22265413, PubMed:22265414, PubMed:22421439, PubMed:25316792, PubMed:29883609). 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 (By similarity). Also regulates apoptosis: apoptosis depends on RIPK1, FADD and CASP8, and is independent of MLKL and RIPK3 kinase activity (By similarity). Phosphorylates RIPK1: RIPK1 and RIPK3 undergo reciprocal auto- and trans-phosphorylation (PubMed:19524513). In some cell types, also able to restrict viral replication by promoting cell death- independent responses (By similarity). In response to Zika virus 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 (By similarity). Itaconate inhibits the activity of succinate dehydrogenase, generating a metabolic state in neurons that suppresses replication of viral genomes (By similarity). RIPK3 binds to and enhances the activity of three metabolic enzymes: GLUL, GLUD1, and PYGL (PubMed:19498109). These metabolic enzymes may eventually stimulate the tricarboxylic acid cycle and oxidative phosphorylation, which could result in enhanced ROS production (PubMed:19498109).
Cellular Location	Cytoplasm, cytosol. Nucleus {ECO:0000250|UniProtKB:Q9QZL0}. Note=Mainly cytoplasmic Present in the nucleus in response to influenza A virus (IAV) infection. {ECO:0000250|UniProtKB:Q9QZL0}
Tissue Location	Highly expressed in the pancreas. Detected at lower levels in heart, placenta, lung and kidney

Background

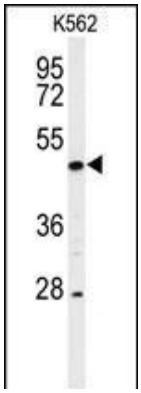
RIPK3 is a member of the receptor-interacting protein (RIP) family of serine/threonine protein kinases, and contains a C-terminal domain unique from other RIP family members. The encoded protein is predominantly localized to the cytoplasm, and can undergo nucleocytoplasmic shuttling dependent on novel nuclear localization and export signals. It is a component of the tumor necrosis factor (TNF) receptor-I signaling complex, and can induce apoptosis and weakly activate the NF-kappaB transcription factor.

References

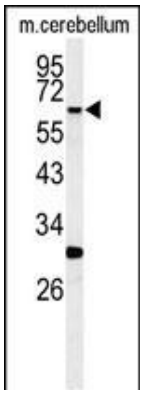
Yu, P.W., et al., Curr. Biol. 9(10):539-542 (1999).
Sun, X., et al., J. Biol. Chem. 274(24):16871-16875 (1999).

Images

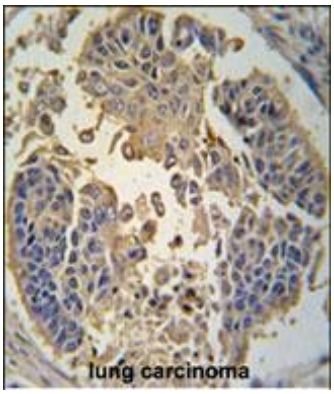
Western blot analysis of hRIPK3-E504 (Cat. #AP7819b) in K562 cell line lysates (35ug/lane). RIPK3 (arrow) was



detected using the purified Pab.



Western blot analysis of hRIPK3-E504 (Cat. #AP7819b) in mouse cerebellum tissue lysates (35ug/lane). RIPK3 (arrow) was detected using the purified Pab.



Formalin-fixed and paraffin-embedded human lung carcinoma tissue reacted with RIP3 (RIPK3) antibody (C-term) (Cat.# AP7819b), which was peroxidase-conjugated to the secondary antibody, followed by DAB staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has not been evaluated.

Citations

- Shifting the balance of autophagy and proteasome activation reduces proteotoxic cell death: a novel therapeutic approach for restoring photoreceptor homeostasis.
- Inhibition of apoptosis protects mice from ethanol-mediated acceleration of early markers of CCl4 -induced fibrosis but not steatosis or inflammation.
- Toll-like receptor 3 signaling attenuates liver regeneration.
- Divergent effects of RIP1 or RIP3 blockade in murine models of acute liver injury.
- Receptor Interacting protein kinase-1 mediates murine acetaminophen toxicity independent of the necrosome and not through necroptosis.
- Cell Death Pathways in Mutant Rhodopsin Rat Models Identifies Genotype-Specific Targets Controlling Retinal Degeneration.
- Differential contribution of complement receptor C5aR in myeloid and non-myeloid cells in chronic ethanol-induced liver injury in mice.

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