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RIP3 Recombinant Rabbit mAb

Catalog # AP99180

Product Information

Application	WB, IHC-P, IHC-F, IF, IP, E
Primary Accession	Q9Y572
Other Accession	Q9Y572
Reactivity	Mouse
Host	Rabbit
Clonality	Recombinant
Calculated MW	56887
Isotype	IgG
Purity	affinity purified by Protein A
Buffer	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
SIMILARITY	Belongs to the protein kinase superfamily. TKL Ser/Thr protein kinase family.Contains 1 protein kinase domain.
SUBUNIT	Interacts (via RIP homotypic interaction motif) with RIPK1 (via RIP homotypic interaction motif); this interaction induces RIPK1 phosphorylation and formation of a RIPK1-RIPK3 necrosis-inducing complex. Upon TNF-induced necrosis, the RIPK1-RIPK3 dimer further interacts with PGAM5 and MLKL; the formation of this complex leads to PGAM5 phosphorylation and increase in PGAM5 phosphatase activity. Binds TRAF2 and is recruited to the TNFR-1 signaling complex. Interacts with PYGL, GLUL and GLUD1; these interactions result in activation of these metabolic enzymes. Interacts with BIRC2/c-IAP1, BIRC3/c-IAP2 and XIAP/BIRC4.
Post-translational modifications	RIPK1 and RIPK3 undergo reciprocal auto- and trans-phosphorylation. Phosphorylation of Ser-199 plays a role in the necroptotic function of RIPK3.Polyubiquitinated with 'Lys-48' and 'Lys-63'-linked chains by BIRC2/c-IAP1 and BIRC3/c-IAP2, leading to activation of NF-kappa-B.
Background Descriptions	The product of this gene 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. [provided by RefSeq, Jul 2008],

Additional Information

Gene ID	11035
Other Names	RIP3; RIPK3_HUMAN; RIPK3; RIP-like protein kinase 3; Receptor-interacting protein 3 (RIP-3); 2.7.11.1; RIPK3_MOUSE; Receptor-interacting protein 3 (RIP-3 | mRIP3); receptor interacting serine/threonine kinase 3; receptor-interacting serine-threonine kinase 3; Receptor-interacting serine/threonine-protein kinase 3 (EC 2.7.11.1) (RIP-like protein kinase 3)

	(Receptor-interacting protein 3) (RIP-3); Receptor-interacting serine/threonine-protein kinase 3; Receptor-interacting protein 3; RIP-3
Dilution	IHC-P=1:50-200,IHC-F=1:50-200,WB=1:500-2000,IF=1:50-200,ELISA=1:5000-10000,IP=1:50-200
Storage	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.

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

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