10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

RIPK3 Antibody

Purified Mouse Monoclonal Antibody (Mab)
Catalog # AM8682b

Product Information

Application	WB, E
Primary Accession	Q9Y572
Reactivity	Human
Predicted	Human
Host	Mouse
Clonality	monoclonal
Isotype	IgG1,κ
Clone Names	2013CT892.86.49
Calculated MW	56887

Additional Information

Gene ID	11035
Other Names	Receptor-interacting serine/threonine-protein kinase 3, 2.7.11.1, RIP-like protein kinase 3, Receptor-interacting protein 3, RIP-3, RIPK3, RIP3
Target/Specificity	This RIPK3 antibody is generated from a mouse immunized with a recombinate protein from the human region of human RIPK3.
Dilution	WB~~1:1000 E~~Use at an assay dependent concentration.
Format	Purified monoclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein G column, 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 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

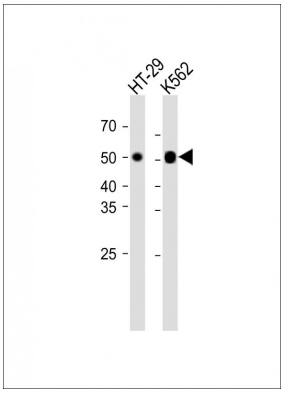
Background

Essential for necroptosis, a programmed cell death process in response to death-inducing TNF-alpha family members. Upon induction of necrosis, RIPK3 interacts with, and phosphorylates RIPK1 and MLKL to form a necrosis-inducing complex. RIPK3 binds to and enhances the activity of three metabolic enzymes: GLUL, GLUD1, and PYGL. These metabolic enzymes may eventually stimulate the tricarboxylic acid cycle and oxidative phosphorylation, which could result in enhanced ROS production.

References

Yu P.W.,et al.Curr. Biol. 9:539-542(1999).
Sun X.,et al.J. Biol. Chem. 274:16871-16875(1999).
Yang Y.,et al.Biochem. Biophys. Res. Commun. 332:181-187(2005).
Heilig R.,et al.Nature 421:601-607(2003).
Ota T.,et al.Nat. Genet. 36:40-45(2004).

Images



All lanes: Anti-RIPK3 Antibody at 1:2000 dilution Lane 1: HT-29 whole cell lysate Lane 2: K562 whole cell lysate Lysates/proteins at 20 µg per lane. Secondary: Goat Anti-Mouse IgG, (H+L), Peroxidase conjugated (ASP1613) at 1/8000 dilution. Observed band size: 46-62 KDa Blocking/Dilution buffer: 5% NFDM/TBST.

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