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

hRIP3 Antibody

Catalog # ASC12188

Product Information

Application	WB, IHC-P, IF, E
Primary Accession	Q9Y572
Other Accession	Q9Y572
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Clone Names	Ripk3
Calculated MW	56887

Additional Information

Gene ID	11035
Alias Symbol	Ripk3
Other Names	hRIP3 Antibody: Rip3, AW107945, 2610528K09Rik, RIP-like protein kinase 3, RIP-3
Target/Specificity	Several isoforms of hRIP3 are known to exist.
Reconstitution & Storage	hRIP3 antibody can be stored at 4˚C for three months and -20˚C, 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	hRIP3 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-alpha 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

hRIP3 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 et al. Curr Biol. 1999;9(10):539-42. Sun et al. J Biol Chem. 1999;274(24):16871-5. Pazdernik et al. Mol Cell Bio. 1999; 19(10):6500-8 (WD0102)