

RIPK3 Antibody (Center)
Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP7184b

Specification

RIPK3 Antibody (Center) - Product Information

Application	WB, IHC-P,E
Primary Accession	O9Y572
Reactivity	Human, Mouse
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Antigen Region	61-91

RIPK3 Antibody (Center) - 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 61-91 amino acids from the Central region of human RIPK3.

Dilution

WB~~1:1000
IHC-P~~1:10~50

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 (Center) is for research use only and not for use in diagnostic or therapeutic procedures.

RIPK3 Antibody (Center) - 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 orthomyxovirus 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

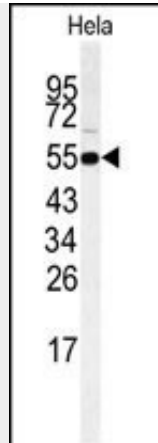
RIPK3 Antibody (Center) - Protocols

Provided below are standard protocols that you may find useful for product applications.

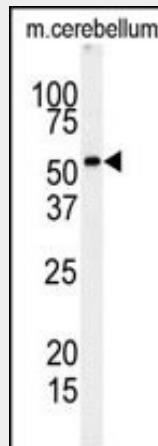
- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

RIPK3 Antibody (Center) - Images

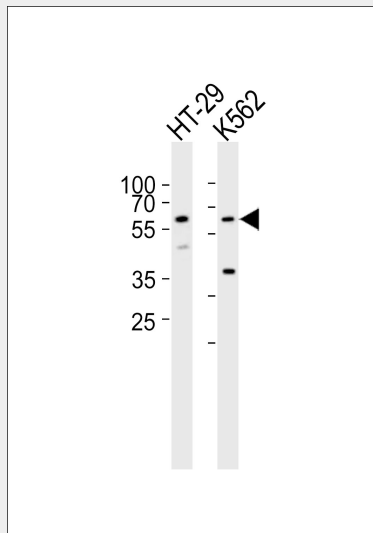




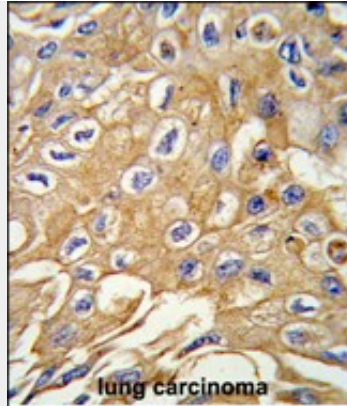
Western blot analysis of RIP3 (RIPK3) Antibody (Center) (Cat.# AP7184b) in HeLa cell line lysates (35ug/lane). RIPK3 (arrow) was detected using the purified Pab.



Western blot analysis of RIP3 (RIPK3) Antibody (Center) (Cat.# AP7184b) in mouse cerebellum tissue lysates (35ug/lane). RIPK3 (arrow) was detected using the purified Pab.



Western blot analysis of lysates from HT-29, K562 cell line (from left to right), using RIPK3 Antibody Center(Cat. #AP7184b). AP7184b was diluted at 1:1000 at each lane. A goat anti-rabbit IgG H&L(HRP) at 1:10000 dilution was used as the secondary antibody. Lysates at 35ug per lane.



Formalin-fixed and paraffin-embedded human lung carcinoma tissue reacted with RIP3 (RIPK3) antibody (Center) (Cat.# AP7184b), 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.

RIPK3 Antibody (Center) - Background

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.

RIPK3 Antibody (Center) - References

Yu P.W., Huang B.C.B., Shen M., Quast J., Chan E., Xu X., Nolan G.P., Payan D.G., Luo Y. *Curr. Biol.* 9:539-542(1999).

Sun X., Lee J., Navas T., Baldwin D.T., Stewart T.A., Dixit V.M.; *J. Biol. Chem.* 274:16871-16875(1999).