

# 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: <u>29883609</u>, PubMed: <u>32657447</u>). 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: <u>19524513</u>, PubMed: <u>22265413</u>, PubMed: <u>22265414</u>, 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: <u>22265414</u>, PubMed: <u>22421439</u>, PubMed: <u>25316792</u>, 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 autoand 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

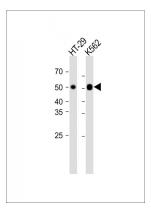
## **Background**

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'.