

**RIPK3 Antibody (C-term) Blocking Peptide**  
**Synthetic peptide**  
**Catalog # BP14554b****Specification**

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**RIPK3 Antibody (C-term) Blocking Peptide - Product Information**Primary Accession [Q9Y572](#)**RIPK3 Antibody (C-term) Blocking Peptide - 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**Format**

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

**Storage**

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

**Precautions**

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

**RIPK3 Antibody (C-term) Blocking Peptide - Protein Information**Name RIPK3 ([HGNC:10021](#))**Function**

Serine/threonine-protein kinase that activates necroptosis and apoptosis, two parallel forms of cell death (PubMed: [19524512](http://www.uniprot.org/citations/19524512)), PubMed: [19524513](http://www.uniprot.org/citations/19524513)), PubMed: [22265413](http://www.uniprot.org/citations/22265413)), PubMed: [22265414](http://www.uniprot.org/citations/22265414)), PubMed: [22421439](http://www.uniprot.org/citations/22421439)), PubMed: [29883609](http://www.uniprot.org/citations/29883609)), PubMed: [32657447](http://www.uniprot.org/citations/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](http://www.uniprot.org/citations/19524512)), PubMed: [19524513](http://www.uniprot.org/citations/19524513)), PubMed: [22265413](http://www.uniprot.org/citations/22265413)), PubMed: [22265414](http://www.uniprot.org/citations/22265414)), PubMed: [22421439](http://www.uniprot.org/citations/22421439)), PubMed: [29883609](http://www.uniprot.org/citations/29883609)), PubMed: [32298652](http://www.uniprot.org/citations/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](http://www.uniprot.org/citations/19524512), PubMed: [19524513](http://www.uniprot.org/citations/19524513), PubMed: [22265413](http://www.uniprot.org/citations/22265413), PubMed: [22265414](http://www.uniprot.org/citations/22265414), PubMed: [22421439](http://www.uniprot.org/citations/22421439), PubMed: [25316792](http://www.uniprot.org/citations/25316792), PubMed: [29883609](http://www.uniprot.org/citations/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](http://www.uniprot.org/citations/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](http://www.uniprot.org/citations/19498109)). These metabolic enzymes may eventually stimulate the tricarboxylic acid cycle and oxidative phosphorylation, which could result in enhanced ROS production (PubMed: [19498109](http://www.uniprot.org/citations/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 (C-term) Blocking Peptide - Protocols

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

- [Blocking Peptides](#)

### RIPK3 Antibody (C-term) Blocking Peptide - Images

### RIPK3 Antibody (C-term) Blocking Peptide - 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-1 signaling complex, and can induce apoptosis and weakly activate the NF-kappaB transcription factor.

**RIPK3 Antibody (C-term) Blocking Peptide - References**

Jugessur, A., et al. PLoS ONE 5 (7), E11493 (2010) :Vandenabeele, P., et al. Sci Signal 3 (115), RE4 (2010) :Cho, Y.S., et al. Cell 137(6):1112-1123(2009)He, S., et al. Cell 137(6):1100-1111(2009)Goyenechea, E., et al. J Nutrigenet Nutrigenomics 2(2):78-84(2009)