

**RIPK1 Antibody (C-term) Blocking Peptide**  
Synthetic peptide  
Catalog # BP7817b**Specification****RIPK1 Antibody (C-term) Blocking Peptide - Product Information**

Primary Accession [Q13546](#)  
Other Accession [NP\\_003795](#)

**RIPK1 Antibody (C-term) Blocking Peptide - Additional Information**

**Gene ID** 8737

**Other Names**

Receptor-interacting  
serine/threonine-protein kinase 1, Cell  
death protein RIP, Receptor-interacting  
protein 1, RIP-1, Serine/threonine-protein  
kinase RIP, RIPK1, RIP, RIP1

**Target/Specificity**

The synthetic peptide sequence used to generate the antibody [<a href=/product/products/AP7817b>AP7817b</a>](#) was selected from the C-term region of human RIPK1 . A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

**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.

**RIPK1 Antibody (C-term) Blocking Peptide - Protein Information****RIPK1 Antibody (C-term) Blocking Peptide - Background**

RIPK1 is a serine/threonine kinase that promotes apoptosis and activation of NF-kappa-B. This protein is required for TNFRSF1A mediated activation of NF-kappa-B. It is proteolytically cleaved by caspase-8 during TNF-induced apoptosis. Cleavage abolishes NF-kappa-B activation and enhances pro-apoptotic signaling through the TRADD-FADD interaction.

**RIPK1 Antibody (C-term) Blocking Peptide - References**

Hsu, H., et al., Immunity 4(4):387-396 (1996). Stanger, B.Z., et al., Cell 81(4):513-523 (1995).

**Name** RIPK1 ([HGNC:10019](#))

**Function**

Serine-threonine kinase which is a key regulator of TNF- mediated apoptosis, necroptosis and inflammatory pathways (PubMed:<a href="http://www.uniprot.org/citations/31827280"

target="\_blank">31827280</a>, PubMed:<a href="http://www.uniprot.org/citations/31827281"

target="\_blank">31827281</a>). Exhibits kinase activity-dependent functions that regulate cell death and kinase-independent scaffold functions regulating inflammatory signaling and cell survival (PubMed:<a href="http://www.uniprot.org/citations/11101870" target="\_blank">11101870</a>,"

PubMed:<a href="http://www.uniprot.org/citations/19524512"

target="\_blank">19524512</a>,"

PubMed:<a href="http://www.uniprot.org/citations/19524513"

target="\_blank">19524513</a>,"

PubMed:<a href="http://www.uniprot.org/citations/29440439"

target="\_blank">29440439</a>,"

PubMed:<a href="http://www.uniprot.org/citations/30988283"

target="\_blank">30988283</a>). Has kinase-independent scaffold functions: upon binding of TNF to TNFR1, RIPK1 is recruited to the TNF-R1 signaling complex (TNF-RSC also known as complex I) where it acts as a scaffold protein promoting cell survival, in part, by activating the canonical NF-kappa-B pathway (By similarity). Kinase activity is essential to regulate necroptosis and apoptosis, two parallel forms of cell death: upon activation of its protein kinase activity, regulates assembly of two death-inducing complexes, namely complex IIa (RIPK1-FADD-CASP8), which drives apoptosis, and the complex IIb (RIPK1-RIPK3-MLKL), which drives necroptosis (By similarity). RIPK1 is required to limit CASP8- dependent TNFR1-induced apoptosis (By similarity). In normal conditions, RIPK1 acts as an inhibitor of RIPK3-dependent necroptosis, a process mediated by RIPK3 component of complex IIb, which catalyzes phosphorylation of MLKL upon induction by ZBP1 (PubMed:<a href="http://www.uniprot.org/citations/19524512" target="\_blank">19524512</a>," PubMed:<a href="http://www.uniprot.org/citations/19524513"

target="\_blank">19524513</a>,  
PubMed:<a href="http://www.uniprot.org/citations/29440439"  
target="\_blank">29440439</a>,  
PubMed:<a href="http://www.uniprot.org/citations/30988283"  
target="\_blank">30988283</a>). Inhibits  
RIPK3- mediated necroptosis via  
FADD-mediated recruitment of CASP8,  
which cleaves RIPK1 and limits TNF-induced  
necroptosis (PubMed:<a href="http://www.uniprot.org/citations/19524512"  
target="\_blank">19524512</a>,  
PubMed:<a href="http://www.uniprot.org/citations/19524513"  
target="\_blank">19524513</a>,  
PubMed:<a href="http://www.uniprot.org/citations/29440439"  
target="\_blank">29440439</a>,  
PubMed:<a href="http://www.uniprot.org/citations/30988283"  
target="\_blank">30988283</a>). Required  
to inhibit apoptosis and necroptosis during  
embryonic development: acts by preventing  
the interaction of TRADD with FADD thereby  
limiting aberrant activation of CASP8 (By  
similarity). In addition to apoptosis and  
necroptosis, also involved in inflammatory  
response by promoting transcriptional  
production of pro-inflammatory cytokines,  
such as interleukin-6 (IL6) (PubMed:<a href="http://www.uniprot.org/citations/31827280"  
target="\_blank">31827280</a>,  
PubMed:<a href="http://www.uniprot.org/citations/31827281"  
target="\_blank">31827281</a>).  
Phosphorylates RIPK3: RIPK1 and RIPK3  
undergo reciprocal auto- and trans-  
phosphorylation (PubMed:<a href="http://www.uniprot.org/citations/19524513"  
target="\_blank">19524513</a>).  
Phosphorylates DAB2IP at 'Ser-728' in a  
TNF-alpha-dependent manner, and thereby  
activates the MAP3K5-JNK apoptotic  
cascade (PubMed:<a href="http://www.uniprot.org/citations/17389591"  
target="\_blank">17389591</a>,  
PubMed:<a href="http://www.uniprot.org/citations/15310755"  
target="\_blank">15310755</a>). Required  
for ZBP1-induced NF-kappa-B activation in  
response to DNA damage (By similarity).

### Cellular Location

Cytoplasm  
{ECO:0000250|UniProtKB:Q60855}. Cell  
membrane

{ECO:0000250|UniProtKB:Q9ZUF4}

### **RIPK1 Antibody (C-term) Blocking Peptide - Protocols**

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

- [Blocking Peptides](#)