

RIP Blocking Peptide
Catalog # PBV10119b**Specification**

RIP Blocking Peptide - Product Information

Primary Accession	Q13546
Gene ID	8737
Calculated MW	75931

RIP Blocking Peptide - Additional Information**Gene ID** 8737**Application & Usage**

The peptide is used for blocking the antibody activity of RIP. It usually blocks the antibody activity completely in Western blot analysis by incubating the peptide with equal volume of antibody for 30-60 minutes at 37°C.

Other Names

Receptor-interacting serine/threonine-protein kinase 1, 2.7.11.1, Cell death protein RIP, Receptor-interacting protein 1, RIP-1, Serine/threonine-protein kinase RIP, RIPK1, RIP, RIP1

Target/Specificity

RIP

Formulation

50 µg (0.5 mg/ml) in phosphate buffered saline (PBS), pH 7.2, containing 50% glycerol, 1% BSA and 0.02% thimerosal.

Reconstitution & Storage

-20 °C

Background Descriptions**Precautions**

RIP Blocking Peptide is for research use only and not for use in diagnostic or therapeutic procedures.

RIP Blocking Peptide - Protein Information**Name** RIPK1 ([HGNC:10019](#))**Function**

Serine-threonine kinase which is a key regulator of TNF- mediated apoptosis, necroptosis and inflammatory pathways (PubMed:32657447, PubMed:<a href="http://www.uniprot.org/citations/31827280"

target="_blank">31827280, PubMed:31827281, PubMed:17703191, PubMed:24144979). Exhibits kinase activity-dependent functions that regulate cell death and kinase-independent scaffold functions regulating inflammatory signaling and cell survival (PubMed:11101870, PubMed:19524512, PubMed:19524513, PubMed:29440439, PubMed:30988283). 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:19524512, PubMed:19524513, PubMed:29440439, PubMed:30988283). Inhibits RIPK3-mediated necroptosis via FADD-mediated recruitment of CASP8, which cleaves RIPK1 and limits TNF-induced necroptosis (PubMed:19524512, PubMed:19524513, PubMed:29440439, PubMed:30988283). 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:31827280, PubMed:31827281). Phosphorylates RIPK3: RIPK1 and RIPK3 undergo reciprocal auto- and trans-phosphorylation (PubMed:19524513). Phosphorylates DAB2IP at 'Ser-728' in a TNF-alpha- dependent manner, and thereby activates the MAP3K5-JNK apoptotic cascade (PubMed:17389591, PubMed:15310755). 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}

RIP Blocking Peptide - 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](#)

RIP Blocking Peptide - Images