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RIPK1 Antibody (N-term) Blocking peptide
Synthetic peptide
Catalog # BP13893a

Specification

RIPK1 Antibody (N-term) Blocking peptide - Product Information

Primary Accession Q13546

RIPK1 Antibody (N-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 AP13893a was selected from the N-term region of 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 (N-term) 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: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:<a href="http://www.uniprot.org/citations/29440439"

target="_blank">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}

RIPK1 Antibody (N-term) Blocking peptide - Protocols

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

- Blocking Peptides

RIPK1 Antibody (N-term) Blocking peptide - Images

RIPK1 Antibody (N-term) Blocking peptide - Background

Essential adapter molecule for the activation of NF-kappa-B. Following different upstream signals (binding of inflammatory cytokines, stimulation of pathogen recognition receptors, or DNA damage), particular RIPK1-containing complexes are formed, initiating a limited number of cellular responses. Upon TNFA stimulation RIPK1 is recruited to a TRADD-TRAF complex initiated by TNFR1 trimerization. There, it is ubiquitinated via 'Lys-63'-link chains, inducing its association with the IKK complex, and its activation through NEMO binding of polyubiquitin chains.

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Kim, S., et al. Cancer Sci. 101(11):2425-2429(2010)Bailey, S.D., et al. Diabetes Care 33(10):2250-2253(2010)Chae, Y.S., et al. J. Cancer Res. Clin. Oncol. (2010) In press :Couch, F.J., et al. Cancer Epidemiol. Biomarkers Prev. 19(1):251-257(2010)Vandenabeele, P., et al. Sci Signal 3 (115), RE4 (2010) :