

Mouse Ripk1 Antibody (Center) Blocking Peptide
Synthetic peptide
Catalog # BP17439c**Specification**

Mouse Ripk1 Antibody (Center) Blocking Peptide - Product InformationPrimary Accession [Q60855](#)**Mouse Ripk1 Antibody (Center) Blocking Peptide - Additional Information****Gene ID** 19766**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, Rinp, Rip

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.

Mouse Ripk1 Antibody (Center) Blocking Peptide - Protein Information**Name** Ripk1 {ECO:0000312|MGI:MGI:108212}**Function**

Serine-threonine kinase which is a key regulator of TNF- mediated apoptosis, necroptosis and inflammatory pathways (PubMed:24813849, PubMed:24813850, PubMed:24557836, PubMed:27819681, PubMed:28842570, PubMed:31511692, PubMed:31827280, PubMed:31827281, PubMed:33397971). Exhibits kinase activity-dependent functions that regulate cell death and kinase-independent scaffold functions regulating inflammatory signaling and cell survival (PubMed:24813849, PubMed:24813850, PubMed:24557836, PubMed:28842570, PubMed:31519886).

target="_blank">31519886, PubMed:31519887). 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 (PubMed:31519886, PubMed:31519887). 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 (PubMed:28842570, PubMed:27819681, PubMed:27819682, PubMed:29440439, PubMed:30988283, PubMed:31519886, PubMed:31519887). RIPK1 is required to limit CASP8- dependent TNFR1-induced apoptosis (PubMed:24813849, PubMed:24813850, PubMed:24557836). 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:24557836, PubMed:27819681, PubMed:27819682, PubMed:31358656). Inhibits RIPK3-mediated necroptosis via FADD-mediated recruitment of CASP8, which cleaves RIPK1 and limits TNF-induced necroptosis (PubMed:31358656). Required to inhibit apoptosis and necroptosis during embryonic development: acts by preventing the interaction of TRADD with FADD thereby limiting aberrant activation of CASP8 (PubMed:30867408, PubMed:30185824). 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 (By similarity). Phosphorylates DAB2IP at 'Ser-728' in a TNF-alpha-dependent manner, and thereby activates the MAP3K5-JNK apoptotic cascade (By similarity). Required for ZBP1-induced NF-kappa-B activation in response to DNA damage (PubMed:12654725, PubMed:19590578).

Cellular Location

Cytoplasm. Cell membrane {ECO:0000250|UniProtKB:Q9ZUF4}

Tissue Location

Found at low levels in all tissues.

Mouse Ripk1 Antibody (Center) Blocking Peptide - Protocols

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

- [Blocking Peptides](#)

Mouse Ripk1 Antibody (Center) Blocking Peptide - Images**Mouse Ripk1 Antibody (Center) 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.

Mouse Ripk1 Antibody (Center) Blocking Peptide - References

Wong, W.W., et al. Cell Death Differ. 17(3):482-487(2010)Kamarajan, P., et al. Mol. Biol. Cell 21(3):481-488(2010)Xu, G., et al. J. Biol. Chem. 285(2):969-978(2010)Haas, T.L., et al. Mol. Cell 36(5):831-844(2009)Chang, M., et al. Nat. Immunol. 10(10):1089-1095(2009)