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RIP Rabbit mAb

Catalog # AP76878

Product Information

Application	WB
Primary Accession	Q13546
Reactivity	Human
Host	Rabbit
Clonality	Monoclonal Antibody
Isotype	IgG
Conjugate	Unconjugated
Purification	Affinity Purified
Calculated MW	75931

Additional Information

Gene ID	8737
Other Names	RIPK1
Dilution	WB~~1:1000
Format	Liquid in 50mM Tris-Glycine(pH 7.4), 0.15M NaCl, 40%Glycerol, 0.01% sodium azide and 0.05% BSA.
Storage	Store at 4°C short term. Aliquot and store at -20°C long term. Avoid freeze/thaw cycles.

Protein Information

Name	RIPK1 (HGNC:10019)
Function	Serine-threonine kinase which is a key regulator of TNF- mediated apoptosis, necroptosis and inflammatory pathways (PubMed:17703191, PubMed:24144979, PubMed:31827280, PubMed:31827281, PubMed:32657447, PubMed:35831301). 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-dependent manner, and thereby activates the MAP3K5-JNK apoptotic cascade (PubMed:15310755, PubMed:17389591). 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}

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.