

Product Name: RIP (16M11) Rabbit Monoclonal Antibody
Catalog #: AMRe17204

Summary

Production Name	RIP (16M11) Rabbit Monoclonal Antibody
Description	Rabbit Monoclonal Antibody
Host	Rabbit
Application	WB
Reactivity	Human

Performance

Conjugation	Unconjugated
Modification	Unmodified
Isotype	IgG
Clonality	Monoclonal
Form	Liquid
Storage	Store at 4°C short term. Aliquot and store at -20°C long term. Avoid freeze/thaw cycles.
Buffer	Supplied in 50mM Tris-Glycine(pH 7.4), 0.15M NaCl, 40%Glycerol, 0.01% New type preservative N and 0.05% BSA.
Purification	Affinity purification

Immunogen

Gene Name	RIPK1
Alternative Names	RIPK1; Cell death protein RIP; RIP1; RIP; RIP-1; Rinp;
Gene ID	8737.0
SwissProt ID	Q13546.Recombinant protein of human RIP

Application

Dilution Ratio	WB: 1:1000
Molecular Weight	76kDa

Background

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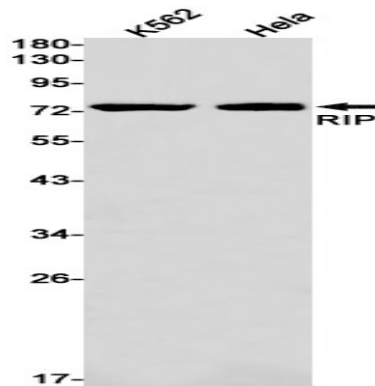
Essential adapter molecule for the activation of NF-kappa-B. Following different upstream signals (binding of inflammatory cytokines, stimulation of pathogen recognition receptors, or DNA damage), particular RIPK1-containing complexes are formed, initiating a limited number of cellular responses. Serine-threonine kinase which is a key regulator of TNF- mediated apoptosis, necroptosis and inflammatory pathways (PubMed:31827280, PubMed:31827281). 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).

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target="_blank">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).

Research Area

Image Data



Western blot detection of RIP in K562,HeLa cell lysates using RIP antibody(1:1000 diluted).

Note

For research use only.