Datasheet for ABIN967985

anti-RIPK1 antibody (AA 385-650)

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Overview

Quantity:	150 µg
Target:	RIPK1
Binding Specificity:	AA 385-650
Reactivity:	Human, Mouse, Rat, Dog, Chicken
Host:	Mouse
Clonality:	Monoclonal
Conjugate:	This RIPK1 antibody is un-conjugated
Application:	Western Blotting (WB), Immunohistochemistry (IHC), Immunoprecipitation (IP), Immunofluorescence (IF)

Product Details

Immunogen:	Human RIP aa. 385-650
Clone:	38-RIP
Isotype:	IgG2a
Cross-Reactivity:	Mouse (Murine), Rat (Rattus), Dog (Canine), Chicken
Characteristics:	1. Since applications vary, each investigator should titrate the reagent to obtain optimal results. 2. Please refer to us for technical protocols. 3. Caution: Sodium azide yields highly toxic hydrazoic acid under acidic conditions. Dilute azide compounds in running water before discarding to avoid accumulation of potentially explosive deposits in plumbing. 4. Source of all serum proteins is from USDA inspected abattoirs located in the United States.

Product Details

Purification:	The monoclonal antibody was purified from tissue culture supernatant or ascites by affinity chromatography.

Target Details

Target:	RIPK1
Alternative Name:	RIP (RIPK1 Products)
Background:	Binding or cross linking of the Fas antigen (also known as APO-1 and CD95) is known to elicit apoptosis in susceptible cells. Fas is a member of a family of cell surface receptors which includes tumor necrosis factor receptors (TNF-R, and TNF-R2) and nerve growth factor receptors (NGF-R), CD40, OX40, CD30, CD27, and 4-1BB. Several members of this family have been shown to regulate or induce cell death (TNF-R1 and TNF-R2). A 74 kDa member of this family protein named RIP (Receptor Interacting Protein) contains an N-terminal region with homology to protein kinases and a C-terminal region containing a cytoplasmic death domain present in both Fas and TNF-R1. Both Fas and RIP have been shown to require this death domain to induce apoptosis and overexpression of RIP has been shown to induce cell death in transfected cells. Synonyms: Receptor Interacting Protein
Molecular Weight:	74 kDa
Pathways:	NF-kappaB Signaling, Apoptosis, Caspase Cascade in Apoptosis, TLR Signaling, Activation of Innate immune Response, Inositol Metabolic Process, Positive Regulation of Endopeptidase Activity, Hepatitis C, Protein targeting to Nucleus, Toll-Like Receptors Cascades, Negative Regulation of intrinsic apoptotic Signaling, SARS-CoV-2 Protein Interactome, Ubiquitin Proteasome Pathway

Application Details

Comment:	Related Products: ABIN968536, ABIN967389
Restrictions:	For Research Use only

Handling

Format:	Liquid
Concentration:	250 µg/mL
Buffer:	Aqueous buffered solution containing BSA, glycerol, and ≤0.09 % sodium azide.

Handling

Preservative:	Sodium azide
Precaution of Use:	This product contains Sodium azide: a POISONOUS AND HAZARDOUS SUBSTANCE which should be handled by trained staff only.
Storage:	-20 °C
Storage Comment:	Store undiluted at -20° C.

Publications

Product cited in:

Devin, Lin, Yamaoka, Li, Karin, Liu Zg: "The alpha and beta subunits of IkappaB kinase (IKK) mediate TRAF2-dependent IKK recruitment to tumor necrosis factor (TNF) receptor 1 in response to TNF." in: **Molecular and cellular biology**, Vol. 21, Issue 12, pp. 3986-94, (2001) (PubMed).

Fulda, Meyer, Debatin: "Metabolic inhibitors sensitize for CD95 (APO-1/Fas)-induced apoptosis by down-regulating Fas-associated death domain-like interleukin 1-converting enzyme inhibitory protein expression." in: **Cancer research**, Vol. 60, Issue 14, pp. 3947-56, (2000) (PubMed).

Lewis, Devin, Miller, Lin, Rodriguez, Neckers, Liu: "Disruption of hsp90 function results in degradation of the death domain kinase, receptor-interacting protein (RIP), and blockage of tumor necrosis factor-induced nuclear factor-kappaB activation." in: **The Journal of biological chemistry**, Vol. 275, Issue 14, pp. 10519-26, (2000) (PubMed).

Stanger, Leder, Lee, Kim, Seed: "RIP: a novel protein containing a death domain that interacts with Fas/APO-1 (CD95) in yeast and causes cell death." in: **Cell**, Vol. 81, Issue 4, pp. 513-23, (1995) (PubMed).

Takahashi, Tanaka, Brannan, Jenkins, Copeland, Suda, Nagata: "Generalized lymphoproliferative disease in mice, caused by a point mutation in the Fas ligand." in: **Cell**, Vol. 76, Issue 6, pp. 969-76, (1994) (PubMed).

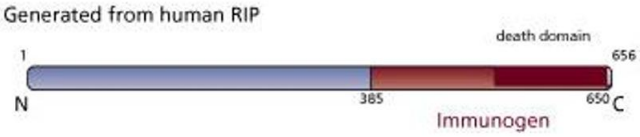
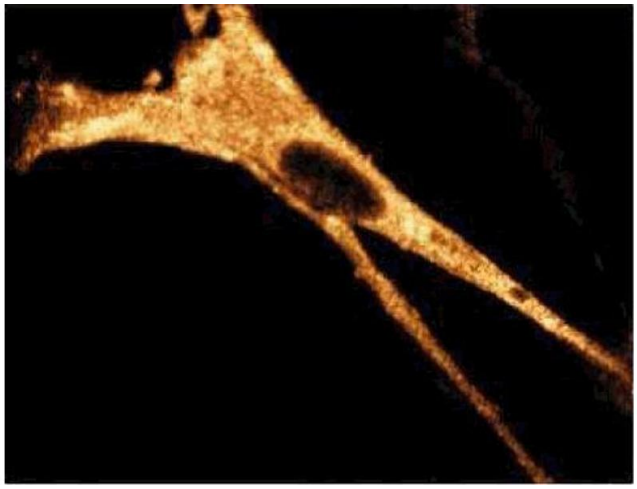


Image 1.

Western Blotting

Image 2. Western blot analysis of RIP on a human endothelial cell lysate. Lane 1: 1:1000, lane 2: 1:2000, lane 3: 1:4000 dilution of the mouse anti-RIP antibody.



Immunofluorescence

Image 3. Immunofluorescence staining of WI-38 cells (Human lung fibroblasts, ATCC CCL-75).