

RIP (C-12): sc-133102

BACKGROUND

In contrast to growth factors which promote cell proliferation, FAS ligand (FAS-L) and the tumor necrosis factors (TNFs) rapidly induce apoptosis. Cellular response to FAS-L and TNF is mediated by structurally related receptors containing a conserved "death domain" and belonging to the TNF receptor superfamily. TRADD, FADD and RIP are FAS/TNF-R1 interacting proteins that contain a death domain homologous region (DDH). TRADD (TNF-R1-associated death domain) and FADD (FAS-associated death domain) associate with the death domains of both FAS and TNF-R1 via their DDH regions. Overexpression of TRADD leads to NF κ B activation and apoptosis in the absence of TNF. Overexpression of FADD causes apoptosis, which can be blocked by the cow pox protein CrmA, suggesting that FADD lies upstream of ICE and possibly other serine proteases. The receptor interacting protein, RIP, associates with FAS exclusively via its DDH and this association is abrogated in *lpr* mutants. Unlike TRADD and FADD, RIP contains a putative amino terminal kinase domain.

CHROMOSOMAL LOCATION

Genetic locus: RIPK1 (human) mapping to 6p25.2; Ripk1 (mouse) mapping to 13 A3.3.

SOURCE

RIP (C-12) is a mouse monoclonal antibody raised against amino acids 465-671 mapping at the C-terminus of RIP (receptor interacting protein) of human origin.

PRODUCT

Each vial contains 200 μ g IgG $_1$ kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

RIP (C-12) is available conjugated to agarose (sc-133102 AC), 500 μ g/0.25 ml agarose in 1 ml, for IP; to HRP (sc-133102 HRP), 200 μ g/ml, for WB, IHC(P) and ELISA; to either phycoerythrin (sc-133102 PE), fluorescein (sc-133102 FITC), Alexa Fluor[®] 488 (sc-133102 AF488), Alexa Fluor[®] 546 (sc-133102 AF546), Alexa Fluor[®] 594 (sc-133102 AF594) or Alexa Fluor[®] 647 (sc-133102 AF647), 200 μ g/ml, for WB (RGB), IF, IHC(P) and FCM; and to either Alexa Fluor[®] 680 (sc-133102 AF680) or Alexa Fluor[®] 790 (sc-133102 AF790), 200 μ g/ml, for Near-Infrared (NIR) WB, IF and FCM.

APPLICATIONS

RIP (C-12) is recommended for detection of RIP of mouse and human origin by Western Blotting (starting dilution 1:100, dilution range 1:100-1:1000), immunoprecipitation [1-2 μ g per 100-500 μ g of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for RIP siRNA (h): sc-36426, RIP siRNA (m): sc-36427, RIP shRNA Plasmid (h): sc-36426-SH, RIP shRNA Plasmid (m): sc-36427-SH, RIP shRNA (h) Lentiviral Particles: sc-36426-V and RIP shRNA (m) Lentiviral Particles: sc-36427-V.

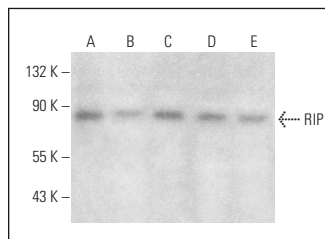
Molecular Weight of RIP: 74 kDa.

Positive Controls: T24 cell lysate: sc-2292 or 3T3-L1 cell lysate: sc-2243.

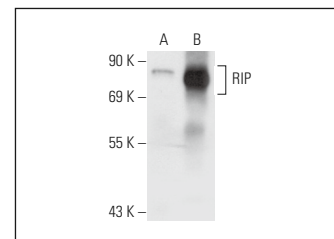
STORAGE

Store at 4° C, ****DO NOT FREEZE****. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

DATA



RIP (C-12): sc-133102. Western blot analysis of RIP expression in 3T3-L1 (A), K-562 (B), SK-N-MC (C), MOLT-4 (D) and Raji (E) whole cell lysates.



RIP (C-12): sc-133102. Western blot analysis of RIP expression in JEG-3 (A) and T24 (B) whole cell lysates.

SELECT PRODUCT CITATIONS

- Buck, M. and Chojkier, M. 2011. C/EBP β -Thr217 phosphorylation signaling contributes to the development of lung injury and fibrosis in mice. *PLoS ONE* 6: e25497.
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- Weiss, R., et al. 2013. IL-24 sensitizes tumor cells to TLR3-mediated apoptosis. *Cell Death Differ.* 20: 823-833.
- Yurdagül, A., et al. 2016. Oxidized LDL induces FAK-dependent RSK signaling to drive NF κ B activation and VCAM-1 expression. *J. Cell Sci.* 129: 1580-1591.
- Cho, E., et al. 2018. Antitumor activity of HPA3P through RIPK3-dependent regulated necrotic cell death in colon cancer. *Oncotarget* 9: 7902-7917.
- Tripathi, D., et al. 2018. Alcohol enhances type 1 interferon- α production and mortality in young mice infected with *Mycobacterium tuberculosis*. *PLoS Pathog.* 14: e1007174.
- Corsetti, G., et al. 2019. Autophagy and oncosis/necroptosis are enhanced in cardiomyocytes from heart failure patients. *Med. Sci. Monit. Basic Res.* 25: 33-44.
- Gaba, A., et al. 2019. The NS1 protein of Influenza A Virus participates in necroptosis by interacting with MLKL and increasing its oligomerization and membrane translocation. *J. Virol.* 93: e01835-18.
- Yu, W.N., et al. 2019. Citronellol induces necroptosis of human lung cancer cells via TNF- α pathway and reactive oxygen species accumulation. *In Vivo* 33: 1193-1201.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

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