SANTA CRUZ BIOTECHNOLOGY, INC.

RIP (H-207): sc-7881

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

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 NFκB activation and apoptosis in the absence of TNF. Overexpression of FADD causes apoptosis, which can be blocked by the bovine 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 (H-207) is a rabbit polyclonal 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 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

APPLICATIONS

RIP (H-207) is recommended for detection of RIP of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500), immunohistochemistry (including paraffin-embedded sections) (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: HeLa whole cell lysate: sc-2200, T24 cell lysate: sc-2292 or JEG-3 whole cell lysate: sc-364255.

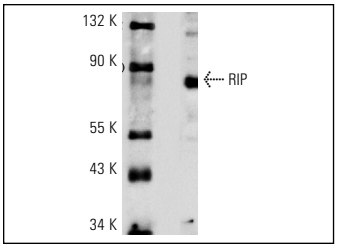
STORAGE

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

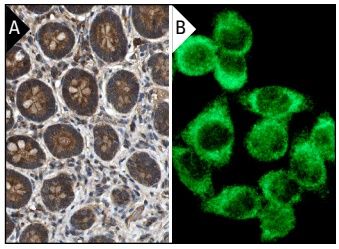
RESEARCH USE

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

DATA



RIP (H-207): sc-7881. Western blot analysis of RIP expression in HeLa whole cell lysate.



RIP (H-207): sc-7881. Immunoperoxidase staining of formalin fixed, paraffin-embedded human small intestine tissue showing cytoplasmic staining of glandular cells magnification. Kindly provided by The Swedish Human Protein Atlas (HPA) program (**A**). Immunofluorescence staining of methanol-fixed HeLa cells showing cytoplasmic staining (**B**).

SELECT PRODUCT CITATIONS

1. Guiet, C., et al. 2000. Caspase recruitment domain (CARD)-dependent cytoplasmic filaments mediate Bcl10-induced NFκB activation. J. Cell Biol. 148: 1131-1140.
2. Zorde-Khvalevsky, E., et al. 2009. Toll-like receptor 3 signaling attenuates liver regeneration. Hepatology 50: 198-206.
3. Zhang, P., et al. 2010. Targeting a novel N-terminal epitope of death receptor 5 triggers tumor cell death. J. Biol. Chem. 285: 8953-8966.
4. Ndour, P.A., et al. 2010. Inhibition of tumor necrosis factor-induced phenotypes by short intracellular versions of latent membrane protein-1. Cell. Signal. 22: 303-313.
5. Knox, P.G., et al. 2011. The death domain kinase RIP1 links the immunoregulatory CD40 receptor to apoptotic signaling in carcinomas. J. Cell Biol. 192: 391-399.
6. Guan, Y.J., et al. 2011. Phospho-SXXE/D motif mediated TNF receptor 1-TRADD death domain complex formation for T cell activation and migration. J. Immunol. 187: 1289-1297.
7. Ndour, P.A., et al. 2012. Inhibition of latent membrane protein 1 impairs the growth and tumorigenesis of latency II Epstein-Barr virus-transformed T cells. J. Virol. 86: 3934-3943.
8. Inomata, M., et al. 2012. Regulation of Toll-like receptor signaling by NDP52-mediated selective autophagy is normally inactivated by A20. Cell. Mol. Life Sci. 69: 963-979.