RIP3 polyclonal antibody

A member in the RIP kinase family was identified and designated RIP3 (Receptor-interacting serine/threonine protein kinase-3). RIP3 contains an N-terminal kinase domain but, unlike RIP or RIP2, lacks the C-terminal death or CARD domain. RIP3 binds to RIP and TNF-R1, mediates TNF-R1 induced apoptosis, and attenuates RIP and TNF-R1 induced NF-κB activation.

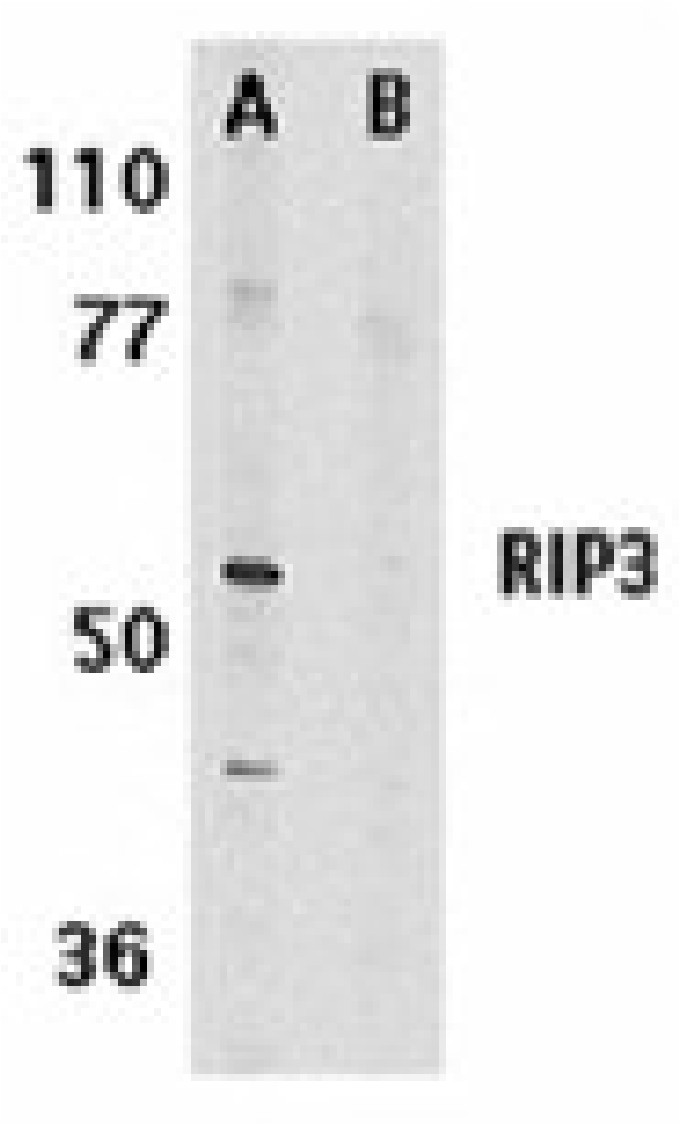
This antibody is covered by our Worry-Free Guarantee.

Citations: 40 View Online »

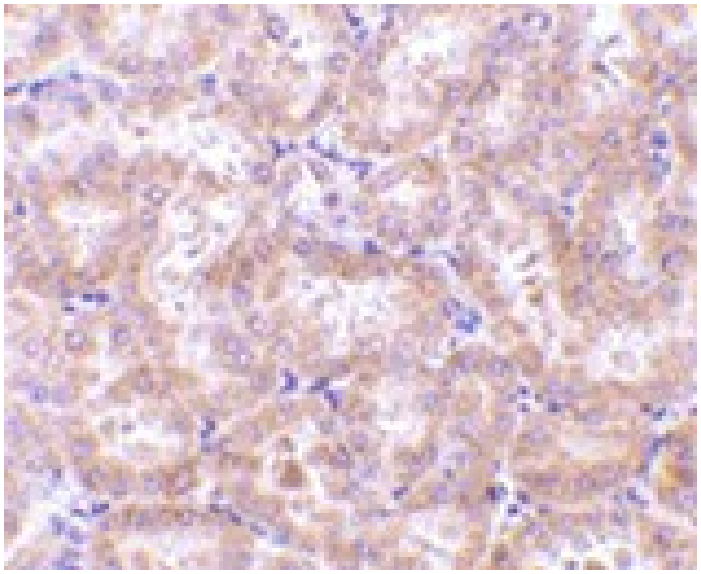
Ordering Information Order Online »

ADI-905-242-100	100µg

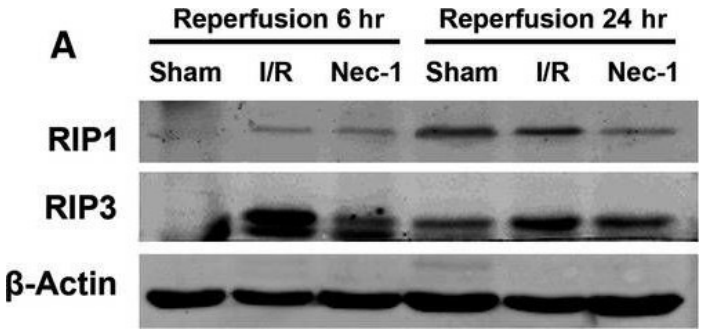
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Western blot analysis of RIP3 in mouse 3T3 whole cell lysate in the absence (A) or presence (B) of blocking peptide with RIP3 antibody at 1µg/ml.

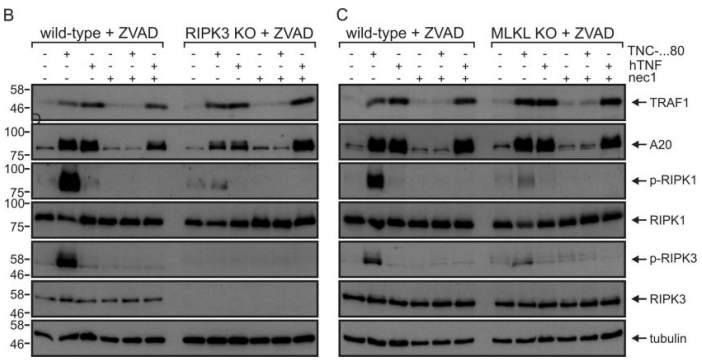


Immunohistochemistry analysis of RIP3 in rat kidney tissue with RIP3 antibody at 5µg/ml.



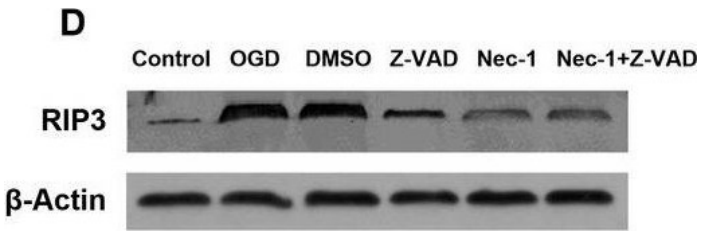
Necrostatin-1 inhibits necroptosis-related protein expressions and protects the intestine independent of apoptosis in vivo. (A and B) Western blot and quantification showed increased RIP1 and RIP3 protein expression levels after intestinal I/R. (C and D) Western blot and quantification showed increased MLKL protein expression levels after intestinal I/R. (E and F) MLKL recruitment to RIP1 was significantly decreased after Nec-1 treatment. (G and H) Pre-treatment with Nec-1 did not affect caspase-3 cleavage. The data are shown as the means ± S.D. (n = 8 per group). *P < 0.05, **P < 0.01 compared with sham group; #P < 0.05, ##P < 0.01 compared with I/R group.

Image collected and cropped by CiteAb under a CC-BY license from the following publication: Necroptosis is a key mediator of enterocytes loss in intestinal ischaemia/reperfusion injury. *J Cell Mol Med* (2017)



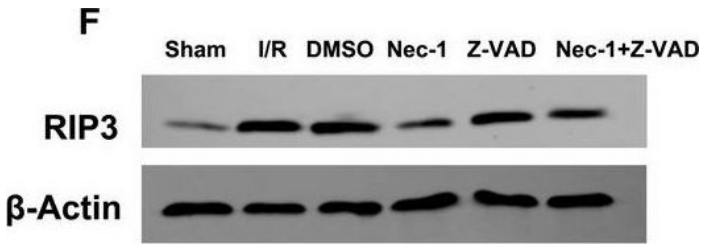
TNFR2-induced upregulation of A20 and TRAF1 is not an epiphenomenon of TNFR2-triggered necroptosis.a Wild-type, RIPK3-, and MLKL-knockout macrophages were stimulated as indicated with human TNF (100 ng/ml), TNC-sc(mu)TNF80 (200 ng/ml), and ZVAD (20 μM). Cell viability was quantified after 36 h using MTT. Data points of five independent experiments were shown with mean ± SEM (***p < 0.001). b, c RIPK3- (b) and MLKL- (c) knockout macrophages along with wild-type macrophages were treated overnight with the indicated combinations of 200 ng/ml TNC-sc(mu)TNF80, 100 ng/ml human TNF, 20 μM ZVAD, and necrostatin-1 (45 μM). Total cell lysates were prepared and analyzed by western blotting. d Wild-type and TNFR2-knockout macrophages were stimulated for 7 h with human TNF (100 ng/ml) or TNC-sc(mu)TNF80 (200 ng/ml) in the presence and absence of ZVAD (20 μM), and total cell lysates were analyzed by western blotting for RIPK1 phosphorylation. e The various macrophage types were stimulated for 36 h with the indicated mixtures of TNC-sc(mu)TNF80 (200 ng/ml), ZVAD (20 μM), and necrostatin-1 (45 μM). IL-1β in the supernatants was determined by ELISA assay. Shown are the mean ± SEM of five or six independent experiments. ***p < 0.001

Image collected and cropped by CiteAb under a CC-BY license from the following publication: TNFR2 unlocks a RIPK1 kinase activity-dependent mode of proinflammatory TNFR1 signaling. *Cell Death Dis* (2018)



Necrostatin-1 decreases IEC-6 cell death and pro-inflammatory cytokine gene expression after OGD in vitro. Cultured IEC-6 cell injury was induced by depriving culture media of oxygen and glucose (OGD). (A) Viability after different time courses of OGD. (B) Immunofluorescence for TUNEL staining (green, bar denotes 20 μm) and the quantification of TUNEL-positive cells per ×20 field in IEC-6 cells (C). (D and E) Western blot and quantification show that RIP3 proteins were expressed at a higher level in the OGD, DMSO and Z-VAD groups but were attenuated after Nec-1 treatment. (F and G) Q-PCR for TNF-α and IL-1β mRNA levels in OGD-challenged IEC-6 cells after Z-VAD and Nec-1 treatment. The data are shown as the means ± S.D. (n = 6 per group). *P < 0.05, **P < 0.01 compared with control group, #P < 0.05, ##P < 0.01 compared with OGD group and DMSO group, δP < 0.05 compared with Z-VAD group and Nec-1 group.

Image collected and cropped by CiteAb under a CC-BY license from the following publication: Necroptosis is a key mediator of enterocytes loss in intestinal ischaemia/reperfusion injury. *J Cell Mol Med* (2017)



Increased protection from intestinal I/R injury by the combined blockade of necroptosis and apoptosis after 1 hr of ischaemia/24 hrs of reperfusion in vivo. (A) Histopathologic changes of the intestinal mucosa. Haematoxylin and eosin stained small intestine. Magnification is ×200, bar denotes 100 mm. (B) Injury scores of the intestinal mucosa morphology. (C) Intestinal cellular injury was evaluated by serum DAO activity. (D and E) Z-VAD with/without Nec-1 treatment decreased the caspase-3 cleavage. (F and G) Treatment with Z-VAD alone had no effect on RIP3 up-regulation. Caspase inhibition shifted intestinal I/R-induced epithelial cell death from apoptosis to necroptosis. The images are representative for each group. The data are shown as the means ± S.D. (n = 8 per group). *P < 0.05, **P < 0.01 compared with sham group, ##P < 0.01 compared with I/R group and DMSO group, δP < 0.05 compared with Nec-1 group and Z-VAD group.

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Handling & Storage

Short Term Storage	+4°C
Long Term Storage	-20°C
Shipping	Blue Ice

Regulatory Status RUO - Research Use Only

Product Details

Alternative Name	Receptor-interacting serine/threonine protein kinase 3, RIP-like protein kinase 3, Receptor-interacting protein 3
Application	IHC (PS), WB
Application Notes	Detects a band of ~57kDa by Western blot.
Formulation	Liquid. In PBS containing 0.02% sodium azide.
Host	Rabbit
Immunogen	Synthetic peptide corresponding to a portion of mouse RIP3.
Purity Detail	Peptide affinity purified.
Recommendation Dilutions/Conditions	Immunohistochemistry (paraffin sections, 5µg/ml)Western Blot (0.5-1.0µg/ml)Suggested dilutions/conditions may not be available for all applications.Optimal conditions must be determined individually for each application.
Species Reactivity	Mouse, Rat
UniProt ID	Q9QZL0
Worry-free Guarantee	**This antibody is covered by our Worry-Free Guarantee .**

Last modified: May 29, 2024

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