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BOSTER BIOLOGICAL TECHNOLOGY
3942 B Valley Ave, Pleasanton, CA 94566
888-466-3604 | support@bosterbio.com | www.bosterbio.com

Anti-RIP3/RIPK3 Antibody Picoband®

Catalog Number: PA2242

About RIPK3

Receptor-interacting serine/threonine-protein kinase 3 (RIPK3), also known as RIP3 is an enzyme that in humans is encoded by the RIPK3 gene. This gene is mapped to 14q12. The product of this gene is a member of the receptor-interacting protein (RIP) family of serine/threonine protein kinases, and contains a C-terminal domain unique from other RIP family members. The encoded protein is predominantly localized to the cytoplasm, and can undergo nucleocytoplasmic shuttling dependent on novel nuclear localization and export signals. It is a component of the tumor necrosis factor (TNF) receptor-I signaling complex, and can induce apoptosis and weakly activate the NF-kappaB transcription factor.

Overview

Product Name	Anti-RIP3/RIPK3 Antibody Picoband®
Reactive Species	Human
Description	Boster Bio Anti-RIP3/RIPK3 Antibody catalog # PA2242. Tested in WB applications. This antibody reacts with Human. The brand Picoband indicates this is a premium antibody that guarantees superior quality, high affinity, and strong signals with minimal background in Western blot applications. Only our best-performing antibodies are designated as Picoband, ensuring unmatched performance.
Application	WB
Clonality	Polyclonal
Formulation	Each vial contains antibody formulated with stabilizing components, 0.9mg NaCl, 0.2mg Na2HPO4, 0.05mg Thimerosal, 0.05mg NaN3. *This antibody is supplied in a stabilized formulation. Compatibility with conjugation reactions depends on the chemistry of the conjugation method used. For conjugation methods that are not compatible with the stabilizing components present in this formulation, a carrier-free antibody format is required.
Storage Instructions	Store at -20˚C for one year from date of receipt. After reconstitution, at 4˚C for one month. It can also be aliquotted and stored frozen at -20˚C for six months. Avoid repeated freeze-thaw cycles.
Host	Rabbit
Uniprot ID	Q9Y572

Technical Details

Immunogen	A synthetic peptide corresponding to a sequence at the N-terminus of human RIP3.
Recommended Detection Systems	Boster recommends Enhanced Chemiluminescent Kit with anti-Rabbit IgG (EK1002) for Western blot.
Cross Reactivity	No cross-reactivity with other proteins

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Isotype	Rabbit IgG
Form	Lyophilized
Concentration	Adding 0.2 ml of distilled water will yield a concentration of 500 ug/ml.
Purification	Immunogen affinity purified.
Suggested Dilutions	Western blot, 0.1-0.5ug/ml, Human

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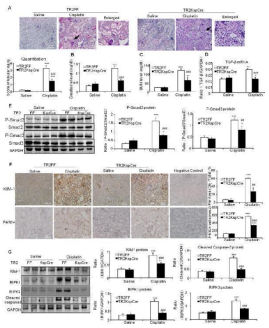
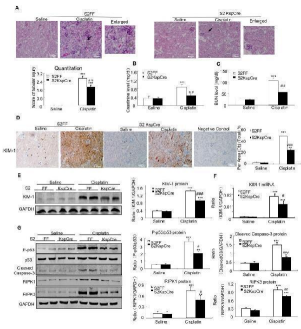
Anti-RIP3/RIPK3 Antibody Picoband® (PA2242) Images

Anti-RIP3 antibody, PA2242, Western blottingLane 1: PANC Cell LysateLane 2: SW620 Cell LysateLane 3: SKOV-3 Cell LysateLane 4: M231 Cell Lysate



Conditional knockout of TGF-betaRII prevented cisplatin-induced renal injury and apoptotic signaling in vivo . A: Periodic acid-Schiff (PAS) staining and quantitative analysis show conditional knockout of TGF-betaRII reduced renal injury in cisplatin nephropathy. B: Creatinine assay. C: BUN assay. Serum creatinine and BUN show conditional knockout of TGF-betaRII prevented decline of renal function in cisplatin nephropathy. D. Real-time PCR data show conditional knockout of TGF-betaRII reduced TGF-beta mRNA level in cisplatin-induced nephropathy. E. Western blot analysis of phospho-Smad2 and phospho-Smad3. F. Immunohistochemistry and quantitative data show conditional knockout of TGF-betaRII reduced KIM-1 protein and F4/80+ macrophages infiltration in cisplatin nephropathy. G. Western blot analysis of KIM-1, RIPK1, RIPK3, cleaved caspase-3. Data represent mean ± SEM for 6-8 mice. **P



Conditional knockout of Smad2 prevented cisplatin-induced renal injury, decline of renal function and attenuated signaling molecules regulating programmed cell death in vivo . A: PAS staining and quantitative analysis show conditional knockout of Smad2 reduced renal injury in cisplatin-induced AKI mice. B: Creatinine assay. C: BUN assay. Serum creatinine and BUN show conditional deletion of Smad2 prevented decline of renal function in cisplatin nephropathy. D: Immunohistochemistry and quantitative data show conditional knockout of Smad2 reduced KIM-1 in cisplatin-induced nephropathy. E and F: Western blot and Real-time PCR analysis of KIM-1. G: Western blot of P-p53, p53, RIPK1, RIPK3 and cleaved caspase-3. Data represent mean ± SEM for 6-8 mice. **P

Knockdown of Smad2 attenuates cisplatin-induced renal injury, inflammation response, and programmed cell death in established nephrotoxic AKI model. A: PAS staining and quantitative analysis show knockdown of Smad2 reduced renal injury in cisplatin nephropathy. B: Creatinine assay. C: BUN assay. Serum creatinine and BUN show knockdown of Smad2 prevented decline of renal function in cisplatin nephropathy. D: Real-time PCR data show knockdown of

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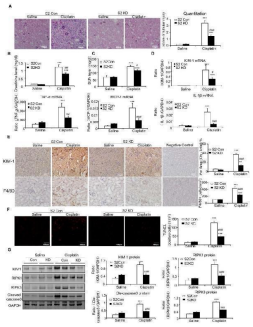
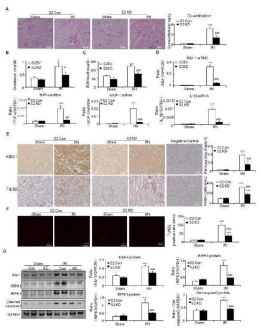


Smad2 reduced mRNA of TNF-alpha, IL-1beta, MCP-1 and KIM-1. E: Immunohistochemistry and quantitative data show knockdown of Smad2 reduced KIM-1 protein and F4/80+ macrophages in cisplatin-induced nephropathy. F: TUNEL assay. Knockdown of Smad2 reduced apoptosis in injured kidney. G: Western blot analysis of KIM-1, RIPK1, RIPK3, cleaved caspase-3. Data represent mean ± SEM for 6 mice. *P



Knockdown of Smad2 attenuates renal injury, inflammation response, and programmed cell death in established ischemic AKI model. A: PAS staining and quantitative analysis show knockdown of Smad2 reduced renal injury in I/R-induced AKI model. B: Creatinine assay. C: BUN assay. Serum creatinine and BUN show knockdown of Smad2 prevented decline of renal function. D: Real-time PCR data show knockdown of Smad2 reduced mRNA of TNF-alpha, IL-1beta, MCP-1 and KIM-1. E: Immunohistochemistry and quantitative data show knockdown of Smad2 reduced KIM-1 protein and F4/80+ macrophages infiltration in injured kidney. F: TUNEL assay. G: Western blot analysis of KIM-1, RIPK1, RIPK3, cleaved caspase-3. Data represent mean ± SEM for 6 mice. *P

1 Publications Citing This Product

1. PubMed ID: -, Smad3-Targeted Therapy Protects against Cisplatin-Induced AKI by Attenuating Programmed Cell Death and Inflammation via a NOX4-Dependent Mechanism. Qin Yang,Li Gao,Xiao-wei Hu,Jia-nan Wang,Yao Zhang,Yu-hang Dong,Hui Yao Lan,Xiao-ming Meng

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Anti-RIP3/RIPK3 Antibody

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