Product Datasheet

TRAF3IP2 Antibody - BSA Free
NB100-56740

Unit Size: 0.1 mg

Store at 4C short term. Aliquot and store at -20C long term. Avoid freeze-thaw cycles.

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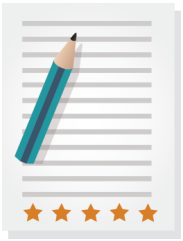
Updated 9/9/2025 v.20.1



NB100-56740

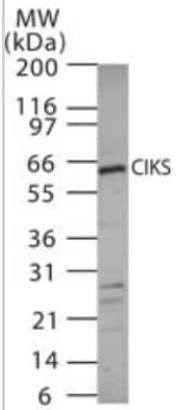
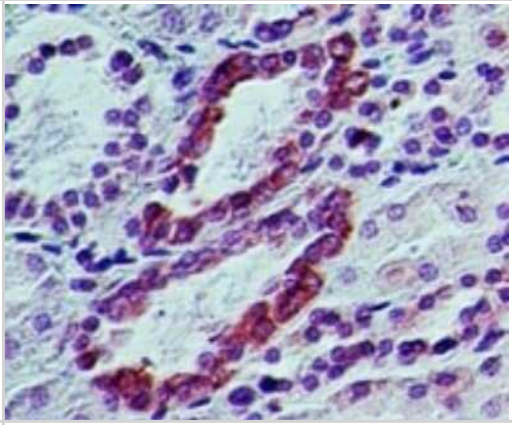
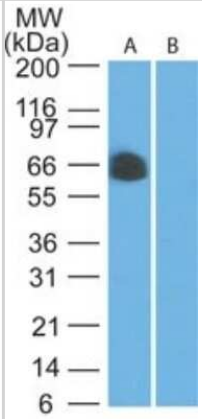
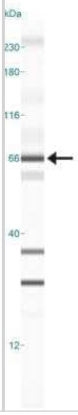
TRAF3IP2 Antibody - BSA Free

Product Information	
Unit Size	0.1 mg
Concentration	1.0 mg/ml
Storage	Store at 4C short term. Aliquot and store at -20C long term. Avoid freeze-thaw cycles.
Clonality	Polyclonal
Preservative	0.05% Sodium Azide
Isotype	IgG
Purity	Protein G purified
Buffer	PBS

Product Description	
Description	Novus Biologicals Rabbit TRAF3IP2 Antibody - BSA Free (NB100-56740) is a polyclonal antibody validated for use in IHC, WB, Simple Western and IP. Anti-TRAF3IP2 Antibody: Cited in 12 publications. All Novus Biologicals antibodies are covered by our 100% guarantee.
Host	Rabbit
Gene ID	10758
Gene Symbol	TRAF3IP2
Species	Human, Mouse
Reactivity Notes	Immunogen displays the following percentage of sequence identity for these species: human (100%), rat (88%), and mouse (82%).
Immunogen	This antibody was developed against a synthetic peptide (QDLPRPLRSREFPQFEP) corresponding to amino acids 225-241 of human TRAF3IP2.

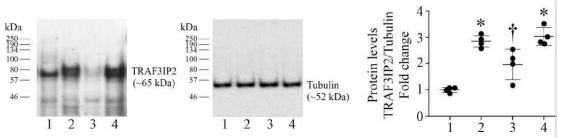
Product Application Details	
Applications	Western Blot, Simple Western, Immunohistochemistry-Paraffin, Immunohistochemistry, Immunoprecipitation
Recommended Dilutions	Western Blot 1-2 ug/ml, Simple Western 1:20, Immunohistochemistry, Immunoprecipitation reported in scientific literature (PMID 24561578), Immunohistochemistry-Paraffin
Application Notes	An approx. 60 kDa band is observed. In Simple Western only 10 - 15 uL of the recommended dilution is used per data point. See Simple Western Antibody Database for Simple Western validation: Tested in Hek293 lysate, separated by Size, antibody dilution of 1:10, apparent MW was 63 kDa.

Images

Western Blot: TRAF3IP2 Antibody [NB100-56740] - Analysis of CIKS using NB100-56740 on 15 ugs of mouse kidney cell lysate.	
Immunohistochemistry-Paraffin: TRAF3IP2 Antibody [NB100-56740] - Analysis of ACT1 in human kidney tissue using ACT1 antibody at 5 ug/ml.	
Western Blot: TRAF3IP2 Antibody [NB100-56740] - Analysis of ACT1 in human kidney lysate in the A) absence and B) presence of immunizing peptide using ACT1 antibody at 2 ug/ml.	
Simple Western: TRAF3IP2 Antibody [NB100-56740] - Simple Western lane view shows a specific band for TRAF3IP2 in 0.5 mg/ml of Hek293 lysate. This experiment was performed under reducing conditions using the 12-230 kDa separation system.	

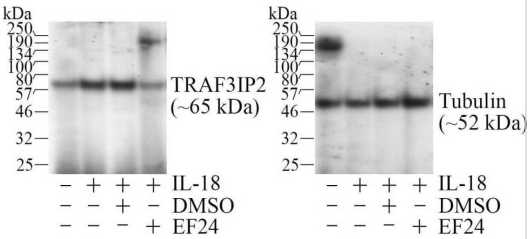
IL-18 inhibits miR-30a and miR-342 expression via stress-activated kinases. (A–C) IL-18 inhibits miR-30a and miR-342 expression via stress-activated kinases. Quiescent ASMCs were treated with inhibitors of either p38 MAPK (SB239063, 10 μM in DMSO for 1 h), ERK1/2 (SCH772984 10 μM in DMSO for 1 h) or JNK (SP600125, 20 μM in DMSO for 1 h) prior to IL-18 addition at 10 ng/mL for 30 min (experimental design in (A)). (B,C) Fresh DMSO (0.1%) served as a solvent control. miR-30a and miR-342 expressions were analyzed by TaqMan® Advanced miRNA assays, with U6 serving as a loading control. (B) * p < 0.001 vs. untreated, † p < at least 0.01 vs. IL-18 or IL-18+DMSO (n = 11), (C) * p < 0.01 vs. untreated, † p < at least 0.05 vs. IL-18 or IL-18+DMSO (n = 5). (D–F) miR-30a mimic inhibits IL-18-induced TRAF3IP2 expression. ASMC were transfected with miR-30a mimic (80 nM), made quiescent and then exposed to IL-18 at 10 ng/mL for 2 h (experimental design in (D)). TRAF3IP2 mRNA expression was analyzed by RT-qPCR (E) and its protein levels by Western blotting (F). (G–I) miR-342 mimic restores IL-18-induced RECK suppression. ASMCs were transfected with miR-342 mimic (80 nM), made quiescent and then exposed to IL-18 at 10 ng/mL for 6 h (experimental design in (G)). RECK mRNA expression was analyzed by RT-qPCR (H) and its protein levels by Western blotting (I). (F,I) While a representative immunoblot is shown, the intensities of immunoreactive bands from 4 independent experiments were semiquantified by densitometry and are summarized as mean +/- SEM on the right. (E,F,H,I) * p < 0.05, † at least p < 0.01 vs. untreated controls (n = 4). Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/39451191), licensed under a CC-BY license. Not internally tested by Novus Biologicals.

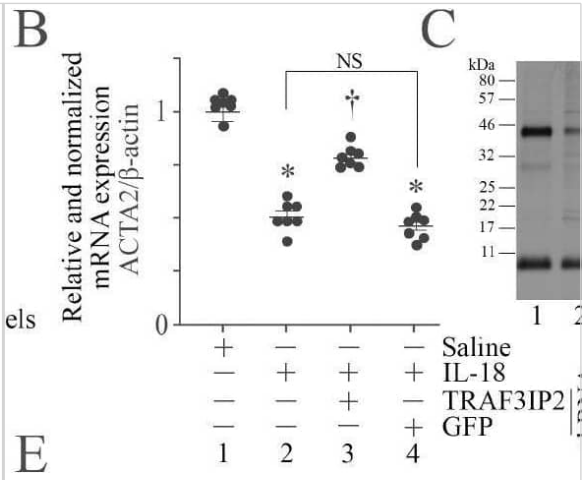
F



EF24 reverses IL-18-induced upregulation in TRAF3IP2 expression and RECK suppression. (A–C) EF24 blunts IL-18-induced TRAF3IP2 expression. Quiescent ASMCs were treated with EF24 (2.5 μM in DMSO for 1 h) prior to IL-18 addition at 10 ng/mL for 3 h (experimental design in (A)). DMSO alone (0.025%) served as a solvent control. TRAF3IP2 mRNA expression was analyzed by RT-qPCR (B) and its protein levels by Western blotting (C). (D–F) EF24 restores IL-18-induced RECK suppression. Quiescent ASMCs were treated with EF24 (2.5 μM in DMSO for 1 h) prior to IL-18 addition at 10 ng/mL for 6 h (experimental design in (D)). RECK mRNA expression was analyzed by RT-qPCR (E) and its protein levels by Western blotting (F). (C,F) While a representative immunoblot is shown, the intensities of immunoreactive bands from 3–4 independent experiments were semiquantified by densitometry and are summarized as mean +/- SEM on the right. (B,E) * p < at least 0.01 vs. Untreated; † p < at least 0.05 vs. IL-18 or IL-18+DMSO (n = 3–4), (C,F) * p < 0.05 vs. Untreated; † p < 0.05 vs. IL-18 or IL-18+DMSO. Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/39451191), licensed under a CC-BY license. Not internally tested by Novus Biologicals.

C





TRAF3IP2 knockdown restores SMC marker expression and inhibits ASMC proinflammatory phenotype without affecting cell viability. (A–G) Silencing TRAF3IP2 restores IL-18-mediated suppression in SMC markers, but inhibits the expression of proinflammatory phenotype markers, without significantly modulating cell viability. ASMCs were transduced with adenoviral TRAF3IP2 shRNA (moi10 for 48 h), made quiescent and then treated with IL-18 (10 ng/mL for 48 h; experimental design in (A)). Expressions of the SMC markers ACTA2 (B,C) and MYH11 (D,E) were analyzed by both RT-qPCR (B,D) and Western blotting (C,E). The proinflammatory phenotype markers Galectin 3, Olr1, VCAM, CCL2, IL-6, IL-8, and TNF-α were analyzed by RT-qPCR using TaqMan™ probes (F). Cell viability was assessed by analyzing cleaved caspase-3 levels using a commercially available Caspase-3 (Cleaved) Human ELISA (G). H2O2 (100 μM for 18 h) served as a positive control and induced a significant increase in cleaved capase-3 levels. (C,E) While a representative immunoblot is shown, the intensities of immunoreactive bands from three independent experiments were semiquantified by densitometry and are summarized on the right. (B,D,F,G) * p < at least 0.01 vs. Untreated; † p < 0.01 vs. IL-18 or IL-18+GFP (n = 6 or 7). (C,E) * p < 0.05 vs. Untreated; † p < 0.05 vs. IL-18 or IL-18+GFP (n = 3). Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/39451191), licensed under a CC-BY license. Not internally tested by Novus Biologicals.

Publications

Higashi, Y;Dashek, R;Delafontaine, P;Rector, RS;Chandrasekar, B; EF24, a Curcumin Analog, Reverses Interleukin-18-Induced miR-30a or miR-342-Dependent TRAF3IP2 Expression, RECK Suppression, and the Proinflammatory Phenotype of Human Aortic Smooth Muscle Cells Cells 2024-10-10 [PMID: 39451191]

Dashek R, Higashi Y, Das N et al. Effects of Empagliflozin on Intermittent Hypoxia-Induced TRAF3IP2-Dependent Human Aortic Smooth Muscle Cell Proliferation Medical Research Archives 2022-11-02 (WB, Human)

Sukhanov S, Higashi Y, Yoshida T et al. The SGLT2 inhibitor Empagliflozin attenuates interleukin-17A-induced human aortic smooth muscle cell proliferation and migration by targeting TRAF3IP2/ROS/NLRP3/Caspase-1-dependent IL-1beta and IL-18 secretion J Virol 2020-11-04 [PMID: 33160017]

Tovell H SGK3 activity and regulation in PI3K-Akt pathway inhibitor resistance in breast cancer Thesis 2020-01-01 (WB, Human)

Das NA, Carpenter AJ, Belenchia A, et al. Empagliflozin reduces high glucose-induced oxidative stress and miR-21-dependent TRAF3IP2 induction and RECK suppression, and inhibits human renal proximal tubular epithelial cell migration and epithelial-to-mesenchymal transition Cell. Signal. 2019-12-17 [PMID: 31862399] (WB, Human)

Das NA, Carpenter AJ, Yashida T et al. TRAF3IP2 mediates TWEAK/TWEAKR-induced pro-fibrotic responses in cultured cardiac fibroblasts and the heart J Mol Cell Cardiol. 2018-07-05 [PMID: 29981796] (WB, Mouse)

Mummidi S, Das NA, Carpenter AJ et al. Metformin inhibits aldosterone-induced cardiac fibroblast activation, migration and proliferation in vitro, and reverses aldosterone + salt-induced cardiac fibrosis in vivo. J Mol Cell Cardiol. 2017-09-01 [PMID: 27423273] (WB, Mouse)

Yariswamy M, Yoshida T, Valente AJ et al. Cardiac-Restricted Overexpression of TRAF3 Interacting Protein 2 (TRAF3IP2) Results in Spontaneous Development of Myocardial Hypertrophy, Fibrosis and Dysfunction. J Biol Chem. [PMID: 27466370] (WB, Mouse)

Padilla J, Carpenter A, Das ND et al. TRAF3IP2 mediates high glucose-induced endothelin-1 production as well as endothelin-1-induced inflammation in endothelial cells. Am. J. Physiol. Heart Circ. Physiol. 2017-09-29 [PMID: 28971844] (WB)

Sakamuri SS, Valente AJ, Siddesha JM et al. TRAF3IP2 mediates aldosterone/salt-induced cardiac hypertrophy and fibrosis. Mol. Cell. Endocrinol. 2016-04-01 [PMID: 27040306] (WB, Mouse)

Somanna NK, Yariswamy M, Garagliano JM et al. Aldosterone-induced cardiomyocyte growth, and fibroblast migration and proliferation are mediated by TRAF3IP2 Cell. Signal. 2015-07-04 [PMID: 26148936] (WB, Mouse)

Valente AJ, Irimpen AM, Siebenlist U et al. OxLDL induces endothelial dysfunction and death via TRAF3IP2: inhibition by HDL3 and AMPK activators Free Radic Biol Med 2014-02-25 [PMID: 24561578] (WB, IP, Human)

More publications at http://www.novusbio.com/NB100-56740

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NB7160	Goat anti-Rabbit IgG (H+L) Secondary Antibody [HRP]
NBP2-24891	Rabbit IgG Isotype Control

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