Product Datasheet

VE-Cadherin Antibody (BV14) - BSA Free
NBP1-43347-0.1mg

Unit Size: 0.1 mg

Store at 4C. Do not freeze.

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Publications: 7

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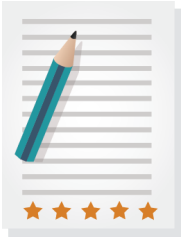
Updated 11/12/2025 v.20.1



NBP1-43347-0.1mg

VE-Cadherin Antibody (BV14) - BSA Free

Product Information

Unit Size	0.1 mg
Concentration	0.5 mg/ml
Storage	Store at 4C. Do not freeze.
Clonality	Monoclonal
Clone	BV14
Preservative	0.09% Sodium Azide
Isotype	IgG2b
Purity	Protein A or G purified
Buffer	PBS (pH 7.2)

Product Description

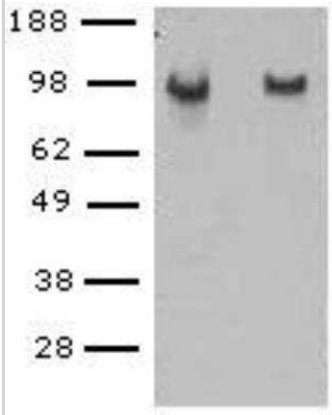
Description	Novus Biologicals Rat VE-Cadherin Antibody (BV14) - BSA Free (NBP1-43347) is a monoclonal antibody validated for use in IHC, WB, ICC/IF and IP. Anti-VE-Cadherin Antibody: Cited in 7 publications. All Novus Biologicals antibodies are covered by our 100% guarantee.
Host	Rat
Gene ID	1003
Gene Symbol	CDH5
Species	Human, Mouse
Reactivity Notes	Use in Mouse reported in scientific literature (PMID:34179146).
Marker	Endothelial Cell Marker
Immunogen	This antibody is made against mouse VE-Cadherin

Product Application Details

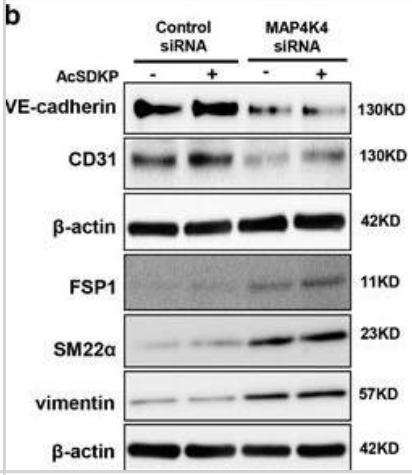
Applications	Western Blot, Immunocytochemistry/ Immunofluorescence, Immunohistochemistry, Immunohistochemistry-Frozen, Immunoprecipitation
Recommended Dilutions	Western Blot 2 ug/ml, Immunohistochemistry 1:10-1:500, Immunocytochemistry/ Immunofluorescence, Immunoprecipitation 1:10-1:500, Immunohistochemistry-Frozen 1:10-1:500
Application Notes	Use in WB reported in scientific literature (PMID:34179146)

Images

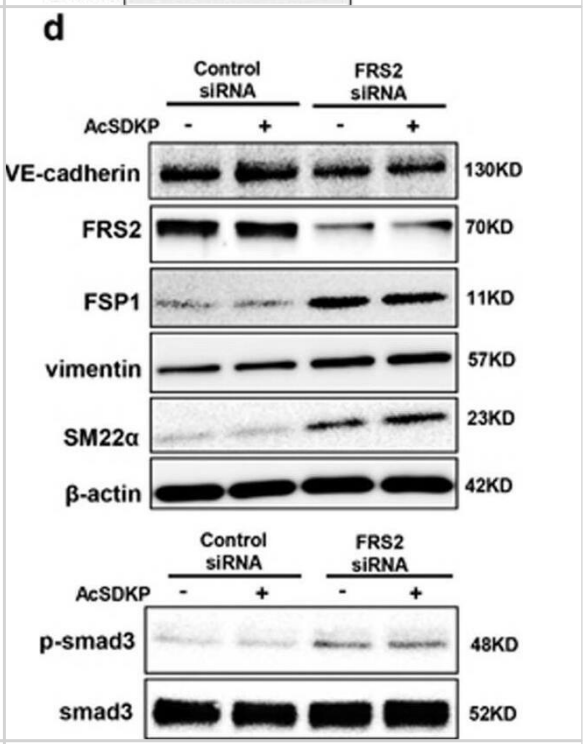
Western Blot: VE-Cadherin Antibody (BV14) [NBP1-43347] - Non-reduced (left) and reduced (right) bEnd.3 cell line lysates were loaded at 1x10^5 cells/lane, probed with 2 ug/mL of Anti-Mouse CD144 (VE-Cadherin) Purified and revealed with Anti-Rat IgG HRP.



MAP4K4 deficiency induces TGFβ/smad signaling and EndMT via activation of integrin β1. (a) HMVECs were transfected with MAP4K4 siRNA (100 nM) for 48 h. Next, the cells were treated with or without AcSDKP for 2 h. The p-smad3/smad3 pathway was analyzed by western blot. Densitometric analysis of the p-smad3/smad3 levels was performed, with n=3 for each group. (b) HMVECs were treated with MAP4K4 siRNA for 48 h with or without AcSDKP treatment. The VE-cadherin, CD31, FSP1, SM22α and vimentin protein levels were analyzed by western blot. (c) HMVECs were transfected with MAP4K4 siRNA for 48 h in the presence or absence of TGFβ2 with or without AcSDKP. The integrin β1 level was analyzed by western blot Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/28771231), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



AcSDKP suppresses TGFβ/smad signaling and EndMT through the FGFR1/FRS2 pathway. (a) HMVECs were treated with N-FGFR1 for 48 h, and the FGFR1, TGFβR1 and TGFβR2 protein levels were analyzed by western blot. (b) HMVECs were treated with TGFβ2 in the presence or absence of N-FGFR1 for 15 min with or without AcSDKP preincubation. The p-smad3 and TGFβR1 protein levels were analyzed by western blot. Densitometric analysis of the p-smad3/smad3 and TGFβR1/β-actin levels (n=3) in each group was performed. (c) HMVECs were incubated with either N-FGFR1 in the presence or absence of TGFβ2 for 48 h with or without preincubation with AcSDKP for 2 h or with N-FGFR1 in the presence or absence of TGFβ2 for 48 h with or without 24 h of incubation with FGF2 (50 ng/ml). The CD31, SM22α, FSP1 and α-SMA protein levels were analyzed by western blot. (d) HMVECs were transfected with FRS2 siRNA (100 nM) for 48 h with or without AcSDKP preincubation. The VE-cadherin, FSP1, vimentin, SM22α and p-smad3 levels were analyzed by western blot. (e) HMVECs were treated with N-FGFR1 for 48 h or 15 min in the presence or absence of N-TGFβ (1, 2, 3) (1.0 μg/ml). The CD31, VE-cadherin, SM22α, FSP1, TGFβR1, TGFβR2 and p-smad3 levels were analyzed by western blot Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/28771231), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



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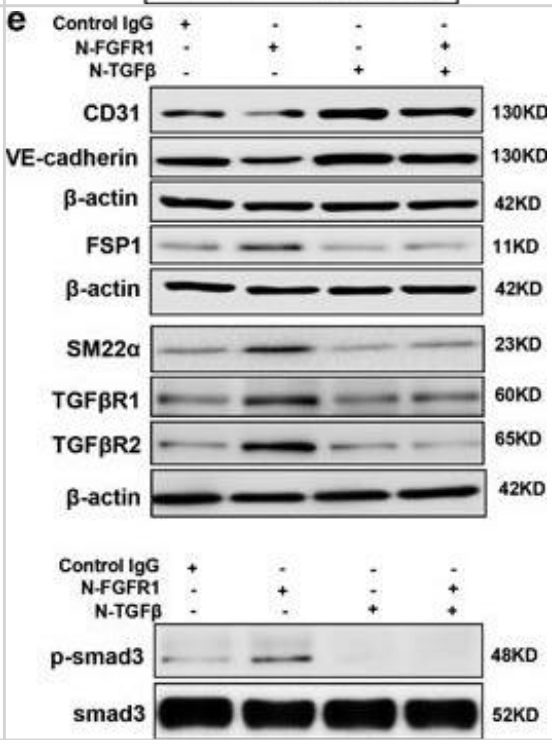
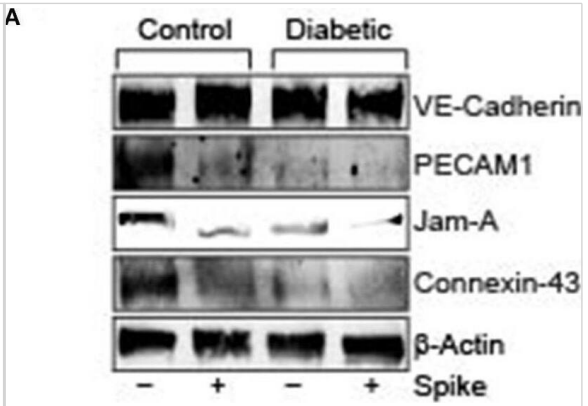
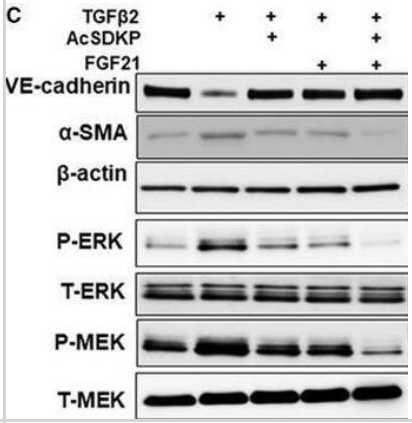


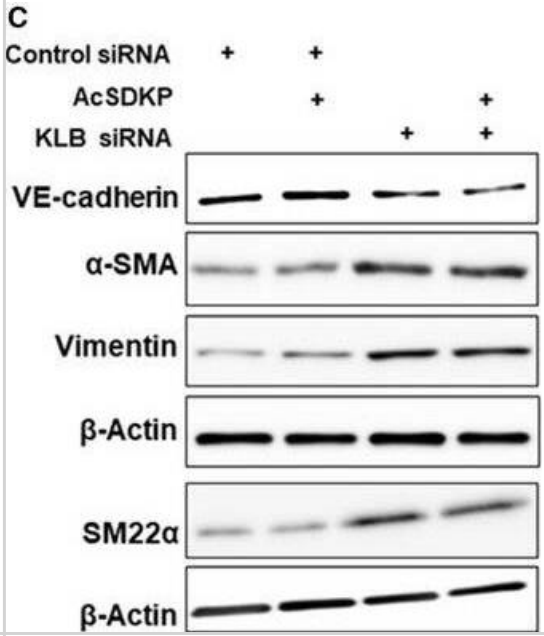
Spike-induced degradation of endothelial junctional proteins is greater in arteries of diabetic mice. (A) Representative Western blot indicating expression of endothelial junctional proteins in arteries isolated from control or diabetic mice. (B) Mean data indicating fold change in protein expression. n = 4 for each, *P < 0.05 vs. untreated control, δP < 0.05 vs. untreated diabetic, #P < 0.05 vs. Spike-treated control. Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/34179146), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



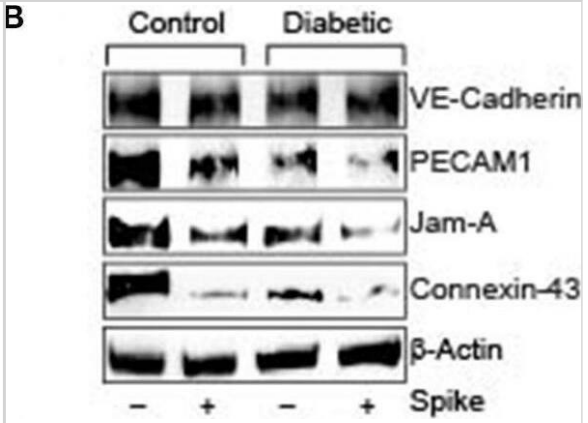
Either FGF19 or FGF21 enhanced the inhibitory effect of AcSDKP on EndMT and MEK/ERK pathway. In the presence of AcSDKP, HMVECs were stimulated by TGFβ2 with or without FGF19 (100 ng·mL−1) or FGF21 (100 ng·mL−1) treatment. The levels of VE□cadherin/β□Actin, α□SMA/β□Actin, P□ERK/T□ERK, and P□MEK/T□MEK were examined by western blot analysis (A, C) and quantified (B, D). The data represent mean +/- SD. Three independent experiments were performed for each result. ANOVA with Tukey's multiple comparisons test was applied. Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/30972974), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



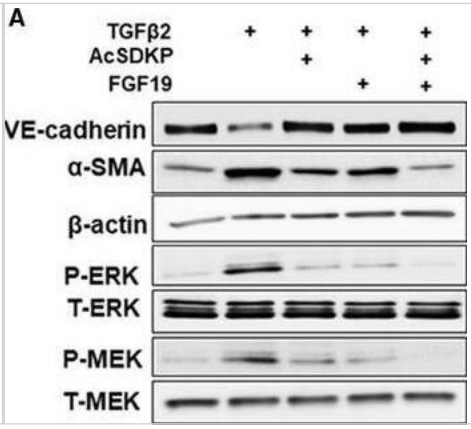
KLB deficiency led to EndMT in HMVECs. (A) Subconfluent HMVECs were transfected with KLB siRNA or control siRNA. Six hours later, the medium was replaced with an experimental medium, followed by N□FGFR1 (1.5 mg·mL−1) treatment. At 48 h, the cells were harvested for western blot analysis. The results are from three repeated experiments. (B) The same treated HMVECs (as in A) cultured on 8□well culture slides were subjected to immunofluorescence staining with an anti□α□SMA antibody and DAPI (scale bar, 100 μm). Six different fields were observed for each slide. (C) HMVECs with or without preincubation with AcSDKP (100 nm) for 2 h were transfected with KLB siRNA or control siRNA for 48 h. The expression of EndMT markers, including VE□cadherin, α□SMA, vimentin, and SM22α, was assessed by western blotting and quantified (D) by imagej software (GE Healthcare Life Sciences, Uppsala, Sweden). The data represent mean +/- SD and are representative of three independent experiments. One□way ANOVA with Tukey's multiple comparisons test was used for statistical analysis. Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/30972974), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



SARS-CoV-2 spike protein (Spike) increases endothelial permeability by downregulation of junctional proteins in diabetic endothelial cells. (A) Endothelial transwell permeability assay, mean data expressed as fold change. n = 6 for each, *P < 0.05 vs. untreated control, δP < 0.05 vs. untreated diabetic, #P < 0.05 vs. Spike-treated control. (B) Representative Western blot indicating expression of endothelial junctional proteins in endothelial cell culture with or without Spike treatment. (C) Mean data indicating fold change in protein expression. n = 6 for each, *P < 0.05 vs. untreated control, δP < 0.05 vs. untreated diabetic, #P < 0.05 vs. Spike-treated control. Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/34179146), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



Either FGF19 or FGF21 enhanced the inhibitory effect of AcSDKP on EndMT and MEK/ERK pathway. In the presence of AcSDKP, HMVECs were stimulated by TGFβ2 with or without FGF19 (100 ng·mL−1) or FGF21 (100 ng·mL−1) treatment. The levels of VE□cadherin/β□Actin, α□SMA/β□Actin, P□ERK/T□ERK, and P□MEK/T□MEK were examined by western blot analysis (A, C) and quantified (B, D). The data represent mean +/- SD. Three independent experiments were performed for each result. ANOVA with Tukey's multiple comparisons test was applied. Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/30972974), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



Publications

Li J, Shi S, et al. FGFR1 is critical for the anti-endothelial mesenchymal transition effect of N-acetyl-seryl-aspartyl-lysyl-proline via induction of the MAP4K4 pathway. Cell Death Dis 2017-08-03 [PMID: 28771231] (WB, Rabbit)

Raghavan S, Kenchappa DB,Leo MD SARS-CoV-2 Spike Protein Induces Degradation of Junctional Proteins That Maintain Endothelial Barrier Integrity Frontiers in cardiovascular medicine 2021-06-11 [PMID: 34179146] (WB, Mouse)

Gao R, Kanasaki K, Li J et al. Beta klotho is essential for the anti-endothelial mesenchymal transition effects of N-acetyl-seryl-aspartyl-lysyl-proline FEBS Open Bio. [PMID: 30972974] (WB, ICC/IF, Human, Mouse)

Luo M, Flood EC, Almeida D et al. Annexin A2 supports pulmonary microvascular integrity by linking vascular endothelial cadherin and protein tyrosine phosphatases J. Exp. Med. 2017-07-10 [PMID: 28694388] (WB, Mouse)

Crosby CV, Fleming PA, Argraves WS, Corada M, Zanetta L, Dejana E, Drake CJ. VE-cadherin is not required for the formation of nascent blood vessels but acts to prevent their disassembly. Blood;105(7):2771-6. 2005-04-01 [PMID: 15604224]

Liao F, Li Y, O'Connor W, Zanetta L, Bassi R, Santiago A, Overholser J, Hooper A, Mignatti P, Dejana E, Hicklin DJ, Bohlen P. Monoclonal antibody to vascular endothelial-cadherin is a potent inhibitor of angiogenesis, tumor growth, and metastasis. Cancer Res;60(24):6805-10. 2000-12-15 [PMID: 11156369]

Corada M, Mariotti M, Thurston G, Smith K, Kunkel R, Brockhaus M, Lampugnani MG, Martin-Padura I, Stoppacciaro A, Ruco L, McDonald DM, Ward PA, Dejana E. Vascular endothelial-cadherin is an important determinant of microvascular integrity in vivo. Proc Natl Acad Sci U S A;96(17):9815-20. 1999-08-17 [PMID: 10449777] (IF/IHC)

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NB7115	Goat anti-Rat IgG (H+L) Secondary Antibody [HRP]
DDXCR03	Rat IgG2b Isotype Control

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