Product Datasheet

Transgelin/TAGLN/SM22 alpha Antibody - BSA Free
NBP1-33003

Unit Size: 0.1 ml

Aliquot and store at -20C or -80C. Avoid freeze-thaw cycles.

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Publications: 4

Protocols, Publications, Related Products, Reviews, Research Tools and Images at:
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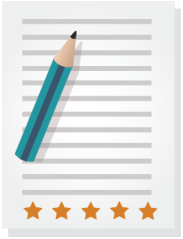
Updated 9/25/2025 v.20.1



NBP1-33003

Transgelin/TAGLN/SM22 alpha Antibody - BSA Free

Product Information

Unit Size	0.1 ml
Concentration	Concentrations vary lot to lot. See vial label for concentration. If unlisted please contact technical services.
Storage	Aliquot and store at -20C or -80C. Avoid freeze-thaw cycles.
Clonality	Polyclonal
Preservative	0.025% Proclin 300
Isotype	IgG
Purity	Antigen Affinity-purified
Buffer	PBS, 1% BSA, 20% Glycerol
Target Molecular Weight	23 kDa

Product Description

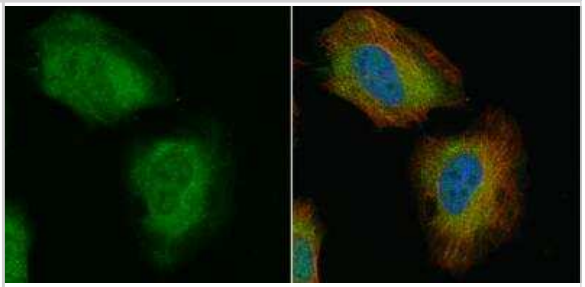
Description	Novus Biologicals Rabbit Transgelin/TAGLN/SM22 alpha Antibody - BSA Free (NBP1-33003) is a polyclonal antibody validated for use in IHC, WB, ICC/IF and IP. Anti-Transgelin/TAGLN/SM22 alpha Antibody: Cited in 4 publications. All Novus Biologicals antibodies are covered by our 100% guarantee.
Host	Rabbit
Gene ID	6876
Gene Symbol	TAGLN
Species	Human, Mouse, Rat
Reactivity Notes	Immunogen displays the following percentage of sequence identity for non-tested species: Chicken (85%).
Immunogen	Recombinant protein encompassing a sequence within the center region of human Transgelin/TAGLN/SM22 alpha. The exact sequence is proprietary.

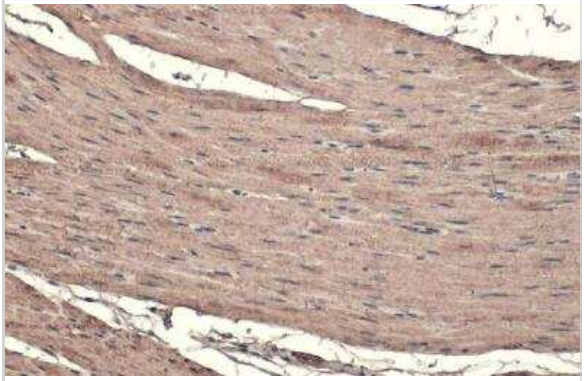
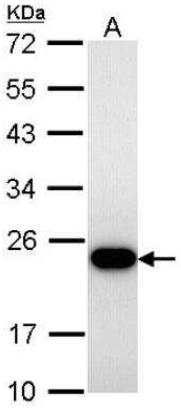
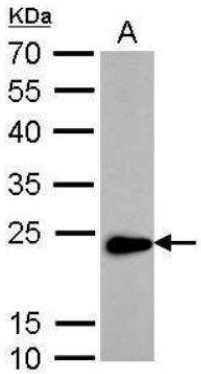
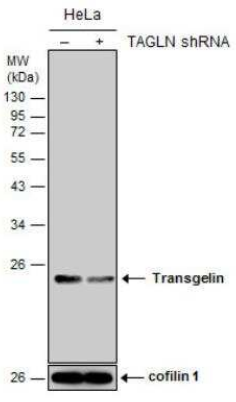
Product Application Details

Applications	Western Blot, Immunohistochemistry-Paraffin, Immunocytochemistry/ Immunofluorescence, Immunohistochemistry, Immunoprecipitation
Recommended Dilutions	Western Blot 1:500-1:10000, Immunohistochemistry 1:100-1:1000, Immunocytochemistry/ Immunofluorescence 1:100-1:1000, Immunoprecipitation 1:100-1:500, Immunohistochemistry-Paraffin 1:100-1:1000

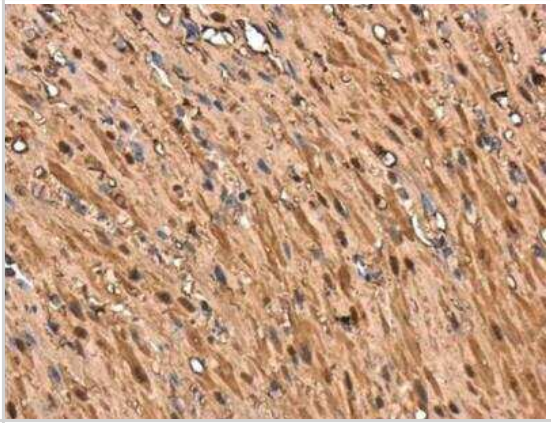
Images

Immunocytochemistry/Immunofluorescence: Transgelin/TAGLN/SM22 alpha Antibody [NBP1-33003] - HeLa cells were fixed in 4% paraformaldehyde at RT for 15 min. Green: Transgelin protein stained by Transgelin antibody diluted at 1:500. Red: alpha Tubulin, a cytoskeleton marker, stained by alpha Tubulin antibody [GT114] diluted at 1:1000. Blue: Hoechst 33342 staining.

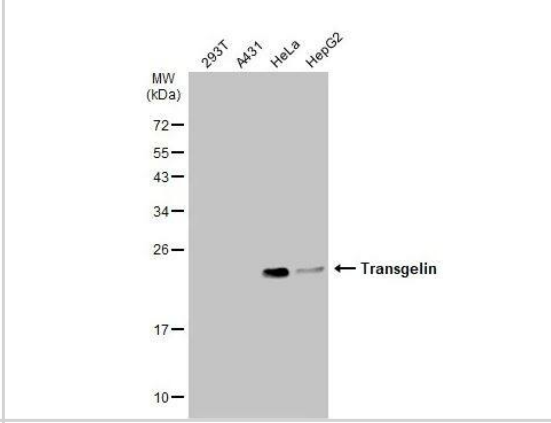


Immunohistochemistry-Paraffin: Transgelin/TAGLN/SM22 alpha Antibody [NBP1-33003] - Mouse smooth muscle. Transgelin stained by Transgelin antibody diluted at 1:500. Antigen Retrieval: Citrate buffer, pH 6.0, 15 min.	
Western Blot: Transgelin/TAGLN/SM22 alpha Antibody [NBP1-33003] - 30 ug C8D30 whole cell lysate/extract 12% SDS-PAGE gel, antibody dilution 1:1000.	
Western Blot: Transgelin/TAGLN/SM22 alpha Antibody [NBP1-33003] - A. 30 ug Rat-2 whole cell lysate/extract 12 % SDS-PAGE Transgelin antibody dilution: 1:5000	
Western Blot: Transgelin/TAGLN/SM22 alpha Antibody [NBP1-33003] - Non-transfected (-) and transfected (+) HeLa whole cell extracts (15 ug) were separated by 12% SDS-PAGE, and the membrane was blotted with Transgelin antibody.	

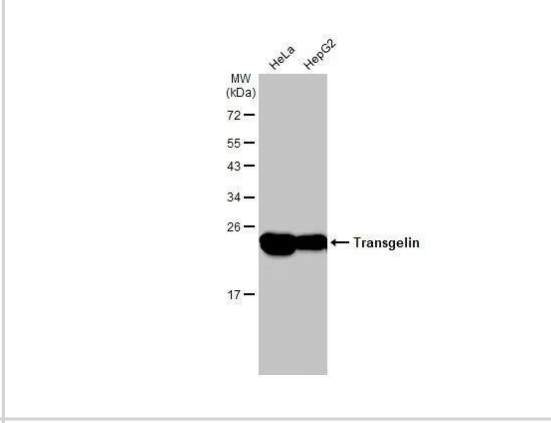
Immunohistochemistry-Paraffin: Transgelin/TAGLN/SM22 alpha Antibody [NBP1-33003] - Rat stomach. Transgelin antibody diluted at 1:500. Antigen Retrieval: Citrate buffer, pH 6.0, 15 min



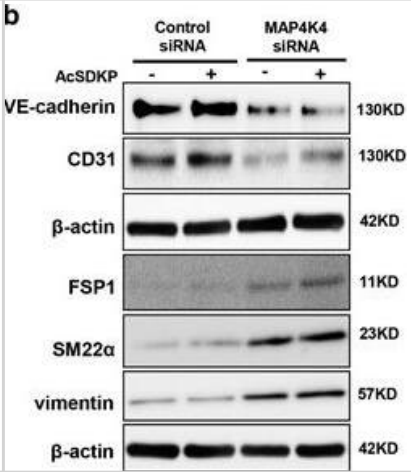
Western Blot: Transgelin/TAGLN/SM22 alpha Antibody [NBP1-33003] - Various whole cell extracts (30 ug) were separated by 12% SDS-PAGE, and the membrane was blotted with Transgelin antibody diluted at 1:1000. The HRP-conjugated anti-rabbit IgG antibody (NBP2-19301 was used to detect the primary antibody.



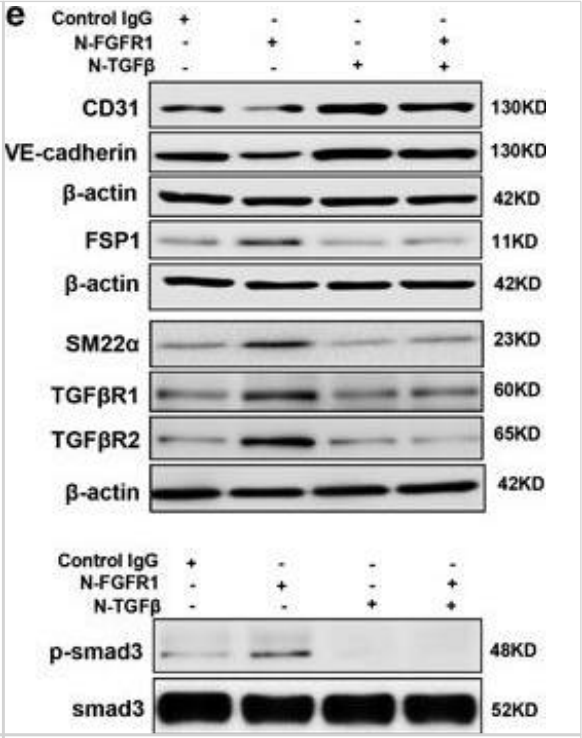
Various whole cell extracts (30 ug) were separated by 12% SDS-PAGE, and the membrane was blotted with Transgelin antibody (NBP1-33003) diluted at 1:1000. The HRP-conjugated anti-rabbit IgG antibody was used to detect the primary antibody.



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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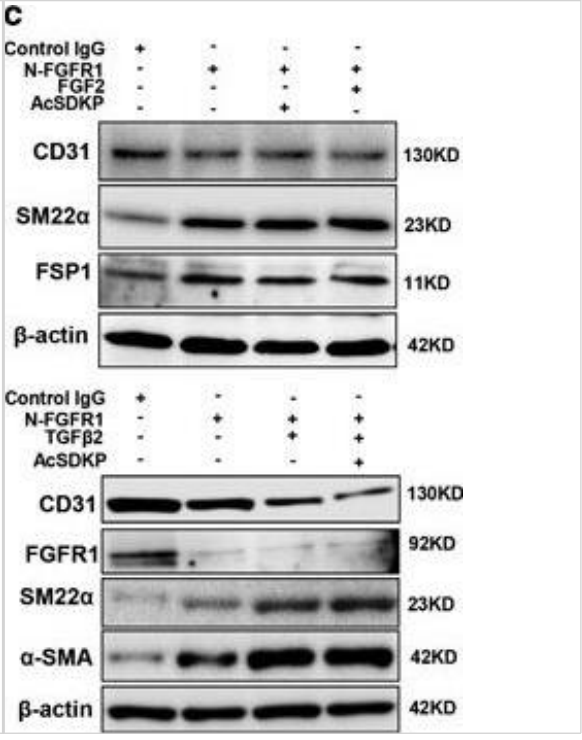
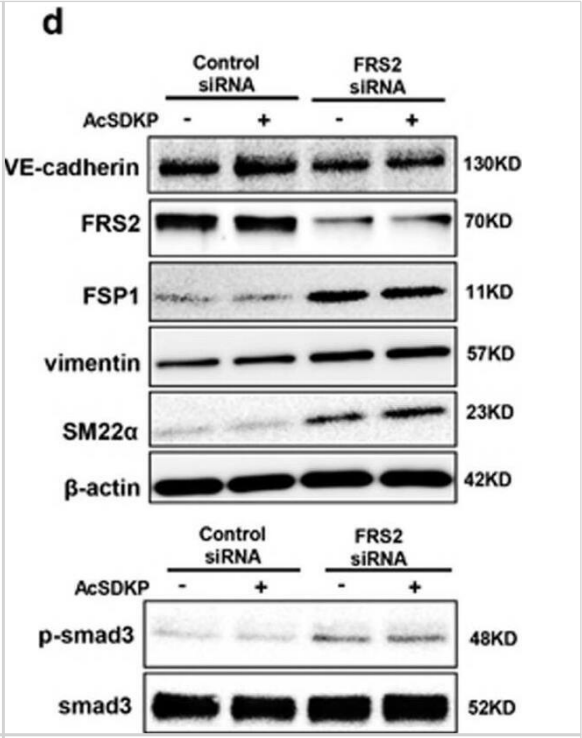
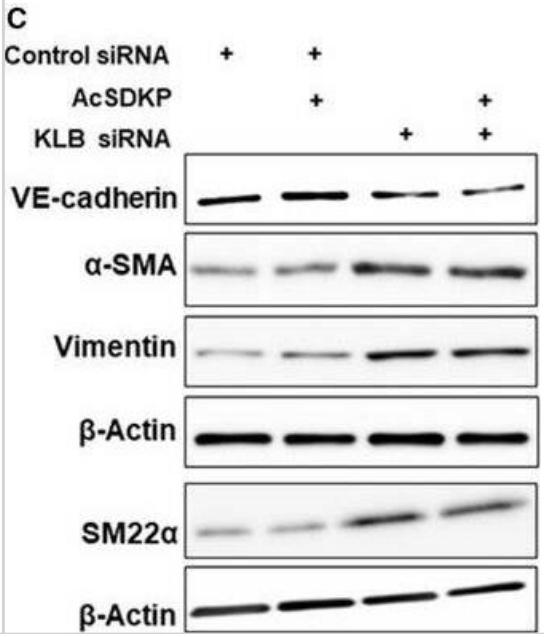


AcSDKP suppresses TGFβ/smad signaling and EndMT through the FGFR1/FRS2 pathway. (a) HMVECs were treated with N-FGFR1 for 48 h, and the FGFR1, TGFβR1 and TGFβR2 protein levels were analyzed by western blot. (b) HMVECs were treated with TGFβ2 in the presence or absence of N-FGFR1 for 15 min with or without 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.



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.



Publications

Cao T, Jiang Y, Li D et al. H19/TET1 axis promotes TGF-beta signaling linked to endothelial-to-mesenchymal transition FASEB J. 2020-05-06 [PMID: 32374060]

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, Human)

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, Mouse)

Shi S, Srivastava SP, Kanasaki M et al. Interactions of DPP-4 and integrin beta1 influences endothelial-to-mesenchymal transition Kidney Int. 2015-04-01 [PMID: 25830763] (WB, Human)

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

Limitations

This product is for research use only and is not approved for use in humans or in clinical diagnosis. Primary Antibodies are guaranteed for 1 year from date of receipt.

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