Product Datasheet

Snail Antibody - BSA Free

NBP2-29626

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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Publications: 2

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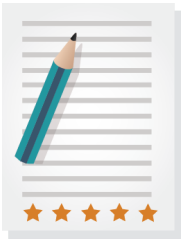
Updated 9/9/2025 v.20.1



NBP2-29626

Snail Antibody - BSA Free

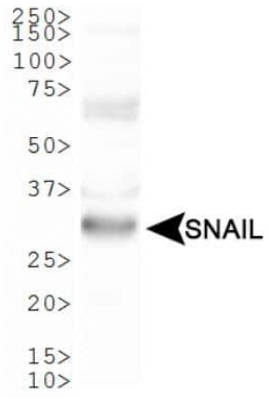
Product Information	
Unit Size	0.1 mg
Concentration	1.0 mg/ml
Storage	Store at 4C short term. Aliquot and store at -20C long term. Avoid freeze-thaw cycles.
Clonality	Polyclonal
Preservative	0.02% Sodium Azide
Isotype	IgG
Purity	Immunogen affinity purified
Buffer	PBS

Product Description	
Description	Novus Biologicals Rabbit Snail Antibody - BSA Free (NBP2-29626) is a polyclonal antibody validated for use in WB. Anti-Snail Antibody: Cited in 1 publication. All Novus Biologicals antibodies are covered by our 100% guarantee.
Host	Rabbit
Gene ID	6615
Gene Symbol	SNAI1
Species	Human, Mouse, Primate
Reactivity Notes	Predicted to react with rat, mink, feline and hamster based on 100% sequence homology.
Immunogen	Synthetic peptide made to an internal portion of human SNAIL protein (between residues 200-300) [UniProt O95863]

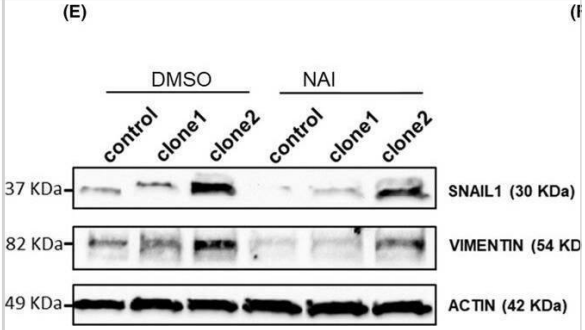
Product Application Details	
Applications	Western Blot
Recommended Dilutions	Western Blot 1.0 ug/ml

Images

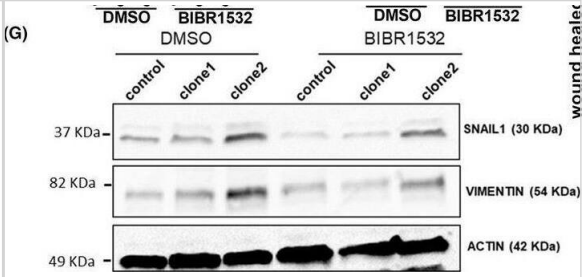
Western Blot: SNAIL Antibody [NBP2-29626] - Western blot analysis of SNAIL in HeLa whole cell extract.



microRNA□196a (miR□196a) effects are mediated by the NFκB signaling pathway. (A) Pathway enrichment meta□analysis of miRTarBase predicted targets for miR□196a. (B) Gene expression of the miR□196a validated target NFKBIA in control and OE33 miR□196a overexpressing clones. (C) Activity of the NFκB pathway in control and OE33 miR□196a clones. Cells were transfected with a plasmid containing 4xNFκB response elements controlling the expression of the downstream luciferase gene. After 48 h, protein extract was obtained and luciferase activity was quantified using the Dual□Glo Luciferase Assay System. (D) Reversion of the mesenchymal phenotype of control and OE33 miR□196a overexpressing clones after inhibition of NFκB. Cells were treated with 10 μm of NAI or DMSO as control for 18 h. Scale bar, 100 μm. (E) Western Blot images showing the inhibition of the increase in both SNAIL1 and VIMENTIN protein levels, and in cell motility (F) of control and OE33 miR□196a overexpressing clones after inhibition of NFκB signaling using 10 μm of NAI or DMSO as control for 18 h. Data are mean + standard error of the mean of three independent experiments. **P < 0.01 and ***P < 0.001 for analysis of variance (ANOVA), plus Bonferroni post□test. ns, not significant. Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/40955778), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



microRNA□196a (miR□196a) effects are mediated by TERT. (A–D) TERT siRNA interference reduces the expression of epithelial□to□mesenchymal transition markers (EMT) in miR□196a overexpressing OE33 clones both at mRNA and protein levels. Cells were transfected using Lipofectamine 2000 with TERT siRNA or control siRNA (ctrl siRNA) and gene expression was analyzed after 48 h by qPCR (A–C). Protein expression in these cells was analyzed by western blot (D). (E–G) Reduction of miR□196a□mediated increase in the expression of EMT markers, both at mRNA and protein levels, upon TERT inhibition. Control cells and OE33 clones overexpressing miR□196a were treated with 20 μm of BIBR1532 or DMSO as control for 18 h, and gene and protein expression were analyzed. (H) Reduction of miR□196a□mediated increase in cell motility upon TERT inhibition. Control cells and OE33 clones overexpressing miR□196a were treated with 20 μm of BIBR1532 or DMSO as control for 18 h, and cell motility was analyzed by wound□healing assay. Data are mean + standard error of the mean of three independent experiments. **P < 0.01 and ***P < 0.001 for analysis of variance (ANOVA), plus Bonferroni post□test. ns, not significant. Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/40955778), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



microRNA□196a (miR□196a) induces epithelial□to□mesenchymal transition (EMT) and cell motility via c□MYC protein accumulation. (A) c□MYC mRNA levels in control cells and OE33 miR□196a overexpressing clones. (B) Western blot of c□MYC protein in control cells and OE33 miR□196a overexpressing clones. (C) VCP mRNA levels in control cells and OE33 miR□196a overexpressing clones. (D) Luciferase reporter assay in control and miR□196a overexpressing OE33 clones. Wild type (wt) 3′ untranslated region of VCP mRNA (vcp mRNA 3′ UTR) or deletion of putative mir□196a binding sequences (Δ196a) were cloned downstream of the luciferase (luc) gene. Luciferase constructs were then transfected in control cells or OE33 miR□196a overexpressing clones, and luciferase activity was quantified using the Dual□Glo Luciferase Assay System after 48 h. (E) Reversal of the mesenchymal phenotype of OE33 miR□196a overexpressing clones after inhibition of c□MYC activity. Cells were plated in six well plates and treated with 50 µm of 10 074□G5 or DMSO as control for 18 h. Scale bar, 100 µm. (F) Western blot of SNAIL1 and VIMENTIN protein levels in control cells or OE33 miR□196a overexpressing clones, and TERT mRNA levels in control cells and OE33 miR□196a overexpressing clones upon c□MYC inhibition (H). Cells were plated in six well plates and treated with 50 µm of 10 074□G5 or DMSO as control for 18 h. Data are mean + standard error of the mean of three independent experiments. **P < 0.01 and ***P < 0.001 for analysis of variance (ANOVA), plus Bonferroni post□test. ns, not significant. Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/40955778), licensed under a CC-BY license. Not internally tested by Novus Biologicals.

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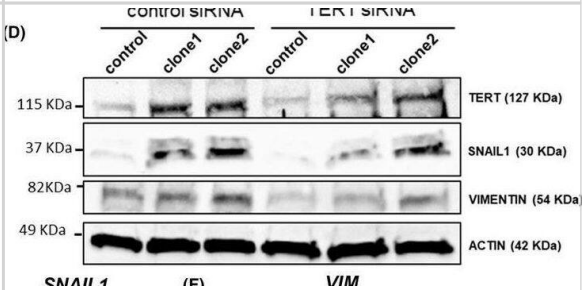
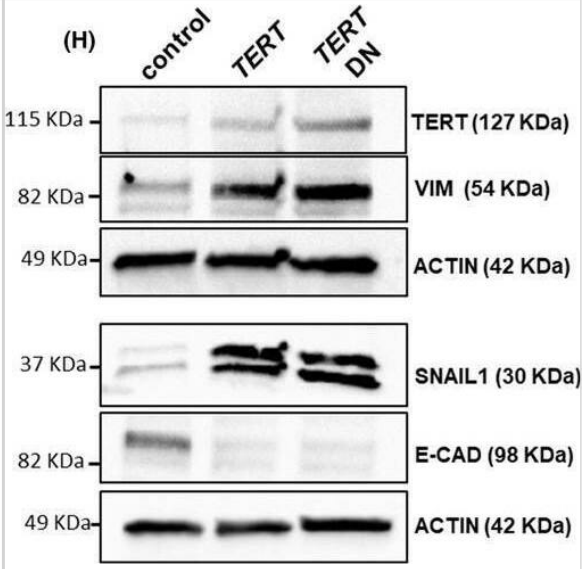


microRNA□196a (miR□196a) up□regulates TERT expression, and TERT increases NFκB activity and produces epithelial□to□mesenchymal transition (EMT) in Esophageal Adenocarcinoma cells in a telomere lengthening□independent manner. (A) Expression of TERT, the main component of the telomerase complex, by qPCR in control and OE33 mir196a overexpressing cells. (B) TERT expression in OE33 control and OE33 clones stably overexpressing TERT or a dominant negative form of TERT (TERT DN). (C) Phenotype switch in OE33 TERT and TER□DN overexpressing clones. (D) TERT overexpression increases NFκB signaling activity. Control or OE33 clones stably overexpressing TERT or TERT DN were transfected with a plasmid containing 4xNFκB response elements controlling the expression of downstream luciferase gene. After 48 h, protein extract was obtained, and luciferase activity was quantified using the Dual□Glo Luciferase Assay System. (E–H) qPCR and western blots of control and OE33 TERT and TER□DN overexpressing clones showing the expression of EMT markers at both mRNA and protein levels. Scale bar, 100 μm. Data are mean + standard error of the mean of three independent experiments. **P < 0.01 and ***P < 0.001 for analysis of variance (ANOVA), plus Bonferroni post□test. Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/40955778), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



Publications

García-Castillo J, Martínez-Cáceres C, Bernabé-García M et al. MicroRNA 196a contributes to the aggressiveness of esophageal adenocarcinoma through the MYC/TERT/NFκB axis. Molecular oncology 2025-09-16 [PMID: 40955778]

Mondal SK, Jinka S, Pal K et al. Glucocorticoid Receptor-Targeted Liposomal Codelivery of Lipophilic Drug and Anti-Hsp90 Gene: Strategy to Induce Drug-Sensitivity, EMT-Reversal, and Reduced Malignancy in Aggressive Tumors. Mol. Pharm. 2016-07-05 [PMID: 27184196] (WB, Mouse)

Procedures

Western Blot protocol for Snail Antibody (NBP2-29626)
Snail Antibody:
Western Blot Protocol

1. Perform SDS-PAGE on samples to be analyzed, loading 40 ug of total protein per lane.
2. Transfer proteins to membrane according to the instructions provided by the manufacturer of the membrane and transfer apparatus.
3. Stain according to standard Ponceau S procedure (or similar product) to assess transfer success, and mark molecular weight standards where appropriate.
4. Rinse the blot.
5. Block the membrane using standard blocking buffer for at least 1 hour.
6. Wash the membrane in wash buffer three times for 10 minutes each.
7. Dilute primary antibody in blocking buffer and incubate 1 hour at room temperature.
8. Wash the membrane in wash buffer three times for 10 minutes each.
9. Apply the diluted HRP conjugated secondary antibody in blocking buffer (as per manufacturers instructions) and incubate 1 hour at room temperature.
10. Wash the blot in wash buffer three times for 10 minutes each (this step can be repeated as required to reduce background).
11. Apply the detection reagent of choice in accordance with the manufacturers instructions.

*Note: Tween-20 can be added to the blocking or antibody dilution buffer at a final concentration of 0.05-0.2%.

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Products Related to NBP2-29626

NBP2-33376H	Blue Marker Antibody (6F4-F6) [HRP]
HAF008	Goat anti-Rabbit IgG Secondary Antibody [HRP]
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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