

Product Datasheet

BLAME/SLAMF8 Antibody NBP2-26110

Unit Size: 0.1 mg

Store at -20C. Avoid freeze-thaw cycles.

www.novusbio.com



technical@novusbio.com

Publications: 1

Protocols, Publications, Related Products, Reviews, Research Tools and Images at:
www.novusbio.com/NBP2-26110

Updated 9/9/2025 v.20.1

Earn rewards for product
reviews and publications.

Submit a publication at www.novusbio.com/publications

Submit a review at www.novusbio.com/reviews/destination/NBP2-26110



NBP2-26110**BLAME/SLAMF8 Antibody**

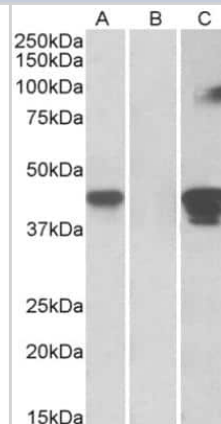
Product Information	
Unit Size	0.1 mg
Concentration	0.5 mg/ml
Storage	Store at -20C. Avoid freeze-thaw cycles.
Clonality	Polyclonal
Preservative	0.02% Sodium Azide
Isotype	IgG
Purity	Immunogen affinity purified
Buffer	Tris saline (20 mM Tris pH 7.3, 150 mM NaCl), 0.5% BSA

Product Description	
Description	Novus Biologicals Goat BLAME/SLAMF8 Antibody (NBP2-26110) is a polyclonal antibody validated for use in IHC, WB and ELISA. Anti-BLAME/SLAMF8 Antibody: Cited in 1 publication. All Novus Biologicals antibodies are covered by our 100% guarantee.
Host	Goat
Gene ID	56833
Gene Symbol	SLAMF8
Species	Human
Reactivity Notes	Expected from sequence similarity: Mouse, Rat, Bovine
Immunogen	Peptide with sequence C-TLYHSRFLGRAQ corresponding to internal region according to NP_064510.1.

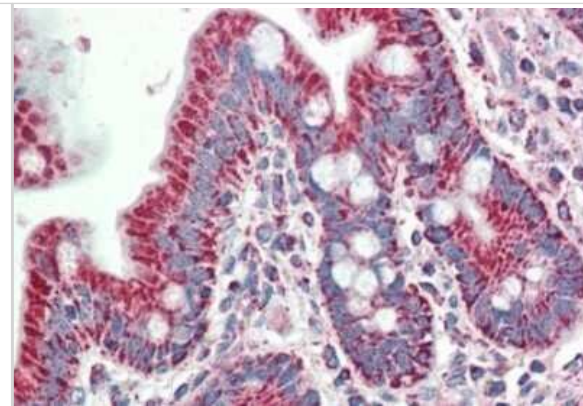
Product Application Details	
Applications	Western Blot, Immunohistochemistry-Paraffin, Immunohistochemistry, Peptide ELISA
Recommended Dilutions	Western Blot 1:100 - 1:2000, Immunohistochemistry 0.5-1ug/ml, Immunohistochemistry-Paraffin, Peptide ELISA Detection limit 1:8000
Application Notes	Western blot: In transfected HEK293 transiently expressing Human SLAMF8 (myc and DYKDDDDK tagged), a band of approx. 45kDa is observed. No bands are observed in mock-transfected HEK293 and the same band is observed using anti-myc antibody. IHC: Paraffin embedded Human Small Intestine.

Images

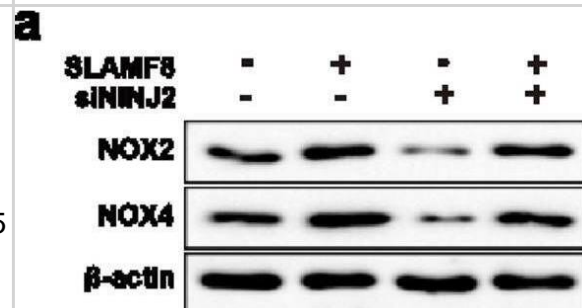
Western Blot: BLAME/SLAMF8 Antibody [NBP2-26110] - HEK293 lysate (10 ug protein in RIPA buffer) overexpressing Human SLAMF8 with C-terminal MYC tag probed with NBP2-26110 (1 ug/ml) in Lane A and probed with anti-MYC Tag (1/1000) in lane C. Mock-transfected HEK293 probed with NBP2-26110 (1 ug/ml) in Lane B. Primary incubations were for 1 hour. Detected by chemiluminescence.



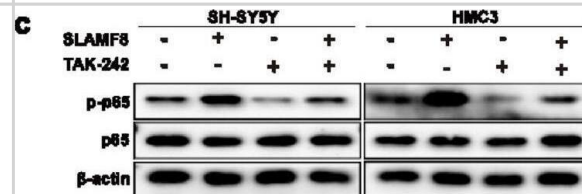
Immunohistochemistry-Paraffin: BLAME/SLAMF8 Antibody [NBP2-26110] - 5ug/ml staining of paraffin embedded Human Small Intestine. Steamed antigen retrieval with citrate buffer pH 6, AP-staining.



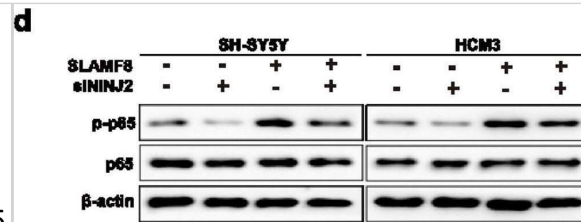
(a,b) Knockdown of NINJ2 significantly abolished SLAMF8-induced oxidative stress, reduced ROS levels (DCF and MitoSOX Red), and decreased NOX2 and NOX4 expression in A β 1-42-treated SH-SY5Y cells. (c) Similarly, knockdown of NINJ2 significantly reduced the SLAMF8-treated neuroinflammation in LPS-treated HMC3 cells. (d) In SLAMF8-stably overexpressing AD cell models, transient knockdown of NINJ2 with siNINJ2 for 48 h significantly abolished SLAMF8-induced p65 phosphorylation in the TLR4/NF- κ B signaling pathway. Data are presented as mean \pm standard deviation. One-way ANOVA with Tukey's post hoc test was used for multiple group comparisons, and a paired t-test was used for comparisons between two groups. "*" indicates $p \leq 0.05$, and "***" indicates $p \leq 0.01$. Image collected and cropped by CiteAb from the following open publication (<https://pubmed.ncbi.nlm.nih.gov/40394132>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



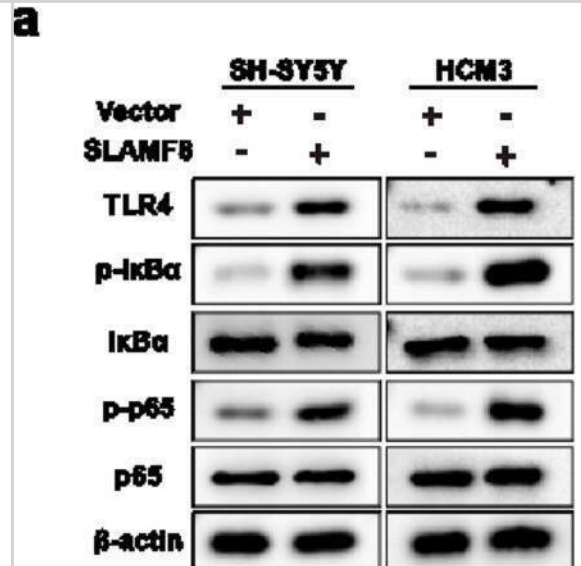
(a) The overexpression of SLAMF8 in AD cell models increased the levels of TLR4/NF- κ B signaling-related markers (TLR4, p-I κ B α /I κ B α ratio) and the phospho-p65/p65 ratio, as confirmed by Western blot. Quantification of the Western blot results was performed and normalized against β -actin ($n = 3$) (a1). (b) Conversely, knockdown of SLAMF8 produced the opposite effect and the data were quantified and normalized to β -actin ($n = 3$). (b1) The TLR4/NF- κ B signaling inhibitor TAK-242 suppressed TLR4 expression (c,c1) and mitigated the increase in ROS (d,e) and neuroinflammation (f) caused by SLAMF8 overexpression in AD cell models. Data are presented as mean \pm standard deviation. One-way ANOVA with Tukey's post hoc test was used for multiple group comparisons, and a paired t-test was used for comparisons between two groups. "*" indicates $p \leq 0.05$, and "***" indicates $p \leq 0.01$. Image collected and cropped by CiteAb from the following open publication (<https://pubmed.ncbi.nlm.nih.gov/40394132>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



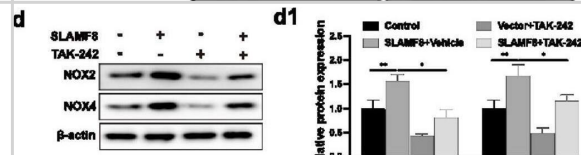
(a,b) Knockdown of NINJ2 significantly abolished SLAMF8-induced oxidative stress, reduced ROS levels (DCF and MitoSOX Red), and decreased NOX2 and NOX4 expression in A β 1-42-treated SH-SY5Y cells. (c) Similarly, knockdown of NINJ2 significantly reduced the SLAMF8-treated neuroinflammation in LPS-treated HMC3 cells. (d) In SLAMF8-stably overexpressing AD cell models, transient knockdown of NINJ2 with siNINJ2 for 48 h significantly abolished SLAMF8-induced p65 phosphorylation in the TLR4/NF- κ B signaling pathway. Data are presented as mean \pm standard deviation. One-way ANOVA with Tukey's post hoc test was used for multiple group comparisons, and a paired t-test was used for comparisons between two groups. "*" indicates $p \leq 0.05$, and "***" indicates $p \leq 0.01$. Image collected and cropped by CiteAb from the following open publication (<https://pubmed.ncbi.nlm.nih.gov/40394132>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



(a) The overexpression of SLAMF8 in AD cell models increased the levels of TLR4/NF- κ B signaling-related markers (TLR4, p-I κ B α /I κ B α ratio) and the phospho-p65/p65 ratio, as confirmed by Western blot. Quantification of the Western blot results was performed and normalized against β -actin ($n = 3$) (a1). (b) Conversely, knockdown of SLAMF8 produced the opposite effect and the data were quantified and normalized to β -actin ($n = 3$). (b1) The TLR4/NF- κ B signaling inhibitor TAK-242 suppressed TLR4 expression (c,c1) and mitigated the increase in ROS (d,e) and neuroinflammation (f) caused by SLAMF8 overexpression in AD cell models. Data are presented as mean \pm standard deviation. One-way ANOVA with Tukey's post hoc test was used for multiple group comparisons, and a paired t-test was used for comparisons between two groups. "*" indicates $p \leq 0.05$, and "***" indicates $p \leq 0.01$. Image collected and cropped by CiteAb from the following open publication (<https://pubmed.ncbi.nlm.nih.gov/40394132>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



(a) The overexpression of SLAMF8 in AD cell models increased the levels of TLR4/NF- κ B signaling-related markers (TLR4, p-I κ B α /I κ B α ratio) and the phospho-p65/p65 ratio, as confirmed by Western blot. Quantification of the Western blot results was performed and normalized against β -actin ($n = 3$) (a1). (b) Conversely, knockdown of SLAMF8 produced the opposite effect and the data were quantified and normalized to β -actin ($n = 3$). (b1) The TLR4/NF- κ B signaling inhibitor TAK-242 suppressed TLR4 expression (c,c1) and mitigated the increase in ROS (d,e) and neuroinflammation (f) caused by SLAMF8 overexpression in AD cell models. Data are presented as mean \pm standard deviation. One-way ANOVA with Tukey's post hoc test was used for multiple group comparisons, and a paired t-test was used for comparisons between two groups. "*" indicates $p \leq 0.05$, and "***" indicates $p \leq 0.01$. Image collected and cropped by CiteAb from the following open publication (<https://pubmed.ncbi.nlm.nih.gov/40394132>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



Publications

Davila S, Froeling FE, Tan A et al. New genetic associations detected in a host response study to hepatitis B vaccine. *Genes Immun* 2010-04-01 [PMID: 20237496]



Novus Biologicals USA

10730 E. Briarwood Avenue
Centennial, CO 80112
USA
Phone: 303.730.1950
Toll Free: 1.888.506.6887
Fax: 303.730.1966
nb-customerservice@bio-techne.com

Bio-Techne Canada

21 Canmotor Ave
Toronto, ON M8Z 4E6
Canada
Phone: 905.827.6400
Toll Free: 855.668.8722
Fax: 905.827.6402
canada.inquires@bio-techne.com

Bio-Techne Ltd

19 Barton Lane
Abingdon Science Park
Abingdon, OX14 3NB, United Kingdom
Phone: (44) (0) 1235 529449
Free Phone: 0800 37 34 15
Fax: (44) (0) 1235 533420
info.EMEA@bio-techne.com

General Contact Information

www.novusbio.com
Technical Support: nb-technical@bio-techne.com
Orders: nb-customerservice@bio-techne.com
General: novus@novusbio.com

Products Related to NBP2-26110

NBP2-33376H	Blue Marker Antibody (6F4-F6) [HRP]
HAF017	Rabbit anti-Goat IgG Secondary Antibody [HRP (Horseradish Peroxidase)]
HAF109	Donkey anti-Goat IgG Secondary Antibody [HRP (Horseradish Peroxidase)]
NB410-28088-1mg	Goat 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.

For more information on our 100% guarantee, please visit www.novusbio.com/guarantee

Earn gift cards/discounts by submitting a review: www.novusbio.com/reviews/submit/NBP2-26110

Earn gift cards/discounts by submitting a publication using this product:
www.novusbio.com/publications



