# Product Datasheet

## TRANCE/TNFSF11/RANK L Antibody (8A7B9) - BSA Free
## NBP2-61813

Unit Size: 0.1 ml

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

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**Publications: 1**

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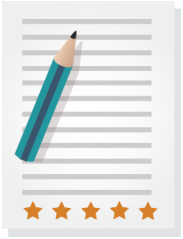
Updated 9/9/2025 v.20.1





# NBP2-61813

TRANCE/TNFSF11/RANK L Antibody (8A7B9) - BSA Free

## Product Information

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|---|---|
| **Unit Size** | 0.1 ml |
| **Concentration** | 1 mg/ml |
| **Storage** | Store at 4C short term. Aliquot and store at -20C long term. Avoid freeze-thaw cycles. |
| **Clonality** | Monoclonal |
| **Clone** | 8A7B9 |
| **Preservative** | 0.05% Sodium Azide |
| **Isotype** | IgG1 |
| **Purity** | Protein G purified |
| **Buffer** | PBS |
| **Target Molecular Weight** | 35.5 kDa |

## Product Description

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|---|---|
| **Description** | Novus Biologicals Mouse TRANCE/TNFSF11/RANK L Antibody (8A7B9) - BSA Free (NBP2-61813) is a monoclonal antibody validated for use in IHC, WB, ELISA, Flow and ICC/IF. Anti-TRANCE/TNFSF11/RANK L Antibody: Cited in 1 publication. All Novus Biologicals antibodies are covered by our 100% guarantee. |
| **Host** | Mouse |
| **Gene ID** | 8600 |
| **Gene Symbol** | TNFSF11 |
| **Species** | Human, Monkey |
| **Immunogen** | Purified recombinant fragment of human TRANCE/TNFSF11/RANK L (AA: 74-308) expressed in E. Coli. |

## Product Application Details

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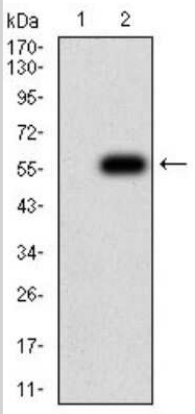
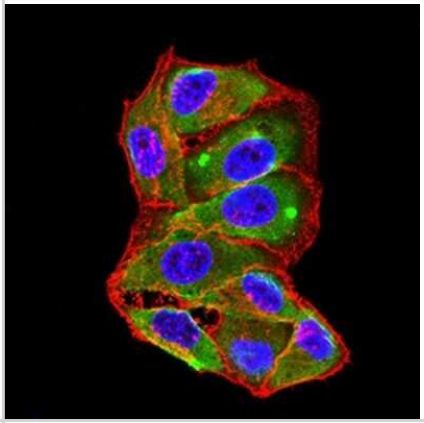
| **Applications** | Western Blot, Immunohistochemistry-Paraffin, ELISA, Flow Cytometry, Immunocytochemistry/ Immunofluorescence, Immunohistochemistry |
| **Recommended Dilutions** | Western Blot 1:500-1:2000, Flow Cytometry 1:200-1:400, ELISA 1:10000, Immunohistochemistry 1:200-1:1000, Immunocytochemistry/ Immunofluorescence 1:100-1:500, Immunohistochemistry-Paraffin 1:200-1:1000 |

## Images

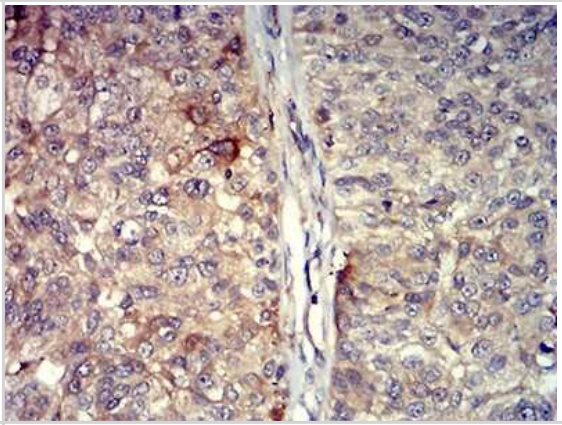
Western Blot: TRANCE/TNFSF11/RANK L Antibody (8A7B9) [NBP2-61813] - Analysis using TNFSF11 mAb against HEK293 (1) and TNFSF11 (AA: 74-308)-hIgGFc transfected HEK293 (2) cell lysate.



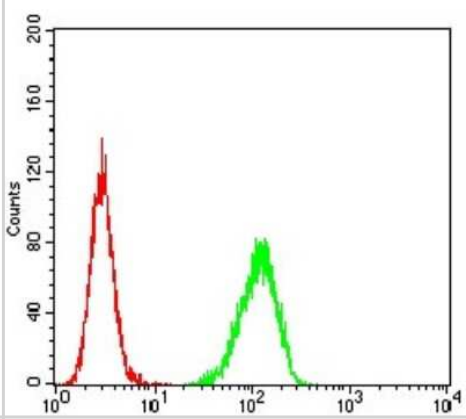
Immunocytochemistry/Immunofluorescence: TRANCE/TNFSF11/RANK L Antibody (8A7B9) [NBP2-61813] - Analysis of Hela cells using TNFSF11 mouse mAb (green). Blue: DRAQ5 fluorescent DNA dye. Red: Actin filaments have been labeled with Alexa Fluor- 555 phalloidin. Goat anti-Mouse IgG (H+L) DyLight 488 secondary antibody was used.



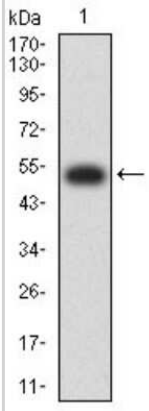
Immunohistochemistry-Paraffin: TRANCE/TNFSF11/RANK L Antibody (8A7B9) [NBP2-61813] - Analysis of bladder cancer tissues using TNFSF11 mouse mAb with DAB staining.



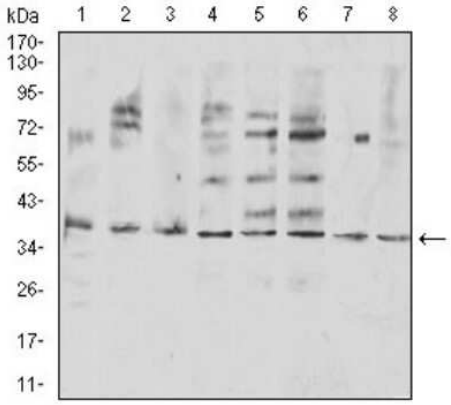
Flow Cytometry: TRANCE/TNFSF11/RANK L Antibody (8A7B9) [NBP2-61813] - Analysis of Hela cells using TNFSF11 mouse mAb (green) and negative control (red).



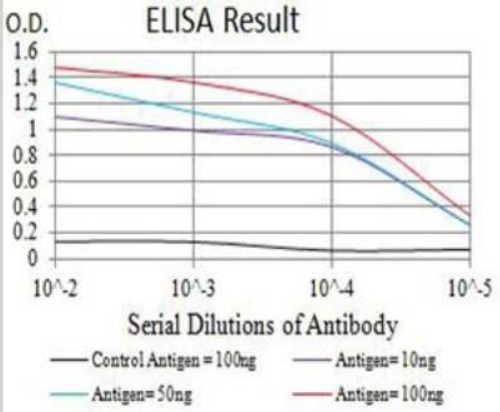

Western Blot: TRANCE/TNFSF11/RANK L Antibody (8A7B9) [NBP2-61813] - Analysis using TNFSF11 mAb against human TNFSF11 (AA: 74 -308) recombinant protein. (Expected MW is 52.6 kDa)



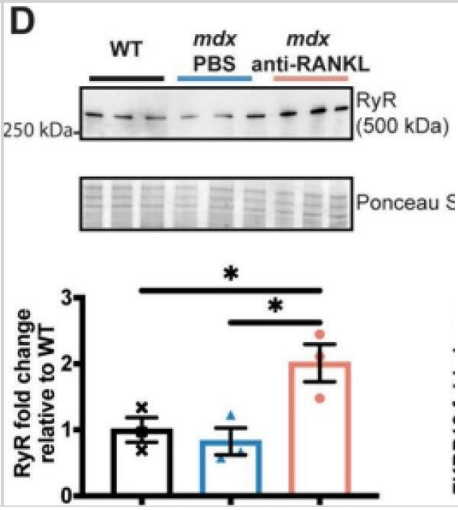
Western Blot: TRANCE/TNFSF11/RANK L Antibody (8A7B9) [NBP2-61813] - Analysis using TNFSF11 mouse mAb against COS7 (1), Hela (2), U937 (3), HL-60 (4), Raji (5), Ramos (6), Jurkat (7), and SW480 (8) cell lysate.




ELISA: TRANCE/TNFSF11/RANK L Antibody (8A7B9) [NBP2-61813] - Black line: Control Antigen (100 ng);Purple line: Antigen (10ng); Blue line: Antigen (50 ng); Red line:Antigen (100 ng)




An anti-RANKL treatment increases SERCA activity and modulates intracellular calcium homeostasis regulators in the dystrophic heart. SERCA activity (A), western blot analyses of SERCA2a (B), phospholamban (PLN) (C), Ryanodine (RyR) (D), and FKBP12 (E) protein levels in hearts from WT, PBS-injected mdx, and anti-RANKL-treated mdx mice. Results are expressed as means +/- SEM (* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$, and **** $p < 0.0001$). Shown are an analysis of variance two-way ANOVA with a Bonferroni correction (A) and an analysis of variance one-way ANOVA with a Tuckey correction for western blots (B–E). N = 11 for WT, mdx-PBS, and mdx-anti-RANKL for SERCA activity (A); n = 3–8 for WT, mdx-PBS, and mdx-anti-RANKL for Western blots (B–E). Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/37296659), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



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| An anti-RANKL treatment reduces the cardiomyocyte surface and inhibits cardiac hypertrophy mediators in dystrophic mice. The heart tissues were sectioned and were incubated with laminin (green), rhodamine-phalloidin (red), and DAPI (blue) markers to label the cardiomyocyte membrane, F-actin filaments, and nuclei, respectively, at 20× magnification (A). The cardiomyocyte mean cross-sectional area (CSA) (A) and CSA distribution (B). Western blot analyses of pNFκB (C), NFκB (D), pPI3K (E), and PI3K (F) protein levels. The scale bar in A represents 0.05 mm. Results are expressed as means +/- SEM (* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$, and **** $p < 0.0001$). Shown are an analysis of variance one-way ANOVA with a Tuckey correction for the CSA and western blots (A,D–F), and an analysis of variance two-way ANOVA with a Bonferroni correction for a distribution analysis (B). N = 3 for WT, n = 6 for mdx-PBS, and n = 9 for mdx-anti-RANKL for the cardiomyocyte CSA (A) and distribution (B). N = 3–6 for WT, n = 5–10 for mdx-PBS, and n = 5–9 for mdx-anti-RANKL for the western blots (C–F). Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/37296659), licensed under a CC-BY license. Not internally tested by Novus Biologicals. | |
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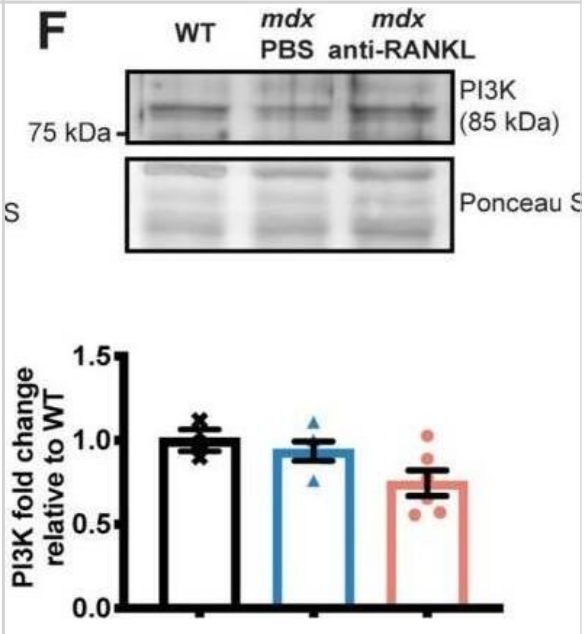
| An anti-RANKL treatment reduces the cardiomyocyte surface and inhibits cardiac hypertrophy mediators in dystrophic mice. The heart tissues were sectioned and were incubated with laminin (green), rhodamine-phalloidin (red), and DAPI (blue) markers to label the cardiomyocyte membrane, F-actin filaments, and nuclei, respectively, at 20× magnification (A). The cardiomyocyte mean cross-sectional area (CSA) (A) and CSA distribution (B). Western blot analyses of pNFκB (C), NFκB (D), pPI3K (E), and PI3K (F) protein levels. The scale bar in A represents 0.05 mm. Results are expressed as means +/- SEM (* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$, and **** $p < 0.0001$). Shown are an analysis of variance one-way ANOVA with a Tuckey correction for the CSA and western blots (A,D–F), and an analysis of variance two-way ANOVA with a Bonferroni correction for a distribution analysis (B). N = 3 for WT, n = 6 for mdx-PBS, and n = 9 for mdx-anti-RANKL for the cardiomyocyte CSA (A) and distribution (B). N = 3–6 for WT, n = 5–10 for mdx-PBS, and n = 5–9 for mdx-anti-RANKL for the western blots (C–F). Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/37296659), licensed under a CC-BY license. Not internally tested by Novus Biologicals. |  |

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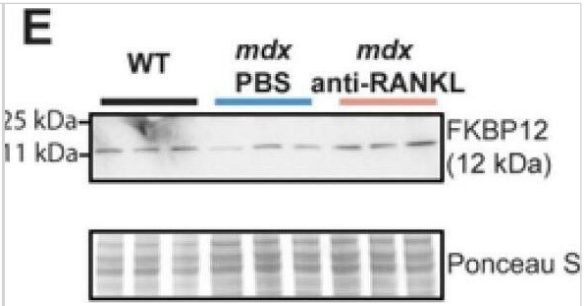
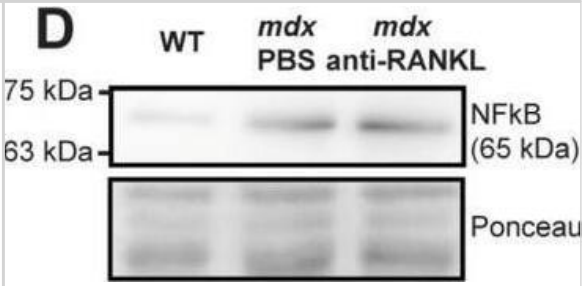
| An anti-RANKL treatment increases SERCA activity and modulates intracellular calcium homeostasis regulators in the dystrophic heart. SERCA activity (A), western blot analyses of SERCA2a (B), phospholamban (PLN) (C), Ryanodine (RyR) (D), and FKBP12 (E) protein levels in hearts from WT, PBS-injected mdx, and anti-RANKL-treated mdx mice. Results are expressed as means +/- SEM (* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$, and **** $p < 0.0001$). Shown are an analysis of variance two-way ANOVA with a Bonferroni correction (A) and an analysis of variance one-way ANOVA with a Tuckey correction for western blots (B–E). N = 11 for WT, mdx-PBS, and mdx-anti-RANKL for SERCA activity (A); n = 3–8 for WT, mdx-PBS, and mdx-anti-RANKL for Western blots (B–E). Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/37296659), licensed under a CC-BY license. Not internally tested by Novus Biologicals. |  |
| An anti-RANKL treatment reduces the cardiomyocyte surface and inhibits cardiac hypertrophy mediators in dystrophic mice. The heart tissues were sectioned and were incubated with laminin (green), rhodamine-phalloidin (red), and DAPI (blue) markers to label the cardiomyocyte membrane, F-actin filaments, and nuclei, respectively, at 20× magnification (A). The cardiomyocyte mean cross-sectional area (CSA) (A) and CSA distribution (B). Western blot analyses of pNFκB (C), NFκB (D), pPI3K (E), and PI3K (F) protein levels. The scale bar in A represents 0.05 mm. Results are expressed as means +/- SEM (* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$, and **** $p < 0.0001$). Shown are an analysis of variance one-way ANOVA with a Tuckey correction for the CSA and western blots (A,D–F), and an analysis of variance two-way ANOVA with a Bonferroni correction for a distribution analysis (B). N = 3 for WT, n = 6 for mdx-PBS, and n = 9 for mdx-anti-RANKL for the cardiomyocyte CSA (A) and distribution (B). N = 3–6 for WT, n = 5–10 for mdx-PBS, and n = 5–9 for mdx-anti-RANKL for the western blots (C–F). Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/37296659), licensed under a CC-BY license. Not internally tested by Novus Biologicals. |  |

## Publications

Marcadet L, Juracic ES, Khan N et al. RANKL Inhibition Reduces Cardiac Hypertrophy in mdx Mice and Possibly in Children with Duchenne Muscular Dystrophy Cells 2023-06-03 [PMID: 37296659] (IHC-Fr, Mouse)

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## Products Related to NBP2-61813

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|---|---|
| NBP2-33376H | Blue Marker Antibody (6F4-F6) [HRP] |
| HAF007 | Goat anti-Mouse IgG Secondary Antibody [HRP] |
| NB7539 | Goat anti-Mouse IgG (H+L) Secondary Antibody [HRP] |
| NBP1-97005-0.5mg | Mouse IgG1 Isotype Control (MG1) |

**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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