Product Datasheet

SEC16A Antibody
NB100-1799

Unit Size: 0.1 ml

Store at 4C. Do not freeze.

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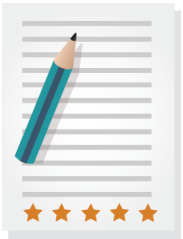
Publications: 11

Protocols, Publications, Related Products, Reviews, Research Tools and Images at:
www.novusbio.com/NB100-1799

Updated 9/9/2025 v.20.1



NB100-1799

SEC16A Antibody

Product Information

Unit Size	0.1 ml
Concentration	0.2 mg/ml
Storage	Store at 4C. Do not freeze.
Clonality	Polyclonal
Preservative	0.09% Sodium Azide
Isotype	IgG
Purity	Immunogen affinity purified
Buffer	TBS and 0.1% BSA

Product Description

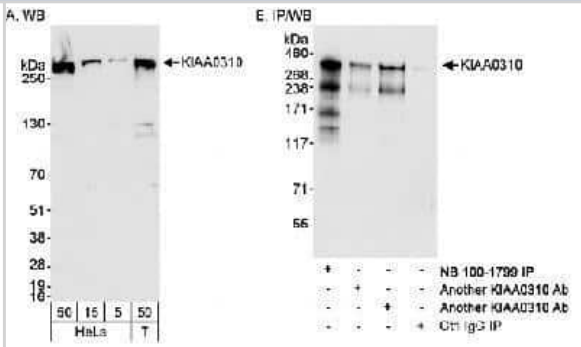
Description	Novus Biologicals Rabbit SEC16A Antibody (NB100-1799) is a polyclonal antibody validated for use in WB, ICC/IF and IP. Anti-SEC16A Antibody: Cited in 10 publications. All Novus Biologicals antibodies are covered by our 100% guarantee.
Host	Rabbit
Gene ID	9919
Gene Symbol	SEC16A
Species	Human, Mouse, Rat, Primate
Reactivity Notes	Primate reactivity reported in (PMID: 22875974), Rat reactivity reported in (PMID: 22740409). Mouse reactivity reported in scientific literature (DOI 10.21007/etd.cghs.2016.0419).
Immunogen	The immunogen recognized by this antibody maps to a region between residues 600 and 650 of human KIAA0310 using the numbering given in entry O15027 (GeneID 9919).

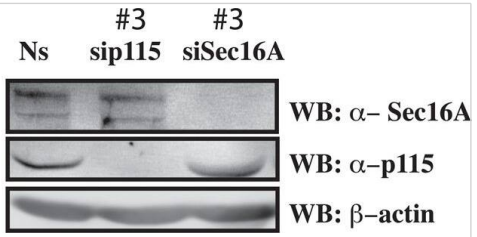
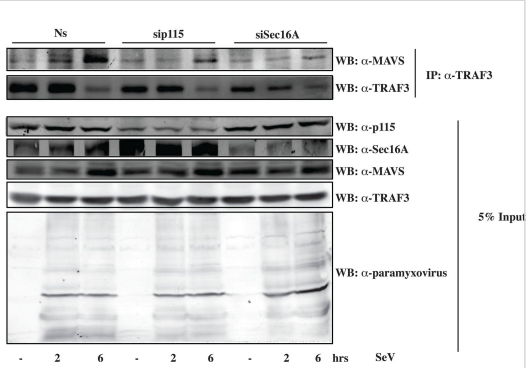
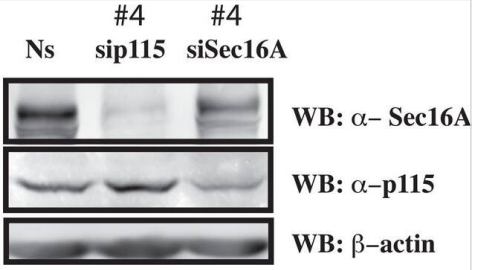
Product Application Details

Applications	Western Blot, Immunocytochemistry/ Immunofluorescence, Immunoprecipitation
Recommended Dilutions	Western Blot 1:2000-1:10000, Immunocytochemistry/ Immunofluorescence 1:10-1:2000, Immunoprecipitation 1-4 ug/mg of lysate
Application Notes	ICC/IF reactivity reported in (PMID: 22740409).

Images

Western Blot: SEC16A Antibody [NB100-1799] - Detection of Human KIAA0310 on HeLa whole cell lysate using NB100-1799. KIAA0310 was also immunoprecipitated by 2 other antibodies.



Requirement of Sec16A and p115 for optimal type I IFN innate immune response in cells exposed to cytosolic DNA and RNA sensor ligands. (A–D) HeLa cells were transfected with nonsilencing (Ns) RNA duplexes or two different sets of siRNA duplexes that specifically target p115 or Sec16A as indicated. 72 h post-transfection, cells were left untreated (Ctl) or stimulated with poly I:C (2.5 ug/ml), poly dA:dT (1 ug/ml) or SeV (200 HAU/ml) for 6 h to 8 h. RNA was extracted and analyzed by RT-qPCR using primers for ifnβ, ifit1, oas1. Data are means +/− S.D. (n=3). * Significantly below the induction response; * P<0.05, ** P<0.01, *** P<0.001. (B and D) Cellular extracts were also prepared and subjected to immunoblot analysis using indicated antibodies. One of three independent experiments with similar results is shown. Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/22792062), licensed under a CC-BY license. Not internally tested by Novus Biologicals.	B 
Sec16A and p115 are required for the proper positioning of TRAF3 along the mitochondrial network.(A) Confocal microscopy of HeLa cells transfected with 40 nM nonsilencing RNA duplexes (panels 1 and 2) or 40 nM siRNA duplexes that specifically target Sec16A (panels 3 and 4) or p115 (panels 5 and 6) and stained for MAVS and endogenous TRAF3 upon no treatment (panels 1, 3 and 5) or SeV infection (200 HAU/ml) for 4 h (panels 2, 4 and 6). Arrows indicate the colocalization of TRAF3 with MAVS. Bars represent 5 um. One of three independent experiments with similar results is shown. (B) p115 and Sec16A were silenced in HeLa cells as described in (A) and infected with SeV for indicated periods of time. Whole-cell lysates were subjected to immunoprecipitation using an anti-TRAF3 (H-20) antibody followed by immunoblotting for the presence of MAVS and TRAF3. Immunoblot analysis against p115, Sec16A, TRAF3 and SeV proteins are also shown (Input). One of two independent experiments with similar results is shown. (C) Densitometric analysis of the binding activity of MAVS to TRAF3 presented in Figures 6B. Data represent the ratio of immunoprecipitated MAVS over immunoprecipitated TRAF3 and are means +/− S.D. of two experiments. Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/22792062), licensed under a CC-BY license. Not internally tested by Novus Biologicals.	B 
Requirement of Sec16A and p115 for optimal type I IFN innate immune response in cells exposed to cytosolic DNA and RNA sensor ligands. (A–D) HeLa cells were transfected with nonsilencing (Ns) RNA duplexes or two different sets of siRNA duplexes that specifically target p115 or Sec16A as indicated. 72 h post-transfection, cells were left untreated (Ctl) or stimulated with poly I:C (2.5 ug/ml), poly dA:dT (1 ug/ml) or SeV (200 HAU/ml) for 6 h to 8 h. RNA was extracted and analyzed by RT-qPCR using primers for ifnβ, ifit1, oas1. Data are means +/− S.D. (n=3). * Significantly below the induction response; * P<0.05, ** P<0.01, *** P<0.001. (B and D) Cellular extracts were also prepared and subjected to immunoblot analysis using indicated antibodies. One of three independent experiments with similar results is shown. Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/22792062), licensed under a CC-BY license. Not internally tested by Novus Biologicals.	D 

Publications

Wang B. Dissecting the Physiological Roles of ULK1/2 in the Mouse Brain Thesis. 2016-01-01 (Mouse)

Rayl M, Truitt M, Held A et al. Penta-EF-Hand Protein Peflin Is a Negative Regulator of ER-To-Golgi Transport. PLoS One 2016-06-08 [PMID: 27276012] (WB)

van Zuylen WJ, Doyon P, Clement JF et al. Proteomic profiling of the TRAF3 interactome network reveals a new role for the ER-to-Golgi transport compartments in innate immunity. PLoS Pathog 2012-01-01 [PMID: 22792062]

Zacharogianni M, Kondylis V, Tang Y et al. ERK7 is a negative regulator of protein secretion in response to amino-acid starvation by modulating Sec16 membrane association. EMBO J 2011-08-01 [PMID: 21847093]

Smith JL, McBride CM, Nataraj PS et al. Trafficking-deficient hERG K+channels linked to long QT syndrome are regulated by a microtubule-dependent quality control compartment in the ER. Am J Physiol Cell Physiol 2011-07-01 [PMID: 21490315]

Hughes H, Stephens DJ. Sec16A defines the site for vesicle budding from the endoplasmic reticulum on exit from mitosis. J Cell Sci 2010-12-01 [PMID: 21045114]

Hughes H, Budnik A, Schmidt K et al. Organisation of human ER-exit sites: requirements for the localisation of Sec16 to transitional ER. J Cell Sci 2009-08-01 [PMID: 19638414]

Townley AK, Feng Y, Schmidt K et al. Efficient coupling of Sec23-Sec24 to Sec13-Sec31 drives COPII-dependent collagen secretion and is essential for normal craniofacial development. J Cell Sci 2008-09-01 [PMID: 18713835]

Abrami L, Kunz B, Iacovache I et al. Palmitoylation and ubiquitination regulate exit of the Wnt signaling protein LRP6 from the endoplasmic reticulum. Proc Natl Acad Sci U S A 2008-04-01 [PMID: 18378904]

Trahey M, Oh HS, Cameron CE, Hay JC. Poliovirus infection transiently increases COPII vesicle budding J Virol 2012 -06-27 [PMID: 22740409] (ICC/IF, Rat)

Baird NL, York J, Nunberg JH. Arenavirus infection induces discrete cytosolic structures for RNA replication J Virol 2012-08-08 [PMID: 22875974] (Primate)

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Products Related to NB100-1799

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

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