

Goat anti-Rabbit IgG (H+L) Cross-Adsorbed Secondary Antibody, Alexa Fluor™ 405

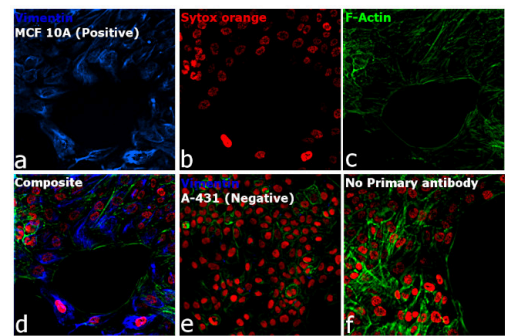
Product Details	
Size	1 mg
Species Reactivity	Rabbit
Host/Isotype	Goat / IgG
Class	Polyclonal
Type	Secondary Antibody
Conjugate	Alexa Fluor™ 405
Excitation/Emission Max	401/422 nm
Immunogen	Gamma Immunoglobins Heavy and Light chains
Form	Liquid
Concentration	2 mg/mL
Purification	purified
Storage buffer	PBS, pH 7.5
Contains	5mM sodium azide
Storage conditions	4° C, store in dark
RRID	AB_221605

Applications	Tested Dilution	Publications
Western Blot (WB)	-	0 Publication
Immunohistochemistry (IHC)	1-10 µg/mL	0 Publication
Immunohistochemistry (Paraffin) (IHC (P))	-	0 Publication
Immunohistochemistry (PFA fixed) (IHC (PFA))	-	0 Publication
Immunohistochemistry (Frozen) (IHC (F))	-	0 Publication
Immunocytochemistry (ICC/IF)	1-10 µg/mL	0 Publication
Flow Cytometry (Flow)	1-10 µg/mL	0 Publication
Functional Assay (Functional)	-	0 Publication
Miscellaneous PubMed (Misc)	-	0 Publication

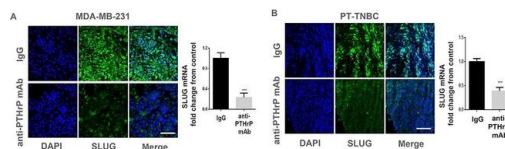
Product Specific Information

Product will be shipped at Room Temperature.

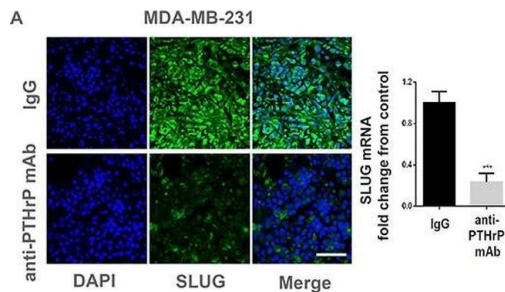
Rabbit IgG (H+L) Cross-Adsorbed Secondary Antibody (A-31556) in ICC/IF
Immunofluorescence analysis of Goat anti-Rabbit IgG (H+L) Cross-Adsorbed Secondary Antibody, Alexa Fluor 405 (Product # A-31556) was performed using MCF 10A (positive model) and A-431 (negative model) cells stained with Vimentin Polyclonal Antibody (Product # PA5-27231). The cells were fixed with 4% paraformaldehyde for 10 minutes, permeabilized with 0.1% Triton™ X-100 for 10 minutes, blocked with 1% BSA for 1 hour and labeled with 2 µg/mL primary antibody for 3 hours at room temperature. Goat anti-Rabbit IgG (H+L) Cross-Adsorbed Secondary Antibody, Alexa Fluor 405 (Product # A-31556) in 0.1% BSA in PBS for 45 minutes at room temperature, was used for detection of Vimentin in the cytoplasm (Panel a: blue). Nuclei (Panel b: red) were stained with SYTOX™ Orange Nucleic Acid Stain (Product # S11368). F-actin was stained with Alexa Fluor® 488 Phalloidin (Product # A12379, 1:300) (Panel c: green). Panel d represents the composite image. The specificity of the secondary antibody was proved by the absence of signal in A-431 (negative model for vimentin) due to no primary antibody binding (Panel e). Nonspecific staining was not observed with secondary antibody alone (panel f). The images were captured at 20X magnification.



Rabbit IgG (H+L) Cross-Adsorbed Secondary Antibody (A-31556) in ICC/IF
Inhibition of EMT and CSC markers in bone lesions following in vivo administration of anti-PTHrP mAb: IF staining and mRNA level for Slug in MDA-MB-231 PthlhWT injected mice (A) and PT-TNBC PthlhWT injected mice (B). IF staining and mRNA level for ALDH1 in MDA-MB-231 PthlhWT injected mice (C) and PT-TNBC PthlhWT injected mice (D). IF staining and mRNA level for E-cadherin in MDA-MB-231 PthlhWT injected mice (E) and PT-TNBC PthlhWT injected mice (F). IF staining and mRNA level for vimentin in MDA-MB-231 PthlhWT injected mice (G) and PT-TNBC PthlhWT injected mice (H). Scale bar = 100 mm, n = 10, p < 0.001. (I) Diagram summarizing mechanistic involvement of PTHrP in EMT and CSC number control and therapeutic implications. In TNBC cells in vitro, Pthlh ablation inhibits vimentin, Slug, ALDH1, and CD49f, enhances E-cadherin and lowers CD44/CD24 ratio and mammosphere formation, while our anti-PTHrP mAb inhibits KI67 and cell survival and motility. In vivo, the mAb inhibits vimentin, Slug, and ALDH1 in TNBC xenografts and increases E-cadherin, leading to growth inhibition of established TNBC bone tumors. Image collected and cropped by CiteAb from the following publication (<https://pubmed.ncbi.nlm.nih.gov/35720668>), licensed under a CC BY license.



Rabbit IgG (H+L) Cross-Adsorbed Secondary Antibody (A-31556) in ICC/IF
Inhibition of EMT and CSC markers in bone lesions following in vivo administration of anti-PTHrP mAb: IF staining and mRNA level for Slug in MDA-MB-231 PthlhWT injected mice (A) and PT-TNBC PthlhWT injected mice (B). IF staining and mRNA level for ALDH1 in MDA-MB-231 PthlhWT injected mice (C) and PT-TNBC PthlhWT injected mice (D). IF staining and mRNA level for E-cadherin in MDA-MB-231 PthlhWT injected mice (E) and PT-TNBC PthlhWT injected mice (F). IF staining and mRNA level for vimentin in MDA-MB-231 PthlhWT injected mice (G) and PT-TNBC PthlhWT injected mice (H). Scale bar = 100 mm, n = 10, p < 0.001. (I) Diagram summarizing mechanistic involvement of PTHrP in EMT and CSC number control and therapeutic implications. In TNBC cells in vitro, Pthlh ablation inhibits vimentin, Slug, ALDH1, and CD49f, enhances E-cadherin and lowers CD44/CD24 ratio and mammosphere formation, while our anti-PTHrP mAb inhibits KI67 and cell survival and motility. In vivo, the mAb inhibits vimentin, Slug, and ALDH1 in TNBC xenografts and increases E-cadherin, leading to growth inhibition of established TNBC bone tumors. Image collected and cropped by CiteAb from the following publication (<https://pubmed.ncbi.nlm.nih.gov/35720668>), licensed under a CC BY license.



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MAPK4 inhibits the early aberrant activation of B cells in rheumatoid arthritis by promoting the IRF4-SHIP1 signaling pathway. *Cell Death Dis* (2025)

Nicotinamide mononucleotide restores impaired metabolism, endothelial cell proliferation and angiogenesis in old sedentary male mice. *iScience* (2025)

Basal forebrain innervation of the amygdala: an anatomical and computational exploration. *Brain Struct Funct* (2025)

RAB18 regulates extrahepatic siRNA-mediated gene silencing efficacy. *Mol Ther Nucleic Acids* (2024)

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