

Anti-VASP (C-terminal region) Antibody Catalog # AN2006

Specification

Anti-VASP (C-terminal region) Antibody - Product Information

Application	WB
Primary Accession	P50552
Host	Mouse
Clonality	Mouse Monoclonal
Isotype	IgG1
Calculated MW	39830

Anti-VASP (C-terminal region) Antibody - Additional Information

Gene ID 7408

Other Names

vasodilator-stimulated phosphoprotein

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

Anti-VASP (C-terminal region) Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Shipping

Blue Ice

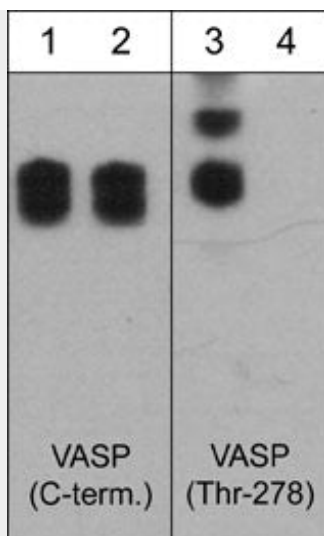
Anti-VASP (C-terminal region) Antibody - Protocols

Provided below are standard protocols that you may find useful for product applications.

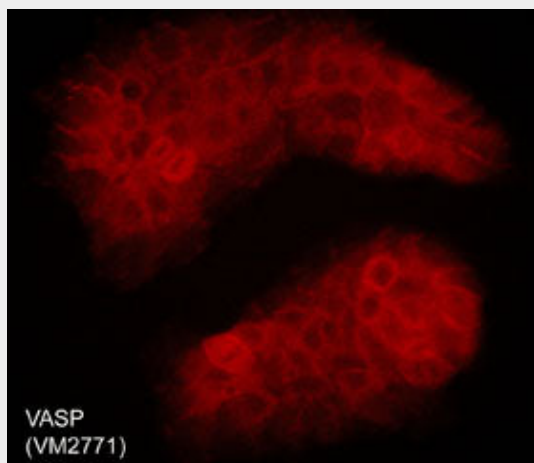
- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

Anti-VASP (C-terminal region) Antibody - Images





Western blot image of human A431 cells stimulated with calyculin A (100 nM) for 30 min. The blots were untreated (lanes 1 & 3) or treated with lambda phosphatase (lanes 2 & 4), then probed with mouse monoclonal VASP (C-term.) antibody (lanes 1 & 2) or rabbit polyclonal VASP (Thr-278) phospho-specific antibody (lanes 3 & 4).



Immunocytochemical labeling of VASP in aldehyde-fixed and NP-40-permeabilized A431 cells. The cells were labeled with mouse monoclonal VASP (C-terminal region) antibody, then the antibody was detected using appropriate secondary antibody conjugated to DyLight® 594.

Anti-VASP (C-terminal region) Antibody - Background

Actin filament tethering and bundling are important mechanisms involved in actin superstructure assembly. The ENA/VASP family includes VASP, mena, and Ena-Vasp-like (EVL). These multidomain proteins localize to the leading edge of filopodia where they associate with AFs, interact with profilin, and compete with capping proteins at the barbed end of AFs. Artificial relocation of VASP from the plasma membrane to mitochondrial membranes inhibits filopodial formation and axon branching, while deletion of all three ENA/VASP proteins produces defects in cortical axon-tract formation. Regulation of VASP protein activity occurs through phosphorylation at Ser-157, Ser-239, and Thr-278. AMPK phosphorylates Thr-278, leading to impaired actin stress fiber assembly and changes in cell morphology.