

p-VASP (16C2): sc-101439

BACKGROUND

The Wiskott-Aldrich syndrome (WAS) is characterized by thrombocytopenia, eczema, defects in cell-mediated and humoral immunity and a propensity for lymphoproliferative diseases. The syndrome is the result of a mutation in the gene encoding a proline-rich protein termed WASP. A distantly related protein, VASP (vasodilator-stimulated phosphoprotein), is involved in the maintenance of cytoarchitecture by interacting with Actin-like filaments. VASP shares a limited degree of homology with the amino-terminus of WASP, which is frequently mutated in WAS patients. An established substrate of cAMP and cGMP dependent kinases, VASP is phosphorylated on a regulatory serine residue 157 and localizes to focal adhesions, microfilaments and highly active regions of the plasma membrane. VASP is also phosphorylated on Serine 239 by cGMP-dependent protein kinase.

REFERENCES

1. Reinhard, M., et al. 1992. The 46/50 kDa phosphoprotein VASP purified from human platelets is a novel protein associated with Actin filaments and focal contacts. *EMBO J.* 11: 2063-2070.
2. Butt, E., et al. 1994. CAMP- and cGMP-dependent protein kinase phosphorylation sites of the focal adhesion vasodilator-stimulated phosphoprotein (VASP) *in vitro* and in intact human platelets. *J. Biol. Chem.* 269: 14509-14517.

CHROMOSOMAL LOCATION

Genetic locus: VASP (human) mapping to 19q13.32; Vasp (mouse) mapping to 7 A3.

SOURCE

p-VASP (16C2) is a mouse monoclonal antibody raised against synthetic VASP of human origin, phosphorylated at Serine 239.

PRODUCT

Each vial contains 50 µg IgG₁ in 0.5 ml of 2 x PBS with < 0.1% sodium azide, 0.1% gelatin, PEG, and sucrose.

APPLICATIONS

p-VASP (16C2) is recommended for detection of Ser 239 phosphorylated VASP of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500), flow cytometry (1 µg per 1 x 10⁶ cells) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for VASP siRNA (h): sc-29516, VASP siRNA (m): sc-36809, VASP shRNA Plasmid (h): sc-29516-SH, VASP shRNA Plasmid (m): sc-36809-SH, VASP shRNA (h) Lentiviral Particles: sc-29516-V and VASP shRNA (m) Lentiviral Particles: sc-36809-V.

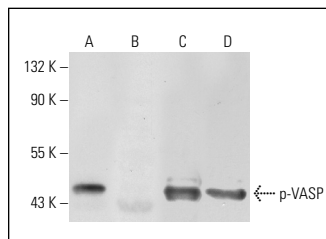
Molecular Weight of p-VASP: 50 kDa.

Positive Controls: VASP (h): 293T Lysate: sc-114829, human platelet extract: sc-363773 or NIH/3T3 + forskolin cell lysate: sc-24741.

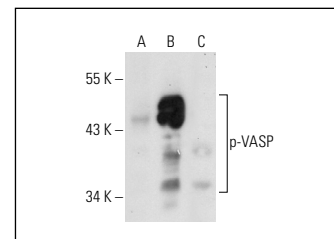
STORAGE

Store at 4° C, ****DO NOT FREEZE****. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

DATA



Western blot analysis of VASP phosphorylation in untreated (A, C), and lambda protein phosphatase (sc-200312A) treated (B, D) human platelet extract. Antibodies tested include p-VASP (16C2): sc-101439 (A, B) and VASP (A-11): sc-46668 (C, D).



p-VASP (16C2): sc-101439. Western blot analysis of VASP phosphorylation in non-transfected (A), untreated human VASP transfected: sc-114829 (B) and lambda protein phosphatase (sc-200312A) treated human VASP transfected: sc-114829 (C) 293T whole cell lysates.

SELECT PRODUCT CITATIONS

1. Korkmaz, B., et al. 2011. Activation of MEK1/ERK1/2/iNOS/sGC/PKG pathway associated with peroxynitrite formation contributes to hypotension and vascular hyporeactivity in endotoxemic rats. *Nitric Oxide* 24: 160-172.
2. Kim, M., et al. 2013. GLP-1 receptor activation and Epac2 link atrial natriuretic peptide secretion to control of blood pressure. *Nat. Med.* 19: 567-575.
3. Limmer, F., et al. 2015. Regulation of the Na⁺-K⁺-2Cl⁻ cotransporter (NKCC2) by cGMP/cGKI after furosemide administration. *FEBS J.* 282: 3786-3798.
4. Murat, N., et al. 2016. Resveratrol protects and restores endothelium-dependent relaxation in hypercholesterolemic rabbit corpus cavernosum. *J. Sex. Med.* 13: 12-21.
5. Cesarini, V., et al. 2017. Type 5 phosphodiesterase regulates glioblastoma multiforme aggressiveness and clinical outcome. *Oncotarget* 8: 13223-13239.
6. Zosen, D.V., et al. 2018. ERK1/2 inhibition increases dopamine release from differentiated PC12 cells. *Neurosci. Lett.* 684: 6-12.
7. Xia, C., et al. 2019. MRP14 enhances the ability of macrophage to recruit T cells and promotes obesity-induced Insulin resistance. *Int. J. Obes.* 43: 2434-2447.
8. Qin, L., et al. 2021. Chlorogenic acid alleviates hyperglycemia-induced cardiac fibrosis through activation of the NO/cGMP/PKG pathway in cardiac fibroblasts. *Mol. Nutr. Food Res.* 65: e2000810.
9. de la Fuente-Alonso, A., et al. 2021. Aortic disease in Marfan syndrome is caused by overactivation of sGC-PRKG signaling by NO. *Nat. Commun.* 12: 2628.

RESEARCH USE

For research use only, not for use in diagnostic procedures.