SANTA CRUZ BIOTECHNOLOGY, INC.

p-VASP (5C6): sc-101440

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

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.
3. Reinhard, M., et al. 1995. Identification, purification and characterization of a zyxin-related protein that binds the focal adhesion and microfilament protein VASP (vasodilator-stimulated phosphoprotein). Proc. Natl. Acad. Sci. USA 92: 7956-7960.
4. Remold-O'Donnell, E., et al. 1996. Defects in Wiskott-Aldrich syndrome blood cells. Blood 87: 2621-2631.
5. Stewart, D.M., et al. 1996. Studies of the expression of the Wiskott-Aldrich syndrome protein. J. Clin. Invest. 97: 2627-2634.
6. Schindelhauer, D., et al. 1996. Wiskott-Aldrich syndrome: no strict genotype-phenotype correlations but clustering of missense mutations in the amino-terminal part of the WASP gene product. Hum. Genet. 98: 68-76.

CHROMOSOMAL LOCATION

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

SOURCE

p-VASP (5C6) is a mouse monoclonal antibody raised against synthetic VASP of human origin, phosphorylated at serine 157.

PRODUCT

Each vial contains 50 µg IgG_1 in 0.5 ml of 2 x PBS with < 0.1% sodium azide, 0.1% gelatin, PEG, and sucrose.

STORAGE

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

APPLICATIONS

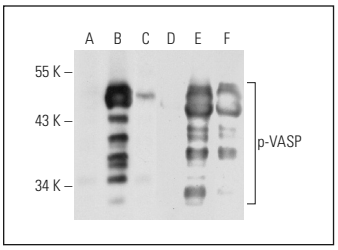
p-VASP (5C6) is recommended for detection of Ser 157 phosphorylated VASP of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500), flow cytometry (1 µg per 1 x 10^6 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.

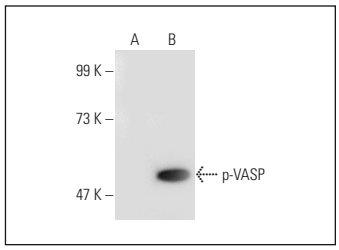
Molecular Weight of p-VASP: 50 kDa.

Positive Controls: VASP (h): 293T Lysate: sc-114829, NIH/3T3 + forskolin cell lysate: sc-24741 or VASP (m): 293T Lysate: sc-127758.

DATA



Western blot analysis of VASP phosphorylation in non-transfected: sc-117752 (**A**,**D**), untreated human VASP transfected: sc-114829 (**B**,**E**) and lambda protein phosphatase (sc-200312A) treated human VASP transfected: sc-114829 (**C**,**F**) 293T whole cell lysates. Antibodies tested include p-VASP (5C6): sc-101440 (**A**,**B**,**C**) and VASP (A-11): sc-46668 (**D**,**E**,**F**).



p-VASP (5C6): sc-101440. Western blot analysis of VASP phosphorylation in non-transfected: sc-117752 (**A**) and mouse VASP transfected: sc-127759 (**B**) 293T whole cell lysates.

SELECT PRODUCT CITATIONS

1. Niimi, K., et al. 2012. β2-Agonists upregulate PDE4 mRNA but not protein or activity in human airway smooth muscle cells from asthmatic and nonasthmatic volunteers. Am. J. Physiol. Lung Cell. Mol. Physiol. 302: L334-L342.

RESEARCH USE

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

PROTOCOLS

See our web site at www.scbt.com for detailed protocols and support products.