SANTA CRUZ BIOTECHNOLOGY, INC.

p-VASP (Ser 239): sc-23507

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.

CHROMOSOMAL LOCATION

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

SOURCE

p-VASP (Ser 239) is available as either goat (sc-23507) or rabbit (sc-23507-R) polyclonal antibody raised against a short amino acid sequence containing Ser 239 phosphorylated VASP of human origin.

PRODUCT

Each vial contains 100 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-23507 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

p-VASP (Ser 239) 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) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

p-VASP (Ser 239) is also recommended for detection of correspondingly phosphorylated VASP in additional species, including canine and bovine.

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 whole cell lysate: sc-363773 or NIH/3T3 + forskolin cell lysate: sc-24741.

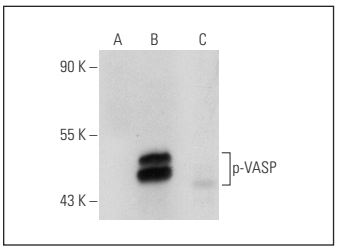
STORAGE

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

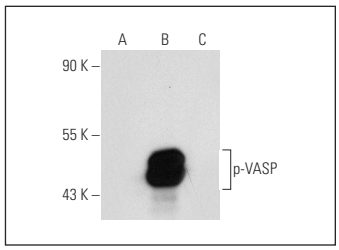
RESEARCH USE

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

DATA



p-VASP (Ser 239): sc-23507. Western blot analysis of VASP phosphorylation in non-transfected: sc-117752 (**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.



p-VASP (Ser 239)-R: sc-23507-R. Western blot analysis of VASP phosphorylation in non-transfected: sc-117752 (**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. Rivero-Vilches, F.J., et al. 2003. Differential relaxing responses to particulate or soluble guanylyl cyclase activation on endothelial cells: a mechanism dependent on PKG-I α activation by NO/cGMP. Am. J. Physiol., Cell Physiol. 285: C891-C898.
2. Matsumoto, T., et al. 2007. Mechanisms underlying the chronic pioglitazone treatment-induced improvement in the impaired endothelium-dependent relaxation seen in aortas from diabetic rats. Free Radic. Biol. Med. 42: 993-1007.
3. Isenberg, J.S., et al. 2008. Thrombospondin-1 stimulates platelet aggregation by blocking the antithrombotic activity of nitric oxide/cGMP signaling. Blood 111: 613-623.
4. Mouchaers, K.T., et al. 2009. Endothelin receptor blockade combined with phosphodiesterase-5 inhibition increases right ventricular mitochondrial capacity in pulmonary arterial hypertension. Am. J. Physiol. Heart Circ. Physiol. 297: H200-H207.
5. Sánchez-Ruiloba, L., et al. 2014. Protein kinase D interacts with neuronal nitric oxide synthase and phosphorylates the activatory residue serine 1412. PLoS ONE 9: 1-17.
6. Aicart-Ramos, C., et al. 2014. Protein kinase D activity controls endothelial nitric oxide synthesis. J. Cell Sci. 127: 3360-3372.

PROTOCOLS

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