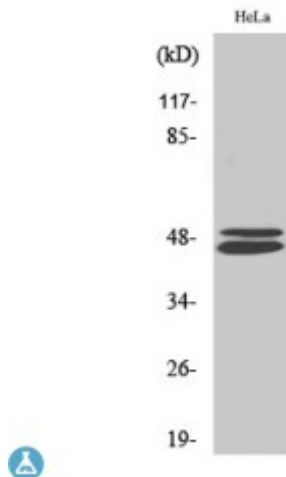


Anti-VASP antibody



Description	Rabbit polyclonal to VASP.
Model	STJ96219
Host	Rabbit
Reactivity	Human, Mouse, Rat, Simian
Applications	ELISA, IF, IHC, WB
Immunogen	Synthesized peptide derived from human VASP around the non-phosphorylation site of S238.
Immunogen Region	180-260 aa
Gene ID	7408
Gene Symbol	VASP
Dilution range	WB 1:500-1:2000IHC 1:100-1:300IF 1:200-1:1000ELISA 1:20000
Specificity	VASP Polyclonal Antibody detects endogenous levels of VASP protein.
Tissue Specificity	Highly expressed in platelets.
Purification	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen.
Note	For Research Use Only (RUO).
Protein Name	Vasodilator-stimulated phosphoprotein VASP
Molecular Weight	46/50 kDa
Clonality	Polyclonal

Conjugation	Unconjugated
Isotype	IgG
Formulation	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Concentration	1 mg/ml
Storage Instruction	Store at -20°C, and avoid repeat freeze-thaw cycles.
Database Links	HGNC:12652OMIM:601703
Alternative Names	Vasodilator-stimulated phosphoprotein VASP
Function	Ena/VASP proteins are actin-associated proteins involved in a range of processes dependent on cytoskeleton remodeling and cell polarity such as axon guidance, lamellipodial and filopodial dynamics, platelet activation and cell migration. VASP promotes actin filament elongation. It protects the barbed end of growing actin filaments against capping and increases the rate of actin polymerization in the presence of capping protein. VASP stimulates actin filament elongation by promoting the transfer of profilin-bound actin monomers onto the barbed end of growing actin filaments. Plays a role in actin-based mobility of <i>Listeria monocytogenes</i> in host cells. Regulates actin dynamics in platelets and plays an important role in regulating platelet aggregation.
Sequence and Domain Family	The EVH2 domain is comprised of 3 regions. Block A is a thymosin-like domain required for G-actin binding. The KLKR motif within this block is essential for the G-actin binding and for actin polymerization. Block B is required for F-actin binding and subcellular location, and Block C for tetramerization.; The WH1 domain mediates interaction with XIRP1.
Cellular Localization	Cytoplasm. Cytoplasm, cytoskeleton. Cell junction, focal adhesion. Cell junction, tight junction Cell projection, lamellipodium membrane. Cell projection, filopodium membrane. Targeted to stress fibers and focal adhesions through interaction with a number of proteins including MRL family members. Localizes to the plasma membrane in protruding lamellipodia and filopodial tips. Stimulation by thrombin or PMA, also translocates VASP to focal adhesions. Localized along the sides of actin filaments throughout the peripheral cytoplasm under basal conditions. In pre-apoptotic cells, colocalizes with MEFV in large specks (pyroptosomes).
Post-translational Modifications	Major substrate for cAMP-dependent (PKA) and cGMP-dependent protein kinase (PKG) in platelets. The preferred site for PKA is Ser-157, the preferred site for PKG/PRKG1, Ser-239. In ADP-activated platelets, phosphorylation by PKA or PKG on Ser-157 leads to fibrinogen receptor inhibition. Phosphorylation on Thr-278 requires prior phosphorylation on Ser-157 and Ser-239. In response to phorbol ester (PMA) stimulation, phosphorylated by PKC/PRKCA. In response to thrombin, phosphorylated by both PKC and ROCK1. Phosphorylation at Thr-278 by AMPK does not require prior phosphorylation at Ser-157 or Ser-239. Phosphorylation at Ser-157 by PKA is required for localization to the tight junctions in epithelial cells. Phosphorylation modulates F-actin binding, actin filament elongation and platelet activation. Phosphorylation at Ser-322 by AMPK also alters actin filament binding. Carbon monoxide (CO) promotes phosphorylation at Ser-157, while nitric oxide (NO) promotes phosphorylation at Ser-157, but also at Ser-239. Response to NO and CO is blunted in platelets from diabetic

patients, and VASP is not phosphorylated efficiently at Ser-157 and Ser-239.

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