

Product name:	WASP (phospho Tyr290) Rabbit Polyclonal Antibody
Cat number:	ABN05628
Conjugate:	Unconjugated
Size:	100µL
Clone:	Polyclonal
Concentration:	1mg/ml
Host:	Rabbit
Isotype:	IgG
Immunogen:	The antiserum was produced against synthesized peptide derived from human WASP around the phosphorylation site of Tyr290. AA range:256-305
Reactivity:	Human,Mouse
Applications:	WB 1:500-1:2000,IHC 1:100-1:300,ICC/IF 1:50-1:200,ELISA 1:5000-1:10000
Molecular Weight:	60kDa
Purification:	Affinity purification
Form:	Liquid
Buffer:	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% New type preservative N.
Storage:	Store at 4°C short term. Aliquot and store at -20°C for 12 months. Avoid freeze/thaw cycles.

Background:

The Wiskott-Aldrich syndrome (WAS) family of proteins share similar domain structure, and are involved in transduction of signals from receptors on the cell surface to the actin cytoskeleton. The presence of a number of different motifs suggests that they are regulated by a number of different stimuli, and interact with multiple proteins. Recent studies have demonstrated that these proteins, directly or indirectly, associate with the small GTPase, Cdc42, known to regulate formation of actin filaments, and the cytoskeletal organizing complex, Arp2/3. Wiskott-Aldrich syndrome is a rare, inherited, X-linked, recessive disease characterized by immune dysregulation and microthrombocytopenia, and is caused by mutations in the WAS gene. The WAS gene product is a cytoplasmic protein, expressed exclusively in hematopoietic cells, which show signalling and cytoskeletal abnormalities in WAS patients. A disease:Defects in WAS are a cause of X-linked severe congenital neutropenia (XLN) [MIM:300299]. XLN is an X-linked immunodeficiency syndrome characterized by recurrent major bacterial infections, severe congenital neutropenia, and monocytopenia.,disease:Defects in WAS are the cause of thrombocytopenia type 1 (THC1) [MIM:313900]. Thrombocytopenia is defined by a decrease in the number of platelets in circulating blood, resulting in the potential for increased bleeding and decreased ability for clotting.,disease:Defects in WAS are the cause of Wiskott-Aldrich syndrome (WAS) [MIM:301000]; also known as eczema-thrombocytopenia-immunodeficiency syndrome. WAS is an X-linked recessive immunodeficiency characterized by eczema, thrombocytopenia, recurrent infections, and bloody diarrhea. Death usually occurs before age 10.,domain:The CRIB (Cdc42/Rac-interactive-binding) region binds to the C-terminal WH2 domain in the autoinhibited state of the protein. Binding of Rho-type GTPases to the CRIB induces a conformation change and leads to activation.,domain:The WH1 (Wasp homology 1) domain may bind a Pro-rich ligand.,function:Effector protein for Rho-type GTPases, providing a link with the Arp2/3 complex that regulates the structure and dynamics of the actin cytoskeleton. Important for efficient actin polymerization. Possible regulator of lymphocyte and platelet function.,online information:WAS mutation db,online information:Wiskott-Aldrich syndrome protein entry,similarity:Contains 1 CRIB domain.,similarity:Contains 1 WH1 domain.,similarity:Contains 1 WH2 domain.,subunit:Binds to CDC42, RAC, NCK, FYN, SRC kinase FGR, BTK, ABL, PSTPIP1, WIP, and to the p85 subunit of PLC-gamma. Binds the Arp2/3 complex.,tissue specificity:Expressed predominantly in the thymus. Also found, to a much lesser extent, in the spleen.,