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WAS Antibody(Center)

Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP19544c

Product Information

Application	WB, E
Primary Accession	P42768
Other Accession	NP_000368.1
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Clone Names	RB40707
Calculated MW	52913
Antigen Region	205-234

Additional Information

Gene ID	7454
Other Names	Wiskott-Aldrich syndrome protein, WASp, WAS, IMD2
Target/Specificity	This WAS antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 205-234 amino acids from the Central region of human WAS.
Dilution	WB~~1:1000 E~~Use at an assay dependent concentration.
Format	Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.
Storage	Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	WAS Antibody(Center) is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	WAS
Synonyms	IMD2
Function	Effector protein for Rho-type GTPases that regulates actin filament reorganization via its interaction with the Arp2/3 complex (PubMed:12235133,

PubMed:12769847, PubMed:16275905). Important for efficient actin polymerization (PubMed:12235133, PubMed:16275905, PubMed:8625410). Possible regulator of lymphocyte and platelet function (PubMed:9405671). Mediates actin filament reorganization and the formation of actin pedestals upon infection by pathogenic bacteria (PubMed:18650809). In addition to its role in the cytoplasmic cytoskeleton, also promotes actin polymerization in the nucleus, thereby regulating gene transcription and repair of damaged DNA (PubMed:20574068). Promotes homologous recombination (HR) repair in response to DNA damage by promoting nuclear actin polymerization, leading to drive motility of double-strand breaks (DSBs) (PubMed:29925947).

Cellular Location Cytoplasm, cytoskeleton. Nucleus

Tissue Location Expressed predominantly in the thymus. Also found, to a much lesser extent, in the spleen.

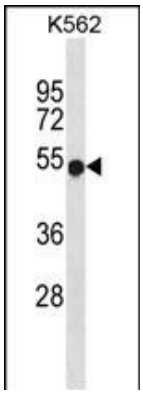
Background

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 transcript variant arising as a result of alternative promoter usage, and containing a different 5' UTR sequence, has been described, however, its full-length nature is not known.

References

Burns, S.O., et al. Blood 115(26):5355-5365(2010)
Taylor, M.D., et al. Sci Transl Med 2 (37), 37RA44 (2010) :
Rajmohan, R., et al. FEMS Yeast Res. 9(8):1226-1235(2009)
Dovas, A., et al. J. Cell. Sci. 122 (PT 21), 3873-3882 (2009) :
Ameratunga, R., et al. N. Z. Med. J. 122(1304):46-53(2009)

Images



WAS Antibody (Center) (Cat. #AP19544c) western blot analysis in K562 cell line lysates (35ug/lane).This demonstrates the WAS antibody detected the WAS protein (arrow).