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WAS Antibody(Center)
Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP19544c

Specification

WAS Antibody(Center) - Product Information

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

WAS Antibody(Center) - 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.

WAS Antibody(Center) - 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

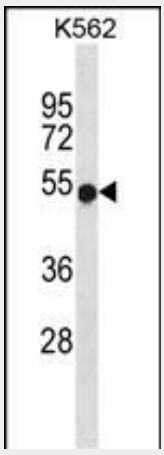
Tissue Location
Expressed predominantly in the thymus. Also found, to a much lesser extent, in the spleen.

WAS Antibody(Center) - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

WAS Antibody(Center) - 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).

WAS Antibody(Center) - 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.

WAS Antibody(Center) - 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) :
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