

**WAS Antibody (Center) Blocking Peptide**  
**Synthetic peptide**  
**Catalog # BP9873c****Specification**

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**WAS Antibody (Center) Blocking Peptide - Product Information**Primary Accession [P42768](#)**WAS Antibody (Center) Blocking Peptide - Additional Information****Gene ID** 7454**Other Names**

Wiskott-Aldrich syndrome protein, WASp, WAS, IMD2

**Format**

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

**Storage**

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

**Precautions**

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

**WAS Antibody (Center) Blocking Peptide - 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:<a href="http://www.uniprot.org/citations/12235133" target="\_blank">12235133</a>, PubMed:<a href="http://www.uniprot.org/citations/12769847" target="\_blank">12769847</a>, PubMed:<a href="http://www.uniprot.org/citations/16275905" target="\_blank">16275905</a>). Important for efficient actin polymerization (PubMed:<a href="http://www.uniprot.org/citations/8625410" target="\_blank">8625410</a>, PubMed:<a href="http://www.uniprot.org/citations/12235133" target="\_blank">12235133</a>, PubMed:<a href="http://www.uniprot.org/citations/16275905" target="\_blank">16275905</a>). Possible regulator of lymphocyte and platelet function (PubMed:<a href="http://www.uniprot.org/citations/9405671" target="\_blank">9405671</a>). Mediates actin filament reorganization and the formation of actin pedestals upon infection by pathogenic bacteria (PubMed:<a href="http://www.uniprot.org/citations/18650809" target="\_blank">18650809</a>). 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:<a href="http://www.uniprot.org/citations/20574068" target="\_blank">20574068</a>). 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:<a href="http://www.uniprot.org/citations/29925947" target="\_blank">29925947</a>).

**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) Blocking Peptide - Protocols**

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

- [Blocking Peptides](#)

**WAS Antibody (Center) Blocking Peptide - Images****WAS Antibody (Center) Blocking Peptide - 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.

**WAS Antibody (Center) Blocking Peptide - References**

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) Cammer, M., et al. J. Biol. Chem. 284(35):23302-23311(2009)Zhang, J., et al. J. Biol. Chem. 284(32):21659-21669(2009)Ameratunga, R., et al. N. Z. Med. J. 122(1304):46-53(2009)