

**ABL2 Antibody (R432) Blocking peptide**  
**Synthetic peptide**  
**Catalog # BP3018b****Specification**

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**ABL2 Antibody (R432) Blocking peptide - Product Information**Primary Accession [P42684](#)**ABL2 Antibody (R432) Blocking peptide - Additional Information****Gene ID 27****Other Names**

Abelson tyrosine-protein kinase 2, Abelson murine leukemia viral oncogene homolog 2, Abelson-related gene protein, Tyrosine-protein kinase ARG, ABL2, ABLL, ARG

**Target/Specificity**

The synthetic peptide sequence used to generate the antibody [AP3018b](/product/products/AP3018b) was selected from the R432 region of human ABL. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

**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.

**ABL2 Antibody (R432) Blocking peptide - Protein Information****Name** ABL2**Synonyms** ABLL, ARG**Function**

Non-receptor tyrosine-protein kinase that plays an ABL1- overlapping role in key processes linked to cell growth and survival such as cytoskeleton remodeling in response to extracellular stimuli, cell motility and adhesion and receptor endocytosis. Coordinates actin remodeling through tyrosine phosphorylation of proteins controlling cytoskeleton dynamics like MYH10 (involved in movement); CTTN (involved in signaling); or TUBA1 and TUBB (microtubule subunits). Binds directly F-actin and regulates actin cytoskeletal structure through its F-actin- bundling activity. Involved in the regulation of cell adhesion and motility through phosphorylation of key regulators of these processes such as CRK, CRKL, DOK1 or ARHGAP35. Adhesion-dependent phosphorylation of ARHGAP35 promotes its association with RASA1, resulting in recruitment of ARHGAP35 to the

cell periphery where it inhibits RHO. Phosphorylates multiple receptor tyrosine kinases like PDGFRB and other substrates which are involved in endocytosis regulation such as RIN1. In brain, may regulate neurotransmission by phosphorylating proteins at the synapse. ABL2 also acts as a regulator of multiple pathological signaling cascades during infection. Pathogens can hijack ABL2 kinase signaling to reorganize the host actin cytoskeleton for multiple purposes, like facilitating intracellular movement and host cell exit. Finally, functions as its own regulator through autocatalytic activity as well as through phosphorylation of its inhibitor, ABL1. Positively regulates chemokine-mediated T-cell migration, polarization, and homing to lymph nodes and immune-challenged tissues, potentially via activation of NEDD9/HEF1 and RAP1 (By similarity).

**Cellular Location**

Cytoplasm, cytoskeleton {ECO:0000250|UniProtKB:Q4JIM5}

**Tissue Location**

Widely expressed.

**ABL2 Antibody (R432) Blocking peptide - Protocols**

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

- [Blocking Peptides](#)

**ABL2 Antibody (R432) Blocking peptide - Images****ABL2 Antibody (R432) Blocking peptide - Background**

ABL2 is a cytoplasmic tyrosine kinase which is closely related to but distinct from ABL1. The similarity of the proteins includes the tyrosine kinase domains and extends amino-terminal to include the SH2 and SH3 domains. ABL2 is expressed in both normal and tumor cells. The ABL2 gene product is expressed as two variants bearing different amino termini, both approximately 12-kb in length. The peptide used to generate this antibody is 100% conserved between ABL2 and ABL1.

**ABL2 Antibody (R432) Blocking peptide - References**

Cao, C., et al., Biochemistry 42(35):10348-10353 (2003). Cao, C., et al., J. Biol. Chem. 278(32):29667-29675 (2003). Kruh, G.D., et al., Proc. Natl. Acad. Sci. U.S.A. 87(15):5802-5806 (1990). Kruh, G.D., et al., Science 234(4783):1545-1548 (1986). Bianchi, C., et al., FEBS Lett. 527 (1-3), 216-222 (2002).