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ABL1 Antibody (N-term K219) Blocking Peptide

Synthetic peptide
Catalog # BP7694a

Specification

ABL1 Antibody (N-term K219) Blocking Peptide - Product Information

Primary Accession P00519

ABL1 Antibody (N-term K219) Blocking Peptide - Additional Information

Gene ID 25

Other Names
Tyrosine-protein kinase ABL1, Abelson murine leukemia viral oncogene homolog 1, Abelson tyrosine-protein kinase 1, Proto-oncogene c-Abl, p150, ABL1, ABL, JTK7

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP7694a was selected from the N-term region of human ABL1. 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.

ABL1 Antibody (N-term K219) Blocking Peptide - Protein Information

Name ABL1

Synonyms ABL, JTK7

Function
Non-receptor tyrosine-protein kinase that plays a role in many key processes linked to cell growth and survival such as cytoskeleton remodeling in response to extracellular stimuli, cell motility and adhesion, receptor endocytosis, autophagy, DNA damage response and apoptosis. Coordinates actin remodeling through tyrosine phosphorylation of proteins controlling cytoskeleton dynamics like WASF3 (involved in branch formation); ANXA1 (involved in membrane anchoring); DBN1, DBNL, CTTN, RAPH1 and ENAH (involved in signaling); or MAPT and PXN (microtubule-binding proteins). Phosphorylation of WASF3 is critical for the stimulation of lamellipodia formation and cell migration. Involved in the regulation of cell adhesion and motility through phosphorylation of key regulators of these processes such as BCAR1, CRK, CRKL, DOK1, EFS or NEDD9 (PubMed:<a

href="http://www.uniprot.org/citations/22810897" target="_blank">22810897). Phosphorylates multiple receptor tyrosine kinases and more particularly promotes endocytosis of EGFR, facilitates the formation of neuromuscular synapses through MUSK, inhibits PDGFRB-mediated chemotaxis and modulates the endocytosis of activated B-cell receptor complexes. Other substrates which are involved in endocytosis regulation are the caveolin (CAV1) and RIN1. Moreover, ABL1 regulates the CBL family of ubiquitin ligases that drive receptor down-regulation and actin remodeling. Phosphorylation of CBL leads to increased EGFR stability. Involved in late-stage autophagy by regulating positively the trafficking and function of lysosomal components. ABL1 targets to mitochondria in response to oxidative stress and thereby mediates mitochondrial dysfunction and cell death. In response to oxidative stress, phosphorylates serine/threonine kinase PRKD2 at 'Tyr-717' (PubMed:28428613). ABL1 is also translocated in the nucleus where it has DNA-binding activity and is involved in DNA-damage response and apoptosis. Many substrates are known mediators of DNA repair: DDB1, DDB2, ERCC3, ERCC6, RAD9A, RAD51, RAD52 or WRN. Activates the proapoptotic pathway when the DNA damage is too severe to be repaired. Phosphorylates TP73, a primary regulator for this type of damage- induced apoptosis. Phosphorylates the caspase CASP9 on 'Tyr-153' and regulates its processing in the apoptotic response to DNA damage. Phosphorylates PSMA7 that leads to an inhibition of proteasomal activity and cell cycle transition blocks. ABL1 acts also as a regulator of multiple pathological signaling cascades during infection. Several known tyrosine-phosphorylated microbial proteins have been identified as ABL1 substrates. This is the case of A36R of Vaccinia virus, Tir (translocated intimin receptor) of pathogenic E.coli and possibly Citrobacter, CagA (cytotoxin-associated gene A) of H.pylori, or AnkA (ankyrin repeat-containing protein A) of A.phagocytophilum. Pathogens can highjack ABL1 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, ABI1. Regulates T-cell differentiation in a TBX21-dependent manner (By similarity). 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). Phosphorylates TBX21 on tyrosine residues leading to an enhancement of its transcriptional activator activity (By similarity).

Cellular Location
Cytoplasm, cytoskeleton. Nucleus. Mitochondrion. Note=Shuttles between the nucleus and cytoplasm depending on environmental signals. Sequestered into the cytoplasm through interaction with 14-3-3 proteins. Localizes to mitochondria in response to oxidative stress (By similarity).

Tissue Location
Widely expressed.

ABL1 Antibody (N-term K219) Blocking Peptide - Protocols

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

- Blocking Peptides

ABL1 Antibody (N-term K219) Blocking Peptide - Images

ABL1 Antibody (N-term K219) Blocking Peptide - Background

The ABL1 protooncogene encodes a cytoplasmic and nuclear protein tyrosine kinase that has been implicated in processes of cell differentiation, cell division, cell adhesion, and stress response. Activity of c-Abl protein is negatively regulated by its SH3 domain, and deletion of the SH3 domain turns ABL1 into an oncogene. The t(9;22) translocation results in the head-to-tailfusion of the BCR and ABL1 genes present in many cases of chronic myelogeneous leukemia. The DNA-binding activity of the ubiquitously expressed ABL1 tyrosine kinase is regulated by CDC2-mediated

phosphorylation, suggesting a cell cycle function for ABL1.

ABL1 Antibody (N-term K219) Blocking Peptide - References

Donaldson, L.W., et al., Proc. Natl. Acad. Sci. U.S.A. 99(22):14053-14058 (2002).Pisabarro, M.T., et al., J. Mol. Biol. 281(3):513-521 (1998).Nam, H.J., et al., Structure 4(9):1105-1114 (1996).Chissoe, S.L., et al., Genomics 27(1):67-82 (1995).Gosser, Y.Q., et al., Structure 3(10):1075-1086 (1995).