c-ABL Antibody – Polyclonal

PRODUCT OVERVIEW

Product Name	c-Abl, Polyclonal Antibody
Full Product Name	c-Abl Polyclonal Antibody
Product Gene Name	anti-c-Abl antibody
Clonality	Polyclonal
Molecular Weight	124,955 Da
OMIM	189980
3D Structure	ModBase 3D Structure for P00519
ISO Certification	Manufactured in an ISO 9001:2015 Certified Laboratory.
Related Product Information for anti-c-Abl antibody	MBS8521567 is an antibody targeting c-Abl. Also known as c-Abl Polyclonal Antibody.
Other Notes	Small volumes of anti-c-Abl antibody vial(s) may occasionally become entrapped in the seal of the product vial during shipment and storage. If necessary, briefly centrifuge the vial on a tabletop centrifuge to dislodge any liquid in the container`s cap. Certain products may require to ship with dry ice and additional dry ice fee may apply.

ADDITIONAL DETAILS

NCBI/Uniprot data below describe general gene information for c-Abl. It may not necessarily be applicable to this product.

NCBI GI #	62362414
NCBI GeneID	25
NCBI Accession #	NP_005148.2
UniProt Primary Accession #	P00519
UniProt Secondary Accession #	Q13869; Q13870; Q16133; Q17R61; Q45F09; A3KFJ3
UniProt Related Accession #	P00519
GenBank Nucleotide #	NM_005157.5
NCBI Official Full Name	tyrosine-protein kinase ABL1 isoform a
NCBI Official Synonym Full Names	ABL proto-oncogene 1, non-receptor tyrosine kinase
NCBI Official Symbol	ABL1
NCBI Official Synonym Symbols	ABL; JTK7; p150; c-ABL; v-abl; CHDSKM; c-ABL1; bcr/abl
UniProt Protein Name	Tyrosine-protein kinase ABL1
UniProt Synonym Protein Names	Abelson murine leukemia viral oncogene homolog 1; Abelson tyrosine-protein kinase 1; Proto-oncogene c-Abl; p150
Protein Family	Probable protein
UniProt Gene Name	ABL1
UniProt Synonym Gene Names	ABL; JTK7
NCBI Protein Information	tyrosine-protein kinase ABL1

NCBI Summary for c-Abl	This gene is a protooncogene that encodes a protein tyrosine kinase involved in a variety of cellular processes, including cell division, adhesion, differentiation, and response to stress. The activity of the protein is negatively regulated by its SH3 domain, whereby deletion of the region encoding this domain results in an oncogene. The ubiquitously expressed protein has DNA-binding activity that is regulated by CDC2-mediated phosphorylation, suggesting a cell cycle function. This gene has been found fused to a variety of translocation partner genes in various leukemias, most notably the t(9;22) translocation that results in a fusion with the 5' end of the breakpoint cluster region gene (BCR; MIM:151410). Alternative splicing of this gene results in two transcript variants, which contain alternative first exons that are spliced to the remaining common exons. [provided by RefSeq, Aug 2014]
UniProt Comments for c-Abl	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. 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. Phosphorylates TBX21 on tyrosine residues leading to an enhancement of its transcriptional activator activity ().
Research Articles on c-Abl	**1.** This is the first report evaluating the role of SOD2 in native and T351-mutated BCR-ABL-expressing cells and in a large cohort of chronic myeloid leukemia patients. In leukemic cells silenced for SOD2 expression a specific down-regulation of the expression of PRDX2 gene was found.
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