

Product name:	Abl1 (phospho Thr735) Rabbit Polyclonal Antibody
Cat number:	ABN04194
Conjugate:	Unconjugated
Size:	100µL
Clone:	Polyclonal
Concentration:	1mg/ml
Host:	Rabbit
Isotype:	IgG
Immunogen:	The antiserum was produced against synthesized peptide derived from human ABL1 around the phosphorylation site of Thr735. AA range:701-750
Reactivity:	Human,Mouse,Rat,Monkey
Applications:	WB 1:500-1:2000,IHC 1:100-1:300,ICC/IF 1:50-1:200,ELISA 1:5000-1:10000
Molecular Weight:	125(200kDa BCR-ABL complex)
Purification:	Affinity purification
Form:	Liquid
Buffer:	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% New type preservative N.
Storage:	Store at 4°C short term. Aliquot and store at -20°C for 12 months. Avoid freeze/thaw cycles.

Background:

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. [prcatalytic activity:ATP + a [protein]-L-tyrosine = ADP + a [protein]-L-tyrosine phosphate.,cofactor:Magnesium or manganese.,disease:A chromosomal aberration involving ABL1 is a cause of chronic myeloid leukemia (CML) [MIM:608232]. Translocation t(9;22)(q34;q11) with BCR. The translocation produces a BCR-ABL found also in acute myeloid leukemia (AML) and acute lymphoblastic leukemia (ALL).,enzyme regulation:Stabilized in the inactive form by an association between the SH3 domain and the SH2-TK linker region, interactions of the amino-terminal cap, and contributions from an amino-terminal myristoyl group and phospholipids. Activated by autophosphorylation as well as by SRC-family kinase-mediated phosphorylation. Activated by RIN1 binding to the SH2 and SH3 domains. Inhibited by imatinib mesylate (Gleevec) which is used for the treatment of chronic myeloid leukemia (CML).,function:Regulates cytoskeleton remodeling during cell differentiation, cell division and cell adhesion. Localizes to dynamic actin structures, and phosphorylates CRK and CRKL, DOK1, and other proteins controlling cytoskeleton dynamics. Regulates DNA repair potentially by activating the proapoptotic pathway when the DNA damage is too severe to be repaired.,online information:Abl entry,PTM:Phosphorylated by PRKDC (By similarity). DNA damage-induced activation of c-Abl requires the function of ATM and Ser-446 phosphorylation. Isoform IB is myristoylated on Gly-2. Phosphorylation on Thr-735 is required for binding 14-3-3 proteins for cytoplasmic translocation.,similarity:Belongs to the protein kinase superfamily. Tyr protein kinase family.,similarity:Belongs to the protein kinase superfamily. Tyr protein kinase family. ABL subfamily.,similarity:Contains 1 protein kinase domain.,similarity:Contains 1 SH2 domain.,similarity:Contains 1 SH3 domain.,subcellular location:The myristoylated c-ABL protein is reported to be nuclear. Sequestered into the cytoplasm through interaction with 14-3-3 proteins.,subunit:Interacts with SORBS1 following insulin stimulation. Found in a trimolecular complex containing CDK5 and CABLES1. Interacts with CABLES1 and PSTPIP1. Interacts with ZDHHC16 (By similarity). Interacts with INPPL1/SHIP2. Interacts with the 14-3-3 proteins, YWHAB, YWHAE, YWHAG, YWHAH, SFN AND YWHAZ; the interaction with 14-3-3 proteins requires phosphorylation on Thr-735 and, sequesters ABL1 into the cytoplasm.,tissue specificity:Widely expressed.,