SANTA CRUZ BIOTECHNOLOGY, INC.

c-Abl (24-11): sc-23

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

The Abl oncogene was initially identified as the viral transforming gene of Abelson murine leukemia virus (A-MuLV). The major translational product of c-Abl has been identified as a protein with tyrosine kinase activity and an SH2 domain. The Abl oncogene is implicated in several human leukemias including 90-95% of chronic myelocytic leukemia (CML), 20-25% of adult acute lymphoblastic leukemia (ALL) and 2-5% of pediatric ALL. In these leukemias the c-Abl protooncogene undergoes a (9;22) chromosomal translocation producing the Philadelphia (Ph1) chromosome. The molecular consequence of this translocation is the generation of a chimeric Bcr/c-Abl mRNA encoding activated Abl protein-tyrosine kinase. The Bcr gene has been shown to encode a GTPase-activating protein (GAP) specific for the Ras-related GTP-binding protein, p21rac.

CHROMOSOMAL LOCATION

Genetic locus: ABL1 (human) mapping to 9q34.12, BCR (human) mapping to 22q11.23; Abl1 (mouse) mapping to 2 B, Bcr (mouse) mapping to 10 B5.3.

SOURCE

c-Abl (24-11) is a mouse monoclonal antibody raised against a region within the c-Abl p120 C-terminus domain.

PRODUCT

Each vial contains 100 µg IgG_1 in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

APPLICATIONS

c-Abl (24-11) is recommended for detection of c-Abl p120 and chimeric Bcr/Abl proteins in CML and ALL of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for c-Abl siRNA (h): sc-29843, c-Abl siRNA (m): sc-29844, c-Abl siRNA (r): sc-270357, c-Abl shRNA Plasmid (h): sc-29843-SH, c-Abl shRNA Plasmid (m): sc-29844-SH, c-Abl shRNA Plasmid (r): sc-270357-SH, c-Abl shRNA (h) Lentiviral Particles: sc-29843-V, c-Abl shRNA (m) Lentiviral Particles: sc-29844-V and c-Abl shRNA (r) Lentiviral Particles: sc-270357-V.

Molecular Weight of c-Abl: 120 kDa.

Molecular Weight of Bcr/Abl fusion protein: 210 kDa.

Positive Controls: chemically-treated HCT-116 whole cell lysate, K-562 whole cell lysate: sc-2203 or RAW 264.7 whole cell lysate: sc-2211.

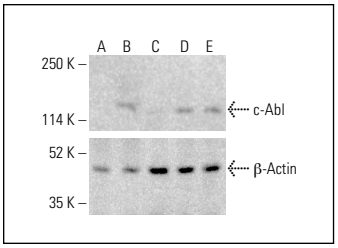
STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

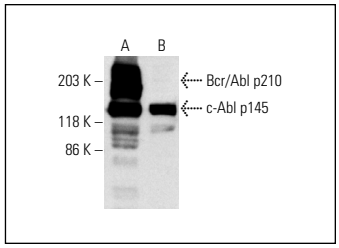
RESEARCH USE

For research use only, not for use in diagnostic procedures.

DATA



c-Abl (24-11): sc-23. Western blot analysis of c-Abl expression in untreated K-562 (**A**), chemically-treated K-562 (**B**), untreated HCT-116 (**C**) and chemically-treated HCT-116 (**D**, **E**) whole cell lysates. β-Actin (C4): sc-47778 used as loading control. Detection reagent used: m-IgG Fc BP-HRP: sc-525409.



c-Abl (24-11): sc-23. Western blot analysis of c-Abl expression in K-562 (**A**) and RAW 264.7 (**B**) whole cell lysates.

SELECT PRODUCT CITATIONS

1. Yamanashi, Y., et al. 1997. Identification of the Abl- and rasGAP-associated 62 kDa protein as a docking protein, Dok. Cell 88: 205-211.
2. García-Tuñón, I., et al. 2017. The CRISPR/Cas9 system efficiently reverts the tumorigenic ability of BCR/ABL *in vitro* and in a xenograft model of chronic myeloid leukemia. Oncotarget 8: 26027-26040.
3. Wang, Z., et al. 2018. Analysis of cellular tyrosine phosphorylation via chemical rescue of conditionally active Abl kinase. Biochemistry 57: 1390-1398.
4. Kashiwagi, S., et al. 2019. Localization of Bcr-Abl to stress granules contributes to its oncogenic function. Cell Struct. Funct. 44: 195-204.
5. Omsland, M., et al. 2020. Tyrosine kinase inhibitors and interferon-α increase tunneling nanotube (TNT) formation and cell adhesion in chronic myeloid leukemia (CML) cell lines. FASEB J. 34: 3773-3791.
6. Luo, M., et al. 2021. Chemoproteomics-enabled discovery of covalent RNF114-based degraders that mimic natural product function. Cell Chem. Biol. 28: 559-566.e15.
7. Chen, C.W., et al. 2022. Activin A downregulates the CD69-MT2A axis via p38MAPK to induce erythroid differentiation that sensitizes Bcr-Abl-positive cells to imatinib. Exp. Cell Res. 417: 113219.
8. Chandía-Cristi, A., et al. 2023. c-Abl tyrosine kinase is required for BDNF-induced dendritic branching and growth. Int. J. Mol. Sci. 24: 1944.
9. Chandía-Cristi, A., et al. 2024. Prophylactic treatment with the c-Abl inhibitor, neurotinib, diminishes neuronal damage and the convulsive state in pilocarpine-induced mice. Cell Rep. 43: 114144.

CONJUGATES

See **c-Abl (8E9): sc-56887** for c-Abl antibody conjugates, including AC, HRP, FITC, PE, and Alexa Fluor® 488, 546, 594, 647, 680 and 790.