SANTA CRUZ BIOTECHNOLOGY, INC.

Vav (H-211): sc-7206

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

The Vav gene was originally identified on the basis of its oncogenic activation during the course of gene transfer assays. The major translational product of the Vav proto-oncogene has been identified as a protein containing an array of structural motifs. Contained within its amino terminus are a helix-loop-helix domain and a leucine zipper motif similar to that of Myc family proteins; deletion of this region of p95Vav causes its oncogenic activation. In addition, p95Vav contains an SH2 domain, which could indicate its role as a substrate for tyrosine kinases. Expression of p95Vav is limited exclusively to cells of hematopoietic origin, including those of the erythroid, lymphoid and myeloid lineages. These results suggest that p95Vav may represent a new type of signal transduction molecule involved in the transduction of tyrosine phosphorylation signaling into transcriptional events.

CHROMOSOMAL LOCATION

Genetic locus: VAV1 (human) mapping to 19p13.3; Vav1 (mouse) mapping to 17 D.

SOURCE

Vav (H-211) is a rabbit polyclonal antibody raised against amino acids 110-320 of Vav p95 of human origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

APPLICATIONS

Vav (H-211) is recommended for detection of Vav p95 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), immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Vav (H-211) is also recommended for detection of Vav p95 in additional species, including canine, bovine and porcine.

Suitable for use as control antibody for Vav siRNA (h): sc-29517, Vav siRNA (m): sc-29518, Vav shRNA Plasmid (h): sc-29517-SH, Vav shRNA Plasmid (m): sc-29518-SH, Vav shRNA (h) Lentiviral Particles: sc-29517-V and Vav shRNA (m) Lentiviral Particles: sc-29518-V.

Molecular Weight of Vav: 95 kDa.

Positive Controls: Jurkat whole cell lysate: sc-2204, MOLT-4 cell lysate: sc-2233 or CTLL-2 cell lysate: sc-2242.

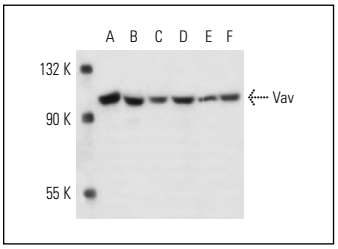
STORAGE

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

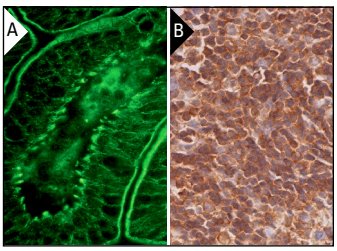
RESEARCH USE

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

DATA



Vav (H-211): sc-7206. Western blot analysis of Vav expression in Jurkat (**A**), MOLT-4 (**B**), CTLL-2 (**C**), HL-60 (**D**), GM-CSF-treated K-562 (**E**) and CCRF-CEM (**F**) whole cell lysates.



Vav (H-211): sc-7206. Immunofluorescence staining of normal mouse intestine frozen section showing membrane and cytoplasmic staining (**A**). Immunoperoxidase staining of formalin fixed, paraffin-embedded human lymph node tissue showing cytoplasmic staining of cells in germinal and non-germinal centers (**B**).

SELECT PRODUCT CITATIONS

1. Brown, A., et al. 1999. Activation of the PAK-related kinase by human immunodeficiency virus type 1 Nef in primary human peripheral blood lymphocytes and macrophages leads to phosphorylation of a PIX-p95 complex. J. Virol. 73: 9899-9907.
2. Salazar-Fontana, L.I., et al. 2003. CD28 engagement promotes actin polymerization through the activation of the small Rho GTPase Cdc42 in human T cells. J. Immunol. 171: 2225-2232.
3. Wang, Y., et al. 2004. Entire mitogen activated protein kinase (MAPK) pathway is present in preimplantation mouse embryos. Dev. Dyn. 231: 72-87.
4. Patrussi, L., et al. 2005. Cooperation and selectivity of the two Grb2 binding sites of p52Shc in T-cell antigen receptor signaling to Ras family GTPases and Myc-dependent survival. Oncogene 24: 2218-2228.
5. Costa, C., et al. 2007. Negative feedback regulation of Rac in leukocytes from mice expressing a constitutively active phosphatidylinositol 3-kinase γ. Proc. Natl. Acad. Sci. USA 104: 14354-14359.
6. Peterson, M.E. and Long, E.O. 2008. Inhibitory receptor signaling via tyrosine phosphorylation of the adaptor Crk. Immunity 29: 578-588.
7. Stevens, C.N., et al. 2010. T cell receptor early signalling complex activation in response to interferon-α receptor stimulation. Biochem. J. 428: 429-437.
8. Kim, H.S., et al. 2010. Synergistic signals for natural cytotoxicity are required to overcome inhibition by c-Cbl ubiquitin ligase. Immunity 32: 175-186.