SANTA CRUZ BIOTECHNOLOGY, INC.

Raf-B (H-145): sc-9002

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Several serine/threonine protein kinases have been implicated as intermediates in signal transduction pathways. These include ERK/MAP kinases, ribosomal S6 kinase (Rsk) and Raf-1. Raf-1 is a cytoplasmic protein with intrinsic serine/threonine activity. It is broadly expressed in nearly all cell lines tested to date and is the cellular homolog of v-Raf, the product of the transforming gene of the 3611 strain of murine sarcoma virus. The unregulated kinase activity of the v-Raf protein has been associated with transformation and mitogenesis, while the activity of Raf-1 is normally suppressed by a regulatory N-terminal domain. Raf-A, a second member of the Raf gene family of serine/threonine protein kinases, exhibits substantial homology to Raf-1 within the kinase domain of the two molecules, but less homology elsewhere. Expression of Raf-B is highly restricted, with highest levels in the cerebrum and testis.

CHROMOSOMAL LOCATION

Genetic locus: BRAF (human) mapping to 7q34; Braf (mouse) mapping to 6 B1.

SOURCE

Raf-B (H-145) is a rabbit polyclonal antibody raised against amino acids 12-156 mapping at the N-terminus of Raf-B 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

Raf-B (H-145) is recommended for detection of Raf-B 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).

Raf-B (H-145) is also recommended for detection of Raf-B in additional species, including bovine.

Suitable for use as control antibody for Raf-B siRNA (h): sc-36368, Raf-B siRNA (m): sc-63294, Raf-B siRNA (r): sc-61894, Raf-B shRNA Plasmid (h): sc-36368-SH, Raf-B shRNA Plasmid (m): sc-63294-SH, Raf-B shRNA Plasmid (r): sc-61894-SH, Raf-B shRNA (h) Lentiviral Particles: sc-36368-V, Raf-B shRNA (m) Lentiviral Particles: sc-63294-V and Raf-B shRNA (r) Lentiviral Particles: sc-61894-V.

Molecular Weight of Raf-B isoforms: 95/62 kDa.

Positive Controls: A-431 whole cell lysate: sc-2201, NIH/3T3 whole cell lysate: sc-2210 or HL-60 whole cell lysate: sc-2209.

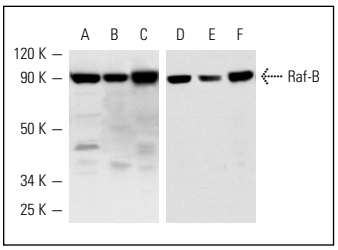
STORAGE

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

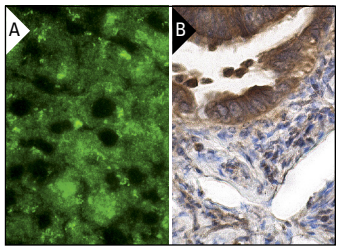
RESEARCH USE

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

DATA



Western blot analysis of Raf B expression in A-431 (**A**,**D**), HL-60 (**B**,**E**) and NIH/3T3 (**C**,**F**) whole cell lysates. Antibodies tested include Raf-B (H-145): sc-9002 (**A**-**C**) and Raf-B (F-7): sc-5284 (**D**-**F**).



Raf-B (H-145): sc-9002. Immunofluorescence staining of normal mouse liver frozen section showing cytoplasmic staining (**A**). Immunoperoxidase staining of formalin fixed, paraffin-embedded human fallopian tube tissue showing cytoplasmic and weak nuclear staining of glandular cells (**B**).

SELECT PRODUCT CITATIONS

1. Brummer, T., et al. 2002. Inducible gene deletion reveals different roles for B-Raf and Raf-1 in B cell antigen receptor signalling. EMBO J. 21: 5611-5622.
2. Pardo, O.E., et al. 2006. FGF-2 protects small cell lung cancer cells from apoptosis through a complex involving PKCε, B-Raf and S6K2. EMBO J. 25: 3078-3088.
3. Baitei, E.Y., et al. 2009. Aberrant BRAF splicing as an alternative mechanism for oncogenic B-Raf activation in thyroid carcinoma. J. Pathol. 217: 707-715.
4. Gangopadhyay, S.S., et al. 2009. Smooth muscle archvillin is an ERK scaffolding protein. J. Biol. Chem. 284: 17607-17615.
5. Baitei, E.Y., et al. 2009. Aberrant BRAF splicing as an alternative mechanism for oncogenic B-Raf activation in thyroid carcinoma. J. Pathol. 217: 707-715.
6. Galabova-Kovacs, G. and Baccarini, M. 2010. Deciphering signaling pathways *in vivo:* the Ras/Raf/MEK/ERK cascade. Methods Mol. Biol. 661: 421-431.
7. Ritt, D.A., et al. 2010. Impact of feedback phosphorylation and Raf heterodimerization on normal and mutant B-Raf signaling. Mol. Cell. Biol. 30: 806-819.
8. Albers, C., et al. 2011. An RNAi-based system for loss-of-function analysis identifies Raf1 as a crucial mediator of Bcr-Abl-driven leukemogenesis. Blood 118: 2200-2210.