

K-Ras-2B (C-19): sc-521

BACKGROUND

The mammalian Ras (also designated v-Ha-Ras, Harvey rat sarcoma viral oncogene homolog, HRAS1, K-Ras, N-Ras, RASH1 or c-bas/has) gene family consists of the Harvey and Kirsten Ras genes (c-H-Ras1 and c-K-Ras2), an inactive pseudogene of each (c-H-Ras2 and c-K-Ras1) and the N-Ras gene. The three Ras oncogenes, H-Ras, K-Ras and N-Ras, encode proteins with GTP/GDP binding and GTPase activity. Ras proteins alternate between an inactive form bound to GDP and an active form bound to GTP, activated by a guanine nucleotide-exchange factor (GEF) and inactivated by a GTPase-activating protein (GAP). Ras nomenclature originates from the characterization of human DNA sequences homologous to cloned DNA fragments containing oncogenic sequences of a type C mammalian retrovirus, the Harvey strain of murine sarcoma virus (HaMSV), derived from the rat. Under normal conditions, Ras family members influence cell growth and differentiation events in a subcellular membrane compartmentalization-based signaling system. Oncogenic Ras can deregulate processes that control both cell proliferation and apoptosis. The Ras superfamily of GTP hydrolysis-coupled signal transduction relay proteins can be subclassified into Ras, Rho, Rab and ARF families.

CHROMOSOMAL LOCATION

Genetic locus: KRAS (human) mapping to 12p12.1; Kras (mouse) mapping to 6 G3.

SOURCE

K-Ras-2B (C-19) is an affinity purified rabbit polyclonal antibody raised against a peptide mapping at the C-terminus of K-Ras-2B of human origin.

PRODUCT

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

Blocking peptide available for competition studies, sc-521 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

K-Ras-2B (C-19) is recommended for detection of K-Ras-2B p21 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).

K-Ras-2B (C-19) is also recommended for detection of K-Ras-2B p21 in additional species, including equine, canine, bovine and porcine.

Suitable for use as control antibody for K-Ras siRNA (h): sc-35731, K-Ras siRNA (m): sc-43876, K-Ras shRNA Plasmid (h): sc-35731-SH, K-Ras shRNA Plasmid (m): sc-43876-SH, K-Ras shRNA (h) Lentiviral Particles: sc-35731-V and K-Ras shRNA (m) Lentiviral Particles: sc-43876-V.

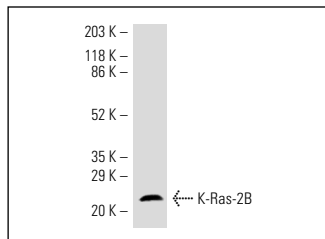
Molecular Weight of K-Ras-2B: 21 kDa.

Positive Controls: KNRK whole cell lysate: sc-2214 or HeLa whole cell lysate: sc-2200.

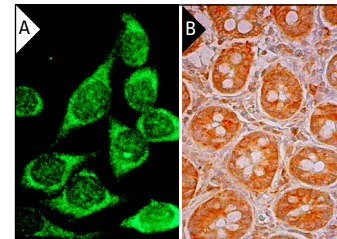
STORAGE

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

DATA



K-Ras-2B (C-19): sc-521. Western blot analysis of K-Ras-2B expression in KNRK whole cell lysate.



K-Ras-2B (C-19): sc-521. Immunofluorescence staining of methanol-fixed HeLa cells showing cytoplasmic staining (A). Immunoperoxidase staining of formalin fixed, paraffin-embedded human colon tissue showing cytoplasmic staining of glandular cells and endothelial cells (B).

SELECT PRODUCT CITATIONS

1. Fan, J., et al. 1997. K-Ras modulates the cell cycle via both positive and negative regulatory pathways. *Oncogene* 14: 2595-2607.
2. Pells, S., et al. 1997. Developmentally regulated expression of murine K-Ras isoforms. *Oncogene* 15: 1781-1786.
3. Fuentes-Calvo, I., et al. 2010. Analysis of K-Ras nuclear expression in fibroblasts and mesangial cells. *PLoS ONE* 5: e8703.
4. Cho, K.J., et al. 2011. Therapeutic levels of the hydroxymethylglutaryl-coenzyme A reductase inhibitor lovastatin activate Ras signaling via phospholipase D2. *Mol. Cell. Biol.* 31: 1110-1120.
5. Wang, Q., et al. 2011. Focal adhesions and Ras are functionally and spatially integrated to mediate IL-1 activation of ERK. *FASEB J.* 25: 3448-3464.
6. Yang, G., et al. 2013. RAS promotes tumorigenesis through genomic instability induced by imbalanced expression of Aurora-A and BRCA2 in midbody during cytokinesis. *Int. J. Cancer* 133: 275-285.
7. Agudo-Ibanez, L., et al. 2015. H-Ras distribution and signaling in plasma membrane microdomains are regulated by acylation and deacylation events. *Mol. Cell. Biol.* 35: 1898-1914.

RESEARCH USE

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

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