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Produktinformation

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Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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SANTA CRUZ BIOTECHNOLOGY, INC.

N-Ras (F155): sc-31

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

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: NRAS (human) mapping to 1p13.2; Nras (mouse) mapping to 3 F2.2.

SOURCE

N-Ras (F155) is a mouse monoclonal antibody raised against recombinant N-Ras p21 of human origin.

PRODUCT

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

APPLICATIONS

N-Ras (F155) is recommended for detection of N-Ras 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) and immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500).

Suitable for use as control antibody for N-Ras siRNA (h): sc-36004, N-Ras siRNA (m): sc-36005, N-Ras shRNA Plasmid (h): sc-36004-SH, N-Ras shRNA Plasmid (m): sc-36005-SH, N-Ras shRNA (h) Lentiviral Particles: sc-36004-V and N-Ras shRNA (m) Lentiviral Particles: sc-36005-V.

Molecular Weight of N-Ras: 21 kDa.

Positive Controls: N-Ras (m): 293T Lysate: sc-121908, A-431 whole cell lysate: sc-2201 or Jurkat whole cell lysate: sc-2204.

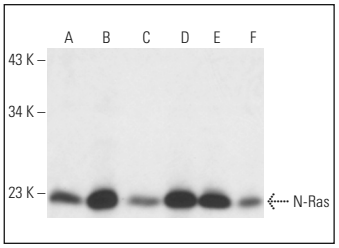
STORAGE

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

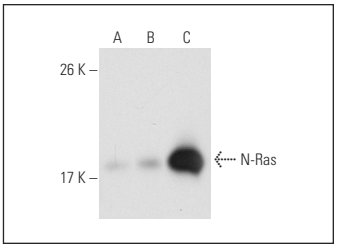
RESEARCH USE

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

DATA



N-Ras (F155): sc-31. Western blot analysis of N-Ras expression in HeLa (**A**), A-431 (**B**), Jurkat (**C**), NIH/3T3 (**D**), NRK (**E**) and F9 (**F**) whole cell lysates.



N-Ras (F155): sc-31. Western blot analysis of N-Ras expression in non-transfected 293T: sc-117752 (**A**), mouse N-Ras transfected 293T: sc-121908 (**B**) and HeLa (**C**) whole cell lysates.

SELECT PRODUCT CITATIONS

1. Clark, W., et al. 2000. v-Jun overrides the mitogen dependence of S-phase entry by deregulating retinoblastoma protein phosphorylation and E2F-pocket protein interactions as a consequence of enhanced cyclin E-Cdk2 catalytic activity. Mol. Cell. Biol. 20: 2529-2542.
2. Gupta, S., et al. 2000. Dissection of Ras-dependent signaling pathways controlling aggressive tumor growth of human fibrosarcoma cells: evidence for a potential novel pathway. Mol. Cell. Biol. 20: 9294-9306.
3. Wu, W., et al. 2003. VEGF receptor expression and signaling in human bladder tumors. Oncogene 22: 3361-3370.
4. Oliveira, J.B., et al. 2007. NRAS mutation causes a human autoimmune lymphoproliferative syndrome. Proc. Natl. Acad. Sci. USA 104: 8953-8958.
5. Steger, D.J. 2008. DOT1L/KMT4 recruitment and H3K79 methylation are ubiquitously coupled with gene transcription in mammalian cells. Mol. Cell. Biol. 28: 2825-2839.
6. Rubio, I., et al. 2010. TCR-induced activation of Ras proceeds at the plasma membrane and requires palmitoylation of N-Ras. J. Immunol. 185: 3536-3543.
7. Omerovic, J., et al. 2010. Phosphatome profiling reveals PTPN2, PTPRJ and PTEN as potent negative regulators of PKB/Akt activation in Ras-mutated cancer cells. Biochem. J. 426: 65-72.
8. Cho, K.J., et al. 2011. Therapeutic levels of the hydroxmethylglutaryl-coenzyme A reductase inhibitor lovastatin activate ras signaling via phospholipase D2. Mol. Cell. Biol. 31: 1110-1120.
9. Kaplan, F.M., et al. 2012. SHOC2 and CRAF mediate ERK1/2 reactivation in mutant NRAS-mediated resistance to RAF Inhibitor. J. Biol. Chem. 287: 41797-41807.

CONJUGATES

See **pan Ras (C-4): sc-166691** for pan Ras antibody conjugates, including AC, HRP, FITC, PE, Alexa Fluor® 488 and Alexa Fluor® 647.