

H-Ras (259): sc-35

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.

SOURCE

H-Ras (259) is a rat monoclonal antibody raised against full length Ras p21 of human origin.

PRODUCT

Each vial contains 200 µg IgG₁ in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin. Also available azide-free for blocking H-Ras binding of Raf, sc-35 L, 200 µg/0.1 ml.

H-Ras (259) is available conjugated to agarose (sc-35 AC), 500 µg/0.25 ml agarose in 1 ml, for IP; to HRP (sc-35 HRP), 200 µg/ml, for WB, IHC(P) and ELISA; to either phycoerythrin (sc-35 PE), fluorescein (sc-35 FITC), Alexa Fluor® 488 (sc-35 AF488), Alexa Fluor® 546 (sc-35 AF546), Alexa Fluor® 594 (sc-35 AF594) or Alexa Fluor® 647 (sc-35 AF647), 200 µg/ml, for WB (RGB), IF, IHC(P) and FCM; and to either Alexa Fluor® 680 (sc-35 AF680) or Alexa Fluor® 790 (sc-35 AF790), 200 µg/ml, for Near-Infrared (NIR) WB, IF and FCM.

In addition, H-Ras (259) is available conjugated to Alexa Fluor® 405 (sc-35 AF405), 100 µg/2 ml, for IF, IHC(P) and FCM.

Blocking peptide available for competition studies, sc-35 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% stabilizer protein).

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APPLICATIONS

H-Ras (259) is recommended for detection of antigenic determinants common to H-, K- and 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), flow cytometry (1 µg per 1 x 10⁶ cells) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

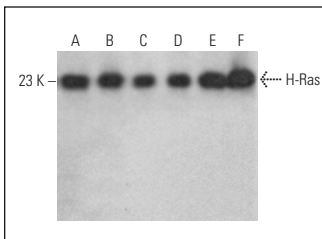
Molecular Weight of H-Ras: 21 kDa.

Positive Controls: MCF7 whole cell lysate: sc-2206.

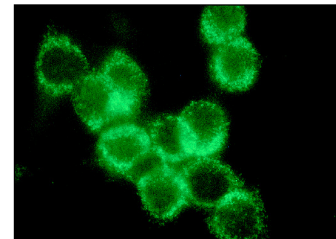
STORAGE

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

DATA



H-Ras (259): sc-35. Western blot analysis of H-Ras expression in K-562 (A), HeLa (B), Jurkat (C), NIH/3T3 (D), A-431 (E) and MCF7 (F) whole cell lysates.



H-Ras (259) AF488: sc-35 AF488. Immunofluorescence staining of methanol-fixed HeLa cells showing cytoplasmic localization.

SELECT PRODUCT CITATIONS

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3. Suen, K.L., et al. 1995. Lack of evidence for the activation of the Ras/Raf mitogenic pathway by 14-3-3 proteins in mammalian cells. *Oncogene* 11: 825-831.
4. Maher, J., et al. 1995. Evidence for cell-specific differences in transformation by N-, H- and K-ras. *Oncogene* 11: 1639-1647.
5. Jung, J.U. and Desrosiers, R.C. 1995. Association of the viral oncoprotein STP-C488 with cellular ras. *Mol. Cell. Biol.* 15: 6506.
6. Lebowitz, P., et al. 1995. Evidence that farnesyltransferase inhibitors suppress Ras transformation by interfering with Rho activity. *Mol. Cell. Biol.* 15: 6613-6622.
7. Albanese, C., et al. 1995. Transforming p21^{ras} mutants and c-Ets-2 activate the cyclin D1 promoter through distinguishable regions. *J. Biol. Chem.* 270: 23589-23597.
8. Vega, S.L., et al. 2015. Organizational metrics of interchromatin speckle factor domains: integrative classifier for stem cell adhesion & lineage signaling. *Integr. Biol.* 7: 435-446.
9. Saha, S.K., et al. 2016. KRT19 directly interacts with β-catenin/RAC1 complex to regulate NUMB-dependent NOTCH signaling pathway and breast cancer properties. *Oncogene* 36: 332-349.
10. García-Pérez, D., et al. 2017. Acute morphine, chronic morphine, and morphine withdrawal differently affect pleiotrophin, midkine, and receptor protein tyrosine phosphatase β₂ regulation in the ventral tegmental area. *Mol. Neurobiol.* 54: 495-510.

RESEARCH USE

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