

# Ras GAP (B4F8): sc-63

## BACKGROUND

The mammalian c-H-, c-K- and N-Ras proto-oncogenes encode ubiquitously expressed proteins. Ras GAP, also designated, Ras p21 protein activator (GTPase activating protein) 1; Ras p21 protein activator 1; and triphosphatase-activating protein, can exist in either a physiologically quiescent GDP-binding state or a GTP-binding signal-emitting state. Oncogenic Ras GAP proteins are trapped in the excited signal-emitting state because the mechanism normally employed to delimit their excitation period, hydrolysis of their bound GTP to GDP, is impaired as a result of specific mutations. Interaction of Ras GAP with GTPase activating protein (GAP) can increase hydrolysis of Ras GAP-bound GTP by as much as 1,000-fold. The product of the neurofibromatosis type 1 gene (NF1) has also been shown to exhibit Ras GAP activity, and proteins that stimulate the GTPase activity of three other low molecular weight GTPases, including Rho, Rab 3A and Rap 1, have also been described.

## CHROMOSOMAL LOCATION

Genetic locus: RASA1 (human) mapping to 5q14.3; (mouse) mapping to 13 C3.

## SOURCE

Ras GAP (B4F8) is a mouse monoclonal antibody raised against full length human recombinant Ras GAP expressed in Sf9 insect cells.

## PRODUCT

Each vial contains 200 µg IgG<sub>2a</sub> kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Ras GAP (B4F8) is available conjugated to agarose (sc-63 AC), 500 µg/0.25 ml agarose in 1 ml, for IP; to HRP (sc-63 HRP), 200 µg/ml, for WB, IHC(P) and ELISA; to either phycoerythrin (sc-63 PE), fluorescein (sc-63 FITC), Alexa Fluor® 488 (sc-63 AF488), Alexa Fluor® 546 (sc-63 AF546), Alexa Fluor® 594 (sc-63 AF594) or Alexa Fluor® 647 (sc-63 AF647), 200 µg/ml, for WB (RGB), IF, IHC(P) and FCM; and to either Alexa Fluor® 680 (sc-63 AF680) or Alexa Fluor® 790 (sc-63 AF790), 200 µg/ml, for Near-Infrared (NIR) WB, IF and FCM.

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## APPLICATIONS

Ras GAP (B4F8) is recommended for detection of Ras GAP p120 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 10<sup>6</sup> cells) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for Ras GAP siRNA (h): sc-29467, Ras GAP siRNA (m): sc-36394, Ras GAP shRNA Plasmid (h): sc-29467-SH, Ras GAP shRNA Plasmid (m): sc-36394-SH, Ras GAP shRNA (h) Lentiviral Particles: sc-29467-V and Ras GAP shRNA (m) Lentiviral Particles: sc-36394-V.

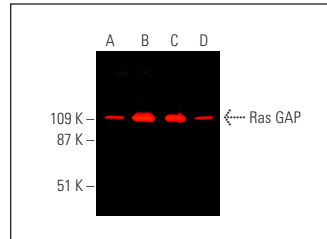
Molecular Weight of Ras GAP: 120 kDa.

Positive Controls: NIH/3T3 whole cell lysate: sc-2210, A-431 whole cell lysate: sc-2201 or KNRK whole cell lysate: sc-2214.

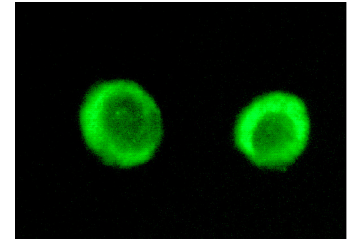
## STORAGE

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

## DATA



Ras GAP (B4F8): sc-63. Near-infrared western blot analysis of Ras GAP expression in NIH/3T3 (A), KNRK (B), 3611-RF (C) and A-431 (D) whole cell lysates. Blocked with UltraCruz® Blocking Reagent: sc-516214. Detection reagent used: m-IgGκ: BP-CFL 790: sc-516181.



Ras GAP (B4F8): sc-63. Immunofluorescence staining of methanol-fixed NIH/3T3 cells showing cytoplasmic staining.

## SELECT PRODUCT CITATIONS

- Zhang, B. and Roth, R.A. 1992. The Insulin receptor-related receptor. Tissue expression, ligand binding specificity, and signaling capabilities. *J. Biol. Chem.* 267: 18320-18328.
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- Sharma, S.B., et al. 2014. MicroRNAs 206 and 21 cooperate to promote Ras-extracellular signal-regulated kinase signaling by suppressing the translation of RASA1 and SPRED1. *Mol. Cell. Biol.* 34: 4143-4164.
- Chiang, H.M., et al. 2014. Hydroalcoholic extract of *Rhodiola rosea L.* (*Crassulaceae*) and its hydrolysate inhibit melanogenesis in B16F0 cells by regulating the CREB/MITF/tyrosinase pathway. *Food Chem. Toxicol.* 65: 129-139.
- Barcelo, C., et al. 2014. Phosphorylation at Ser-181 of oncogenic KRAS is required for tumor growth. *Cancer Res.* 74: 1190-1199.
- Dominguez, D., et al. 2015. Centrosome aberrations in human mammary epithelial cells driven by cooperative interactions between p16<sup>INK4a</sup> deficiency and telomere-dependent genotoxic stress. *Oncotarget* 6: 28238-28356.
- Yamani, L., et al. 2015. Nck1 deficiency improves pancreatic β cell survival to diabetes-relevant stresses by modulating PERK activation and signaling. *Cell. Signal.* 27: 2555-2567.
- Ercilla, A., et al. 2016. New origin firing is inhibited by APC/CCdh1 activation in S-phase after severe replication stress. *Nucleic Acids Res.* 44: 4745-4762.

## RESEARCH USE

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