SANTA CRUZ BIOTECHNOLOGY, INC.

C3G (C-19): sc-869

BACKGROUND

Ras p21 is the prototype of a superfamily of GTPases that is involved in the regulation of a wide variety of cellular processes. Ras signals in its GTP-bound form but is "turned off" when bound to GDP. When unregulated or constitutively turned on by mutations, Ras signaling contributes to malignant transformation. The switch between active and inactive Ras is controlled by GTPase-activating proteins (GAPs) and guanine nucleotide exchange factors (GEFs). C3G was isolated in a screen for proteins that could bind the SH3 domain of the Crk proto-oncogene product. The carboxy-terminus of the C3G protein displays significant sequence similarity to Ras-GRF/Cdc25Mm and mSos and can substitute for Cdc25 function in *S. cerevisiae*. These observations strongly suggest that C3G is a GEF for Ras and is involved in the regulation of Ras signaling through Crk. The C3G gene maps to human chromosome 9q34.13 in proximity to the gene that encodes c-Abl, a proto-oncogene that regulates Crk.

CHROMOSOMAL LOCATION

Genetic locus: RAPGEF1 (human) mapping to 9q34.13; Rapgef1 (mouse) mapping to 2 B.

SOURCE

C3G (C-19) is an affinity purified rabbit polyclonal antibody raised against a peptide mapping at the C-terminus of C3G 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.

Blocking peptide available for competition studies, sc-869 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

C3G (C-19) is recommended for detection of C3G of mouse, rat and human origin by Western Blotting (starting dilution 1:100, dilution range 1:50-1:500), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:25, dilution range 1:25-1:250) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

C3G (C-19) is also recommended for detection of C3G in additional species, including equine, canine, bovine, porcine and avian.

Suitable for use as control antibody for C3G siRNA (h): sc-29863, C3G siRNA (m): sc-29864, C3G shRNA Plasmid (h): sc-29863-SH, C3G shRNA Plasmid (m): sc-29864-SH, C3G shRNA (h) Lentiviral Particles: sc-29863-V and C3G shRNA (m) Lentiviral Particles: sc-29864-V.

Molecular Weight of C3G: 121 kDa.

Positive Controls: HeLa whole cell lysate: sc-2200, K-562 whole cell lysate: sc-2203 or KNRK whole cell lysate: sc-2214.

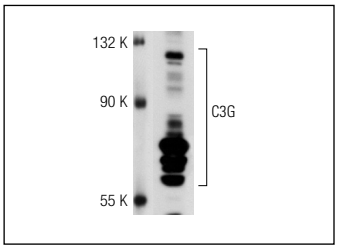
RESEARCH USE

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

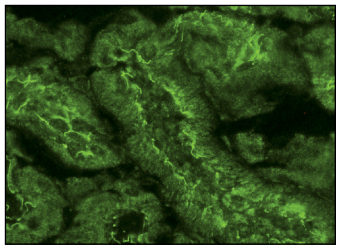
STORAGE

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

DATA



C3G (C-19): sc-869. Western blot analysis of C3G expression in K-562 whole cell lysate.



C3G (C-19): sc-869. Immunofluorescence staining of normal mouse kidney frozen section showing cytoplasmic staining.

SELECT PRODUCT CITATIONS

1. van den Berghe, N., et al. 1997. Biochemical characterization of C3G: an exchange factor that discriminates between Rap1 and Rap2 and is not inhibited by Rap1A(S17N). Oncogene 15: 845-850.
2. Ahmad, S., et al. 1997. The type I interferon receptor mediates tyrosine phosphorylation of the CrkL adaptor protein. J. Biol. Chem. 272: 29991-29994.
3. Boussiotis, V.A., et al. 1997. Maintenance of human T cell anergy: blocking of IL-2 gene transcription by activated Rap 1. Science 278: 124-128.
4. Park, T.J. and Curran, T. 2008. Crk and Crk-like play essential overlapping roles downstream of disabled-1 in the Reelin pathway. J. Neurosci. 28: 13551-13562.
5. Feng, L. and Cooper, J.A. 2009. Dual functions of Dab1 during brain development. Mol. Cell. Biol. 29: 324-332.
6. Schönherr, C., et al. 2010. Anaplastic lymphoma kinase activates the small GTPase Rap1 via the Rap1-specific GEF C3G in both neuroblastoma and PC12 cells. Oncogene 29: 2817-2830.
7. He, Y., et al. 2011. The non-receptor tyrosine kinase Lyn controls neutrophil adhesion by recruiting the CrkL-C3G complex and activating Rap1 at the leading edge. J. Cell Sci. 124: 2153-2164.
8. Ferrando, I.M., et al. 2012. Identification of targets of c-Src tyrosine kinase by chemical complementation and phosphoproteomics. Mol. Cell. Proteomics 11: 355-369.