Store at -200

## p190-A RhoGAP (C59F7) Rabbit



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Source/Isotype: Applications: Reactivity: Sensitivity: MW (kDa): **UniProt ID:** Entrez-Gene Id: WB, IP HMR Hm Mk Endogenous 190 Rabbit IgG #Q9NRY4 2909 **Product Usage Application** Dilution Information Western Blotting 1:1000 Immunoprecipitation 1:200 Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than **Storage** 0.02% sodium azide. Store at  $-20^{\circ}$ C. Do not aliquot the antibody.

Specificity / Sensitivity Source / Purification

p190-A RhoGAP (C59F7) Rabbit mAb detects endogenous levels of total p190-A RhoGAP protein.

Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues near the carboxy terminus of human p190-A RhoGAP.

**Background** 

Rho family GTPases are key regulators of diverse processes such as cytoskeletal organization, cell growth and differentiation, transcriptional regulation, and cell adhesion/motility. The activities of these proteins are controlled primarily through guanine nucleotide exchange factors (GEFs) that facilitate the exchange of GDP for GTP, promoting the active (GTP-bound) state, and GTPase activating proteins (GAPs) that promote GTP hydrolysis and the inactive (GDP-bound) state (1,2).

The p190 RhoGAP proteins are widely expressed Rho family GAPs. p190-A has been characterized as a tumor suppressor, and research studies have shown that loss or rearrangement of the chromosomal region containing the gene for p190-A is linked to tumor development (3,4). p190-A binds the mitogen-inducible transcription factor TFII-I, sequestering it in the cytoplasm and inhibiting its activity. Phosphorylation of p190-A at Tyr308 reduces its affinity for TFII-I, relieving the inhibition (5). p190-A can also inhibit growth factor-induced gliomas in mice (6) and affect cleavage furrow formation and cytokinesis in cultured cells (7).

Mice lacking p190-B RhoGAP show excessive Rho activation and a reduction in activation of the transcription factor CREB (8). Cells deficient in p190-B display defective adipogenesis (9). There is increasing evidence that p190 undergoes tyrosine phosphorylation, which activates its GAP domain (9-11). Levels of tyrosine phosphorylation are enhanced by Src overexpression (10,11). IGF-I treatment downregulates Rho through phosphorylation and activation of p190-B RhoGAP, thereby enhancing IGF signaling implicated in adipogenesis (9).

## **Background References**

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- 7. Su, L. et al. (2003) J Cell Biol 163, 571-82.
- 8. Sordella, R. et al. (2002) Dev Cell 2, 553-65.
- 9. Sordella, R. et al. (2003) Cell 113, 147-58.
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## **Species Reactivity**

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

## Western Blot Buffer

IMPORTANT: For western blots, incubate membrane with diluted primary antibody in 5% w/v BSA, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

1/1/24, 7:12 AM

p190-A RhoGAP (C59F7) Rabbit mAb (#2860) Datasheet Without Images Cell Signaling Technology

**Applications Key** 

**Cross-Reactivity Key** 

WB: Western Blotting IP: Immunoprecipitation

information.

H: human M: mouse R: rat Hm: hamster Mk: monkey Vir: virus Mi: mink C: chicken Dm: D. melanogaster X: Xenopus Z: zebrafish B: bovine Dg: dog Pg: pig Sc: S. cerevisiae Ce: C. elegans Hr: horse

GP: Guinea Pig Rab: rabbit All: all species expected

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