Revision 1

Store at -20C

#2860

p190-A RhoGAP (C59F7) Rabbit mAb

Cell Signaling TECHNOLOGY®

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For Research Use Only. Not for Use in Diagnostic Procedures.

Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W, IP	H M R Hm Mk	Endogenous	190	Rabbit IgG	#Q9NRY4	2909

Product Usage Information

Application	Dilution
Western Blotting	1:1000
Immunoprecipitation	1:200

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at –20°C. Do not aliquot the antibody.

Specificity/Sensitivity

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

Source / Purification

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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4. Tikoo, A. et al. (2000) *Gene* 257, 23-31.
5. Jiang, W. et al. (2005) *Mol Cell* 17, 23-35.
6. Wolf, R.M. et al. (2003) *Genes Dev* 17, 476-87.
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.
10. Chang, J.H. et al. (1995) *J Cell Biol* 130, 355-68.
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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.

Applications Key

W: Western Blotting **IP:** Immunoprecipitation

Cross-Reactivity Key

H: Human **M:** Mouse **R:** Rat **Hm:** Hamster **Mk:** Monkey

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Limited Uses