SANTA CRUZ BIOTECHNOLOGY, INC.

RECK (H-300): sc-28918

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

RECK (reversion-inducing-cysteine-rich protein with Kazal motifs) is a membrane anchored glycoprotein that binds to and inhibits the proteolytic activity of matrix metalloproteinase-9 (MMP-9). The enzymatic activity of MMP-9 facilitates tumor invasion by proteolytically digesting the extracellular matrix, thereby enabling tumor growth, expansion and metastasis. RECK inhibits the secretion and activation of MMP-9 into the extracellular matrix, which results in the inhibition of tumor growth. RECK contains multiple EGF-like repeats and serine-protease inhibitor-like domains. The expression of RECK is suppressed in several tumors and oncogenically transformed cells, suggesting that the loss of RECK activity correlates with transformed phenotypes. Transcriptional activation of RECK is potentially negatively regulated by the Sp1 family of transcription factors, as it contains two Sp1 binding motifs in the promoter region, and, in cells transformed with the ras oncogene, the Sp1 promoter region is essential for repressing RECK gene expression.

CHROMOSOMAL LOCATION

Genetic locus: RECK (human) mapping to 9p13.3; Reck (mouse) mapping to 4 B1.

SOURCE

RECK (H-300) is a rabbit polyclonal antibody raised against amino acids 644-943 mapping near the C-terminus of RECK 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.

APPLICATIONS

RECK (H-300) is recommended for detection of RECK 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) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

RECK (H-300) is also recommended for detection of RECK in additional species, including equine, canine and porcine.

Suitable for use as control antibody for RECK siRNA (h): sc-39718, RECK siRNA (m): sc-39719, RECK shRNA Plasmid (h): sc-39718-SH, RECK shRNA Plasmid (m): sc-39719-SH, RECK shRNA (h) Lentiviral Particles: sc-39718-V and RECK shRNA (m) Lentiviral Particles: sc-39719-V.

Molecular Weight of RECK: 110 kDa.

Positive Controls: AT-3 whole cell lysate or WI-38 whole cell lysate: sc-364260.

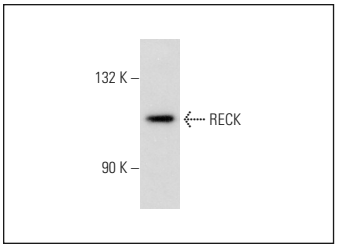
STORAGE

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

RESEARCH USE

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

DATA



RECK (H-300): sc-28918. Western blot analysis of RECK expression in AT-3 whole cell lysate.

SELECT PRODUCT CITATIONS

1. Visigalli, D., et al. 2009. The carboxyl terminal trimer of procollagen I induces pro-metastatic changes and vascularization in breast cancer cells xenografts. BMC Cancer 9: 59.
2. Vinothini, G., et al. 2009. Evaluation of molecular markers in a rat model of mammary carcinogenesis. Oncol. Res. 17: 483-493.
3. Priyadarsini, R.V., et al. 2009. The neem limonoids azadirachtin and nimbolide inhibit hamster cheek pouch carcinogenesis by modulating xenobiotic-metabolizing enzymes, DNA damage, antioxidants, invasion and angiogenesis. Free Radic. Res. 43: 492-504.
4. Nagini, S., et al. 2009. Of humans and hamsters: a comparative evaluation of carcinogen activation, DNA damage, cell proliferation, apoptosis, invasion, and angiogenesis in oral cancer patients and hamster buccal pouch carcinomas. Oral Oncol. 45: e31-e37.
5. Vidya Priyadarsini, R., et al. 2011. The flavonoid quercetin modulates the hallmark capabilities of the hamster bucal pouch carcinogenesis. Nutr. Cancer 63: 218-226.
6. Vinothini, G., et al. 2011. Correlation of matrix metalloproteinases and their inhibitors with hypoxia and angiogenesis in premenopausal patients with adenocarcinoma of the breast. Clin. Biochem. 44: 969-974.
7. Manikandan, P., et al. 2011. Eugenol inhibits cell proliferation via NFκB suppression in a rat model of gastric carcinogenesis induced by MNNG. Invest. New Drugs 29: 110-117.
8. Thiyagarajan, P., et al. 2014. Dietary chlorophyllin abrogates TGFβ signaling to modulate the hallmark capabilities of cancer in an animal model of forestomach carcinogenesis. Tumour Biol. 35: 6725-6737.