

p-Ret (Tyr 1062)-R: sc-20252-R

BACKGROUND

The Ret proto-oncogene is structurally related to the growing family of tyrosine kinase transmembrane receptors and is involved in GDNF signaling. By alternative splicing, two isoforms of the Ret proto-oncogene product are generated that differ from each other by having either 9 or 51 carboxy-terminal amino acids. The Ret gene products include two glycosylated proteins and, in tunicamycin-treated cells, a non-glycosylated protein consistent with the predicted Ret molecular weight based on sequence analysis. Tumor-specific rearrangements of the Ret proto-oncogene have been identified in papillary thyroid carcinomas leading to the formation of different transforming fusion proteins sharing the tyrosine kinase domain of Ret. In contrast to the Ret proto-oncogene, the rearranged forms are constitutively phosphorylated on tyrosine and are translocated from the membrane to the cytoplasm. The putative binding site for either SH2 and PTB domains has been identified as Tyr 586 of Ret/Ptc2 (Tyr 1062 on proto-Ret). Tyr 1062 shows features of a multifunctional docking site and Shc activation plays a key role in the transforming pathways triggered by Ret/Ptc oncoproteins.

CHROMOSOMAL LOCATION

Genetic locus: RET (human) mapping to 10q11.21; Ret (mouse) mapping to 6 F1.

SOURCE

p-Ret (Tyr 1062)-R is a rabbit polyclonal antibody raised against a short amino acid sequence containing phosphorylated Tyr 1062 of Ret 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-20252 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

p-Ret (Tyr 1062)-R is recommended for detection of Tyr 1062 phosphorylated Ret 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).

p-Ret (Tyr 1062)-R is also recommended for detection of correspondingly phosphorylated tyr on Ret in additional species, including equine, canine, bovine and porcine.

Suitable for use as control antibody for Ret siRNA (h): sc-36404, Ret siRNA (m): sc-36405, Ret siRNA (r): sc-156121, Ret shRNA Plasmid (h): sc-36404-SH, Ret shRNA Plasmid (m): sc-36405-SH, Ret shRNA Plasmid (r): sc-156121-SH, Ret shRNA (h) Lentiviral Particles: sc-36404-V, Ret shRNA (m) Lentiviral Particles: sc-36405-V and Ret shRNA (r) Lentiviral Particles: sc-156121-V.

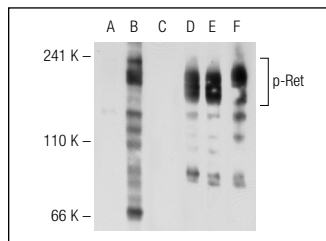
Molecular Weight of precursor p-Ret: 150 kDa.

Molecular Weight of mature p-Ret: 170 kDa.

STORAGE

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

DATA



Western blot analysis of Ret phosphorylation in untreated (A,D), pervanadate treated (B,E) and lambda protein phosphatase (sc-200312A) and pervanadate treated (C,F) TT whole cell lysate. Antibodies tested include p-Ret (Tyr 1062)-R: sc-20252-R (A,B,C) and Ret (H-300): sc-13104 (D,E,F).

SELECT PRODUCT CITATIONS

1. Drosten, M., et al. 2004. Role of MEN2A-derived RET in maintenance and proliferation of medullary thyroid carcinoma. *J. Natl. Cancer Inst.* 96: 1231-1239.
2. Petrangolini, G., et al. 2006. Apoptotic cell death induction and angiogenesis inhibition in large established medullary thyroid carcinoma xenografts by Ret inhibitor RPI-1. *Biochem. Pharmacol.* 72: 405-414.
3. Mise, N., et al. 2006. Evaluation of potential mechanisms underlying genotype-phenotype correlations in multiple endocrine neoplasia type 2. *Oncogene* 25: 6637-6647.
4. Oppenheimer, O., et al. 2007. The Ret oncogene is a critical component of transcriptional programs associated with retinoic acid-induced differentiation in neuroblastoma. *Mol. Cancer Ther.* 6: 1300-1309.
5. Akeno-Stuart, N., et al. 2007. The RET kinase inhibitor NVP-AST487 blocks growth and calcitonin gene expression through distinct mechanisms in medullary thyroid cancer cells. *Cancer Res.* 67: 6956-6964.
6. He, Z., et al. 2008. GDNF upregulates c-Fos transcription via the Ras/ERK 1/2 pathway to promote mouse spermatogonial stem cell proliferation. *Stem Cells* 26: 266-278.
7. Cincinelli, R., et al. 2008. Synthesis, modeling, and Ret protein kinase inhibitory activity of 3- and 4-substituted β -carboline-1-ones. *J. Med. Chem.* 51: 7777-7787.
8. Cassinelli, G., et al. 2009. Ret/PTC1-driven neoplastic transformation and proinvasive phenotype of human thyrocytes involve Met induction and β -catenin nuclear translocation. *Neoplasia* 11: 10-21.
9. Song, R., et al. 2010. Angiotensin II-induced activation of c-Ret signaling is critical in ureteric bud branching morphogenesis. *Mech. Dev.* 127: 21-27.

RESEARCH USE

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