

RET Antibody (N-term Q28)
Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP7669c

Specification

RET Antibody (N-term Q28) - Product Information

Application	WB, FC,E
Primary Accession	P07949
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Antigen Region	13-44

RET Antibody (N-term Q28) - Additional Information

Gene ID 5979

Other Names

Proto-oncogene tyrosine-protein kinase receptor Ret, Cadherin family member 12, Proto-oncogene c-Ret, Soluble RET kinase fragment, Extracellular cell-membrane anchored RET cadherin 120 kDa fragment, RET, CDHF12, CDHR16, PTC, RET51

Target/Specificity

This RET antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 13-44 amino acids from the N-terminal region of human RET.

Dilution

WB~~1:1000

FC~~1:10~50

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.

Storage

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

RET Antibody (N-term Q28) is for research use only and not for use in diagnostic or therapeutic procedures.

RET Antibody (N-term Q28) - Protein Information

Name RET {ECO:0000303|PubMed:2660074, ECO:0000312|HGNC:HGNC:9967}

Function Receptor tyrosine-protein kinase involved in numerous cellular mechanisms including

cell proliferation, neuronal navigation, cell migration, and cell differentiation in response to glia cell line- derived growth family factors (GDNF, NRTN, ARTN, PSPN and GDF15) (PubMed:[20064382](#), PubMed:[20616503](#), PubMed:[20702524](#), PubMed:[21357690](#), PubMed:[21454698](#), PubMed:[24560924](#), PubMed:[28846097](#), PubMed:[28846099](#), PubMed:[28953886](#), PubMed:[31118272](#)). In contrast to most receptor tyrosine kinases, RET requires not only its cognate ligands but also coreceptors, for activation (PubMed:[21994944](#), PubMed:[23333276](#), PubMed:[28846097](#), PubMed:[28846099](#), PubMed:[28953886](#)). GDNF ligands (GDNF, NRTN, ARTN, PSPN and GDF15) first bind their corresponding GDNFR coreceptors (GFRA1, GFRA2, GFRA3, GFRA4 and GFRAL, respectively), triggering RET autophosphorylation and activation, leading to activation of downstream signaling pathways, including the MAPK- and AKT-signaling pathways (PubMed:[21994944](#), PubMed:[23333276](#), PubMed:[24560924](#), PubMed:[25242331](#), PubMed:[28846097](#), PubMed:[28846099](#), PubMed:[28953886](#)). Acts as a dependence receptor via the GDNF-GFRA1 signaling: in the presence of the ligand GDNF in somatotrophs within pituitary, promotes survival and down regulates growth hormone (GH) production, but triggers apoptosis in absence of GDNF (PubMed:[20616503](#), PubMed:[21994944](#)). Required for the molecular mechanisms orchestration during intestine organogenesis via the ARTN-GFRA3 signaling: involved in the development of enteric nervous system and renal organogenesis during embryonic life, and promotes the formation of Peyer's patch-like structures, a major component of the gut-associated lymphoid tissue (By similarity). Mediates, through interaction with GDF15-receptor GFRAL, GDF15-induced cell-signaling in the brainstem which triggers an aversive response, characterized by nausea, vomiting, and/or loss of appetite in response to various stresses (PubMed:[28846097](#), PubMed:[28846099](#), PubMed:[28953886](#)). Modulates cell adhesion via its cleavage by caspase in sympathetic neurons and mediates cell migration in an integrin (e.g. ITGB1 and ITGB3)-dependent manner (PubMed:[20702524](#), PubMed:[21357690](#)). Also active in the absence of ligand, triggering apoptosis through a mechanism that requires receptor intracellular caspase cleavage (PubMed:[21357690](#)). Triggers the differentiation of rapidly adapting (RA) mechanoreceptors (PubMed:[20064382](#)). Involved in the development of the neural crest (By similarity). Regulates nociceptor survival and size (By similarity). Phosphorylates PTK2/FAK1 (PubMed:[21454698](#)).

Cellular Location

Cell membrane; Single-pass type I membrane protein. Endosome membrane; Single-pass type I membrane protein Note=Predominantly located on the plasma membrane (PubMed:[23333276](#), PubMed:[9575150](#)). In the presence of SORL1 and GFRA1, directed to endosomes (PubMed:[23333276](#)).

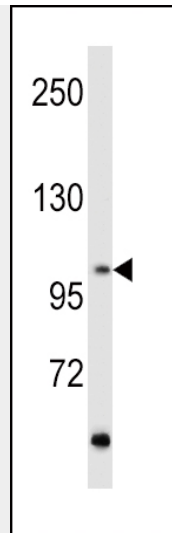
RET Antibody (N-term Q28) - Protocols

Provided below are standard protocols that you may find useful for product applications.

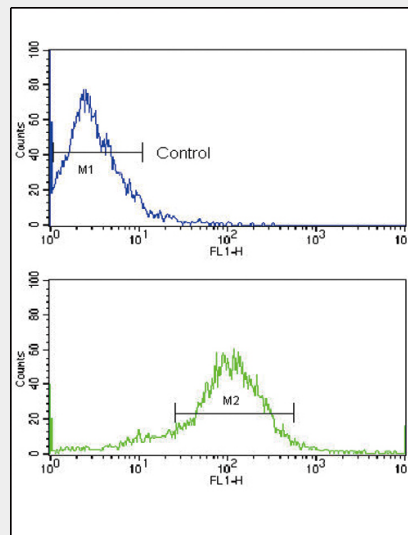
- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

RET Antibody (N-term Q28) - Images





Western blot analysis of hRET-G28 (Cat. #AP7669c) in MCF7 cell line lysates (35ug/lane). RET (arrow) was detected using the purified Pab.



Flow cytometric analysis of MCF-7 cells using RET Antibody (N-term Q28) (bottom histogram) compared to a negative control (top histogram). FITC-conjugated goat-anti-rabbit secondary antibodies were used for the analysis.

RET Antibody (N-term Q28) - Background

RET, a member of the cadherin superfamily, is one of the receptor tyrosine kinases, which are cell-surface molecules that transduce signals for cell growth and differentiation. This protein plays a crucial role in neural crest development, and the gene can undergo oncogenic activation *in vivo* and *in vitro* by cytogenetic rearrangement. Mutations are associated with the disorders multiple endocrine neoplasia, type IIA, multiple endocrine neoplasia, type IIB, Hirschsprung disease, and medullary thyroid carcinoma.

RET Antibody (N-term Q28) - References

- Da Silva, A.M., et al., *J. Clin. Endocrinol. Metab.* 88(11):5438-5443 (2003).
- McWhinney, S.R., et al., *J. Clin. Endocrinol. Metab.* 88(10):4911-4916 (2003).
- D'Alessio, A., et al., *Endocrinology* 144(10):4298-4305 (2003).
- Soares, P., et al., *Oncogene* 22(29):4578-4580 (2003).

Punales, M.K., et al., J. Clin. Endocrinol. Metab. 88(6):2644-2649 (2003).