SunLong Biotech Co.,LTD
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[Primary Antibody]

Rabbit Anti-Ret antibody

Catalog Number: SL6270R

FOR RESEARCH USE ONLY

[DATASHEET]

Host:	Rabbit
Isotype:	IgG
Clonality:	Polyclonal
Target:	Ret
Immunogen:	KLH conjugated synthetic peptide derived from human Ret/HSCR1: 711-756/1114
Purification:	affinity purified by Protein A
Concentration:	1mg/ml
Storage:	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol. Shipped at 4℃. Store at -20 °C for one year. Avoid repeated freeze/thaw cycles.
Applications:	IHC-P=1:100-500 IHC-F=1:100-500 IF=1:100-500 （Paraffin sections need antigen repair） not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Reactivity:	Rat(predicted:Human,Mouse,Chicken,Dog,Cow,Rabbit)
Predicted MW.:	124kDa
Subcellular Location:	cytoplasmic The cell membrane
Background:	This gene, a member of the cadherin superfamily, encodes one of the receptor tyrosine kinases, which are cell-surface molecules that transduce signals for cell growth and differentiation. This gene plays a crucial role in neural crest development, and it can undergo oncogenic activation in vivo and in vitro by cytogenetic rearrangement. Mutations in this gene are associated with the disorders multiple endocrine neoplasia, type IIA, multiple endocrine neoplasia, type IIB, Hirschsprung disease, and medullary thyroid carcinoma. Two transcript variants encoding different isoforms have been found for this gene. Additional transcript variants have been described but their biological validity has not been confirmed. [provided by RefSeq, Jul 2008] **Function:** Receptor tyrosine-protein kinase involved in numerous cellular mechanisms including cell proliferation, neuronal navigation, cell migration,

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	and cell differentiation upon binding with glial cell derived neurotrophic factor family ligands. Phosphorylates PTK2/FAK1. Regulates both cell death/survival balance and positional information. Required for the molecular mechanisms orchestration during intestine organogenesis; 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. 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. Involved in the development of the neural crest. Active in the absence of ligand, triggering apoptosis through a mechanism that requires receptor intracellular caspase cleavage. Acts as a dependence receptor; 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. Regulates nociceptor survival and size. Triggers the differentiation of rapidly adapting (RA) mechanoreceptors. Mediator of several diseases such as neuroendocrine cancers; these diseases are characterized by aberrant integrins-regulated cell migration. **Subunit:** Phosphorylated form interacts with the PBT domain of DOK2, DOK4 and DOK5. The phosphorylated form interacts with PLCG1 and GRB7. Interacts (not phosphorylated) with CC PTK2/FAK1 (via FERM domain). Extracellular cell-membrane anchored RET cadherin fragments form complex in neurons with reduced trophic status, preferentially at the contact sites between somas. Interacts with AIP in the pituitary gland; this interaction prevents the formation of the AIP-survivin complex. Binds to ARTN. Interacts (inactive) with CBLC and CD2AP; dissociates upon activation by GDNF which increases CBLC:CD2AP interaction. **Subcellular Location:** Cell membrane; Single-pass type I membrane protein; Endosome membrane; **Post-translational modifications:** Autophosphorylated on C-terminal tyrosine residues upon ligand stimulation. Dephosphorylated by PTPRJ on Tyr-905, Tyr-1015 and Tyr-1062.5 Publications Proteolytically cleaved by caspase-3. The soluble RET kinase fragment is able to induce cell death. The extracellular cell-membrane anchored RET cadherin fragment accelerates cell adhesion in sympathetic neurons. DISEASE: Defects in RET may be a cause of colorectal cancer (CRC) [MIM:114500]. Defects in RET are a cause of Hirschsprung disease type 1 (HSCR1)

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	[MIM:142623]. HSCR1 is a disorder of neural crest development characterized by the absence of intramural ganglion cells in the myenteric and submucosal plexuses of the gastrointestinal tract, often resulting in intestinal obstruction. Total colonic aganglionosis and total intestinal Hirschsprung disease also occur. Occasionally, MEN2A or FMTC occur in association with HSCR1. Defects in RET are the cause of medullary thyroid carcinoma (MTC) [MIM:155240]. MTC is a rare tumor derived from the C cells of the thyroid. Three hereditary forms are known, that are transmitted in an autosomal dominant fashion: (a) multiple neoplasia type 2A (MEN2A), (b) multiple neoplasia type IIB (MEN2B) and (c) familial MTC (FMTC), which occurs in 25-30% of MTC cases and where MTC is the only clinical manifestation. Defects in RET are the cause of multiple neoplasia type 2B (MEN2B) [MIM:162300]. MEN2B is an uncommon inherited cancer syndrome characterized by predisposition to MTC and phaeochromocytoma which is associated with marfanoid habitus, mucosal neuromas, skeletal and ophtalmic abnormalities, and ganglioneuromas of the intestine tract. Then the disease progresses rapidly with the development of metastatic MTC and a pheochromocytome in 50% of cases. Defects in RET are a cause of susceptibility to pheochromocytoma (PCC) [MIM:171300]. A catecholamine-producing tumor of chromaffin tissue of the adrenal medulla or sympathetic paraganglia. The cardinal symptom, reflecting the increased secretion of epinephrine and norepinephrine, is hypertension, which may be persistent or intermittent. Defects in RET are the cause of multiple neoplasia type 2A (MEN2A) [MIM:171400]; also known as multiple neoplasia type 2 (MEN2). MEN2A is the most frequent form of medullary thyroid cancer (MTC). It is an inherited cancer syndrome characterized by MTC, phaeochromocytoma and/or hyperparathyroidism. Defects in RET are a cause of thyroid papillary carcinoma (TPC) [MIM:188550]. TPC is a common tumor of the thyroid that typically arises as an irregular, solid or cystic mass from otherwise normal thyroid tissue. Papillary carcinomas are malignant neoplasm characterized by the formation of numerous, irregular, finger-like projections of fibrous stroma that is covered with a surface layer of neoplastic epithelial cells. Note=Chromosomal aberrations involving RET are found in thyroid papillary carcinomas. Inversion inv(10)(q11.2;q21) generates the RET/CCDC6 (PTC1) oncogene; inversion inv(10)(q11.2;q11.2) generates the RET/NCOA4 (PTC3) oncogene; translocation t(10;14)(q11;q32) with GOLGA5 generates the RET/GOLGA5 (PTC5) oncogene; translocation t(8;10)(p21.3;q11.2) with

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	PCM1 generates the PCM1/RET fusion; translocation t(6;10)(p21.3;q11.2) with RFP generates the Delta RFP/RET oncogene; translocation t(1;10)(p13;q11) with TRIM33 generates the TRIM33/RET (PTC7) oncogene; translocation t(7;10)(q32;q11) with TRIM24/TIF1 generates the TRIM24/RET (PTC6) oncogene. The PTC5 oncogene has been found in 2 cases of PACT in children exposed to radioactive fallout after Chernobyl. A chromosomal aberration involving TRIM27/RFP is found in thyroid papillary carcinomas. Translocation t(6;10)(p21.3;q11.2) with RET. The translocation generates TRIM27/RET and delta TRIM27/RET oncogenes. Defects in RET are a cause of renal adysplasia (RADYS) [MIM:191830]; also known as renal agenesis or renal aplasia. Renal agenesis refers to the absence of one (unilateral) or both (bilateral) kidneys at birth. Bilateral renal agenesis belongs to a group of perinatally lethal renal diseases, including severe bilateral renal dysplasia, unilateral renal agenesis with contralateral dysplasia and severe obstructive uropathy. Defects in RET are a cause of congenital central hypoventilation syndrome (CCHS) [MIM:209880]; also known as congenital failure of autonomic control or Ondine curse. CCHS is a rare disorder characterized by abnormal control of respiration in the absence of neuromuscular or lung disease, or an identifiable brain stem lesion. A deficiency in autonomic control of respiration results in inadequate or negligible ventilatory and arousal responses to hypercapnia and hypoxemia. **Similarity:** Belongs to the protein kinase superfamily. Tyr protein kinase family. Contains 1 cadherin domain. Contains 1 protein kinase domain. **Database links:** **Entrez Gene:** 5979 Human **Entrez Gene:** 19713 Mouse **Entrez Gene:** 24716 Rat Omim: 164761 Human **SwissProt:** P07949 Human **SwissProt:** P35546 Mouse **SwissProt:** G3V9H8 Rat **Unigene:** 350321 Human **Unigene:** 57199 Mouse

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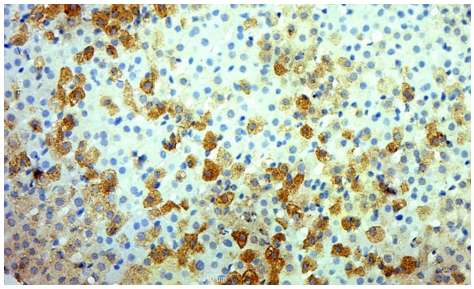
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[VALIDATION IMAGES]



Paraformaldehyde-fixed, paraffin embedded (Rat adrenal gland); Antigen retrieval by boiling in sodium citrate buffer (pH6.0) for 15min; Block endogenous peroxidase by 3% hydrogen peroxide for 20 minutes; Blocking buffer (normal goat serum) at 37°C for 30min; Antibody incubation with (Ret) Polyclonal Antibody, Unconjugated (SL6270R) at 1:500 overnight at 4°C, followed by a conjugated secondary (sp-0023) for 20 minutes and DAB staining.

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