SunLong Biotech Co.,LTD
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[Primary Antibody]

Rabbit Anti-phospho-Ret (Tyr1062)antibody

Catalog Number: SL6271R

FOR RESEARCH USE ONLY

[DATASHEET]

Host:	Rabbit
Isotype:	IgG
Clonality:	Polyclonal
Target:	phospho-Ret (Tyr1062)
Immunogen:	KLH conjugated Synthesised phosphopeptide derived from human Ret around the phosphorylation site of Tyr1062: KL(p-Y)GM
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:	WB=1:500-2000 IHC-P=1:100-500 IHC-F=1:100-500 IF=1:100-500 ELISA=1:5000-10000 （Paraffin sections need antigen repair） not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Reactivity:	(predicted: Human, Mouse, Rat)
Predicted MW.:	119kDa
Subcellular Location:	The cell membrane Secretory protein
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]

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	Function: Receptor tyrosine-protein kinase involved in numerous cellular mechanisms including cell proliferation, neuronal navigation, cell migration, 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)

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	[MIM:114500]. Defects in RET are a cause of Hirschsprung disease type 1 (HSCR1) [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. **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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