

Human RET Kinase Protein (His Tag)



Sino Biological
Biological Solution Specialist

Catalog Number: 11997-H08H1

General Information

Gene Name Synonym:

CDHF12; CDHR16; HSCR1; MEN2A; MEN2B; MTC1; PTC; RET-ELE1; RET51

Protein Construction:

A DNA sequence encoding the extracellular domain of human RET (P07949-1) (Met 1-Arg 635) was fused with a polyhistidine tag at the N-terminus.

Source: Human

Expression Host: HEK293 Cells

QC Testing

Purity: > 92 % as determined by SDS-PAGE

Endotoxin:

< 1.0 EU per µg of the protein as determined by the LAL method

Stability:

Samples are stable for up to twelve months from date of receipt at -70 °C

Predicted N terminal: Leu 29

Molecular Mass:

The recombinant human RET consists of 618 amino acids and has a calculated molecular mass of 69.1 kDa. The apparent molecular mass of the protein is approximately 110-120 kDa in SDS-PAGE under reducing conditions.

Formulation:

Lyophilized from sterile PBS, pH 7.4

Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as protectants before lyophilization. Specific concentrations are included in the hardcopy of COA. Please contact us for any concerns or special requirements.

Usage Guide

Storage:

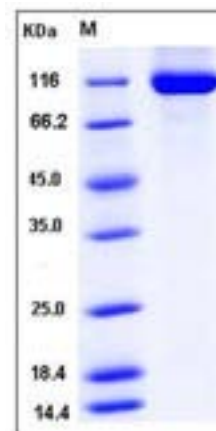
Store it under sterile conditions at -20°C to -80°C upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

Avoid repeated freeze-thaw cycles.

Reconstitution:

Detailed reconstitution instructions are sent along with the products.

SDS-PAGE:



Protein Description

RET proto-oncogene, also known as RET, is a cell-surface molecule that transduce signals for cell growth and differentiation. It contains 1 cadherin domain and 1 protein kinase domain. RET proto-oncogene belongs to the protein kinase superfamily, tyr protein kinase family. RET proto-oncogene is 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. It phosphorylates PTK2/FAK1 and regulates both cell death/survival balance and positional information. RET is required for the molecular mechanisms orchestration during intestine organogenesis; involved in the development of enteric nervous system and renal organogenesis during embryonic life; promotes the formation of Peyer's patch-like structures; modulates cell adhesion via its cleavage; involved in the development of the neural crest. RET proto-oncogene is active in the absence of ligand, triggering apoptosis. RET 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. It also regulates nociceptor survival and size; triggers the differentiation of rapidly adapting (RA) mechanoreceptors; mediated several diseases such as neuroendocrine cancers. Defects in RET may cause colorectal cancer, hirschsprung disease type 1, medullary thyroid carcinoma, multiple neoplasia type 2B, susceptibility to pheochromocytoma, multiple neoplasia type 2A, thyroid papillary carcinoma and congenital central hypoventilation syndrome.

References

- 1.Schulten HJ, *et al.* (2011) Mutational screening of RET, HRAS, KRAS, NRAS, BRAF, AKT1, and CTNNB1 in medullary thyroid carcinoma. *Anticancer Res.* 31(12):4179-83.
- 2.Ciampi R, *et al.* (2012) Chromosome 10 and RET gene copy number alterations in hereditary and sporadic Medullary Thyroid Carcinoma. *Mol Cell Endocrinol.* 348(1):176-82.
- 3.Garcia-Lavandeira M, *et al.* (2012) Craniopharyngiomas express embryonic stem cell markers (SOX2, OCT4, KLF4, and SOX9) as pituitary stem cells but do not coexpress RET/GFRA3 receptors. *J Clin Endocrinol Metab.* 97(1):E80-7.

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