RET Protein, Human (sf9, His-GST)

Cat. No.:	HY-P73678
Synonyms:	Proto-oncogene tyrosine-protein kinase receptor Ret; RET; CDHF12; CDHR16
Species:	Human
Source:	Sf9 insect cells
Accession:	P07949 (H658-S1114)
Gene ID:	5979
Molecular Weight:	Approximately 70 kDa

PROPERTIES

Biological Activity	The specific activity was determined to be 17 nmol/min/mg using synthetic TRK-C-derived Peptide (R11-VYSTDYYRLFNPS) as substrate.
Appearance	Solution.
Formulation	Supplied as a 0.2 μm filtered solution of 20 mM Tris, 500 mM NaCl, 25% gly, 0.5 mM TCEP, 0.5 mM GSH, pH 8.0.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	N/A.
Storage & Stability	Stored at -80°C for 1 year. It is stable at -20°C for 3 months after opening. It is recommended to freeze aliquots at -80°C for extended storage. Avoid repeated freeze-thaw cycles.
Shipping	Shipping with dry ice.

DESCRIPTION

Background	The RET protein, a receptor tyrosine-protein kinase, plays a crucial role in diverse cellular processes such as cell proliferation, neuronal navigation, migration, and differentiation upon binding with glial cell-derived neurotrophic factor family ligands. It phosphorylates PTK2/FAK1 and regulates the delicate balance between cell death and survival, as well as positional information. Essential for the molecular coordination during intestine organogenesis, RET is involved in the development of the enteric nervous system, renal organogenesis, and promotes the formation of Peyer's patch-like structures in the gut-associated lymphoid tissue. Furthermore, RET modulates cell adhesion, mediated by caspase cleavage in sympathetic neurons, and facilitates cell migration in an integrin-dependent manner. Operating as a dependence receptor, it triggers apoptosis in the absence of the ligand GDNF in somatotrophs but promotes survival and downregulates growth hormone production in its presence. RET is a key mediator in various diseases, particularly neuroendocrine cancers characterized by aberrant integrins-regulated cell migration. Additionally, it mediates GDF15-induced cell signaling in the brainstem through interaction with GFRAL, resulting in the inhibition of food intake and activation of MAPK- and AKT-signaling pathways. Notably, isoform 1 in complex with GFRAL induces higher activation of the MAPK-signaling pathway compared to isoform 2 in the same complex.

Caution: Product has not been fully validated for medical applications. For research use only.

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