TrkB Protein, Rat (HEK293, His)

Cat. No.:	HY-P73574
Synonyms:	BDNF/NT-3 Growth Factors Receptor; Trk-B; NTRK2; TRKB
Species:	Rat
Source:	HEK293
Accession:	Q63604-2 (C32-H429)
Gene ID:	25054
Molecular Weight:	Approximately 67 kDa

PROPERTIES

Biological Activity	Measured by its binding ability in a functional ELISA. Immobilized rat NTRK2-His at 10 μg/mL (100 μL/well) can bind mouse BDNF and the EC_{50} is 12.2-28.6 ng/mL.
Appearance	Solution.
Formulation	Supplied as a 0.2 μm filtered solution of PBS, pH 7.4.
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	TrkB, a receptor tyrosine kinase, intricately orchestrates the development and maturation of both the central and peripheral nervous systems, exerting regulatory control over various neuronal processes, including survival, proliferation, migration, differentiation, and synapse formation. Functioning as a receptor for BDNF/brain-derived neurotrophic factor and NTF4/neurotrophin-4, it can alternatively bind NTF3/neurotrophin-3, modulating neuron survival through NTRK2. Upon ligand binding, TrkB undergoes homodimerization, autophosphorylation, and activation, initiating a cascade of downstream events. It recruits, phosphorylates, and activates diverse effectors such as SHC1, FRS2, SH2B1, SH2B2, and PLCG1, which coordinate distinct signaling pathways, including the GRB2-Ras-MAPK cascade for neuronal differentiation, the Ras-PI3 kinase-AKT1 pathway for growth and survival, and PLCG1-regulated pathways for synaptic plasticity. TrkB's involvement in learning and memory encompasses the regulation of short-term synaptic function and long-term potentiation. Additionally, through PLCG1, it activates NF-Kappa-B and transcription of survival-related genes, enabling the suppression of anoikis—a form of apoptosis resulting from the loss of cell-matrix interactions. Notably, TrkB may also contribute to neurotrophin-dependent calcium signaling in glial cells.

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

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