

Product Data Sheet

TrkB Protein, Rat (HEK293, Fc)

Cat. No.: HY-P73575

Synonyms: BDNF/NT-3 Growth Factors Receptor; Trk-B; NTRK2; TRKB

Species:

HEK293 Source:

Accession: Q63604 (M1-H429)

Gene ID: 25054

Molecular Weight: Approximately 120 kDa

PROPERTIES	
Biological Activity	The enzyme activity of this recombinant protein is testing in progress, we cannot offer a guarantee yet.
Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.2 μ m filtered solution of PBS, pH 7.4. Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 μg/mL in ddH ₂ O.
Storage & Stability	Stored at -20°C for 2 years. After reconstitution, it is stable at 4°C for 1 week or -20°C for longer (with carrier protein). It is recommended to freeze aliquots at -20°C or -80°C for extended storage.
Shipping	Room temperature in continental US; may vary elsewhere.

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.

 $\label{lem:caution:Product} \textbf{Caution: Product has not been fully validated for medical applications. For research use only.}$

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