

TrkA Protein, Rat (HEK293, Fc)

Cat. No.:	HY-P73459
Synonyms:	High affinity nerve growth factor receptor; Trk-A; NTRK1; MTC; TRK
Species:	Rat
Source:	HEK293
Accession:	P35739 (M1-P418)
Gene ID:	59109
Molecular Weight:	92-102 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/ μ g, determined by LAL method.
Reconstitution	It is not recommended to reconstitute to a concentration less than 100 μ g/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

The TrkA protein, a receptor tyrosine kinase, plays a vital role in the development and maturation of the central and peripheral nervous systems by regulating the proliferation, differentiation, and survival of sympathetic and sensory neurons. Serving as a high-affinity receptor for NGF, its primary ligand, TrkA can also be activated by NTF3/neurotrophin-3, though NTF3 specifically supports axonal extension through NTRK1 without influencing neuron survival. Upon dimeric NGF ligand-binding, TrkA undergoes homodimerization, autophosphorylation, and activation, subsequently recruiting, phosphorylating, and/or activating downstream effectors such as SHC1, FRS2, SH2B1, SH2B2, and PLCG1. These effectors regulate distinct yet overlapping signaling cascades, steering cell survival and differentiation. Through SHC1 and FRS2, TrkA activates a GRB2-Ras-MAPK cascade controlling cell differentiation and survival, while through PLCG1, it modulates NF-Kappa-B activation and the transcription of genes crucial for cell survival. Additionally, through SHC1 and SH2B1, TrkA controls a Ras-PI3 kinase-AKT1 signaling cascade, further contributing to the regulation of cell survival. In the absence of ligand and activation, TrkA may promote cell death, underscoring the dependence of neuron survival on trophic factors.

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

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