Recombinant Mouse TrkA/NTRK1 Protein (His Tag)

Catalog Number: PKSM040355



Note: Centrifuge before opening to ensure complete recovery of vial contents.

Description

Synonyms C80751;Tkr;trk;TrkA

Species Mouse

Expression Host HEK293 Cells
Sequence Met1-Gly420
Accession NP_001028296.1

Calculated Molecular Weight 43.7 kDa
Observed molecular weight 69 kDa
Tag C-His

Bioactivity Measured by its ability to inhibit NGF-induced proliferation of TF-1 human

erythroleukemic cells. The ED50 for this effect is typically 0.2-1 µg/mL in the

presence of 10 ng/mL of recombinant mouse NGF.

Properties

Purity > 95 % as determined by reducing SDS-PAGE.

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

Storage Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to

-80°C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots

of reconstituted samples are stable at < -20°C for 3 months.

Shipping This product is provided as lyophilized powder which is shipped with ice packs.

Formulation Lyophilized from sterile PBS, pH 7.4

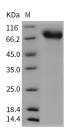
Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as

protectants before lyophilization.

Please refer to the specific buffer information in the printed manual.

Reconstitution Please refer to the printed manual for detailed information.

Data



> 95 % as determined by reducing SDS-PAGE.

Background

TRKA is a member of the neurotrophic tyrosine kinase receptor (NTKR) family. It is a membrane-bound receptor that, upon neurotrophin binding, phosphorylates itself and members of the MAPK pathway. Isoform TrkA-III promotes angiogenesis and has oncogenic activity when overexpressed. Isoform TrkA-I is found in most non-neuronal tissues. Isoform TrkA-II is primarily expressed in neuronal cells. TrkA-III is specifically expressed by pluripotent neural stem and neural crest progenitors. The presence of NTRK1 leads to cell differentiation and may play a role in specifying sensory

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neuron subtypes. Mutations in TRKA gene have been associated with congenital insensitivity to pain, anhidrosis, selfmutilating behavior, mental retardation and cancer. It was originally identified as an oncogene as it is commonly mutated in cancers, particularly colon and thyroid carcinomas. TRKA is required for high-affinity binding to nerve growth factor (NGF), neurotrophin-3 and neurotrophin-4/5 but not brain-derived neurotrophic factor (BDNF). Known substrates for the Trk receptors are SHC1, PI 3-kinase, and PLC-gamma-1. NTRK1 has a crucial role in the development and function of the nociceptive reception system as well as establishment of thermal regulation via sweating. It also activates ERK1 by either SHC1- or PLC-gamma-1-dependent signaling pathway. Defects in NTRK1 are a cause of congenital insensitivity to pain with anhidrosis and thyroid papillary carcinoma.

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