

Trk B Polyclonal Antibody
Catalog # AP72926**Specification**

Trk B Polyclonal Antibody - Product Information

Application	WB, IHC-P
Primary Accession	Q16620
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal

Trk B Polyclonal Antibody - Additional Information**Gene ID** 4915**Other Names**

NTRK2; TRKB; BDNF/NT-3 growth factors receptor; GP145-TrkB; Trk-B; Neurotrophic tyrosine kinase receptor type 2; TrkB tyrosine kinase; Tropomyosin-related kinase B

Dilution

WB~~Western Blot: 1/500 - 1/2000. Immunohistochemistry: 1/100 - 1/300. ELISA: 1/20000. Not yet tested in other applications.

IHC-P~~N/A

Format

Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.09% (W/V) sodium azide.

Storage Conditions

-20°C

Trk B Polyclonal Antibody - Protein Information**Name** NTRK2**Synonyms** TRKB**Function**

Receptor tyrosine kinase involved in the development and the maturation of the central and the peripheral nervous systems through regulation of neuron survival, proliferation, migration, differentiation, and synapse formation and plasticity (By similarity). Receptor for BDNF/brain-derived neurotrophic factor and NTF4/neurotrophin-4. Alternatively can also bind NTF3/neurotrophin-3 which is less efficient in activating the receptor but regulates neuron survival through NTRK2 (PubMed:<<http://www.uniprot.org/citations/15494731>>15494731, PubMed:<<http://www.uniprot.org/citations/7574684>>7574684). Upon ligand- binding, undergoes homodimerization, autophosphorylation and activation (PubMed:<<http://www.uniprot.org/citations/15494731>>15494731). Recruits, phosphorylates and/or activates several downstream effectors including SHC1, FRS2, SH2B1, SH2B2 and PLCG1 that regulate distinct overlapping

signaling cascades. Through SHC1, FRS2, SH2B1, SH2B2 activates the GRB2-Ras-MAPK cascade that regulates for instance neuronal differentiation including neurite outgrowth. Through the same effectors controls the Ras-PI3 kinase-AKT1 signaling cascade that mainly regulates growth and survival. Through PLCG1 and the downstream protein kinase C-regulated pathways controls synaptic plasticity. Thereby, plays a role in learning and memory by regulating both short term synaptic function and long-term potentiation. PLCG1 also leads to NF-Kappa-B activation and the transcription of genes involved in cell survival. Hence, it is able to suppress anoikis, the apoptosis resulting from loss of cell-matrix interactions. May also play a role in neutrophin-dependent calcium signaling in glial cells and mediate communication between neurons and glia.

Cellular Location

Cell membrane; Single-pass type I membrane protein. Endosome membrane {ECO:0000250|UniProtKB:P15209}; Single-pass type I membrane protein {ECO:0000250|UniProtKB:P15209}. Early endosome membrane {ECO:0000250|UniProtKB:P15209}. Cell projection, axon {ECO:0000250|UniProtKB:Q63604}. Cell projection, dendrite {ECO:0000250|UniProtKB:Q63604}. Cytoplasm, perinuclear region {ECO:0000250|UniProtKB:Q63604}. Postsynaptic density {ECO:0000250|UniProtKB:P15209}. Note=Internalized to endosomes upon ligand-binding. {ECO:0000250|UniProtKB:P15209}

Tissue Location

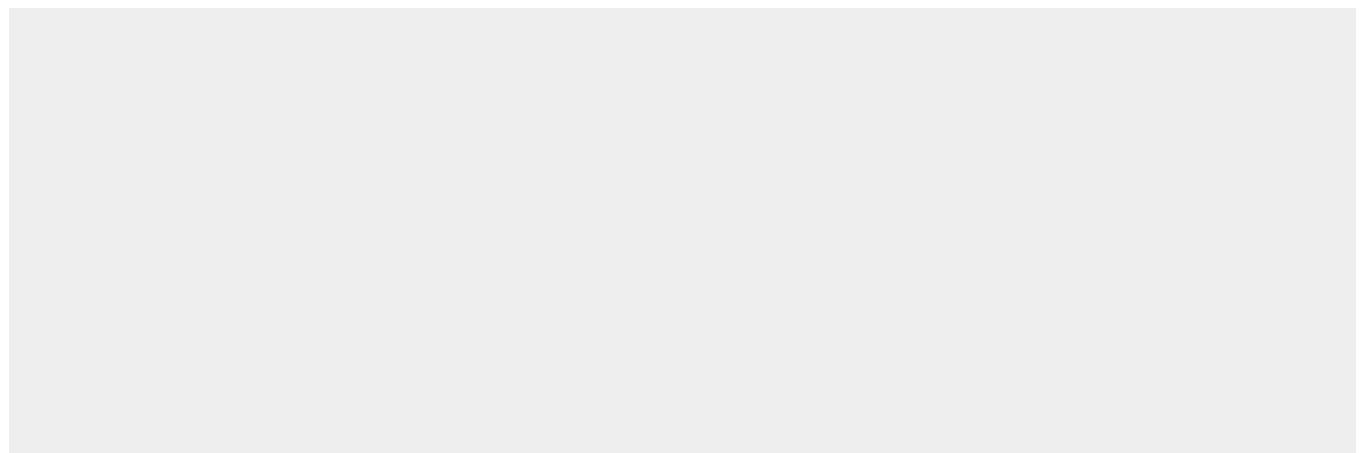
Isoform TrkB is expressed in the central and peripheral nervous system. In the central nervous system (CNS), expression is observed in the cerebral cortex, hippocampus, thalamus, choroid plexus, granular layer of the cerebellum, brain stem, and spinal cord. In the peripheral nervous system, it is expressed in many cranial ganglia, the ophthalmic nerve, the vestibular system, multiple facial structures, the submaxillary glands, and dorsal root ganglia Isoform TrkB-T1 is mainly expressed in the brain but also detected in other tissues including pancreas, kidney and heart. Isoform TrkB-T-Shc is predominantly expressed in the brain.

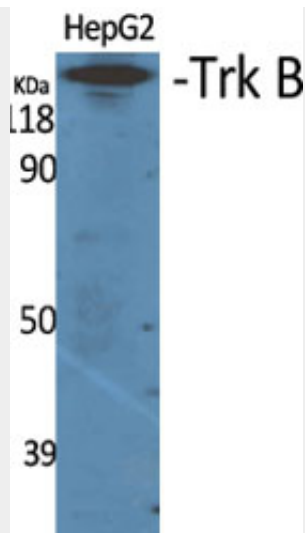
Trk B Polyclonal Antibody - Protocols

Provided below are standard protocols that you may find useful for product applications.

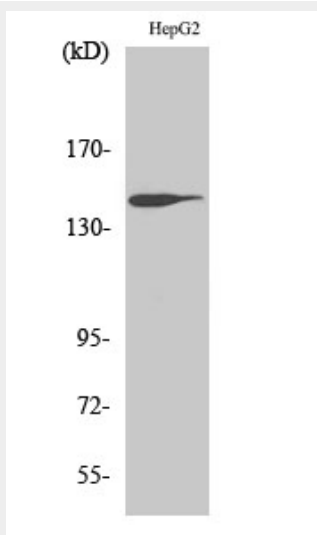
- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

Trk B Polyclonal Antibody - Images

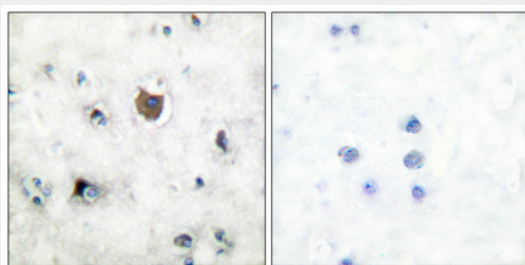




Western Blot analysis of various cells using Trk B Polyclonal Antibody diluted at 1:500. Secondary antibody was diluted at 1:20000



Western Blot analysis of HepG2 cells using Trk B Polyclonal Antibody diluted at 1:500. Secondary antibody was diluted at 1:20000



Immunohistochemical analysis of paraffin-embedded Human breast cancer. Antibody was diluted at 1:100(4°,overnight). High-pressure and temperature Tris-EDTA,pH8.0 was used for antigen retrieval. Negative contrl (right) obtained from antibody was pre-absorbed by immunogen peptide.

Trk B Polyclonal Antibody - Background

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differentiation, and synapse formation and plasticity. Receptor for BDNF/brain-derived neurotrophic factor and NTF4/neurotrophin- 4. Alternatively can also bind NTF3/neurotrophin-3 which is less efficient in activating the receptor but regulates neuron survival through NTRK2. Upon ligand-binding, undergoes homodimerization, autophosphorylation and activation. Recruits, phosphorylates and/or activates several downstream effectors including SHC1, FRS2, SH2B1, SH2B2 and PLCG1 that regulate distinct overlapping signaling cascades. Through SHC1, FRS2, SH2B1, SH2B2 activates the GRB2-Ras-MAPK cascade that regulates for instance neuronal differentiation including neurite outgrowth. Through the same effectors controls the Ras-PI3 kinase-AKT1 signaling cascade that mainly regulates growth and survival. Through PLCG1 and the downstream protein kinase C-regulated pathways controls synaptic plasticity. Thereby, plays a role in learning and memory by regulating both short term synaptic function and long-term potentiation. PLCG1 also leads to NF-Kappa-B activation and the transcription of genes involved in cell survival. Hence, it is able to suppress anoikis, the apoptosis resulting from loss of cell-matrix interactions. May also play a role in neurotrophin- dependent calcium signaling in glial cells and mediate communication between neurons and glia.