

## TRKB Antibody (C-term) Blocking Peptide

Synthetic peptide Catalog # BP7687a

## **Specification**

## TRKB Antibody (C-term) Blocking Peptide - Product Information

Primary Accession

<u>016620</u>

# TRKB Antibody (C-term) Blocking Peptide - Additional Information

**Gene ID 4915** 

#### **Other Names**

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

## **Target/Specificity**

The synthetic peptide sequence used to generate the antibody <a href=/product/products/AP7687a>AP7687a</a> was selected from the C-term region of human TRKB . A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

### **Format**

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

#### Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

#### **Precautions**

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

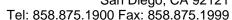
### TRKB Antibody (C-term) Blocking Peptide - Protein Information

### 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:<a href="http://www.uniprot.org/citations/7574684" target="\_blank">7574684</a>, PubMed:<a href="http://www.uniprot.org/citations/15494731" target="\_blank">15494731</a>(a>). Upon ligand- binding, undergoes homodimerization, autophosphorylation and activation (PubMed:<a href="http://www.uniprot.org/citations/15494731")





target="\_blank">15494731</a>). 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.

## TRKB Antibody (C-term) Blocking Peptide - Protocols

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

# Blocking Peptides

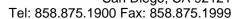
TRKB Antibody (C-term) Blocking Peptide - Images

## TRKB Antibody (C-term) Blocking Peptide - Background

TRKB, a member of the insulin receptor subfamily of Tyr protein kinases, is a receptor for brain-derived neurotrophic factor (BDNF), neurotrophin-3 and neurotrophin-4/5 but not nerve growth factor (NGF). This Type I membrane protein is involved in the development and/or maintenance of the nervous system. This is a tyrosine-protein kinase receptor. Known substrates for the TRK receptors are SHC, PI-3 kinase, and PLC-gamma-1. Isoform TrkB is widely expressed, mainly in the nervous tissue. In the 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 ophtalmic nerve, the vestibular system, multiple facial structures, the submaxillary glands, and dorsal root ganglia. Isoform TrkB-T1 is expressed in multiple tissues, mainly in brain, pancreas, kidney and heart. Isoform TrkB-T-Shc is predominantly expressed in brain. TRKB is subject to ligand-mediated auto-phosphorylation. The structure contains 2 immunoglobulin-like C2-type domains and 2 leucine-rich (LRR) repeats.

### TRKB Antibody (C-term) Blocking Peptide - References







Stoilov, P., et al., Biochem. Biophys. Res. Commun. 290(3):1054-1065 (2002). Strausberg, R.L., et al., Proc. Natl. Acad. Sci. U.S.A. 99(26):16899-16903 (2002).Nakagawara, A., et al., Genomics 25(2):538-546 (1995). Haniu, M., et al., Arch. Biochem. Biophys. 322(1):256-264 (1995). Allen, S.J., et al., Neuroscience 60(3):825-834 (1994).