

Trk A (Phospho Tyr791) Rabbit pAb

CatalogNo: YP0954

Key Features

Host Species

- Rabbit

Reactivity

- Human, Mouse, Rat

Applications

- IHC, IF, ELISA

MW

- 140-180kD (Observed)

Isotype

- IgG

Storage

Storage* -15°C to -25°C/1 year (Do not lower than -25°C)

Formulation Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.

Recommended Dilution Ratios

IHC 1:100-1:300

ELISA 1:5000

IF 1:50-200

Basic Information

Clonality Polyclonal

Immunogen Information

Immunogen The antiserum was produced against synthesized peptide derived from human Trk A around the phosphorylation site of Tyr791. AA range: 747-796

Specificity Phospho-Trk A (Y791) Polyclonal Antibody detects endogenous levels of Trk A protein only when phosphorylated at Y791. The name of modified sites may be influenced by many factors, such as species (the modified site was not originally found in human samples) and the change of protein sequence (the previous protein sequence is incomplete, and the protein sequence may be prolonged with the development of protein sequencing technology). When naming, we will use the "numbers" in historical reference to keep the sites consistent with the reports. The antibody binds to the following modification sequence (lowercase letters are modification sites): PVyLD

| Target Information

Gene name NTRK1 MTC TRK TRKA

Protein Name High affinity nerve growth factor receptor

Organism	Gene ID	UniProt ID
Human	4914;	P04629;
Mouse	18211;	Q3UFB7;
Rat	59109;	P35739;

**Cellular
Localization**

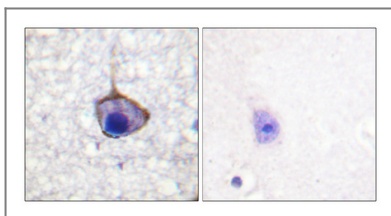
Cell membrane ; Single-pass type I membrane protein . Early endosome membrane ; Single-pass type I membrane protein . Late endosome membrane ; Single-pass type I membrane protein . Recycling endosome membrane ; Single-pass type I membrane protein . Rapidly internalized after NGF binding (PubMed:1281417). Internalized to endosomes upon binding of NGF or NTF3 and further transported to the cell body via a retrograde axonal transport. Localized at cell membrane and early endosomes before nerve growth factor (NGF) stimulation. Recruited to late endosomes after NGF stimulation. Colocalized with RAPGEF2 at late endosomes. .

Tissue specificity 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.

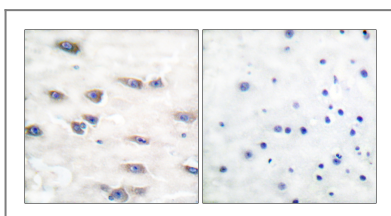
Function

Alternative products:Both isoforms have similar biological properties,Catalytic activity:ATP + a [protein]-L-tyrosine = ADP + a [protein]-L-tyrosine phosphate.,Caution:The sequence shown here is derived from an Ensembl automatic analysis pipeline and should be considered as preliminary data.,Disease:Chromosomal aberrations involving NTRK1 are a cause of thyroid papillary carcinoma (PACT) [MIM:188550]. Intrachromosomal rearrangement that links the protein kinase domain of NTRK1 to the 5'-end of the TPR gene forms the fusion protein TRK-T1. TRK-T1 is a 55 kDa protein reacting with antibodies against the C-terminus of the NTRK1 protein.,Disease:Chromosomal aberrations involving NTRK1 are a cause of thyroid papillary carcinoma (PACT) [MIM:188550]. Translocation t(1;3)(q21;q11) with TFG generates the TRKT3 (TRK-T3) transcript by fusing TFG to the 3'-end of NTRK1; a rearrangement with TPM3 generates the TRK transcript by fusing TPM3 to the 3'-end of NTRK1.,Disease:Defects in NTRK1 are a cause of congenital insensitivity to pain with anhidrosis (CIPA) [MIM:256800]. CIPA is characterized by a congenital insensitivity to pain, anhidrosis (absence of sweating), absence of reaction to noxious stimuli, self-mutilating behavior, and mental retardation. This rare autosomal recessive disorder is also known as congenital sensory neuropathy with anhidrosis or hereditary sensory and autonomic neuropathy type IV or familial dysautonomia type II.,Domain:The extracellular domain mediates interaction with NGFR.,Domain:The transmembrane domain mediates interaction with KIDINS220.,Function: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. Has a crucial role in the development and function of the nociceptive reception system as well as establishment of thermal regulation via sweating. Activates ERK1 by either SHC1- or PLC-gamma-1-dependent signaling pathway.,PTM:Ligand-mediated auto-phosphorylation. Interaction with SQSTM1 is phosphotyrosine-dependent.,similarity:Belongs to the protein kinase superfamily. Tyr protein kinase family.,similarity:Belongs to the protein kinase superfamily. Tyr protein kinase family. Insulin receptor subfamily.,similarity:Contains 1 protein kinase domain.,similarity:Contains 2 Ig-like C2-type (immunoglobulin-like) domains.,similarity:Contains 3 LRR (leucine-rich) repeats.,subcellular location:Endocytosed to the endosomes upon treatment of cells with NGF.,subunit:Exists in a dynamic equilibrium between monomeric (low affinity) and dimeric (high affinity) structures. Binds SH2B2. Interacts with SQSTM1 which bridges NTRK1 to NGFR. Interacts with KIDINS220 and NGFR. Can form a ternary complex with NGFR and KIDINS220 and this complex is affected by the expression levels of KIDINS220. An increase in KIDINS220 expression leads to a decreased association of NGFR and NTRK1.,tissue specificity:Isoform TrkA-II is primarily expressed in neuronal cells; isoform TrkA-I is found in non-neuronal tissues.,

Validation Data



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.



Immunohistochemistry analysis of paraffin-embedded human brain, using Trk A (Phospho-Tyr791) Antibody. The picture on the right is blocked with the phospho peptide.

| Contact information

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Please scan the QR code to access additional product information:
Trk A (Phospho Tyr791) Rabbit pAb

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