

## NTRK2

**Reactivity:**Human Mouse Rat

**Tested applications:**WB IHC

**Recommended Dilution:**WB 1:500 - 1:2000 IHC 1:50 - 1:200

**Calculated MW:**90kDa

**Observed MW:**Refer to Figures

**Immunogen:**

Recombinant protein of human NTRK2

**Storage Buffer:**

Store at -20. Avoid freeze / thaw cycles. Buffer: PBS with 0.02% sodium azide, 50% glycerol, pH7.3.

**Synonym:**

GP145-TrkB; TRKB;

**Catalog #:**A2099

**Antibody Type:**

Polyclonal Antibody

**Species:**Rabbit

**Gene ID:**4915

**Isotype:**IgG

**Swiss Prot:**Q16620

**Purity:**Affinity purification

For research use only.

**Background:**

The family of Trk receptor tyrosine kinases consists of TrkA, TrkB, and TrkC. While the sequence of these family members is highly conserved, they are activated by different neurotrophins: TrkA by NGF, TrkB by BDNF or NT4, and TrkC by NT3 (1). Neurotrophin signaling through these receptors regulates a number of physiological processes, such as cell survival, proliferation, neural development, and axon and dendrite growth and patterning (1). In the adult nervous system, the Trk receptors regulate synaptic strength and plasticity. TrkA regulates proliferation and is important for development and maturation of the nervous system (2). Phosphorylation at Tyr490 is required for Shc association and activation of the Ras-MAP kinase cascade (3,4). Residues Tyr674/675 lie within the catalytic domain, and phosphorylation at these sites reflects TrkA kinase activity (3-6). Point mutations, deletions, and chromosomal rearrangements (chimeras) cause ligand-independent receptor dimerization and activation of TrkA (7-10). TrkA is activated in many malignancies including breast, ovarian, prostate, and thyroid carcinomas (8-13). Research studies suggest that expression of TrkA in neuroblastomas may be a good prognostic marker as TrkA signals growth arrest and differentiation of cells originating from the neural crest (10).

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