# TrkB (80G2) Rabbit mAb



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#### For Research Use Only. Not for Use in Diagnostic Procedures.

Applications: IHC-P	Reactivity:	Sensitivity: Endogenous	<b>MW (kDa):</b> 140	Source/Isotype: Rabbit IgG	UniProt ID: #Q16620	Entrez-Gene Id 4915	
Product Usage Information	Ap	Application				Dilution	
	lmı	Immunohistochemistry (Paraffin)				1:2560	
Storage	•	Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 μ 0.02% sodium azide. Store at –20°C. Do not aliquot the antibody.				erol and less than	
Specificity / Sensitiv		TrkB (80G2) Rabbit mAb detects endogenous levels of total TrkB protein. The antibody does not cross-react with TrkA.					
Species predicted to	o Mou	Mouse, Rat					

react based on 100% sequence homology:

Source / Purification

Monoclonal antibody is produced by immunizing animals with a synthetic peptide surrounding Pro50 of human TrkB.

## **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).

The phosphorylation sites are conserved between TrkA and TrkB: Tyr490 of TrkA corresponds to Tyr512 in TrkB, and Tyr674/675 of TrkA to Tyr706/707 in TrkB of the human sequence (14). TrkB is overexpressed in tumors, such as neuroblastoma, prostate adenocarcinoma, and pancreatic ductal adenocarcinoma (15). Research studies have shown that in neuroblastomas, overexpression of TrkB correlates with an unfavorable disease outcome when autocrine loops signaling tumor survival are potentiated by additional overexpression of brain-derived neurotrophic factor (BDNF) (16-18). An alternatively spliced truncated TrkB isoform lacking the kinase domain is overexpressed in Wilms' tumors and this isoform may act as a dominant-negative regulator of TrkB signaling (17).

#### **Background References**

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#### **Species Reactivity**

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

## **Applications Key**

**IHC-P:** Immunohistochemistry (Paraffin)

### **Cross-Reactivity Key**

H: human M: mouse R: rat Hm: hamster Mk: monkey Vir: virus Mi: mink C: chicken Dm: D. melanogaster X: Xenopus Z: zebrafish B: bovine Dg: dog Pg: pig Sc: S. cerevisiae Ce: C. elegans Hr: horse

GP: Guinea Pig Rab: rabbit All: all species expected

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