

**#3376** Store at -20C

## TrkC (C44H5) Rabbit mAb


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

| Applications:        | Reactivity: | Sensitivity: | MW (kDa): | Source/Isotype: | UniProt ID: | Entrez-Gene Id: |
|----------------------|-------------|--------------|-----------|-----------------|-------------|-----------------|
| WB, IP, IHC-P, IF-IC | H M R       | Endogenous   | 145, 100  | Rabbit IgG      | #Q16288     | 4916            |

### Product Usage Information

#### Application

Western Blotting  
Immunoprecipitation  
Immunohistochemistry (Paraffin)  
Immunofluorescence (Immunocytochemistry)

#### Dilution

1:1000  
1:50  
1:1000  
1:1600

### Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at -20°C. Do not aliquot the antibody.

### Specificity / Sensitivity

TrkC (C44H5) Rabbit mAb detects endogenous levels of total TrkC protein. The antibody does not cross-react with TrkA or TrkB.

### Source / Purification

Monoclonal antibody is produced by immunizing animals with a synthetic peptide surrounding Gly50 of human TrkC.

### 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 TrkC: Tyr490 of TrkA corresponds to Tyr516 in TrkC, and Tyr674/675 of TrkA to Tyr709/710 in TrkC of the human sequence (14). Research studies have demonstrated altered TrkC expression and corresponding gene mutations in various forms of cancer, with increased expression as a potential positive prognostic indicator in patients with medulloblastoma (15).

### Background References

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**Species Reactivity**

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

**Western Blot Buffer**

**IMPORTANT:** For western blots, incubate membrane with diluted primary antibody in 5% w/v BSA, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

**Applications Key**

**WB:** Western Blotting **IP:** Immunoprecipitation **IHC-P:** Immunohistochemistry (Paraffin)  
**IF-IC:** Immunofluorescence (Immunocytochemistry)

**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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