

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human TrkC in direct ELISAs and Western blots. In Western blots, no cross-reactivity with recombinant human TrkB is observed.
Source	Monoclonal Mouse IgG _{2B} Clone # 75219
Purification	Protein A or G purified from ascites
Immunogen	Mouse myeloma cell line NS0-derived recombinant human TrkC Cys32-Asp428 Accession # Q96CY4
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied as a 0.2 µm filtered solution in PBS.

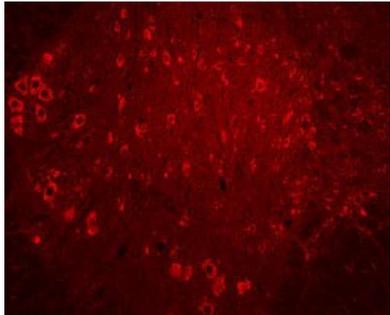
APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. General Protocols are available in the Technical Information section on our website.

	Recommended Concentration	Sample
Western Blot	1 µg/mL	Recombinant Human TrkC Fc Chimera (Catalog # 373-TC)
Immunohistochemistry	8-25 µg/mL	See Below

DATA

Immunohistochemistry



TrkC in Rat Spinal Cord. TrkC was detected in perfusion fixed frozen sections of rat spinal cord using 15 µg/mL Mouse Anti-Human TrkC Monoclonal Antibody (Catalog # MAB373) overnight at 4 °C. Tissue was stained (red). View our protocol for [Fluorescent IHC Staining of Frozen Tissue Sections](#).

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.5 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 6 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

The neurotrophins, including NGF, BDNF, NT-3, and NT-4/5, constitute a group of structurally related, secreted proteins that play an important role in the development and function of the nervous system. The biological activities of the neurotrophins are mediated by binding to and activating two unrelated receptor types: the p75 neurotrophin receptor (p75NTR) and the Trk family of receptor tyrosine kinases (1, 2). p75NTR is a member of the tumor necrosis factor receptor superfamily (TNFRSF) and has been designated TNFRSF16. It binds all neurotrophins with low-affinity to transduce cellular signaling pathways that synergize or antagonize those activated by the Trk receptors. Three Trk family proteins, TrkA, TrkB, and TrkC, exhibiting different ligand specificities, have been identified. TrkA binds NGF and NT-3, TrkB binds BDNF, NT-3 and NT-4/5, and TrkC only binds NT-3 (1-2). All Trk family proteins share a conserved, complex subdomain organization consisting of a signal peptide, two cysteine-rich domains, a cluster of three leucine-rich motifs, and two immunoglobulin-like domains in the extracellular region, as well as an intracellular region that contains the tyrosine kinase domain (3). Natural splice variants of the different Trks, lacking the first cysteine-rich domain, the first and second or all three of the leucine-rich motifs, or the tyrosine kinase domain, have been described (4). At the protein sequence level, Trks are highly conserved between species with the extracellular domains of human and mouse TrkC's showing 94% amino acid sequence identity (5). The proteins also exhibit cross-species activity. The primary location of TrkC expression is in the nervous system and, specifically, in regions of the CNS. Low level TrkC expression has also been observed in a wide variety of tissues outside the nervous system (6).

References:

1. Huang, E.J. and L.F. Reichardt. (2003) *Annu. Rev. Biochem.* **72**:(epub ahead of print).
2. Dechant, G. (2001) *Cell Tissue Res.* **305**:229.
3. Schneider, R. and M. Schweiger (1991) *Oncogene* **6**:1807.
4. Ninkina, N. *et al.* (1997) *J. Biol. Chem.* **272**:13019.
5. Menn, B. *et al.* (1998) *J. Comp. Neurol.* **401**:47.
6. Shelton, D. *et al.* (1995) *J. Neurosci.* **15**:477.