

TrkA and TrkB Antibody Sampler Kit



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1 Kit (5 x 20 microliters)

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

Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
Phospho-TrkA (Tyr490)/TrkB (Tyr516) (C35G9) Rabbit mAb	4619	20 µl	140 kDa	Rabbit IgG
Phospho-TrkA (Tyr674/675)/TrkB (Tyr706/707) (C50F3) Rabbit mAb	4621	20 µl	140 kDa	Rabbit IgG
TrkA (12G8) Rabbit mAb	2510	20 µl	140 kDa	Rabbit IgG
TrkB (80E3) Rabbit mAb	4603	20 µl	90, 140 kDa	Rabbit IgG
Trk (pan) (A7H6R) Rabbit mAb	92991	20 µl	120-140 kDa	Rabbit IgG
Anti-rabbit IgG, HRP-linked Antibody	7074	100 µl		Goat

Please visit cellsignal.com for individual component applications, species cross-reactivity, dilutions, protocols, and additional product information.

Description

The TrkA and TrkB Antibody Sampler Kit provides an economical means to investigate the Trk family of tyrosine kinase receptors. The kit contains enough primary and secondary antibodies to perform four Western blots with each antibody.

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.

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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