Revision 1

Store at +4C

#7106

PathScan® Total TrkB Sandwich ELISA Kit

Cell Signaling TECHNOLOGY®

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UniProt ID: #Q16620 **Entrez-Gene Id:** #4915

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

Product Includes	Product #	Quantity	Color	Storage Temp
TMB Substrate	7004	11 ml	Colorless	+4C
STOP Solution	7002	11 ml	Colorless	+4C
Sealing Tape	54503	2 ea		+4C
ELISA Wash Buffer (20X)	9801	25 ml	Colorless	+4C
ELISA Sample Diluent	11083	25 ml	Blue	+4C
Cell Lysis Buffer (10X)	9803	15 ml	Yellowish	-20C

Kit contents scale proportionally with size, except sealing tape.
Example: The V1 kit contains 5X the listed quantities above, but will exclude the sealing tape.

The microwell plate is supplied as 12 8-well modules - Each module is designed to break apart for 8 tests.

Description

The PathScan® Total TrkB Sandwich ELISA Kit is a solid phase sandwich enzyme-linked immunosorbent assay (ELISA) that detects transfected levels of total TrkB protein. A TrkB Mouse Antibody has been coated onto the microwells. After incubation with cell lysates, TrkB (phospho and nonphospho) is captured by the coated antibody. Following extensive washing, a TrkB Rabbit Detection Antibody is added to detect tyrosine phosphorylation of the captured TrkB protein. Anti-rabbit IgG HRP-Linked Antibody is then used to recognize the bound detection antibody. HRP substrate, TMB, is added to develop color. The magnitude of the absorbance for this developed color is proportional to the quantity of TrkB protein.

Specificity/Sensitivity

CST's PathScan® Total TrkB Sandwich ELISA Kit #7106 detects transfected levels of total TrkB protein. The kit sensitivity is shown in figure 2. This kit detects proteins from the indicated species, as determined through in-house testing, but may also detect homologous proteins from other species.

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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Limited Uses

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