Data Sheet (Cat.No.TMAC-04133)

Anti-TrkA/B/C Antibody (9N825)

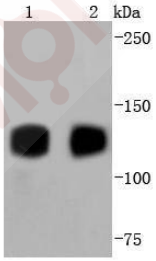
Product Details

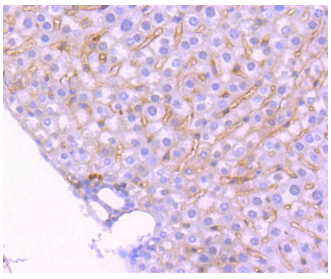
Ig Type:	IgG
Reactivity:	Human,Mouse,Rat
Conjugation:	Unconjugated
Molecular Weight:	Theoretical: 140 kDa.
Clone:	9N825
Purification:	ProA affinity purified

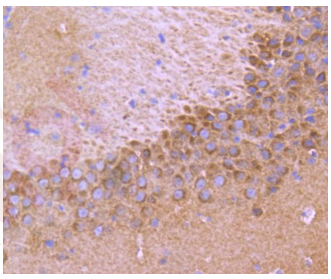
Applications

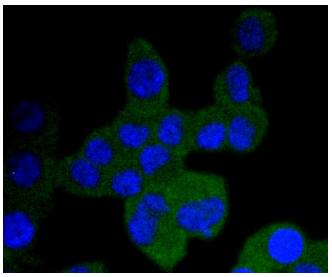
Verified Activity:

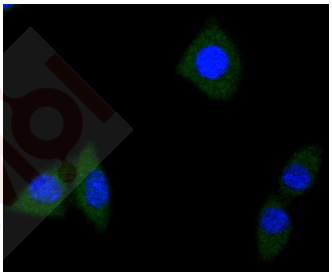
1. Western blot analysis of TrkA+B+C on different lysates using anti-TrkA+B+C antibody at 1/1,000 dilution. Positive control: Lane 1: Rat brain, Lane 2: Mouse brain.
2. Immunohistochemical analysis of paraffin-embedded mouse liver tissue using anti-TrkA+B+C antibody. Counter stained with hematoxylin.
3. Immunohistochemical analysis of paraffin-embedded mouse brain tissue using anti-TrkA+B+C antibody. Counter stained with hematoxylin.
4. ICC staining TrkA+B+C in N2A cells (green). The nuclear counter stain is DAPI (blue). Cells were fixed in paraformaldehyde, permeabilised with 0.25% Triton X100/PBS.
5. ICC staining TrkA+B+C in SH-SY-5Y cells (green). The nuclear counter stain is DAPI (blue). Cells were fixed in paraformaldehyde, permeabilised with 0.25% Triton X100/PBS.











Application: ICC,IHC,WB

Recommended: WB: 1:1000-5000; IHC: 1:50-200; ICC: 1:50-200

Properties

Stability & Storage: Store at -20°C or -80°C for 12 months. Avoid repeated freeze-thaw cycles.

Shipping: Shipping with blue ice.

Antigen Details

Immunogen: Recombinant Protein

Synonyms: neurotrophic tyrosine kinase, receptor, type 1

Research 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. 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. Point mutations, deletions, and chromosomal rearrangements (chimeras) cause ligand-independent receptor dimerization and activation of TrkA. TrkA is activated in many malignancies including breast, ovarian, prostate, and thyroid carcinomas. TrkB is overexpressed in tumors such as neuroblastoma, prostate adenocarcinoma and pancreatic ductal adenocarcinoma. In neuroblastomas overexpression of TrkB correlates with unfavorable disease outcome when autocrine loops signaling tumor survival are potentiated by additional overexpression of brain-derived neurotrophic factor (BDNF). An alternatively spliced truncated TrkB isoform lacking the kinase domain is overexpressed in Wilms?? tumors and this isoform may act as a dominant-negative to TrkB signaling. Altered TrkC expression and corresponding gene mutations are seen in various forms of cancer, with increased expression a positive prognostic indicator in patients with medulloblastoma.

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