



Product Information Sheet

Polyclonal Anti- Trk-A

Catalogue No. PA1321

Lot No. 09L01

Ig type rabbit IgG

Size 100µg/vial

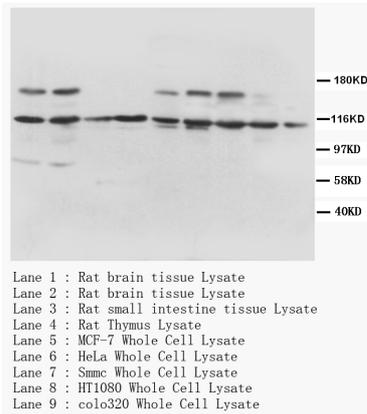
Specificity

Human, rat, mouse.

No cross reactivity with other proteins.

Recommended application

Western blot



Immunogen

A synthetic peptide corresponding to a sequence at the middle region of human Trk-A, identical to the related rat and mouse sequence.

Purity

Immunogen affinity purified.

Application

	Concentration	Tested Species	Concluded Species	Antigen Retrieval
WB	1µg/ml	Hu, Rat	Ms	-
IHC-P	-	-	-	-
IHC-F	-	-	-	-
ICC	-	-	-	-

Other applications have not been tested.

Optimal dilutions should be determined by end user.

Contents

Each vial contains 5mg BSA, 0.9mg NaCl, 0.2mg Na₂HPO₄, 0.05mg Thimerosal, 0.05mg NaN₃.

Reconstitution

0.2ml of distilled water will yield a concentration of 500µg/ml.

Storage

At -20°C for one year. After reconstitution, at 4°C for one month. It can also be aliquotted and stored frozen at -20°C for longer time.

To reorder contact us at:

Antagene, Inc.

Toll Free: 1(866)964-2589

email: Info@antageneinc.com

BACKGROUND

Trk A (Neurotrophic tyrosine kinase receptor A) is the high affinity catalytic receptor for the neurotrophin, Nerve Growth Factor (NGF). Higher affinity binding of NGFR can be achieved by association with higher molecular mass, low-affinity neurotrophin receptors, namely the tropomyosin receptor kinases, TRKA (NTRK1), TRKB (NTRK2), and TRKC (NTRK3). TRKA, TRKB, and TRKC are specific for or 'preferred by' NGF, NTF4 (Neurotrophin-4) and BDNF, and NTF3 (Neurotrophin-3), respectively. NTF3 also binds to TRKA and TRKB, but with significantly lower affinity. The absence of TrkA (NGFR) expression was associated with a strong increase in the Sp3 repressor short isoform(s) and a lack of the Sp3 activator long isoform. Sp3 is a bifunctional transcription factor that has been reported to stimulate or repress the transcription of numerous genes.¹ Indo et al. (1996) concluded that defects in TRKA cause CIPA (Congenital insensitivity to pain with anhidrosis) and that the NGF-TRKA system has a crucial role in the development and function of the nociceptive reception as well as establishment of thermoregulation via sweating in humans. These results also implicate genes encoding other TRK and neurotrophin family members as candidates for developmental defect(s) of the nervous system.²

REFERENCE

1. Lambiase A, Merlo D, Mollinari C, Bonini P, Rinaldi AM, D' Amato M, Micera A, Coassin M, Rama P, Bonini S, Garaci E (November 2005). "Molecular basis for keratoconus: lack of TrkA expression and its transcriptional repression by Sp3". *Proc. Natl. Acad. Sci. U.S.A.* 102 (46): 16795–800.
2. Indo, Y.; Tsuruta, M.; Hayashida, Y.; Karim, M. A.; Ohta, K.; Kawano, T.; Mitsubuchi, H.; Tonoki, H.; Awaya, Y.; Matsuda, I. : Mutations in the TRKA/NGF receptor gene in patients with congenital insensitivity to pain with anhidrosis. *Nature Genet.* 13: 485-488, 1996.