**Polyclonal Anti- Trk-A (Sepharose Bead Conjugate)**

**Catalogue No.** PA1321-S

**Lot No.** 09L01

**Ig type:** rabbit IgG

**Size:** 100µg/vial

**Specificity**

Human, rat, mouse. No cross reactivity with other proteins.

**Recommended application**

*(Immunoprecipitation(IP)*

**Immunogen**

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

**Purification**

Immunogen affinity purified.

**Formulation**

50% slurry in PBS pH 7.2 with 0.01mg $NaN_3a_3$ preservative.

**Storage**

Store at 4˚C for frequent use.

**Description:**

This Antagene antibody is immobilized via covalent binding of primary amino groups to N-hydroxysuccinimide (NHS)-activated sepharose beads. It is useful for immunoprecipitation assays

**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 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.



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