Product Information Sheet

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

Catalogue No. PA1321-M

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.

Purity

Immunogen affinity purified.

Contents

Each vial contains 1mg/ml Magnetic Bead in PBS, pH 7.2, 0.05mg NaN_3.

Storage

Store at 4˚C for frequent use.

Description

This Antagene antibody is immobilized by the covalent reaction of hydrazinonicotinamide-modified antibody with formylbenzamide-modified magnetic beads. It is useful for immunoprecipitation

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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