

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human Tenascin R in direct ELISAs and Western blots. In direct ELISAs and Western blots, less than 1% cross-reactivity with recombinant human Tenascin C is observed.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant human Tenascin R isoform 1 Glu34-Phe1358 Accession # Q92752
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied as a 0.2 µm filtered solution in PBS.

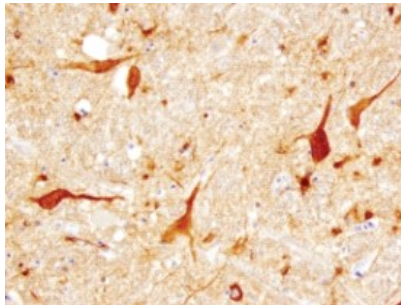
APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. *General Protocols* are available in the *Technical Information* section on our website.

	Recommended Concentration	Sample
Western Blot	0.1 µg/mL	Recombinant Human Tenascin R (Catalog # 3865-TR)
Immunohistochemistry	5-15 µg/mL	See Below

DATA

Immunohistochemistry



Tenascin R in Human Brainstem. Tenascin R was detected in immersion fixed paraffin-embedded sections of human brainstem (medulla) using 1.7 µg/mL Goat Anti-Human Tenascin R Antigen Affinity-purified Polyclonal Antibody (Catalog # AF3865) overnight at 4 °C. Tissue was stained with the Anti-Goat HRP-DAB Cell & Tissue Staining Kit (brown; Catalog # CTS008) and counterstained with hematoxylin (blue). View our protocol for [Chromogenic IHC Staining of Paraffin-embedded Tissue Sections](#).

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.2 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> • 12 months from date of receipt, -20 to -70 °C as supplied. • 1 month, 2 to 8 °C under sterile conditions after reconstitution. • 6 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

Tenascin R (TNR) is an extracellular matrix glycoprotein belonging to the tenascin family of adhesion proteins (1-3). TNR is expressed in the central nervous system by oligodendrocytes and selected inhibitory interneurons. It shows highest expression during the postnatal period of active myelination and promotes neurite outgrowth and synaptic functions (1, 2). It is essential for formation of perineuronal nets, the mesh-like network of extracellular matrix (ECM) molecules that surrounds some neurons (4). The 180 kDa, 1327 amino acid (aa) form of human TNR contains a signal sequence, three heptad repeats that mediate coiled-coil trimer formation, five EGF-like repeats, nine fibronectin type III repeats (FN), and a C-terminal Ca²⁺-binding fibrinogen-related domain. TNR isoform 2 (160 kDa) lacks a portion of FN#6 (aa 773-862) (3). Mature human TNR isoform 1 shows 94%, 94%, 93%, 93%, and 76% aa identity with bovine, mouse, rat, canine, and chicken TNR, respectively. Experiments using recombinant TNR fragments indicate that EGF-like domains are counteradhesive for neurons and microglia and contribute to their migration (1, 5-7). This region interacts with immunoglobulin superfamily molecules including contactin, phosphacan and voltage-gated sodium channel β subunits. However, the fibronectin domains are adhesive for the lectican family of chondroitin sulfate proteoglycans (brevican, aggrecan, versican and neurocan; FN 3-5), contactin (FN 2-3) and sodium channel β subunits (FN 6-8) (6-9). These adhesive interactions can compete with each other, but can also contribute to crosslinking of lecticans and contactin with other ECM molecules to form perineuronal nets (9, 10). Post-translational modification of TNR can differ with time and location (11). Notably, glycosylation may include GalNAc-4-SO₄, O-linked sialylated glycans, "brain-type" neutral N-glycans and the HNK-1 carbohydrate epitope that is thought to be involved in regulation of synaptic plasticity (11, 12).

References:

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