Human Tenascin C Antibody

Monoclonal Rat IgG_{2A} Clone # 391819

Catalog Number: MAB3358

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human Tenascin C in direct ELISAs. No cross-reactivity with recombinant human Tenascin R or recombinant mouse Tenascin C is observed.
Source	Monoclonal Rat IgG_{2A} Clone # 391819
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant human Tenascin C Ser186-Pro625 Accession # NP_002151
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 either lyophilized or as a 0.2 μm filtered solution in PBS.

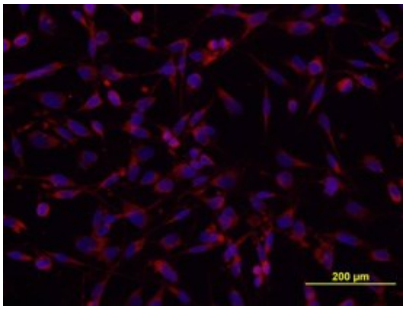
APPLICATIONS

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
Immunocytochemistry	8-25 µg/mL	See Below

DATA

Immunocytochemistry



Tenascin C in U-118-MG Human Cell Line. Tenascin C was detected in immersion fixed U-118-MG human glioblastoma/astrocytoma cell line using Human Tenascin C Monoclonal Antibody (Catalog # MAB3358) at 10 µg/mL for 3 hours at room temperature. Cells were stained red and counterstained with DAPI (blue).

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.5 mg/mL in sterile PBS. For liquid material, refer to CoA for concentration.
Shipping	Lyophilized product is shipped at ambient temperature. Liquid small pack size (-SP) is shipped with polar packs. Upon receipt, store immediately at the temperature recommended below.
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** • 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.

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BACKGROUND

Tenascin C, also known as hexabrachion, cytotactin, neuronectin, GMEM, JI, myotendinous antigen, glioma-associated-extracellular matrix antigen, and GP 150-225, is a member of the Tenascin family of extracellular matrix proteins. It is secreted as a disulfide-linked homohexamer whose subunits can vary in size from approximately 200 kDa to over 300 kDa due to differences in glycosylation (1). Rotary-shadowed electron micrographs of the purified molecule show six strands joined to one another at one end in a globular domain with each arm terminating in a knob-like structure (2, 3). The human Tenascin C monomer is synthesized as a precursor with a 22 amino acid (aa) signal sequence and a 2179 aa mature chain. The mature chain consists of a coiled-coil region (aa 118-145), followed by 15 EGF-like domains, 15 fibronectin type-III domains, and a fibrinogen C-terminal domain. In addition, there are 23 potential sites of N-linked glycosylation. Alternative splicing within the fibronectin type-III repeats produces six isoforms for human Tenascin C. Mature human Tenascin C (isoform 1) shares 84% aa sequence identity with mature mouse Tenascin C. In the developing embryo, Tenascin C is expressed during neural, skeletal, and vascular morphogenesis (1, 2). In the adult, it virtually disappears with continued basal expression detectable only in tendon-associated tissues (1, 2). However, great up-regulation in expression occurs in tissues undergoing remodeling processes seen during wound repair and neovascularization or in pathological states such as inflammation or tumorigenesis (1, 4, 5). Biologically, Tenascin C functions as an adhesion-modulatory extracellular matrix protein (1, 4-8). Specifically, it antagonizes the adhesive effects of fibronectin, and impacts the ability of fibroblasts to deposit and contract the matrix by affecting the morphology and signaling pathways of adherent cells (5-7). Tenascin C acts by blocking syndecan-4 binding at the edges of the wound and by suppressing fibronectin-mediated activation of RhoA and focal adhesion kinase (FAK) (4-8). Tenascin C thus promotes epidermal cell migration and proliferation during wound repair.

References:

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