

11-255-C025

Monoclonal Antibody to GFAP Purified Antibody (0.025 mg)

Clone:	GF-01
lsotype:	Mouse IgG2b
Specificity:	The antibody GF-01 reacts with GFAP, the principal marker of astroglial cells in the central nervous system, which is specifically expressed in satellite cells in peripheral ganglia and in non myelinating Schwann cells in peripheral nerves. The GFAP protein runs on gels at ~55 kDa protein, usually associated with lower Mw bands which are thought to be proteolytic fragments and alternate transcripts from the single gene.
Regulatory Status:	RUO
Immunogen:	Pellet of porcine brain cold-stable proteins after depolymerization of microtubules.
Species Reactivity:	Human, Porcine, Feline (Cat)
Negative Species:	Rat
Application:	Immunohistochemistry (paraffin sections) Recommended dilution:10 µg/ml Positive tissue: human brain (cortex, cerebellum) The antibody GF-01 strongly stains astrocytes in human brain tissue sections but it is essentially negative on mouse and rat tissues. Immunoprecipitation Western Blotting Immunocytochemistry Recommended dilution: 5-10 µg/ml
Purity:	> 95% (by SDS-PAGE)
Purification:	Purified from ascites by protein-A affinity chromatography.
Concentration:	1 mg/ml
Storage Buffer:	Phosphate buffered saline (PBS) with 15 mM sodium azide, approx. pH 7.4
Storage / Stability:	Store at 2-8°C. Do not freeze. Do not use after expiration date stamped on vial label.
Expiration:	See vial label
Lot Number:	See vial label

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Background:

Glial Fibrillary Acidic Protein (GFAP) was discovered by Bignami et al. (1972) as a major fibrous protein of multiple sclerosis plaques. It was subsequently found to be a member of the 10 nm or intermediate filament protein family, specifically the intermediate filament protein family Class III, which also includes peripherin, desmin and vimentin.

GFAP is heavily, and specifically, expressed in astrocytes and certain other astroglia in the central nervous system, in satellite cells in peripheral ganglia, and in non-myelinating Schwann cells in peripheral nerves. In addition, neural stem cells frequently strongly express GFAP. It is also found in the lens epithelium, Kupffer cells of the liver, in some cells in salivary tumors and has been reported in erythrocytes.

Although its function is not fully understood, GFAP protein is probably involved in controlling the shape and movement of astrocytes. The protein probably also plays a significant role in the interactions of astrocytes with other cells, which are required for the formation and maintenance of the insulating layer (myelin) that covers nerve cells. Additionally, GFAP protein may assist in maintaining the protective barrier that allows only certain substances to pass between blood vessels and the brain (blood-brain barrier).

In adults, GFAP levels increase as a result of the proliferation of astrocytes that occurs in a response to a variety of physical, chemical and etiological insults, including Alzheimer's disease, epilepsy and multiple sclerosis.

Antibodies to GFAP are therefore very useful as markers of astrocytic cells and neural stem cells and for distinguishing of neoplasms of astrocytic origin from other neoplasms in the central nervous system.

Finally, Alexander's disease was recently shown to be caused by point mutations in protein coding region of the GFAP gene (Brenner et al., 2001). All forms of Alexander disease are characterized by the presence of Rosenthal fibers, which are GFAP containing cytoplasmic inclusions found in astrocytes.

References: *Bignami A, Eng LF, Dahl D, Uyeda CT: Localization of the glial fibrillary acidic protein in astrocytes by immunofluorescence. Brain Res. 1972 Aug 25;43(2):429-35.

*Brenner M, Johnson AB, Boespflug-Tanguy O, Rodriguez D, Goldman JE, Messing A: Mutations in GFAP, encoding glial fibrillary acidic protein, are associated with Alexander disease. Nat Genet. 2001 Jan;27(1):117-20.

*Lukas Z, Draber P, Bucek J, Draberova E, Viklicky V, Staskova Z: Expression of vimentin and glial fibrillary acidic protein in human developing spinal cord. Histochem J. 1989 Dec;21(12):693-701.

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