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Catalogue# Prot-r-GFAP: Recombinant human Glial Fibrillary Acidic Protein (GFAP)

Background: Glial Fibrillary Acidic Protein (GFAP) was discovered by Amico Bignami and coworkers as a major fibrous protein of multiple sclerosis plaques (2). It was subsequently found to be a member of the 10nm or intermediate filament protein family, specifically the intermediate filament protein family Class III, which also includes peripherin, desmin and vimentin. The most common isotype of the GFAP protein runs on gels at ~55 kDa protein, usually associated with lower molecule weight bands which are proteolytic fragments and less abundant alternate transcripts from the single gene. GFAP is strongly and specifically expressed in astrocytes and certain other astroglia in the central nervous system, in satellite cells in peripheral ganglia, and in nonmyelinating Schwann cells in peripheral nerves. In many damage and disease states GFAP expression is heavily upregulated in astrocytes. In addition neural stem cells frequently strongly express GFAP. Antibodies to GFAP are therefore very useful as markers of astrocytic cells and neural stem cells. In addition many types of brain tumor, presumably derived from astrocytic cells, heavily express GFAP. Finally, Alexander's disease was recently shown to be caused by point mutations in protein coding region of the GFAP gene (3). All forms of Alexander disease are characterized by the presence of Rosenthal fibers, which are GFAP containing cytoplasmic inclusions found in astrocytes. See also reference 4 for some important earlier work. There has been considerable recent interest in GFAP due to it's potential use as a damage and degeneration biomarker, since it can be detected in blood and CSF following various kinds of CNS damage and disease states (5). Our recombinant form of GFAP is an excellent protein standard for such experiments. Since the rodent protein is somewhat different in primary sequence from the human protein, we have also generated a recombinant form of that, PROT-r-GFAP-rat. This form is therefore a more appropriate standard for studies of rodent models of traumatic and neurodegerative states. The HGNC name for this protein is GFAP.

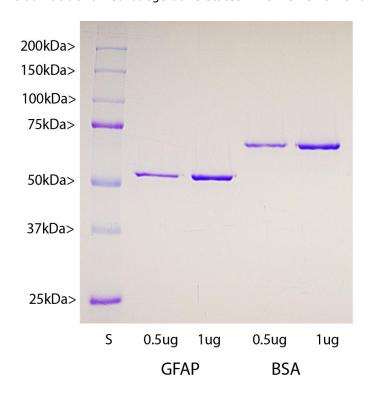


Figure: GFAP based on the human Isotype I sequence was expressed in and purified from *E. coli* using standard methods. Lane S shows protein standards of the indicated molecular weights and in the next two lanes are the indicated amounts of recombinant human GFAP. The next two lanes show similar amounts of BSA protein standard.

References:

- 1. Leung, C. L. and Liem, R. K. H. Isolation of intermediate filaments. <u>Curr. Prot. Cell Biol. 3:Unit 3.23 doi: 10.1002/0471143030.cb0323s31 (2006).</u>
- 2. Bignami A, Eng LF, Dahl D, Uyeda CT. Localization of the glial fibrillary acidic protein in astrocytes by immunofluorescence. Brain Res. 43:429-35 1972.
- 3. Brenner M, Johnson AB, Boespflug-Tanguy O, Rodriguez D, Goldman JE and Messing A. Mutations in GFAP, encoding glial fibrillary acidic protein, are associated with Alexander disease. Nat Genet 27:117-20 2001
- 4. Liem RKH, Yen SH, Salomon GD and Shelanski ML. Intermediate filaments in nervous tissues. <u>J Cell Biol</u> 79:637-745 (1978).
- 5. Schiff L1, Hadker N, Weiser S, Rausch C. A literature review of the feasibility of glial fibrillary acidic protein as a biomarker for stroke and traumatic brain injury. Mol Diagn Ther 16:79-92 (2012).

Limitations: This product is for research use only and is not approved for use in humans or in clinical diagnosis.

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