Glial fibrillary acidic protein

Glial fibrillary acidic protein is a polypeptide, MW =49,000 Daltons. Stains intermediate filaments classically identified in astrocytes/ astrocytic tumors. However, is also typically expressed in ependymomas, oligodendrogliomas (especially in mini gemistocytes and gliofibrillary oligodendrocytes), and some choroid plexus papillomas.

It is encoded by the GFAP gene in humans.

First described in 1971, GFAP is a type III intermediate filament (IF) protein that maps, in humans, to 17q21.

It is expressed by numerous cell types of the central nervous system (CNS) including astrocytes, and ependymal cells.

GFAP has also been found to be expressed in glomeruli and peritubular fibroblasts taken from rat kidneys Leydig cells of the testis in both hamsters and humans, human keratinocytes, human osteocytes and chondrocytes and stellate cells of the pancreas and liver in rats.

It is closely related to its non-epithelial family members, vimentin, desmin, and peripherin, which are all involved in the structure and function of the cell's cytoskeleton. GFAP is thought to help to maintain astrocyte mechanical strength, as well as the shape of cells but its exact function remains poorly understood, despite the number of studies using it as a cell marker. Glial fibrillary acidic protein (GFAP) was named and first isolated and characterized by Lawrence F. Eng in 1969.

Ferrari-Souza et al. assessed 121 individuals across the aging and Alzheimer's disease clinical spectrum with positron emission tomography (PET) brain imaging for Aβ ([18F]AZD4694) and tau ([18F]MK-6240), as well as CSF GFAP and YKL-40 measures. They observed that higher CSF GFAP levels were associated with elevated Aβ-PET but not tau-PET load. By contrast, higher CSF YKL-40 levels were associated with elevated tau-PET but not Aβ-PET burden. Structural equation modeling revealed that CSF GFAP and YKL-40 mediate the effects of Aβ and tau, respectively, on hippocampal atrophy, which was further associated with cognitive impairment. The results suggest the existence of distinct astrocyte biomarker signatures in response to brain Aβ and tau accumulation, which may contribute to the understanding of the complex link between reactive astrogliosis heterogeneity and AD progression [1].

Plasma GFAP in presymptomatic and symptomatic familial Alzheimer's disease: a longitudinal cohort study [2].

Blood ubiquitin C-terminal hydrolase (UCH-L1) and GFAP are increased early after stroke and distinct biomarker-specific release profiles are associated with stroke characteristics and type. Ren et al confirmed the potential of GFAP as a tool for early rule-in of ICH, while UCH-L1 was not clinically useful [3].

Glial fibrillary acidic protein (GFAP) might play an important role in the aggressiveness of Glioblastoma and also contributed to its poor overall survival. A study aimed to test (1) the associations between GFAP single nucleotide polymorphisms (SNPs) and Glioblastoma cells chemoresistance and metastasis, and (2) the molecular mechanism accounting for their effects. Four tagging SNPs of GFAP were initially genotyped in 667 subjects and the significant SNP was further analyzed via online bioinformatics tools. SNP rs11558961 was found to be significantly associated with Glioblastoma susceptibility. It was predicted to influence microRNA(miR)-139 binding to 3'UTR of GFAP gene. In functional experiments, we found that cells transfected with rs11558961 G-allele constructs had lower baseline luciferase activities and were more responsive to miR-139 changes, compared to C-allele constructs. Moreover, rs11558961 C>G variant reduced the chemoresistance of Glioblastoma cells and migration capability. In conclusion, rs11558961 might influence the chemoresistance and progression of Glioblastoma cells via promoting the binding of miR-139, ultimately decrease the susceptibility of Glioblastoma. This investigation will shed light on the optimizing for clinical trial design and individualizing of therapeutic plans [4].

Glial fibrillary acidic protein in Traumatic brain injury

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