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Gene Section
Review

FABP7 (fatty acid binding protein 7, brain)

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Abstract

Review on FABP7, with data on DNA/RNA, on the protein encoded and where the gene is implicated.

Identity

Other names: B-FABP, BLBP, FABPB, MRG

HGNC (Hugo): FABP7

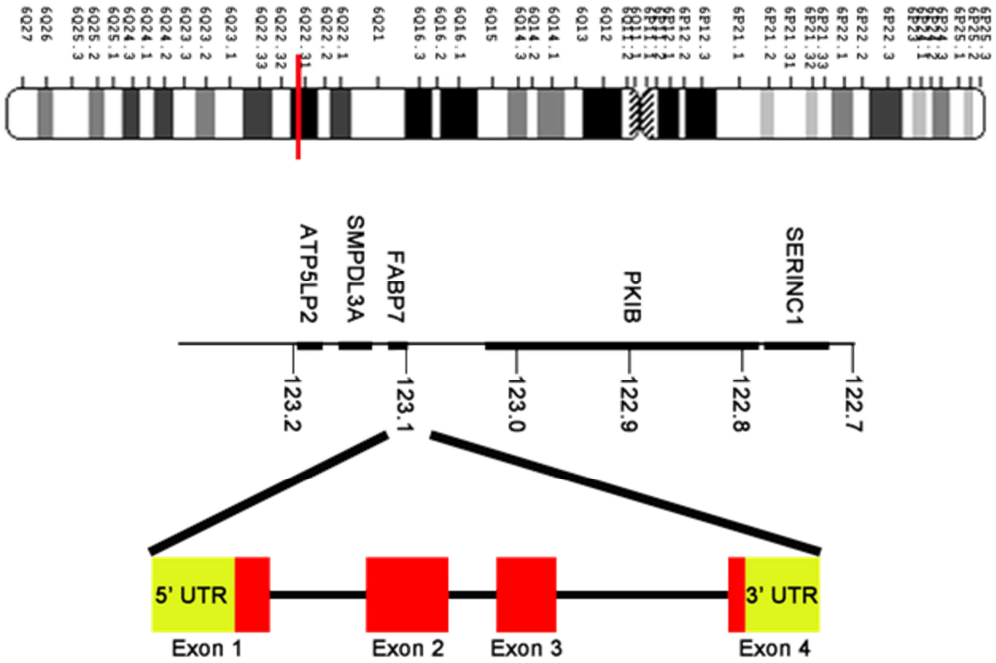
Location: 6q22.31

Local order: PKIB → **FABP7** → SMPDL3A.

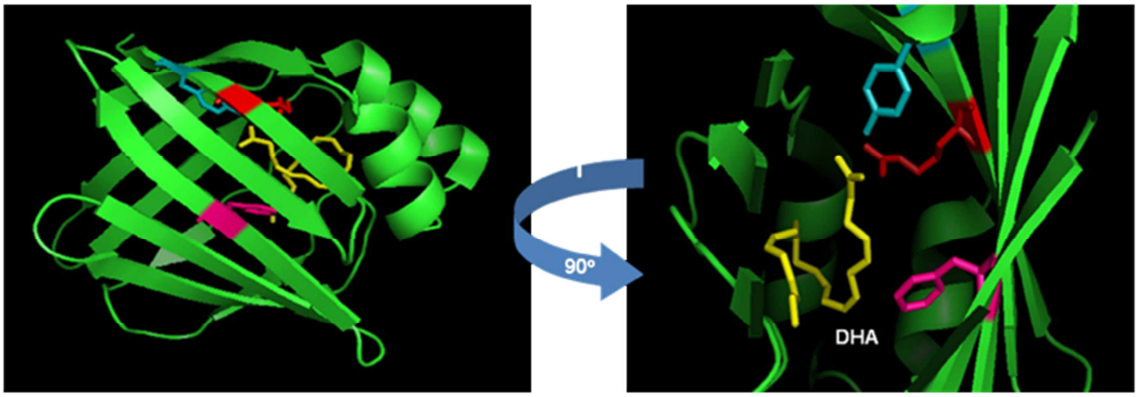
DNA/RNA

Description

The FABP7 gene is 4,5 kb long and contains 4 exons, all of which contain coding sequences.
The following FABP7 SNPs have been validated: 7 in the 3' UTR, 6 in the 5' UTR, 5 missense and 4 SNPs in the coding region that don't alter the amino acid code (dbSNP).



FABP7 gene. The FABP7 gene is located on chromosome 6 in the region of q22-q23 on the positive strand. Neighboring genes are indicated.



Crystal structure of FABP7 bound to DHA. The structure of FABP7 is similar to that of other FABPs and consists of two N-terminal α-helices attached to a β-barrel motif (left). Three amino acids are predicted to be important for fatty acid binding: F104 (fuchsia), arginine 126 (red) and Y128 (teal) based on the structure of FABP7 bound to DHA. DHA is shown in yellow (right). Structural data were obtained from the Protein Data Bank (PDB ID: 1FE3) (Balendiran et al., 2000) and rendered using PyMOL (Beaulieu, 2012).

Based on EST data, FABP7 RNA is most highly expressed in the fetus, followed by adult brain, eye, connective tissue, bone, heart, kidney, mammary gland, skin, uterus, lung and testis.
Transcription regulators: Members of the nuclear factor I (NFI) family regulate the transcription of the FABP7 gene (Bisgrove et al., 2000; Brun et al., 2009).
The phosphorylation state of NFI determines its regulatory activity, with FABP7 transcription up-regulated by hypophosphorylated NFI (Bisgrove et al., 2000; Brun et al., 2013). Other transcription factors implicated in the regulation of FABP7 include Notch (Anthony et al., 2005), PAX6 (Arai et al., 2005; Numayama-Tsuruta et al., 2010; Liu et al., 2012b), and POU-domain protein PBX-1 (Josephson et al., 1998).
Furthermore, ligands of peroxisome proliferator-activated receptors (PPARs) such as clofibrate and omega-3 docosahexaenoic acid (DHA) have been shown to up-regulate FABP7 expression (Nasrollahzadeh et al., 2008; Venkatachalam et al., 2012).
Post-transcriptional regulation: The 3' untranslated region of FABP7 contains phylogenetically conserved cytoplasmic polyadenylation elements (CPE) which have been implicated in the trafficking and localized translation of FABP7 at perisynaptic processes of astrocytic cells (Gerstner et al., 2012).

Pseudogene

A predicted FABP7 pseudogene is located on chromosome 1 (NCBI nucleotide database NG_029025.1). There are two inferred human FABP7 pseudogenes listed in the Rat Genome Database (http://rgd.mcw.edu/rgdweb/report/gene/main.html?id=5132511 on chromosome 1 and http://www.rgd.mcw.edu/rgdweb/report/gene/main.html?id=6481032 on chromosome 2).

Protein

Description

FABP7 is a member of the intracellular lipid-binding protein family. FABP7 is a 132 amino acid polypeptide with an estimated molecular mass of 15 kDa.
It has a beta-clam structure made up of ten anti-parallel beta sheets capped by two alpha helices. Fatty acid ligands reside inside the beta-clam structure.

Expression

FABP7 is expressed in radial glial cells during brain development (Feng et al., 1994). FABP7 persists in specific regions of the mature mouse brain, including glia limitans, in radial glial cells of the hippocampal dentate gyrus and Bergman glial cells (Kurtz et al., 1994). FABP7 is also expressed in glial cells of the peripheral nervous system, and ensheathing cells of the olfactory nerve (Kurtz et al., 1994).

Localisation

The FABP7 protein is found in both the cytoplasm and nucleus of normal radial glial cells (Feng et al., 1994) and tumor cells (Liang et al., 2006; Slipicevic et al., 2008). FABP7 is also found in perisynaptic processes of astrocytes with localized translation of FABP7 at these sites (Gerstner et al., 2012).

Function

Recombinant human FABP7 exhibits the highest affinity for the polyunsaturated omega-3 fatty acids α-linolenic acid, eicosapentaenoic acid, docosahexaenoic acid, and for monounsaturated omega-9 oleic acid (Kd from 28 to 53 nM) and moderate affinity for the polyunsaturated omega-6 fatty acids, linoleic acid and arachidonic acid (AA) (Kd from 115 to 206 nM) (Balendiran et al., 2000).

FABP7 has low binding affinity for saturated long chain fatty acids. Human FABP7 enhances DHA trafficking to the nucleus (Mita et al., 2010).
FABP7 is required for the establishment of the radial glial fiber system along which neurons migrate in order to reach their correct destination in the developing brain (Feng et al., 1994). FABP7 is also required for the maintenance of neuroepithelial cells in rat cortex (Arai et al., 2005). FABP7 knock-out mice have a structurally normal brain; however, the mice show enhanced anxiety and increased fear memory, as well as decreased DHA in neonatal brain and increased AA in adult brain amygdala (Owada et al., 2006).

Homology

Human FABP7 amino acid sequence is 86,4% identical to mouse FABP7, 90,9% identical to chicken FABP7, 82,6% identical to zebrafish FABP7a and 78% identical to zebrafish FABP7b. Human FABP7 shows variable sequence identity with the other FABP paralogues, with the lowest identity to FABP1 (27,6%) and highest identity to FABP3 (65,9%).

Mutations

Note
With the exception of SNPs, no mutations in the FABP7 gene have been reported.

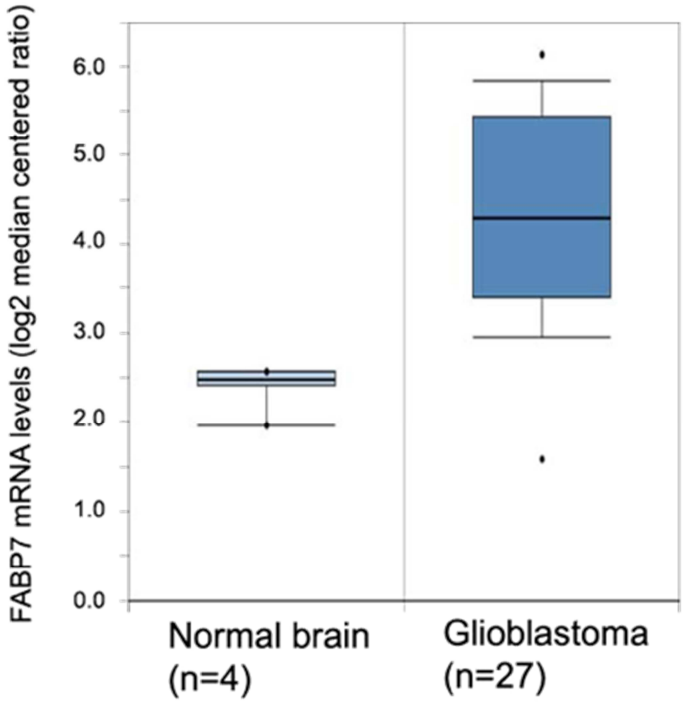
Implicated in

Malignant glioma (grades III and IV astrocytoma) / glioblastoma multiforme (grade IV astrocytoma)

Note
FABP7 was first reported to be expressed in malignant glioma cell lines and malignant glioma tumour tissue in 1998 (Godbout et al., 1998). Liang et al. (2005) used gene expression profiling to demonstrate that FABP7 RNA levels were elevated in glioblastoma tumours compared to normal brain. These authors showed that elevated levels of nuclear FABP7 protein were associated with decreased survival in patients with glioblastoma multiforme, particularly in younger patients. Subsequent analysis of 123 glioblastomas by Kaloshi et al. (2007) revealed a correlation between nuclear FABP7, EGFR amplification and more invasive tumours. De Rosa et al. (2012) also showed a correlation between elevated FABP7 levels and decreased survival in patients with glioblastoma multiforme.
Transfection of a FABP7 expression construct into the SF767 malignant glioma cell line results in increased cell migration (Liang et al., 2005). A role for FABP7 in malignant glioma cell migration was confirmed by Mita et al. (2007) who used human U87 malignant glioma cell lines stably transfected with a FABP7 expression construct to demonstrate a correlation between FABP7 expression and increased cell migration.
In agreement with a role for FABP7 in migration and infiltration, FABP7 was found to be preferentially expressed at sites of infiltration and surrounding blood vessels in glioblastoma multiforme (Mita et al., 2007).
Growth of malignant glioma cell lines in the presence of polyunsaturated fatty acids omega-3 DHA and omega-6 AA indicates that the ratio of AA:DHA affects migration in FABP7-positive cells, with a higher DHA:AA ratio resulting in decreased migration (Mita et al., 2010). These results suggest that glioblastoma tumour growth and infiltration may be controlled by increasing levels of DHA in tumour tissue (Elsherbiny et al., 2013).
Neurospheres derived from glioblastoma multiforme express high levels of FABP7, suggesting the presence of FABP7-positive neural stem-like cells in glioblastoma (De Rosa et al., 2012).
In keeping with this possibility, FABP7 is preferentially expressed in the subset of glioblastoma tumour cells that express the neural stem cell marker CD133 (Liu et al., 2009). Knock-down of FABP7 in glioblastoma-derived neurosphere cultures results in decreased cell migration and reduced proliferation (De Rosa et al., 2012).
The FABP7 promoter has been shown to be hypomethylated in glioblastoma tumours compared to normal brain (Etcheverry et al., 2010).

Breast cancer

Note
MRG (mammary-derived growth inhibitor-related gene), later shown to be identical to FABP7 (Hohoff and Spener, 1998), was reported to be expressed in normal and benign breast tissue but only rarely in breast cancer (1 of 10 infiltrative breast cancers and 2 of 12 ductal carcinomas in situ) (Shi et al., 1997). Transfection of a MRG expression construct into the MDA-MB-231 breast cancer cell line suppressed cell proliferation and tumour growth in an orthotopic mouse model (Shi et al., 1997). Subsequent work showed that MRG over-expression induced differentiation in human breast cancer cells and that treatment of breast cancer cells with DHA causes MRG-dependent growth inhibition (Wang et al., 2000).



FABP7 mRNA levels are upregulated in human glioblastoma. Comparison of FABP7 mRNA levels in normal human brain versus glioblastoma tissues. Database obtained from Oncomine website (www.oncomine.org; Bredel Brain 2).

Preferential expression of FABP7 in estrogen receptor-negative breast cancer compared to estrogen receptor-positive breast cancer has been reported by four separate groups (Tang et al., 2010; Zhang et al., 2010; Graham et al., 2011; Liu et al., 2012a). In an analysis of 176 primary breast cancers, Liu et al. (2012) found a correlation between elevated FABP7 levels and poor prognosis. These authors further showed that depletion of FABP7 in FABP7-positive/estrogen-negative MDA-MB-435S, reduced cell growth and sensitized the cells to growth inhibition by DHA. In addition, FABP7 was found to mediate DHA-induced retinoid-X-receptor beta (RXRβ) activation in triple-negative BT-20 breast cancer cells as well as MDA-MB-435S cells. In a study of 899 invasive breast cancer cases, Zhang et al. (2010) showed that basal breast cancers (estrogen/progesterone receptor-negative, HER2-negative) that were FABP7-positive had significantly better outcomes than basal breast cancers that were FABP7-negative. Analysis of the subcellular localization of FABP7 in 1249 unselected and 245 estrogen receptor-negative invasive breast cancers revealed both nuclear and cytoplasmic staining patterns, with nuclear FABP7 associated with a high histological grade, stage, mitotic frequency, as well as basal and triple-negative status (Alshareeda et al., 2012). Within the basal category, elevated levels of nuclear FABP7 were associated with longer disease-free survival. In light of the proposed roles for nuclear FABPs in making their fatty acid ligands available to nuclear receptors such as PPARs, understanding the roles of cytoplasmic and nuclear FABP7 will help elucidate its biological functions in breast cancer.

Renal cell carcinoma

Note

FABP7 RNA and protein are up-regulated in renal cell carcinoma compared to normal kidney tissue (Seliger et al., 2005; Teratani et al., 2007; Domoto et al., 2007). Analysis of a tissue microarray containing 272 renal cell carcinomas showed significantly lower levels of FABP7 in grades 3 and 4 compared to grades 1 and 2 renal cell carcinomas (Tölle et al., 2009). No correlation was found between patient survival and FABP7 staining intensity. In agreement with malignant glioma experiments, knock-down of FABP7 in human kidney carcinoma cells resulted in decreased cell migration (Tölle et al., 2011).

The regulation of FABP7 in renal cell carcinoma has been addressed by analysing the FABP7 promoter (Takaoka et al., 2011). This analysis indicates that BRN2 (POU3F2) and nuclear factor I (NFI) may be regulating the expression of FABP7 in renal cell carcinoma.

Melanoma

Note

FABP7 been reported to be both down-regulated in melanoma compared to benign nevi (de Wit et al., 2005) and widely expressed in melanoma (Goto et

al., 2010). FABP7 immunostaining of 149 primary melanomas revealed an association between FABP7 expression and tumour thickness, as well as a trend towards increased relapse-free survival for patients who had tumors with low cytoplasmic FABP7 levels (Slipicevic et al., 2008). Knock-down of FABP7 in human melanoma cells resulted in decreased cell proliferation and invasion. There was no association between the nuclear expression of FABP7 and patient survival in this study (Slipicevic et al., 2008).
Gene expression analysis of 87 primary melanomas and 68 metastatic melanoma, combined with immunohistochemical analysis of 37 paired primary and metastatic melanomas, showed significantly decreased FABP7 levels in metastatic melanoma compared to primary tumor tissue (Goto et al., 2010). In metastatic melanoma, FABP7 mRNA expression was associated with decreased relapse-free survival and overall survival (Goto et al., 2010). Loss of heterozygosity analysis using microsatellite markers specific to the FABP7 gene revealed that 10 of 20 metastatic melanomas (and 0 of 14 primary melanomas) had undergone loss of one FABP7 allele, leading the authors to postulate that genomic instability that favors loss of FABP7 expression may lead to better prognosis.

Neurological disorders

Note

FABP7 is overexpressed in the brains of Down syndrome patients and has been postulated to contribute to Down syndrome-associated neurological disorders (Sánchez-Font et al., 2003).
Pelsers et al. (2004) measured FABP7 levels in various parts of the adult human brain, with a range of 0,8 µg/g wet weight in the striatum and 3,1 µg/g in the frontal lobe. Measurement of FABP7 and FABP3 levels in the serum of patients with minor brain injuries identified both these FABPs as more sensitive at detecting brain injury than markers currently in use for this purpose. Similarly, serum FABP7 and FABP3 served as markers for individuals who had undergone ischaemic stroke (Wunderlich et al., 2005). FABP7 levels were also elevated in the serum of patients with neurodegenerative diseases such as Alzheimer's disease, Parkinson's disease and other cognitive disorders. Although elevated levels of FABP7 were found in only one-third of patients, FABP7 is still the most discriminatory serum marker identified to date (Teunissen et al., 2011). The authors propose that elevated levels of FABP7 in serum may reflect damage to the central nervous system.
FABP7-deficient mice have characteristics associated with schizophrenia such as decreased prepulse inhibition and shortened startle response latency (Watanabe et al., 2007). FABP7 RNA levels in the postmortem brains of male patients with schizophrenia were up-regulated in the dorsolateral prefrontal cortex. Furthermore, single nucleotide polymorphism (SNP) analysis revealed an association between missense polymorphism Thr61Met 182C>T) and male patients with schizophrenia (Watanabe et al., 2007).
In a separate study, FABP7 SNPs F704, F705 and F709 showed nominal association with bipolar disorder (Iwayama et al., 2010). Analysis of 6 FABP7 variants identified by polymorphic screen failed to identify any associations with autism or schizophrenia in 285 autistic and 1060 schizophrenic patients of Japanese descent (Maekawa et al., 2010).

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