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Gene Section
Review

FKBP8 (FK506 binding protein 8, 38kDa)

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Identity

Other names: FKBP38, FKBPr38

HGNC (Hugo): FKBP8

Location: 19p13.11

DNA/RNA

Description

FKBP8 gene is located on chromosome 19 at 19p13.1.

FKBP8 gene ranges from 18642563 to 18654886 on reverse strand with a total length of 12323 bp (Thierry-Mieg and Thierry-Mieg 2006).
FKBP8 gene contains 22 distinct introns (18gt-ag, 4gc-ag) (Thierry-Mieg and Thierry-Mieg 2006).

Transcription

Transcription of FKBP8 gene produces 18 different mRNAs.
Most of these forms are produced by alternatively splicing, while one is an unspliced form (Thierry-Mieg and Thierry-Mieg 2006).

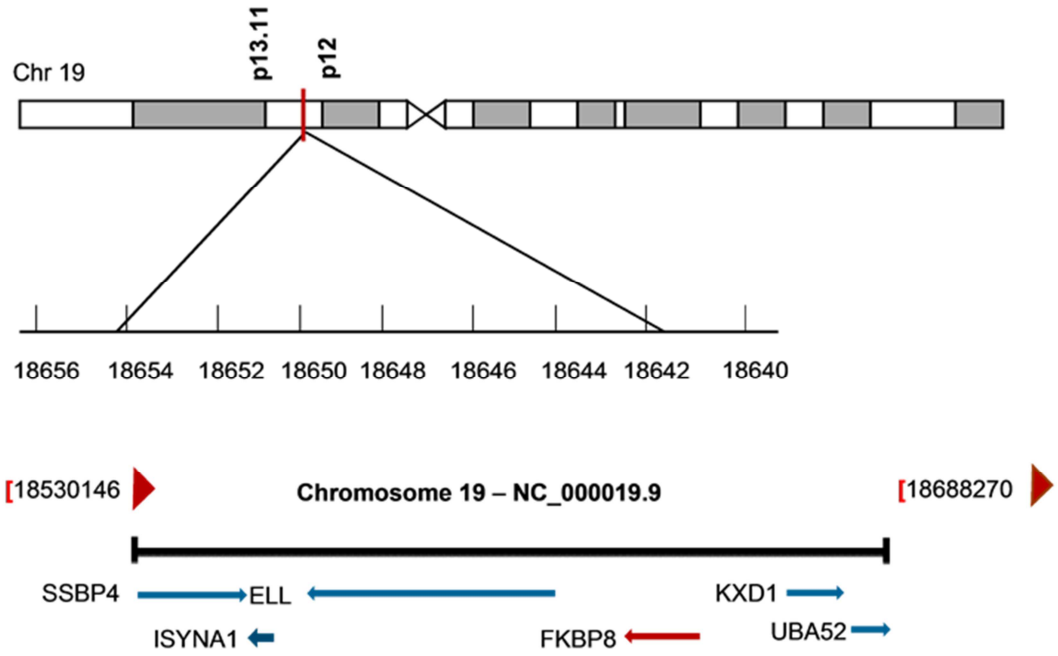

Figure 1: Schematic diagram of FKBP8 location on chromosome 19. Chromosome 19 is represented with the banding pattern. FKBP8 is located at 19p13.1 and ranges from 18642563 to 18654886 bp on reverse strand. The region surrounding FKBP8 gene is enlarged. Genes are represented by arrows in the direction of transcription. Distances shown are in kilobases.

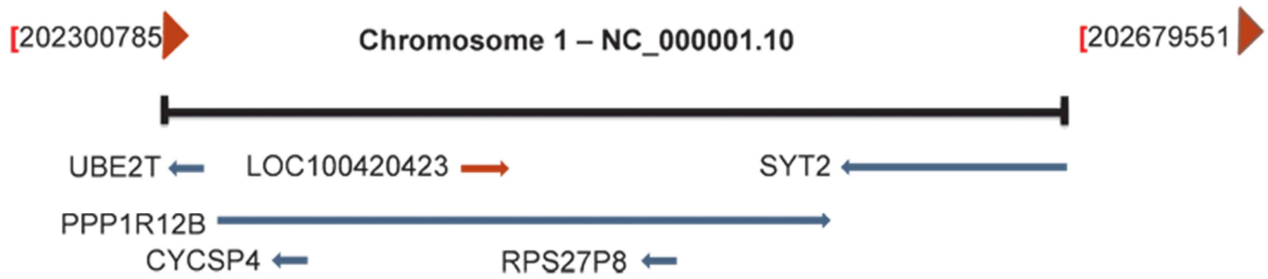

Figure 2: Schematic diagram of FKBP8 pseudogene location. Pseudo gene (100420423) is mapped on chromosome 1q32.1. It extends in the intronic region between phosphatase 1 regulatory subunit 12B exons (10 and 11). Pseudo gene ranges from 202407524-202408693 bp from pter.

Figure 3: FKBP8 structural organization.

Protein

Note

Protein name: FKBP8, FKBP38, Peptidyl-prolyl cis-trans isomerase.

Description

FKBP8 is a non-canonical FKBP family member, which shows Ca^{+2}-calmodulin dependent peptidylprolylisomerase (PPIase) activity.

Unlike other FKBP family members, FKBP8 binds to FK506 only upon activation by Ca^{+2}-saturated calmodulin. FKBP8 contains a glutamate rich domain (ERD), FKBP domain, tetratricopeptide repeat region (TPR domain) interspersed by a consensus leucine-zipper (LZ) repeat region followed by calmodulin and a transmembrane domain (Figure 3).

FKBP8 through its multiple domains interact with other leucine-zipper or coiled-coil proteins and forms multimers (Lam et al., 1995).

Ca^{+2}-saturated calmodulin positively regulates the PPIase activity of FKBP8 (Edlich et al., 2005). Both C-terminal regions of calmodulin and FKBP8 interact with each other; the N-terminal regions of both proteins also interact with each other, while calcium interacts with negatively charged aspartate residues in β4-α1 loop (L147-I153 residues).

These interactions could modulate the enzymatic activity of FKBP8 (Edlich et al., 2007; Maestre-Martinez et al., 2011).

Interaction of FKBP8 with its substrate proteins such as Bcl-2 and Hsp90 is primarily dependent on formation of Ca^{+2}-calmodulin/FKBP8 complex.

Expression

FKBP8 is widely expressed with varying levels of distribution in different tissues.

FKBP8 is highly expressed in the brain tissues; moderately in heart, lung, skeletal muscle, pancreas, while it is expressed marginally in placenta and liver tissues (Bulgakov et al., 2004; Kang et al., 2005a).

Localisation

FKBP8 anchors mitochondrial and ER membranes with its trans-membrane domain and is exposed to the cytosol.

Function

FKBP8 plays important roles in cellular process involving protein folding and trafficking, apoptosis, proteasomal degradation, neural tube patterning, viral replication, metastasis, invasion and neurodegenerative processes (see below for details).

Homology

FKBP8 gene is well conserved across species ranging from primates to non-primates including invertebrates.

Mutations

Somatic

Two types of somatic mutations - A222G mis-sense mutation and V118V silent mutations have been characterized in ovarian carcinoma cell lines (Bamford et al., 2004; Forbes et al., 2010).

Implicated in

Cell size regulation

Note

FKBP8 plays an important role in tuberous sclerosis (TSC) mediated autosomal disorders. Human TSC1 and TSC2 genes regulate the cell size reduction while the dominant TSC2 mutant increases the cell size. Microarray studies revealed that ectopic overexpression of TSC1 or TSC2 (the wild type) induced high levels of FKBP8 while overexpression of TSC1 mutant 127 or TSC2ΔRL were unable to trigger increase in FKBP8 levels. Selective inhibition of FKBP8 by specific antisense oligonucleotide treatment showed the loss of TSC gene ability to control cell size (Figure 4A) (Rosner et al., 2003).

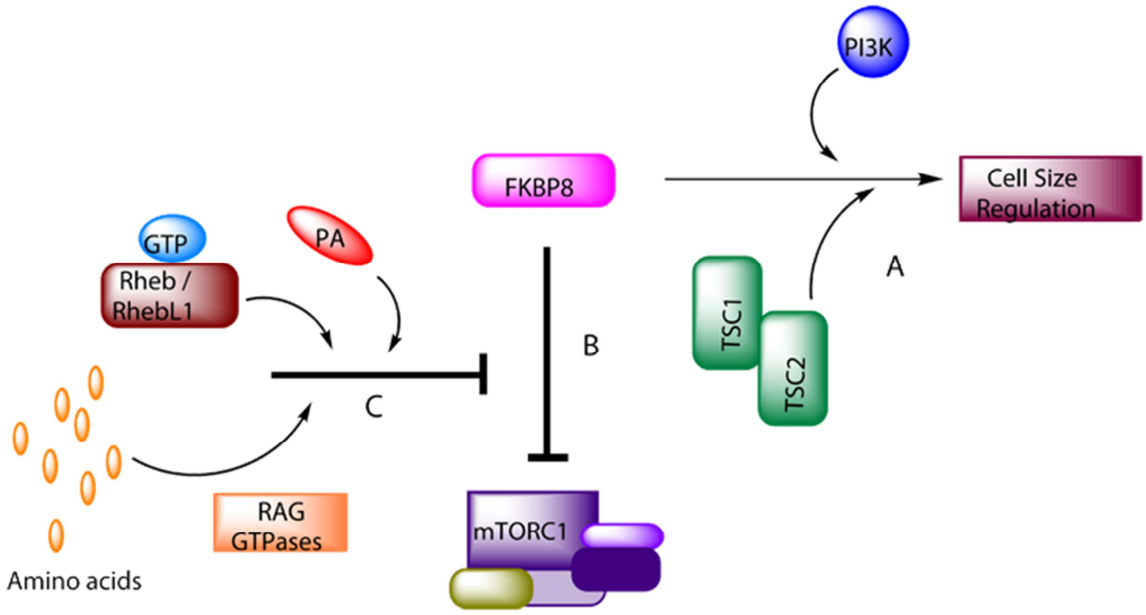

Figure 4: Importance of FKBP8 in cell size regulation and mTOR signaling. (A) FKBP8 together with PI3K maintains the integrity of TSC-mediated regulation of cell size. **(B)** FKBP8 functions as an endogenous mTORC1 inhibitor. **(C)** Excess amino acids, phosphatidic acid and GTP bound Rheb/RhebL1 protein complex(-es) antagonize FKBP8-mediated mTOR inhibition.

mTOR signaling

Note

The mammalian target of rapamycin (mTOR) signaling is implicated in multiple processes such as cancer, mitochondrial biogenesis, hypoxia signaling, and cell cycle progression. FKBP8 functions as an endogenous inhibitor of mTOR by inhibiting mTORC1 activity both in vitro and in vivo (Bai et al., 2007; Wang et al., 2008; Uhlenbrock et al., 2009) (Figure 4B).

FKBP8 has been shown to inhibit mTOR activity in the presence of insulin only under an amino acid-deprived state. However, in the presence of excess amino acids, FKBP8 fails to inhibit mTOR. An excess of amino acids serves to stimulate the FAT domain of mTOR via a Rag-dependent mechanism and thereby antagonizes FKBP8-mediated mTOR inhibition (Dunlop et al., 2009). Furthermore, protein complexes like the GTP-bound Rheb/RhebL1 complex and signaling molecules such as excess amino acids and phosphatidic acid can modulate this FKBP8-mTOR interaction (Figure 4C) (Yoon et al., 2011).

Apoptosis

Note

FKBP8 plays pivotal roles in modulating apoptosis by protecting Bcl-2 from caspase dependant degradation. FKBP8 by interacting with the flexible loop domain of Bcl-2 stabilizes Bcl-2 levels and prevents apoptosis (Kang et al., 2008). Thus FKBP8 enhances cell survival, promotes tumorigenesis and contributes to chemoresistance (Kang et al., 2005b; Kang et al., 2008; Choi et al., 2010; Choi and Yoon, 2011).

On the other hand, Presenilins (PS1/PS2) and Hsp90 antagonize the chaperone effects of FKBP8. Presenilins blocks FKBP8-Bcl-2 interactions in a γ-secretase independent manner and thereby increase the susceptibility to apoptosis by promoting Bcl-2 degradation (Wang et al., 2005).

Hsp90 negatively regulates FKBP8/Ca^{+2}-calmodulin complex by preventing its interaction with Bcl-2 and controls programmed cell death of neuroblastoma cells (Erdmann et al., 2007) (Figure 5A).

Proteasomal degradation

Note

FKBP8 influences proteasomal degradation by directly interacting with almost all the subunits of 26S proteasome via its TPR domain (Nakagawa et al., 2007) (Figure 5C). FKBP8 could probably serve to modulate proteasomal degradation of its substrate proteins like phosphatase of regenerating liver 3 (PRL-3) and prolylhydroxylase-2. FKBP8 interacts with PRL-3 and modulates the stability of PRL-3 by promoting the degradation of PRL-3 via proteasomal pathway and thus suppresses PRL-3 mediated p53 activity and cell proliferation (Choi et al., 2011).

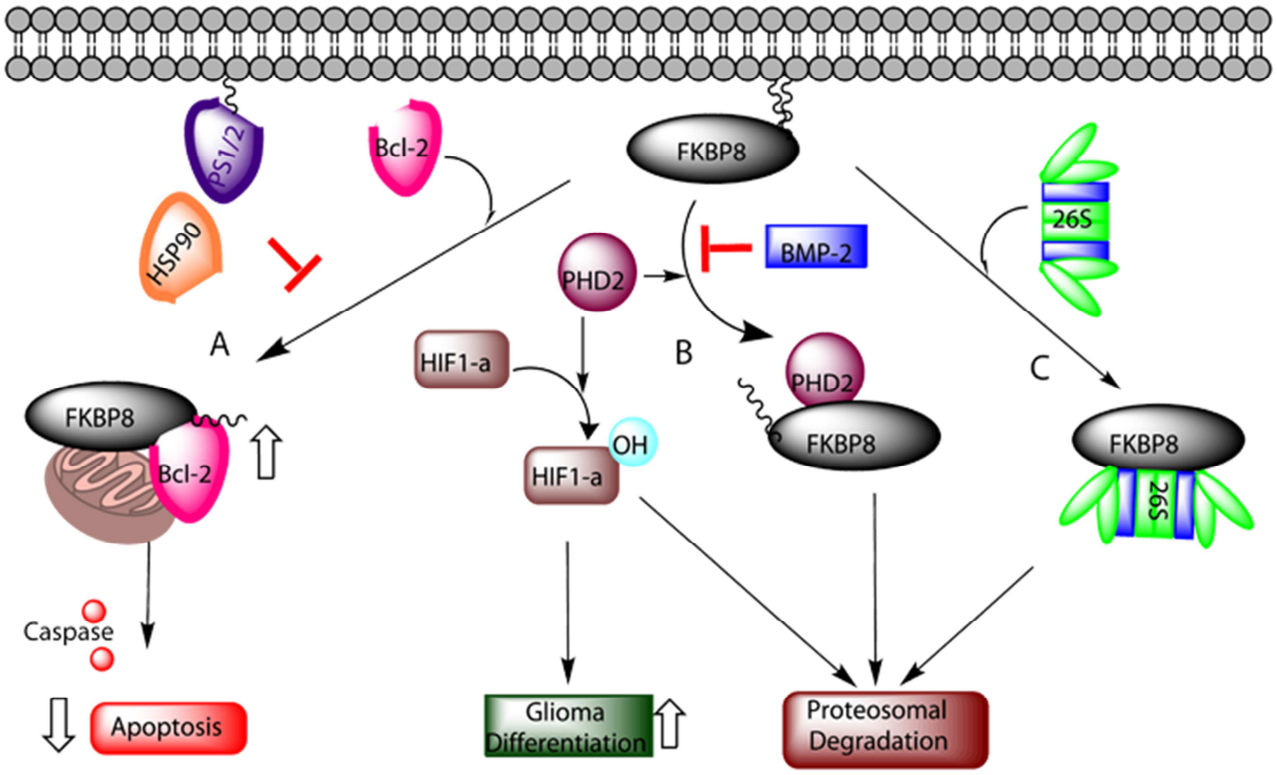

Figure 5: FKBP8 influences the stability of substrate proteins. (A) FKBP8 protects Bcl-2 by targeting it to mitochondria and preventing its caspase mediated degradation. **(B)** FKBP8 promotes proteasomal degradation of PHD2 and thereby enhances stability and transcriptional activity of HIF-1a. Growth factors like BMP-2 counteract this FKBP8-mediated proteasomal degradation of PHD2 thereby decreasing HIF-1a levels and committing the differentiation of glioma cells. **(C)** FKBP8 regulates proteasomal degradation by interacting with the 26S proteasome.

FKBP8 with its glutamate region domain (ERD) specifically binds to prolyl-4-hydroxylase domain containing protein PHD-2. The membrane anchor targets FKBP8-PHD-2 complex to mitochondrial and ER membranes and promotes its proteasomal degradation and maintains in vivo levels of PHD-2 (Barth et al., 2009). FKBP8 mediated modulation of PHD-2-HIF-1a interaction plays key roles in regulating hypoxia responses. Depletion of FKBP8 prolongs PHD-2 stability; elevates its hydroxylation activity, leading to degradation and reduction of HIF-1a transcriptional activity (Figure 5B) (Barth et al., 2007).

Given that hypoxia plays a key role in the development of gliomas like glioblastoma multiforme (GBM), its modulation may present an alternative approach in the therapeutic intervention of gliomas. For example, growth factors like BMP-2 has been recently been used for GBM treatment. BMP-2, by lowering HIF-1a levels (via FKBP8 inhibition) and activating mTOR signaling, alters the activity of succinic dehydrogenase. This, in turn, prevents the proliferation of glioma cells and commits the cells to differentiation (Figure 5B) (Pistollato et al., 2009).

Negative regulator of Shh signaling and development of neural tubes

Note

Sonic Hedgehog (Shh) signaling regulates neural patterning of central nervous system by altering the genes that mediate dorso-lateral and ventral fates (Briscoe and Ericson, 2001). FKBP8 gene knock out studies have revealed that it functions as a negative regulator of Shh signaling. Hedgehog signal transduction occurs mainly by modulating the activities of GLI2 transcriptional factors. FKBP8 primarily acts in a cell autonomous fashion and modulates hedgehog pathway independent of upstream activator smoothened but dependent on kinesin-2 motor subunit kif3a (which mediates in intra flagellar transport (IFT) and cilia assembly) (Figure 6). FKBP8 depletion modifies the neural progenitors-BMP signaling causing non-autonomous effects on neural patterning (Bulgakov et al., 2004; Cho et al., 2008).

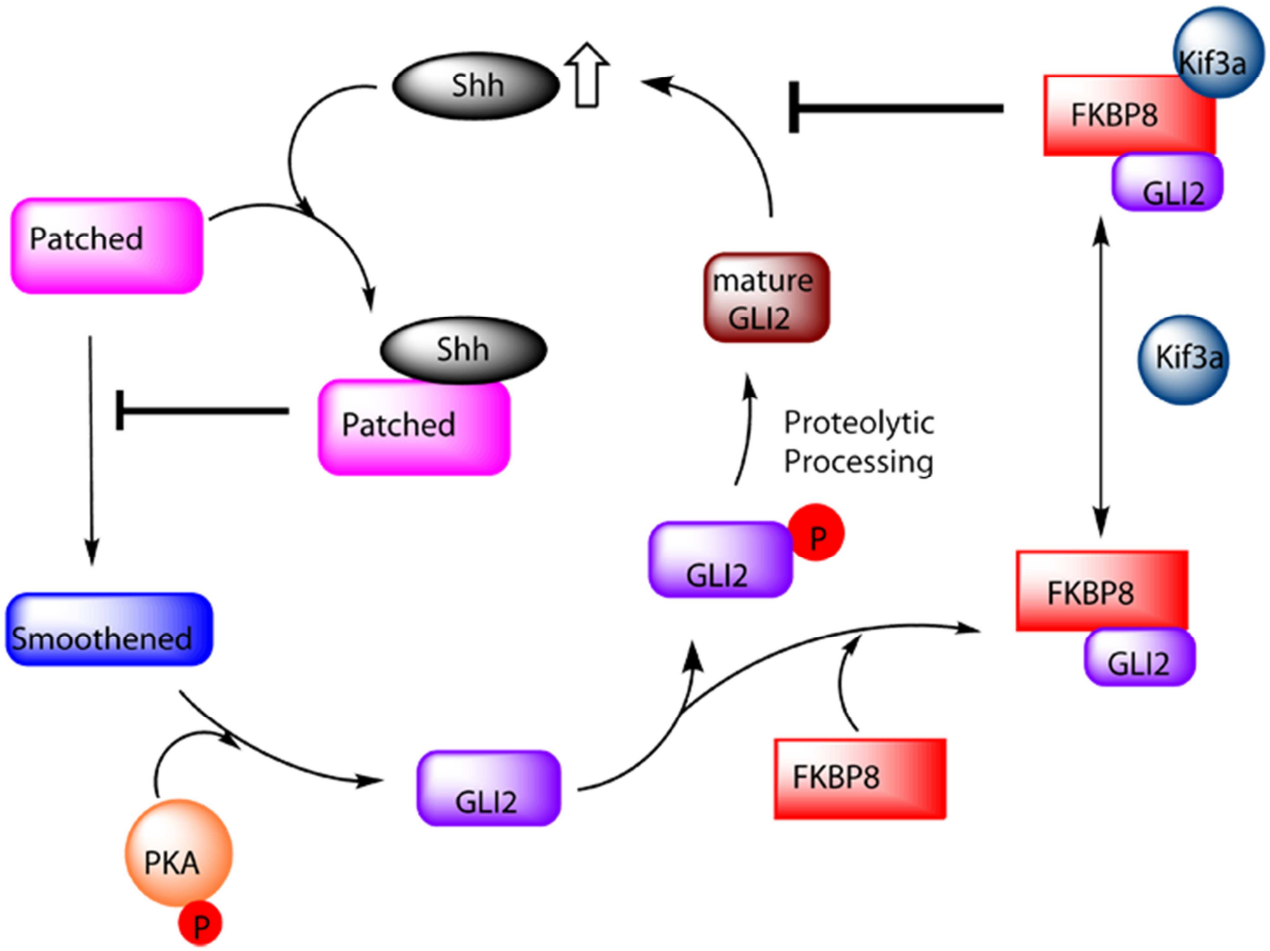

Figure 6: FKBP8 antagonizes Shh signaling independent of ligand (Shh) binding to Patched. FKBP8 binds to GLI2 transcription factors and in conjunction with Kif3a inhibits the hedgehog secretions by preventing proteolytic processing of GLI2 transcription factor.

Development and neuroprotective roles

Note

FKBP8 plays a critical role in the development of various organs. FKBP8$^{(-/-)}$ mice shows several developmental defects that includes improper eye development, spina bifida, skeletal defects, defective dorsal root ganglion and disorganized neural epithelium. The extension of nerve fibers in spinal cord is also abnormal in FKBP8 null embryos. Shirane et al. have shown that abnormal nerve extension in FKBP8$^{(-/-)}$ mice is mediated by the hyperphosphorylation of Protrudin. Thus, it is likely that FKBP8 plays an important role in regulating protrudin-dependent neurite extension (Shirane et al., 2008; Saita et al., 2009).

Neurodegenerative disorders

Note

Recent studies have highlighted the role of FKBP8 in modulating neurodegenerative 'amyloidoses' disorders like Parkinson's disease. Stable overexpression of FKBP8 has been shown to enhance the aggregation of α-synuclein and cell death in neuronal cell culture model suggesting its probable role in Parkinson disease (Deleersnijder et al., 2011; Chattopadhaya et al., 2011). Selective inhibition of FKBP8 by specific inhibitor N-(N', N'-dimethylcarboxamidomethyl) cycloheximide (DM-CHX) has shown promise in achieving neuronal protection in a rat model of transient focal cerebral ischemia. DM-CHX not only protected neurons from ischemic challenge but also induced neural stem cell proliferation and neuronal differentiation suggesting potential role of FKBP8 in neuronal cells (Edlich et al., 2006).

Chaperonic role in biogenesis of membrane proteins

Note

FKBP8 plays key roles in modulating the biogenesis of membrane proteins such as HERG, CFTR. FKBP8 functions as a co-chaperone assisting maturation and trafficking of human ether-a-go-go-related gene (HERG), a voltage dependent potassium channel. Mutations in HERG, for example F805C, causes long QT syndrome which is characterized by a prolonged QT interval and increased susceptibility to cardiac

arrhythmia. FKBP8 knock down shows reduction in HERG trafficking, while its overexpression rescues the mutant F805C HERG trafficking (Walker et al., 2007). Similarly, mutations such as ΔF508 in cystic fibrosis transmembrane conductance regulator (CFTR, a chloride ion channel) alters the biogenesis, trafficking or stability of CFTR and disrupts the functioning of chloride ion channel. FKBP8 plays a rate limiting role as a co-chaperone on maturation and biogenesis of CFTR. FKBP8 by maintaining steady state levels of HSP90 regulates the biogenesis, maturation, trafficking and post-translational folding of both wild type and ΔF508 CFTR proteins (Wang et al., 2006b; Banasavadi-Siddegowda et al., 2011).

Invasion and adhesion - cancer cell progression

Note

Gene expression analysis on B16-F10 cells treated with rapamycin or FKBP8 overexpression highlighted the role of FKBP8 gene during tumor cell invasion. FKBP8 overexpression prevents tumor cell invasion by up-regulation of anti-invasive Syndecan (Sdc1) levels and suppression of pro-invasive MMP9 (Fong et al., 2003).

Viral replication

Note

Both in vitro and in vivo studies have shown that FKBP8 binds to HCV NS5. FKBP8 through its TPR domain binds tightly to BH-domain (Bcl-2- homology domain) of HCV NS5. Immunoprecipitation studies showed that FKBP8 forms a heteromeric complex with NS5 and Hsp90. Furthermore, fluorescence and electron microscopy have revealed that FKBP8 partially colocalizes with NS5 into web like cytoplasmic structures, which are probable sites of viral replication and might play an important role in HCV replication (Okamoto et al., 2006; Wang et al., 2006a; Okamoto et al., 2008).

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