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Gene Section
Review

S100A7 (S100 calcium binding protein A7)

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Identity

Other names: PSOR1, S100A7c
HGNC (Hugo): S100A7
Location: 1q21.3
Local order: S100A7 is located on chromosome 1cen-q21 between D1Z5 and MUC1 (Borglum et al., 1995).
Note: S100A7 is also known as psoriasin, psoriasin 1, S100 calcium binding protein A7, S100-A7, S100A7c, and PSOR1.
S100A7, a member of the S100 family, was first identified as a protein upregulated in psoriasis (Madsen et al., 1991).

DNA/RNA

Note
S100A7 is located on chromosome 1q21 within the epidermal differentiation complex.

Description

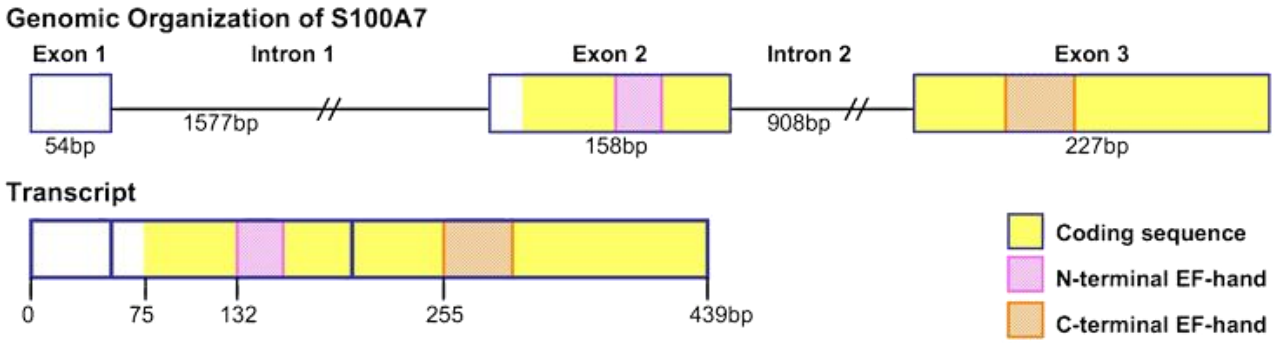
The S100A7 gene has 3 exons and 2 introns with a genomic structure similar to other S100 family members. Exon 1 encodes the 5' untranslated region while exons 2 and 3 contain the protein coding sequence. Exon 2 encodes the start codon and the non-canonical N-terminal EF-hand while exon 3 encodes the carboxyl-terminal EF-hand.

Transcription

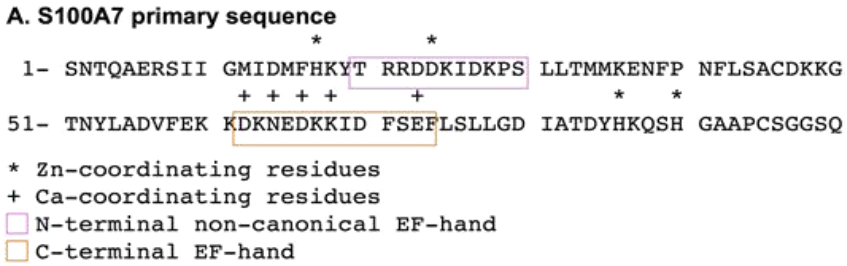
The S100A7 gene encodes for a single constitutively spliced transcript. An EST has been reported in which an alternative promoter is used to produce an identical S100A7 mRNA (See Ensembl, UCSC genome browser).

Pseudogene

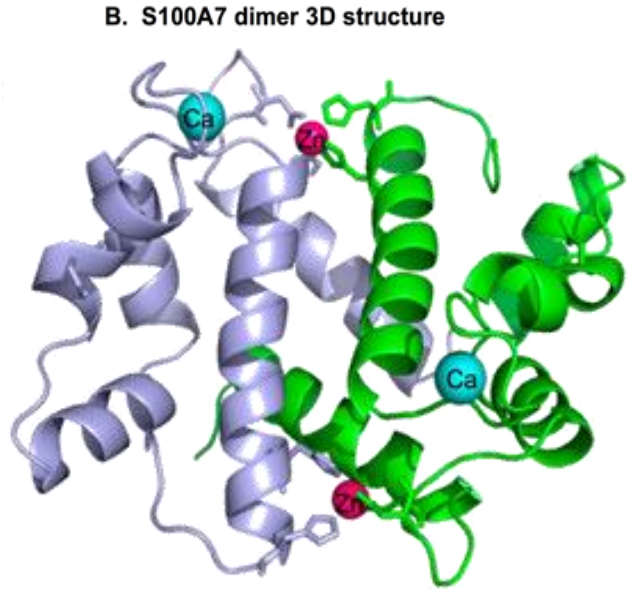
Five copies of S100A7 in the human genome have been reported including the closely related paralog S100A15 (also known as S100A7A) (Kulski et al., 2003; Wolf et al., 2003). Two of the five reported copies of S100A7, S100A7d (S100A7P1) and S100A7e (S100A7P2), are proposed to be non-coding pseudogenes (Kulski et al., 2003; Marenholz et al., 2006).



The S100A7 genomic organization includes 3 exons and 2 introns with exons 2 and 3 containing the protein encoding sequence (Semprini et al., 1999). The EF-hand domains are highlighted (Burgisser et al., 1995).



A. S100A7 primary sequence highlighting the calcium- and zinc-binding residues and the EF-hand domains.



B. The 3D structure of zinc- and calcium-bound S100A7 dimer (2psr).

Protein

Note

S100A7 is a member of the S100 family of calcium-binding signaling proteins. S100A7 has both intracellular and extracellular functions.

Description

S100A7 is a small 11.4 kDa protein containing a C-terminal canonical calcium-binding EF-hand motif and an N-terminal non-canonical EF-hand motif which is characteristic of the S100 protein family. S100A7 forms a homodimer with one Ca^{2+} ion bound by the canonical EF-hand motif in each monomer and two Zn^{2+} ions located at the dimer interface (Brodersen et al., 1999). S100A7 monomers and putative higher order multimers have been observed in both psoriatic and healthy epidermis (Ruse et al., 2001).

Expression

S100A7 is present at low levels in healthy skin, however it is highly upregulated in psoriatic epidermal keratinocytes (Madsen et al., 1991). E. Coli has been shown to induce S100A7 expression in keratinocytes (Gläser et al., 2005).

S100A7 expression is upregulated in several cancers including skin, breast, lung, head, neck, cervix, bladder and gastric cancer (for review see Emberley et al., 2004).

S100A7 expression is induced in MCF10 cells by stresses such as serum deprivation and cell confluency (Enerback et al., 2002).

S100A7 is repressed by BRCA1 in a c-myc dependent manner in HCC-BR116 cells (Kennedy et al., 2005). 17beta-estradiol treatment increased S100A7 expression in an estrogen receptor beta dependent manner in MCF-7 cells (Skliris et al., 2007). Epidermal Growth Factor induces S100A7 expression in MCF-7 and MDA-MB-468 cells (Paruchuri et al., 2008).

S100A7 expression is induced by proinflammatory cytokines in skin and breast cancer cells. S100A7 expression is enhanced in human keratinocytes by stimulation with the cytokine IL-22 in combination with IL-17 or IL-17F (Liang et al., 2006). Oncostatin-M was shown to induce S100A7 expression in human epidermal cell skin equivalents (Gazel et al., 2006). S100A7 expression is induced by the cytokines oncostatin-M and IL-6 in MCF-7, TD47 and MDA-MB-468 cell lines (West and Watson, 2010).

Localisation

S100A7 is localized to the cytoplasm, nucleus, cell periphery and is also secreted from cells.
In keratinocytes, S100A7 is observed in the cytoplasm when untreated and at the cell periphery upon stimulation with calcium (Ruse et al., 2003). S100A7 is expressed at low levels or is not detected in healthy breast cells. In breast cancer cells, however, S100A7 is observed in the nucleus and cytoplasm and is also secreted (Al-Haddad et al., 1999; Enerback et al., 2002).

Function

S100A7 has been shown to function as a chemotactic factor for neutrophils and CD4+ T cells (Jinquan et al., 1996). S100A7 binds RAGE (receptor for advanced glycation end products) in a zinc-dependent manner and is proposed to mediate chemotaxis in a RAGE-dependent manner (Wolf et al., 2008). S100A7 present in skin functions as a Zn-dependent antimicrobial towards E.Coli (Glaser et al., 2005). S100A7 has also been shown to play an antibacterial role in wound healing (Lee and Eckert, 2007). S100A7 is a substrate for transglutaminase (Ruse et al., 2001).
S100A7 interacts, co-purifies and colocalizes in the cytoplasm with epidermal-type fatty acid-binding protein (E-FABP), a protein which is also upregulated in psoriasis (Hagens et al., 1999; Ruse et al., 2003). S100A7 has been shown to interact with RanBPM by yeast two-hybrid and co-immunoprecipitation studies in breast cancer cells (Emberley et al., 2002). S100A7 has been shown to interact with the multifunctional signalling protein, Jab1, yeast two-hybrid and co-immunoprecipitation studies in breast cancer cells (Emberley et al., 2003). The Jab1-S100A7 interaction and downstream effects were disrupted by mutation of a Jab1-binding site (Emberley et al., 2003; West et al., 2009).

Homology

S100A7 is a member of the S100 family of vertebrate proteins. Among the S100 family, S100A7 is the most divergent (Burgisser et al., 1995) with the exception of a recently identified paralog S100A715 (or S100A7A), with which it shares 93% similarity (Wolf et al., 2003). A bovine ortholog to S100A7, Bosd3 (Virtanen, 2006) and equine ortholog (Leeb et al., 2005) have also been reported. The mouse S100A7, which has 40% similarity (Webb et al., 2005), has been assigned the designation mouse S100A15 (Wolf et al., 2006).

Mutations

Note

An allergy associated polymorphism of S100A7 (rs3014837) has been reported (Bryborn et al., 2008).

Implicated in

Psoriasis and other skin diseases

Note

S100A7 is associated with inflammation in several skin diseases (Algermissen et al., 1996). S100A7 was originally identified as a protein secreted from psoriatic skin (Madsen et al., 1991). S100A7 is also overexpressed in skin lesions of patients with lichen sclerosus (Gambichler et al., 2009), acne inversa (Schlapbach et al., 2009), and middle ear cholesteatoma (Kim et al., 2009).

Non-melanoma skin cancer

Note

S100A7 may play a role in the progression of skin cancer. S100A7 expression is not observed in healthy epidermis. When S100A7 levels were studied by immunohistochemistry in squamous cell carcinoma skin lesions, higher levels of expression were found in pre-invasive squamous cell carcinoma in situ compared to invasive squamous cell carcinoma (Alowami et al., 2003). In a separate study, S100A7 mRNA levels, determined by real-time PCR, were upregulated in pre-cancerous skin lesions and epithelial skin tumours including basal cell carcinoma and squamous cell carcinoma (Moubayed et al., 2007).

Melanoma

Note

S100A7 protein was observed at higher levels in the urine of melanoma patients compared to healthy controls (Brouard et al., 2002), although S100A7 was not detected in melanoma cells (Petersson et al., 2009).

Ductal carcinoma in situ (DCIS) and breast cancer

Note

S100A7 was first associated with primary breast cancer (Moog-Lutz et al., 1995). Later studies identified S100A7 as one of the most highly expressed genes in DCIS, a key stage before the transition to invasive breast cancer (Leygue et al., 1996; Enerback et al., 2002). When S100A7 is expressed in later stages of breast cancer it is associated with the agressive estrogen-negative tumors and poor prognosis (Al-Haddad et al., 1999; Emberley et al., 2004). In vivo mouse model studies have shown that S100A7 promotes tumorigenesis (Emberley et al., 2003; Krop et al., 2005). Several of the tumorigenic effects of S100A7, including upregulation of NF-kappaB, PI3K-Akt, and AP-1 as well as promotion of cell survival, are mediated by the interaction of S100A7 with Jab1 (Emberley et al., 2003; Emberley et al., 2005).

Epithelial ovarian cancer

Note

S1007 mRNA and protein levels are upregulated in epithelial ovarian carcinoma tissue compared to normal and benign ovary tissue (Gagnon et al., 2008). Autoantibodies to S100A7 were detected at higher levels in the plasma of early and late-stage ovarian cancer patients compared to healthy controls (Gagnon et al., 2008). S100A7 autoantibodies may be useful as a biomarker for epithelial ovarian cancer (for review see Piura and Piura, 2009).

Lung squamous cell carcinoma

Note

S100A7 is associated with non-small lung squamous cell carcinoma metastasis to the brain (Zhang et al., 2007). Proteomic studies identified S100A7 as a protein upregulated in a brain metastasis lung squamous cell carcinoma cell line and S100A7 overexpression was confirmed in brain metastasis tissues (Zhang et al., 2007).

Bladder squamous cell carcinoma

Note

S100A7 was detected in bladder squamous cell carcinoma tumors and also in the urine of patients with bladder squamous cell carcinoma (Celis et al., 1996; Ostergaard et al., 1997). As a result, S100A7 has been proposed to be a potential biomarker for bladder squamous cell carcinoma (Celis et al., 1996; Ostergaard et al., 1997; Ostergaard et al., 1999).

Oral squamous cell carcinoma

Note

S100A7 is associated with oral squamous cell carcinoma (Zhou et al., 2008; Kesting et al., 2009). RT-PCR and immunofluorescence studies showed that S100A7 mRNA and protein levels respectively are up-regulated in oral squamous cell carcinoma tissues compared to normal oral tissues (Kesting et al., 2009).

Head-and-neck squamous cell carcinoma

Note

S100A7 is a highly upregulated biomarker in head-and-neck squamous cell carcinomas (Ralhan et al., 2008).

Gastric cancer

Note

SAGE (serial analysis of gene expression) studies identified S100A7 as one of the top twenty genes upregulated in gastric cancer (El-Rifai et al., 2002). Further mining of publicly available SAGE, virtual Northern Blot, and microarray data confirmed the association of S100 proteins such as S100A7 with gastric cancer (Liu et al., 2008).

Chronic rhinosinusitis

Note

Chronic rhinosinusitis (CRS) is characterized by a persistant inflammation of the nasal mucosa. It has been proposed that the antibacterial function of S100A7 play a role in protecting against the environmental factors that contribute to chronic rinosinusitis (for review see Tieu et al., 2009). Reduced levels of S100A7 were detected in the nasal lavage fluid of patients with allergic rhinitis when compared to controls (Bryborn et al., 2005). A polymorphism (RS3014837) has been linked with allergic individuals in Sweden (Bryborn et al., 2008).

Systemic sclerosis (SSc)

Note

S100A7 is upregulated in the saliva of patients with systemic sclerosis when compared to healthy individuals and has been proposed as a potential biomarker for systemic sclerosis with pulmonary involvement (Giusti et al., 2007; Baldini et al., 2008).

Alzheimer's disease

Note

A recent study has suggested that S100A7 is a potential biomarker for Alzheimer's disease. Increased levels of S100A7 were detected in the cerebralspinal fluid and brain of patients with Alzheimer's disease (Qin et al., 2009).

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