

Gene Section

Short Communication

S100BP (S100P binding protein)

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Identity

Other names: S100BPBR

HGNC (Hugo): S100BPB

Location: 1p35.1

DNA/RNA

Description

There are two different transcript variants of S100BP. Variant 1 is 4317 base pairs and contains 7 exons. Variant 2 is 1483 base pairs and also contains 7 exons.

This is shown in the below alignment. The coding sequence for each isoform is indicated in red, with the non-coding sequence in grey.

Unmatched base pairs between the two isoforms are highlighted in green and the position of a missing CAG codon in isoform B is circled. The end of the isoform B sequence is indicated by a bold line.

Protein

Description

S100BP isoform A codes for a 45 kDa protein and isoform B for 37 kDa protein.

Secondary structure analysis of the S100BP protein sequence was performed using the secondary structure consensus prediction tool (NPS@, Lyon, France). Based on three different methods: DSC (Discrimination of protein Secondary structure Class), MLRC (Multivariate Linear Regression Combination) and PHD neural network system, S100BP was shown to be largely unstructured and unfolded, as seen below ("h" represents a helix, "c" a coil and "e" an extended strand).

Expression

S100BP is expressed in various normal tissues including prostate and lung and in both the endocrine and exocrine pancreas.

S100BP is also expressed in malignant tissues such as liver hepatocellular carcinoma and thyroid carcinoma (Lines et al., 2012).

In pancreas, S100BP is expressed in decreasing levels as cancer develops and progresses, which is an inverse pattern of expression of its binding partner, S100P.

This is shown on picture below (a= acinar cells, d= ducts; PanIN= pancreatic intraepithelial neoplasia, a precursor lesion to pancreatic cancer, black arrows= PanIN-1, red arrows= PanIN-2; PDAC= pancreatic cancer).

Localisation

Predominantly nuclear.

Function

The exact functions of S100BP are currently unknown, however it has been shown that S100BP can bind to the metastasis related protein S100P (Downen et al., 2005).

Homology

No homology to any currently described protein is seen.

Mutations

Note

No mutations have yet been reported.

Exon 1 10 20 30 40 50 60 70 80 90 100

Isoform A CTGGTGTGGCCCGCTGACTCGGGTAGGGGAGGTCGGG GAGGGGGCGATAAAATGCCGCAGGGGGCCGG AGTGAGGCCAGTCTGTTCCGCCAGGCTTTG
 Isoform B CTGGTGTGGCCCGCTGACTCGGGTAGGGGAGGTCGGG GAGGGGGCGATAAAATGCCGCAGGGGGCCGG AGTGAGGCCAGTCTGTTCCGCCAGGCTTTG
 Consensus CTGGTGTGGCCCGCTGACTCGGGTAGGGGAGGTCGGG GAGGGGGCGATAAAATGCCGCAGGGGGCCGG AGTGAGGCCAGTCTGTTCCGCCAGGCTTTG

Exon 2 110 120 130 140 150 160 170 180 190 200

Isoform A GCCCGGCTTCGGAGCAAGGTGGGAGTCAATCATTTTGC AAGTCTCTGAAAGG AACAGCTAGCAGGAA CTGAACCTTTTTCC ATTTGGTCTCGTGGC
 Isoform B GCCCGGCTTCGGAGCAAGGTGGGAGTCAATCATTTTGC AAGTCTCTGAAAGG AACAGCTAGCAGGAA CTGAACCTTTTTCC ATTTGGTCTCGTGGC
 Consensus GCCCGGCTTCGGAGCAAGGTGGGAGTCAATCATTTTGC AAGTCTCTGAAAGG AACAGCTAGCAGGAA CTGAACCTTTTTCC ATTTGGTCTCGTGGC

Exon 3 210 220 230 240 250 260 270 280 290 300

Isoform A AAAGGCAGAGATTGCTCCAGCAGCTCCACACAAAATGATGTGCTCACGGGTGCCCTCTGAACAGTCTCTGGTACCTCTCTTG CCTAAGACGGGTGCC
 Isoform B AAAGGCAGAGATTGCTCCAGCAGCTCCACACAAAATGATGTGCTCACGGGTGCCCTCTGAACAGTCTCTGGTACCTCTCTTG CCTAAGACGGGTGCC
 Consensus AAAGGCAGAGATTGCTCCAGCAGCTCCACACAAAATGATGTGCTCACGGGTGCCCTCTGAACAGTCTCTGGTACCTCTCTTG CCTAAGACGGGTGCC

310 320 330 340 350 360 370 380 390 400

Isoform A CCATTTTCTTGGGATTCCTTGGATGAGGATGGATGGATGACTCCTTGCTGGAGCTGTCAAGGGGAGAG AAGATGATGGTGTGTAAATACACAGAGG
 Isoform B CCATTTTCTTGGGATTCCTTGGATGAGGATGGATGGATGACTCCTTGCTGGAGCTGTCAAGGGGAGAG AAGATGATGGTGTGTAAATACACAGAGG
 Consensus CCATTTTCTTGGGATTCCTTGGATGAGGATGGATGGATGACTCCTTGCTGGAGCTGTCAAGGGGAGAG AAGATGATGGTGTGTAAATACACAGAGG

410 420 430 440 450 460 470 480 490 500

Isoform A AAGAGATTGATGCACTGTTGAAGGAAGATGACCCATCATATGAGCAGTCTTCTGGGGAAGATGATGGTGG GCATGTTGAGAGGG AGAAGAGGGGAGTCA
 Isoform B AAGAGATTGATGCACTGTTGAAGGAAGATGACCCATCATATGAGCAGTCTTCTGGGGAAGATGATGGTGG GCATGTTGAGAGGG AGAAGAGGGGAGTCA
 Consensus AAGAGATTGATGCACTGTTGAAGGAAGATGACCCATCATATGAGCAGTCTTCTGGGGAAGATGATGGTGG GCATGTTGAGAGGG AGAAGAGGGGAGTCA

510 520 530 540 550 560 570 580 590 600

Isoform A AATTTCTACTT GATACTCCCCGAGAGAAAATTTCATCGTAC AGCCTGGGACCAGT A GCTGAGACTCCTGAC CTCTTCAAACACTCT CAGCTAAGTACATCA
 Isoform B AATTTCTACTT GATACTCCCCGAGAGAAAATTTCATCGTAC AGCCTGGGACCAGT A GCTGAGACTCCTGAC CTCTTCAAACACTCT CAGCTAAGTACATCA
 Consensus AATTTCTACTT GATACTCCCCGAGAGAAAATTTCATCGTAC AGCCTGGGACCAGT A GCTGAGACTCCTGAC CTCTTCAAACACTCT CAGCTAAGTACATCA

610 620 630 640 650 660 670 680 690 700

Isoform A AGTGGTCATGGACCAGCTCATACTA AACCATTAAACAGACGCTCTGTACTAGAAA AGAATCTTATAAAGTAACTGTTGCACCATTTAATCCAAACAGTTT
 Isoform B AGTGGTCATGGACCAGCTCATACTA AACCATTAAACAGACGCTCTGTACTAGAAA AGAATCTTATAAAGTAACTGTTGCACCATTTAATCCAAACAGTTT
 Consensus AGTGGTCATGGACCAGCTCATACTA AACCATTAAACAGACGCTCTGTACTAGAAA AGAATCTTATAAAGTAACTGTTGCACCATTTAATCCAAACAGTTT

710 720 730 740 750 760 770 780 790 800

Isoform A GTGATGCTCTGCTTGTAAAGGACGAGACTGATTCGTTCCAAAGATACTGAAAAMCTCTCTTCCCTTGAGAGAGAGATGAGAGATGGTCTTAGCCCCAAA
 Isoform B GTGATGCTCTGCTTGTAAAGGACGAGACTGATTCGTTCCAAAGATACTGAAAAMCTCTCTTCCCTTGAGAGAGAGATGAGAGATGGTCTTAGCCCCAAA
 Consensus GTGATGCTCTGCTTGTAAAGGACGAGACTGATTCGTTCCAAAGATACTGAAAAMCTCTCTTCCCTTGAGAGAGAGATGAGAGATGGTCTTAGCCCCAAA

810 820 830 840 850 860 870 880 890 900

Isoform A TGAAGCAAACTTTGTAATCTGAAGGGATCAGCCCCAATAACTCTGCTTGG AATGGGCCCCAGCTCTCTTCTCAAACAACTAACTTTCAACAGACT
 Isoform B TGAAGCAAACTTTGTAATCTGAAGGGATCAGCCCCAATAACTCTGCTTGG AATGGGCCCCAGCTCTCTTCTCAAACAACTAACTTTCAACAGACT
 Consensus TGAAGCAAACTTTGTAATCTGAAGGGATCAGCCCCAATAACTCTGCTTGG AATGGGCCCCAGCTCTCTTCTCAAACAACTAACTTTCAACAGACT

910 920 930 940 950 960 970 980 990 1000

Isoform A GTCTCTGATAAAAATATGCCTGACAGTGAGAACCCCTACGTCTGTATTCTCTGGATCTCAGACCATTGAGAGACTCCTAATATGGAGTTATCCTGACAGAA
 Isoform B GTCTCTGATAAAAATATGCCTGACAGTGAGAACCCCTACGTCTGTATTCTCTGGATCTCAGACCATTGAGAGACTCCTAATATGGAGTTATCCTGACAGAA
 Consensus GTCTCTGATAAAAATATGCCTGACAGTGAGAACCCCTACGTCTGTATTCTCTGGATCTCAGACCATTGAGAGACTCCTAATATGGAGTTATCCTGACAGAA

Exon 4 1010 1020 1030 1040 1050 1060 1070 1080 1090 1100

Isoform A ATGGTGGTTCACACAAGTCAAGTTGTGAANTGAGATCTCTGTTGTTCCACCTCATCAAACAAATGAGTCTTTAAACAAGGATTCTGGGAAGATGAA
 Isoform B ATGGTGGTTCACACAAGTCAAGTTGTGAANTGAGATCTCTGTTGTTCCACCTCATCAAACAAATGAGTCTTTAAACAAGGATTCTGGGAAGATGAA
 Consensus ATGGTGGTTCACACAAGTCAAGTTGTGAANTGAGATCTCTGTTGTTCCACCTCATCAAACAAATGAGTCTTTAAACAAGGATTCTGGGAAGATGAA

Exon 5 1110 1120 1130 1140 1150 1160 1170 1180 1190 1200

Isoform A AGGCCATGAGAGAAGACTAGGCCAAAGTCATTCTGTCTCAAACTAAGAC CAGGACTAATGTTCCGACGTTTTACAGTCAAACTAGAACAGCAGAGG
 Isoform B AGGCCATGAGAGAAGACTAGGCCAAAGTCATTCTGTCTCAAACTAAGAC CAGGACTAATGTTCCGACGTTTTACAGTCAAACTAGAACAGCAGAGG
 Consensus AGGCCATGAGAGAAGACTAGGCCAAAGTCATTCTGTCTCAAACTAAGAC CAGGACTAATGTTCCGACGTTTTACAGTCAAACTAGAACAGCAGAGG

Exon 6 1210 1220 1230 1240 1250 1260 1270 1280 1290 1300

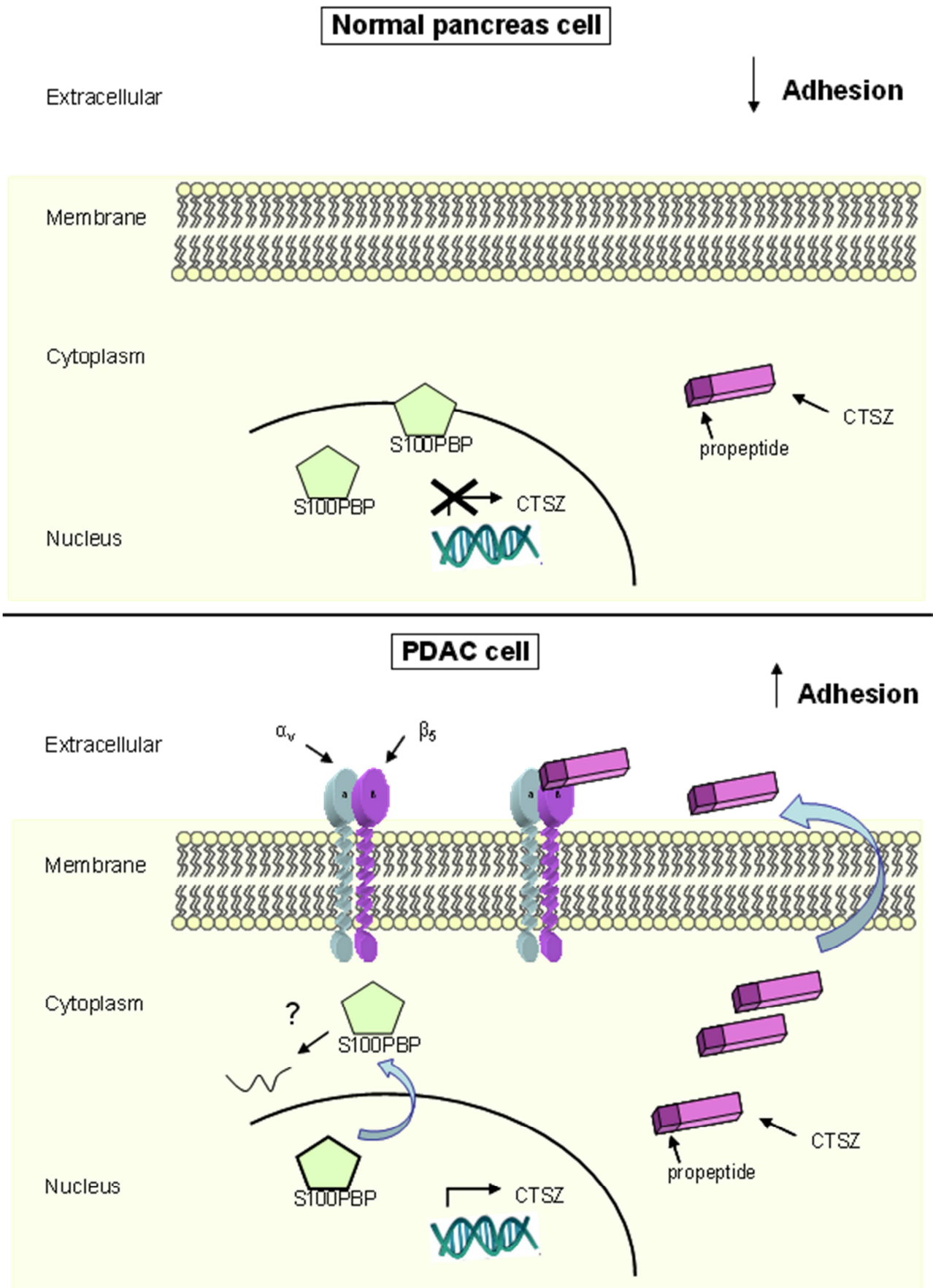
Isoform A CAGCTTTATCTCAGGAGTCTCATATAGAAGACC CAGAGGACACTAACC AAGGATCTCGGGGG AGCTTTTGCCCTTATGGATCAAGTTCAATC
 Isoform B CAGCTTTATCTCAGGAGTCTCATATAGAAGACC CAGAGGACACTAACC AAGGATCTCGGGGG AGCTTTTGCCCTTATGGATCAAGTTCAATC
 Consensus CAGCTTTATCTCAGGAGTCTCATATAGAAGACC CAGAGGACACTAACC AAGGATCTCGGGGG AGCTTTTGCCCTTATGGATCAAGTTCAATC

Exon 7 1310 1320 1330 1340 1350 1360 1370 1380 1390 1400

Isoform A ATATGACGACTCAAAAATGGCAGCATCCTTTCGGACCTCAC CAGCGGA ACTACGC CCGCCGACAGAAAATCTGCAAAAGATACAGTCTGACTCAGTGGGT
 Isoform B ATATGACGACTCAAAAATGGCAGCATCCTTTCGGACCTCAC CAGCGGA ACTACGC CCGCCGACAGAAAATCTGCAAAAGATACAGTCTGACTCAGTGGGT
 Consensus ATATGACGACTCAAAAATGGCAGCATCCTTTCGGACCTCAC CAGCGGA ACTACGC CCGCCGACAGAAAATCTGCAAAAGATACAGTCTGACTCAGTGGGT

1410 1420 1430 1440 1450 1460 4310

Isoform A TGACAGGAACATGCGAAGCCACCATCGGTTCCAGCGTCTCCAGACTTCTCGTACAGTTAATTTG
 Isoform B TGACAGGAACATGCGAAGCCACCATCGGTTCCAGCGTCTCCAGACTTCTCGTACAGTTAATTTG
 Consensus TGACAGGAACATGCGAAGCCACCATCGGTTCCAGCGTCTCCAGACTTCTCGTACAGTTAATTTG



Implicated in

Pancreatic ductal adenocarcinoma (PDAC)

Note

Overexpression of S100PBP in FA6 pancreatic cancer cells that show low levels of endogenous S100PBP expression and silencing of S100PBP in MiaPaCa2 cells (high levels of endogenous S100PBP) showed no effect on proliferation or wound healing.

While cell migration was not affected in majority of tested pancreatic cancer cell lines after modulation of S100PBP expression, significant changes in invasion (increase in MiaPaCa2 and Panc1 cells after S100PBP silencing and decrease in Rwp1 cells after overexpression) were seen. However, the most affected cellular function after modulation of S100PBP expression was adhesion. Loss of S100PBP causes an increase in pancreatic cancer cell adhesion to extracellular matrix proteins which was mediated by cysteine protease Cathepsin Z (CTSZ) and the integrin $\alpha\beta 5$ (Lines et al., 2012).

Schematic and simplified diagram of the putative mechanism behind S100PBP mediated changes in pancreatic cell adhesion is shown below: in normal

cells where S100PBP is highly expressed, low levels of CTSZ are present; no or very little $\alpha\beta 5$ is seen on the cellular surface. In cancer cells (PDAC), S100PBP levels decrease, which results in an increase in CTSZ expression; CTSZ is then secreted and can interact with $\alpha\beta 5$, promoting thus the adhesion.

Disease

Pancreatic cancer.

References

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