TRIAP1 (TP53 regulated inhibitor of apoptosis 1)

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Identity

Other names: HSPC132; p53CSV; P53CSV; WF-1
HGNC (Hugo): TRIAP1
Location: 12q24.31

DNA/RNA

Description
2452 bases, starts at 119366147 and ends at 119368598 bp from promoter with minus strand orientation.

Transcription
This gene contains 2 introns which transcription gives 3 different mRNAs, 2 alternatively spliced variants and 1 unspliced form that encodes good proteins (see figure 1).

Protein

Note
The P53CSV protein is involved in programmed cell death. It contains a p53-binding site and it is induced when cells are at low genotoxic stress. It is probably involved in cell survival by interaction between Apaf-1 (apoptosis protease activating factor 1) and heat shock protein 70 (Hsp70) with subsequent inhibition of caspase-9 activation.

Figure 1.
**Description**

This protein contains 76 amino acids and has 8786 (Da) of weight.

![Figure 2.](image)

**Localisation**

The protein is localized in cytoplasm and perinuclear region.

**Function**

P53CSV is a novel p53-target gene. This gene can modulate apoptotic pathways by interaction with heat shock protein 70 (HSP70), preventing the induction of apoptosis. When cells are submitted to low levels of genotoxic stress, it is an important player in P53-mediated cell survivor pathway (Park and Nakamura, 2005; Felix et al., 2009). P53CSV can inhibit apoptosis through interaction with APAF1 and HSP70 complex.

**Mutations**

**Note**

There are two identified alterations until now. One of them is located at position 270 of mRNA and the allele G (guanine) is switched to the allele C (cytosine) at position 77 of the amino acid sequence protein. The other one is a synonymous alteration localized at position 160 of mRNA involving the protein residue Leucine. The allele C (cytosine) is switched to the allele T (thymine) at position 40 of the amino acid sequence protein (NCBI).

**Implicated in**

**Multiple myeloma**

**Note**

Felix et al. (2009), described that P53CSV gene was upregulated in multiple myeloma SAGE (serial analysis of gene expression) library when compared to normal/reactive plasma cells.

![Figure 3.](image)
They suggested that the interaction between P53CSV/Hsp70 should be evaluated as a potential target for multiple myeloma patients. Real time quantitative PCR analyses confirmed upregulation of P53CSV in 90% of multiple myeloma plasma samples cells.

**Inflammatory stress**

**Note**

Staib et al. (2005) reported P53CSV expression in colon carcinoma cells in the course of inflammatory responses associated with four microenvironmental components: nitric oxide, hydrogen peroxide, DNA replication arrest, and hypoxia.

**Solid cancers**

**Note**

Yu Kun et al. (2008), using a genome-wide computational strategy identified genes exhibiting precise transcriptional control in solid tumors and evaluated if they linked to multiple cancer-related pathways such as metastatic and invasive potential. siRNA knockdown of five genes supports the existence of precisely controlled genes in solid tumors, including P53CSV.

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**Figure 4.** Hypothetical P53CSV mechanism of action in interaction with heat shock protein 70 in normal and tumor cells (Felix et al., 2009)

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**References**


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*This article should be referenced as such: