RUVBL2 (RuvB-like 2 (E. coli))

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Identity

Other names: CGI-46; ECP51; INO80J; REPTIN; RVB2; Reptin52; Rvb2; TAP54-beta; THI2; TIP48; TIP49B
HGNC (Hugo): RUVBL2
Location: 19q13.33

DNA/RNA

Description
15 exons, 14 introns (Parfait et al., 2000).

Transcription
1518bp mRNA with 463aa open reading frame.

Protein

Description
463 amino acids, 52 kDa.
RUVBL2 belongs to the AAA+ ATPase super-family (ATPases associated with diverse cellular activities) sharing conserved Walker A and B motifs, arginine fingers, and sensor domains. The monomers contain two domains, which are involved in ATP binding and hydrolysis respectively. RUVBL2 assembles into an hexameric structure with a central channel.
RUVBL2 interacts with RUVBL1 to form a dodecamer (Puri et al., 2007). This RUVBL1/ RUVBL2 complex displays a significant ATPase activity and is likely one of the functional forms of the proteins. Sumoylation of RUVBL2 has been reported on Lys456 in invasive prostate cancer cells (Kim et al., 2006).
RUVBL2 is phosphorylated on an ATM/ATR consensus site following DNA damage (Matsuoka et al., 2007).

Expression

Expression of RUVBL2 is ubiquitous but especially abundant in thymus and testis (Salzer et al., 1999; Parfait et al., 2000).
RUVBL2 is overexpressed in hepatocellular carcinoma (Rousseau et al., 2007). Overexpression of RUVBL2 in several cancers and its possible role in human cancers has been reported (reviewed in Huber et al., 2008).

Localisation

Cytoplasm and nucleus.

Function

RUVBL2 interacts with c-myc (Wood et al., 2000) and also modulates transcriptional regulation by the beta-catenin/TCF-LEF complex (Bauer et al., 2000) and ATF2 (Cho et al., 2001). RUVBL2 participates in the remodelling of chromatin as a member of several complexes such as TIP60 (Ikura et al., 2000), INO80 (Jin et al., 2005), SRCAP (Cai et al., 2005).
It is also involved in transcriptional regulation (reviewed in Gallant, 2007), DNA repair (Gospodinov et al., 2008), snoRNP biogenesis (Watkins et al., 2002), and telomerase activity (Venteicher et al., 2008). RUVBL2 silencing in fibroblasts induces a senescent phenotype (Chan et al., 2005).

Implicated in

Hepatocellular carcinoma (HCC)

Disease
RUVBL2 was found to be overexpressed in 75% of cases in a series of 96 human HCC studied with real-time RT-PCR (Rousseau et al., 2007). It was also increased in a smaller 15 cases series (Iizuka et al., 2006). No mutations in the coding sequence were identified (Rousseau et al., 2007).
Prognosis
Overexpression of RUVBL2 was an independent factor of poor prognosis (Rousseau et al., 2007).

Oncogenesis
RUVBL2 depletion with siRNAs led to HCC cell growth arrest and apoptosis, whereas over-expression in HCC cells allowed these cells to give rise to more progressive tumors in xenografts than control cells (Rousseau et al., 2007).

Colon cancer
Disease
RUVBL2 was overexpressed in a series of 18 colon cancers (Graudens et al., 2006).

Melanoma
Disease
RUVBL2 was overexpressed in a series of 45 melanomas (Talantov et al., 2005).

Bladder carcinoma
Disease
RUVBL2 was overexpressed in a series of 108 bladder carcinomas (Sanchez-Carbayo et al., 2006).

Prostate cancer
Oncogenesis
In conjunction with beta-catenin, RUVBL2 represses the expression of the anti-metastasis gene KAI-1 (Kim et al., 2005) and is involved in the invasive phenotype of cultured prostate cancer cells (Kim et al., 2006).

References


Watkins NJ, Dickmanns A, Lührmann R. Conserved stem II of the box C/D motif is essential for nucleolar localization and is required, along with the 15.5K protein, for the hierarchical assembly of the box C/D snoRNP. Mol Cell Biol. 2002 Dec;22(23):8342-52


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