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Gene Section
Mini Review

JUND (proto-oncogene)

Fei Chen

Health Effects Laboratory Division, NIOSH, 1095 Willowdale Rd, Morgantown, WV 26505, USA (FC)

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Identity

Other names: JUN-D proto-oncogene

HGNC (Hugo): JUND

Location: 19p13.1-p12

DNA/RNA

Description

The gene for JUND is located on the region of chromosome 19p13.1-p12 covering 1409bp. Similar to other members of JUN family, the JunD gene is also intronless.

Protein

Description

The JUND protein contains 347 amino acids with a predicted molecular weight 35.2 kD. From N-terminus to C-terminus, JUND has a JNK phosphorylating motif (Ser90/Ser100), DNA binding domain, nuclear localization signal (NLS), and a leucine zipper domain. Since this protein lacks the JNK docking site, JUND can only be weakly phosphorylated by JNK.

Although the JunD gene has no introns and produces a single transcript, the JUND mRNA translates two JUND protein isoforms, JUND-L and JUND-S. By using a different in-frame translational initiation site, the third AUG codon in JUND mRNA, the short version of JUND, JUND-S, was generated that lacks the N-terminal 43 amino acids. Due to this N-terminal truncation, the JUND-S is unable to associate with Menin, another tumor suppressor protein.

Expression

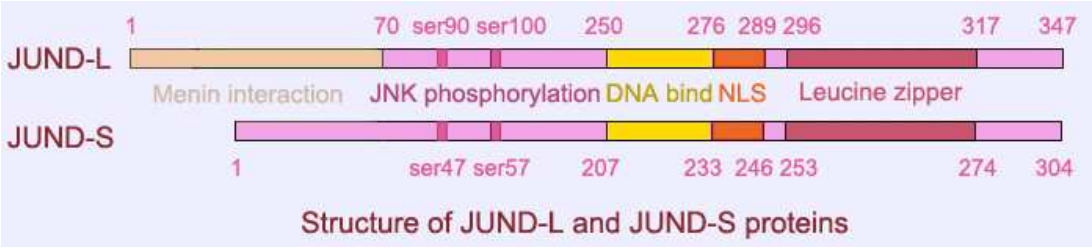
JUND is the most broadly expressed member of the JUN family but expressed at low level.

Localisation

The subcellular location of this protein is most likely in the nucleus. Less likely possibilities are in the cytoplasm and in the mitochondria.

Function

JUND is a member of the JUN family of basic region leucine zipper (bZIP) DNA-binding proteins. Analysis of the protein expression levels demonstrated an opposite expression pattern between JUN and JUND. When cells entry into the G0 phase of the cell cycle by serum starvation, JUN level decreases and JUND level increases. Similar to JUNB, JUND has been shown as an antagonist of JUN in the induction of cyclin D1. Therefore, increasing the abundance of JUND may maintain the cells in a quiescent state. Transformation studies demonstrated that excess JUND protein could partially suppress the transformed phenotype mediated by JUN in cooperation with Ras. The effect of JUND in development appears to be marginal. Mice lacking JUND are viable with only mild defects in growth and spermatogenesis, whereas mice lacking JUN or JUNB die in embryo.



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