STK11 (serine/threonine kinase 11)

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Identity

Other names: LKB1; PJS (Peutz-Jeghers syndrome)
HGNC (Hugo): STK11
Location: 19p13.3

DNA/RNA

Description
Spans 23 kb; 10 exons, transcribed in telomere to centromere direction.

Transcription
Transcripts of 3.0 and 3.3 kb mainly.

Protein

Description
436 amino acids, 48 kDa; N-term with a nuclear localization domain and a putative cytoplasmic retention signal, a kinase domain, and a C-term domain that is phosphorylated on a Ser by the cAMP-dependant protein kinase.

Expression
Wide, in particular during embryonic development.

Localisation
Found in both the nucleus and the cytoplasm, but predominantly nuclear.

Function
Serine/threonine protein kinase of unknown function; expression of LKB1 results in an inhibition of cell growth by inducing G1 arrest.

Mutations

Germinal
Most mutations are null alleles; they are dispersed through the entire gene.

Somatic
Many of the polyps that develop in Peutz-Jeghers syndrome (see below) show loss of heterozygosity; somatic mutations have been tested and rarely found in the following sporadic cancers: pancreas, colon, stomach, breast, uterine cervix, ovary, testis, melanomas. The inactivation of the LKB1 can also occur through promoter hypermethylation.

Implicated in

Peutz-Jeghers syndrome (PJS)

Disease
Syndrome associating mucocutaneous melanotic pigmentation, intestinal polyposis, and an increased risk of cancers (small intestine, stomach, pancreas, colon, esophagus, ovary, uterus, breast, and lung).

Hybrid/Mutated gene
A majority of Peutz-Jeghers patients show mutation in STK11; there is however genetic heterogeneity in this disease.

Oncogenesis
STK11 is affected by biallelic inactivation in tumors of Peutz-Jeghers syndrome patients.

References


Marignani PA, Kanai F, Carpenter CL. LKB1 associates with Brd1 and is necessary for Brd1-induced growth arrest. J Biol Chem. 2001 Aug 31;276(35):32415-8

Sapkota GP, Kieloch A, Lizcano JM, Lain S, Arthur JS, Williams MR, Morrice N, Deak M, Alessi DR. Phosphorylation of the protein kinase mutated in Peutz-Jeghers cancer syndrome, LKB1/STK11, at Ser431 by p90(RSK) and cAMP-dependent protein kinase, but not its farnesylation at Cys(433), is essential for LKB1 to suppress cell growth. J Biol Chem. 2001 Jun 1;276(22):19469-82


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