The long form of CDK2 arises via alternative splicing and forms an active protein kinase with cyclins A and E

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Abstract

We have reinvestigated the long form of cyclin-dependent kinase (CDK)2 that is expressed in many rodent cells. We show that the mRNA encoding CDK2L arises by alternative splicing and that the encoded protein can bind to, and be activated by, cyclins A and E. The complex of CDK2L with cyclin A has about half the specific activity of the equivalent CDK2-cyclin A complex. Also, CDK2L--cyclin A is inhibited to the same extent and by the same concentrations of p21(CIP1) as CDK2--cyclin A. The nucleotide sequences of intron V in the human and murine CDK2 genes, where the sequences encoding the 48-residue insert in CDK2L are located, show very high conservation in the position of the alternatively spliced exon and its surroundings. Despite this, we were not able to detect significant expression of CDK2L in human cell lines, although a low level is expressed in COS-1 cells from monkeys.