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Investigating the Contribution of the Oncogenic Cyclin D1b Isoform to the Aggressive Growth Properties of SV40 T Antigen Transformed Human Diploid Fibroblasts

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Abstract

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Cyclin D1 is a driving force of the cell cycle by the regulation of S-phase entry. Cyclin D1 complexes with kinase CDK4/6 to phosphorylate the retinoblastoma tumor suppressor protein resulting in release of the E2F transcription factor. Over-expression or promotion of the alternatively spliced oncogenic D1b isoform is correlated with cancer. To determine the contribution of cyclin D1 to virally induced cancers, this study used immortalized and virally transformed HDFs. RT-PCR showed more D1a RNA produced compared to D1b and no significant difference in the amount of the isoforms in transformed cell lines. Immunoblotting and immunofluorescence suggested that the cyclin is predominantly cytoplasmic, suggesting that little to no cyclin D1b is being produced at the protein level. Results are surprising but indicate that even though splicing factors are upregulated in transformed cells, they do not result in cyclin D1b production; thus, D1b does not contribute to aggressive growth in HDFs.