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Identification Of Key Genes And Carcinogenic Pathways In Hepatitis B Virus-associated Hepatocellular Carcinoma Through Bioinformatics Analysis

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Background : The mechanisms of development of hepatocellular carcinoma (HCC) in hepatitis B virus (HBV)-infected patients remain unclear. The present study sought to identify the genes and mechanisms involved in the development of HBV-associated HCC.

Methods : The GSE121248 gene dataset, which included 70 HCCs and 37 adjacent liver tissues, was downloaded from the Gene Expression Omnibus database. Differentially expressed genes (DEGs) in HCCs and adjacent liver tissues were identified, and gene ontology and Kyoto Encyclopedia of Genes and Genome pathway enrichment analyses were performed.

Results : Of the 134 DEGs identified, 34 were up-regulated and 100 down-regulated in HCCs. The 34 up-regulated DEGs were mainly involved in nuclear division, organelle fission, spindle and midbody formation, histone kinase activity, and the p53 signaling pathway, whereas the 100 down-regulated DEGs were involved in steroid and hormone metabolism; the collagen-coated extracellular matrix; oxidoreductase activity; and activity on paired donors, including the incorporation or reduction of molecular oxygen, monooxygenase activity, and retinol metabolism. Analyses of protein-protein interaction networks with a high degree of connectivity identified significant modules containing 14 hub genes, including ANLN, ASPM, BUB1B, CCNB1, CDK1, CDKN3, ECT2, HMMR, NEK2, PBK, PRC1, RACGAP1, RRM2, and TOP2A, which were mainly associated with nuclear division, organelle fission, spindle formation, protein serine/threonine kinase activity, the p53 signaling pathway, and the cell cycle.

Conclusions : This study identified key genes and carcinogenic mechanisms that play essential roles in the development of HBV-associated HCC. This may provide important evidence in the development of diagnostic and therapeutic targets for HCC.

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