The direct and indirect roles of HBV in liver cancer: prospective markers for HCC screening and potential therapeutic targets.

Abstract:
Chronic hepatitis B virus (HBV) infection remains the number one risk factor for hepatocellular carcinoma (HCC), accounting for more than 600,000 deaths/year. Despite highly effective antiviral treatment options, chronic hepatitis B (CHB), subsequent end-stage liver disease and HCC development remain a major challenge worldwide. In CHB, liver damage is mainly caused by the influx of immune cells and destruction of infected hepatocytes, causing necro-inflammation. Treatment with nucleoside/nucleotide analogues can effectively suppress HBV replication in patients with CHB and thus decrease the risk for HCC development. Nevertheless, the risk of HCC in treated patients showing sufficient suppression of HBV DNA replication is significantly higher than in patients with inactive CHB, regardless of the presence of baseline liver cirrhosis, suggesting direct, long-lasting, predisposing effects of HBV. Direct oncogenic effects of HBV include integration in the host genome, leading to deletions, cis/trans-activation, translocations, the production of fusion transcripts and generalized genomic instability, as well as pleiotropic effects of viral transcripts (HBsAg and HBx). Analysis of these viral factors in active surveillance may allow early identification of high-risk patients, and their integration into a molecular classification of HCC subtypes might help in the development of novel
therapeutic approaches.

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