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# HBV AND HEPATOCARCINOGENESIS IN TRANSLATIONAL MEDICINE

**Xiaodong Zhang**
Nankai University, China

HCC is the fifth-most common cancer and the third leading cause of cancer death worldwide. HBV infection is one of the major causes of HCC. HBx plays critical roles in the development of liver cancer. Our group has reported that HBx modulates oncogene YAP via CREB to promote growth of hepatoma cells. HBx promotes the development of liver fibrosis and hepatoma through down-regulation of miR-30e targeting *P4HA2* mRNA up-regulates Lin28A/Lin28B through Sp1/c-Myc to enhance the proliferation of hepatoma cells. Up-regulated long non-coding RNA HULC by HBx enhances growth of hepatoma cells via down-regulating p18. HULC modulates abnormal lipid metabolism in hepatoma cells through a miR-9–mediated RXRA signaling pathway. *MicroRNA-520e* suppresses growth of hepatoma cells by targeting the NF-κBinducing kinase (NIK). Therapeutically, anti-HBV drugs suppress the growth of HBV-related hepatoma cells via down-regulation of hepatitis B virus X protein. Our findings provide new insights into the mechanism by which HBV promotes the development of HCC. Our findings develop novel targets for anti-HCC therapy. Current antiviral therapies inhibit cytoplasmic HBV genomic replication, but rarely achieve a cure because they do not directly target nuclear HBV covalently closed circular DNA (cccDNA), the genomic form that serves as a HBV replication intermediate and viral persistence reservoir. We report that HBx-elevated MSL2 modulates HBV cccDNA through inducing degradation of APOBEC3B to enhance hepatocarcinogenesis.

zhangxd@nankai.edu.cn