Calcium signaling in hepatitis B virus infection and its potential as a therapeutic target

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Video Byte

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Abstract

The ubiquitous second messenger calcium (Ca^{2+}) interacts with numerous cellular proteins to regulate physiological processes. Ca^{2+} also participates in a variety of diseases, including hepatitis B virus (HBV) infection. HBV infection is a major cause of fibrosis, cirrhosis, and hepatocellular carcinoma. Recent studies have demonstrated that HBV infection elevates levels of intracellular Ca^{2+} and this elevation is primarily dependent on the HBV protein HBX. The activation of Ca^{2+} signaling contributes to viral replication in HBV-infected cells. The importance of Ca^{2+} signaling in HBV infection makes controlling intracellular Ca^{2+} a promising therapeutic target. Early studies have suggested that binding cytosolic Ca^{2+} or inhibiting Ca^{2+} channels reduces viral replication, but current research is largely derived from in vitro cellular models and needs to be confirmed in animal models and human patients. The influence Ca^{2+} signaling has on the subsequent development of HBV-associated diseases, like hepatitis, is also largely unexplored.