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Targeting apolipoprotein E could be key to eliminating hepatitis B virus

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Human apolipoprotein E (apoE) promotes hepatitis B virus (HBV) infection and production, according to a study published Aug 8 in the open-access journal *PLOS Pathogens* by Luhua Qiao and Guangxiang (George) Luo of the University of Alabama at Birmingham School of Medicine. The findings suggest that inhibitors interfering with apoE biogenesis, secretion, and/or binding to receptors may serve as antivirals for the elimination of chronic HBV infection.

HBV chronically infects about 240 million people worldwide, posing a major global health problem. The virus is a common cause of liver diseases, including chronic hepatitis, steatosis, fibrosis, cirrhosis, and hepatocellular carcinoma. The current standard antiviral therapy effectively inhibits HBV replication but does not eliminate the virus. Moreover, little is known about the importance of host factors in HBV infection, assembly, and release. To address this gap in knowledge, Qiao and Luo set out to determine the association of apoE with HBV and its importance in the HBV life cycle.

In the new study, the researchers show that apoE, which is known to play an important role in hepatitis C virus (HCV) infection, also promotes HBV infection and production. They found that human apoE is enriched in HBV and is incorporated onto the virus envelope. HBV infection was efficiently blocked by an apoE-specific monoclonal antibody or by silencing apoE expression and apoE gene knockout. Moreover, down-regulation of apoE expression or knockout of the apoE gene from the HBV-producing liver cells severely impaired HBV production. The

authors speculate that apoE may play a role in persistent HBV infection by evading host immune responses, similar to its role in the HCV life cycle.

More information: Luhua Qiao et al, Human apolipoprotein E promotes hepatitis B virus infection and production, *PLOS Pathogens* (2019). DOI: 10.1371/journal.ppat.1007874