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Intrinsic Immune Response in HBV/HDV-infected Cells and the Activation of Associated Innate Immune Cells

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Introduction

Hepatitis B virus and hepatitis D virus are significant viral pathogens that cause chronic liver disease, often leading to cirrhosis and hepatocellular carcinoma. The immune system plays a crucial role in both controlling these infections and in the pathogenesis of liver disease. The intrinsic immune response in HBV and HDV-infected cells, along with the activation of innate immune cells, is a key component of the body's defense mechanisms. However, the viruses have evolved strategies to evade the immune system, leading to persistent infection and chronic liver disease in many individuals. This article explores the intrinsic immune responses in cells infected with HBV and HDV and the activation of associated innate immune cells [1,2].

Description

Hepatitis B virus is a hepatotropic DNA virus that primarily infects hepatocytes (liver cells). The virus has a complex lifecycle that involves integration of its DNA into the host genome, which can lead to chronic infection and liver damage. HBV infection is a major cause of liver cirrhosis and hepatocellular carcinoma, and chronic infection often leads to a failure of the immune system to clear the virus. Hepatitis D virus is a defective RNA virus that requires HBV for replication. HDV is unique because it relies on the HBV surface antigen for its propagation, and it can exacerbate the severity of HBV-related liver disease. Infected individuals often have more severe outcomes, including an increased risk of liver failure and hepatocellular carcinoma. Both HBV and HDV infections provoke an immune response, but the viruses have developed mechanisms to evade or suppress host immunity, leading to chronic infection and liver damage. Once viral components are recognized, host cells activate a variety of signaling pathways designed to limit viral replication. The primary antiviral response involves the production of type I interferons, such as IFN-αand IFN-α which activate antiviral genes within the infected cell and surrounding cells. These genes induce a state of cellular resistance to viral replication and help control the spread of the virus. In the case of HBV, the viral DNA is less easily recognized than RNA, making it harder for cells to trigger an immediate interferon response. This is a significant reason why HBV is able to establish chronic infections. HDV, with its RNA genome, is more readily detected by host PRRs, but the virus has evolved mechanisms to evade immune detection by modulating the host's antiviral responses [3-5].

Conclusion

The immune response to HBV and HDV infections is complex and involves both intrinsic immune responses in infected cells and the activation of various innate immune cells. The interplay between viral evasion strategies and host immune responses is a major factor in the progression to chronic infection and liver disease. Although the innate immune system plays an essential role in controlling viral replication, HBV and HDV have evolved mechanisms to subvert this response, leading to persistent infections and liver damage. Understanding these immune responses and the strategies these viruses use to evade detection is crucial for developing more effective therapies and vaccines for HBV and HDV infections.

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Conflict of Interest

None.

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