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HBV/HDV-infected Cells' Intrinsic Immune Response and Associated Innate Immune Cell Activation

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Introduction

Hepatitis B virus and Hepatitis D virus are two major pathogens responsible for liver diseases, ranging from acute hepatitis to chronic liver conditions, including cirrhosis and hepatocellular carcinoma. HBV, a DNA virus, primarily infects hepatocytes, while HDV, a defective RNA virus, requires HBV for replication. Infected cells are the primary targets for both the adaptive and innate immune responses, but the ability of these viruses to modulate and evade host immune detection complicates their pathogenesis. The interplay between viral infections and the host's immune system is central to disease progression and outcome. This article explores the intrinsic immune responses of HBV/HDV-infected cells, their interactions with innate immune mechanisms, and how this affects the activation of innate immune cells. HBV is a highly infectious virus that primarily infects the liver. The viral genome is circular and partially double-stranded DNA, and its replication cycle involves reverse transcription. HBV infection can result in chronic or acute disease, with chronic infection carrying the risk of cirrhosis, liver failure, and hepatocellular carcinoma. The immune system's failure to clear HBV in chronic infection is linked to immune tolerance or dysfunction, allowing persistent viral replication. HDV is a satellite virus that cannot replicate independently and requires co-infection with HBV for its lifecycle. HDV is an RNA virus that encodes a single protein, the hepatitis delta antigen which is essential for the replication of the viral genome. HDV infections typically lead to more severe liver diseases than HBV alone, as the virus accelerates the progression of cirrhosis and is associated with a higher risk of liver failure and HCC [1,2].

Description

Upon recognition of viral PAMPs, infected cells initiate an innate immune response by producing type I interferons. These interferons are potent antiviral cytokines that act in an autocrine and paracrine manner to inhibit viral replication and modulate immune responses. Chronic HBV infection is characterized by the failure to mount an effective interferon response, which may be due to several factors, including immune exhaustion, viral immune evasion strategies, and the suppression of PRR signaling. For HDV, the presence of HBV can further dampen the interferon response, contributing to the chronicity and more severe outcomes of HDV co-infection. Dendritic cells are pivotal in the initiation of adaptive immune responses, as they process and present viral antigens to T cells. In HBV and HDV infections, dendritic cells can capture viral particles and activate T cell responses, but their function is often impaired in chronic infections. In chronic HBV infection, DCs may present viral antigens in a way that induces tolerance rather than activation, potentially contributing to the persistence of the infection. Moreover, HBV has been shown to alter the maturation of DCs, impairing their ability to activate T cells effectively. The HBV genome remains largely episomal, making it less detectable by cytoplasmic sensors like RIG-I. Furthermore, HBV's ability to produce large amounts of viral particles that do not contain viral DNA allows it to evade immune detection. Additionally, the HBV core protein inhibits the activation of interferon responses by binding to key components of the innate immune signaling machinery. However, it has mechanisms to suppress these responses, including interference with RIG-I activation and the inhibition of interferon production in infected cells. Moreover, HDV's reliance on HBV for replication means that some immune responses may be dampened due to the immune tolerance induced by HBV [3-5].

Conclusion

HBV and HDV infections trigger complex intrinsic immune responses in hepatocytes, involving multiple pattern recognition receptors and signaling pathways. However, both viruses have evolved mechanisms to evade detection and dampen the host's immune response, leading to chronic infections and liver damage. Innate immune cells, such as NK cells, dendritic cells, macrophages, and neutrophils, are critical for controlling HBV and HDV infections, yet they too are often functionally impaired in chronic infections. Understanding the interplay between HBV/HDV-infected cells and the innate immune system is crucial for developing more effective therapeutic strategies to combat these infections. Strategies aimed at boosting the innate immune response, restoring NK and dendritic cell function, or overcoming viral immune evasion could hold the key to achieving better outcomes for patients with chronic HBV and HDV infections.

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Conflict of Interest

None.

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