



Dendritic cells in chronic viral hepatitis: Did they loose or can they help to win the battle?

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Hepatitis B virus (HBV) and hepatitis C virus (HCV) can cause chronic liver disease and may elicit progressive liver injury leading to increased risk of developing liver cirrhosis, liver failure and liver cancer. Worldwide more than 500 million people are chronically infected with HBV and/or HCV. Accumulating evidence that inadequate immune responses to HBV and HCV are responsible for viral persistence has led to the search for immune escape strategies of these viruses. Given the central role of dendritic cells (DC) in initiating and shaping anti-viral T cell responses, it is important to understand how these cells interact with hepatitis viruses and their products, and what the functional consequences are of this interaction. This will help to understand the lack of strong anti-viral immunity as observed in patients chronically infected with HBV or HCV, and may eventually aid in developing effective anti-viral treatment leading to immune control over the viruses.

Our studies aim to understand the functional consequence of virus-DC interaction, especially HBV. To obtain insight into the interaction of HBV with DC subsets and the functional consequences, we analyzed liver tissue and peripheral blood of HBV-infected patients. Patient-derived DC subsets displayed an impaired function compared to DC present in healthy controls. Although HBV seems unable to infect DC, we demonstrated that DC from chronic HBV patients contain HBV-DNA and HBV proteins. In vitro studies from our group revealed that HBV can alter the function of myeloid and plasmacytoid DC. I will summarize our findings, provide information on the possible underlying molecular mechanisms, and discuss the possibility to use DC as tools or targets to treat chronic infections.