Innate and Adaptive Immunity in Hepatitis B

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Hepatitis B virus (HBV) is a largely non-cytopathic DNA virus with liver disease pathogenesis that is believed to be mediated by host innate and adaptive immune responses. HBV can display stealth characteristics with only limited innate immune activation (e.g. type I IFNs) during initial infection, although innate immune components including NK, NKT and platelets may contribute to HBV pathogenesis. T cells play a critical role in HBV clearance: CD8 T cells directly eliminate the virus-infected hepatocytes via cytopathic and non-cytopathic mechanisms; CD4 T cells regulate the overall antiviral adaptive immune response. B cell-mediated neutralizing antibody response to viral envelope limits viral spread and provides protective immunity against re-infection. In patients with chronic HBV infection, the antiviral effector T cell response is dampened by various immune regulatory or tolerance mechanisms (e.g. PD-1, Tregs). The balance between the antiviral immune effector and regulatory responses may define the level of virus control, liver inflammation and disease pathogenesis. This presentation highlights the host immune effector and regulatory response that may be relevant in the natural history, therapeutic outcome and disease pathogenesis in HBV-infected patients.