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Hepatocellular carcinoma: The role of host immunity in the regulation of proliferative responses

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Hepatocellular carcinoma (HCC) is the most common primary liver malignancy, with over 600,000 cases annually worldwide. Cirrhosis of the liver is a major driver in the pathogenesis of HCC in addition to direct proliferative stimuli from hepatitis viruses. Liver cirrhosis is pivotal to HCC development and associated long term impact of chronic inflammatory states that it stimulates in the liver influences the host environment by a perpetual activation of inflammatory pathways predisposes in some cases to dysregulation of cell proliferation, differentiation and survival mechanisms, culminating in malignancy. Recently, novel therapies that target PD1 or PDL1 and help to up regulate MHC-1 assisted markers on the malignant cells has shown exceptional promise for various malignancies including HCC. The basis of its success in HCC may depend on how these drugs are affected by the cirrhotic state compared to non-cirrhotic conditions. Learning how survival/proliferative and immune signaling pathways communicate in the background of cirrhosis compared with other conditions will give new insights into the impact of these pathways on the development and progression of HCC as well as influence the future of HCC therapeutic development. In our discussion we will: Provide an overview of the major etiologic factors associated with the development of HCC; review the major biological and molecular pathways that have been shown to be important in the pathogenesis of HCC and review the current therapies that are in use or in study for treatment; review the data which demonstrates the impact of host inflammation on the pathogenesis of HCC in the absence versus presence of liver cirrhosis and; summarize the widely used systemic therapies in HCC and refractory HCC and highlight the more promising/actively studied therapies that show reasonable promise in improving the outcomes of patients with advanced and or refractory HCC.

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