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IMPACT OF HEPATITIS C VIRUS: UPDATE ON PATHOGENESIS OF COMPLICATIONS AND TREATMENT STRATEGIES

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Hepatitis C (HCV) is a global infection due to *Hepacivirus*, a member of the Flaviviridae family. Parenteral routes including blood transfusion, injecting drug and exposure to medical procedures usually transmit infection; however, the virus can be transmitted maternally and in some patients, no known risk factor is identifiable. Over 170 million persons worldwide are infected and chronic infection leads to cirrhosis, hepatocellular carcinoma and increased all-cause mortality. The majority of infected patients maintain a chronic infection leading to several hepatic and non-hepatic complications. Linked to progressive infection is the presence of significant fibrosis within the liver and several risk factors predict which patients are likely to develop complications such as cirrhosis, hepatocellular carcinoma (HCC), and increased all-cause mortality. HCC is the fifth most common cancer in males, seventh in females and is a major cause of cancer related death. Eighty-five percent of cases occur in the developing world and HCV is a leading predisposition. The pathogenesis of HCV related HCC is complex and unlike hepatitis B virus, HCV does not integrate into the host genome. However, HCV does dysregulate cellular proliferation and differentiation pathways, creates chronic inflammation and inhibits tumor suppressor gene activity. Our laboratory has focused on two aspects of HCC carcinogenesis, namely cancer like stem cells and chronic inflammation. Hepatoma cells expressing a HCV subgenomic replicon express several cancers like stem cell markers, especially doublecortin-like kinase (DCLK1), a microtubule kinase that is a putative marker for intestinal and pancreatic cancers. Expression of DCLK-1 is linked to HCV replication and tumorigenesis in xenograft models, and DCLK-1 is identifiable in tissue and plasma derived from patients with HCV associated cirrhosis and HCC. SiRNA knockdown of DCLK-1 inhibits tumor growth in animal models suggesting that DCKL-1 might be a therapeutic marker. Additionally, total RNA analysis of FCA4 cells, which also express a HCV subgenomic replicon, reveals upregulation of DCLK-1 and a number of pro-inflammatory markers including S100A9 and SMARCA. These cells generate tumors in xenograft models that express DCLK-1, AFP and S100A9 and siRNA knockdown of DCLK-1 abrogates tumorigenesis and S100A9 expression. Over the last several years, there has been significant progress in the treatment of HCV infection. New, pan-genotypic direct antiviral agents (DAA) have vastly improved our treatment strategies not only achieving cure in a large percentage of patients, but also showing promise in reducing the complications of HCV infection.

Biography

Michael S. Bronze, M.D. was appointed Professor and Chairman, Department of Medicine, University of Oklahoma Health Sciences Center effective July 1, 2000. He was named the Stewart G. Wolf Professor in Internal Medicine in 2004 and David Ross Boyd Professor in 2011. He is board certified in Internal Medicine and Infectious Diseases. He completed his medical school training at the University of Tennessee, Memphis in 1982. His internship and residency were completed at the University of Tennessee, Memphis and served and additional year as Chief Medical Resident.

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