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Protein found that slows hepatitis C growth in liver cells

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Biomedical researchers have identified a cellular protein that interferes with hepatitis C virus replication, a finding that ultimately may help scientists develop new drugs to fight the virus.

The anti-hepatitis C activity of the protein, called “p21-activated kinase 1” (PAK1), was discovered by scientists at the University of Texas Medical Branch at Galveston (UTMB), who describe their findings in an article in the current issue of the Journal of Biological Chemistry. In addition to presenting the researchers’ discovery that PAK1 controls the rate at which hepatitis C virus replicates, the paper describes the biochemical pathways that lead to PAK1 activation and the specific mechanisms by which PAK1 interferes with the ability of hepatitis C to hijack liver cells and make more copies of itself.

“Our findings reveal a novel cellular control pathway that regulates the growth of hepatitis C virus within the cell,” said Dr. Stanley M. Lemon, director of the National Institutes of Health-funded Hepatitis C Research Center at UTMB and of the academic medical center’s Institute for Human Infections and Immunity. Lemon, senior author of the Journal of Biological Chemistry paper, added, “Understanding this better is likely to suggest new approaches to therapy for this difficult to treat disease."

Hepatitis C chronically infects approximately 170 million people worldwide. The most effective treatment for the virus, interferon-based therapy, eradicates the virus less than 50 percent of the time and causes debilitating side effects. Those for whom that treatment fails are at high

risk for fatal cirrhosis or liver cancer. In the United States, about half of all liver cancer cases occur in people infected by hepatitis C virus.

The UTMB scientists reported their findings in the April 20 issue of the Journal of Biological Chemistry. Their article is entitled “P21-activated Kinase 1 Is Activated through the Mammalian Target of Rapamycin/p70 56 Kinase Pathway and Regulates the Replication of Hepatitis C Virus in Human Hepatoma Cells.”

Source: University of Texas Medical Branch at Galveston