Result: The human pancreas cDNA library was constructed successfully. The reconstructed bait plasmid (pGBKTT-HCV NS4B) was transformed into yeast cells AH109 successfully. Eight proteins interacting with HCV NS4B were found. Conclusion: Some of the eight pancreatic proteins may be related with metabolisms of glucose and lipid.

**PP-135** The efficiency of blood-borne HCV amplification in different three dimensional cell culture systems in vitro

Y. Qi1, Q.L. Jin1, X.Y. Wen1, J.Q. Niu1. 1Division of liver diseases, the first hospital of Jilin University, Changchun, China

Background: Immortalized human hepatocytes HuS-E/2 cells cultured in Hollow fiber (HF) support the blood-borne hepatitis C virus (HCVbb) infection in vitro. But several limits of the HF system stand in the way of its usage for HCV research. This study is to evaluate the efficiency of different three dimensional (3-D) culture systems for HCVbb amplification in vitro.

Methods: HuS-E/2 cells were cultured in different 3-D cell culture systems, HF, hydrocell plate (HP) and mebiol gel (MG). Serum and plasma from hepatitis C patients were used for infection of the cells. Plasma was pre-treated with Ca2+ containing solution for the infection. Cell proliferation was monitored with XTT assay. HCV amplification in 3-D cultured cells was measured with real time PCR.

Results: Stable cell proliferation was observed in both HF and MG system. HCVbb amplification in HuS-E/2 cells which were cultured in HF, HP and MG exhibited the similar patterns although the titer in cells of different systems is variational.

Conclusion: All three 3-D systems can support the proliferation of HCVbb. The selection of the system for research depends on other factors such as the objective of the research, culture condition and so on.

**PP-136** Antiviral therapy of patients with decompensated cirrhosis associated with hepatitis C virus infection

F.P. Ji1*, H. Deng1. 1Department of Infectious Disease, the Second Affiliated Hospital, College of Medicine, Xi’an Jiaotong University, China

Patients with hepatitis C virus (HCV)-related decompensated cirrhosis are associated with a poor prognosis. Although liver transplantation (LT) offers an effective treatment, HCV reinfection of the transplanted graft is a critical and almost inevitable complication with major influence on graft- and patient survival. Antiviral therapy of this patient population is difficult, the use of interferon and ribavirin might expose these patients to severe treatment-related side effects as a large proportion of them have pre-existing hematological cytopenias. However, antiviral treatment in patients with advanced liver cirrhosis is a potential option for two reasons: first, clearing or suppressing HCV before LT may reduce or eliminate the risk of recurrent hepatitis C in the transplanted liver and thereby improve survival; second, clearing HCV in cirrhotic patient may halt disease progression and avoid the need for transplantation. Based on AASLD and ESAL guidelines, antiviral therapy in this patient population is generally recommended, but indication, optimal timing, dose and duration of therapy are not clearly defined. In this article, the results obtained by antiviral regimens administered to HCV-related decompensated cirrhosis are discussed.

**PP-137** Suppression of HCV replication in hepatocytes through a selective induction of IRF7

Q.L. Jin1*, X.Y. Wen1, C. Yang1, J.Q. Niu1. 1Department of Hepatology, First Hospital, Jilin University, Changchun, China

Background: A high risk of chronicity is the major concern of HCV infection and chronic infection often leads to liver cirrhosis and hepatocellular carcinoma. Although proportion of patients achieving a sustained virological response has been increased by the introduction of combination therapy of pegylated-IFN-alpha and ribavirin, still half of the patients exhibits no response to the therapy. One of the mechanisms for the establishment of persistent infection of HCV is the escape from the host immune system. To eliminate HCV from the hepatocytes of the patients without possible cytotoxicity due to anover induction of host immunity, we generated a therapeutic construct, cMR3, by which type I IFN is selectively induced in HCV infected cells.

Methods: The cMR3 is composed of the N-terminal part of the interferon regulatoryfactor 7 (IRF7) possessing a dominant active function, the sequences specifically cleaved by HCV NS3/4A protease and an ER anchor, and exhibits apotent IFN inducing activity in HCV infected cells. After cleavage by the HCV protease, the processed cMR3 migrates into the nucleus and activates various IFN promoters including IFN-alpha6, IFNbeta, and IFN stimulated response element.

Results: The specific activation of the IFN promoters was observed in both HCV replicon cells and JFH1 virus infected cells upon introduction of the cMR3 but not in cells infected with JEVor DENV. Expression of viral protein and viral RNA replication were also impaired by the introduction of cMR3 into the HCV replicon cells.

Conclusion: These results suggest that the selective expression of type I IFN in the hepatocytes infected with HCV by the introduction of the cMR3 might be feasible to eliminate HCV from the chronic hepatitis C patients without liver damage.

**PP-138** CD44v6 expression in HCV-infected cells and the correlation with apoptosis resistance

X.Y. Wen1*, Q.L. Jin1, Y. Qi1, C. Yang1, J.Q. Niu1. 1Department of Hepatology, First Hospital, Jilin University, Changchun, China

Background: Hepatitis C virus (HCV) infects about 170 million people worldwide and is a major cause of chronic hepatitis, cirrhosis, and hepatocellular carcinoma (HCC). Several mechanisms have been proposed for the incidence of HCC, including apoptosis resistance. CD44, an integral cell membrane glycoprotein and adhesion molecule, plays a critical role in a number of important cellular function including activating anti-apoptosis signal pathway. The aim of this study were to investigate the expression of CD44v6 in liver cancer tissues and to determine its correlation with apoptosis resistance via upregulated activity of the PI3K/AKT pathway.

Methods: We examined the expression of CD44 in liver cancer tissues in patients with HCV-related HCC compared with in liver tissues in health controls by Western blot and the expression of CD44v6 in HCV replicon and JFH1-infected cells compared with in Huh7 by FACS. Furthermore, we investigated the apoptosis of CD44 knockdown HCV replicon and HCV-infected cells treated by ActD compared with that in negative control cells.

Results: HCV-related HCC liver cancer tissues expressed significantly higher CD44 protein levels than tissues in health controls. Significantly higher CD44v6 were observed in HCV