Methods: A model of HBV is described by: x, y, v, z which represent uninfected cells, infected cells, free virus, and CTL cells respectively. Here the infected cells were divided into two parts with one part having drug resistance and the other part having none. The model includes 11 parameters: \( i, d, b, a, p, k_1, k_2, u, c, b, t \). During treatment of AD, we chose \( \{ i, d, b, a, p, k_1, k_2, u, c, b, t \} = \{4.621e5, 6.9e-3, 7.08e-11, 6.9e-3, 1.5e-9, 1, 0.93, 0.08, 0.35, 2.251e-2, 10.6\} \). During treatment of ETV, we choose \( \{ k_2, u, t \} = \{0.99, 0.18, 1.6 \} \) with other parameters unchanged. Here the change of the three parameters can imply the difference of therapy effect of AD and ETV.

Results: The simulation data of our model are qualitatively agreement with the clinical ones, especially the HBV DNA decrease when switch to ETV treatment.

Conclusion: The results show that our time-delay therapy model may possibly capture the dynamics of HBV infection and anti-HBV infection treatment under different drug.

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**PP-103** Immune responses elicited in hepatitis B virus transgenic mice by B7-H1 and HBsAg vaccine

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Background: The prevalence and infection hepatitis B virus is a serious problem in the world. Recent studies showed that the PD-1/PD-L pathway contributed directly to T cell dysfunction and lack of viral control in established chronic infection. In the present study, we try to improve the immune effect of HBsAg vaccination in HBV transgenic mice by blocking PD-1/PD-L pathway in vivo and to seek new methods for the treatment of chronic hepatitis B.

Methods: Fifteen HBV Tg mice were randomized into three groups with 5 mice each, and designated as HBsAg vaccine group, vaccine and B7-H1 group and control group, respectively. The anti-HBs of the three groups were detected by ELISA. The splenocytes of the mice were stimulated with HBsAg (10μg/ml). The concentrations of IL-2 and IFN-γ were detected by ELISA. The number of IFN-γ secreting cells was measured by ELISPOT.

Results: The anti-B7-H1 in serum was detected in the mice immunized with B7-H1 and the antibody titer increased with the immune times. There was no difference of anti-HBs positive rate between HBsAg group and HBsAg+B7-H1 group at 6th week and 8th week after immunization. Concentrations of IL-2 and IFN-γ were elevated in both HBsAg group and HBsAg+B7-H1 group than that in control group (\( P < 0.05 \)). Moreover, The numbers of HBs-specific IFN-γ secreting T cells is also increased in both the HBsAg group and HBsAg+B7-H1 group when compared with the control group, and the outcomes showed no difference between the two experimental groups (\( P > 0.05 \)).

Conclusion: (1) HBsAg vaccine can increase the cellular and humoral immune response in HBV Tg mice and reconstruct immune response to HBsAg. (2) B7-H1 vaccine can not enhance the humoral and cell immune responses in HBV Tg mice immunized with HBsAg.

**PP-104** Analysis of prognostic factors in patients with HBV-related liver failure and construction of a prognostic model to predict patient survival

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Objective: The aim of this study was to analyze variables that may affect survival of patients with HBV-related liver failure, identify the independent predictors for development of a prognostic survival model, and evaluate its predictive power.

Methods: In this retrospective cohort study, 569 patients were diagnosed with HBV-related liver failure between January 2004 and December 2007. Univariate and multivariate Cox proportional hazards regression analyses were applied to such variables as age, sex, disease complications, biochemical markers, coagulation markers, and HBV DNA loads to identify independent risk factors for establishment of a prognostic survival model, and 79 confirmed cases of HBV-related liver failure were used to evaluate the model’s predictive capacity.

Results: Hepatic encephalopathy, pulmonary infection, upper gastrointestinal bleeding (UGIB), albumin, AST, creatinine, international normalized ratio (INR) were determined to be independent risk factors (\( P < 0.01 \)); accordingly, the prognostic index (PI) = \( 4.98 \times \text{hepatic encephalopathy} + 4.57 \times \text{pulmonary infection} + 4.41 \times \text{upper gastrointestinal bleeding} - 9.69 \times \text{Ln(Alb}[\text{g/L}]) + 2.46 \times \text{Ln(ALT}[\text{U/L}]) + 5.18 \times \text{Ln(Cr}[\text{mmol/L}]) + 3.35 \times \text{Ln(INR)} - 15.36 \), which was able to accurately predict the 90-d survival.

Conclusions: Survival of patients with HBV-related liver failure can be accurately predicted based on seven independent predictors: hepatic encephalopathy, pulmonary infection, upper gastrointestinal bleeding (UGIB), albumin, AST, creatinine, and INR. Thus, these factors might be of reference value in the choice of clinical treatment.

**PP-105** miR-338–3p is down-regulated by the wild and mutant HBxs and inhibits proliferation by targeting CyclinD1

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Background: Hepatocellular carcinoma (HCC) is one of the most common malignancies. Numerous studies have suggested the potentially oncogenic roles of wild or mutant hepatitis B virus X protein (HBx) in hepatocarcinogenesis. To date, however, it remains unknown if HBx regulates the expression of microRNAs (miRNAs) which negatively regulate gene expression, playing essential roles in cell differentiation and carcinogenesis.

Methods: MiRNA microarrays were employed to compare the expression of cellular miRNAs in the LO2 cell line that have been successfully established transfecting with recombinant plasmid pcDNA3.0/HBx-d382, pcDNA/HBx and plasmid pcDNA3.0 previously. Real-time Quantitative PCR was used to confirm the chip results. MiR-338–3p was functionally characterized in LO2 cells with transiently altered miR-338–3p expression. The direct target of miR-338–3p was validated using 3’UTR-reporter assay.

Results: Compared with the LO2/pC DNA3.0 cell, a total of 1 miRNAs exhibited higher expression and 5 miRNAs demonstrated lower expression in the LO2/HBx-d382 cell, while 4 up-regulated and 11 down-regulated miRNAs were observed in LO2/HBx cell. MiR-338–3p, which both down-
regulated by wild and mutant HBx, demonstrated a lower level in the LO2/HBx-d382 cell. Further characterization of miR-338-3p revealed that it negatively regulated cellular proliferation. Cell cycle analysis showed that miR-338-3p induced cell cycle arrest at the G0/G1 phase. A dual-luciferase reporter assay demonstrated that the 3’UTR of CyclinD1 were directly bound to miR-338-3p and western blotting analysis further indicated that miR-338-3p down-regulated the expression of CyclinD1.

Conclusion: This study demonstrates that HBx can influence cellular miRNA expression. The deregulation of the expression of miR-338-3p by HBx may represent a potential novel pathway which HBx acts to deregulate cell proliferation leading to hepatocarcinogenesis.

**PP-106** Relationship between HBsAg, HBCAg expression and serum HBV DNA level in 140 patients with chronic hepatitis B

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Objective: The aim of this study was to investigate the relationship between HBsAg, HBCAg expression and serum HBV DNA level.

Methods: The expression of HBsAg, HBCAg in the livers of 140 patients with chronic hepatitis B was detected by immunohistochemistry. The level of serum hepatitis B virus DNA (HBV DNA) was tested. Statistical significance was assessed using One-Way analysis of variance (ANOVA).

Results: Serum HBV DNA level in 13 patients with HBsAg (− −), 108 patients with (++++) and 19 patients with (++++++) was 5.313±1.874 copies log10/ml, 6.010±0.216 copies log10/ml and 5.664±1.548 copies log10/ml respectively (P=0.408). Serum HBV DNA level in 42 patients with HBsAg (− −), 79 patients with (++) and 19 patients with (++++++) was 5.886±1.997 copies log10/ml, 5.968±2.020 copies log10/ml and 5.634±1.551 copies log10/ml respectively (P=0.800).

Conclusions: The expression of HBsAg, HBCAg in the liver does not correlate with serum HBV DNA level.

**PP-107** Cause analysis of chronic HBV infected patients without antiviral therapy in the Pearl River Delta region

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Objectives: To analyze the causes of the patients with chronic Hepatitis B without taking antiviral therapy and the strategies of dealing with it.

Method: We make long-term observation on the patients with chronic Hepatitis B Virus (HBV) infection, who were voluntarily to be followed up in our clinic department, and analyze statistically the objective and subjective causes of patient without taking antiviral therapy.

Results: In total eligible 951 cases, 424 cases didn’t receive the antiviral therapy. 105 out of 424 cases had the indications of the antiviral therapy (105/424, 24.8%), the other 319 cases had no indications of the therapy (319/424, 75.2%). The ratio of female (124/202, 61.4%) who didn’t get the antiviral therapy was significant higher than that of male (300/749, 40.0%). 49 out of 105 cases who had the indications of the antiviral therapy worried about the unhealthful effect on their fertility by the antiviral drugs and put off antiviral therapy (49/105, 46.7%); 31 out of 105 cases could not pay for the antiviral therapy (31/105, 29.5%); 19 out of 105 cases queried the safety of the antiviral drugs and uncertainty of course of the treatment (19/105, 18.1%). 6 out of 105 cases were because of poor compliance (6/105, 5.7%).

Conclusions: No antiviral indications was the main cause of the untreated group. The causes of that patients with indications didn’t receive antiviral therapy were that worrying about their fertility, fees of the treatment hard to bear, querying the safety of the antiviral drugs and uncertainty of course of the treatment, and poor compliance. Formal long-term follow up by the clinicians, good communications between clinicians and patients and health education might improve the effects of anti-HBV treatment.

**PP-108** Investigation on serum HBV viral loads and the changes of liver pathological features in 158 patients with chronic hepatitis B

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Objective: To investigate the relationship between serum HBV DNA loads and liver pathological changes in the patients with chronic hepatitis B.

Methods: The relationship among HBV DNA loads, live histological inflammation grades and fibrosis stages of 158 cases was analyzed.

Results: The serum HBV DNA loads in HBeAg-positive group with inflammation grades G0 (6 patients), G1 (74 patients) and G3,4 (25 patients) were 5.580±1.098 copies log10/ml, 6.520±2.004 copies log10/ml and 6.950±1.467 copies log10/ml respectively. There was no significant difference in patients of three inflammation grades (P=0.250). The serum HBV DNA loads in HBeAg-positive group with liver tissues fibrosis stages of S0,1 (23 patients), S2 (56 patients), S3,4 (26 patients) were 6.599±1.832 copies log10/ml, 6.559±2.012 copies log10/ml, 6.562±1.601 copies log10/ml respectively, the difference was not significant (P=0.996). The serum HBV DNA loads in HBeAg-negative group with inflammation grades G0,1 (8 patients), G2 (17 patients) and G3,4 (28 patients) were 2.132±1.875 copies log10/ml, 4.745±2.250 copies log10/ml and 5.581±2.305 copies log10/ml respectively. The serum HBV DNA level in patients with G2 and G3,4, inflammation grades was significant higher than in patients with G0,1 inflammation grades (P=0.001). The serum HBV DNA loads in HBeAg-negative group with liver tissues fibrosis stages of S0,1 (10 patients), S2 (45 patients), S3,4 (18 patients) were 2.689±3.225 copies log10/ml, 5.127±1.833 copies log10/ml, 5.375±2.410 copies log10/ml respectively. The serum HBV DNA level in patients with fibrosis stages of S2 and S3,4 was significant higher than in patients with fibrosis stages of S0,1 (P=0.005).

Conclusions: The serum HBV DNA level does not correlate with the inflammation grades and fibrosis stages of liver tissues in HBeAg-positive patients. The serum HBV DNA loads display a positive correlation with the inflammation grades and fibrosis stages of liver tissues in HBeAg-negative patients.

**PP-109** Association of IL-6 gene polymorphism and its levels in HBV related hepatocellular carcinoma progression in India

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Objectives: Hepatitis B Virus (HBV) infection is a primary risk factor for hepatocellular carcinoma (HCC), the fifth most frequent cancer, worldwide. The present study was undertaken to analyze the association of IL-6 (~572) and...