Concurrent Sessions

**Concurrent Session 1 – Management of Hepatitis B**

**I-14** Treatment of hepatitis B virus  
M. Omata*. Department of Gastroenterology, University of Tokyo, Japan

HBV was discovered in 1964 and HBV suppression by treatment started in late 1970 by Dr. Greenberg using Interferon. However, until 1998 when Dr. CL Lai reported one year experience of Lamivudine in N Engl J Med, the treatment of HBV has not become common practice. That time, I wrote critical comment on this article by Editorial in N Engl J Med (1998; 339: 114–5). Because the majority of the patients treated had infection more than thirty years or sometimes even fifty years. And only one year suppression is not obviously sufficient to change the natural course of HBV infection. Subsequently, very high resistance rate was noted in the Lamivudine treatment and long-term efficacy was blurred.

And now, experiences of three to five years of drug use were gathered and several drugs seem so far very safe and potential suppressor of HBV replication for long time. Now the question is raised how long it can continue to suppress HBV before the emergence of HBV resistant mutant. In my talk, I will compare the potency and durability of several drugs and emergency of resistant strains. In addition, I will address the validations of new APASL guideline by our own patients’ data and on the Japanese experiences of the improvement of liver history, treated by nucleoside analogues for 3 years. We know envision the use of these drugs for longer than five years. I personally feel if these drugs can suppress longer than ten years, probably the natural course of HBV infection could be drastically changed in the majority of HBV carriers.

**I-15** From the pathogenesis to the therapeutic strategy of chronic hepatitis B infection  
G.Q. Wang*. Department of Infectious Diseases & Center for Liver Diseases, Peking University First Hospital, Beijing 100034, China

Chronic HBV infection is a severe public health problem in the world, especially in China and Asia-Pacific Area. Although some new drugs developed in the treatment of chronic hepatitis B, such as pegylated interferon, lamivudine, adefovir, entecavir and tenbivudine, not all the patients benefit from them, especially the sustain response is not satisfied.

There are two main reasons for the HBV infection as an intractable disease. One is the HBV cccDNA, the template for the replication of virus, that no drugs have worked on it. Another is the immune tolerance or dysfunction of the host immune system on HBV. The final elimination of the HBV cccDNA depends upon the immune system, that is, the viral specific cytotoxic T lymphocyte (CTL) and cytokines contribute the sustain viral inhibition by inducing the apoptosis of the viral infected hepatocytes, and the exhaustion of the cccDNA reservoir by sustain inhibition of the viral replication. So, immune modulation treatment that can provoke the immune system will play a very important role in the control of HBV infection.

As the sustain HBV inhibition rely on the immune system, the immune modulatory agents should be considered as the main therapeutic strategy of chronic HBV infection. Unfortunately, only Interferon α including pegylated interferon will be used in the young patients with low level HBV DNA and elevated ALT, and Thymosin α1 can be one of candidate as a immunomodulatory agent. As the nucleos(t)ide acid has a strong potency of inhibiting the virus but no immunomodulatory effect, the combination with two type agents will be a potential strategy in the future. The development of new agents with immunomodulatory effect may change the outcome of chronic hepatitis B patients, but there is a long way to go.

**Concurrent Session 2 – Bacterial infection**

**I-16** Prevalence of dupA of Helicobacter pylori strains isolated from Shanghai patients and its clinical implications  
H. Lu*. Shanghai Renji Hospital affiliated to Shanghai Jiao Tong University School of Medicine, Shanghai Institution of Digestive Disease, Shanghai, China

Aims: To determine the prevalence of duodenal ulcer promoting (dupA) gene of Helicobacter pylori in patients with various gastroduodenal diseases in Shanghai and to explore the association between the gene and other putative virulence factors.

Methods: H. pylori were isolated from gastric biopsies of patients with chronic gastritis, duodenal ulcer (DU), gastric ulcer (GU), or non-cardia gastric carcinoma. The dupA, cagA, vacA, iceA and babA2 genotypes were determined by polymerase chain reaction. Histological features of gastric mucosal biopsy specimens were graded based on the scoring system proposed by the updated Sydney system.

Results: Isolates from 360 patients including 133 with chronic gastritis, 101 with DU, 47 with GU, and 79 with non-cardia gastric carcinoma were examined. The dupA gene was detected in 35.3% (127/360) and the prevalence DU patients was significantly greater than that in gastric cancer or GU patients (45.5% vs. 24.1% and 23.4%, P<0.05). Patients infected with dupA-positive strains had