**Concurrent Session 15: Detection and Clinical Significance of Viral Infection**

**Sunday, July 16, 2011, 11:15–12:45**

**Meeting Room 310**

**CS15.1** CMV in transplant recipients  
B.-H. Tan*. Department of Internal Medicine, Singapore General Hospital, Singapore  
CMV has long been described as the Achilles heel of organ transplantation. CMV may produce direct effects such as fever, and end-organ damage, such as retinitis and colitis. CMV is also thought to produce indirect effects such as rejection, diabetes, and perhaps even graft atherosclerosis (in heart transplant recipients). Advances in viral diagnostics, plus the availability of ganciclovir and valganciclovir, however, have made CMV less fearsome than it used to be. In this lecture, we will review the data on both the prophylactic and pre-emptive modes of CMV prevention in organ transplant recipients, and ask ourselves if the technology that is now enabling us to measure host immunity to CMV has a role in clinical management.

**CS15.2** Drug design and development against EV71 and other enteroviruses  
T.W. Lin*. State Key Laboratory on Stress Cell Biology, School of Life Sciences, Xiamen University, China  
Enteroviruses are a family of single stranded, positive sense RNA virus of Picornaviridae. Although most of the enterovirus-associated diseases are mild and asymptomatic, some member in the family can cause severe diseases and death, especially in the young and immunocompromised. Enteroviruses are the leading cause of aseptic meningitis which in turn is the most common central nervous system infection. Enterovirus 71 (EV71), for example, is an important pathogen besides polioviruses of the family. It is emerging as the most significant neurotropic enterovirus in some area of the world in outbreaks and epidemics. This virus circulates in US, and 26% of the adults tested in a study had antibody. EV71 was the leading cause of infectious diseases in China in 2010. The outbreaks of EV71-associated diseases have been reported in the United States, Australia, Sweden, Japan, Bulgaria, Hungary, Malaysia, and other countries. It has been associated with a variety of clinical diseases, including hand, foot and mouth disease, herpangina, aseptic meningitis, encephalitis, poliomylitis-like paralysis, and even fatal pulmonary edema or hemorrhage. Enterovirus-associated disease can be both acute and chronic. The chronic diseases include dermatomyositis, polymyositis, dilated cardiomyopathy, and diabetes mellitus. There is an urgent need to develop therapeutics against EV71 in particular, and enteroviruses in general.

Upon infection, a polyprotein is translated from the single open reading in the genome of an enterovirus, which is processed into mature proteins by virally encoded proteinases. These proteinases are not only vital to the propagation of the virus but important factors in limiting host defense against the virus infection as well. We carry out structure-based screening, design, and development of inhibitors against the enterovirus infection using the proteinases as the targets. The lead compounds were generated after hits were identified by virtual screening, structure characterization, and medicinal chemistry. The lead compounds were improved by iterations of structure-based design, chemical synthesis, and functional assay. The resulting inhibitors are shown to be capable of inhibiting virus replication and restore host functions.

**Concurrent Session 16: End Stage Liver Disease and Complications**

**Sunday, July 16, 2011, 11:15–12:45**

**Meeting Room 311A**

**CS16.1** Management of the complications of liver cirrhosis  
H. You*. Liver Research Center, Beijing Friendship Hospital, Capital Medical University, Beijing, China  
Abstract not available

**CS16.2** Improving survival in patients with decompensated cirrhosis  
D.N. Amarapurkar*. Department of Gastroenterology and Hepatology, Bombay Hospital & Medical Research Centre, Mumbai, India  
Liver cirrhosis is defined as development of regenerative nodules surrounded by fibrous septa in response to chronic liver injury. This leads to vascular remodeling and giving rise to portal hypertension and end stage liver disease. Liver transplantation is the only treatment which improves both longevity and quality of life in patients with decompensated liver cirrhosis. However every patient with decompensated liver cirrhosis is not eligible for transplantation and it is not available for majority of the patients. Our current understanding of natural history, pathophysiology and treatment of complication has resulted in improved management quality of life and life expectancy in patients with decompensated liver cirrhosis. Median survival of patients with compensated cirrhosis is 12 years while that of decompensated patients is reduced to 2 years. Approximately 5 to 7% of the patients change from compensated stage to decompensated stage. Portal hypertension (PH) is a universal consequence of cirrhosis responsible for most of the complications like esophagogastric varices, variceal bleeding, ascites, spontaneous bacterial peritonitis, heporenal syndrome and hepatic encephalopathy. PH in cirrhosis is defined by hepatic venous pressure gradient (HVPG) more than 5mm of mercury. HVPG is indirect measure of portal pressure. Now it is clear that HVPG more