**PP-128** Polysaccharopeptide stimulated human peripheral blood mononuclear cells activation and promoted anti-hepatitis B virus effect in vitro

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Background: The mechanism of anti-hepatitis B virus effects of PSP is unclear.

Methods: Human peripheral blood mononuclear Cells (PBMCs) obtained from healthy volunteer were co-cultured with PSP. The ratio of CD4+/CD8+ and the expression of CD3+/HLA-DR+ and CD19+/CD69+ of PBMCs were assessed by flow cytometry at 72 hours, meanwhile, detecting the levels of IFN-γ, IL-12 and IgG in supernatant by ELISA. Then HepG2.2.15 cells were divided into four groups (Table 1). HBVDNA load and the levels of HBsAg and HBeAg in supernatant were detected by fluorescent-quantitative PCR and Abbott-chemiluminescent microparticle immunoassay respectively at 96 h.

Results: At 72h, the ratio of CD4+/CD8+ in PBMCs+PSP group was significantly higher than PBMCs group (p<0.05), and the expression of CD3+/HLA-DR+ and CD19+/CD69+ in PBMCs+PSP group were higher than PBMCs group, however, have no statistical significance. PSP could increase IFN-γ, IL-12 and IgG (Graph 1). PSP couldn’t inhibit HBV replication directly; PBMCs activated by PSP reduced HBsAg and HBeAg secretion significantly, however, have no remarkable inhibitory effects of HBVDNA (Table 1).

Conclusion: The PSP could activate T and B lymphocytes and increased IFN-γ and IL-12 levels of PBMCs, The effect of HBsAg and HBeAg inhibition owe to the immune response of PBMCs activated by PSP.

**PP-129** Clinical picture of acute viral hepatitis A in Mongolian children with HBsAg

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Background: HBV infection is endemic in Mongolia. So, Mongolian Government launched HBV vaccination as mandatory immunization since 1991. After that, the incidence acute viral hepatitis and mortality from acute liver failure were dramatically decreased. But, it was not stopped concurrent acute viral hepatitis A and B in practice.

Purpose of study: To study the clinical picture of acute viral hepatitis A in children with HBsAg.

Material and Method: In this study, we selected 130 children (age 1–15; male 84) with acute icteric viral hepatitis, whom positive detected HBsAg. In 102 children detected only anti-HA-igm (1st group), in 8 also anti-HAV-igm and anti-Hbc-igm (2nd group) in 20 detected anti-Hbc-igm but not anti-HAV-igm (3rd group). Study was conducted at Communicable Diseases Centre of Mongolia in 2005–2009.

Results: In children for 1st group were severe form 7.8%; moderate severe form –23.5% cases, then in 2nd group were severe form 50.0%; moderate severe form were 12.5%; in 3rd group were fulminant form 25.0%, severe form 25.0% and moderate severe form were 15.0%; There was case of fulminant liver failure only in acute HBV infection. The course of clinic manifestations were depending on study groups shown in the table.

### Table 1. Effect of PBMCs activated by PSP on HBsAg, HBeAg and HBV DNA at 96 h (X±S)

<table>
<thead>
<tr>
<th>Treatment group</th>
<th>HBsAg level (IU/ml)</th>
<th>HBsAg inhibition rate</th>
<th>HBeAg level (S/CO)</th>
<th>HBeAg inhibition rate</th>
<th>HBV-DNA inhibition rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>(PBMCs co-cultured with PSP + HepG2.2.15 cells)</td>
<td>4.13±0.92**</td>
<td>50.63%**</td>
<td>24.89±13.67</td>
<td>35.05%**</td>
<td>-4.78%</td>
</tr>
<tr>
<td>Un-treated group (PBMCs cultured without PSP + HepG2.2.15 cells)</td>
<td>5.86±1.32*</td>
<td>29.40%*</td>
<td>33.29±19.29</td>
<td>39.26%*</td>
<td>-2.87%</td>
</tr>
<tr>
<td>PSP group (PBMCs cultured with PSP + HepG2.2.15 cells)</td>
<td>8.64±1.09</td>
<td>-2.88%</td>
<td>50.24±19.99</td>
<td>1.72%*</td>
<td>-12.28%</td>
</tr>
<tr>
<td>Control group (HepG2.2.15 cells)</td>
<td>8.41±0.66</td>
<td>-</td>
<td>51.67±21.51</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Prolongation of jaundice: 4 days 6 days 12 days
Prolongation of jaundice: 7 days 11 days 12 days
Normalization of GTP: 20.2 days 24.6 days 33.4 days

Conclusion: The course of acute icteric viral hepatitis A in children with HBsAg predominantly mild as HAV infection.

**PP-130** Development and application of Reverse Dot Blot (RDB) hybridization assay for HBV Pol/RT resistant mutation

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Objectives: The purpose of this study was to develop a method for detecting the mutations at YMDD by reverse dot blot (RDB) hybridization assay, which was specific, sensitive and simple.

Methods: Using LiP A technology principle, we designed a groups of capture probes with PolyT tail, fixed on the NC membrane and developed the method Reverse Dot Blot (RDB) hybridization assay. Sera were collected from patients with hepatitis B who were predicted to develop viral breakthrough during lamivudine treatment at infectious department of NanFang Hospital, between 2006 and 2007. For 134 samples with YMDD mutation detected by

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**Abstracts, 4th DICID**

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PP-130 Development and application of Reverse Dot Blot (RDB) hybridization assay for HBV Pol/RT resistant mutation