**BASL abstracts**

**Method** Null responders (<1-log_{10} HCV RNA decrease at week-4 or <2-log_{10} at week-12), partial responders (=2-log_{10} decrease at week 12, detectable at week 24), patients with viral breakthrough and relapsers from PROVE1/2/3 PR arms were eligible for treatment. Initially all patients received T 750 mg q8h plus PR at standard doses for 12 weeks, followed by 12 weeks of PR (T12/PR24). Protocol was amended to allow partial responders, viral breakthroughs and relapsers with undetectable HCV RNA at weeks 4 and 12 (eRVR) to receive T12/PR24. Partial responders, viral breakthroughs and relapsers with detectable HCV RNA at week 4 and/or week 12 and null responders received an additional 24 weeks of PR (T12/PR48).

**Results** Of 117 patients included in an ITT analysis, 97 (83%) had baseline HCV RNA=800 000 IU/mL, (69) 59% had genotype subtype 1a, 44 (38%) had cirrhosis or bridging fibrosis, and 9 (8%) were black. Viral breakthrough and relapse rates occurred in 25%, 23% of prior null responders; 10%, 22% of prior partial responders; 15%, 0% of prior viral breakthroughs; and 0%, 4% of prior relapsers.

**Conclusion** Patients with prior relapse, breakthrough and partial response exhibited high SVR rates after 24 weeks of telaprevir-based regimen. High SVR rates were also observed in patients with previous null response after 48 weeks of therapy.

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**Abstract P66 Table 1 Results: Patients achieving SVR**

<table>
<thead>
<tr>
<th></th>
<th>T12/PR24</th>
<th>T12/PR48</th>
<th>Unassigned</th>
</tr>
</thead>
<tbody>
<tr>
<td>All</td>
<td>47 (59)</td>
<td>18 (52)</td>
<td>2 (100)</td>
</tr>
<tr>
<td>Prior null responders: n/N, %</td>
<td>3/2 (13)</td>
<td>16/28 (57)</td>
<td></td>
</tr>
<tr>
<td>Prior partial responders: n/N, %</td>
<td>15/25 (60)</td>
<td>0/3 (0)</td>
<td>1/1 (100)</td>
</tr>
<tr>
<td>Prior relapsers: n/N, %</td>
<td>23/25 (92)</td>
<td>2/3 (67)</td>
<td>1/1 (100)</td>
</tr>
<tr>
<td>Prior viral breakthrough: n/N, %</td>
<td>6/7 (86)</td>
<td>0/1 (0)</td>
<td></td>
</tr>
</tbody>
</table>

*One prior partial responder and one prior relaper who discontinued all treatment prior to reaching week 12 of dosing were designated "unassigned" to treatment group.

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**P68 PACIFIC: A PHASE III, RANDOMISED, MULTICENTRE, DOSE ESCALATION, EFFICACY AND SAFETY STUDY EXAMINING THE EFFECTS OF TREATMENT WITH PEGLATERFERON ALFA-2A IN PATIENTS WITH CHILD'S A OR B CIRRHOSIS IN CHRONIC HEPATITIS C VIRUS INFECTION**

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**Introduction** Trials have found conflicting results about the efficacy of pegylated interferon α (PINFN2a), with or without pretreatment including ribavirin, as an antifibrotic agent in patients with established cirrhosis due to persistent HCV infection. We have investigated the use of an escalating dose of PINF2a monotherapy for 48 weeks in the treatment of patients with established cirrhosis due to persistent HCV infection.

**Method** A multicentre, randomised prospective controlled trial of escalating dose PINF2a treatment of patients with HCV infection and Child's A or B cirrhosis. 39 patients were enrolled at 5 UK centres and randomised to standard clinical care, or 48 weeks treatment with PINF2a at 90 mcg p.w. escalating each month by 45 mcg to 180 mcg p.w. if tolerated and followed for 140 weeks. Primary outcomes were liver related death; “liver related morbidity” including varical haemorrhage, ascites and SBP, hepatocellular cancer, transplantation and all cause mortality. Secondary outcomes were health related quality of life (HRQL).

**Results** There was no significant difference in the baseline characteristics between treatment and control groups (male 71.77%; mean age 55.25;1; Child’s score 5.5:5.5:5.5; MELD 8:23:9:79). Treatment was well tolerated. 15/17 (88%) completed 48 weeks treatment; 1 at 45 mcg; 1 at 90 mcg; 2 at 135 mcg; 11 at 180 mcg.

There were no differences between groups in HAPOL except pain scores that were increased in the treatment group (Score=5.7:7.8:5.6; p<0.01). Recruitment to the study was halted by the DSCM on publication of HALT-C and EPIC trial results.

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**P67 COMBINED INNATE AND ADAPTIVE IMMUNE RESPONSES ARE NEEDED TO CONTROL HEPATITIS B VIRUS REPLICATION IN CHILDREN WITH INFANCY-ACQUIRED INFECTION**

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**Introduction** Innate/adaptive immunity interplay is crucial to control hepatitis B virus (HBV) replication. In infancy-acquired chronic hepatitis B, low viral load is associated with high numbers of natural killer cells (NKC), high expression of their activation markers/receptors (CD69, CD107a, CD161 and NKG2D) and possibly of their inhibitory receptor NKG2A, but there is no information on NKC receptors (CD69, CD107a, CD161 and NKG2D) and possibly of their expression. High expression of their activation markers/indicators of specific immune response were tested by IFN-g intracellular staining after PBMC incubation with HBV core antigen (HBCAg) and HBV DNA viral load by real-time PCR.

**Results** Frequency of NKC producing IFN-g only (CD107a+/IFN-g+), polyfunctional NKC (CD107a+/IFN-g+) and NKC expressing NKG2A or NKG2D only (CD107a-NKG2A/D+) was higher in group D than in groups A–C, both ex-vivo (%CD107a+/IFN-g+: 24.5, 7.3, 11.3, 16.3, p=0.03) (%CD107a+/IFN-g+: 25.5, 14.6, 18.6, 20.2, p=0.04) (%CD107a+/NKG2A+: 10.9, 5.1, 4.5, p=0.02) and (%CD107a+/NKG2D+: 27.3 vs 16.7, 20.2, 22.6, p=0.05) and post-K562 (CD107a+/IFN-g+: 31.5 vs 13.2, 16.4, 19.6, p=0.04) (%CD107a+/NKG2A+: 12.3 vs 3.7, 4.1, 5.6, p=0.05) and (%CD107a+/NKG2D+: 35.3 vs 20.1, 22.6, 23.4, p=0.05)). % NKG2D+CD107a+/NKC was higher in group D than in groups A–C ex-vivo (32.4 vs 17.1, 20.1, 22.4, p=0.05) and post-K562 (39.7 vs 17.9, 21.7, 26.2, p=0.04), while NKG2A+/CD107a+/NKC number was similar in all groups. % HBCAg-specific IFN-g producing cells was higher in group D than groups A–C (CD4+/IFN-g+: 7.2±2 vs 2.3±0.3, 2.7±0.5, 3.1±0.9, p=0.04). Polyfunctional NKC CD107a+/IFN-g+ number correlated with that of HBCAg-specific IFN-g producing cells (r=0.5, p=0.04) and negatively with HBV DNA viral load (r=−0.42, p=0.05).

**Conclusion** High numbers of NKC producing IFN-g, polyfunctional, and with high NKG2D expression are associated with low HBV DNA replication. The strong correlations between polyfunctional NKC and HBV-specific T-helper 1 cells and HBV DNA viral load indicate a joint action between innate and adaptive immunity in controlling HBV infection.