due to anaemia occurred in 3.3%, 0.8% and 0.6% in T3PR, T12PR and control arms, respectively. 

**Conclusion** A significantly greater proportion of patients achieved SVR with 12-week and 8-week telaprevir-based combination regimens (75% and 69%, respectively), compared with PR48 control arm (44%, p<0.0001). The safety and tolerability profile of telaprevir in the ADVANCE trial was consistent with the profile previously reported, with an improvement in treatment discontinuation rates due to adverse events, including rash and anaemia. These first Phase 3 results confirm the clinical benefit previously reported in Phase 2.

**Abstract P70 Table 1 Viral response**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Univariate OR (95% CI)</th>
<th>p Value</th>
<th>Multivariate OR (95% CI)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients achieving RVR, n (%)</td>
<td>242 (66)</td>
<td>246 (68)</td>
<td>34 (9)</td>
<td></td>
</tr>
<tr>
<td>Patients with HCV RNA undetectable at end of treatment (EOT), n (%)</td>
<td>295 (81)</td>
<td>314 (87)</td>
<td>229 (63)</td>
<td></td>
</tr>
<tr>
<td>Patients achieving SVR, n (%)</td>
<td>250 (69)*</td>
<td>271 (75)*</td>
<td>158 (44)</td>
<td></td>
</tr>
<tr>
<td>Difference in SVR rates, TRV arms vs control, % (95% CI)</td>
<td>25 (18 to 32)</td>
<td>31 (24 to 38)</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Patients with relapse, n (%)</td>
<td>28 (9)</td>
<td>27 (9)</td>
<td>64 (28)</td>
<td></td>
</tr>
</tbody>
</table>

*p<0.0001, Denominator is number of patients with HCV RNA undetectable at EOT.

**Abstract P71 Table 1 Cox regression hazard analysis of predictors of 3 months liver related deaths or re-LT**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Univariate OR (95% CI)</th>
<th>p Value</th>
<th>Multivariate OR (95% CI)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>D7 AST ≥500 IU/l</td>
<td>2.188 (1.162 to 4.121)</td>
<td>0.0159</td>
<td>1.807 (0.953 to 3.425)</td>
<td>0.071</td>
</tr>
<tr>
<td>D1 lactate &gt;3 mmol/l</td>
<td>2.500 (1.739 to 3.594)</td>
<td>&lt;0.0001</td>
<td>1.939 (1.400 to 2.872)</td>
<td>0.001</td>
</tr>
<tr>
<td>D7 bilirubin &gt;100 μmol/l</td>
<td>1.530 (1.149 to 2.036)</td>
<td>0.0037</td>
<td>1.469 (1.101 to 1.960)</td>
<td>0.0094</td>
</tr>
<tr>
<td>D7 on vasopressors</td>
<td>5.241 (3.143 to 8.739)</td>
<td>&lt;0.0001</td>
<td>4.067 (2.395 to 6.996)</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

**Conclusion** The new model is simple to use and significantly improved the sensitivity of detection of severe ELGD. Validation in another cohort of LT patients is warranted.

**Transplant**

**P71** A MODEL TO IMPROVE PERFORMANCE OF CURRENT CATEGORY 9 UK LISTING CRITERIA: EARLY LIVER DYSFUNCTION. A SINGLE CENTRE COHORT
doi:10.1136/gutjnl-2011-300857a.71

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**Introduction** Current super urgent criteria for listing for early liver graft dysfunction (ELGD) in the UK (category 9, C9C) is defined as fulfilling 2 out of 4 of the following criteria within 7 days post liver transplant (LT): AST >10000 IU/l, INR >3, Lactate >3 mmol/l and absence of bile production. We demonstrated that these criteria have critically low sensitivity in predicting early post LT death or need for re-LT (Al-Freah, et al. Hepatology 2009; 50 Suppl 4:A145).

**Aim** To develop an improved predictive model for early re-LT or death using early post-LT clinical parameters.

**Method** Retrospective study of all patients transplanted at our centre 1 January 2000 to 31 December 2008. Daily clinical and laboratory parameters for the first 7 days post LT were reviewed. These included AST, bilirubin, INR, lactate, vasopressor requirement and/or haemolisation.

**Results** Over the study period, 1286 patients underwent first LT at our centre. Patients excluded 28 because of re-LT for hepatic artery thrombosis (22), died on table (5) and one re-LT because of donor cancer. We analysed data on 1258 patients (median age 51 (16–74) years (16–74), 60% male). The most common aetiology was viral hepatitis in 303 patients (24%) and alcohol related liver disease in 227 patients (18%); 181 patients (14.4%) with hepatitis C, 158 (12.6%) with hepatitis B, with a 3 month liver related outcome met this criterion compared to 16 who met C9C. This gave sensitivity 68% (58–77%), specificity 67% (64–70%), LR+ 2.08 (1.77–2.45) and LR– 0.48 (0.36–0.63).

**P72** LIVER TRANSPLANTATION FOR FAMILIAL AMYLOIDOSIS: LONG-TERM DATA FROM THE FAMILIAL AMYLOID POLYNEUROPATHY WORLD TRANSPLANT REGISTRY (FAPWTR)
doi:10.1136/gutjnl-2011-300857a.72

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**Introduction** Liver transplantation (LT) is the only available treatment for familial amyloid polyneuropathy (FAP). The Familial Amyloid Polyneuropathy World Transplant Registry (FAPWTR), established shortly after LT was introduced as potential treatment for FAP in 1990, is a centralised service based in Karolinska Institute in Sweden for the collection, monitoring and analysis of international data on LT for FAP.

**Aim** We present here the long-term FAPWTR results on the 20 years anniversary of LT for FAP.

**Results** Between April 1990 and January 2010, data on 1782 liver transplant procedures and regular follow-up were reported to the FAP registry from 70 transplant centres in 18 countries. Annual international transplant activity for FAP has remained stable at 80–120 procedures since 1996. Among those 866 liver transplants were performed in Portugal, 216 in France, 150 in Sweden, followed by USA 79, UK 78, Brazil 77, Spain 74, Japan 65. The Mediterranean Val63Met transthyretin (TTR) mutation was identified in 85% of cases. A further 50 different variants were reported, collectively referred to as non-ValMet30, and additionally a dozen of non-TTR Val30Met variants. Median age at LT was 30 years). Of patients in the Val50Met group 98% received isolated LT, while 11% of non-Val30Met variants. Median age at LT was 38 years (range 21–72 years), 57% of patients were male. Median disease duration prior to transplantation was 5 years (range 0–30 years). Of patients in the Val50Met group 98% received isolated LT, while 11% of non-Val50Met cases required either simultaneous (9%) or sequential heart and liver transplant (2%). Overall 1-, 5-, 10- and 15-year survival after LT in the entire FAP population including all variants was 86.9%, 81.8%, 77.6% and 71%. Five-year and 10-year survival in the Val50Met group was 80.9% and 73.4%, respectively, significantly superior to 57.8% and 43.9% in the non-Val50Met group (p<0.001).
Commonest cause of death was cardiac related events (24%), followed by sepsis (23%) or liver related complications (14%). Disease duration prior to transplantation, initial presentation with autonomic rather than peripheral neuropathy, TTR mutation, and modified body mass index (mBMI) of <600, indicating poor nutritional status, were identified as significant factors influencing survival after LT (p<0.01).

Conclusion Liver transplantation is rational and effective treatment for FAP with excellent long-term outcomes and 10-year survival >70%. Type of mutation, nutritional status, disease duration and degree of autonomic involvement are significant prognostic factors.

P74 HEPATOCYTE TRANSPLANTATION IN RATS WITH ACUTE LIVER FAILURE USING CELLS LABELLED WITH A CLINICAL GRADE MRI CONTRAST AGENT

doi:10.1136/gutjnl-2011-300857a.74

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Introduction Hepatocyte transplantation is being evaluated as an alternative to orthotopic liver transplant. However, the fate of hepatocytes after transplantation is not well defined.

Aim The aims of the study were to: (1) investigate the possibility of labelling hepatocytes in vitro using superparamagnetic iron oxide nanoparticles (SPIOs), (2) determine the effects of labelling on cell viability and function, and (3) perform in vivo experiments on tracking labelled cells by MRI.

Method Human and rat hepatocytes were labelled in culture for 16 h with clinical SPIOs (12.5 µg Fe/ml) and protamine sulphate (5 µg/ml) as a transfection agent. Cellular iron uptake was determined using Prussian blue staining, and quantified by a ferrozine-based assay. Cell viability and function were assessed using LDH leakage, mitochondrial dehydrogenase activity, [14C]-leucine incorporation, albumin and urea assays. Effects of labelled cells on T2-weighted images were assessed in vitro using a 7-T MR scanner. Intrasplenic transplantation of 2×107 male rat hepatocytes labelled with SPIOs (n=4) or non-labelled (n=4) was performed in female recipients 28–30 h after acute liver failure induction by intraperitoneal injection of D-galactosamine. Hepatocytes were also marked with the fluorescent dye CM-Dil. A control group (n=4) received medium injection only. T2*-weighted gradient-echo images at 7-T MRI were acquired at day 7 post-acute liver failure induction. Transplanted cells were detected in the liver by PCR for the Y-chromosome (Sry-2 gene) and histological analysis.

Results Mean intracellular iron concentrations were 11.4±SE1.1 pg/cell in human and 8.6±0.3 pg/cell in rat hepatocytes. Cell viability and metabolic function were not significantly affected at these SPIO concentrations. In vitro MRI of SPIO-labelled cells (2000 cells/ml) induced a 50% change in T2 relaxivity compared to non-labelled cells. SPIOs were detected in rat liver as a decrease in the MRI signal intensity 6 days after transplantation in the three survivors. On histology most of the SPIO particles were located in Kupffer cells, indicating the loss of iron oxide particles from hepatocytes. In keeping with this, labelled cells could not be detected in the liver by the fluorescent dye or by PCR for Sry-2 gene.

Conclusion Optimum conditions to label human and rat hepatocytes with SPIOs were determined, which did not affect cell viability or metabolic function, and were sufficient for in vitro MRI detection. However, the clearance of hepatocytes after transplantation limits the value of MRI for assessing long-term hepatocyte engraftment.

P75 IMPAIRED CARDIORESPIRATORY RESERVE IN PRIMARY BILIARY CIRRHOSIS PATIENTS UNDERGOING LIVER TRANSPLANT ASSESSMENT

doi:10.1136/gutjnl-2011-300857a.75

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Introduction It has been previously shown that PBC patients have bioenergetic abnormality in both peripheral and cardiac muscle. In