Abstract PTU-098 Figure

**Graph 2** – The degree to which GPs felt liver investigations were important in the clinical management of liver disease in primary care.

For investigations that GPs graded as ‘Very Important and Essential’ many were not, in ‘real-life’ primary care practice, documented in patient records (Williams et al. 2012).

**Graph 3** – The degree to which potential barriers might influence GP capacity to manage liver disease in primary care.

Only 6% of GPs stated they had a ‘special interest’ in liver disease and no GP stated that someone else in the practice took the lead on liver disease.

**Graph 4** – Reasons for GPs not having a ‘special interest’ in liver disease

However, as ‘generalists’, 83% of GPs felt they needed more educational support via protected learning sessions, improved national guidelines and joint specialist-GP clinics.

**Conclusion** This survey revealed that many GPs, despite not having a ‘special interest’ in liver disease, would welcome greater educational support from specialists and improved national guidelines. The barriers most cited as influencing GP capacity to manage liver disease are surmountable and should be the focal point for new integrated care pathways.

**Disclosure of Interest** None Declared

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**PTU-099** DISCOVERY OF POTENTIAL PLASMA BIOMARKERS OF CHOLANGIOCARCINOMA UTILISING SURFACE-ENHANCED LASER DESORPTION/IonIZATION TIME-OF-FLIGHT MASS SPECTROMETRY (SELDI-TOF MS)

doi:10.1136/gutjnl-2013-304907.189

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**Introduction** Cholangiocarcinoma (CC) is a malignant neoplasm of the bile duct. Diagnosis of CC is hampered by the inadequate performance of current plasma markers of disease, particularly in patients with preexisting primary sclerosing cholangitis (PSC). We aimed to identify potential new protein biomarkers of CC

**Methods** In an initial discovery study, blood plasma samples from 18 subjects with CC, 17 with PSC and 10 healthy controls were subjected to SELDI-TOF MS. Comparisons of m/z peak intensity were made between groups using the Mann-Whitney U test. Differentiating m/z peaks were then confirmed in a further validation study of 81 subjects with CC, 54 with PSC and 90 healthy controls. Pearson’s correlation was used to investigate the relationship of each m/z peak’s intensity to routine laboratory indices. Diagnostic performance was investigated using receiver operator characteristic area-under-the-curve (ROC-AUC) analyses. Multiple linear regression was used to investigate the performance of differentiating m/z peaks, as well as the combination of m/z peaks with routine laboratory markers (including CA19–9).

**Results** Seven differentially expressed m/z peaks were identified in the CC group and these were subsequently confirmed in the validation study (p = 2.6 × 10−4 to 9.4 × 10−13). The intensity of the seven m/z peaks of interest did not correlate with creatinine, ALP, bilirubin, CRP, white cell count or CA19–9. A panel of three peaks discriminated CC from PSC subjects with ROC-AUC of 0.76 (sensitivity 75%, specificity 64%). A panel of five peaks discriminated CC subjects from healthy controls with ROC-AUC of 0.90 (sensitivity 95%, specificity 74%). Addition of routine laboratory indices did not change the diagnostic performance of these models significantly.

**Conclusion** SELDI-TOF has been used to successfully identify seven m/z peaks that are differentially intense in CC subjects (total n = 99), when compared to PSC subjects (n = 64) and healthy controls (n = 107). These peaks appear to be independent of standard markers of renal impairment, cholestasis, sepsis and inflammation, as well as CA19–9. Individuality, and more so in combination, these peaks exceed the expected diagnostic performance of CA19–9, particularly in discriminating CC from PSC. Work to identify the proteins represented by these m/z peaks is ongoing.

**Disclosure of Interest** None Declared

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**PTU-100** DECOMPENSATED ALCOHOLIC LIVER DISEASE (ALD) IS ASSOCIATED WITH STARTING HEAVY DRINKING AT AN OLDER AGE: A CASE-CONTROL STUDY

doi:10.1136/gutjnl-2013-304907.190

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**Introduction** Decompensated alcoholic liver disease (ALD) is associated with starting heavy drinking at an older age: a case-control study.
**Introduction** The relationship between development of ALD (which affects only 10–15% of heavy drinkers) and rate, duration and age of onset of alcohol consumption is incompletely understood. We have previously reported on total lifetime alcohol consumption in two cohorts of heavy drinkers (>60 Units/wk(M) or >40 Units/wk(F) for ≥5 years): one (patients) with decompensated ALD (Child Grade B or C, negative tests for other liver diseases) and one (controls) without serious liver disease on clinical, laboratory and ultrasound examination. Here, we aimed to compare alcohol consumption patterns in these cohorts in more detail.

**Methods** Subjects (330 patients, 234 male, mean age 48 yr and 235 heavy-drinking controls, 187 male, mean age 48 yr) completed a lifetime alcohol questionnaire. Alcohol consumption was calculated at home and outside home, and during Monday-Thursday and Friday-Sunday. Data were summed over each stable drinking period during the subject’s lifetime. We calculated (a) total duration, and age at start and at cessation of all periods during which the subject drank >0, >40, >80, >120 and >160 units(U)/wk and (b) percent of drinking career engaged in: regular drinking, drinking <5 days per week, weekend drinking and not drinking.

**Results**

<table>
<thead>
<tr>
<th>Units/wk</th>
<th>Duration (yr)*</th>
<th>Age started (yr)*</th>
<th>Duration (yr)</th>
<th>Age started (yr)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;0</td>
<td>30 (23–36)</td>
<td>17 (16–18)+</td>
<td>30 (23–36)</td>
<td>16 (15–18)</td>
</tr>
<tr>
<td>&gt;80</td>
<td>12 (6–19)</td>
<td>29 (21–38)+</td>
<td>13 (7–22)</td>
<td>25 (18–33)</td>
</tr>
<tr>
<td>&gt;120</td>
<td>3 (0–12)</td>
<td>32 (24–41)+</td>
<td>4 (0–10)</td>
<td>28 (21–37)</td>
</tr>
<tr>
<td>&gt;160</td>
<td>0 (0–7)</td>
<td>33 (27–41)+</td>
<td>0 (0–7)</td>
<td>30 (24–39)</td>
</tr>
</tbody>
</table>

* median (interquartile range). ++: p < 0.001, +++: p = 0.02, +++++: p = 0.017 by Mann-Whitney test for patients vs controls.

Neither total duration of periods consuming >0, >40, >80, >120, and >160 U alcohol/wk (table) mean weekly consumption during those periods (not shown) differed significantly between patients and controls. However, patients first started drinking over each level at an older age than did controls (table). The relationships between ALD and age of starting drinking >0, >40, >80, >120 and >160 U/week persisted in multivariate analysis (p = 0.00–0.015). Subjects spent 5 (63–97)% of their careers in regular drinking, with no case-control differences.

**Conclusion** Development of decompensated ALD in heavy drinkers is associated with starting heavy drinking at an older age.

**Disclosure of Interest** None Declared.