Pancreatic and Liver Free Papers:

240 FAS-LIGAND PROTECTS ISLET CELLS FROM APOPTOSIS IN CHRONIC PANCREATITIS

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Background: Exocrine (acinar) destruction and endocrine (islet) preservation in chronic pancreatitis (CP) is related to differential apoptotic indices within these cellular compartments.

Aim: To define the role of cell cycle regulatory proteins in the control of pancreatic epithelial cell apoptosis.

Methods: Formalin-fixed, paraffin-embedded tissue from six cases of CP and eight normal controls (N) were studied using immunohistochemistry for Bcl-2, Bax, retinoblastoma protein (Rb) and Fas ligand (Fas-L). Labelling patterns were assessed using semi-quantitative (intensity-proportion [I-P]) or fully quantitative (labelling index [LI]) analysis.

Results: Rb protein was expressed at a higher level by acinar cells in CP than N (mean acinar cell Rb LI 1.45% in CP and 0.437% in N; p<0.05) but this difference was not observed within islet cells. Bcl-2 was more strongly expressed by acinar than islet cells in CP (4.20±3.4 in acini and 2.83 in islets; p<0.01) while Bax was strongly expressed by a subset of islet cells and weakly by centroacinar cells (N mean Bax I-P score 4.88 in acini and 0.375 in islets; p<0.05). Fas-L was more strongly expressed by islet than acinar cells in CP and N (e.g. mean Fas-L I-P score 5.67 in islets and 2.83 in acini; p<0.01).

Conclusion: Strong Bcl-2 expression by acinar cells and of Bax by islet cells was an unexpected result and indicates complex control of apoptosis within these cell populations. Increased Rb protein expression by acinar cells in CP may differentially promote apoptosis within these cells. Fas-L expression by islet cells may protect islets from apoptosis by promoting apoptosis of cytotoxic T-lymphocytes.

241 THE EFFECT OF SELECTIVE INHIBITION OF INDUCIBLE NITRIC OXIDE SYNTHASE ON THE OUTCOME OF SEVERE ACUTE PANCREATITIS

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Background: Nitric oxide (NO), produced by inducible NO synthase (iNOS), has been established as a key inflammatory mediator in systemic inflammatory response syndrome leading to multiple organ failure.

Aim: To examine the effect of treatment with the selective iNOS inhibitor L-N6-iminoethyl-lysine (L-NIL) on the course of severe acute pancreatitis (AP) in the rat, both local pancreatic and systemic organ failure.

Methods: Acute severe necrotising pancreatitis was induced in 10 adult male Wistar rats by a combination of biliary and pancreatic duct infusion of glycodeoxycholic acid and arterial infusion of caerulein. Data were compared to those in sham operated controls (n=10). The effect of L-NIL treatment was examined in a disease group (n=10) and in a control group (n=10). L-NIL was administered two hours after induction of pancreatitis. Blood pressure, PaO2, and serum creatinine were measured at 7 h. Pancreatic sections were examined histologically and morphometric measurement of areas of acinar cell necrosis as a percentage of total acinar tissue area was performed.

Results: Compared with controls at 7 h, the pancreatitis group displayed increased mean arterial blood pressure [mean (s.e.m.) 77.8 (3.7) versus 108.2 (2.2) mmHg, P<0.0001], reduced PaO2 [66.7 (1.9) versus 115.2 (3.6) mmHg, P<0.0001], and raised serum creatinine [132.5 (20.6) versus 41.8 (2.5) µmol/L, P<0.0001]. Treatment with L-NIL corrected the reduction in blood pressure [mean (s.e.m.) 113.5 (3.4) mmHg, P<0.0001] and ameliorated the reduction of

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INCREASED SYSTEMIC NITRIC OXIDE IN PATIENTS
WITH SEVERE ACUTE PANCREATITIS IS ASSOCIATED WITH ALTERED GUT MACROMOLECULAR
PERMEABILITY: AN ENDOTOXIN MEDIATED INFLAMMATORY RESPONSE?

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Background: There is considerable evidence implicating nitric oxide (NO) as a critical mediator of the systemic inflammatory response to bacterial endotoxaemia. Severe acute pancreatitis (AP) is associated with alterations in intestinal permeability, and bacterial translocation may in part contribute to the local and systemic manifestations of this disease. However, the mechanisms remain speculative and the role of NO in humans with AP has not been studied.

Methods: Patients with a clinical and biochemical diagnosis of AP were studied within 72 hours of onset of abdominal pain. The 24-hr urinary nitrite excretion, reflecting NO production, was measured using the Greiss reaction. The ratio of renal excretion of the enterally administered polynitrogen glycol (PEG) 3350/400 was measured to determine intestinal macromolecular permeability. The IgM:IgG EndoCaB ratio was used as a marker of systemic endotoxin exposure.

Results: Of the 17 patients, 15 had acute severe AP (Grade 3-4) and 2 had AP in the context of chronic pancreatitis (Grade 2). The NO production was significantly increased in patients with severe AP compared with mild AP (r = 0.8, p < 0.01). In patients with severe disease, urine nitrite excretion was significantly increased in patients with severe attacks (median 20.6 µmol/L) compared with mild attacks (median 15.7, p = 0.003) and the latter was significantly greater in healthy controls (median 6.3, p = 0.004). PEG excretion ratios were significantly increased in patients with severe attacks compared to mild attacks (r = 0.8, p < 0.01). In patients with severe disease, urine nitrite excretion demonstrated a positive and significant correlation with intestinal macromolecular permeability and the IgM:IgG EndoCaB ratio (r = 0.7, p = 0.006, and r = 0.8, p = 0.001 respectively).

Conclusion: Systemic NO is increased in AP, and is greatest amongst patients who develop a severe attack. The strong correlation with altered gut permeability and endotoxin exposure suggests that the mechanism may be mediated through bacterial up-regulation of inducible nitric oxide synthase activity.

EVALUATION OF ENDOSCOPIC ULTRASOUND
VERSUS HELICAL COMPUTED TOMOGRAPHY IN THE
ASSESSMENT OF PANCREATIC MASSES: RESULTS
FROM A UK CENTRE

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Introduction: Endoscopic ultrasound (EUS) is an emerging diagnostic modality which has been shown to be more accurate in the diagnosis, and locoregional staging of pancreatic carcinoma than conventional computed tomography (CT). The aim of this study was to determine the local staging accuracy of EUS, compared with helical computed tomography (HCT) in a United Kingdom centre.

Patients, materials and methods: Between September 1999 and October 2000, 43 consecutive patients underwent radial EUS (GF-UM20) and HCT for assessment of pancreatic masses. Of these, 17 patients underwent both EUS and surgery. EUS included gastric lymph node clearance in all cases, and portal vein resection where necessary. Tumour size, portal venous invasion, and nodal involvement were assessed and compared with histopathological examination of the resected specimens as the reference standard. The χ² test for paired data was used to compare groups.

Conclusion: EUS is accurate in the local, and nodal staging of pancreatic masses, and is sensitive in the assessment of portal vein invasion.

HEPATOcyTES DERIVED FROM BONE MARROW
STEM CELLS SHOW POLYpLOIDIZATION IN HUMANS
AND MICE

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Background and aims: Using a technique to detect Y-chromosomes within human liver we have previously demonstrated in patients that have received sex mis-matched bone marrow or liver transplants that human hepatocytes can be derived from bone marrow stem cells. These cells may provide an alternative source of hepatocytes following liver damage. Using the technique of Y-chromosome detection we have examined the potential for hepatocyte polyploidization in bone marrow recipients. This is of relevance as polyploidization is an integral feature of hepatocyte replication.

Method: Beta3-integrin-deficient mice were used as they develop cirrhotic liver damage that results in a persistent low-grade liver regeneration. Six-week old female recipient mice underwent whole
**STEM CELL FACTOR LIMITS LIVER INJURY INDUCED BY PARACETAMOL POISONING**

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**Background and aims:** Paracetamol (acetaminophen) remains a common cause of acute liver failure and emergency liver transplantation in both the UK and USA. The cytokine networks involved in the pathogenesis of paracetamol induced acute liver injury are poorly understood. Stem cell factor (SCF) is a cytokine involved in cell growth and repair which was studied in a murine model of paracetamol poisoning.

**Methods:** CBA mice were injected IP with paracetamol solution following an 8 hour fast. SCF was quantified by ELISA, neutralized with anti-SCF antibodies and constituted with recombinant SCF (rSCF).

**Results:** Paracetamol injection lead to significant reduction in hepatic SCF concentration measured by ELISA (control 2100+300ng/gm liver, mean+sem, paracetamol 970+170, n=7, p<0.05) at 24 hours. Injection of 200mg/kg paracetamol was associated with 80% survival at 96 hours, but inhibition of SCF in this “sublethal” model was associated with survival of only 40% at 96 hours (p<0.05, n=20 in each group): this was associated with significantly increased areas of liver necrosis at 96 hours in the survivors (control 11.6+5.4 % total area of liver, mean+sem, anti-SCF treated 25.7+8.5, n=5, p<0.05). Conversely administration of rSCF (1ug improved survival of mice injected with 300mg/kg paracetamol improved survival from 30% at 96 hours to 90% (p<0.05, n=20 in each group) which was associated with decreased hepatic histology at days 1, 2, 4 and 6 in the rSCF treated group. SCF treatment was correlated with significant reduction in cytochrome P4502E1 expression in both murine liver and culture murine cell lines.

**Conclusions:** Hepatic SCF plays an important role in modulating paracetamol induced liver injury in a murine model. A potential mechanism for this effect is the inhibition of paracetamol activation by cytochrome P4502E1. Other mechanisms such as an effect of SCF on hepatocyte proliferation are currently under investigation.

**LIVER DISEASE MORTALITY IN THE BLACK COUNTRY 1993–1999**

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**Background:** Treatment of advanced liver failure is a major burden on healthcare resources. To determine incidence of liver disease mortality and any underlying trends we analysed data in three boroughs of the West Midlands “Black Country” (Wolverhampton, Dudley and Sandwell, total population 845,000) from 1993–1999.

**Methods:** Public health mortality files were analysed for liver-related deaths using ICD reference codes and keyword searches. Case notes were analysed in cases of liver disease of unspecified cause.

**Results:** There was a stepwise increase in liver-related mortality from 6.6 per 10^5 population in 1993 to 13.9 per 10^5 in 1999. This increase was exclusively due to alcoholic liver disease (ALD); incidence 3.1 per 10^5 in 1993 rising threefold to 9.3 per 10^5 in 1999, whilst mortality due to other defined liver diseases was stable at 0.5 per 10^5. In Wolverhampton and Sandwell (which have large Asian communities) ALD mortality rates in Asian and white populations were similar but Asian subjects died at an earlier age (median age at death 46 years vs. 55 yrs in whites, p<0.001). These data probably underestimate true ALD mortality since unspecified liver disease (around 20% of all liver mortality) was due to ALD in 62% of cases as judged by case note analysis; also ALD deaths increased by 8% when accounting for ALD “misclassified” as other diseases. Finally, in-patient liver disease episodes increased stepwise from 41 per 10^5 population in 1993 to 57 per 10^5 in 1999 which correlated significantly with mortality rates (p=0.74, p=0.01).

**Conclusions:** Liver disease mortality due to alcohol has increased dramatically in recent years, and if sustained this trend has important implications for gastroenterologists and public health specialists.

**CORRELATION BETWEEN CEREBRAL PROTON MAGNETIC RESONANCE SPECTROSCOPY (1H MRS) AND NEUROPSYCHOMETRIC ABNORMALITIES IN PATIENTS WITH CHRONIC HEPATITIS C (HCV) INFECTION**

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Patients with chronic HCV score lower on quality of life scales compared to normal individuals and patients with chronic hepatitis B (HBV). We examine the hypothesis that a direct cerebral effect of HCV underlies this. Computerised and paper-based psychometric tests batteries were administered to 26 patients with histologically mild HCV hepatitis and 10 HCV antibody+ve, PCR-ve age, sex and intelligence-matched controls. 12/26 patients vs 0/10 controls (p=0.01) and 8/24 patients vs 0/10 controls (p=0.07) were impaired

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on computer and on paper-based batteries respectively. There were no differences according to a history of intravenous drug abuse (IVDA). In vivo [1H] MR spectra were acquired from vessels in the basal ganglia (BG), white matter (WM) and occipital grey matter (GM) with a 1.5T spectroscopy system in 3 patient groups: 1) 30 patients with biopsy-proven mild HCV infection (mean age 44 yr, 47% M, mean liver necroinflammatory score 2.4/18, mean fibrosis score 1.6/6); 67% had a history of IVDA; 2) 12 hepatitis B eAg+ve patients without cirrhosis, none IVDA+ve; 3) 29 healthy controls (mean age 42 yr, 52% M), none IVDA+ve.

Results: A significant elevation in BG and WM choline/creatinine (Cho/cr) was seen in the HCV group compared to the other groups (*p<0.005).

Abstract 249, Table 1

<table>
<thead>
<tr>
<th>Mean (SD)</th>
<th>HCV</th>
<th>HBV</th>
<th>Volunteers</th>
</tr>
</thead>
<tbody>
<tr>
<td>BG Cho/Cr</td>
<td>1.17 (0.14)*</td>
<td>1.04 (0.14)</td>
<td>1.06 (0.13)</td>
</tr>
<tr>
<td>WM Cho/Cr</td>
<td>1.35 (0.22)*</td>
<td>1.16 (0.12)</td>
<td>1.18 (0.14)</td>
</tr>
</tbody>
</table>

No difference was seen in the HCV group according to IVDA+ve status. Fourteen HCV patients also had psychometric testing, showing correlations between BG Cho/ Cr and indices of sustained attention (*r=0.699, p=0.005) and quality of working memory (*r=0.544, p=0.04).

Conclusion: Both cognitive impairment and cerebral metabolite abnormalities are seen in patients with mild HCV hepatitis. This may be due to a cerebral effect of systemic cytokines or direct infection of the CNS by HCV (as in HIV infection where similar [1H] MRS abnormalities are seen).

250 CHARACTERISING THE EFFECTS OF THYROID HORMONE ON THE LIVER: A NOVEL APPROACH TO INCREASING LIVER MASS

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Background: Liver growth can occur as part of two distinct mechanisms: 1) Compensatory Regeneration: Following resection, viral/ drug injury; 2) Direct Hyperplasia: A primary mitogen increasing liver mass directly.

Aim: a) To assess if thyroid hormone acts as a primary mitogen. b) To characterise the effects of thyroid hormone when administered prior to a 70% partial hepatectomy (PH)—testing a primary mitogen with an established model of regeneration.

Method: a) Male Sprague-dawley rats (n=7 per group) were injected with a single dose of triiodothyronine (T3) and sacrificed at intervals of 1,2,4,7,10 and 14 days. A control group received vehicle only twenty four hours prior to sacrifice. All animals were assayed 24 hrs after partial hepatectomy. b) A 70% partial hepatectomy was performed on Sprague-Dawley rats (n=5 per group) on days 1,3 and 10 following a single dose of T3. A control group received vehicle only twenty four hours prior to PH. All animals were sacrificed 24 hrs after partial hepatectomy. Cell Proliferation — Assessed by bromodeoxyuridine (BRDU) incorporation into nuclei and immunohistochemical recognition.

Results: a) Liver mass was increased in animals treated with T3 as compared with controls. Maximum effect was seen on day 10 with a 20% increase in liver mass (p<0.05). A corresponding increase in total DNA (p<0.05) and liver protein (p<0.05) was seen at this time point. Proliferation — Peaked at day 1: 7% hepatocytes labelled compared to <1% in controls (p<0.01). b) In animals treated with T3 three and ten days prior to partial hepatectomy the liver weight at sacrifice was greater (p<0.05) than controls. There was a corresponding increase in liver protein (p<0.05) and total DNA (p<0.05) at these time points. Proliferation — Peaked when T3 was administered 24 hrs prior to PH 36% hepatocytes labelled compared to 26% in controls (p<0.01).

Conclusions: Thyroid hormone acts as a mitogen and synergistically with a 70 % partial hepatectomy. This raises the possibility of preoperative T3 administration to perform larger liver resections.

251 HEPATIC CO-FACTORS IN END-STAGE, ALCOHOL RELATED, LIVER DISEASE

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Alcohol consumption is well recognised as an independent cause of cirrhosis and as an accentuating factor in the pathogenesis of advanced liver disease in hepatitis C carriers. However, a history of alcohol consumption may contribute to erroneous diagnoses or the failure to recognise significant co-factors. We undertook a retrospective analysis of these variables in 159 patients referred for liver transplantation with a primary diagnosis of alcoholic liver disease between 1996 and 2000.

Of the 159 patients, 31 (20%) had recognised chronic viral hepatitis (24 anti-HCV positive, 5 HBsAg positive, 2 anti-HCV & HBsAg positive). These 31 patients were younger than the remaining 128 patients without viral hepatitis (mean 49 vs 52 years, P = 0.03) and younger than a separate cohort of 79 non-alcohol abusing hepatitis C carriers with end-stage liver disease (mean 49 vs 53 years, P = 0.01). In 12 out of the 128 non-viral alcohol abusers the primary diagnosis was not alcoholic liver disease: 2 Autoimmune hepatitis, 2 Caroll’s disease, 2 Nodular Regenerative Hyperplasia, 2 PBC, 2 PSC, 1 Sarcoidosis, 1 Haemochromatosis (C282Y homozygous). In a further 16 patients co-factors were identified: 14 hepatic siderosis (grade 3/4), 2 alpha-1 antitrypsin deficiency (MZ phenotype). Of the siderotic patients only 2 had HFE gene mutations detected, 1 had aceruloplasminaemia. Hepatocellular cancer was identified in 23 patients, 7 were discovered incidentally in the explanted liver.

Mean serum IgA was significantly higher in the alcohol cohort than the separate hepatitis C cohort (5 vs 7 g/l, P = 0.0002), but no other discriminatory laboratory variables were identified.

This study reveals a 10% erroneous diagnosis rate in patients referred with a diagnosis of alcoholic liver disease. It confirms the accelerated progression to cirrhosis in hepatitis C carriers abusing alcohol. Significant co-factors were present in a further 13%; this went unrecognised in most cases before transplantation. Despite advances in screening for hepatocellular carcinoma, 30% of the tumours were undiagnosed before transplantation. These data suggest a need for a more circumspective assessment of alcohol consuming patients with end-stage liver disease.

252 THALIDOMIDE BUT NOT PENTOXIFYLLINE LOWERS PORTAL PRESSURE IN HUMAN ALCOHOLIC CIRRHOSIS

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Introduction: Blockade of tumour necrosis factor alpha (TNFα) activity reduces portal pressure in portal vein-ligated rats. We investigated the ability of two inhibitors of TNFα production, thalidomide and pentoxifylline, to reduce portal pressure in humans.

Methods: Abstinent patients with stable alcoholic cirrhosis and oesophageal varices were treated for two weeks with open-label pentoxifylline 1800mg daily (n=9) or thalidomide 200mg daily (n=10). Portal and systemic haemodynamics were measured invasively. The hepatic venous pressure gradient (HVPG-mmHg) was calculated by subtracting free from wedged hepatic venous pressure gradients.

Results: Thalidomide (T) consistently reduced HVPG (Table 1) and increased hepatic blood flow 1164 (738–2260) ml/min to 1505 (1054–2943) ml/min. Hepatic vascular resistance fell from 1498 (460–2546) to 559 (269–1039) dynes/sec/cm5 (p=0.028). Pentoxifylline (P) had no effect. Side effects led to dose reduction/withdrawal in 4 (T) and 5 (P). Two patients (T) withdrew without reason. Data for patients completing two weeks treatment are expressed as median (range) and analysed using Wilcoxon signed ranks test (*p=0.028).

Abstract 252, Table 1

<table>
<thead>
<tr>
<th>N Age</th>
<th>Child</th>
<th>ABC</th>
<th>HVPG before</th>
<th>HVPG after</th>
<th>Change in HVPG</th>
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<tbody>
<tr>
<td>P</td>
<td>6</td>
<td>51</td>
<td>(45–64)</td>
<td>3/2/1</td>
<td>19.2 (14.3–24.7)</td>
</tr>
<tr>
<td>T</td>
<td>6</td>
<td>59</td>
<td>(45–72)</td>
<td>3/3/0</td>
<td>19.7 (9.3–23.5)</td>
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</table>
TIPS and BUDD–CHIARI SYNDROME


Background/aims: Patients with Budd–Chiari who are not amenable to hepatic vein recanalisation are usually treated either medically, with surgical shunt formation or liver transplantation. We reviewed the outcome of 17 patients who were treated with TIPS.

Methods: A retrospective case note analysis was undertaken of all patients with Budd–Chiari Syndrome who had a TIPS attempted.

Results: TIPS was attempted in 17 patients with a median age of 43 years (range 20–59) and median length of history 9 weeks (range 2–364 weeks). An underlying aetiology was found in 16 patients. 15 patients had a technically successful TIPS performed, neither of the 2 patients developed adverse sequelae following failed TIPS attempt. Of the 15 patients, one died soon after TIPS and one died of a subdural haematoma 6 weeks later. The remaining 13 have been followed up for a median of 31 months and have had excellent symptom resolution and have required an average of 0.92 re-interventions per patient.

Conclusion: In appropriately selected patients with Budd–Chiari, TIPS can provide a definitive treatment at least in the medium term.

RANDOMISED CONTROLLED TRIAL OF TRANSJUGULAR INTRAHEPATIC PORTOSYSTEMIC STENT-HUNTING (TIPS) VERSUS TIPS AND VARICEAL BANDING (VBL) IN THE PREVENTION OF VARICEAL REBLEEDING

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TIPS is highly successful in treating variceal haemorrhage and preventing variceal rebleeding. We have previously shown TIPS to be superior to VBL in the prevention of variceal rebleeding. However patients with a TIPS require regular radiological surveillance in order to ensure patency. It is not known whether the combination of TIPS and VBL can prevent the need for regular surveillance without compromising efficacy.

Aims: To compare the efficacy of TIPS against that of TIPS in combination with VBL in the prevention of variceal haemorrhage.

Methods: Between 1996–2000 eligible patients who required a TIPS following an oesophageal variceal bleed were randomised to the TIPS only arm (n=36, group 1) or TIPS and banding arm (n=31, group 2). Average follow up was 24 and 26 months respectively. In group 1 patients underwent regular portography and TIPS surveillance was performed for the first year only with VBL to stent dilatation or parallel shunt insertion if required. In group 2 patients with a TIPSS require regular radiological surveillance in order to ensure patency. It is not known whether the combination of TIPS and VBL can prevent the need for regular surveillance without compromising efficacy.

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