

increased the number of cells per field of view in the livers of mice following transplantation (1.6 vs 0.7, $p=0.017$).

Conclusion β 1-integrin blocking antibodies increase survival of isolated hepatocytes and improve their ability to remain adherent to HSEC under flow resulting in increased engraftment of transplanted human hepatocytes in mouse liver. The use of β 1-integrin blocking antibodies may provide a means to increase the efficacy of human hepatocyte transplantation.

Competing interests None declared.

REFERENCES

1. Pinkse, et al. *J Hep* 2005.
2. Kocken, et al. *Lab Invest* 1997.

PMO-116 EUS GUIDED SAMPLING OF PANCREATIC MALIGNANCY

doi:10.1136/gutjnl-2012-302514b.116

¹D Lloyd,* ²A Higginson, ³B Stacey, ¹H Shepherd, ¹H Gordon. ¹Department of Gastroenterology, Royal Hampshire County Hospital, Winchester; ²Department of Radiology, Portsmouth Hospitals NHS Trust, Portsmouth; ³Department of Gastroenterology, Southampton University Teaching Hospital Foundation Trust, Southampton, UK

Introduction Endoscopic ultrasound (EUS) guided sampling of malignant pancreatic lesions is increasingly performed to confirm malignancy prior to chemotherapy and/or radiotherapy. Historically lesions have been sampled by fine needle aspiration (FNA) yielding cells for cytological analysis. Cook Medical has recently developed the ProCore needle for EUS guided fine needle biopsy (FNB). This study compared the diagnostic yield of Procore FNB and FNA in patients undergoing EUS guided sampling of suspected pancreatic malignancy in a tertiary EUS centre.

Methods All patients with suspected pancreatic malignancy undergoing EUS guided tissue sampling over a 1-year period from 1st January 2011 to 31st December 2011 were retrospectively identified from endoscopy records. Note was made of whether FNA or FNB were performed. Electronic records were reviewed to determine the results of FNA/FNB. Standard statistical tests were used to compare the diagnostic yield of FNA and FNB.

Results EUS guided sampling was performed on 51 suspected malignant pancreatic mass lesions. FNA was performed on 27 occasions; FNB was performed on 40 occasions. Fifteen lesions were sampled by both FNA and FNB. FNA yielded a sample sufficient for cytological analysis in 19 (70%) cases. Of these samples, cytology confirmed malignancy in 17 (89%) of cases. FNB yielded a sample sufficient for histological analysis in 27 (68%) cases. Of these, histology confirmed malignancy in 26 (96%) cases. There was no statistically significant difference in either the yield of analysable tissue or the yield of positive cytology/histology between FNA and FNB. In cases where both FNA and FNB were performed, both modalities confirmed malignancy in eight cases (53%) and both modalities failed to yield diagnostic tissue in three cases (20%). In two cases FNA was positive with insufficient tissue from FNB, and in two cases FNB was positive with FNA yielding insufficient tissue. The overall yield of FNB was only one in five of patients undergoing repeat sampling where the initial sample had been non-diagnostic, compared to two in three for FNA.

Conclusion An advantage of Procore FNB is that cytology specimen preparation in endoscopy is not required and the larger sample allows more extensive histological assessment. The overall positive yield of FNB in patients who underwent EUS guided sampling of a suspected pancreatic malignancy was 65% compared to 63% for FNA. The limitation to higher yield appears to be acquisition of sufficient tissue for histological analysis. The yield of FNB in repeat sampling is low suggesting that combined FNA & FNB should be

performed in such situations, ideally with on site microscopy assessment to ensure adequate tissue acquisition.

Competing interests None declared.

PMO-117 PERTURBATION OF THE MITOCHONDRIAL NETWORK ARCHITECTURE IN AN IN VITRO MODEL OF ALCOHOL-INDUCED LIVER TOXICITY

doi:10.1136/gutjnl-2012-302514b.117

¹E Palma,* ^{2,3}D Clemens, ¹R Williams, ¹S Chokshi. ¹Institute of Hepatology, London, UK; ²Veteran Affairs Medical Center; ³University of Nebraska Medical Center, Omaha, USA

Introduction Mitochondria are central to many cellular processes and are dynamic organelles that exist as a network in the form of elongated filaments and respond to the demands of the cell through cycles of fusion (binding of mitochondria) and fission (mitochondrial-fragmentation) which are driven primarily through multiple mitochondrial shaping proteins. Mitochondrial function is intimately associated with their morphology and while mitochondrial dysfunction has been previously correlated with alcohol consumption, there is a paucity of understanding regarding the impact of alcohol on the dynamic balance between fusion/fission and on mitochondrial morphology. The aim of this study was to investigate the impact of alcohol-induced liver damage on mitochondrial morphology, dynamics and to identify the precise mechanisms driving these changes.

Methods Ethanol metabolising-human hepatoma cell lines VL-17A (positive for alcohol-dehydrogenase and CYP2E1) were cultured in the presence of increasing doses of ethanol (EtOH), reflecting real-life alcohol consumption. Cells were cultured with EtOH at 10 mM (safe levels), 50 mM, 250 mM (high levels) and 500 mM (highly toxic levels). Cultures were incubated in presence/absence of EtOH for 24, 48, 72 and 96 h. Post-treatment the levels of mitochondrial shaping proteins including Mitofusin-1 (Mfn-1), Mitofusin-2 (Mfn-2) and Dynamin-related protein-1 (Drp-1) were analysed by detecting protein and mRNA levels. Dynamic changes in the morphology of mitochondria were assessed by confocal microscopy.

Results In the absence of alcohol, we observed no changes in the mitochondrial shaping proteins and no changes in the mitochondrial network over time. At 24 h, cells treated with 50 mM ethanol induced profound modifications in the mitochondrial network with a spot-like presentation which correlated with increased levels of Drp-1. At higher toxic levels of 500 mM, the cells display features of mitochondrial toxicity characterised by fragmentation reflecting the high level of cell death observed with this concentration. This toxicity was associated with reduced expression of Mfn-1 and Mfn-2.

Conclusion For the first time we show that alcohol can profoundly perturb the equilibrium between fusion and fission which directly affects the mitochondrial morphology. This study reveals a novel finding in the pathogenesis of alcohol-induced liver toxicity.

Competing interests None declared.

PMO-118 THE EFFECTS OF TH17 CYTOKINES ON LIVER PARENCHYMAL CELLS SHAPE THE MICROENVIRONMENT FOR LOCAL GENERATION OF TH17/TC17 IN INFLAMMATORY LIVER DISEASE

doi:10.1136/gutjnl-2012-302514b.118

¹E Humphreys,* ¹R Bhogal, ¹M Munir, ¹G Muirhead, ²B Eksteen, ¹S Afford, ¹Y Oo, ¹D Adams. ¹Immunity & Infection, University of Birmingham, Birmingham, UK; ²Immunity & Infection, University of Alberta, Calgary, Canada

Introduction IL-17 secreting T cells have been implicated in autoimmunity, inflammatory disease and provide a link between the