correlated with reduced bone density and female sex, but neither vitamin D status nor pre-transplant fragility fractures.

Discussion Liver transplant recipients are commonly deficient in Vitamin D, and at high risk of reduced bone density and fragility fractures both pre- and post-transplantation. Further evaluation of factors predisposing recipients to fragility fractures, and adherence to national and local guidelines on optimising bone health, are required.

P053 INVESTIGATION OF THE ORIGIN OF SERUM NEUTROPHIL GELATINASE ASSOCIATED LIPOCALIN (NGAL) FOLLOWING LIVER ISCHAEMIA REPERFUSION AND RESULTANT ACUTE KIDNEY INJURY IN MICE

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Ischaemia reperfusion (IR) injury to the liver is independently associated with development of acute kidney injury in patients following liver transplantation. Neutrophil Gelatinase Associated Lipocalin (NGAL) is an early marker of primary Acute Kidney Injury (AKI) in mice and humans. Using a mouse model of remote ischaemic preconditioning, we investigated whether serum NGAL concentration correlates with development of AKI following ischaemia reperfusion of the liver and the origin of serum NGAL in this setting.

24 C56BL/6J mice were divided into 4 groups (6 mice per group): a) sham b) liver ischaemia reperfusion (IR) c) remote ischaemic preconditioning (RIPC) d) RIPC + IR.

RIPC was achieved by interval clamping of the right femoral vessels with sham mice undergoing vessel exposure without clamping. Subsequent partial liver ischaemia was achieved by cross-clamping the inflow to left and middle liver lobes for 45 minutes followed by 2 hours of reperfusion. Sham mice underwent laparotomy without clamping. Plasma samples were collected by exsanguination at the end of 2 hours reperfusion and the liver and left kidney were harvested, fixed in formalin and paraffin embedded. Plasma transaminase levels were used as a surrogate marker of liver IR injury. Serum creatinine was used as a marker of AKI and NGAL levels were also performed. Tissue specimens were processed for quantitative PCR, NGAL immunohistochemistry and NGAL/F4/80 dual staining.

Abstract P053 Figure 1 NGAL immunohistochemistry (IHC) of mouse kidney specimen in 'A' demonstrates staining within the apical third of tubular cells, indicating possible uptake of NGAL from tubular filtrate. This is in keeping with established literature. 'B' demonstrates dual staining of F4/80 positive cells (brown stain) and NGAL (pink stain) in a mouse liver specimen. Co-localisation of F4/80 and NGAL is depicted by yellow stain. This indicates partial NGAL expression by F4/80 (Kupffer) cells, with no hepatocyte NGAL expression. As depicted in this slide, at least one other cell type expresses NGAL within the liver

Comparing mice in group b) with the other three groups, serum transaminase levels correlated with both serum creatinine and serum NGAL, indicating that liver IR is significantly associated with liver injury, development of AKI and an increase in serum NGAL concentration in mice.

Quantitative PCR identified no difference in NGAL RNA expression between the groups. This was supported by NGAL immunohistochemistry findings. Liver and kidney specimens demonstrated no difference in the number of NGAL positive cells or intensity of NGAL staining between different groups of mice (figure 1). In the kidney, NGAL staining within the apical third of tubular cells suggested uptake of NGAL from tubular filtrate. In the liver, NGAL staining spared hepatocytes but demonstrated partial co-localisation with F4/80, a marker for Kupffer cells.

We demonstrated an association between liver IR injury, AKI and serum NGAL that is present only 2 hours post reperfusion in mice. Interestingly NGAL does not appear to originate from either the liver or the kidney within this time frame, in contrast to other published data indicating production of NGAL by hepatocytes at later time points.

P054 LONG TERM SURVIVAL AND HEALTHCARE RESOURCE USE IN PATIENTS WITH HEPATIC ENCEPHALOPATHY RECEIVING RIFAXIMIN- α TREATMENT: A RETROSPECTIVE OBSERVATIONAL EXTENSION STUDY WITH LONG TERM FOLLOW-UP (IMPRESS II)

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Hepatic encephalopathy (HE) is associated with high mortality and considerable resource use including recurrent hospital admissions in people with cirrhosis. IMPRESS was a 12-month observational study of 207 patients with HE, demonstrating addition of rifaximin- α led to reduced hospitalisations, Emergency Department attendances and 30-day readmissions.¹ Data on long-term (1–5 year) outcomes with rifaximin- α use is scarce. An extension to IMPRESS was conducted to evaluate outcomes up to 5 years (IMPRESS II).

Data were obtained from medical records of 138 patients from 9/13 centres included in IMPRESS, including demographic, clinicopathological, treatment and resource use for the period from rifaximin- α initiation (index) to 5 years post-index or death. Long-term survival is reported herein.

Overall, 62% (85/138) of patients were male; mean (standard deviation [SD]) age at cirrhosis diagnosis, HE diagnosis and index was 58.2 (12.1 [n=133]), 60.2 (11.7), and 60.9 (11.6), respectively. At index, 9%, 50% and 41% of patients (n=127) had albumin-bilirubin (ALBI) grade I, II and III,