signalling, in regulating intestinal epithelial apoptosis in vivo have not previously been investigated.

**Aims** To assess susceptibility to intestinal apoptosis and the associated molecular changes in mice with germline deletions of three individual NF-kB family members.

**Methods** Intestinal apoptosis was induced in male c-Rel-null, NFkB1-null and NFkB2-null mice and their wild-type (C57BL/6) counterparts by 8Gy \( \gamma \)-irradiation (n=6 per group). Apoptosis was assessed on a cell positional basis from H/E stained sections. The mRNA expression of 10 key apoptosis regulating genes (BCL2, BCL-XL, c-IAP2 and XIAP) was assessed by real time PCR (n=4 per group). Statistical comparisons were by ANOVA with Bonferroni post-hoc tests.

**Results** Basal small intestinal crypt apoptosis was significantly increased in NFkB2-null relative to C57BL/6 mice. In addition, small intestinal and colonic crypt apoptotic indices were both significantly increased (up to threefold) in NFkB1-null and NFkB2-null mice 4.5 h after 8Gy \( \gamma \)-irradiation relative to wild-type and c-Rel-null mice. Untreated NFkB1-null and NFkB2-null small intestine showed reduced mRNA expression of the anti-apoptotic genes BCL2, BCL-XL, c-IAP2 and XIAP. Following irradiation, NFkB1-null mice showed significant increases in the mRNA of the pro-apoptotic genes TRAIL, Caspase12 (in both small intestine and colon) and BAK (small intestine only) compared to wild-type mice. Significant increases in the mRNA of the pro-apoptotic genes Caspase12 and FAS-L were also seen in irradiated NFkB2-null small intestine and colon relative to wild-type mice.

**Conclusion** c-Rel expression does not appear to regulate susceptibility to intestinal epithelial apoptosis in vivo. NFkB1 and NFkB2 deletion both caused increased susceptibility to intestinal apoptosis and this was associated with altered expression of TRAIL, Caspase12, BAK and FAS-L. These NFkB family members may therefore also regulate the susceptibility of intestinal epithelia to other consequences of DNA damage such as cancer.

**Competing interests** None declared.

**REFERENCE**

BASL plenary session

**OC-022** EMBOLISATION OF INFLOW TO ALLOW SAFER LIVER RESECTION—IS MORE, BETTER?

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R Lochan,* A Vallance, J French, R Charnley, B Jaques, J Rose, S White, D Manas. HPB Liver Transplantation Surgery, FHH, Newcastle Upon Tyne, UK

**Introduction** Portal vein embolisation (PVE) is now an established technique to increase the future liver volume/remnant (FLR) prior to liver resection. For those patients where hypotrophy is still considered insufficient complete uni-lateral embolisation incorporating both portal and hepatic artery embolisation (HAE) has been less frequently reported. The aim of this study was to evaluate the feasibility of sequential PV/HAE embolisation to increase the FLR prior to liver resection.

**Methods** All HPB patients are discussed at a weekly MDT meeting to decide on appropriate management decisions including the necessity for FLR augmentation. PVE is performed by initially obtaining a portogram by percutaneous trans-hepatic puncture. Selective embolisation of the necessary portal veins are then obtained using a combination of coils and glue etc. Embolisation of Segment 4 PV branches are performed on a selective basis. HAE embolisation is performed by mapping arterial inflow and selectively embolising the desired segments planned for resection while carefully preserving the FLR. The aim of this study was to evaluate the feasibility/safety of PVE with sequential HAE over a 5-year period (January 2006–May 2011).

**Results** 50 patients (M:F = 38:12) underwent a right PVE; 33 (66%) progressed to liver resection. Reasons for inoperability (34%) following PVE (n=17) were (1) Small FLR, (n=6) all underwent HAE (with five proceeding to resection) (2) extra-hepatic disease (n=7) (3) progression of hepatic disease (n=4). The median FLR of those who progressed to resection following PVE, by CT volumetry, was 384.5 cc (330–490), significantly more than those who did not 237 cc (110–210). HAE increased the FLR by a further 100 cc. An R0 resection was achieved in 25 patients (76%), including 4/5 (80%) of sequential patients. Following PVE and sequential embolisation; 9/33 (27%) and 3/5 (60%) suffered serious complications (Clavien-Dindo 3 or 4). There were six post operative deaths including 5/33 (15%) after PVE and 1 (20%) following sequential embolisation respectively.

**Conclusion** PVE is an increasingly used technique to increase the FLR allowing a significant proportion of patients an R0 resection despite initially being considered inoperable. Nevertheless at least 20% of patients will also have progression of disease. Patients who do not achieve adequate hypertrophy can potentially have HA embolisation to increase the FLR by a further 100 cc but perhaps at the expense of increasing post-operative complications.

**Competing interests** None declared.