(ECM). Hepatic progenitor cell (HPC) activation occurs in the context of severe liver injury. ECM stiffness has been shown to direct differentiation in mesenchymal stem cells. However, the effect of mechanical factors, such as ECM stiffness on HPC responses is poorly characterised. We examined the effect of ECM stiffness on HPC proliferation and differentiation.

**Methods** Experiments were undertaken using a murine HPC line (BMOL) and primary murine HPCs. Cell culture experiments were performed using a system of laminin-coated polyacrylamide (PA) gels to support culture. The stiffness of the PA supports (expressed as shear modulus) was altered across a physiological range (1–12 kPa) for BMOL cells cultured on stiff (12 kPa) supports. Quantitative PCR revealed that BMOL cells cultured on soft (1 kPa) supports were 7.1-fold (p < 0.01) and 11.5-fold (p < 0.001), respectively, than cells cultured on 1 kPa supports. Similarly, in experiments with primary cells, the PI of murine HPCs was 1.7-fold higher (p < 0.05) when cells were cultured on stiff (12 kPa) vs soft (1 kPa) supports. Quantitative PCR revealed that BMOL cells cultured on soft (1 kPa) supports up-regulate hepatocyte markers, including albumin (1.5-fold, p < 0.01) and CYP7A1 (1.6-fold, p < 0.01), and down-regulate the HPC/biliary marker cytokeratin-19 (0.6-fold, p < 0.01), relative to cells on stiff (12 kPa) supports. There was no significant change in expression of the biliary epithelial cell markers aquaporin-1 and γ-glutamyl-transferase.

**Conclusion** Physiological changes in ECM stiffness lead to alterations in HPC morphology, proliferation and differentiation. Increased ECM stiffness (as would be encountered in an injured or fibrotic liver) promotes HPC proliferation and expression of the HPC/biliary marker cytokeratin-19. In contrast, a low-stiffness environment is associated with a reduction in cell proliferation and up-regulation of hepatocyte-specific markers. These results suggest that mechanical factors, such as ECM stiffness might regulate HPC responses following liver injury.

**Competing interests** None declared.

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**ENDOGENOUS PRODUCTION OF ANTIBIOTICS BY MESENCHYMAL STEM CELLS AND THE POTENTIAL VALUE IN CROHN’S FISTULA HEALING**

**Methods** Experiments were undertaken using a murine HPC line (BMOL) and primary murine HPCs. Cell culture experiments were performed using a system of laminin-coated polyacrylamide (PA) gels to support culture. The stiffness of the PA supports (expressed as shear modulus) was altered across a physiological range (1–12 kPa) for BMOL cells cultured on stiff (12 kPa) supports. Quantitative PCR revealed that BMOL cells cultured on soft (1 kPa) supports were 7.1-fold (p < 0.01) and 11.5-fold (p < 0.001), respectively, than cells cultured on 1 kPa supports. Similarly, in experiments with primary cells, the PI of murine HPCs was 1.7-fold higher (p < 0.05) when cells were cultured on stiff (12 kPa) vs soft (1 kPa) supports. Quantitative PCR revealed that BMOL cells cultured on soft (1 kPa) supports up-regulate hepatocyte markers, including albumin (1.5-fold, p < 0.01) and CYP7A1 (1.6-fold, p < 0.01), and down-regulate the HPC/biliary marker cytokeratin-19 (0.6-fold, p < 0.01), relative to cells on stiff (12 kPa) supports. There was no significant change in expression of the biliary epithelial cell markers aquaporin-1 and γ-glutamyl-transferase.

**Conclusion** Physiological changes in ECM stiffness lead to alterations in HPC morphology, proliferation and differentiation. Increased ECM stiffness (as would be encountered in an injured or fibrotic liver) promotes HPC proliferation and expression of the HPC/biliary marker cytokeratin-19. In contrast, a low-stiffness environment is associated with a reduction in cell proliferation and up-regulation of hepatocyte-specific markers. These results suggest that mechanical factors, such as ECM stiffness might regulate HPC responses following liver injury.

**Competing interests** None declared.

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**BSG INFORMATION GROUP SYMPOSIUM & FREE PAPERS: “SOCIAL MEDIA AND APPS: NEW OPPORTUNITIES, NEW RISKS”**

**Introduction** Illness blogs are online accounts of the course of a disease: they are unsolicited first person narratives, that are publicly accessible allowing author-reader interactivity. Expressive writing improves quality of life and scores in patients with irritable bowel syndrome. We sought to compare the accessibility and contents of illness blogs written by patients with ulcerative colitis (UC) and Crohn’s disease (CD): hypothesising that psychological distress, reportedly more common in IBD than the general population, and active disease would be the key reasons for a post.

**Methods** Using the search terms “Crohn’s” and “Ulcerative colitis” with “Blog” and the internet search engine Google, we identified 12 consecutive UC and Crohn’s disease illness narratives. All posts written in the preceding year were included. We then undertook a structured quantitative analysis; including an assessment of the readability of posts using the Flesch reading ease (FRE) score and author-reader interactivity. Semi-quantitative analyses of excerpts of the contents of each post were undertaken using Dedoose software to identify narrative themes: wherever possible, for each post, disease activity was recorded.

**Results** 1297 and 1009 excerpts were made from 642 and 499 posts from the illness blogs written by patients with UC and CD, respectively. There were no differences in the mean [SD] number of