exocytosis; cytokine production and cellular response to cytokine stimulus. DSP analysis provided additional spatial transcriptomic data, highlighting differential inflammatory signature expression between the tumour and tumour capsule.

**Conclusion**
Our results demonstrate that the phenotype of tumour endothelium contributes to pathways which promote immune privilege in HCC. Spatial transcriptomics can provide further insight of how endothelial profiling correlates with immune cell infiltration in HCC. We have identified several new genes which need further validation but could be novel therapeutic targets that reprogramme the tumour endothelium and boost the efficacy of current immunotherapies.

**P62**
**PROGNOSTIC VALUE AND POTENTIAL IMMUNOREGULATORY ROLE OF SCARF1 IN HEPATOCELLULAR CARCINOMA**

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The immune microenvironment plays a key role in determining the pathogenesis and progression of hepatocellular carcinoma (HCC). Some immune cell subsets promote tumour growth whilst others drive anti-tumour responses. Elucidating the mechanisms by which distinct immune subsets are recruited to the HCC microenvironment could lead to novel immunotherapies. Immune cell recruitment follows a generalized stepwise process, termed the adhesion cascade. We have previously shown that liver endothelial cells express a number of atypical adhesion molecules, such as scavenger receptors, which contribute to immune cell selectivity within the adhesion cascade and thus shape the hepatic immune microenvironment. Scavenger receptor class F member 1 (SCARF1) is thought to play an important role in the selective recruitment of CD4+ T cells to liver sinusoidal endothelial cells during chronic liver disease; however, its contribution to the pathophysiology of hepatocellular carcinoma (HCC) is currently unknown. In this study, we investigated the expression of SCARF1 in HCC tumours and explored its potential role in the recruitment of CD4+ T cells to the tumour microenvironment.

Using TGCA (The Cancer Genome Atlas) RNA-sequencing datasets, we identified the downregulation of SCARF1 expression in HCC tumours, compared to non-tumorous tissues, and validated these findings with immunohistochemical staining of HCC resection specimens. We next explored the relationship of SCARF1 expression with tumour progression and found that a loss of SCARF1 expression was associated with aggressive tumour biology. Following this, we evaluated the prognostic value of SCARF1 expression in HCC tumours and demonstrated that high SCARF1 expression in HCC tumour tissues correlates with a better overall survival, disease-free survival and progression-free survival. Next, via a combination of TGCA data analysis and dual colour immunofluorescent staining, we confirmed that SCARF1 within HCC was largely associated with tumour endothelial cells. We then undertook flow-based recruitment assays under physiological levels of shear stress with primary liver-derived endothelial cells and purified CD4+ T cell subsets. We demonstrated that SCARF1 mediated a role in the specific recruitment of effector CD4+ T cells (CD4+CD25-) across liver endothelium, rather than immunosuppressive regulatory T cell subsets.

Our data suggests that endothelial SCARF1 expression in tumour biopsies may provide critical prognostic information and may potentially help in selecting HCC patients for immunotherapy trials. Regulating SCARF1 levels could itself be a novel immunotherapeutic approach that re-programmes the microenvironment of HCC tumours by promoting effector CD4+ T cell infiltration.

**P63**
**‘GET TESTED LEEDS’: TESTING FOR HEPATITIS B, HEPATITIS C AND HIV IN AN URBAN EMERGENCY DEPARTMENT VIA NOTIONAL CONSENT**

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**Background**
Both Hepatitis B and C are underdiagnosed and Public Health England (PHE) estimates there are around 143,000 people living with Hepatitis C (HCV) in the UK, with around two thirds of these undiagnosed. In inner cities, the frequency of chronic Hepatitis B (HBV) infection is increasing because of migration from high prevalence areas. With this in mind and NHS England’s plan to eliminate Hepatitis C within the decade, increased testing for the viruses is vital.

‘Get Tested LeEDs’ implemented blood borne virus (BBV) testing in the Emergency Department (ED) from October 2018 to March 2020.

**Method**
Patients attending the ED aged between 16 and 65 and having U&Es taken were offered BBV testing via notional consent (posters and leaflets). HIV, Hepatitis B and Hepatitis C testing was done unless the patient declined or did not have capacity. Viral hepatitis testing included HCV antibody (HCV Ab) and HBV surface antigen (HbsAg). Positive HCV Ab had reflexed HCV RNA and confirmed HbsAg positive had a full serology panel. Positive results were electronically reported to specialist nurses and results given to patients face to face where possible.

**Results (Data from Oct 2018 to July 2019)**
There was a total of 112,479 attendances, with 28,178 (25.1%) having U&Es taken. Of these, 16,053 (57%) had BBV testing. There were 345 HCV Ab positive cases (2.1%) of which 156 (1%) were HCV RNA positive (45% of HCV Ab positive were HCV RNA positive).

Of the HCV RNA positive cases, 76 were new diagnoses, 72 had been lost to follow up and 8 were currently in care. Of the 148 patients with a new diagnosis or lost to follow up, 51 patients so far (34%) have been commenced on treatment for Hepatitis C.

There were also 73 HbsAg positive cases of which 35 were new diagnoses, 34 were currently in care and 4 had been lost to follow up. 24 out of 35 patients with a new diagnosis (69%) were subsequently reviewed in a specialist clinic.

**Conclusion**
The ‘Get Tested LeEDs’ initiative has shown that testing for BBV in the ED, alongside the traditional settings (hostels, prisons and substance misuse clinics), offers additional access to this cohort of the population. However, maintaining contact, adherence to regular follow up and treatment is often difficult in this population. Nevertheless, with recent advances in the treatment of viral hepatitis, every opportunity needs to be taken to identify and treat.