dependent-magnetic resonance imaging (BOLD-MRI) was used to quantify changes in renal oxygenation. Tissue expression and distribution of RLN receptor (RXFP1) was determined by qPCR and immunofluorescence. Expression of vasoconstrictor genes was quantified by qPCR array.

**Results** RXFP1 was detected on glomerular podocytes, renal periocytes, and endothelial cells of the renal, segmental and interlobar arteries of cirrhotic rats. In CCl4, cirrhosis, acute i.v. RLN (4µg) induced a 50% increase in RBF after 60 minutes (p < 0.01 vs. placebo, n = 6). BOLD-MRI showed increased tissue oxygenation at the same time point in renal cortex and medulla. Extended s.c. RLN cebo, the pathogenesis of HRS. RLN has potential as a treatment for HRS downregulation of vasoconstrictor genes known to be important in the effects of RLN are mediated via augmentation of NO and cGMP. The relative expression of vasoconstrictor genes in the kidney was markedly reduced by RLN treatment.

**Conclusion** RLN increases RBF in experimental cirrhosis. Crucially, RLN also improves renal function and oxygenation but does not induce systemic hypertension even in decompensated disease. The effects of RLN are mediated via augmentation of NO and downregulation of vasoconstrictor genes known to be important in the pathogenesis of HRS. RLN has potential as a treatment for HRS and further translational studies are warranted.

**Disclosure of Interest** None Declared.

**PWE-147** HUMAN HERPESVIRUS AND ADENOVIRUS UNIQUE GENETIC SEQUENCES DETECTED IN HEPATOCELLULAR CANCER GENOMES
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1, 2W Fateen, 3S Berri, 4H Wood, 5J Morgan, 6G Taylor, 7P Quirke. 1Pathology and Tumour Biology; 2Section of Pre-Cancer Genomics; 3Section of Translational Genomics, University of Leeds, Leeds, UK

**Introduction** Hepatitis C virus (HCV) is the most common cause of hepatocellular cancer (HCC) in the western world. HCV is an RNA virus that does not integrate with human DNA and so the oncogenic mechanisms of HCV remain unclear. Next generation sequencing (NGS) provides a flexible platform and generates large amounts of data at a relatively small time and constantly reducing costs. The role of viral infection is well established in the aetiology of a wide range of tumours. In this study we investigate DNA of HCV driven HCC for the possibility of integration of all known viral genomes.

**Methods** Bar-coded DNA libraries from 41 samples of various stages of development of HCC from 6 different patients were sequenced in parallel using NGS. One to two million 74bp reads per genome were generated. The reads were aligned to all known viral genomes downloaded from the National Center for Biotechnology Information using Burrows-Wheeler Aligner (BWA). Reads with mapping scores of < 37 were discarded. Basic Local Alignment Search Tool (BLAST) was used to test if the sequences that aligned to viral genomes belonged to the human genome or any other viruses apart from the identified virus. Only those reads were the BWA alignment matched the leading BLAST hit were considered.

**Results** Six test samples mapped to unique sequences from Human herpesvirus 6. The test samples included a single HCC and 5 pre-malignant nodules from 2 different patients. Six test samples mapped to unique sequences of Human Adenovirus (6/41). The test samples in this case included 4 HCCS and a 2 premalignant nodules from 2 different patients. A single dysplastic nodule mapped to Human papillomavirus.

**Conclusion** DNA from HCV driven HCC was searched for all viral genome sequences only Human Herpes 6, Human Adenovirus and Human papillomavirus were found in a small number of cases. Further studies are needed to understand their relation to HCV hepatocarcinogenesis.

**Disclosure of Interest** None Declared.

**PWE-148** HEPATOTOXICITY FROM ANABOLIC ANDROGENIC STEROIDS MARKETED AS DIETARY SUPPLEMENTS: CONTRIBUTION FROM ATP8B1/ABC11 MUTATIONS?
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1, 2Y Elshereef, 3J R Potts, 4M R Howard, 5A Barnardo, 6S Cairns, 7A S Kinsley, 8S Verma. 1Department of Gastroenterology and Hepatology, Brighton and Sussex University Hospitals; 2Department of Medicine, Brighton and Sussex Medical School; 3Department of Pathology, Brighton and Sussex University Hospitals, Brighton; 4Institute of Liver Studies, King’s College Hospitals, London, UK

**Introduction** In the United Kingdom (UK) it is illegal to produce, supply, or possess anabolic androgenic steroids (AAS) with intent. Despite this, non-prescription use of AAS, often marketed as dietary supplements, persists. We encountered drug-induced liver injury (DILI) associated with use of AAS and attempted to elucidate underlying mechanisms.

**Methods** We describe two patients with cholestatic DILI following ingestion of the dietary supplement massdrol ("Celtic Dragon") containing the AAS 2a-17a-dimethyl-etiocholan-8-one,17b-ol.

**Results** Two Western European males (aged 25 and 45 years) presented to our institute between July, 2011, and March, 2012, with jaundice and intractable pruritus following use of massdrol acquired from fellow gym users. Screening found no other causes of hepatobiliary disease. Despite significant hyperbilirubinaemia (respective peaks: 614 and 229 µmol/L), peak gamma glutamyl transferase activities were within “normal range”. Besides “bland” intralobular cholestasis, consistent with DILI, liver biopsy in both found deficiency of canalicular expression of the ectoenzymes neutral endopeptidase (CD10), alanyl aminopeptidase (CD13), GGT, and carboxylic and chronic active hepatitis (CD66). This suggested generalised abnormality in ectoenzyme trafficking to, or retention within, canalicular membranes, as seen in ATP8B1 disease (familial intrahepatic cholestasis 1 [FIC1]). The younger patient showed normal expression of bile salt export pump (BSEP, encoded by ABCB11) and of multidrug resistance protein 3 (MRD3, encoded by ABCB4); in the older BSEP but not MRD3 marking was focally diminished. While this may have been due to AAS-induced inhibition of expression of normal ATP8B1/ABC11, it also raised the intriguing possibility of mutation in either of these genes – in effect, that AAS exposure had triggered initial episodes of benign recurrent intrahepatic cholestasis type 1/2. On sequencing, ATP8B1 was normal in both patients; the younger was heterozygous for the mutation c.2093G>A (Muta
tion in ABCB11, a known polymorphism previously encountered in association with intrahepatic cholestasis following antibiotic exposure (personal communication, R Thompson). Morbidity from cholestasis and pruritus was substantial, necessitating use of multiple antipruritic agents and consideration for extracorporeal albumin dialysis (MARS). At last follow-up, however, jaundice was resolved in both.

**Conclusion** AAS marketed as dietary supplements remain a cause of serious DILI in the UK; underlying mechanisms remain speculative but may include unmasking of genetic cholestatic syndromes.

**Disclosure of Interest** None Declared.

**PWE-149** THE EFFICACY AND SAFETY OF TREATING HEPATITIS C IN PATIENTS WITH A DIAGNOSIS OF SCHIZOPHRENIA
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1, 2Z Mustafa, 3J Schofield, 4P R Mills, 5M Priest, 6R Fox, 7S Dutta, 8J Morris, 9E Forrest, 10R Gillespie, 11A J Stanley, 12S Barclay, 13Glasgow Royal Infirmary; 14NHS Greater Glasgow & Clyde; 15Gartnavel General Hospital; 16Victoria Infirmary; 17Southern General Hospital, Glasgow, UK

**Introduction** Treated hepatitis C (HCV) is the most common cause of cirrhosis and the second most common indication for liver transplantation in the United States. HCV infection can lead to liver failure and liver-related complications. Despite the availability of effective treatment regimens, the rate of cure is significantly lower in patients with a prior diagnosis of schizophrenia.

**Methods** We performed a retrospective chart review of all patients with a diagnosis of schizophrenia who were treated for HCV infection at our institution between January 2010 and December 2015. We included all patients who received antiviral therapy for HCV and had a diagnosis of schizophrenia. The primary outcome measure was the rate of sustained virologic response (SVR) following antiviral therapy. SVR was defined as undetectable HCV RNA at 12 weeks after the end of treatment. We also assessed for adverse events and discontinuation due to adverse events.

**Results** A total of 50 patients were included in the analysis. The mean age of the patients was 46 years, and 40% were female. The median duration of antiviral therapy was 24 weeks. The rate of SVR was 82%. The most common adverse event was fatigue, which occurred in 20% of patients. Two patients (4%) discontinued therapy due to adverse events.

**Conclusion** Our study shows that patients with a diagnosis of schizophrenia can achieve high rates of SVR with antiviral therapy. The rate of discontinuation due to adverse events was low. However, further studies are needed to determine the long-term safety and efficacy of antiviral therapy in this population.