progressors. Importantly, no significant difference in the proportion exhibiting fibrosis progression was found between those with NAFL or NASH at index biopsy (10/27 (37%) vs. 36/83 (43%) p = 0.65). 12/27 (44%) with NAFL at baseline progressed to NASH at follow-up biopsy, whereas 6/75 (8%) with NASH regressed to NAFL. Weight change was a significant factor associated with inter-biopsy change in disease activity measured by NAFLD activity score (r = 0.23 p = 0.026). Of 10 patients with NAFL who had fibrosis progression, 3 progressed by 1 stage, 5 by 2 stages and 2 by 3 stages; all had NASH on the follow-up biopsy. Of concern, 6 of 27 (22%) patients with baseline NAFL had reached stage 3 fibrosis at the follow up biopsy, but none were cirrhotic. Among the patients with NAFL, 80% of those who had fibrosis progression were diabetic at the time of follow-up liver biopsy compared with 25% of non-progressors (p = 0.005). The FIB-4 score was the only significant baseline factor that predicted fibrosis progression (OR 2.1 [95% CI: 1.1–3.9], p = 0.02). However, the AUROC was only 0.63 (p = 0.04).

Conclusion Contrary to current dogma, this study suggests that NAFL is not entirely benign and has the potential to progress to NASH and clinically significant fibrosis, particularly if patients develop diabetes.

Disclosure of Interest None Declared.

**BMSG 2014 abstracts**

**PTH-091 TESTING FOR HEPATITIS C IN HIGH RISK IMMIGRANTS – FINDINGS FROM A SINGLE PRACTICE WITH A LARGE IMMIGRANT POPULATION**

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Introduction Chronic Hepatitis C virus (HCV) is a major cause of liver cirrhosis with a worldwide prevalence of 3%. The UK prevalence is between 0.4–1.0% however pockets of higher prevalence will exist in areas with large immigrant populations. Chronic liver disease in HCV infected patients is costly to the NHS. The Hepatitis C Action plan recommends all ethnic minorities from countries where HCV is endemic be offered screening; however testing of this group remains haphazard. Our aims were to determine the number of high risk individuals tested for HCV by interrogation of a Primary Care database in a single GP surgery located in an area with a large immigrant population. Secondary aims include establishing the reason for testing, prevalence of HCV in the tested population and treatment outcomes.

Methods We used 4 search terms in the primary care database SystmOne to identify our target population: age (>18), ethnic code, place of birth and language spoken. We then applied Read Codes pertaining to HCV to this population to determine the number already tested. The electronic medical records of all individuals tested positive for HCV were reviewed to answer secondary aims.

Results There were 4256 individuals registered age >18. 75% (3210) qualified as the target population, 18% (718) were excluded because of lack of demographic data, 7% (328) originated from low risk countries. We identified 16 read codes pertaining to HCV and these generated 247 ‘hits’ and identified that 6% of the target population had been tested for HCV (115M, 79F). Indications for testing were: isolated raised ALT/bilirubin/ALP/AST 45%, contact testing 12%, mixed raised LFTs 9%, generally unwell 9%, screening pre DMARD therapy 7%, illicit drug use 7%, patient request 3%, other indication 3%, indication unknown 2%, medical intervention overseas 1%, other abnormal bloods 1%. Proactive screening took place in 1%. The prevalence of HCV in the tested population was 7.7% (15/194 M, 6F). 73% (11/15) received treatment, 9/11 (82%) achieved an SVR, 1/11 (9%) was termed ‘non-responder’ but achieved SVR on re-treatment, 1/11 (9%) had no response to treatment and the course terminated prematurely, with subsequent spontaneous clearance of the virus. 4/15 had not received treatment: 2 patients were considered high risk for treatment in view of co-morbidities, 1 failed to attend appointments and 1 was recently diagnosed.

Conclusion This study confirms that testing is reactive rather than proactive highlighting the need for a screening programme dedicated to high risk populations. GP work load, prioritisation of chronic diseases forming part of QOF and poor understanding of HCV all exist as possible barriers to screening.

Disclosure of Interest None Declared.

**Pancreas**

**PTH-092 INVESTIGATING THE ROLE OF PHYSICAL ACTIVITY IN PANCREATIC CANCER – THE AGE AT WHICH THIS IS MEASURED IS IMPORTANT IN AETIOLOGICAL STUDIES AND IS INDEPENDENT OF BODY MASS INDEX**

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Introduction There are plausible biological mechanisms for how increased physical activity (PA) may prevent pancreatic cancer, although most studies do not report an inverse association. We investigated whether this may be related to the age at which PA is measured, using a validated questionnaire, and whether the effect of PA is independent of body mass index (BMI).

Methods 23,639 participants, aged 40–74 years were recruited into the EPIC-Norfolk cohort study between 1993 and 1997. These participants completed validated questionnaires on both occupational and leisure time PA. From this, four levels of PA index were derived. The cohort was monitored for up to 17 years to identify those participants who developed pancreatic cancer. The hazard ratios (HRs) of developing cancer were estimated using Cox regression and adjusted for covariates (age, gender, cigarette smoking status and type 2 diabetes). Each analysis was first performed in those recruited of all ages and then in those younger and older than 60 years at recruitment.

Results Within 17 years, 88 participants developed pancreatic cancer (55% female, median age of diagnosis 73 years, range 52–89 years). There was no association between PA and risk of pancreatic cancer in the whole cohort (hazard ratio (HR) = 1.03, 95% CI: 0.84–1.27). However, in those recruited at younger than 60 years (n = 29 cases), higher levels of PA were associated with a decreased risk (highest vs. lowest category HR = 0.27, 95% CI: 0.07–0.99, trend HR = 0.75, 95% CI: 0.53–1.06, p = 0.11). When BMI was included, the associations were similar (highest vs. lowest category HR = 0.25, 95% CI: 0.07–0.93, trend HR = 0.73, 95% CI: 0.51–1.03, p = 0.08). In participants aged greater than 60 years (n = 59 cases), higher PA was associated with a non significant, increased risk both when BMI was unaccounted for
(highest vs. lowest category HR=1.98, 95% CI: 0.94–4.16, p = 0.07, trend HR=1.23, 95% CI: 0.96–1.57, p = 0.10) and when BMI was included (trend HR=1.21, 95% CI: 0.94–1.55, p = 0.13).

Conclusion The association between PA and cancer risk is dependent on the age at which PA is measured. This possibly reflects occupational activity and differences in general medical health with age or residual confounding. The associations were similar when adjusted for BMI, suggesting an independent mechanism of PA. If the inverse association of increased PA in younger participants is causal, one in six cases of pancreatic cancer might be prevented by encouraging more PA. Aetiological studies should measure PA at different ages when investigating pancreatic cancer.

Disclosure of Interest None Declared.

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