Aims & Methods: The stool and serum samples from 52 NAFLD patients and 38 healthy controls were collected. qPCR analysis of Akkermansia muciniphila, Faecalibacterium prausnitzii, Lactobacillus spp., Bifidobacterium spp., and Bacteroides fragilis group was performed. Serum endotoxin levels were also determined by Chromogenic LAL Assay. Dietary habits were analysed by nutritional questionnaires.

Results: Akkermansia muciniphila and Bacteroides fragilis group were significantly lower in patients with NAFLD as compared with the healthy control (p < 0.0001). No significant difference was determined in terms of Lactobacillus spp., Bifidobacterium spp. and Faecalibacterium prausnitzii counts. Moreover, significantly elevated endotoxin levels were determined in NAFLD patients (9.04 EU/mL in NAFLD group; 2.75 EU/mL in control group; p < 0.005).

Conclusion: Akkermansia muciniphila and Bacteroides fragilis group has been known to have beneficial effects on gut barrier function. These two bacterial groups were decreased in Turkish NAFLD patients. Decreased levels of these bacteria were also shown in metabolic syndrome, which is frequently associated with NAFLD. NAFLD patients have also increased endotoxin levels which indicate a translocation of bacterial products as a result of increased gut permeability.

Disclosure of Interest: All authors have declared no conflicts of interest.

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and quickness of hepatocyte repopulation in liver were higher in the group of animal after PH. Number of desmin + cells increased more in the experimental group compared with the control one. In both groups a SMA-positive cells were not detected.

Conclusion: Genetically modified HSCs, which is transduced by RGF and FGF-4 genes, save their viability after transplantation into the rat after PH, migrate and integrate into the liver parenchyma. These cells have a positive influence on the process of liver regeneration without the risk of liver fibrosis.

Disclosure of Interest: All authors have declared no conflicts of interest.

P0018 ANGIOTENSIN II TYPE 1 RECEPTOR GENE A1166C POLYMORPHISM IS ASSOCIATED WIT NON ALCOHOLIC FATTY LIVER DISEASE AND PREDICTS ITS SEVERITY

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Introduction: The pathogenesis of non alcoholic fatty liver disease (NAFLD) has not been well demonstrated yet, however, genetic predisposition is probably of major importance. Angiotension II type I receptor (AGTR1) have been known to be involved in the process of liver fibrosis and metabolic syndrome.

Aims & Methods: This study aimed to investigate the association between AGTR1 A1166C polymorphism and NAFLD. A cross-sectional study was conducted between March 2014 and March 2015 among healthy adult individuals referred to our radiology clinic for abdominal ultrasonography. NAFLD was diagnosed by an expert radiologist based on the presence of these ultrasonographic findings: hepatorenal echo contrast, liver brightness, deep attenuation, vascular blurring and the absence of hepatitis B surface antigen or antibody tests, 1) high hepatitis C virus, 2) alcohol consumption (>20 g/day), 3) history of other causes of liver disease, and 4) medications known to produce fatty liver disease during the last six months prior to the study. Participants’ characteristics and their lab data including liver function tests, lipid profile, fasting plasma glucose (FPG), were also recorded. AGTR1 A1166C polymorphism was checked in subjects with NAFLD and healthy controls using TaqMan allelic discrimination method.

Results: Eighty subjects with NAFLD were compared with 88 individuals without NAFLD. Mean of all anthropometric indices including BMI, waist height, waist circumference and hip circumference were significantly higher in subjects with NAFLD compared to those without NAFLD (P < 0.05). Mean total cholesterol was significantly higher in subjects with NAFLD in comparison to the controls in univariate analysis (P = 0.018). Higher serum ALT was also a predictor of NAFLD (38.56 ± 17.61 versus 20.76 ± 6.40 IU/L) (P = 0.0001). Metabolic syndrome was detected in 31 (53.44%) individuals in NAFLD group and in 27 (19.01%) in control group (P < 0.001) (OR: 3.31, 95% CI: 1.84-6.66). Multivariate logistic regression analysis of risk factors showed that body mass index (BMI), metabolic syndrome, waist circumference, hip circumference and serum ALT were independent predictors of NAFLD in our study population. The frequency of AA and CC genotypes of AGTR1 gene was significantly higher in patients with NAFLD compared to controls (P = 0.029 and P = 0.042 respectively). Furthermore, C allele was more detected in subjects with NAFLD compared to healthy controls (OR: 2.1; 95% CI: 1.23-3.61, P = 0.006). CC genotype (OR: 10.62, 95% CI: 1.05-106.57, P: Value: 0.045) and C allele (OR: 6.81-95% 5C1: 1.42- 32.48, P:Value: 0.016) were also predictors of severe fatty liver disease in our study population.

Conclusion: Our results provide the first evidence that AGTR1 gene A1166C polymorphism not only is associated with NAFLD and but also may predict its severity. Multivariate regression analysis for the NAFLD predictors.

OR (95% CI) P-value

<table>
<thead>
<tr>
<th>Body mass index (BMI)</th>
<th>OR (95% CI) P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>7.74 (1.25-3.73)</td>
<td>0.005</td>
</tr>
<tr>
<td>Waist circumference</td>
<td>9.26 (0.68-0.92)</td>
</tr>
<tr>
<td>Hip circumference</td>
<td>5.21 (1.02-1.39)</td>
</tr>
<tr>
<td>Metabolic syndrome</td>
<td>15.21 (9.74 -14.43)</td>
</tr>
<tr>
<td>Alamineaminotransferase (ALT)</td>
<td>26.46 (1.19-1.50)</td>
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</tbody>
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Disclosure of Interest: All authors have declared no conflicts of interest.

P0019 PATIENTS WITH POLYCYSTIC LIVERS MORE THAN TWO TIMES THE NORMAL SIZE ARE LIKELY TO DEVELOP SYMPTOMS

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Introduction: Progressive growth of hepatic cysts can lead to symptomatic hepato-megaly in polycystic liver disease (PLD).

Aims & Methods: Our primary aim was to determine at which threshold of liver volume patients become symptomatic. As a secondary objective we investigated which symptoms are associated with higher liver volume. We used the PLD questionnaire (PLD-Q), a validated questionnaire that assesses frequency and discomfort of PLD-related symptoms, to determine the symptom burden.

In this study and 159 PLD patients that have completed the PLD-Q and rated themselves as symptomatic or not (NCT02173080), we have defined the PLD-Q cut-off value of being symptomatic with receiver operating characteristic (ROC) analysis. The optimal PLD-Q cut-off score was 31 points with an area under the curve (AUC) of 0.832 (p < 0.001).

Next, we used baseline data of PLD patients from two prospective studies (DIPAK observational study and CURSOR randomized controlled trial (NCT02021110). All patients completed the PLD-Q, and had liver volumes measured with CT or MRI. In order to determine the liver volume cut-off value for being symptomatic, we used the PLD-Q cut-off value from the previous step in another ROC analysis with liver volume as independent variable. Spearman correlations were calculated for liver size and symptoms.

Results: We included 82 of the 131 patients from the prospective studies (main exclusions: no PLD n = 26, no PLD-Q n = 7 or no imaging n = 8). Most patients were female (n = 67) with a mean age of 48 years. Median liver volume was 3879 mL (IQR: 2452 – 5891). Cut-off liver volume for being symptomatic was 3472 mL (AUC 0.805, p < 0.001) with a sensitivity of 80% and a specificity of 73%. This cut-off volume has a positive and negative predictive value of 66% and 82% respectively. Disatisfaction with body size was strongly correlated with liver volume (r = 0.63). Furthermore, pain in rib cage, shortness of breath, limited mobility, anxiety about the future and, problems with intercourse correlated moderately (r = 0.40-0.59). There was a weak correlation with lack of appetite, pain in side and tiredness (r = 0.20-0.39). Nausea (r = 0.17, p = 0.146) and abdominal pain (r = 0.17) were not correlated to liver size.

Conclusion: Patients with liver volumes equivalent to two times the normal size are likely to develop PLD-related symptoms. In patients with smaller livers, other causes that lead to similar symptoms should be considered. Most PLD-related symptoms are associated with larger liver volume, except for nausea and abdominal pain.

Disclosure of Interest: All authors have declared no conflicts of interest.

P0020 LncRNA PROFILE IN NAFLD AND IDENTIFICATION OF A PROTECTIVE NOVAL LncRNA FLRL2 IN NAFLD

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Introduction: Non-alcoholic fatty liver disease (NAFLD) is one of the most prevalent chronic liver diseases worldwide with unclear mechanism. Long non-coding RNAs (lncRNAs) have recently emerged as important regulatory molecules in liver diseases.

Aims & Methods: To further understand the pathogenesis of NAFLD, IncRNA and mRNA microarray was conducted in NAFLD mice model. Potential target genes of significantly changed IncRNA were predicted using bioinformatic algorithms, followed by Gene Ontology analysis and KEGG pathway enrichment analysis. NAFLD mice model and NAFLD AML12 cell model were used in further experiments. Real-time qPCR and Western Blot were adopted in evaluating IncRNA and mRNA expression with certain silence LncRNA or shRNA transfection. The knockdown with FLRL2 was strated to be likely a key player in circadian rhythm targeting Per3, Per2 and Arntl. While FLRL8, FLRL3 and FLRL7 showed their potential role in PPAR signaling pathway through interaction with Fabp5, Lpl and Fads2. Mechanism of FLRL2 as well as its potential target circular rhythm gene Arntl was inves-

ized.

Disclosure of Interest: All authors have declared no conflicts of interest.