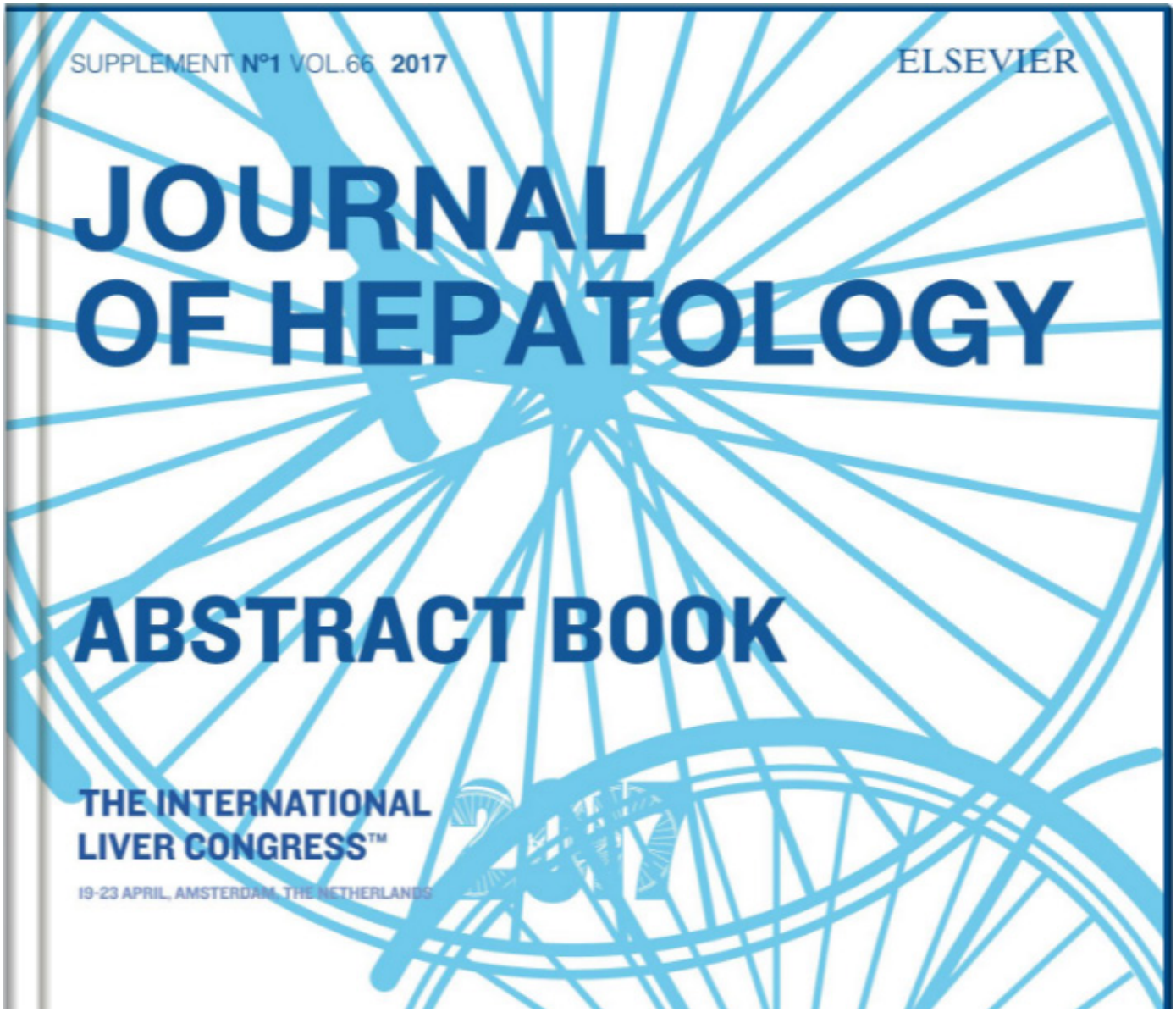

FRI-332

A prospective study to evaluate the efficacy of a standardized low calorie diet according to PNPLA3 genotype in patients with Non Alcoholic Fatty Liver Disease (NAFLD) – 2 months data (interim analysis)

M. Teufelhart[1], H. Hofer[2], H. Haslacher[3], R. Winker[1], B. Meyer[1], P. Ferenci[4], B. Mehl[1], C. Rabitsch[1], S. Nistler[1], M. Trauner[4], T.-M. Scherzer[1]. *[1]Health and Prevention Center, Sanatorium Hera, Vienna; [2]Internal Medicine I, Klinikum Wels, Wels; [3]Laboratory Medicine; [4]Internal Medicine III, Medical University of Vienna, Vienna, Austria*

E-mail: Manuela.teufelhart@hera.co.at

Background and Aims: Steatosis can lead to Non Alcoholic Steatohepatitis, liver cirrhosis, hepatocellular carcinoma as well as aggravation of diabetes and cardiovascular diseases. PNPLA3 is associated with NAFLD. The HEPAFAST® protein shake is developed for the treatment of NAFLD and is characterized by the addition of liver protective substances, such as ß-glucan and cholin. Aim of the study is to analyze the efficacy of a standardized low calorie diet (HEPAFAST®, Bodymed, Germany) and the influence of PNPLA3 genotypes on treatment outcome.

Methods: In this study 81 non cirrhotic patients are stratified according to PNPLA3 genotypes (27 patients per group;CC,CG,GG). All patients receive HEPAFAST® shakes for 2 weeks (1,000 kcal/day) and instructions to follow a low glycemic and insulinemic (LOGI) diet monitored with food diaries for another 6 weeks (EOT). All patients are seen for four time points (baseline, week 2, 2 months and 6 months-follow up). At each time point liver fat content is assessed by a blinded investigator with Fibroscan CAP. Additionally Fatty Liver Index (FLI), waist circumference (WC), BMI and Vitamin D are measured.

Results: To date 38 patients [age 46 ± 9.3 (mean ± SD); m:26,f:12; PNPLA3 genotypes:CC:20,CG:12,GG:6] finished the HEPAFAST® therapy from baseline (bl) to week 2 (wk2), 33 patients completed the LOGI diet for another 6 weeks. All outcome variables significantly decreased from bl to wk2 and further on LOGI diet till EOT: (1) Fibroscan CAP 319.6 ± 29.6 dB/m^2 to 268.9 ± 34.2 ($p < 0.001$) and 244.6 ± 30.6 ($p < 0.001$), (2) FLI 77.4 ± 16.6 to 56.3 ± 20.9 ($p < 0.001$) and 58.4 ± 21.4 ($p < 0.001$), (3) WC 108.5 ± 8.5 cm to 104.8 ± 8.5 ($p < 0.001$) and 102.0 ± 8.5 ($p < 0.001$), (4) BMI 31.8 ± 3.7 to 30.3 ± 3.4 ($p < 0.001$) and 29.6 ± 3.4 ($p < 0.001$), (5) Vitamin D 22.2 ± 9.0 ng/mL to 25.7 ± 9.1 ($p < 0.001$) and 28.6 ± 9.5 ($p < 0.001$). HEPAFAST® was well tolerated. 7 patients finished 6 months follow up, CAP remained low at 250.3 ± 52.2 ($p < 0.019$). Evaluation according to PNPLA3 genotype revealed a lower drop of CAP in CC compared to G-allele carriers till wk 2 [CC:310.8 ± 29.3 to 268.7 ± 40.1 (Δ42.1 ± 34.5) vs. G-allele:329.4 ± 27.4 to 269.1 ± 27.6(Δ60.3 ± 26.6); $p = 0.12$] and EOT [CC:312.2 ± 29.3 to 238.2 ± 33.1 (Δ74.0 ± 30.1) vs. G-allele:335.9 ± 24.8 to 253.3 ± 25.5(Δ82.6 ± 23.8); $p = 0.7$].

Conclusions: A liver specific HEPAFAST® diet is a very effective strategy to significantly lower liver fat in NAFLD patients. To date this effect seems to be more pronounced in patients carrying the PNPLA3 G-allele. CAP and Vitamin D further improve on LOGI diet.