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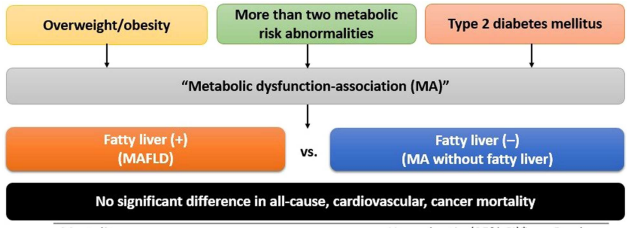
Background: A novel term, metabolic dysfunction-associated fatty liver disease (MAFLD), was proposed by a group of experts. However, it remains unclear whether hepatic steatosis *per se* in MAFLD contributes to an elevated risk of mortality in individual's with overweight/obesity, metabolic dysregulation, or type 2 diabetes mellitus (DM) (referred to as the metabolic dysfunction-association (MA) group), which are known significant risk factors for increased mortality. This study aimed to compare all-cause and cause-specific mortality between the 'MAFLD' group and the 'MA without fatty liver' group. **Methods:** A total of 10,052 participants from NHANES III were included. Fatty liver was diagnosed using ultrasound, and MAFLD was defined based on the criteria proposed by an international panel of experts. Mortality risks were compared between the 'MAFLD' group and the 'MA without fatty liver' group using the Cox proportional hazards model with complex survey design weights, adjusted for demographic and anthropometry factors (age, sex, race, body mass index and waist circumference), social history (education, marriage status, smoking and alcohol history and exercise) and comorbidity variables (hypertension, DM and hyperlipidemia). Linked mortality data, including all-cause, cancer, cardiovascular, and other causes-related mortality, were examined from 1988 through 2019. For liver-related mortality, data were evaluated from 1988 through 2006. **Results:** Over an average follow-up period of 23.0 years, the 'MAFLD' group did not exhibit a significant increase in all-cause mortality (adjusted hazard ratio [95% confidence interval], 1.07 [0.99-1.16], $P = 0.100$), cancer mortality (1.08 [0.86-1.36], $P = 0.508$), or cardiovascular mortality (0.89 [0.78-1.02], $P = 0.084$) compared to the 'MA without fatty liver' group. However, other causes-related mortality, which included liver-related mortality, was higher in the MAFLD group (1.36 [1.14-1.62], $P = 0.001$). This trend persisted in sensitivity analyses conducted on participants without viral hepatitis or heavy alcohol consumption. No significant effect modification was observed according to subgroups. Liver-related mortality, assessed over a 13.8-year follow-up period, showed a marginal increase in the 'MAFLD' group (2.49 [0.99-6.23], $P = 0.052$) compared to the 'MA without fatty liver' group. **Conclusion:** The 'MAFLD' group did not demonstrate an elevated risk of all-cause, cardiovascular, or cancer mortality when compared to the 'MA without fatty liver' group. However, there was a trend toward an increased risk of liver-related mortality in the MAFLD group.



Mortality	Hazard ratio (95% CI)*	*P*-value
All-cause mortality	1.07 (0.99-1.16)	0.100
Cancer mortality	1.080 (0.86-1.36)	0.508
Cardiovascular mortality	0.89 (0.78-1.02)	0.084
Diabetes mellitus-related mortality	1.61 (0.98-2.65)	0.059
Other mortality (including liver-related mortality)	1.36 (1.14-1.62)	0.001
Liver-related mortality**	2.49 (0.99-6.23)	0.052

*Adjusted for age, sex, race, marital status, education, ever-smoking, heavy alcohol consumption, exercise, hypertension, diabetes, hyperlipidemia, waist circumference and body mass index
**Follow-up until 2006

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The following people have nothing to disclose: Min-Sun Kwak, Hyunseok Kim, Mazen Noureddin

2179-A | FIRST RESULTS OF THE EVOLUTION AND THE INFLUENCE OF STATIN TREATMENT ON NAFLD IN A PRIMARY CARE COHORT OVER A 2-YEAR PERIOD

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Background: Non-alcoholic fatty liver disease (NAFLD) has become the most frequent cause of chronic liver disease. Non-alcoholic fatty liver can evolve into non-alcoholic steatohepatitis, fibrosis, cirrhosis, and hepatocellular carcinoma. Data concerning the evolution of NAFLD in a primary care continuum remain scarce. As a therapeutic option, besides lifestyle changes, statins might influence the evolution of steatosis or fibrosis.

Symbols: ♦, Poster of Distinction; ★, Foundation Award Recipient

Therefore, we aimed to map the evolution of NAFLD and the influence of statin treatment in a primary care (PC) cohort assessed by liver stiffness (LSM) and controlled attenuation parameter (CAP™). **Methods:** In a prospective, multicentric cohort study in two large PC practices in Belgium between October 2020 and May 2023, a FibroScan® measurement (for assessment of steatosis by CAP™ and of LSM as a surrogate for fibrosis) and clinical examination (waist circumference (WC) and BMI) was performed at baseline and follow-up. Steatosis was defined as a CAP™ value > 215 dB/m. At the start, lifestyle advice was given. Recent laboratory data, medical background, and medication for both study visits were gathered from the electronic patient file. **Results:** Of the 67 study participants evaluated, 11 (16.4%) were excluded due to treatment with tamoxifen, not being sober, alcohol abuse, or IQR/MED > 30%. In total, 56 (83.6%) participants were included, of whom 21 (37.5%) were men, 55 (94.0%) were of Caucasian origin, and 40 (71.7%) had steatosis. The mean age, median BMI, and mean WC at baseline were 62 ± 10 years, 26.3 ± 6.0 kg/m², and 91.3 ± 12.2 cm, respectively. At follow-up, WC (91.7 ± 12.5 vs. 95.9 ± 11.2 cm; $p < 0.001$) and CAP™ (251.4 ± 62.8 vs. 261.2 ± 56.0 dB/m; $p = 0.005$) were significantly higher while BMI remained unchanged ($p = 0.098$). LSM and the serum level of triglycerides decreased significantly (5.2 ± 2.3 vs. 4.3 ± 1.5 kPa; $p = 0.021$ and 102 ± 62 vs. 87 ± 37 mg/dl; $p = 0.008$). No statistical differences were found for the liver enzymes AST, ALT, and GGT. Twenty of the 56 (64.3%) participants took statins and 2 (3.6%) fibrates. No statistical difference between baseline and follow-up was seen for CAP™ for statin users (246.5 ± 64.1 vs. 265.4 ± 58.1 dB/m; $p = 0.266$) and non-users (254.3 ± 62.9 vs. 258.7 ± 55.9 dB/m; $p = 0.691$). Non-statin users saw a significant decrease in LSM (5.5 ± 2.6 vs. 4.4 ± 1.3 kPa; $p < 0.001$) at follow-up, which was not seen with statin users (5.3 ± 2.0 vs. 4.3 ± 2.2 kPa; $p = 0.179$). **Conclusion:** Overall, we saw a decrease in fibrosis and triglycerides during the two-year follow-up time in a Caucasian PC cohort. No study participant developed decompensated liver disease. However, we did see an increase in steatosis accompanied by an increase in waist circumference, although lifestyle advice was given during the first visit. Moreover, statin use did not influence steatosis or fibrosis evolution, though future research is warranted to further investigate the influence of statin treatment on NAFLD.
Disclosures: Sven Francque – Inventiva: Consultant, No, No; Eisai: Consultant, No, Yes; Siemens Healthcare: Speaking and Teaching, No, Yes; Novo Nordisk: Speaking and Teaching, No, Yes;
The following people have nothing to disclose: Leen Heyens, Wouter Robaeys, Liesbet Vernijns, Anneleen Robaeys, Geert Robaeys

2180-A | FOOD INSECURITY IS ASSOCIATED WITH LOWER NAFLD PREVALENCE BUT GREATER LIVER FIBROSIS IN PEOPLE WITH HIV

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Background: Food insecurity, defined as the economic or social condition of limited or uncertain access to nutritionally adequate foods, is a growing public health problem in the US. In recent years, it has emerged as a risk factor for nonalcoholic fatty liver disease (NAFLD) and advanced liver fibrosis in the general population. However, little is known about the impact of food insecurity on liver disease in people with HIV (PWH). We aimed to examine associations between food insecurity and NAFLD and liver fibrosis prevalence in a diverse multicenter cohort of PWH. **Methods:** PWH aged 20 years on suppressive antiretroviral therapy, HIV RNA < 200 copies/mL, and without chronic viral hepatitis or other known cause of liver disease were screened for NAFLD and fibrosis by vibration controlled transient elastography at 8 US centers. NAFLD was defined as CAP ≥ 263 decibels/m in the absence of self-reported heavy alcohol use and advanced fibrosis was defined as liver stiffness measurement (LSM) ≥ 10 kPa. Food security was measured using the validated Six-Item Short Form US Household Food Security Survey Module, and participants were categorized as being food secure or food insecure. We used multivariable logistic regression to estimate odds ratios (OR) and 95% confidence intervals (CI) of NAFLD and advanced fibrosis by food security status. **Results:** Among 570 PWH, mean age was 54 years, 410 (72%) were male, 26% White, 49% Black, 21% Hispanic, 267 (47%) had BMI ≥ 30 kg/m², and 171 (30%) were diabetic. NAFLD was present in 306 (54%) and advanced fibrosis in 45 (8%) of participants. Food

Symbols: ♦, Poster of Distinction; ★, Foundation Award Recipient