CRC cases developed. NAFLD and MAFLD were each associated with a significantly higher risk of developing CRC. When the Neither-FLD group was the reference, multivariable-adjusted hazard ratios (95% confidence interval) for CRC were 1.16 (1.06-1.28) in the NAFLD-only group, 1.18 (1.16-1.20) in the Both-FLD group, and 1.32 (1.28-1.35) in the MAFLD-only group. The presence of advanced liver fibrosis further increased CRC risk in each FLD group.

Conclusions FLD was associated with a higher risk of CRC development. CRC risk was higher in the presence of MAFLD, especially when accompanied by liver fibrosis

IDDF2021-ABS-0137 THE aMAP SCORE PREDICTS RISK OF HEPATOCELLULAR CARCINOMA DEVELOPMENT IN PATIENTS WITH ALCOHOL-RELATED CIRRHOSIS

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Background The aMAP score (derived from age, male sex, albumin and platelet count) was recently devised to predict hepatocellular carcinoma (HCC) development. It was shown to have excellent performance irrespective of aetiology and ethnicity. Only one cohort of non-viral hepatitis patients (Japanese with mostly non-alcoholic fatty liver disease) was included in the validation set. Hence, the performance of the aMAP score among Caucasians with alcohol-related cirrhosis (ALC) is unknown. We aimed to validate the aMAP score in a cohort of ALC patients.

Methods Participants with ALC from three centres enrolled to a genome-wide association study (Schwantes-An et al. *Hepatology* 2021) were included. All participants had a history of high alcohol consumption. Cirrhosis was defined clinically (portal hypertension or decompensation), using Fibroscan and/or histology. Patients were followed until last liver imaging, HCC development, liver transplantation, or death with the latter two adjusted as competing risks.

Results 270 ALC patients were included: male (72.2%), Caucasian (98.9%), with median age of 56 years, and

compensated cirrhosis (median Child-Pugh score 5). The median aMAP score was 60: 12.6% low-risk (score 0-50), 35.2% medium-risk (50-60) and 52.2% high-risk (>60). After a median follow-up of 41 months, 13 patients developed HCC, 25 received liver transplantation and 106 died. The aMAP score predicted HCC development (hazard ratio 1.11, 95% confidence interval 1.05-1.18, $P<0.001$, IDDF2021-ABS-0137 Figure 1). The C-index for the aMAP score for predicting HCC development was 0.83, 0.78 and 0.80 at one, three and five years respectively which was similar to ADDRESS-HCC (Flemming et al. *Cancer*. 2014) and Ioannou et al.'s (*J Hepatol*. 2019) scores (IDDF2021-ABS-0137 Figure 2A-C). We then examined the predictive value of three genome-wide association-significant risk-increasing alleles (PNPLA3:rs2294915, TM6SF2:rs10401969, HSD17B13:rs10433937) using a genetic risk score. A three-gene score did not predict HCC development at five years (C-index 0.53) or improve the aMAP score performance when combined.

Conclusions We validated the excellent performance of the aMAP score in a cohort of ALC patients and affirmed its applicability across wider aetiologies.

Abstract IDDF2021-ABS-0137 Figure 1

Abstract IDDF2021-ABS-0137 Figure 2