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Authors: Jerzy Jaroszewicz, Karol Żmudka, Marlena Dubatówka, Małgorzata Chlabicz, Paweł Sowa, Urszula Roszkowska, Damian Piotrowski, Karol Kamiński

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Metabolic dysfunction–associated steatotic liver disease and metabolic dysfunction–associated alcohol-related liver disease represent distinct clinical phenotypes and fibrosis risk: insights from a population-based Polish cohort

Jerzy Jaroszewicz[1], Karol Żmudka[1], Marlena Dubatówka[2], Małgorzata Chlabicz[2], Paweł Sowa[2], Urszula Roszkowska[2], Damian Piotrowski[1], Karol Kamiński[2]

1 Department of Infectious Diseases and Hepatology, Medical University of Silesia in Katowice, Katowice, Poland

2 Department of Population Medicine and Lifestyle Diseases Prevention, Medical University of Bialystok, Białystok, Poland

Correspondence to: Jerzy Jaroszewicz, MD, PhD, Department of Infectious Diseases and Hepatology, Medical University of Silesia in Katowice, ul. Ziołowa 45/47, 40-635 Katowice, Poland, phone: +48 32 359 84 52, email: jjaroszewicz@sum.edu.pl

Key words: cardiometabolic, liver diseases, metabolic dysfunction–associated steatotic liver disease, metabolic dysfunction–associated alcohol-related liver disease, Poland

Abstract

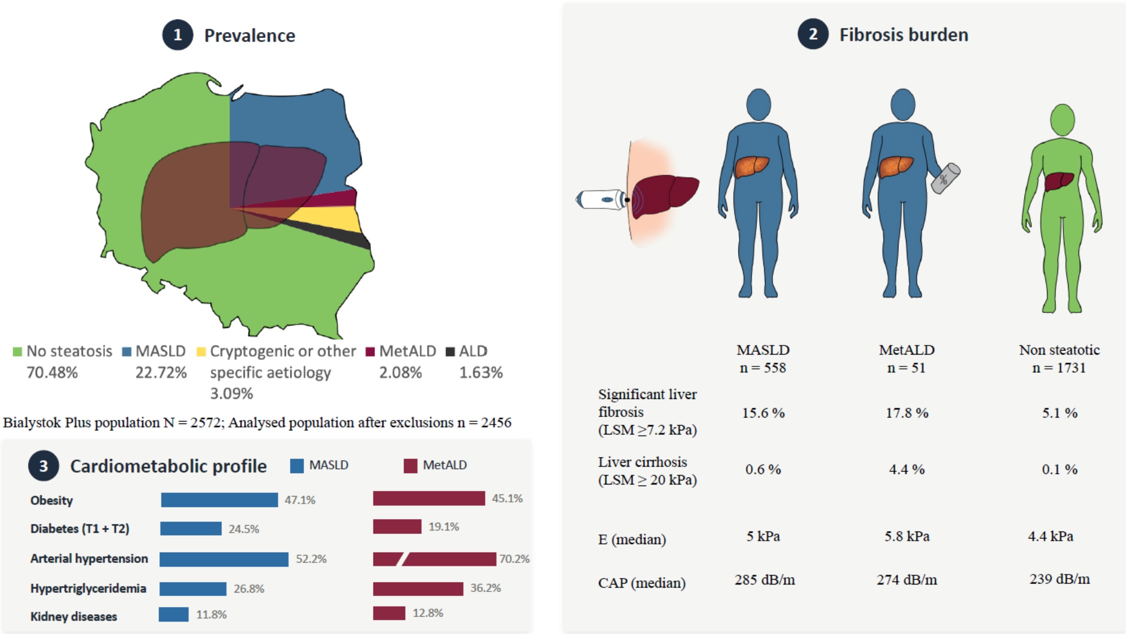
Introduction: Metabolic dysfunction–associated steatotic liver disease (MASLD) and metabolic dysfunction–associated alcohol-related liver disease (MetALD) are increasingly recognized conditions worldwide. However, epidemiological data from Central and Eastern Europe are lacking.

Objectives: This study aimed to assess the prevalence, clinical profiles, and liver fibrosis burden of MASLD and MetALD in a representative population from Central and Eastern Europe.

Patients and methods: This study is part of the Bialystok PLUS (Polish Longitudinal University Study), an ongoing, prospective, population-based cohort study. A random sample of 2456 adult residents from the city of Białystok, Poland, was analyzed. Anthropometric, epidemiological, clinical, and lifestyle-related parameters were assessed. Participants were categorized into MASLD, MetALD, and control groups. Differences in metabolic comorbidities and liver fibrosis severity were evaluated between groups.

Results: The prevalence of MASLD was 22.72%, while MetALD accounted for 2.08% of the population. MASLD was predominantly associated with obesity and diabetes mellitus, whereas MetALD showed a higher prevalence of arterial hypertension and dyslipidemia. Significant liver fibrosis was more frequent in MetALD (17.8%) and MASLD (15.6%) compared to controls (5.1%; $p < 0.001$ and $p = 0.005$). The highest cirrhosis rates were observed in 4.44% of MetALD participants, 0.58% of MASLD participants, and 0.12% of controls ($p = 0.001$). Risk factors for significant fibrosis were analyzed across subgroups.

Conclusions: MASLD and MetALD represent a substantial and distinct health burden not only in the Polish population but likely across Central and Eastern Europe, given shared epidemiological patterns of metabolic dysfunction and alcohol consumption. Understanding the interplay between these factors is crucial for improving diagnosis, treatment strategies, and public health policies in this region.



MASLD and MetALD are prevalent in this Central European population and are characterized by distinct cardiometabolic risk profiles and differential burdens of liver fibrosis.

Introduction

Metabolic dysfunction–associated steatotic liver disease (MASLD) is a newly adopted term that replaces nonalcoholic fatty liver disease (NAFLD), as defined by the 2023 multi-society Delphi consensus and formally endorsed by the European Association for the Study of the Liver (EASL) in 2024 [1]. MASLD is diagnosed when hepatic steatosis coexists with at least one cardiometabolic risk factor (CMRF). Within this framework, metabolic dysfunction–associated alcohol-related liver disease (MetALD) designates individuals who meet MASLD metabolic criteria and consume moderate alcohol (>20 g and <50 g for women and >30 g <60 g for men a day) [2,3], thereby distinguishing them from MASLD (lower intake) and classical ALD. Although MASLD and MetALD partially overlap, emerging evidence indicates that they represent biologically and clinically distinct entities [4]. Importantly, recent evidence suggests that MetALD is associated with increased risks of cardiovascular, cancer, and all-cause mortality when compared to MASLD [5,6].

High-quality epidemiological data on MASLD and MetALD from Central and Eastern Europe remain scarce, despite the region's high combined burden of overweight and obesity (~60% of adults) and excessive alcohol consumption (>10 L pure ethanol per capita annually) [7]. This knowledge gap impedes effective disease surveillance and public health planning. Most prior studies are based in clinical or registry settings; few have compared MASLD versus MetALD in population-based cohorts, and even fewer have incorporated validated measures of liver fibrosis. The recent National Health and Nutrition Examination Survey (NHANES) analysis in the United States (US) was one of the first population-representative studies reporting general (~2.6%) and adolescents (~0.6%) MetALD prevalence [8,9]. Similar nationally representative data have since emerged, for example, from Korea [10], but none originate from Central Europe or include simultaneous assessment of liver stiffness and attenuation parameters.

Therefore, this study aims to estimate the age-specific and age-standardized prevalence of MASLD and MetALD, and to compare their demographic, lifestyle, metabolic, and clinical characteristics, including liver fibrosis, in a population-based cohort from Central Europe. A key strength is its use of the Bialystok PLUS cohort, an ongoing 15-year longitudinal study with extensive multi-system phenotyping [11]; the present analysis represents its baseline, cross-sectional hepatic evaluation, laying groundwork for future longitudinal insights.

Patients and methods

Study population

This study is part of the Bialystok PLUS (Polish Longitudinal University Study), an ongoing, prospective, population-based cohort study designed to investigate the determinants of chronic non-communicable diseases in a representative sample of Białystok residents. The current analysis is based on a baseline cross-sectional assessment of the cohort. Adult participants aged 20 or more years were randomly selected from the official registry of the Mayor's Office of Białystok between January 2017 and April 2024. All participants provided informed consent prior to enrolment. Participants underwent a comprehensive health assessment, including anthropometric measurements, functional tests, laboratory evaluations, and imaging studies (including vibration-controlled transient elastography [VCTE] and ultrasonography). Additionally, biological samples were collected for the analysis of genetic, proteomic, metabolomic, and gut microbiota profiles. Standardized questionnaires were administered to collect detailed information on medical history, medication use, cardiovascular risk factors, and lifestyle habits, in accordance with the European Society of Cardiology guidelines. For the purposes of this study, selected data were used in the analysis. The cohort will be followed prospectively for at least 15 years to monitor the incidence and progression of chronic diseases. Detailed information on the study design and methodology has been published previously [12].

Study participants

Of the initial 2572 participants enrolled in the study, a total of 116 (4.51%) were excluded from further analysis. Specifically, 63 participants (2.45%) were excluded due to missing data on hepatic steatosis assessed by ultrasonography, and 45 (1.75%) were excluded due to a history of, or positive diagnostic markers for, hepatitis B (HBV) or C virus (HCV) infection. Additionally, among those with SLD, 8 (0.31%) participants were excluded due to incomplete data on alcohol consumption. A detailed flowchart illustrating the studied population is presented in Figure 1.

Diagnostic criteria of liver disease

Participants included in the study were initially screened for hepatic steatosis using ultrasound imaging. Those diagnosed with SLD were further classified according to the current EASL and American Association for the Study of Liver Diseases (AASLD) consensus definitions for MASLD[1]. For clarity, respective cardiometabolic risk factors (CMRFs) were labeled as follows: CMRF 1 criteria (body mass index [BMI] + waist circumference); CMRF 2 criteria (diabetes mellitus [DM]); CMRF 3 criteria (blood pressure [BP]); CMRF 4 criteria (triglycerides [TG]); CMRF 5 criteria (high-density lipoprotein [HDL]).

Participants with SLD were assigned to one of the following groups [1,2]: 1) MASLD, defined as the presence of at least one of the mentioned above CMRFs specified in the EASL/AASLD MASLD consensus and weekly alcohol consumption below 140 g for women and 210 g for men; 2) MetALD, defined as the presence of at least one CMRF and alcohol consumption between 140 g and 350 g per week for women, or between 210 g and 420 g per week for men; 3) ALD, defined as alcohol consumption exceeding 350 g per week for women or 420 g per week for men, regardless of the presence of CMRFs; and 4) other SLD, specific etiology SLD and cryptogenic SLD: participants with identifiable causes of liver disease, such as viral hepatitis, were classified into this group, irrespective of alcohol intake or metabolic risk factors. Those with steatosis and no identifiable etiological factor were categorized as cryptogenic SLD. For the purposes of this study, only participants classified as MASLD, MetALD, and a control group without hepatic steatosis were included in further analyses. Participants with ALD or other specific or cryptogenic etiologies of SLD were not analyzed due to the different pathogenesis of their liver disease.

Data acquisition

Ethical approval for this study was provided by the Ethics Committee of the Medical University of Bialystok on March 31, 2016 (R- -I-002/108/2016). All participants provided their written informed consent. The study protocol conforms to the ethical guidelines of the 1975 Declaration of Helsinki.

The data were collected by qualified medical personnel during the participant's one-day visit to the research center. Health assessments and demographic data were obtained from medical interviews and standardized questionnaires, including the Alcohol Use Disorders Identification Test and the Fagerström Test for Nicotine Dependence. Information on prior viral liver infections and other diseases was based on participant statements, confirmed by medical records where available.

Peripheral intravenous blood samples were collected in the examination center in the morning after at least 8 h of fasting. Individuals without diagnosed DM underwent an oral glucose tolerance test (OGTT). The following parameters were assessed: fasting glucose level, glucose level 120 minutes after OGTT, glycated hemoglobin, fasting C-peptide level, lipid profile. Tests for HBV (hepatitis B surface antigen) and HCV (anti-HCV antibodies) infections were performed using the electrochemiluminescence method. The samples were determined on a Cobas device (Roche Diagnostics). Hypercholesterolemia was defined as either a history of hypercholesterolemia or a cholesterol level of ≥200 mg/dL. DM was defined as a history of DM, a glucose level of ≥200 mg/dL 120 minutes after OGTT, or a glycated hemoglobin level of ≥6.5%. The diagnosis of DM in our study was not made on the basis of fasting glucose, due to the availability of only a single measurement.

In addition, participants underwent anthropometric measurements, such as waist, abdominal, and hip circumference, using SECA 201 tapes. The InBody 770 device (InBody Co., Ltd.) was used to measure body weight, while the SECA 264 height meter was used to measure height. Systolic and diastolic blood pressure were measured twice at 5-minute intervals after 5 minutes of sitting using an Omron M6 AC Comfort device (Omron Healthcare CO., Ltd.). Hypertension was classified as a history of hypertension or systolic BP ≥140 mm Hg or diastolic BP ≥90 mm Hg.

Liver steatosis was assessed through ultrasound examination using the ultrasound Vivid 9 (GE Healthcare) with increased echogenicity of liver parenchyma compared to renal cortex parenchyma as criteria. Moreover, VCTE and controlled attenuation parameter (CAP) were both measured by FibroScan 530 Compact (Echosense) in the supine position with the right limb elevated. The examination was performed on the right lobe of the liver in the intercostal space in the median axillary line at the level of the gladius process after obtaining 10 repeated measurements. The examination was preceded by at least 3 h of fasting. Results were evaluated using various cut-off points for assessing fibrosis stage and steatosis grade, as suggested by the manufacturer for nonalcoholic steatohepatitis/NAFLD etiology[13], but also scientific societies [14]. Respective cut-off values are mentioned in the results section.

Statistical analysis

Continuous variables are presented as mean (SD) or median (interquartile range [IQR]), as appropriate depending on data distribution. The normality of distribution for continuous variables was assessed visually using histograms and quantile-quantile (Q-Q) plots. Categorical variables are presented as absolute numbers with corresponding percentages, calculated among participants with available data for each respective variable, unless otherwise specified.

Comparisons of continuous variables across three groups (MASLD, MetALD, and no steatosis) were performed using Welch ANOVA or the Kruskal–Wallis test depending on the data distribution. For variables which differed significantly between three groups post-hoc analysis was conducted. For post-hoc pairwise comparisons, the Games–Howell test was applied following Welch ANOVA, and Dunn test with Holm correction ($k = 3$) was applied following the Kruskal–Wallis test.

For categorical variables, Pearson chi-squared test was used when all expected cell frequencies were ≥5, and Fisher exact test was applied otherwise. For polytomous categorical variables (fibrosis stage F0–F4, steatosis grade S0–S3, BMI categories, and educational attainment), an omnibus Fisher exact test with Monte Carlo simulation ($B = 100\,000$) was performed on the full $r \times c$ contingency table. Post hoc pairwise comparison of the categorical variables was conducted using the same approach with Holm correction applied across the three pairwise comparisons ($k = 3$).

Univariable logistic regression models were used to estimate crude odds ratios (ORs) with two sets of analyses performed. First being the association between selected comorbidities and the presence of MASLD or MetALD, each compared against the no steatosis group, and second being the association between CMRFs and the presence of significant liver fibrosis. P-values were obtained from the logistic regression model. Variables for which an OR could not be estimated were not presented.

Crude prevalence estimates were calculated using the final study population as the denominator, which consisted of 2456 adult participants after applying exclusion criteria. Age-specific and age-standardized prevalence rates were calculated across 5-year age intervals and presented separately by sex. Age-standardized prevalence rates were calculated using the World Health Organization (WHO) world standard population (2001 revision) [15]. All prevalence estimates are expressed per 1000 participants. Crude prevalence estimates are reported with 95% CIs calculated using the Wilson method. Age-standardized prevalence rates are reported with 95% CIs derived using the Dobson method. ORs are reported with 95% CIs calculated using the Wald method. All values of *P* below 0.05 were considered significant. All statistical analyses were performed using R statistical software (version 4.4.1).

Results

Classification of participants

The final study population consisted of 2456 participants after applying exclusion criteria. Participants ranged in age from 20 to 82 years, with a median age of 48 years (IQR: 37–63). A total of 1103 individuals (44.91%) were male. Among the 2456 participants, ultrasound assessments revealed hepatic steatosis in 725 participants (29.52%), while 1731 individuals (70.48%) showed no evidence of steatosis, Figure 1.

Among those with SLD, 40 participants (1.63% of the total cohort) met criteria for ALD based on reported alcohol consumption. The remaining 609 individuals (24.8%) with SLD did not meet ALD criteria and were further subdivided as follows: 558 participants (22.72%) were classified as having MASLD, and 51 participants (2.08%) fulfilled the criteria for the MetALD diagnosis. An additional 76 participants (3.09%) with SLD were classified as having cryptogenic or other specific etiologies. Within this group, 19 individuals (0.77%) had a history of HBV infection, and 3 individuals (0.12%) were suspected of HCV infection. For 54 participants (2.2%), no identifiable etiological factor was determined.

For the purposes of further analyses, only MASLD, MetALD, and the control group without hepatic steatosis were included. Participants with ALD or cryptogenic/specific etiologies of SLD were excluded from further evaluation.

Crude and age-standardized prevalence

The crude prevalence of MASLD was estimated at 227.2 per 1000 inhabitants (95% CI: 211.06–244.19). The age-standardized prevalence of MASLD, adjusted to the WHO world standard population, was 127.64 per 1000 inhabitants (95% CI: 116.13–139.9). For MetALD, the crude prevalence was 20.77 per 1000 inhabitants (95% CI: 15.83–27.2), while the age-standardized prevalence was 12.62 per 1000 inhabitants (95% CI: 9.08–16.99). Importantly, for male participants, disease burden for both MASLD and MetALD was high already in individuals aged 30 years and older.

Detailed age-specific and age-standardized prevalence rates of MASLD and MetALD stratified by sex and across 5-year age intervals are presented in Figure 2A–2D.

Demographic, anthropometric, and lifestyle characteristics

General demographic, anthropometric, educational, employment, and lifestyle characteristics of the study population are summarized in Table 1. Results of post-hoc pairwise comparisons are provided in Supplementary material, *Table S1A–S1C*.

Participants with MASLD were significantly older than those in the control group ($p < 0.001$) and more frequently male ($p = 0.02$). The proportion of males was significantly higher in the MetALD group than in the control group ($p < 0.001$); however, no significant age difference was observed between the groups ($p = 0.4$). Additionally, the MetALD group had a higher proportion of males compared to the MASLD group ($p = 0.009$).

Regarding anthropometric parameters, both MASLD and MetALD participants exhibited poorer body composition and unfavorable fat distribution compared to controls ($p < 0.001$ for both). However, no significant differences in anthropometric measures were found between the MASLD and MetALD groups in post-hoc pairwise analyses (all $p > 0.05$).

Education level distribution did not differ significantly between the groups ($p = 0.08$). Retirement was less frequently reported among control group participants compared to those with MASLD ($p < 0.001$). Alcohol consumption was significantly higher among MetALD participants compared to both MASLD and control groups (both $p < 0.001$). However, there was no significant difference in alcohol consumption between the MASLD group and the control group ($p = 0.35$). Patterns of alcohol preference differed across groups: beer was more commonly consumed in the MetALD group compared both to MASLD and no steatosis (both $p < 0.001$), whereas wine consumption was more frequently reported among control participants than in the MASLD group ($p = 0.02$). A history of smoking was more prevalent among MetALD participants compared to both MASLD ($p = 0.004$) and control groups ($p < 0.001$). No significant differences between groups were observed in current coffee consumption ($p = 0.48$). Lastly, recent weight gain was reported more frequently in the MASLD group compared to controls ($p < 0.001$). However, no significant differences in recent weight gain were observed between the MetALD and control groups ($p = 0.6$), nor between the MASLD and MetALD groups ($p > 0.99$).

Comorbidities and cardiometabolic risk factors

Detailed data on the prevalence of comorbidities and CMRFs across MASLD, MetALD, and control groups are presented in Table 2.

Among the comorbidities analyzed, no significant differences in prevalence were observed between the three groups for neoplasm, myocardial infarction, heart failure, ischemic heart disease, and kidney disease (all $p > 0.05$). In contrast, the prevalence of other cardiometabolic conditions, including arterial hypertension, type 1 and type 2 DM and dyslipidemia differed significantly across groups with BMI classes' distribution differing significantly between the groups ($p < 0.001$). Notably, MASLD was predominantly associated with high prevalence of obesity and DM, whereas MetALD exhibited a high prevalence of arterial hypertension, obesity and hypertriglyceridemia, highlighting distinct cardiometabolic profiles. Univariable ORs with 95% CIs for the presence of MASLD and MetALD in relation to the assessed comorbidities are depicted in Figure 3A and 3B, respectively.

Laboratory findings

Laboratory parameters across the MASLD, MetALD, and control groups are summarized in Table 3. Significant differences were observed in most laboratory parameters across the three study groups, except for platelet count ($p = 0.93$), total cholesterol ($p = 0.3$), and total bilirubin ($p = 0.23$). In post-hoc pairwise comparisons between the MASLD and MetALD groups, only liver enzymes: aspartate aminotransferase (AST) ($p = 0.008$), alanine transaminase ($p = 0.04$), and gamma-glutamyl transferase ($p < 0.001$) showed significant differences, with higher levels observed in the MetALD group.

Liver fibrosis burden and disease progression

The extent of liver fibrosis and disease progression among participants is summarized in Table 4. Fibrosis severity and hepatocellular injury were assessed using vibration-controlled transient elastography (VCTE), alongside composite clinical indices such as Fibrosis-4 (FIB-4), aspartate aminotransferase to platelet ratio, and FibroScan-AST (FAST) score. Hepatic steatosis was assessed by CAP. In the total cohort of 2456 participants, median liver stiffness was 4.6 kPa (IQR: 3.8–5.6). Significant hepatic fibrosis, defined as liver stiffness measurement

(LSM) ≥F2, was identified in 7.4% of the total population, while liver cirrhosis was present in 0.3% of participants.

Comparing across groups, significant hepatic fibrosis (LSM ≥F2) was observed in 15.6% of MASLD participants ($p < 0.001$) and 17.8% of MetALD participants ($p = 0.005$), compared to 5.1% in the control group. However, the proportion of participants with significant hepatic fibrosis did not differ significantly between the MASLD and MetALD groups ($p = 0.87$). Regarding cirrhosis, assessed by VCTE, the prevalence was significantly higher in the MetALD group (4.4%) compared to control participants (0.1%) ($p = 0.01$). No significant difference in cirrhosis prevalence was observed between the MASLD and control groups ($p = 0.11$).

In the MASLD cohort, 27 participants (4.8%) demonstrated LSM values suggestive or highly suggestive of compensated advanced chronic liver disease (LSM ≥10 kPa), compared to 5 (9.8%) in the MetALD group ($p = 0.18$). Applying the Baveno VII consensus criteria [16], which recommend screening endoscopy in patients with compensated cirrhosis and LSM ≥20kPa or platelet count $\leq 150 \times 10^9$/L, 6 out of 27 MASLD participants (22.2%) and 2 out of 5 MetALD participants (40%) met the threshold for variceal screening endoscopy, with no significant difference observed between the two groups ($p = 0.58$).

Furthermore, we assessed which CMRFs were associated with significant hepatic fibrosis within each group (Figure 4). In the MASLD cohort, the presence of CMRF 1 (BMI + WC), 2 (DM), 3 (BP) and 4 (TG) were significantly associated with higher odds of advanced fibrosis. In contrast, among participants without hepatic steatosis, none of the assessed CMRFs showed a significant association with hepatic fibrosis. Importantly, none of the CMRF showed significant associations with hepatic fibrosis in the MetALD group.

Discussion

In this large, population-based study from Central Europe, we report the prevalence, clinical characteristics, and fibrosis risk factors for MASLD and MetALD, leveraging comprehensive phenotyping from the Bialystok PLUS cohort. To our knowledge, this is the first study from Central and Eastern Europe to provide robust epidemiologic data on both entities using standardized definitions aligned with recent EASL and AASLD consensus statements. The prevalence of MASLD in our cohort (22.7% crude; 12.8% age-standardized) is comparable to estimates from Western populations, including the US (NHANES ~25%) and Europe (~23–29%) [8,17,18], but higher than in East Asian cohorts, where reported rates range from 12–20% depending on diagnostic modality and criteria [10]. In contrast, MetALD prevalence (2.1% crude; 1.3% age-standardized) is consistent with reports from the US (~2%) and Korea (~4%), suggesting that moderate alcohol consumption in the presence of metabolic dysfunction represents a relatively small but clinically important phenotype [8,10]. However, the true prevalence may be underestimated due to self-reported alcohol intake, which is prone to under-reporting bias; future studies incorporating objective biomarkers such as phosphatidylethanol (PEth) could help refine these estimates [19]. Beyond under-reporting, it should be noted that alcohol consumption below MetALD diagnostic criteria may exert hepatotoxic effects rendering the boundary between MASLD and MetALD inherently imprecise [10].

Our analysis reveals distinct cardiometabolic profiles between MASLD and MetALD. MASLD was more strongly associated with obesity and type 1 and type 2 DM, aligning with the traditional metabolic pathway to steatosis. MetALD, by contrast, showed a higher prevalence of hypertension and dyslipidemia, a pattern observed in other cohorts and potentially linked to alcohol-induced alterations in lipid metabolism and vascular tone. Importantly, alcohol may potentiate the deleterious effects of metabolic dysfunction on hepatic inflammation and fibrosis via synergistic mechanisms, including oxidative stress, modulation of the gut microbiota, and upregulation of pro-inflammatory cytokines [21].

In our cohort, several demographic and socioeconomic characteristics differed between MASLD, MetALD, and control groups. MASLD participants were older and more often male compared to controls, whereas MetALD cases were predominantly male but did not differ significantly in age from controls. Male sex and older age are regarded as traditional risk factors for MASLD [22]. On the other hand, and notably, our data shows that in men, the prevalence of both MASLD and MetALD began to rise markedly from around the age of 30, suggesting that childhood and early adulthood may represent a critical window for prevention of alcohol use and lifestyle intervention [23,24]. Socioeconomic and lifestyle factors, including occupational status, diet, and physical activity, likely contribute to the observed variation in disease phenotype and progression risk between groups. Particularly, in Central and Eastern Europe, the overlay of obesity and alcohol consumption is worrisome [25], highlighting the need to consider demographic and socioeconomic context when designing targeted prevention and screening programs.

Significant fibrosis (≥F2 by VCTE) was present in ~16% of MASLD and ~18% of MetALD cases, compared to only ~5% in controls. Although the proportion of significant fibrosis did not differ significantly between MASLD and MetALD, both groups demonstrated markedly higher rates compared to controls. Notably, MetALD participants showed a higher proportion meeting the rule-in cut-off for advanced fibrosis (LSM ≥12 kPa) at 11.1%, compared to 2.9% in the MASLD group. This was further reflected in the prevalence of cirrhosis, which accounted for 4.4% of MetALD and 0.6% of MASLD cases.

This finding reinforces previous evidence that even moderate alcohol consumption can accelerate fibrosis progression in the setting of metabolic dysfunction [20,26]. Clinically, these data support routine fibrosis assessment in all participants with MetALD, irrespective of aminotransferase levels. Special attention should be paid to individuals with obesity and metabolic risk factors who report even moderate alcohol use, as they may be at particularly high risk of MASLD progression and cancer mortality, with men generally showing the highest risk [27].

The debate on clinical relevance of steatosis quantification apart from diagnostic importance is still ongoing. Several ultrasonography based algorithms are available, with CAP being applied most often [28]. In the current cohort lowest CAP threshold (<248 dB/m) showed moderate discernment between SLD and non-SLD individuals, although still more than 40% of non-SLD subjects presented higher values. In this context a noninvasive surrogate of metabolic dysfunction-associated steatohepatitis, FAST-score, especially using rule-out criterion showed better accuracy in differentiation between SLD and non-SLD, with less than 2% positive values in non-SLD subjects.

Our results also inform potential screening strategies. Given the relatively high prevalence of MASLD and the significant fibrosis burden in both MASLD and MetALD, targeted screening in primary care using non-invasive scores (eg, FIB-4) followed by elastography in at-risk participants could be a cost-effective approach [1,2,29]. In MetALD, targeted questioning about alcohol consumption in individuals with metabolic risk factors, particularly men, is essential, and any positive cases should undergo fibrosis staging. These strategies may be particularly relevant in Central and Eastern Europe, where the combined burden of overweight/obesity (~60% of adults) and high per capita alcohol consumption creates a fertile ground for both MASLD and MetALD.

Strengths and limitations

This study has notable strengths, including its population-based design, extensive phenotyping, and strict application of current MASLD/MetALD criteria. The Bialystok PLUS cohort also offers the unique advantage of an ongoing 15-year follow-up, enabling future longitudinal analyses of fibrosis progression and outcomes. However, several limitations should be acknowledged. First, the study population is urban, and rural prevalence patterns may differ due to lifestyle and occupational factors. Second, data are from a single city, which may limit generalizability, though Białystok's demographic and socioeconomic profile is representative of much of Eastern Poland and similar Central / Eastern European regions. Third, alcohol intake was self-reported and may be subject to under-reporting bias; objective biomarkers such as PEth were not available in this baseline analysis [19,30].

Conclusions

MASLD and MetALD are prevalent in this Central European population and present distinct cardiometabolic profiles and fibrosis risks. MASLD case finding should prioritize individuals with central obesity and DM, whereas MetALD should be particularly suspected in middle-aged or older men with hypertension, obesity, dyslipidemia, and regular alcohol consumption above low-risk thresholds but below ALD criteria.

All individuals diagnosed with MetALD warrant routine fibrosis assessment due to the high cirrhosis prevalence. Implementing targeted, primary care–based screening strategies combining simple non-invasive scores with elastography could facilitate early detection and reduce the growing burden of liver cirrhosis and hepatocellular carcinoma in Central and Eastern Europe.

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Table 1 Demographic characteristics of the study population

Characteristic		**MASLD**	**MetALD**	**No steatosis**	***P* value**
Men		272 (48.7)	36 (70.6)	744 (43)	<0.001
Age, y, median (IQR)		55 (41–64)	49 (37.5–61.5)	47 (35–62)	<0.001
Anthropometric measures	BMI, kg/m^2, median (IQR)	29.6 (27.11–33.33)	29.48 (26.85–32.9)	25.45 (22.85–28.66)	<0.001
	Waist circumference, cm	96.94 (12.57)	98.02 (13.57)	85.51 (12.6)	<0.001
	Abdominal circumference, cm	103.87 (12.12)	103.85 (13.75)	92.88 (11.92)	<0.001
	Hip circumference, cm, median (IQR)	105.5 (100.38–111.5)	105 (98.25–112)	100 (95–105)	<0.001
Education and employment	Primary education	14 (2.5)	0	39 (2.3)	0.08
	Secondary education	277 (49.7)	18 (36)	769 (44.5)	
	Tertiary education	266 (47.8)	32 (64)	920 (53.2)	
	Employed or self-employed	358 (64.3)	32 (62.7)	1199 (69.4)	0.06

	Retiree or pensioner	180 (32.3)	16 (31.4)	410 (23.7)	<0.001
Lifestyle data	Alcohol consumption in the past 30 d	372 (68)	51 (100)	1188 (70.3)	<0.001
	Beer consumption in the past 30 d	185 (47.9)	44 (86.3)	706 (57.4)	<0.001
	High-proof spirits consumption in the past 30 d	167 (43.3)	31 (60.8)	476 (38.7)	0.003
	Type of alcohol consumed in the past 30 d (liqueurs, fruit infusions, cocktails)	59 (15.2)	10 (19.6)	266 (21.7)	0.02
	Type of alcohol consumed in the past 30 d (wine)	140 (36.3)	15 (29.4)	543 (44.2)	0.004
	Current smoker	95 (29.9)	13 (31)	312 (33.6)	0.46
	Former smoker	321 (59)	42 (82.4)	940 (55.2)	<0.001
	Regular coffee consumption	432 (82.9)	37 (77.1)	1323 (81.1)	0.48
	Weight gain in the past year	232 (42.7)	20 (41.7)	565 (33.4)	<0.001

Data are presented as number (percentage) or mean (SD) unless indicated otherwise.

Abbreviations: BMI, body mass index; IQR, interquartile range; MASLD, metabolic dysfunction–associated steatotic liver disease; MetALD, metabolic dysfunction–associated alcohol-related liver disease

Table 2 Individuals with comorbidities and respective cardiometabolic risk factors in the study population[a]

Variable			MASLD	MetALD	No steatosis	*P* value
Comorbidities	DM T1 + T2		125 (24.5)	9 (19.1)	128 (8.1)	<0.001
	Arterial hypertension		266 (52.2)	33 (70.2)	516 (32.7)	<0.001
	Hypercholesterolemia		356 (69.8)	37 (78.7)	979 (62)	<0.001
	Hypertriglyceridemia		135 (26.8)	17 (36.2)	229 (14.6)	<0.001
	Neoplasm		35 (6.9)	4 (8.5)	94 (6)	0.51
	Myocardial infarction		14 (2.7)	2 (4.3)	30 (1.9)	0.22
	Cardiac failure		4 (0.8)	0	30 (1.9)	0.2
	Ischemic heart disease		7 (1.4)	1 (2.1)	33 (2.1)	0.5
	Kidney disease		60 (11.8)	6 (12.8)	143 (9.1)	0.14
BMI, kg/m^2	Underweight (<18.5)		0	0	43 (2.5)	<0.001
	Normal weight (18.5–24.9)		66 (11.8)	11 (21.6)	725 (41.9)	
	Overweight (25–29.9)		229 (41)	17 (33.3)	655 (37.9)	
	Obesity	class I (30–34.9)	174 (31.2)	17 (33.3)	247 (14.3)	
		class II (35–39.9)	62 (11.1)	5 (9.8)	48 (2.8)	
		class III (≥40)	27 (4.8)	1 (2)	12 (0.7)	
CMRF	CMRF 1 (BMI + WC)		504 (90.3)	42 (82.4)	1017 (58.8)	<0.001
	CMRF 2 (DM)		444 (79.9)	45 (88.2)	939 (55.4)	<0.001
	CMRF 3 (BP)		381 (68.3)	39 (76.5)	840 (48.5)	<0.001
	CMRF 4 (TG)		273 (49)	27 (52.9)	452 (26.2)	<0.001
	CMRF 5 (HDL)		216 (38.8)	20 (39.2)	350 (20.3)	<0.001

Data are presented as number (percentage).

a Each CMRF is labeled according to its main associated factor.

Abbreviations: BP, blood pressure; CMRF, cardiometabolic risk factor; DM, diabetes mellitus; HDL, high-density lipoprotein; TG, triglycerides; WC, waist circumference

Table 3 Laboratory results, vibration-controlled transient elastography measurements, and values of noninvasive indexes of the study population

Variable	MASLD	MetALD	No steatosis	*P* value
WBC count, 10^9/l	5.9 (5.05–7.1)	5.6 (5–6.7)	5.6 (4.9–6.6)	<0.001
RBC count, 10^9/l	4.8 (0.41)	4.84 (0.42)	4.72 (0.42)	<0.001
Hemoglobin, g/dl	13.9 (13.2–14.8)	14.5 (13.7–15.55)	13.8 (12.9–14.8)	<0.001
PLT count, 10^9/l	239 (203.5–278)	244 (204–269)	238 (202–280)	0.93
Fasting glucose, mg/dl	104 (98–115)	105 (98.5–113.5)	98 (92–105)	<0.001
HOMA-IR	3.88 (2.6–5.9)	3.95 (2.54–5.49)	2.26 (1.5–3.23)	<0.001
Total cholesterol, mg/dl	192 (164–222)	198 (163.5–253)	191 (166–219)	0.3
LDL cholesterol, mg/dl	123.6 (96.5–151.1)	129.6 (91.3–156.6)	118 (94.7–143.15)	0.03
HDL cholesterol, mg/dl	54.16 (45–64.6)	55.3 (44.39–74.62)	63.17 (52.95–74.84)	<0.001
Triglycerides, mg/dl	121 (89–173)	130 (90–187.5)	90 (66.9–122.5)	<0.001
AST, U/l	24 (20.3–29.6)	27.7 (22.05–36.85)	21.8 (18.6–25.8)	<0.001
ALT, U/l	27.1 (19.1–37.5)	31.8 (21.1–52.15)	19.3 (14.6–25.8)	<0.001
GGT, U/l	23 (14.6–35.8)	44 (23.95–93.9)	15.3 (11.2–23.7)	<0.001
Total bilirubin, mg/dl	0.51 (0.38–0.7)	0.58 (0.42–0.85)	0.52 (0.39–0.73)	0.23
hsCRP, mg/l	1.46 (0.7–2.85)	1.22 (0.68–2.34)	0.93 (0.42–1.81)	<0.001
Uric acid, mg/dl	5.63 (1.34)	5.92 (1.51)	4.95 (1.21)	<0.001
eGFR, ml/min/1.73 m^2	98.65 (87.44–107.57)	100.73 (85.22–110.29)	100.89 (89.78–111.83)	0.001
Vitamin D, ng/ml	25.24 (19.34–32.22)	25.29 (19–34.38)	26.5 (20.38–35.03)	0.004
Elasticity median, kPa	5 (4.1–6.2)	5.8 (4.8–6.7)	4.4 (3.7–5.4)	<0.001

CAP median, dB/m	285 (246.25–320.75)	274 (227–310)	239 (213–268)	<0.001
FIB-4	1.03 (0.79–1.47)	1.07 (0.81–1.39)	0.94 (0.68–1.35)	<0.001
HSI	40.91 (6.49)	40.14 (6.92)	34.76 (5.7)	<0.001
FLI	61.48 (34.86–84.59)	77.68 (48.18–92.13)	19.63 (7.22–47.34)	<0.001
FAST	0.13 (0.07–0.25)	0.19 (0.08–0.34)	0.07 (0.04–0.12)	<0.001

Data are presented as mean (SD) or median (interquartile range).

SI conversion factors: to convert hemoglobin to mmol/l, multiply by 0.6206; fasting glucose to mmol/l, by 0.0555; total cholesterol to mmol/l, by 0.0259; LDL cholesterol to mmol/l, by 0.0259; HDL cholesterol to mmol/L, by 0.0259; triglycerides to mmol/l, by 0.0113; total bilirubin to μmol/l, by 17.1; uric acid to μmol/l, by 59.48; vitamin D to nmol/l, by 2.5.

Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; CAP, controlled attenuation parameter; eGFR, estimated glomerular filtration rate; FAST, FibroScan-AST score; FIB-4, fibrosis-4 index; FLI, fatty liver index; GGT, gamma-glutamyl transferase; HOMA-IR, homeostasis model assessment of insulin resistance; hsCRP, high-sensitivity C-reactive protein; HSI, hepatic steatosis index; LDL, low-density lipoprotein cholesterol; PLT, platelet; RBC, red blood cell; VCTE, vibration-controlled transient elastography; WBC, white blood cell

Table 4 Disease advancement in the study groups based on the Meta-analysis of Histological Data in Viral Hepatitis scoring systema

Biological process reflected	**Noninvasive test result**	**Cutoff valuesb**	**MASLD**	**MetALD**	**No steatosis**	***P* value**
Fibrosis, extracellular volume fraction	VCTE: LSM - F0	LSM <6.5 (kPa)	397 (76.6)	31 (68.9)	1470 (89.5)	<0.001
	VCTE: LSM - F1	6.5 ≤LSM <7.2 (kPa)	40 (7.7)	6 (13.3)	88 (5.4)	
	VCTE: LSM - F2	7.2 ≤LSM <9.6 (kPa)	50 (9.7)	3 (6.7)	67 (4.1)	

	VCTE: LSM - F3	9.6 ≤LSM <14.5 (kPa)	22 (4.2)	1 (2.2)	13 (0.8)	
	VCTE: LSM - F4	LSM ≥14.5 (kPa)	9 (1.7)	4 (8.9)	4 (0.2)	
	VCTE: LSM ≥ F2	LSM ≥7.2 (kPa)	81 (15.6)	8 (17.8)	84 (5.1)	<0.001
	VCTE: LSM - rule-out cut-off	LSM <8 (kPa)	466 (90)	39 (86.7)	1602 (97.6)	<0.001
	VCTE: LSM - rule-in cut-off	LSM ≥12 (kPa)	15 (2.9)	5 (11.1)	6 (0.4)	<0.001
	VCTE: LSM - cirrhosis	LSM ≥20 (kPa)	3 (0.6)	2 (4.4)	2 (0.1)	0.001
Stress to hepatocytes	FIB-4: rule-out cut-off	FIB-4 <1.3	378 (68.1)	32 (62.7)	1237 (71.9)	0.1
	FIB-4: rule-in cut-off	FIB-4 ≥2.67	18 (3.2)	1 (2)	34 (2)	0.22
Stress to hepatocytes, fibrosis, lipid content	FAST score: rule-out cut-off	FAST score <0.35	446 (86.3)	34 (75.6)	1605 (98.2)	<0.001
	FAST score: rule-in cut-off	FAST score ≥0.67	11 (2.1)	5 (11.1)	2 (0.1)	<0.001
Lipid content	VCTE: CAP - S0	CAP <248 (dB/m)	134 (25.9)	14 (31.1)	945 (57.6)	<0.001
	VCTE: CAP - S1	248 ≤CAP <268 (dB/m)	62 (12)	7 (15.6)	274 (16.7)	
	VCTE: CAP - S2	268 ≤CAP <280 (dB/m)	37 (7.1)	5 (11.1)	102 (6.2)	
	VCTE: CAP - S3	CAP ≥280 (dB/m)	285 (55)	19 (42.2)	321 (19.5)	

Data are presented as number (percentage).

a Targets of selected noninvasive techniques, and suggested thresholds for ruling out / in certain features of steatotic liver disease, and the degree of steatosis

b Cutoff values for fibrosis stages and steatosis grades according to manufacturer recommendations

Abbreviations: LSM, liver stiffness measurement

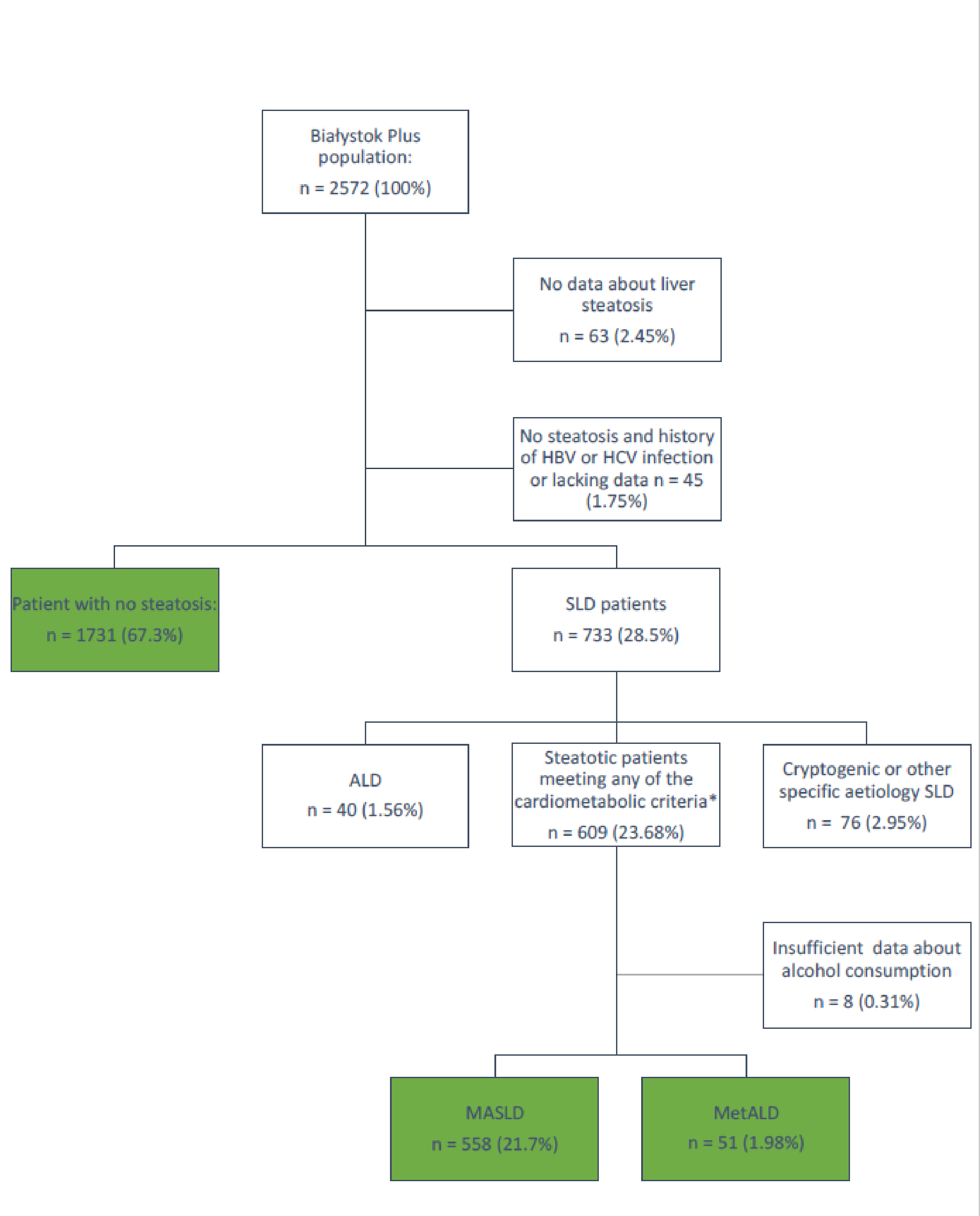

Figure 1 Flowchart of the study selection process

Abbreviations: ALD, alcohol-related liver disease; HBV, hepatitis B virus; HCV, hepatitis C virus; MASLD, metabolic dysfunction-associated steatotic liver disease; MetALD, metabolic dysfunction-associated alcohol-related liver disease; SLD, steatotic liver disease

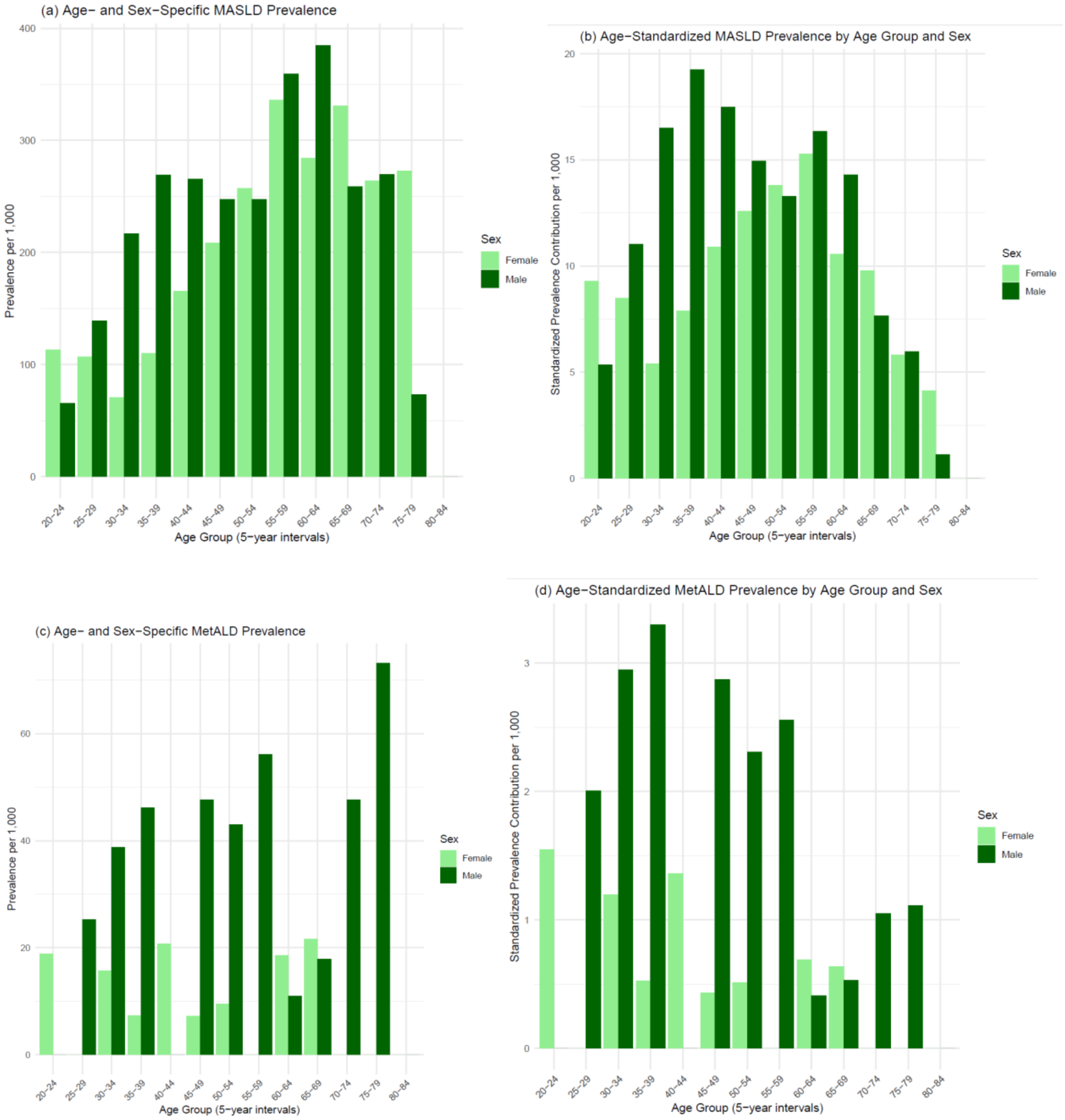

Figure 2 Age-specific and age-standardized prevalence of MASLD (**A**, **B**) and MetALD (**C**, **D**) participants across 5-year age intervals by sex

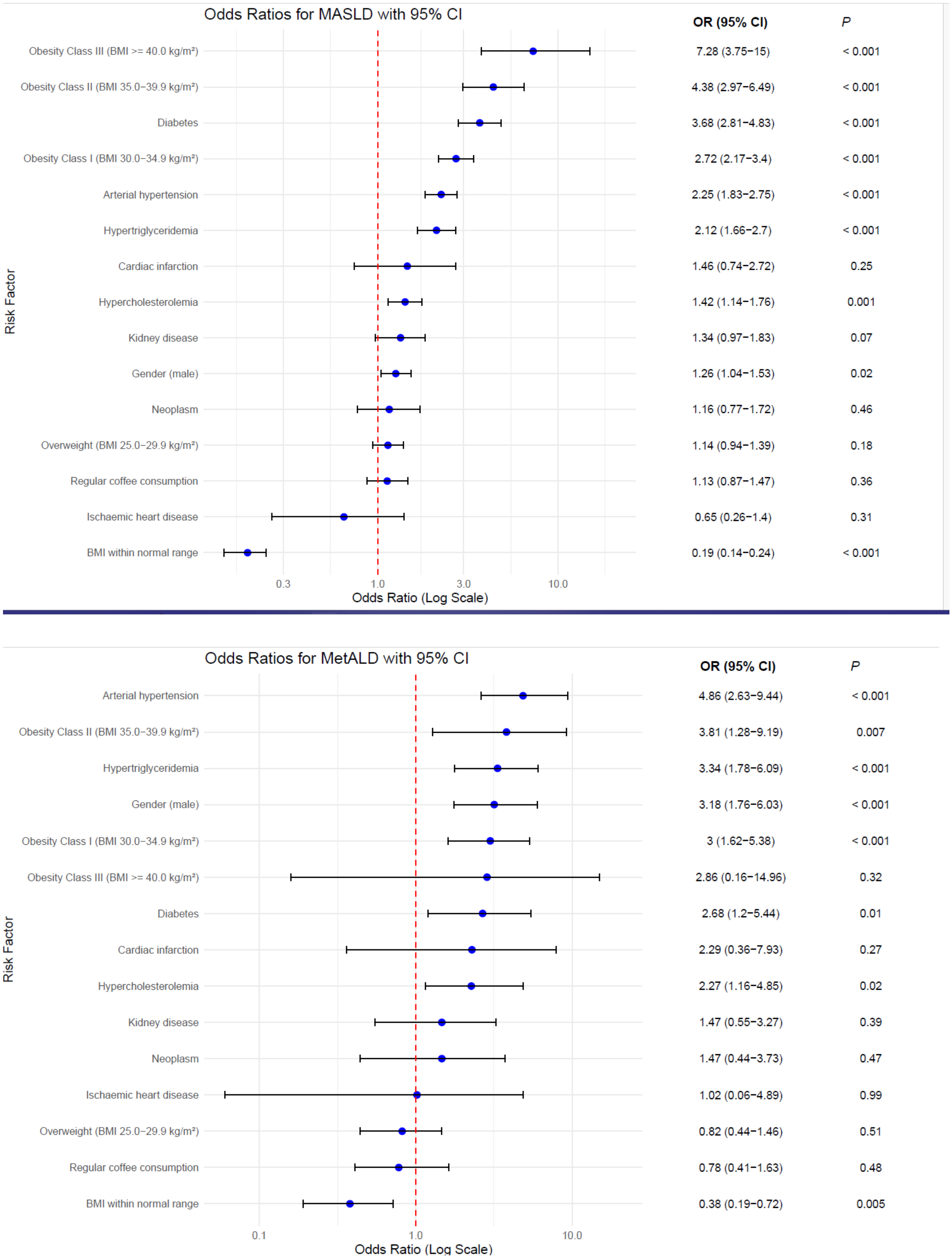

Figure 3 Odds ratios for presence of MASLD (**A**) and MetALD (**B**) among population with selected comorbidities. Crude odds ratios with corresponding 95% CIs and p-values are presented. The x-axis is displayed on a logarithmic scale.

Abbreviations: BMI, body mass index; BP, blood pressure; DM, diabetes mellitus; HDL, high-density lipoprotein; TG, triglycerides; WC, waist circumference

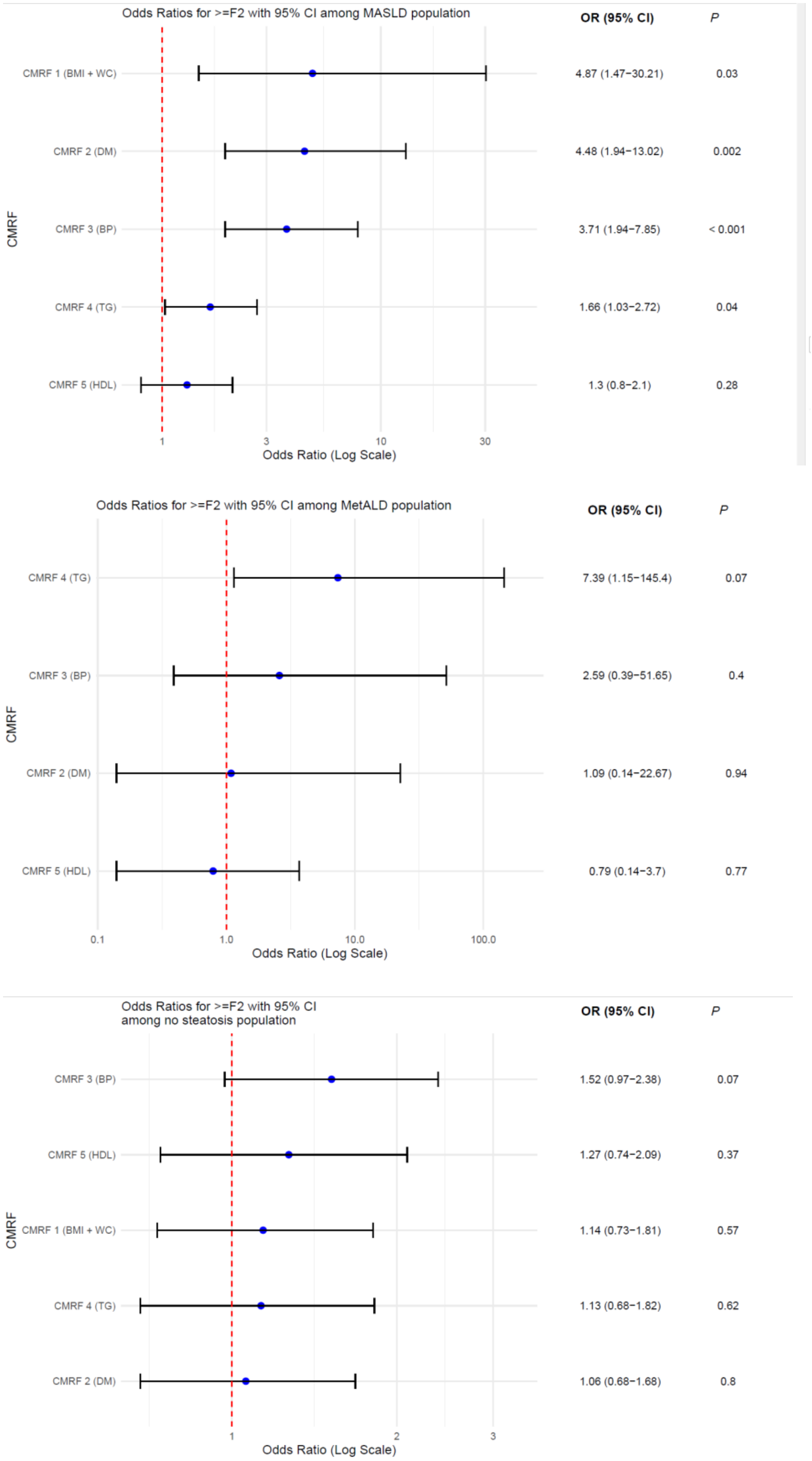

Figure 4 Odds ratios for the presence of significant fibrosis among participants with the presence of CMRFs within the populations of MASLD (**A**), MetALD (**B**), and no liver steatosis

participants (**C**). Crude odds ratios with corresponding 95% CIs and p-values are presented. The x-axis is displayed on a logarithmic scale. For clarity, each CMRF is labeled according to its main associated factor.

Abbreviations: CMRF, cardiometabolic risk factor

Short title: MASLD and MetALD in the Polish population