



## Brief report

## New steatotic liver disease criteria diagnostic performance in an agricultural population in Chile



Maria Spencer-Sandino<sup>a,b</sup>, Franco Godoy<sup>a,b</sup>, Laura Huidobro<sup>b,c</sup>, Danilo Alvares<sup>d</sup>,  
Francisco Cruz<sup>e</sup>, Claudia Marco<sup>a,b</sup>, Macarena Garrido<sup>a,j</sup>, Daniel Cabrera<sup>f,g</sup>, Juan Pablo Arab<sup>h,i</sup>,  
Marco Arrese<sup>i</sup>, Francisco Barrera<sup>i</sup>, Catterina Ferreccio<sup>a,j,\*</sup>

<sup>a</sup> Escuela de Salud Pública, Pontificia Universidad Católica de Chile, Santiago, Chile

<sup>b</sup> Advance Center for Chronic Diseases, ACCDIS, Universidad De Chile and Pontificia Universidad Católica de Chile, Santiago, Chile

<sup>c</sup> Departamento de Ciencias Preclínicas — Facultad de Medicina, Universidad Católica del Maule, Talca, Chile

<sup>d</sup> MRC Biostatistics Unit, University of Cambridge, UK

<sup>e</sup> Departamento de Radiología, Escuela de Medicina, Pontificia Universidad Católica de Chile, Santiago, Chile

<sup>f</sup> Centro de Investigación e Innovación en Biomedicina, Facultad de Medicina, Universidad de los Andes, Santiago, Chile

<sup>g</sup> Facultad de Ciencias Médicas, Universidad Bernardo O'Higgins, Santiago, Chile

<sup>h</sup> Division of Gastroenterology, Hepatology, and Nutrition, Department of Internal Medicine, Virginia Commonwealth University School of Medicine, Richmond 23298, VA, USA

<sup>i</sup> Departamento de Gastroenterología, Escuela de Medicina, Pontificia Universidad Católica de Chile, Santiago, Chile

<sup>j</sup> Instituto de Salud Pública de Chile, Santiago, Chile

## ARTICLE INFO

## Article History:

Received 22 October 2024

Accepted 17 February 2025

Available online 1 May 2025

## Keywords:

Non-alcoholic fatty liver disease (NAFLD)  
Metabolic dysfunction-associated fatty liver disease (MAFLD)  
Steatotic Liver Disease (SLD)

## ABSTRACT

**Introduction and Objectives:** This study aims to assess the performance of Steatotic Liver Disease (SLD) criteria in identifying liver steatosis compared to the NAFLD and MAFLD definitions in an agricultural population in Chile.

**Patients and Methods:** We performed a cross-sectional analysis on the MAUCO cohort, composed of 9,013 individuals aged 38 to 74. Health conditions, socio-demographics, anthropometrics, hepatic ultrasonography, blood pressure, and biological samples were obtained. Participants were classified as NAFLD, MAFLD, or any of the five SLD categories: Metabolic dysfunction-associated steatosis liver disease (MASLD), Metabolic and Alcohol-Associated Liver Disease (MetALD), Alcohol-Associated Liver Disease (ALD), Specific aetiologies, and Cryptogenic. The Framingham cardiovascular risk score and BARD liver fibrosis score were used to assess clinical relevance.

**Results:** Liver steatosis was present in 4,082 participants (45%); SLD criteria captured an additional 176 individuals not classified under NAFLD and 103 not included under MAFLD definition. The main SLD subgroups were MASLD (95%), MetALD (1.9%) and ALD (1.3%). Individuals classified in the MetALD and ALD subgroups exhibited more severe liver steatosis and a higher cardiovascular risk. Notably, participants categorized under specific etiologies and cryptogenic subgroups were younger and had a higher risk for liver fibrosis.

**Conclusions:** The study reveals that SLD offers a more inclusive classification to identify high-risk individuals in the Chilean population, capturing cases that could be missed by NAFLD or MAFLD definitions by using the same resources.

© 2025 Published by Elsevier España, S.L.U. on behalf of Fundación Clínica Médica Sur, A.C. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>)

**Abbreviations:** ALD, Alcohol-Associated Liver Disease; ALT/GOT, Alanine Aminotransferase; AST/GPT, Aspartate Aminotransferase; GGT, Gamma-Glutamyl Transferase; HDL, High-Density Lipoprotein; HOMA, Homeostatic Model Assessment; MAFLD, Metabolic Dysfunction-Associated Fatty Liver Disease; MASLD, Metabolic Dysfunction-Associated Steatosis Liver Disease; MetALD, Metabolic Alcohol-Associated Liver Disease; NAFLD, Non-alcoholic Fatty Liver Disease; SLD, Steatotic Liver Disease

\* Corresponding author.

E-mail address: [cferrecc@uc.cl](mailto:cferrecc@uc.cl) (C. Ferreccio).

<https://doi.org/10.1016/j.aohp.2025.101919>

1665-2681/© 2025 Published by Elsevier España, S.L.U. on behalf of Fundación Clínica Médica Sur, A.C. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>)

## 1. Introduction

Non-alcoholic fatty liver disease (NAFLD) is the most frequent chronic liver disease, with an estimated global prevalence in adults of 24–25% [1]. NAFLD rates are rising, leading to a substantial clinical and economic burden [2,3]. In Latin America, where obesity and type 2 diabetes, major risk factors of NAFLD [4,5], are increasing, the prevalence of NAFLD is believed to be higher than reported [6]. A decade ago, the adult prevalence in Chile was 23% [7], reaching 47.5% in 2019 [8].

**Table 1**  
Inclusion and exclusion criteria for NAFLD, MAFLD, and SLD.

Criteria	NAFLD	MAFLD	SLD				
			MASLD	MetALD	ALD	Specific aetiologies	Cryptogenic
Liver steatosis by Ultrasound	+	+	+	+	+	+	+
BMI <sup>1</sup>		+	+	+			
Waist circumference <sup>2</sup>		+	+	+			
Type 2 diabetes <sup>3</sup>		+	+	+			
Prediabetes <sup>3</sup>		+					
Insuline resistance <sup>3</sup>		+					
Hypertension <sup>4</sup>		+	+	+			
Triglycerides <sup>5</sup>		+	+	+			
HDL <sup>6</sup>		+	+	+			
C-RP <sup>7</sup>		+					
Moderate alcohol consumption <sup>8</sup>	-	+		+			
Excessive alcohol consumption <sup>9</sup>	-	-			+		
Hepatitis <sup>10</sup>	-	+				+	
Hepatotoxic drugs <sup>11</sup>	-	-				+	

+Inclusion criteria  
-Exclusion criteria  
<sup>1</sup> BMI: Body Mass Index,  $\geq 25$  kg/m<sup>2</sup>  
<sup>2</sup> Waist circumference  $\geq 80$  cm women,  $\geq 94$  cm men for SLD and  
<sup>3</sup> For SLD, diabetes is defined as fasting serum glucose  $\geq 100$  mg/dl or 2-hour post-load glucose  $\geq 140$ mg/dl or diagnosis for type 2 diabetes or treatment. For MAFLD, diabetes is defined as fasting glucose  $\geq 126$  mg/dL or specific drug treatment, prediabetes as fasting glucose 100– 125 mg/ dL, and insulin resistance as homeostasis model assessment of insulin resistance [HOMA] score  $\geq 2.5$ .  
<sup>4</sup> Blood pressure  $\geq 130/85$  mm Hg or treatment for hypertension.  
<sup>5</sup> Plasma  $\geq 150$  mg/dl or treatment.  
<sup>6</sup> HDL cholesterol  $\leq 50$  mg/dl women,  $\leq 40$  mg/dl men  
<sup>7</sup> Plasma high-sensitivity C- reactive protein  $\geq 0.2$  mg/dL  
<sup>8</sup> Weekly intake of 140–350 grams for women and 210–420 grams for men or an average daily of 20–50 grams for women and 30–60 grams for men  
<sup>9</sup> Weekly intake of 350 grams for women and 420 grams for men or an average daily of 50 grams for women and 60 grams for men  
<sup>10</sup> History of Hepatitis B or C  
<sup>11</sup> Use of amiodarone > six months, methotrexate > two years, tamoxifen > one year, prednisone > six months, and valproic acid > one and half months.

NAFLD encompasses a wide disease spectrum, from steatosis to fibrosis, potentially progressing to cirrhosis and liver cancer [9]. However, its exclusionary diagnosis can miss patients with diverse etiologies [10]. In 2020, an expert panel proposed the term metabolic dysfunction-associated fatty liver disease (MAFLD), which considered the metabolic context of fatty liver disease [11,12]. Although more inclusive, MAFLD required diabetes, obesity, or two metabolic risk factors, potentially excluding high-risk patients and limiting understanding of the disease [13]. In 2023, the term: Steatotic Liver Disease (SLD) [13] was introduced by three international liver associations and patient representatives. This new definition encompasses all causes of liver steatosis, categorizing them into subgroups with common health contexts and prognoses. This study aimed to assess whether the SLD definition improves liver steatosis identification in our population compared to NAFLD and MAFLD.

2. Patients and Methods

2.1. Study population and design

This study included 9,013 participants aged 38 to 74 years from the MAUCO population-based cohort in Chile; details published elsewhere [14]. Participants' health history, physical examination, laboratory testing (glycemia, insulin, homeostatic model assessment [HOMA], high-density lipoprotein [HDL] cholesterol, triglycerides, aspartate aminotransferase [ALT/GOT], alanine aminotransferase [AST/GPT], gamma-glutamyl transferase [GGT]) and hepatobiliary ultrasound were collected [8,14]. Ultrasounds were performed with the Siemens ACUSON P500TM by a trained technician and under radiologist supervision. Liver steatosis was defined as Normal, Mild, Moderate, or Severe based on the Rumack criteria [15].

2.2. Definitions of NAFLD, MAFLD, and SLD

Participants were categorized according to the three criteria. Table 1 shows the inclusion and exclusion criteria for NAFLD [12], MAFLD [11], and SLD subgroups: Metabolic dysfunction-associated steatosis liver disease (MASLD), Metabolic Alcohol-Associated Liver Disease (MetALD), Alcohol-Associated Liver Disease (ALD), Specific aetiologies, and Cryptogenic [13].

2.3. Risk of severity

To identify individuals with the highest risk health outcome within each group, liver fibrosis risk was assessed using BARD score and also the Framingham cardiovascular risk score was used. The Framingham score considers age, gender, HDL cholesterol, systolic blood pressure, and smoking history, where >10% is classified as risk of developing a cardiovascular disease in 10 years [16]. BARD score considers BMI  $\geq 28$  (1 point), AST/ALT ratio  $\geq 0.8$  (2 points), and diabetes (1 point), categorizing 0–1 as low and 2–4 points as high risk of developing fibrosis [17].

2.4. Statistical analysis

We conducted only descriptive statistical analyses to characterize the different SLD subgroups. Continuous variables are presented as means with standard deviations (SD), while categorical variables are expressed as percentages. The percentage of missing data was minimal; therefore, no data imputation was performed. All analyses were conducted using R (Version 1.4.1106, © 2009–2021, RStudio, PBC) [18].

**Table 2**  
Profile of 9,013 participants allocated in each of the six-steatosis liver disease (SLD) classes.

		a. Normal (n:4,931)	b.MASLD (n:3,892)	c.MetALD (n:77)	d.ALD (n:53)	e.Specific aetiology (n:45)	f.Cryptogenic (n:12)
Sociodemographic							
Women (4,936)		2,730 (55.4)	2,132 (54.7)	26 (33.8)	8 (15.1)	37 (82.2)	3 (25)
Men (4,077)		2,201 (44.6)	1,760 (45.3)	51 (66.2)	45 (84.9)	8 (17.8)	9 (75)
Age, mean $\pm$ SD		53.6 (10.2)	53.8 (9.5)	53.6 (9.9)	52.1 (10.3)	55.9 (9.6)	51.0 (8.5)
Educational level	$\leq 8$	2,357 (48)	1,894 (49)	34 (44)	23 (44.2)	21 (46.7)	9 (75)
	9-12	1,983 (40.4)	1,513 (39.1)	30 (39)	19 (36.5)	18 (40)	1 (8.3)
	$\geq 13$	566 (11.5)	462 (11.9)	13 (16.9)	10 (19.2)	6 (13.3)	2 (16.7)
Health profile							
Hepatitis		4 (0.1)	0 (0)	0 (0)	0 (0)	11 (24.4)	0 (0)
Alcohol <sup>1</sup>	Moderate or worse	124 (2.5)	0 (0)	77 (100)	53 (100)	0 (0)	1 (8.3)
Ultrasound	Mild	0 (0)	2,455 (63)	39 (50.6)	34 (64.2)	28 (62.2)	12 (100)
	Moderate	0 (0)	1,345 (34.5)	34 (44.2)	18 (34)	16 (35.6)	0 (0)
	Severe	0 (0)	95 (2.4)	4 (5.2)	1 (1.9)	1 (2.2)	0 (0)
BMI ( $\geq 25$ kg/m <sup>2</sup> )		3,666 (74.5)	3,740 (96)	75 (97.4)	48 (90.6)	43 (95.6)	0 (0)
Waist circumference <sup>2</sup>		2,478 (50.5)	3,216 (82.7)	62 (80.5)	39 (73.6)	41 (91.1)	0 (0)
Diabetes		457 (9.3)	873 (22.5)	14 (18.2)	7 (13.5)	2 (4.5)	0 (0)
Prediabetes		795 (17)	1,067 (32.1)	30 (44.1)	12 (24.5)	2 (4.8)	0 (0)
HOMA <sup>3</sup>		471 (29.6)	1,010 (69.3)	14 (73.7)	4 (66.7)	15 (68.2)	0 (0)
Hypertension		1,971 (40.1)	2,071 (53.3)	50 (64.9)	36 (67.9)	21 (46.7)	0 (0)
High Blood pressure <sup>4</sup>		1,538 (31.2)	1,589 (40.8)	43 (55.8)	29 (54.7)	5 (11.1)	0 (0)
HDL <sup>5</sup>		2,231 (45.4)	2,417 (62.3)	41 (53.2)	21 (40.4)	7 (15.9)	0 (0)
Triglycerides <sup>6</sup>		1,838 (37.4)	2,325 (59.9)	48 (62.3)	28 (53.8)	6 (13.6)	0 (0)
C-RP <sup>7</sup>		667 (41.9)	940 (64.5)	13 (68.4)	5 (83.3)	16 (72.7)	2 (40)
ALT/GOT, mean $\pm$ SD		27.5 (19.5)	40.5 (29.1)	48.0 (31.1)	53.4 (30.6)	33.5 (27.4)	28.1 (10.0)
AST/GPT, mean $\pm$ SD		25.4 (13.3)	30.8 (17.3)	35.9 (21.0)	53.4 (80.4)	28.0 (13.0)	32.9 (13.2)
GGT, mean $\pm$ SD		32.1 (46.0)	39.5 (48.3)	76.0 (74.6)	55.4 (49.2)	35.3 (17.2)	43.2 (48.0)
BARD <sup>11</sup>	Low	0 (0)	731 (18.8)	14 (18.2)	5 (9.4)	0 (0)	0 (0)
	High	0 (0)	3,162 (81.2)	62 (80.5)	41 (77.4)	10 (22.2)	11 (91.7)
Framingham <sup>11</sup>	>10%/10years	1,145 (23.2)	991 (25.4)	31 (40.3)	25 (47.2)	5 (11.1)	2 (16.7)

Absolute numbers are present with their percentages in parentheses unless the mean value is indicated.

<sup>1</sup> Moderate intake: weekly intake of 140-350 grams for women and 210-420 grams for men or an average daily of 20-50 grams for women and 30-60 grams for men. Excessive intake: weekly intake of 350 grams for women and 420 grams for men or an average daily of 50 grams for women and 60 grams for men

<sup>2</sup> Waist circumference:  $\geq 80$  cm women,  $\geq 94$  cm men.

<sup>3</sup> HOMA score  $\geq 2.5$ .

<sup>4</sup> Blood pressure  $\geq 130/85$  mm Hg.

<sup>5</sup> HDL cholesterol  $\leq 50$  mg/dl women,  $\leq 40$  mg/dl men.

<sup>6</sup> Plasma  $\geq 150$  mg/dl or treatment.

<sup>7</sup> Plasma high-sensitivity C-reactive protein  $\geq 0.2$  mg/dL

<sup>10</sup>History of Hepatitis B or C.

<sup>11</sup> BARD: score of fibrosis; Framingham: Framingham score of cardiovascular disease.

## 2.5. Ethical statement

MAUCO was approved by the ethics committees of Pontificia Universidad Católica de Chile (N° 14-141) and the Maule Regional Service of the Chilean Ministry of Health [14]. Written informed consent was obtained from each participant included in this study.

## 3. Results

Among the 9,013 adults in the MAUCO cohort, 4,082 (45%) had confirmed liver steatosis. As expected, those with liver steatosis had a significantly higher prevalence of obesity, type 2 diabetes, prediabetes, hypertension, and dyslipidemia (Table 2). Of these, 95% were classified as "MASLD," 1.9% "MetALD," 1.3% "ALD," 1.1% "Specific etiology" and 0.3% "Cryptogenic" (Fig. 1).

### 3.1. Transition from NAFLD/MAFLD to SLD

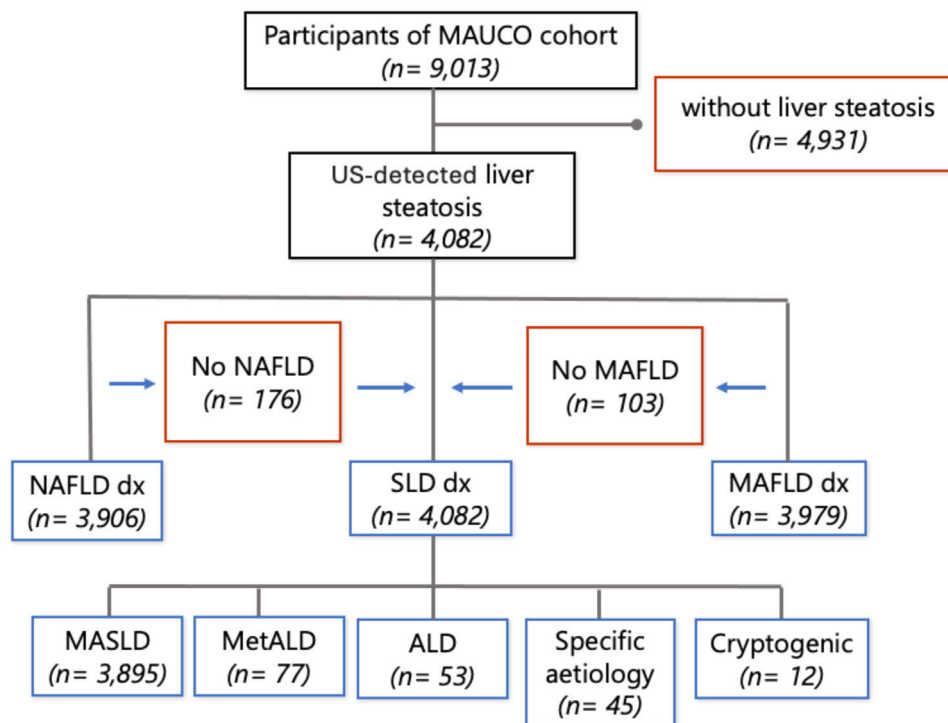
Transitioning from NAFLD to SLD criteria included 176 additional participants excluded under NAFLD criteria (Fig. 1). These participants were predominantly men (59.7%) with a high burden of cardiometabolic risk factors. They also had moderate (38.6%) and severe (3.4%) steatosis, with a high risk of liver fibrosis (86.7%) and cardiovascular disease (35%) (Supplementary table 1).

In contrast, when switching from MAFLD to SLD, the criteria included 103 individuals who had been excluded under MAFLD criteria (Fig. 1). These were mainly women (63.1%) with healthier

cardiometabolic profiles. While only 22.3% had moderate steatosis and 1% severe, 82.1% had a high risk of developing liver fibrosis and 21.6% risk of developing cardiovascular disease (Supplementary table 1).

### 3.2. New SLD classification

MASLD group (95%) was the largest, followed by MetALD (1.9%) and ALD (1.3%). These three groups had similar health profiles, with sex distribution being the primary difference. MASLD was 54.7% of women, while MetALD and ALD were 33.8% and 15.1%, respectively (Table 2). Specific aetiology and Cryptogenic groups, newly incorporated classification, show slightly different profiles. The specific aetiology group was mainly women (82.2%) under 60 (Supplementary table 2) with low rates of chronic diseases. This group includes most participants with a history of hepatitis B and C (24.4%) (Table 2), and 78% of them use hepatotoxic drugs, prednisone the most used (Supplementary table 2). The cryptogenic group is primarily composed of men (75%) under the age of 60 years (Supplementary table 2) with a healthier cardiometabolic profile across the five groups and mild liver steatosis (100%). Across the five groups, MetALD had the highest prevalences of moderate and severe liver steatosis (44.2% and 5.2%, respectively). Regarding severity outcomes, Cryptogenic had the highest risk of developing liver fibrosis (91.7%), followed by MASLD (81.2%) and MetALD (80.5%). The risk of cardiovascular disease was more predominant in ALD and MetALD (47.2% and 40.3%, respectively).



**Fig. 1.** The flowchart illustrates the selection of individuals with ultrasound-confirmed steatosis from the MAUCO cohort. These participants were classified according to NAFLD and MAFLD definitions, with excluded cases highlighted. At the bottom of the diagram, all individuals with confirmed steatosis were reclassified using the updated SLD framework and subcategories.

#### 4. Discussion

Our findings suggest that SLD classification benefits the MAUCO cohort, classifying all patients with liver steatosis regardless of etiology. Shifting from NAFLD to SLD criteria increased the identification of high-risk populations by adding 176 patients. Despite MAFLD having wider criteria than NAFLD [12], it still missed 103 participants at higher risk for liver fibrosis and cardiovascular disease. With liver steatosis affecting a quarter of the global population [19], adopting more inclusive criteria is crucial for diagnosing and treating.

The MASLD group was the most predominant, encompassing most patients with systemic metabolic dysregulation associated with liver steatosis, such as obesity and diabetes [20]. Including participants with at least one cardiometabolic factor effectively captured most patients with a high burden of chronic diseases, particularly in populations with high rates of obesity, diabetes, prediabetes, and hypertension, like those in Chile [21]. Although MAFLD incorporates metabolic disease, it prioritizes overweight/obese individuals and those with diabetes, making it more likely to miss cases with only one chronic disease [10]. Studies from the United States, Brazil, and China demonstrate strong agreement between MASLD and NAFLD/MAFLD. This suggests that transitioning to SLD will not compromise the validity of prior NAFLD and MAFLD research [22–25] and will benefit patients by including a subgroup of high-risk individuals.

Incorporating the MetALD and ALD subgroups clarifies the role of moderate or excessive alcohol consumption in causing steatosis. Previous studies emphasize the importance of distinguishing MetALD from MASLD and ALD due to their distinct pathogenic mechanisms and prognostic outcomes [10]. While MASLD and MetALD share similar health profiles and risks, moderate alcohol consumption with cardiometabolic factors significantly increases the risk of liver fibrosis and cardiovascular disease. Similar findings were reported by the UK Biobank study, with MASLD predominantly affecting women with a higher burden of metabolic syndrome. In contrast, MetALD mainly

affected men with worse liver profiles, characterized by elevated ALT, AST, and GGT levels [26].

The inclusion of Specific aetiology and Cryptogenic groups introduces a key distinction. Our findings show that although the Specific etiology group had high rates of obesity and hypertension, they were excluded from MASLD group because their liver steatosis was primarily linked to a history of hepatitis B and C infections and the use of hepatotoxic drugs—both preventable factors. The frequent overlap of conditions in steatotic liver disease, particularly those linked to metabolic syndrome, infection history, or hepatotoxin use, highlights the need to manage all the steatogenic factors for appropriate patient treatment [20]. On the other hand, the Cryptogenic group lacks a specific etiology for liver steatosis [27]. Our data indicate that while this group had a healthier cardiometabolic profile, it has a high risk of developing liver fibrosis (100%) and cardiovascular disease (16.7%). Similar findings were observed in ELSA-Brazil and Japanese cohorts, where participants previously classified as NAFLD without cardiometabolic profiles under the SLD were placed in the Cryptogenic group [23,28]. In Japan, studies also found that this group was relatively small compared to others and had a healthier profile [28]. They observed that the Cryptogenic group had a lower risk of metabolic complications than other SLD subgroups, emphasizing the need for dedicated studies to better understand the underlying causes of liver steatosis in this population and to develop targeted treatments [28].

Our study has limitations. The diagnosis of liver steatosis was made using ultrasound, which has variable sensitivity and specificity that improves with disease progression and is highly operator-dependent [29]. These limitations may lead to an underestimation of cases. However, current guidelines in the United States, Europe, and Latin America recommend ultrasound due to its practicality, low cost, safety, and wide availability [30–32]. Given the high burden of metabolic syndrome and its strong association with SLD, the high prevalence of SLD found in the MAUCO cohort, detected by ultrasound, appears reasonable. Studies have shown that MASLD and MetALD are



the most prevalent subcategories, are closely linked to metabolic syndrome [13,20], with obesity, diabetes, and hypertension as primary risk factors [12]. Approximately 65% of obese patients and 70% of individuals with type 2 diabetes have MASLD [33]. In our cohort, 84% of participants are overweight or obese, 65% have a high waist circumference, 15% have diabetes, and 46% have hypertension, supporting the high SLD prevalence observed.

Another limitation is the reliance on self-reported alcohol consumption, which may lead to an underestimation of alcohol intake and, consequently, the prevalence of MetALD and ALD. Additionally, the history of Hepatitis B or C infections was self-reported, potentially underestimating past viral infections. However, national data from the Chilean National Health Survey (2009–2010), which included serological testing (HBsAg and anti-HCV), reported a low prevalence of hepatitis B (0.15%) and hepatitis C (0.01%) [34]. Although few cases were self-reported in our study [34], these national prevalence rates suggest that hepatitis rates in our cohort would likely be low. Despite these limitations, a key strength of our study is that it is one of the few population-based studies in Latin America, offering valuable insights into SLD classification within a well-characterized Chilean agricultural cohort.

## 5. Conclusions

In conclusion, transitioning to SLD criteria was beneficial in identifying high-risk patients in our population, particularly those at significant risk of developing liver fibrosis and cardiovascular disease, who should be prioritized for medical and lifestyle interventions. A more inclusive definition raises awareness of the disease among the medical community and the general population.

## Funding

This study was supported by Fondo Nacional de Desarrollo Científico y Tecnológico (FONDECYT) from the government of Chile (grant 1212066 (C.F.), 1211879 (D.C.) and 1241450 (M.A.) and Advanced Center for Chronic Diseases (ACCDIS) FONDAP 15130011 (C.F.), and Beca de Doctorado Nacional ANID 21241360 (M.S.S.).

## Declaration of competing interest

None.

## Acknowledgement

The success of this investigation would not have been possible without the exceptional teamwork of the field staff who oversaw the recruitment, interviews, and data collection. Special thanks to Ricardo Erazo, Cristian Herrera, Ian Reyes, Matias Pozo, Miguel Carrera, Carolina Riveros, and Marjorie Barrera from the Santiago team; Pia Venegas and Jenifer Loyola for coordinating fieldwork; and Fernando Herrera, who obtained the ultrasound tests. Vicente Cid for his help in the preliminary data processing. Appreciation is also expressed to all the participants who agreed to be part of the MAUCO cohort and have been part of this for the past years.

## Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.aohep.2025.101919.

## References

[1] Younossi ZM, Golabi P, de Avila L, Paik JM, Srishord M, Fukui N, et al. The global epidemiology of NAFLD and NASH in patients with type 2 diabetes: a systematic

review and meta-analysis. *J Hepatol* 2019;71:793–801. <https://doi.org/10.1016/j.jhep.2019.06.021>.

[2] Arab JP, Díaz LA, Dirchwolf M, Mark H, Lazarus JV, Vaughan E, et al. NAFLD: Challenges and opportunities to address the public health challenge in Latin America. *Ann Hepatol* 2021;24:100359. <https://doi.org/10.1016/j.aohep.2021.100359>.

[3] Stepanova M, Henry L, Younossi ZM. Economic burden and patient-reported outcomes of nonalcoholic fatty liver disease. *Clin Liver Dis* 2023;27:483–513. <https://doi.org/10.1016/j.cld.2023.01.007>.

[4] Arab JP, Arrese M, Trauner M. Recent insights into the pathogenesis of nonalcoholic fatty liver disease. *Ann Rev Pathol: Mech Disease* 2018;13:321–50. <https://doi.org/10.1146/annurev-pathol-020117-043617>.

[5] Haas JT, Francque S, Staels B. Pathophysiology and mechanisms of nonalcoholic fatty liver disease. *Annu Rev Physiol* 2016;78:181–205. <https://doi.org/10.1146/annurev-physiol-021115-105331>.

[6] Pinto Marques Souza de Oliveira C, Pinchemel Cotrim H, Arrese M. Nonalcoholic fatty liver disease risk factors in Latin American populations: current scenario and perspectives. *Clin Liver Dis (Hoboken)* 2019;13:39–42. <https://doi.org/10.1002/cld.759>.

[7] Riquelme A, Arrese M, Soza A, Morales A, Baudrand R, RM Pérez-ayuso, et al. Non-alcoholic fatty liver disease and its association with obesity, insulin resistance and increased serum levels of C-reactive protein in Hispanics. *Liver Int* 2009;29:82–8. <https://doi.org/10.1111/j.1478-3231.2008.01823.x>.

[8] Ferreccio C, Huidobro A, Cortés S, Bambs C, Toro P, de Wyngard V Van, et al., et al. Cohort profile: The Maule Cohort (MAUCO). *Int J Epidemiol* 2021;49 760–760. <https://doi.org/10.1093/ije/DYAA003>.

[9] Portincasa P, Baffy G. Metabolic dysfunction-associated steatotic liver disease: evolution of the final terminology. *Eur J Intern Med* 2024;124:35–9. <https://doi.org/10.1016/j.ejim.2024.04.013>.

[10] Kim GA, Moon JH, Kim W. Critical appraisal of metabolic dysfunction-associated steatotic liver disease: Implication of Janus-faced modernity. *Clin Mol Hepatol* 2023;29:831–43. <https://doi.org/10.3350/cmh.2023.0277>.

[11] Eslam M, Sanyal AJ, George J, Sanyal A, Neuschwander-Tetri B, Tiribelli C, et al. MAFLD: a consensus-driven proposed nomenclature for metabolic associated fatty liver disease. *Gastroenterology* 2020;158:1999–2014.e1. <https://doi.org/10.1053/j.gastro.2019.11.312>.

[12] Wong R, Fortune BE. The Shift from nonalcoholic fatty liver disease to metabolic dysfunction-associated fatty liver disease. *Clin Liver Dis (Hoboken)* 2022;20:157–61. <https://doi.org/10.1002/cld.1250>.

[13] Rinella ME, Lazarus JV, Ratzliff V, Francque SM, Sanyal AJ, Kanwal F, et al. A multi-society Delphi consensus statement on new fatty liver disease nomenclature. *Ann Hepatol* 2024;29:101133. <https://doi.org/10.1016/j.aohep.2023.101133>.

[14] Ferreccio C, Roa JC, Bambs C, Vives A, Corvalán AH, Cortés S, et al. Study protocol for the Maule Cohort (MAUCO) of chronic diseases, Chile 2014–2024. *BMC Public Health* 2016;16:1–7. <https://doi.org/10.1186/s12889-015-2454-2>.

[15] Rumack CM, Wilson SR, Charboneau JW, Levine D. Diagnostic ultrasound. 4th ed. 2011.

[16] Jahangiri L, Farhangi MA, Rezaei F. Framingham risk score for estimation of 10-years of cardiovascular diseases risk in patients with metabolic syndrome. *J Health Popul Nutr* 2017;36:1–6. <https://doi.org/10.1186/s41043-017-0114-0>.

[17] Harrison SA, Oliver D, Arnold HL, Gogia S, Neuschwander-Tetri BA. Development and validation of a simple NAFLD clinical scoring system for identifying patients without advanced disease. *Gut* 2008;57:1441–7. <https://doi.org/10.1136/gut.2007.146019>.

[18] R Core Team. R: A language and environment for statistical computing. <https://www.r-project.org/2023>.

[19] Kokkorakis M, Boutari C, Katsiki N, Mantzoros CS. From non-alcoholic fatty liver disease (NAFLD) to steatotic liver disease (SLD): an ongoing journey towards refining the terminology for this prevalent metabolic condition and unmet clinical need. *Metabolism* 2023;147. <https://doi.org/10.1016/j.metabol.2023.155664>.

[20] El-Kassas M, Alsawat K, Tharwat M, Labidi A, Medhat MA, Sanai FM, et al. Steatotic liver disease as a new nomenclature for NAFLD from the perspectives of the MENA region: one size fits all this time. *J Hepatol* 2023. <https://doi.org/10.1016/j.jhep.2023.08.012>.

[21] Salud Ministerio de. Encuesta Nacional de Salud 2016–2017 Primeros resultados. Departamento de Epidemiología, División de Planificación Sanitaria, Subsecretaría de Salud Pública 2017:61 <http://web.minsal.cl/wp-content/uploads/2017/11/ENS-2016-17-PRIMEROS-RESULTADOS.pdf>.

[22] Zou H, Ma X, Pan W, Xie Y. Comparing similarities and differences between NAFLD, MAFLD, and MASLD in the general U.S. population. *Front Nutr* 2024;11. <https://doi.org/10.3389/fnut.2024.1411802>.

[23] Perazzo H, Pacheco AG, Griep RH, Gracino R, Goulart AC, da Fonseca M de JM. Changing from NAFLD through MAFLD to MASLD: Similar prevalence and risk factors in a large Brazilian cohort. *J Hepatol* 2023. <https://doi.org/10.1016/j.jhep.2023.08.025>.

[24] Ciardullo S, Carbone M, Invernizzi P, Perseghin G. Exploring the landscape of steatotic liver disease in the general US population. *Liver Int* 2023;43:2425–33. <https://doi.org/10.1111/liv.15695>.

[25] Song SJ, Lai JCT, Wong GLH, Wong VWS, Yip TCF. Can we use old NAFLD data under the new MASLD definition? *J Hepatol* 2024;80:e54–6. <https://doi.org/10.1016/j.jhep.2023.07.021>.

[26] Schneider KM, Schneider CV. A new era for steatotic liver disease: evaluating the novel nomenclature in the UK biobank. *J Hepatol* 2023. <https://doi.org/10.1016/j.jhep.2023.07.007>.

[27] Ye Q, Zou B, Yeo YH, Li J, Huang DQ, Wu Y, et al. Global prevalence, incidence, and outcomes of non-obese or lean non-alcoholic fatty liver disease: a systematic review and meta-analysis. *Lancet Gastroenterol Hepatol* 2020;5:739–52. [https://doi.org/10.1016/S2468-1253\(20\)30077-7](https://doi.org/10.1016/S2468-1253(20)30077-7).

- [28] He L, Zheng W, Qiu K, Kong W, Zeng T. Changing from NAFLD to MASLD: The new definition can more accurately identify individuals at higher risk for diabetes. *J Hepatol* 2024;80:e85–7. <https://doi.org/10.1016/j.jhep.2023.09.035>.
- [29] Castera L, Vilgrain V, Angulo P. Noninvasive evaluation of NAFLD. *Nat Rev Gastroenterol Hepatol* 2013;10:666–75. <https://doi.org/10.1038/nrgastro.2013.175>.
- [30] Rinella ME, Neuschwander-Tetri BA, Siddiqui MS, Abdelmalek MF, Caldwell S, Barb D, et al. AASLD practice guidance on the clinical assessment and management of nonalcoholic fatty liver disease. *Hepatology* 2023;77:1797–835. <https://doi.org/10.1097/HEP.0000000000000323>.
- [31] Berzigotti A, Tsochatzis E, Boursier J, Castera L, Cazzagon N, Friedrich-Rust M, et al. EASL clinical practice guidelines on non-invasive tests for evaluation of liver disease severity and prognosis –2021 update. *J Hepatol* 2021;75:659–89. <https://doi.org/10.1016/j.jhep.2021.05.025>.
- [32] Arab JP, Dirchwolf M, Álvares-da-Silva MR, Barrera F, Benítez C, Castellanos-Fernandez M, et al. Latin American Association for the study of the liver (ALEH) practice guidance for the diagnosis and treatment of non-alcoholic fatty liver disease. *Ann Hepatol* 2020;19:674–90. <https://doi.org/10.1016/j.aohp.2020.09.006>.
- [33] Eslam M, Newsome PN, Sarin SK, Anstee QM, Targher G, Romero-Gomez M, et al. A new definition for metabolic dysfunction-associated fatty liver disease: An international expert consensus statement. *J Hepatol* 2020;73:202–9. <https://doi.org/10.1016/j.jhep.2020.03.039>.
- [34] Encuesta Nacional 2009–2010 n.d. <https://www.minsal.cl/portal/url/item/bcb03d7bc28b64dfe040010165012d23.pdf> (accessed February 13, 2025).