J Biochem Technol (2026) 17(2): 30-44
https://doi.org/10.51847/YbhmIQqB3d
ISSN: 0974-2328

Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD): Clinical Profile, Phenotypes, and Risk Stratification. A Narrative Review

Zalina Yuryevna Sozaeva*, Marina Vsevolodovna Atayeva, Elizaveta Olegovna Salatova, Irina Kazbekovna Alikova, Alexandra Evgenievna Dolgaya, Amin Gasaevich Magomedov, Kamilla Ramisovna Hafsi, Nur-Mukhamed Aslanovich Shomakhov, Elena Mukhadinovna Pshukova, Yulia Nikolaevna Sharaf

Received: 15 January 2026 / Received in revised form: 05 April 2026, Accepted: 08 April 2026, Published online: 25 May 2026

Abstract

Metabolically dysregulated steatotic liver disease (MASLD) affects approximately one-third of the global adult population. This narrative review presents the clinical profile of the contemporary patient with MASLD, focusing on the systemic nature of the disease and risk stratification. MASLD is a systemic metabolic disorder in which hepatic steatosis serves as a marker of insulin resistance, systemic inflammation, and atherogenic dyslipidemia. The leading cause of death in MASLD patients is cardiovascular events, with a two-fold increase in cardiovascular mortality that persists across all fibrosis stages. Disease trajectory is determined by fibrosis stage, comorbid profile (type 2 diabetes, hypertension, dyslipidemia), and phenotype. Three main phenotypes have been identified: classical metabolic (60-70%, dominant cardiovascular risk), lean (15-20%, higher PNPLA3 I148M carriage, faster fibrosis progression, underdiagnosis), and mixed Met-ALD (10–15%, synergistic liver damage from alcohol and metabolic disturbances). Non-invasive fibrosis assessment using FIB-4 followed by elastography is the first step after diagnosis. Patients with fibrosis F3–F4 require aggressive monitoring, including hepatocellular carcinoma screening every 6 months, while patients with F0–F2 require mandatory cardiovascular risk control in primary care. Management priorities differ by phenotype: cardiometabolic control for classical metabolic, fibrosis screening and genetic counseling for lean, and complete alcohol cessation for Met-ALD. Lifestyle modification (weight loss of 5–10%, Mediterranean diet, 150 minutes of physical activity per week) has stronger evidence than any experimental drug. The proposed risk stratification algorithm can be implemented both in primary care and in specialized hepatology centers.

Keywords: MASLD, NAFLD, Fibrosis, Cardiovascular risk, Lean phenotype, Met-ALD

Zalina Yuryevna Sozaeva*, Marina Vsevolodovna Atayeva
Department of Children's Diseases №2, North Ossetian State Medical Academy, Vladikavkaz, Republic of North Ossetia-Alania, Russia.

Elizaveta Olegovna Salatova, Irina Kazbekovna Alikova, Alexandra Evgenievna Dolgaya
Faculty of Pediatrics, North Ossetian State Medical Academy, Vladikavkaz, Republic of North Ossetia-Alania, Russia.

Amin Gasaevich Magomedov, Kamilla Ramisovna Hafsi
Institute of Dentistry, Saratov State Medical University named after V.I. Razumovsky, Saratov, Russia.

Nur-Mukhamed Aslanovich Shomakhov
Faculty of Medicine, Institute of Clinical Medicine named after N.V. Sklifosovsky, First Moscow State Medical University named after I.M. Sechenov, Moscow, Russia.

Elena Mukhadinovna Pshukova
Department of Normal and Pathological Anatomy, Medical Academy, Kabardino-Balkarian State University named after H.M. Berbekov, Nalchik, Republic of Kabardino-Balkaria, Russia.

Yulia Nikolaevna Sharaf
Faculty of Medicine, Medical Academy, Kabardino-Balkarian State University named after H.M. Berbekov, Nalchik, Republic of Kabardino-Balkaria, Russia.

***E-mail:** dr.zayushka@mail.ru

Introduction

Metabolic dysfunction-associated steatotic liver disease (MASLD) is a chronic condition defined by the accumulation of triglycerides in hepatocytes exceeding 5% of liver weight, in the absence of hepatotoxic alcohol consumption (less than 30 g of ethanol per day for men and less than 20 g for women) (Eslam *et al*., 2020; Lazarus *et al*., 2024). MASLD is not a single disease but a spectrum of pathological states ranging from reversible steatosis (fat accumulation without inflammation) to non-alcoholic steatohepatitis (NASH), where inflammation and hepatocyte ballooning appear, and further to fibrosis, cirrhosis, and hepatocellular carcinoma (HCC) (Younossi *et al*., 2016). For a long time, clinicians considered MASLD a benign condition that did not require active intervention, but this view has now been revised (Younossi *et al*., 2018).

According to a 2022 global meta-analysis including over 10 million people from 22 countries, the prevalence of MASLD

among adults is 32.4% (Riazi *et al*., 2022). The highest rates are in the Middle East (38%) and South America (35%), while the lowest rates are in Africa (17%) (Riazi *et al*., 2022). Recent Russian research has focused on emerging pharmacotherapeutic targets for NAFLD, including novel agents targeting insulin resistance, lipotoxicity, and inflammation (Prikhodko *et al*., 2022). Over the past 20 years, the incidence of MASLD has more than doubled, and if current trends continue, the number of patients with MASLD-related cirrhosis will increase by 168% by 2030 (Estes *et al*., 2018). In the Russian Federation, the prevalence of MASLD is estimated at 28–37%, comparable to European data, and MASLD is the most common chronic liver disease in Russia, surpassing viral hepatitis and alcoholic liver disease (Maevskaya *et al*., 2022). The condition is also affecting younger people: the prevalence of MASLD among children in developed countries reaches 7–8%, and among children with obesity, it reaches 34% (Nobili *et al*., 2019; Maevskaya *et al*., 2022).

MASLD is a systemic pathology (Targher *et al*., 2024). First, the disease has a long asymptomatic course. Early stages show no jaundice, no pain, and no pruritus. The most common complaints are non-specific fatigue and right upper quadrant discomfort, which have no diagnostic value (Amangurbanova *et al*., 2023). By the time overt clinical signs appear (ascites, variceal bleeding, hepatic encephalopathy), patients typically already have decompensated cirrhosis (Sanyal *et al*., 2021). Second, the leading cause of death in MASLD patients is not liver complications but cardiovascular events. Prospective cohort studies show that MASLD is associated with a two-fold increase in cardiovascular mortality, a risk that persists across all stages of fibrosis (Zheng *et al*., 2025). The pathophysiological basis includes insulin resistance, systemic inflammation, and atherogenic dyslipidemia, for which fatty liver serves as a marker (Targher *et al*., 2024). Third, 20–30% of patients with NASH develop cirrhosis within 10–15 years, and among patients with MASLD-related cirrhosis, the annual risk of HCC is 2–4% (Le *et al*., 2024). In developed countries, MASLD has become the third leading cause of HCC (Le *et al*., 2024).

The key clinical challenge is the heterogeneity of patients with MASLD (Younossi *et al*., 2018). Disease trajectory differs significantly between individuals and is determined by multiple factors: fibrosis stage, comorbid conditions (type 2 diabetes, hypertension, dyslipidemia), genetic polymorphisms (notably PNPLA3 I148M), and alcohol consumption patterns (Eslam *et al*., 2020; Zheng *et al*., 2025). Thus, MASLD represents a spectrum of pathological states with different prognoses and management approaches. **Figure 1** shows disease progression from reversible steatosis to irreversible stages of fibrosis, cirrhosis, and HCC (Le *et al*., 2024). The main therapeutic window is in the early stages (steatosis and steatohepatitis without advanced fibrosis), whereas at stages F3–F4, intervention options are substantially limited.

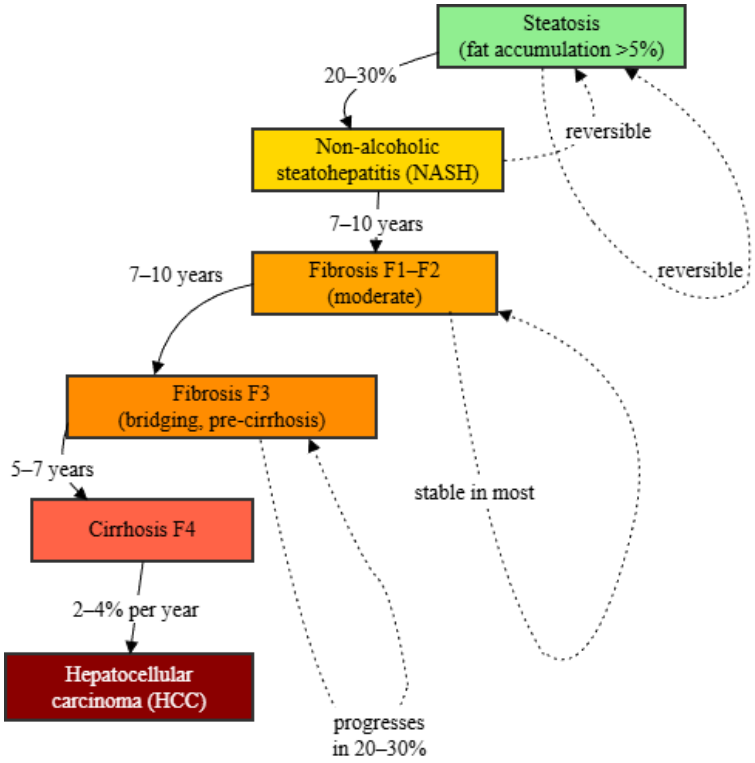

Figure 1. Progression of MASLD (from steatosis to hepatocellular carcinoma).

The figure shows disease stages from steatosis to hepatocellular carcinoma (HCC), including the proportion of patients transitioning to each subsequent stage and the time intervals for progression. Solid arrows indicate disease progression, while dashed arrows indicate possible stabilization or regression. Green (steatosis) and yellow (NASH) mark stages with high potential for reversal through lifestyle modification. Orange and red mark stages where regression is unlikely and therapeutic options are

largely limited to slowing progression. Dark red (HCC) marks the terminal stage with the poorest prognosis. The time intervals shown are average data from cohorts of patients with NASH and may vary significantly depending on risk factors such as type 2 diabetes, genetic polymorphisms, and alcohol consumption. F1–F4 indicate fibrosis stages according to the Kleiner (METAVIR) scale.

Goal of this work: Based on an analysis of current data, including the new MASLD nomenclature (Metabolic Dysfunction-Associated Steatotic Liver Disease), this work aims to present the clinical profile of the contemporary patient with steatotic liver disease associated with metabolic dysfunction (Abdulrahman *et al*., 2023; Zigmantavičius *et al*., 2023; Chakraborty & Rajasekar, 2024; Son & Lee, 2024). The main focus is on the systemic nature of the disease and on patient risk stratification.

Demographic Profile of the Patient and the New Nomenclature

The classic demographic profile of patients with NAFLD, established by population-based studies over the past decade, is now being refined with the introduction of new nomenclature (Guo *et al*., 2025). The average patient profile is as follows. Age is over 40 years, although patients aged 30–35 years with confirmed steatosis are increasingly seen in clinical practice; the peak incidence occurs in the 50–65 year age group (Younossi *et al*., 2023). Gender differences are ambiguous: NAFLD is slightly more common in men, but under MASLD criteria, the proportion of women may increase due to metabolic disturbances associated with the postmenopausal period (Shaheen *et al*., 2021). Body mass index (BMI) typically exceeds 25 kg/m², indicating overweight or obesity. Waist circumference (greater than 94 cm in men and greater than 80 cm in women) indicates abdominal fat distribution, which is most closely associated with insulin resistance (Ross *et al*., 2020).

A key clinical feature is the existence of the "lean" phenotype. Up to 22% of patients with confirmed steatotic liver disease have a normal BMI (<25 kg/m²) (Danpanichkul *et al*., 2025). These patients have no visible signs of obesity, but instrumental imaging reveals excessive visceral obesity (Seko *et al*., 2025). Some data suggest that the risk of fibrosis progression in this patient group is higher than in patients with overt obesity (Ampuero *et al*., 2024). This is likely explained by a higher frequency of pathogenic genetic variants, most notably PNPLA3 I148M (Boeckmans *et al*., 2023).

At the end of 2023, the American Association for the Study of Liver Diseases (AASLD) and the European Association for the Study of the Liver (EASL) proposed replacing the term NAFLD with MASLD (Metabolic Dysfunction-Associated Steatotic Liver Disease) (Rinella *et al*., 2023). This change reflects a shift in diagnostic logic: from a "diagnosis of exclusion" to a "diagnosis of inclusion." MASLD requires the presence of hepatic steatosis plus at least one cardiometabolic risk factor (Kanwal *et al*., 2024). The alcohol criterion is preserved. For patients with metabolic dysfunction and alcohol consumption above the MASLD threshold (more than 20 g/day for women and 30 g/day for men) but below the threshold for isolated alcoholic liver disease, the category MetALD (metabolic dysfunction-associated alcoholic liver disease) has been introduced. This category reflects the synergistic damaging effect of alcohol and metabolic disturbances (Kido *et al*., 2025). The diagnostic criteria for MASLD are presented in **Table 1** (Rinella *et al*., 2023).

Table 1. Diagnostic criteria for MASLD (Rinella *et al.*, 2023)

Parameter	Criterion
Mandatory condition	Presence of hepatic steatosis (confirmed by imaging, biopsy, or biomarkers)
Plus, at least one of the five cardiometabolic risk factors	
1. Anthropometric measures	BMI ≥25 kg/m² *or* waist circumference >94 cm (men) / >80 cm (women)
2. Glucose metabolism	Fasting glucose ≥5.6 mmol/L *or* postprandial glucose ≥7.8 mmol/L *or* HbA1c ≥5.7% *or* established type 2 diabetes
3. Blood pressure	≥130/85 mmHg *or* use of antihypertensive drugs
4. Triglycerides	≥1.7 mmol/L *or* use of lipid-lowering drugs
5. HDL cholesterol	≤1.0 mmol/L (men) / ≤1.3 mmol/L (women) *or* use of lipid-lowering drugs

The shift to MASLD nomenclature has several practical consequences (Kanwal *et al*., 2024). First, it is no longer necessary to exclude other liver diseases. Combined diagnoses are now possible (e.g., MASLD + chronic hepatitis B). Second, the focus shifts from the liver as an isolated organ to systemic metabolic disturbances. Third, the term “steatotic” is considered less stigmatizing than “fatty” (Rinella *et al*., 2023).

Criticisms of the new nomenclature exist. One concern is the difficulty of interpreting clinical studies conducted under the old NAFLD label. Another is the possible invalidity of cardiometabolic risk factor thresholds across different racial and ethnic groups (Teng *et al*., 2023). Nevertheless, the scientific literature of 2024–2025 predominantly uses the term MASLD (Younossi *et al*., 2025).

Thus, analyzing the profile of the contemporary patient requires abandoning the concept of the “NAFLD patient” as an isolated hepatological case. Instead, we must adopt a model of a patient with metabolic dysfunction, for whom the liver is one of several target organs—and often not the most important one for prognosis (Kanwal *et al*., 2024).

Comorbid Profile: Associated Diseases as Key to Prognosis

The leading cause of death in patients with MASLD is not liver complications but cardiovascular disease (Zheng *et al*., 2024; Lim *et al*., 2025). According to prospective cohort studies and meta-

analyses, myocardial infarction and ischemic stroke rank first in the mortality structure of this patient group. Liver cirrhosis and its complications rank second, and hepatocellular carcinoma ranks third (Wong *et al*., 2023). The gap between cardiovascular and liver mortality persists across all stages of fibrosis, except in decompensated cirrhosis, where liver causes become predominant (Carrieri *et al*., 2022). This observation has two implications. First, managing a MASLD patient based solely on liver tests and fibrosis stage ignores the main threat to life. Second, comorbid diseases (arterial hypertension, dyslipidemia, type 2 diabetes mellitus (T2DM), and obesity) are key drivers of both liver damage and overall mortality (Grander *et al*., 2023; Nysather *et al*., 2023).

The link between MASLD and cardiovascular disease has independent pathophysiological mechanisms beyond shared risk factors (Gao *et al*., 2025). The fatty liver secretes pro-inflammatory cytokines (C-reactive protein, tumor necrosis factor-alpha, interleukin-6), coagulation factors (plasminogen activator inhibitor-1), and atherogenic lipoproteins (Miller *et al*., 2025). The resulting systemic inflammatory and procoagulant state damages the vascular endothelium, promotes atherosclerosis, and increases the risk of thrombosis (Byrne & Targher, 2022). In addition, MASLD serves as a marker of severe insulin resistance, which underlies most cardiometabolic disturbances (Hagström *et al*., 2024). Patients with MASLD have significantly higher rates of coronary artery calcification, increased carotid intima-media thickness, and ventricular arrhythmias (Lee *et al*., 2024). Importantly, the elevated cardiovascular risk persists after adjusting for traditional risk factors, allowing MASLD to be considered an independent predictor of cardiovascular events (Zhou *et al*., 2025).

Type 2 diabetes mellitus occupies a special place in the comorbid profile. The prevalence of T2DM among MASLD patients reaches 30–40%, compared to 9–10% in the general population (Ferguson & Finck, 2021). The combination of MASLD and T2DM is associated with a 2.5- to 3-fold higher risk of developing advanced fibrosis (stages F3–F4) compared to patients without diabetes (Genua *et al*., 2023). Chronic hyperglycemia and insulin resistance enhance lipotoxicity, oxidative stress, and hepatocyte apoptosis, accelerating the transition from steatosis to steatohepatitis and fibrosis (Lee *et al*., 2022). Conversely, MASLD in a patient with T2DM increases the risk of microvascular (nephropathy, retinopathy) and macrovascular complications. Thus, T2DM and MASLD form a mutually reinforcing combination (Cho *et al*., 2023).

Arterial hypertension and dyslipidemia are found in 60–80% of patients with MASLD, often in forms resistant to standard therapy (Zisis *et al*., 2025). The characteristic dyslipidemia profile includes elevated triglycerides, decreased HDL cholesterol, and an increased proportion of small, dense LDL particles—the most atherogenic fraction (Stefan *et al*., 2025). Statins, often unjustifiably avoided in patients with "liver disease," are safe in most MASLD patients and may provide additional hepatoprotective effects through anti-inflammatory action (Bharali *et al*., 2021).

Obstructive sleep apnea (OSA) is a common but underrecognized comorbid condition. In patients with obesity and MASLD, the prevalence of OSA can reach 70% (Bonsignore, 2022). Episodes of nocturnal hypoxia followed by reoxygenation trigger oxidative stress and activate pro-inflammatory pathways in the liver, accelerating fibrosis progression (Ji *et al*., 2022). Treating OSA with CPAP therapy may slow the progression of liver damage (Parola & Pinzani, 2024).

Chronic kidney disease (CKD) is closely associated with MASLD. Shared pathophysiological mechanisms include endothelial dysfunction, activation of the renin-angiotensin-aldosterone system, and chronic systemic inflammation (Bilson *et al*., 2024). The risk of developing CKD in MASLD patients is 1.5- to 2-fold higher than in the general population (Yang *et al*., 2021). In patients with MASLD-related cirrhosis, the rate of estimated glomerular filtration rate decline exceeding 40% reaches 3 events per 100 person-years, necessitating regular monitoring of kidney function (Baratta *et al*., 2022).

Endocrine comorbidities include hypothyroidism and polycystic ovary syndrome (PCOS). The prevalence of hypothyroidism in MASLD patients is significantly higher than in the general population, especially among women (Mantovani *et al*., 2024). Hypothyroidism worsens dyslipidemia and slows intrahepatic lipid metabolism. PCOS, characterized by hyperandrogenism and insulin resistance, is associated with a higher prevalence of MASLD in women of reproductive age (Falzarano *et al*., 2022). Identifying and correcting these conditions is an integral part of managing MASLD patients.

In summary, the comorbid profile of a patient with MASLD is a systemic picture where each accompanying disease is linked to the others (Djeagou *et al*., 2025). Cardiovascular risks determine overall mortality. T2DM and obesity drive fibrosis progression. OSA, hypothyroidism, and PCOS act as additional modulators requiring special attention. **Figure 2** presents the pathogenetic links between MASLD and the main comorbid conditions, illustrating that the fatty liver is an active participant in systemic inflammation and metabolic dysregulation, justifying a multidisciplinary approach.

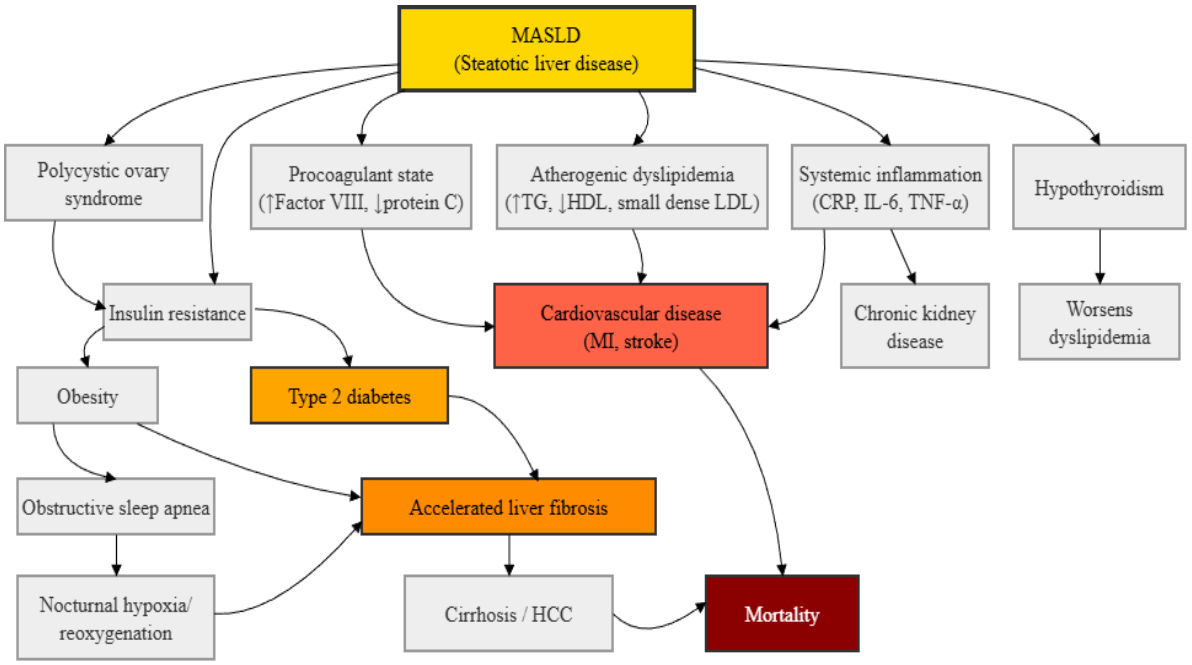

Figure 2. Pathogenetic links between MASLD and major comorbid conditions.
Abbreviations: CRP, C-reactive protein; IL-6, interleukin-6; TNF-α, tumor necrosis factor-alpha; TG, triglycerides; HDL, high-density lipoprotein; LDL, low-density lipoprotein; T2DM, type 2 diabetes mellitus; HCC, hepatocellular carcinoma.
Note: The diagram is not exhaustive and reflects only the most established links. Interactions between individual comorbid conditions (e.g., the effect of T2DM on CKD risk) are not shown to preserve clarity.

The diagram illustrates the central role of MASLD in generating systemic metabolic and inflammatory disturbances. Solid arrows indicate direct pathophysiological links. Dashed arrows indicate links mediated through intermediate mechanisms. Yellow highlights the central condition (MASLD). Orange highlights key comorbid diseases and pathological processes. Red highlights terminal outcomes.

Prognostic Profile: From Steatosis to Cirrhosis

The disease trajectory of MASLD varies substantially between patients (Lekakis & Papatheodoridis, 2024). In some patients, steatosis does not progress for decades. These individuals are highly likely to die from cardiovascular disease without ever developing clinically significant liver damage (Noureddin & Wong, 2023; Sarkar *et al*., 2023; Wang, 2024). In other patients, steatosis transforms relatively quickly (within 5–7 years) into non-alcoholic steatohepatitis (NASH), then into fibrosis and cirrhosis (Fang *et al*., 2024). A third group first comes to attention already at the stage of decompensated cirrhosis (Sanyal *et al*., 2025). The clinical task is to identify patients at high risk of progression as early as possible.

The most powerful prognostic factor for liver outcomes in MASLD is fibrosis stage (Angulo *et al*., 2015). It is the degree of replacement of normal liver parenchyma by connective tissue, rather than the severity of steatosis or aminotransferase levels, that determines the risk of liver complications and mortality (Dulai *et al*., 2017). In a prospective multicenter study by Sanyal *et al*. (2021) including 1,773 patients with biopsy-proven NAFLD, total mortality was 0.32 per 100 person-years in patients with fibrosis stages F0–F2, with virtually no liver complications (Quek *et al*., 2023). At stage F3, mortality increased to 0.89 per 100 person-years. At established cirrhosis (stage F4), mortality reached 1.76 per 100 person-years, and the frequency of decompensation and hepatocellular carcinoma (HCC) increased significantly (Quek *et al*., 2023). Importantly, the rate of cardiovascular events did not differ between fibrosis stages, meaning that elevated cardiovascular risk persists at all stages (Kasper *et al*., 2021). **Figure 3** shows fibrosis stages with corresponding mortality rates and risks of liver complications.

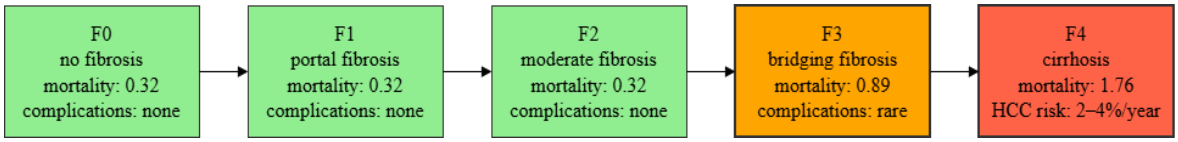

Figure 3. Stages of liver fibrosis in MASLD and associated risks. *Abbreviation:* HCC, hepatocellular carcinoma.

Figure 3 illustrates fibrosis stages according to the METAVIR scale (F0–F4). Each block shows the stage, its characteristics, total mortality (per 100 person-years of follow-up), and the frequency of liver complications. Green highlights stages F0–F2 (low risk of liver complications; mortality is determined primarily by cardiovascular causes). Orange highlights stage F3 (intermediate risk). Red highlights stage F4 (cirrhosis, high risk of decompensation and hepatocellular carcinoma). Arrows indicate the direction of fibrosis progression.

Assessment of fibrosis stage should be the first step after diagnosing MASLD (Pouwels *et al*., 2022). Liver biopsy, while the reference standard, has limitations for routine screening due to invasiveness, cost, and sampling error (Abdelhameed *et al*., 2024). Non-invasive methods are used in clinical practice, primarily calculated indices. The FIB-4 index and the NAFLD Fibrosis Score (NFS) have high negative predictive value (Kang *et al*., 2024). A FIB-4 value below 1.3 reliably excludes advanced fibrosis; such patients can be followed in primary care, with emphasis on controlling cardiometabolic risk factors (Kjaergaard *et al*., 2023). A FIB-4 value above 2.67 requires elastography or, in doubtful cases, biopsy (Mózes *et al*., 2022). Vibration-controlled transient elastography (VCTE) and magnetic resonance elastography (MRE) have established optimal cut-off values for diagnosing advanced fibrosis in patients with NAFLD, with high diagnostic accuracy (Chon *et al*., 2024).

Beyond fibrosis, genetic polymorphisms have prognostic significance (Cherubini *et al*., 2023). The PNPLA3 I148M variant, carried by up to 40% of European-descent individuals and up to 70% in some Latin American populations, is associated with higher liver fat content, accelerated fibrosis progression, and increased HCC risk (Cherubini *et al*., 2021). In homozygous carriers, the risk of advanced fibrosis is 2–3 times higher than in wild-type carriers (Hsueh *et al*., 2022). A young patient with normal BMI and normal liver tests who carries PNPLA3 I148M may have a worse prognosis than an older patient with obesity without this variant (Wang *et al*., 2026). Genetic testing is not yet routine but may be justified in patients with positive family history or aggressive disease without obvious metabolic risk factors (Cherubini *et al*., 2023).

Patient age at the time of steatosis diagnosis is also significant (Alqahtani & Schattenberg, 2021). In patients younger than 40–45 years, longer exposure to metabolic disturbances leads to a higher cumulative risk of progression to cirrhosis by age 60–70 years. In patients older than 65 years, newly diagnosed steatosis without aggressive factors (T2DM, high baseline fibrosis) is likely to remain stable. Age influences therapeutic strategy: more aggressive intervention is indicated for younger patients, while older patients require control of cardiometabolic risks (Alqahtani & Schattenberg, 2021).

Fibrosis progression rate is variable (Huang *et al*., 2023). In patients with NASH, transition by one fibrosis stage takes an average of 7–10 years (Paternostro & Trauner, 2022). However, in patients with T2DM, high histological activity, and genetic predisposition, progression may take 3–5 years, whereas in the absence of these factors, it may take 15–20 years (Huang *et al*., 2023). This requires individualization of follow-up intervals. Low-risk patients (FIB-4 <1.3, no T2DM, no PNPLA3) should be reassessed every 2–3 years. Intermediate-risk patients (FIB-4 1.3–2.67, T2DM or obesity) should be reassessed annually. High-risk patients (FIB-4 >2.67, confirmed fibrosis F3–F4) should be reassessed every 6 months with mandatory liver ultrasound for HCC screening (Koh *et al*., 2024).

Although this review focuses on adults, a brief comment on pediatric MASLD is warranted. The prevalence of MASLD among children in developed countries reaches 7–8%, and among children with obesity, it reaches 34% (Nobili *et al*., 2019). Longitudinal data suggest that pediatric MASLD can progress to advanced fibrosis and cirrhosis by early adulthood, particularly in the presence of type 2 diabetes or PNPLA3 risk alleles (Nobili *et al*., 2019; Cherubini *et al*., 2023). However, pharmacotherapy options for children are extremely limited, and lifestyle modification remains the cornerstone of management. Screening strategies for pediatric populations are not yet standardized, representing a critical gap for future research (Nobili *et al*., 2019).

Synthetic Profile: Patient Phenotypes

Behind the diagnosis of MASLD lies a heterogeneous group of patients (Baratta *et al*., 2022). However, this heterogeneity can be organized into several recurring phenotypes. Each phenotype has a characteristic disease trajectory, specific risks, and consequently requires different management strategies (Cusi *et al*., 2025). Identifying phenotypes is a practical tool for patient stratification in the context of limited consultation time (Houttu *et al*., 2024).

Based on analysis of demographic, comorbid, and genetic data, three main phenotypes have been identified: classical metabolic, lean (genetically driven), and mixed Met-ALD (metabolic dysfunction-associated alcoholic liver disease) (Powell *et al*., 2021; Israelsen *et al*., 2024). Their comparative characteristics are presented in **Table 2.**

Table 2. Comparative characteristics of MASLD phenotypes

Feature	Classical metabolic	Lean	Met-ALD
Proportion among MASLD	60–70%	15–20%	10–15%
BMI	>30 kg/m²	<25 kg/m²	any
Fat distribution	overt abdominal obesity	visceral (hidden)	often abdominal
Type 2 diabetes	very common (40–50%)	rare	less common than classical
Hypertension/dyslipidemia	very common	less common	common
PNPLA3 I148M	population frequency	significantly higher	population frequency
Alcohol consumption	none (<30/20 g/day)	none (<30/20 g/day)	moderate (30–60/20–40 g/day)

Dominant risk	cardiovascular	liver (fibrosis, HCC)	mixed, with an accent on liver damage
Management priorities	BP, lipids, glucose control; weight loss	fibrosis screening, genetic counseling, visceral fat reduction	complete alcohol cessation + metabolic control

Figure 4 shows the estimated distribution of phenotypes in the MASLD patient population (Horn & Tacke, 2024; Zhou *et al*., 2026). The classical metabolic phenotype is dominant (60–70% of cases). However, the lean phenotype and Met-ALD together account for up to one third of patients and require special attention due to their higher risk of liver outcomes and, in the case of the lean phenotype, systematic underdiagnosis (Zhou *et al*., 2026).

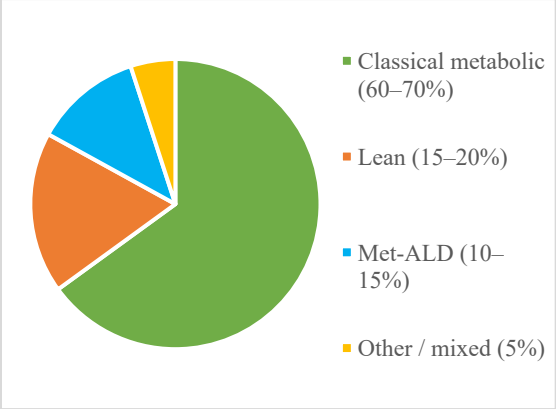

Figure 4. Distribution of phenotypes in the MASLD patient population

Note: The lean phenotype is characterized by normal body mass index (<25 kg/m²) but the presence of visceral obesity and/or carriage of the pathogenic PNPLA3 I148M variant (Das, *et al*., 2025). The Met-ALD phenotype requires the presence of metabolic dysfunction and alcohol consumption at doses of 20–30 g/day for women and 30–60 g/day for men (Portincasa *et al.,* 2024).

The classical metabolic phenotype is the most common. Its key feature is a high frequency of cardiovascular events, which requires priority control of blood pressure, lipids, and glucose (Njei *et al*., 2024). The lean phenotype is characterized by normal BMI but a higher frequency of PNPLA3 I148M carriage and faster fibrosis progression (Das *et al*., 2025). These patients often remain undiagnosed due to the absence of overt signs of obesity (Ramandi *et al*., 2025). The Met-ALD phenotype is distinguished by the synergistic damaging effect of alcohol and metabolic disturbances (Portincasa *et al*., 2024). These patients have higher aminotransferase levels and a significantly higher risk of fibrosis and HCC than in isolated MASLD, while arterial hypertension and T2DM are less common (Babaei *et al*., 2023; Kusumawardani *et al*., 2023; Doddapanen *et al*., 2024; Joungtrakul & Smith, 2024; Shaji *et al*., 2024). The absolute priority for this group is complete alcohol cessation (Elmustafa *et al*., 2025).

The three phenotypes presented do not exhaust the full clinical diversity. Variants with dominant renal comorbidity, severe obstructive sleep apnea as the main driver, or combinations with hypothyroidism or polycystic ovary syndrome are possible (Njei *et al*., 2024). Nevertheless, identifying the three main phenotypes allows the physician to determine the priority specialty for coordinating patient management: cardiologist (classical metabolic phenotype), hepatologist and geneticist (lean phenotype), or addiction specialist (Met-ALD) (Powell *et al*., 2021; Elmustafa *et al*., 2025).

Results and Discussion

The present analysis confirms the initial thesis of heterogeneity within MASLD and the need for a stratified approach (Han *et al*., 2023). The traditional management model, focused primarily on liver tests and exclusion of alternative etiologies, is being replaced by a model in which comorbidity, genetic factors, and fibrosis stage are assessed together, and therapeutic priorities are determined by the dominant risk—cardiovascular, liver, or mixed (Eslam *et al*., 2022).

Data from large cohort studies allow us to state that fibrosis stage is the main predictor of liver outcomes. In contrast, cardiovascular outcomes are less dependent on fibrosis stage and are determined primarily by classical risk factors potentiated by MASLD-associated systemic inflammation (Badmus *et al*., 2023; Quek *et al*., 2023). This has two practical implications. A patient with MASLD and fibrosis F3–F4 requires aggressive monitoring of liver complications, including screening for hepatocellular carcinoma every 6 months (Koh *et al*., 2024). However, this does not negate the need for parallel control of blood pressure, lipid profile, and glycemia. A patient with MASLD and fibrosis F0–F2 should not be considered to have a “benign” course in terms of overall mortality. Their cardiovascular risk remains elevated, and it is this risk that will most likely determine the outcome (Kasper *et al*., 2021).

The introduction of the MASLD nomenclature represents a shift in diagnostic logic from exclusion to inclusion (Rinella *et al*., 2023; Kanwal *et al*., 2024). However, this shift raises several controversial issues. First, the threshold values for cardiometabolic factors adopted by the AASLD/EASL consensus are to some extent arbitrary. Their applicability across different racial and ethnic groups requires additional validation (Aboona *et al*., 2024). For example, the >94 cm waist circumference threshold for men, established for the European population, may be inadequate for individuals of Asian descent (Goedecke *et al*., 2022). Second, clinical studies conducted under the old NAFLD label now require careful interpretation, as the inclusion criteria may have systematically differed from those of the MASLD criteria (Guo *et al*., 2025). Third, the Met-ALD category introduces a new diagnostic threshold (20–30 g of ethanol per day), which is consensus-based and lacks a clear pathophysiological rationale (Zeng *et al*., 2024; Elmustafa *et al*., 2025).

Up to 22% of patients with confirmed steatotic liver disease have a normal BMI (Danpanichkul *et al*., 2025). In routine clinical practice, such patients are systematically underdiagnosed because

physicians typically do not suspect fatty liver disease in a lean individual with normal or borderline liver tests (Ramandi *et al*., 2025). This delays diagnosis until the stage of established fibrosis (Zhou *et al*., 2026). The question of expanding indications for non-invasive fibrosis assessment (e.g., FIB-4 calculation) to all patients with metabolic disturbances regardless of BMI remains open (Sanyal *et al*., 2023). Several authors have proposed including the lean phenotype as a separate category in screening recommendations, but the evidence base for such recommendations is currently insufficient (Ampuero *et al*., 2024; Das *et al*., 2025).

Most of the data cited in this work comes from Western populations (USA, Europe) and Asian cohorts. Extrapolating these data to the Russian population requires caution (Zoncapè *et al*., 2024). The prevalence of obesity and T2DM in Russia is somewhat lower than in the USA, and the pattern of alcohol consumption has its own characteristics (Maevskaya *et al*., 2022). Specialized Russian cohort studies on MASLD are limited, underscoring the need for local epidemiological research (Li *et al*., 2024).

Based on the present analysis, the following algorithm for risk stratification and management of patients with MASLD is proposed **(Figure 5).** The algorithm includes four sequential steps: phenotype assessment, calculation of the FIB-4 index, elastography (if needed), and therapy selection based on the dominant risk (Bansal *et al.,* 2025; EASL, 2024). The algorithm was developed taking into account the availability of the methods and can be implemented both at the primary care level and in specialized hepatology centers (Mózes *et al.*, 2022).

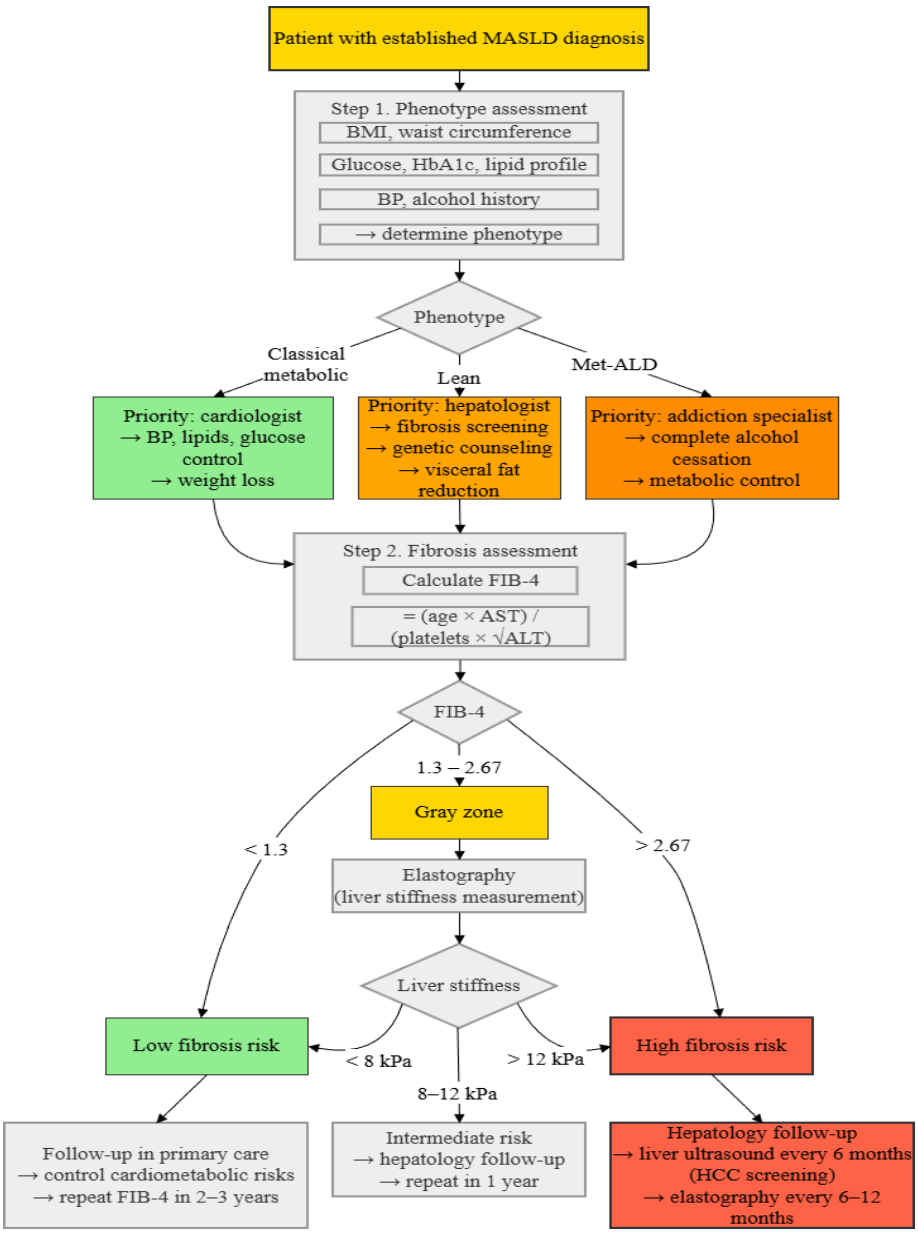

Figure 5. Management algorithm for patients with MASLD: risk stratification and patient routing.

Abbreviations: BMI, body mass index; BP, blood pressure; HbA1c, glycated hemoglobin; AST, aspartate aminotransferase; ALT, alanine aminotransferase; HCC, hepatocellular carcinoma.

Note*: I*n patients with confirmed fibrosis F3–F4 (liver stiffness >12 kPa or FIB-4 >2.67 combined with clinical signs), liver ultrasound for HCC screening is indicated every 6 months, regardless of phenotype (Koh *et al*., 2024)

The flowchart presents the sequence of steps from establishing a diagnosis of MASLD to selecting therapy and determining follow-up frequency (Arroyo-Fernández *et al*., 2023; Mislishayeva *et al*., 2023; Triantafyllopoulos *et al*., 2023; Lin & Mukai, 2024; Miranda *et al*., 2024; Prada *et al*., 2024). The algorithm includes four steps: (1) phenotype assessment based on anthropometric, laboratory, and anamnestic data; (2) determination of the priority specialty for coordinating management (cardiologist, hepatologist, addiction specialist) depending on phenotype; (3) non-invasive assessment of fibrosis stage using the FIB-4 index and, if necessary, elastography; (4) risk stratification and assignment of appropriate follow-up frequency (Hima *et al*., 2024; Ganea *et al*., 2024; Varoneckaitė *et al*., 2024).

Criteria and threshold values:

- FIB-4 < 1.3 — low risk of advanced fibrosis
- FIB-4 1.3–2.67 — gray zone, elastography required
- FIB-4 > 2.67 — high risk of advanced fibrosis (F3–F4)
- Liver stiffness < 8 kPa — low risk
- Liver stiffness 8–12 kPa — intermediate risk
- Liver stiffness > 12 kPa — high risk (F3–F4)

Limitations of the Work

This work has several limitations. First, it is a narrative review, not a systematic review with formal quality assessment of studies (Silva *et al*., 2025). Second, we deliberately did not address pharmacotherapy for MASLD (GLP-1 receptor agonists, ACC inhibitors, FXR agonists), as this is beyond the scope of our focus on patient profile and risk stratification (Wang *et al*., 2021). Third, the proposed phenotypes and algorithm require prospective validation in clinical studies (Wang *et al*., 2025). Nevertheless, we believe that the proposed model is practically applicable now, as it is based on routine parameters (FIB-4 is calculated from standard laboratory tests, elastography is widely available in regional centers, and phenotype identification requires only thorough history taking and basic examination) (Mózes *et al.*, 2022; Kjaergaard *et al.*, 2023).

Conclusion

Non-alcoholic fatty liver disease in its current conceptualization as MASLD is not an isolated hepatological disease but a systemic metabolic pathology in which the liver serves as one of several target organs. The prevalence of MASLD reaches 32% among adults worldwide. Incidence is steadily increasing, and the age of onset is decreasing. However, the main clinical significance of this disease is determined not so much by liver complications themselves, but by the fact that hepatic steatosis serves as a marker of severe insulin resistance, systemic inflammation, and atherogenic dyslipidemia — conditions that are direct causes of cardiovascular death, which ranks first in the mortality structure of patients with MASLD.

Prognosis in patients with MASLD is determined by the interaction of three groups of factors: fibrosis stage (main predictor of liver outcomes), comorbid profile (type 2 diabetes, arterial hypertension, dyslipidemia determine cardiovascular risk), and phenotype (classical metabolic, lean, or mixed Met-ALD). Ignoring any of these components leads to incomplete risk assessment and, consequently, to inadequate monitoring strategies.

Fibrosis stage should be assessed in every patient with MASLD using non-invasive methods (a two-step approach: calculate FIB-4, then elastography if needed). Patients with fibrosis F3–F4 require aggressive monitoring of liver complications (including HCC screening every 6 months), whereas patients with fibrosis F0–F2 can be followed in primary care but with mandatory control of cardiovascular risks.

Comorbid diseases (T2DM, arterial hypertension, dyslipidemia, obesity, obstructive sleep apnea, hypothyroidism, polycystic ovary syndrome) should be actively identified and treated. Prescribing statins and antihypertensive therapy in patients with MASLD is not only safe but necessary to reduce cardiovascular mortality.

Identifying three phenotypes — classical metabolic, lean (genetically driven), and Met-ALD — allows the physician to determine the priority specialty for coordinating patient management (cardiologist, hepatologist/geneticist, addiction specialist, respectively) and to select targeted therapy: control of cardiometabolic factors in the classical phenotype, aggressive fibrosis screening and genetic counseling in the lean phenotype, and complete alcohol cessation in the Met-ALD phenotype.

Lifestyle modification (weight loss of 5–10% for overweight patients, Mediterranean diet with restriction of fructose and simple sugars, physical activity of at least 150 minutes per week) has a higher level of evidence than any experimental pharmacological agent and should be recommended to all patients with MASLD.

The results of this work identify several areas requiring further study. First, validation of the proposed phenotypes and stratification algorithm is needed in prospective Russian cohorts, taking into account the characteristics of the Russian population (alcohol consumption patterns, prevalence of genetic polymorphisms, availability of diagnostic methods). Second, screening strategies for the lean phenotype of MASLD, which is currently systematically underdiagnosed, require development and clinical testing. Third, studies are needed to evaluate the efficacy and safety of new pharmacological agents in Russian populations, considering the comorbid background. Fourth, the development of Russian clinical guidelines for the management of patients with MASLD, adapted to local epidemiological and resource conditions, is advisable (Osonoi & Takebe, 2024; Zhang *et al.*, 2026).

Contemporary management of a patient with MASLD is not about waiting for an "ideal" pharmacological agent. It is about

systematic, consistent work with the patient's metabolic health, based on proper risk stratification and application of proven interventions. Weight loss, control of blood pressure, lipid profile, and glycemia, alcohol cessation, and regular physical activity currently have a higher level of evidence than any experimental therapy. The clinical task is not to limit oneself to documenting hepatic steatosis, but to assess the entire spectrum of metabolic disturbances, refer the patient to appropriate specialists in a timely manner, and not lose sight of the main threat — cardiovascular disease. Behind every liver disease stands a patient with their heart, blood vessels, kidneys, and long-term habits. One must treat the patient, not the organ.

Acknowledgments: The authors would like to thank the medical librarians at North Ossetian State Medical Academy (Vladikavkaz, Republic of North Ossetia-Alania, Russia) for their assistance with the literature search.

Conflict of interest: None

Financial support: None

Ethics statement: As this article is a narrative literature review and does not involve original data collection from human participants or animals, ethical approval was not required. All cited studies were conducted in accordance with their respective ethical standards and the principles of the Declaration of Helsinki.

References

Abdelhameed, F., Kite, C., Lagojda, L., Dallaway, A., Chatha, K. K., Chaggar, S. S., Dalamaga, M., Kassi, E., Kyrou, I., & Randeva, H. S. (2024). Non-invasive scores and serum biomarkers for fatty liver in the era of metabolic dysfunction-associated steatotic liver disease (MASLD): A comprehensive review from NAFLD to MAFLD and MASLD. *Current Obesity Reports, 13*(3), 510–531. doi:10.1007/s13679-024-00574-z

Abdulrahman, B. I., Aldahmash, A. M., Alghamdi, H. H., Alghamdi, A. H., Hamad, T. A. B., & Ruished, A. F. (2023). Prevalence of dental anomalies among patients visiting the Riyadh Elm University Clinics. *Annals of Dental Specialty, 11*(2), 40–45. doi:10.51847/4wajSjZRu8

Aboona, M. B., Faulkner, C., Rangan, P., Ng, C. H., Huang, D. Q., Muthiah, M., Nevah Rubin, M. I., Han, M. A. T., Fallon, M. B., Kim, D., et al. (2024). Disparities among ethnic groups in mortality and outcomes among adults with MASLD: A multicenter study. *Liver International, 44*(6), 1316–1328. doi:10.1111/liv.15880

Alqahtani, S. A., & Schattenberg, J. M. (2021). NAFLD in the elderly. *Clinical Interventions in Aging, 16*, 1633–1649. doi:10.2147/CIA.S295524

Amangurbanova, M., Huang, D. Q., & Loomba, R. (2023). Review article: The role of HSD17B13 on global epidemiology, natural history, pathogenesis and treatment of NAFLD. *Alimentary Pharmacology & Therapeutics, 57*(1), 37–51. doi:10.1111/apt.17292

Ampuero, J., Aller, R., Gallego-Durán, R., Crespo, J., Calleja, J. L., García-Monzón, C., Gómez-Camarero, J., Caballería, J., Lo Iacono, O., Ibañez, L., et al. (2024). The biochemical pattern defines MASLD phenotypes linked to distinct histology and prognosis. *Journal of Gastroenterology, 59*(7), 586–597. doi:10.1007/s00535-024-02098-8

Angulo, P., Kleiner, D. E., Dam-Larsen, S., Adams, L. A., Bjornsson, E. S., Charatcharoenwitthaya, P., Mills, P. R., Keach, J. C., Lafferty, H. D., Stahler, A., et al. (2015). Liver fibrosis, but no other histologic features, is associated with long-term outcomes of patients with nonalcoholic fatty liver disease. *Gastroenterology, 149*(2), 389–397.e10. doi:10.1053/j.gastro.2015.04.043

Arroyo-Fernández, A., Blanco-Fernández, M. A., & Lladó-Jordan, G. (2023). Psychological therapy in overweight and obesity treatment centers. Do the centers include it on their websites? *Journal of Advanced Pharmacy Education & Research, 13*(1), 158–161. doi:10.51847/TCqButfuej

Babaei, E., Shirvani, M., Salehi, L., & Gohari, M. (2023). Late-onset Stargardt disease; a clinical condition may be misdiagnosed: A case report. *Journal of Advanced Pharmacy Education & Research, 13*(2), 128–130. doi:10.51847/FDIEB0V59X

Badmus, O. O., Hinds, T. D., Jr., & Stec, D. E. (2023). Mechanisms linking metabolic-associated fatty liver disease (MAFLD) to cardiovascular disease. *Current Hypertension Reports, 25*(8), 151–162. doi:10.1007/s11906-023-01242-8

Bansal, M. B., Patton, H., Morgan, T. R., Carr, R. M., Dranoff, J. A., & Allen, A. M. (2025). Semaglutide therapy for metabolic dysfunction-associated steatohepatitis: November 2025 updates to AASLD Practice Guidance. *Hepatology*, *83*(5), 1326-1340. doi:10.1097/HEP.0000000000001608

Baratta, F., D'Erasmo, L., Bini, S., Pastori, D., Angelico, F., Del Ben, M., Arca, M., & Di Costanzo, A. (2022). Heterogeneity of non-alcoholic fatty liver disease (NAFLD): Implication for cardiovascular risk stratification. *Atherosclerosis, 357*, 51–59. doi:10.1016/j.atherosclerosis.2022.08.011

Baratta, F., D'Erasmo, L., Di Costanzo, A., Umbro, I., Pastori, D., Angelico, F., & Del Ben, M. (2022). Metabolic syndrome but not fatty liver-associated genetic variants correlates with glomerular renal function decline in patients with non-alcoholic fatty liver disease. *Biomedicines, 10*(3), 720. doi:10.3390/biomedicines10030720

Bharali, P., Anirvan, P., Gogoi, M., & Singh, S. P. (2021). Statins and cancer mortality in NAFLD: Is it too early to rejoice? *Journal of Clinical Gastroenterology, 55*(7), 638–639. doi:10.1097/MCG.0000000000001570

Bilson, J., Mantovani, A., Byrne, C. D., & Targher, G. (2024). Steatotic liver disease, MASLD and risk of chronic kidney disease. *Diabetes & Metabolism, 50*(1), 101506. doi:10.1016/j.diabet.2023.101506

Boeckmans, J., Gatzios, A., Schattenberg, J. M., Koek, G. H., Rodrigues, R. M., & Vanhaecke, T. (2023). PNPLA3 I148M and response to treatment for hepatic steatosis: A systematic review. *Liver International, 43*(5), 975–988. doi:10.1111/liv.15533

Bonsignore, M. R. (2022). Obesity and obstructive sleep apnea. *Handbook of Experimental Pharmacology, 274*, 181–201.

doi:10.1007/164_2021_558

Byrne, C. D., & Targher, G. (2022). Non-alcoholic fatty liver disease-related risk of cardiovascular disease and other cardiac complications. *Diabetes, Obesity and Metabolism, 24*(Suppl 2), 28–43. doi:10.1111/dom.14484

Carrieri, P., Mourad, A., Marcellin, F., Trylesinski, A., Calleja, J. L., Protopopescu, C., & Lazarus, J. V. (2022). Knowledge of liver fibrosis stage among adults with NAFLD/NASH improves adherence to lifestyle changes. *Liver International, 42*(5), 984–994. doi:10.1111/liv.15209

Chakraborty, P., & Rajasekar, A. (2024). Efficacy of linezolid-based hydrogel as local drug in stage II grade A periodontitis: A clinical study. *Annals of Dental Specialty, 12*(1), 1–6. doi:10.51847/BUnKMJeFfH

Cherubini, A., Casirati, E., Tomasi, M., & Valenti, L. (2021). PNPLA3 as a therapeutic target for fatty liver disease: The evidence to date. *Expert Opinion on Therapeutic Targets, 25*(12), 1033–1043. doi:10.1080/14728222.2021.2018418

Cherubini, A., Ostadreza, M., Jamialahmadi, O., Pelusi, S., Rrapaj, E., Casirati, E., Passignani, G., Norouziesfahani, M., Sinopoli, E., Baselli, G., et al. (2023). Interaction between estrogen receptor-α and PNPLA3 p.I148M variant drives fatty liver disease susceptibility in women. *Nature Medicine, 29*(10), 2643–2655. doi:10.1038/s41591-023-02553-8

Chon, Y. E., Jin, Y. J., An, J., Kim, H. Y., Choi, M., Jun, D. W., Kim, M. N., Han, J. W., Lee, H. A., Yu, J. H., et al. (2024). Optimal cut-offs of vibration-controlled transient elastography and magnetic resonance elastography in diagnosing advanced liver fibrosis in patients with nonalcoholic fatty liver disease: A systematic review and meta-analysis. *Clinical and Molecular Hepatology, 30*(Suppl), S117–S133. doi:10.3350/cmh.2024.0392

Cusi, K., Abdelmalek, M. F., Apovian, C. M., Balapattabi, K., Bannuru, R. R., Barb, D., Bardsley, J. K., Beverly, E. A., Corbin, K. D., ElSayed, N. A., et al. (2025). Metabolic dysfunction-associated steatotic liver disease (MASLD) in people with diabetes: The need for screening and early intervention. A consensus report of the American Diabetes Association. *Diabetes Care, 48*(7), 1057–1082. doi:10.2337/dci24-0094

Danpanichkul, P., Suparan, K., Prasitsumrit, V., Ahmed, A., Wijarnpreecha, K., & Kim, D. (2025). Long-term outcomes and risk modifiers of metabolic dysfunction-associated steatotic liver disease between lean and non-lean populations. *Clinical and Molecular Hepatology, 31*(1), 74–89. doi:10.3350/cmh.2024.0631

Das, S., Agarwal, K., Kapoor, N., Lakhani, O. J., & Das Gupta, A. (2025). Emerging concepts in the diagnosis and management of metabolically associated steatotic liver disease. *Current Opinion in Endocrinology, Diabetes and Obesity, 32*(6), 269–278. doi:10.1097/MED.0000000000000935

Djeagou, A., Gunukula, K., Sermani, A., & Vaspari, S. K. (2025). Emerging perspectives in the diagnosis and management of metabolic dysfunction-associated steatotic liver disease (MASLD): A narrative review. *Cureus, 17*(10), e95288. doi:10.7759/cureus.95288

Doddapanen, N., Lakshmegowda, Y. K., Aardhya, S., Rajashekar, R., Doolgindachbaporn, T., & Nagaraju, P. (2024). Environmental education, awareness and environmental ethics among pre-university students of Mysuru city, Karnataka, India. *World Journal of Environmental Biosciences, 13*(2), 13–20. doi:10.51847/nBbI6XJU0H

Dulai, P. S., Singh, S., Patel, J., Soni, M., Prokop, L. J., Younossi, Z., Sebastiani, G., Ekstedt, M., Hagström, H., Nasr, P., et al. (2017). Increased risk of mortality by fibrosis stage in nonalcoholic fatty liver disease: Systematic review and meta-analysis. *Hepatology, 65*(5), 1557–1565. doi:10.1002/hep.29085

Elmustafa, F., Tyagi, M., Kaur, H., Blaney, H., Xie, C., Jalil, S., & Singal, A. K. (2025). Met-ALD and ALD- Does differentiating these impact management and clinical outcomes? *Current Hepatology Reports, 24*(1). doi:10.1007/s11901-025-00712-8

En Li Cho, E., Ang, C. Z., Quek, J., Fu, C. E., Lim, L. K. E., Heng, Z. E. Q., Tan, D. J. H., Lim, W. H., Yong, J. N., Zeng, R., et al. (2023). Global prevalence of non-alcoholic fatty liver disease in type 2 diabetes mellitus: An updated systematic review and meta-analysis. *Gut, 72*(11), 2138–2148. doi:10.1136/gutjnl-2023-330110

Eslam, M., El-Serag, H. B., Francque, S., Sarin, S. K., Wei, L., Bugianesi, E., & George, J. (2022). Metabolic (dysfunction)-associated fatty liver disease in individuals of normal weight. *Nature Reviews Gastroenterology & Hepatology, 19*(10), 638–651. doi:10.1038/s41575-022-00635-5

Eslam, M., Newsome, P. N., Sarin, S. K., Anstee, Q. M., Targher, G., Romero-Gomez, M., Zelber-Sagi, S., Wai-Sun Wong, V., Dufour, J. F., Schattenberg, J. M., et al. (2020). A new definition for metabolic dysfunction-associated fatty liver disease: An international expert consensus statement. *Journal of Hepatology, 73*(1), 202–209. doi:10.1016/j.jhep.2020.03.039

Estes, C., Anstee, Q. M., Arias-Loste, M. T., Bantel, H., Bellentani, S., Caballeria, J., Colombo, M., Craxi, A., Crespo, J., Day, C. P., et al. (2018). Modeling NAFLD disease burden in China, France, Germany, Italy, Japan, Spain, United Kingdom, and United States for the period 2016–2030. *Journal of Hepatology, 69*(4), 896–904. doi:10.1016/j.jhep.2018.05.036

European Association for the Study of the Liver, European Association for the Study of Diabetes, & European Association for the Study of Obesity. (2024). EASL-EASD-EASO clinical practice guidelines on the management of metabolic dysfunction-associated steatotic liver disease (MASLD). *Journal of Hepatology, 81*(3), 492–542. doi:10.1016/j.jhep.2024.04.031

Falzarano, C., Lofton, T., Osei-Ntansah, A., Oliver, T., Southward, T., Stewart, S., & Andrisse, S. (2022). Nonalcoholic fatty liver disease in women and girls with polycystic ovary syndrome. *The Journal of Clinical Endocrinology & Metabolism, 107*(1), 258–272. doi:10.1210/clinem/dgab658

Fang, L., Li, J., Zeng, H., & Liu, J. (2024). Effects of GLP-1 receptor agonists on the degree of liver fibrosis and CRP in non-alcoholic fatty liver disease and non-alcoholic

steatohepatitis: A systematic review and meta-analysis. *Primary Care Diabetes, 18*(3), 268–276. doi:10.1016/j.pcd.2024.03.005

Ferguson, D., & Finck, B. N. (2021). Emerging therapeutic approaches for the treatment of NAFLD and type 2 diabetes mellitus. *Nature Reviews Endocrinology, 17*(8), 484–495. doi:10.1038/s41574-021-00507-z

Ganea, M., Marcu, O. A., & Csep, A. N. (2024). Finite element analysis of trabecular bone response to orthodontic forces in periodontal breakdown. *Asian Journal of Periodontics & Orthodontics, 4*, 121–138. doi:10.51847/DVdnjkDpYE

Gao, X., Chen, T., Zhou, F., Sun, Y., Zhang, J., Li, X., Zhao, W., Li, Y., Shi, Y., Niu, K., et al. (2025). The association between different insulin resistance surrogates and all-cause mortality and cardiovascular mortality in patients with metabolic dysfunction-associated steatotic liver disease. *Cardiovascular Diabetology, 24*(1), 200. doi:10.1186/s12933-025-02758-w

Genua, I., Iruzubieta, P., Rodríguez-Duque, J. C., Pérez, A., & Crespo, J. (2023). NAFLD and type 2 diabetes: A practical guide for the joint management. *Gastroenterología y Hepatología, 46*(10), 815–825. doi:10.1016/j.gastrohep.2022.12.002

Goedecke, J. H., Nguyen, K. A., Kufe, C., Masemola, M., Chikowore, T., Mendham, A. E., Norris, S. A., Crowther, N. J., Karpe, F., Olsson, T., et al. (2022). Waist circumference thresholds predicting incident dysglycaemia and type 2 diabetes in Black African men and women. *Diabetes, Obesity and Metabolism, 24*(5), 918–927. doi:10.1111/dom.14655

Grander, C., Grabherr, F., & Tilg, H. (2023). Non-alcoholic fatty liver disease: Pathophysiological concepts and treatment options. *Cardiovascular Research, 119*(9), 1787–1798. doi:10.1093/cvr/cvad095

Guo, X., Yin, X., Liu, Z., & Wang, J. (2022). Non-alcoholic fatty liver disease (NAFLD) pathogenesis and natural products for prevention and treatment. *International Journal of Molecular Sciences, 23*(24), 15489. doi:10.3390/ijms232415489

Guo, Z., Wu, D., Mao, R., Yao, Z., Wu, Q., & Lv, W. (2025). Global burden of MAFLD, MAFLD-related cirrhosis and MASH-related liver cancer from 1990 to 2021. *Scientific Reports, 15*(1), 7083. doi:10.1038/s41598-025-91312-5

Hagström, H., Shang, Y., Hegmar, H., & Nasr, P. (2024). Natural history and progression of metabolic dysfunction-associated steatotic liver disease. *The Lancet Gastroenterology & Hepatology, 9*(10), 944–956. doi:10.1016/S2468-1253(24)00193-6

Han, S. K., Baik, S. K., & Kim, M. Y. (2023). Non-alcoholic fatty liver disease: Definition and subtypes. *Clinical and Molecular Hepatology, 29*(Suppl), S5–S16. doi:10.3350/cmh.2022.0424

Hima, N., Benarous, D., Louail, B., & Hamadi, W. (2024). The effect of social media on purchasing behaviour through e-marketing: Empirical study for Algerian university students. *Journal of Organizational Behavior Research, 9*(2), 73–86. doi:10.51847/LSsHBTTTHg

Horn, P., & Tacke, F. (2024). Metabolic reprogramming in liver fibrosis. *Cell Metabolism, 36*(7), 1439–1455. doi:10.1016/j.cmet.2024.05.003

Houttu, V., Boulund, U., Troelstra, M., Csader, S., Stols-Gonçalves, D., Mak, A. L., Dijk, A. V., Bouts, J., Winkelmeijer, M., Verdoes, X., et al. (2024). Deep phenotyping of patients with MASLD upon high-intensity interval training. *JHEP Reports, 7*(3), 101289. doi:10.1016/j.jhepr.2024.101289

Hsueh, R. C., Wu, W. J., Lin, C. L., Liu, C. J., Huang, Y. W., Hu, J. T., Wu, C. F., Sung, F. Y., Liu, W. J., & Yu, M. W. (2022). Impact of PNPLA3 p.I148M and hepatic steatosis on long-term outcomes for hepatocellular carcinoma and HBsAg seroclearance in chronic hepatitis B. *Journal of Hepatocellular Carcinoma, 9*, 301–313. doi:10.2147/JHC.S355540

Huang, D. Q., Wilson, L. A., Behling, C., Kleiner, D. E., Kowdley, K. V., Dasarathy, S., Amangurbanova, M., Terrault, N. A., Diehl, A. M., Chalasani, N., et al. (2023). Fibrosis progression rate in biopsy-proven nonalcoholic fatty liver disease among people with diabetes versus people without diabetes: A multicenter study. *Gastroenterology, 165*(2), 463–472.e5. doi:10.1053/j.gastro.2023.04.025

Israelsen, M., Francque, S., Tsochatzis, E. A., & Krag, A. (2024). Steatotic liver disease. *The Lancet, 404*(10464), 1761–1778. doi:10.1016/S0140-6736(24)01811-7

Ji, Y., Liang, Y., Mak, J. C. W., & Ip, M. S. M. (2022). Obstructive sleep apnea, intermittent hypoxia, and non-alcoholic fatty liver disease. *Sleep Medicine, 95*, 16–28. doi:10.1016/j.sleep.2022.04.006

Joungtrakul, J., & Smith, I. D. (2024). Examining the effect of organizational justice on organizational citizenship behavior through the mediation of job commitment. *Journal of Organizational Behavior Research, 9*(2), 105–113. doi:10.51847/PfxUhbw9xY

Kang, Y. W., Baek, Y. H., & Moon, S. Y. (2024). Sequential diagnostic approach using FIB-4 and ELF for predicting advanced fibrosis in metabolic dysfunction-associated steatotic liver disease. *Diagnostics, 14*(22), 2517. doi:10.3390/diagnostics14222517

Kanwal, F., Neuschwander-Tetri, B. A., Loomba, R., & Rinella, M. E. (2024). Metabolic dysfunction-associated steatotic liver disease: Update and impact of new nomenclature on the American Association for the Study of Liver Diseases practice guidance on nonalcoholic fatty liver disease. *Hepatology, 79*(5), 1212–1219. doi:10.1097/HEP.0000000000000670

Kasper, P., Martin, A., Lang, S., Kütting, F., Goeser, T., Demir, M., & Steffen, H. M. (2021). NAFLD and cardiovascular diseases: A clinical review. *Clinical Research in Cardiology, 110*(7), 921–937. doi:10.1007/s00392-020-01709-7

Kido, H., Mizukoshi, E., Yanagi, M., Shihui, L., Seike, T., Nakagawa, H., Yamashima, T., Shiraishi, Y., Ozaki, N., Harada, K., et al. (2025). Abnormalities of intracellular organelles in metabolic dysfunction-associated steatotic disease. *Journal of Gastroenterology, 60*(8), 990–999. doi:10.1007/s00535-025-02257-5

Kjaergaard, M., Lindvig, K. P., Thorhauge, K. H., Andersen, P.,

Hansen, J. K., Kastrup, N., Jensen, J. M., Hansen, C. D., Johansen, S., Israelsen, M., et al. (2023). Using the ELF test, FIB-4 and NAFLD fibrosis score to screen the population for liver disease. *Journal of Hepatology, 79*(2), 277–286. doi:10.1016/j.jhep.2023.04.002

Koh, J. H., Wang, M., Suzuki, H., Muthiah, M., Ng, C. H., & Huang, D. Q. (2024). NAFLD and NAFLD-related HCC in Asia: Burden and surveillance. *Journal of Clinical and Experimental Hepatology, 14*(1), 101213. doi:10.1016/j.jceh.2023.06.013

Kusumawardani, L. A., Andrajati, R., Lathifatuzahra, F., Yusuf, R. A., Azzahrini, F. L., & Ardhianti, I. F. (2023). Prescribing and patient care indicators for drug use evaluation at primary healthcare centers in Indonesia. *Journal of Advanced Pharmacy Education & Research, 13*(2), 53–58. doi:10.51847/DkT6Fry65G

Lazarus, J. V., Newsome, P. N., Francque, S. M., Kanwal, F., Terrault, N. A., & Rinella, M. E. (2024). Reply: A multi-society Delphi consensus statement on new fatty liver disease nomenclature. *Hepatology, 79*(3), E93–E94. doi:10.1097/HEP.0000000000000696

Le, M. H., Le, D. M., Baez, T. C., Dang, H., Nguyen, V. H., Lee, K., Stave, C. D., Ito, T., Wu, Y., Yeo, Y., et al. (2024). Global incidence of adverse clinical events in non-alcoholic fatty liver disease: A systematic review and meta-analysis. *Clinical and Molecular Hepatology, 30*(2), 235–246. doi:10.3350/cmh.2023.0485

Lee, C. H., Lui, D. T., & Lam, K. S. (2022). Non-alcoholic fatty liver disease and type 2 diabetes: An update. *Journal of Diabetes Investigation, 13*(6), 930–940. doi:10.1111/jdi.13756

Lee, H. H., Lee, H. A., Kim, E. J., Kim, H. Y., Kim, H. C., Ahn, S. H., Lee, H., & Kim, S. U. (2024). Metabolic dysfunction-associated steatotic liver disease and risk of cardiovascular disease. *Gut, 73*(3), 533–540. doi:10.1136/gutjnl-2023-331003

Lekakis, V., & Papatheodoridis, G. V. (2024). Natural history of metabolic dysfunction-associated steatotic liver disease. *European Journal of Internal Medicine, 122*, 3–10. doi:10.1016/j.ejim.2023.11.005

Li, Y., Yang, P., Ye, J., Xu, Q., Wu, J., & Wang, Y. (2024). Updated mechanisms of MASLD pathogenesis. *Lipids in Health and Disease, 23*(1), 117. doi:10.1186/s12944-024-02108-x

Lim, T. S., Kwon, S., Bae, S. A., Chon, H. Y., Jang, S. A., Kim, J. K., Kim, C. S., Park, S. W., & Kim, K. M. (2025). Association between handgrip strength and cardiovascular disease risk in MASLD: A prospective study from UK Biobank. *Journal of Cachexia, Sarcopenia and Muscle, 16*(2), e13757. doi:10.1002/jcsm.13757

Lin, P. C., & Mukai, T. (2024). Regenerative periodontal therapy for vital teeth with extensive attachment loss at the root apex: Two cases with 5-year follow-up. *Asian Journal of Periodontics & Orthodontics, 4*, 88–97. doi:10.51847/fWQQVl1I8j

Maevskaya, M. V., Kotovskaya, Y. V., Ivashkin, V. T., Tkacheva, O. N., Troshina, E. A., Shestakova, M. V., Breder, V. V., Geyvandova, N. I., Doshchitsin, V. L., Dudinskaya, E. N., et al. (2022). The national consensus statement on the management of adult patients with non-alcoholic fatty liver disease and main comorbidities. *Terapevticheskii Arkhiv, 94*(2), 216–253. doi:10.26442/00403660.2022.02.201363

Mantovani, A., Csermely, A., Bilson, J., Borella, N., Enrico, S., Pecoraro, B., Shtembari, E., Morandin, R., Polyzos, S. A., Valenti, L., Tilg, H., Byrne, C. D., & Targher, G. (2024). Association between primary hypothyroidism and metabolic dysfunction-associated steatotic liver disease: An updated meta-analysis. *Gut, 73*(9), 1554–1561. doi:10.1136/gutjnl-2024-332491

Miller, D. M., McCauley, K. F., & Dunham-Snary, K. J. (2025). Metabolic dysfunction-associated steatotic liver disease (MASLD): Mechanisms, clinical implications and therapeutic advances. *Endocrinology, Diabetes & Metabolism, 8*(6), e70132. doi:10.1002/edm2.70132

Miranda, L. G., Amigo, T. R., & Ortiz, H. A. D. L. B. (2024). Validating an objective structured clinical examination to enhance assessment of clinical skills in physical therapy students. *Journal of Advanced Pharmacy Education & Research, 14*(2), 16–26. doi:10.51847/c2DlK9b9pQ

Mislishayeva, M. S., Saiyan, L. A., Frolova, P. S., Grinenko, E. V., Abdurakhmanova, I. M., & Chemazokova, M. Z. (2023). Basic principles of pharmacotherapy of diseases of the visual organs. *Journal of Advanced Pharmacy Education & Research, 13*(2), 24–28. doi:10.51847/44gUM9g3fF

Mózes, F. E., Lee, J. A., Selvaraj, E. A., Jayaswal, A. N. A., Trauner, M., Boursier, J., Fournier, C., Staufer, K., Stauber, R. E., Bugianesi, E., et al. (2022). Diagnostic accuracy of non-invasive tests for advanced fibrosis in patients with NAFLD: An individual patient data meta-analysis. *Gut, 71*(5), 1006–1019. doi:10.1136/gutjnl-2021-324243

Njei, B., Ameyaw, P., Al-Ajlouni, Y., Njei, L. P., & Boateng, S. (2024). Diagnosis and management of lean metabolic dysfunction-associated steatotic liver disease (MASLD): A systematic review. *Cureus, 16*(10), e71451. doi:10.7759/cureus.71451

Nobili, V., Alisi, A., Valenti, L., Miele, L., Feldstein, A. E., & Alkhouri, N. (2019). NAFLD in children: New genes, new diagnostic modalities and new drugs. *Nature Reviews Gastroenterology & Hepatology, 16*(9), 517–530. doi:10.1038/s41575-019-0169-z

Noureddin, M., & Wong, V. W. (2023). A revisit of the natural history of nonalcoholic fatty liver disease. *Clinical Gastroenterology and Hepatology, 21*(5), 1152–1153. doi:10.1016/j.cgh.2022.08.019

Nysather, J., Kaya, E., Manka, P., Gudsoorkar, P., & Syn, W. K. (2023). Nonalcoholic fatty liver disease and chronic kidney disease cross talk. *Advances in Kidney Disease and Health, 30*(4), 315–335. doi:10.1053/j.akdh.2023.04.001

Osonoi, S., & Takebe, T. (2024). Organoid-guided precision hepatology for metabolic liver disease. *Journal of Hepatology, 80*(5), 805–821. doi:10.1016/j.jhep.2024.01.002

Parola, M., & Pinzani, M. (2024). Liver fibrosis in NAFLD/NASH: From pathophysiology towards diagnostic and therapeutic strategies. *Molecular Aspects of Medicine, 95*, 101231. doi:10.1016/j.mam.2023.101231

Paternostro, R., & Trauner, M. (2022). Current treatment of non-alcoholic fatty liver disease. *Journal of Internal Medicine, 292*(2), 190–204. doi:10.1111/joim.13531

Portincasa, P., Khalil, M., Mahdi, L., Perniola, V., Idone, V., Graziani, A., Baffy, G., & Di Ciaula, A. (2024). Metabolic dysfunction-associated steatotic liver disease: From pathogenesis to current therapeutic options. *International Journal of Molecular Sciences, 25*(11), 5640. doi:10.3390/ijms25115640

Pouwels, S., Sakran, N., Graham, Y., Leal, A., Pintar, T., Yang, W., Kassir, R., Singhal, R., Mahawar, K., & Ramnarain, D. (2022). Non-alcoholic fatty liver disease (NAFLD): A review of pathophysiology, clinical management and effects of weight loss. *BMC Endocrine Disorders, 22*(1), 63. doi:10.1186/s12902-022-00980-1

Powell, E. E., Wong, V. W., & Rinella, M. (2021). Non-alcoholic fatty liver disease. *The Lancet, 397*(10290), 2212–2224. doi:10.1016/S0140-6736(20)32511-3

Prada, A. M., Cicalău, G. I. P., & Ciavoi, G. (2024). Resin infiltration for white-spot lesion management after orthodontic treatment. *Asian Journal of Periodontics & Orthodontics, 4*, 19–23. doi:10.51847/ZTuGEanCSV

Prikhodko, V. A., Bezborodkina, N. N., & Okovityi, S. V. (2022). Pharmacotherapy for non-alcoholic fatty liver disease: Emerging targets and drug candidates. *Biomedicines, 10*(2), 274. doi:10.3390/biomedicines10020274

Quek, J., Chan, K. E., Wong, Z. Y., Tan, C., Tan, B., Lim, W. H., Tan, D. J. H., Tang, A. S. P., Tay, P., Xiao, J., et al. (2023). Global prevalence of non-alcoholic fatty liver disease and non-alcoholic steatohepatitis in the overweight and obese population: A systematic review and meta-analysis. *The Lancet Gastroenterology & Hepatology, 8*(1), 20–30. doi:10.1016/S2468-1253(22)00317-X

Ramandi, A., Diehl, A. M., Sanyal, A. J., & de Jong, Y. P. (2025). Experimental models to investigate PNPLA3 in liver steatosis. *Liver International, 45*(5), e70091. doi:10.1111/liv.70091

Riazi, K., Azhari, H., Charette, J. H., Underwood, F. E., King, J. A., Afshar, E. E., Swain, M. G., Congly, S. E., Kaplan, G. G., & Shaheen, A. A. (2022). The prevalence and incidence of NAFLD worldwide: A systematic review and meta-analysis. *The Lancet Gastroenterology & Hepatology, 7*(9), 851–861. doi:10.1016/S2468-1253(22)00165-0

Rinella, M. E., Lazarus, J. V., Ratziu, V., Francque, S. M., Sanyal, A. J., Kanwal, F., Romero, D., Abdelmalek, M. F., Anstee, Q. M., Arab, J. P., et al. (2023). A multisociety Delphi consensus statement on new fatty liver disease nomenclature. *Journal of Hepatology, 79*(6), 1542–1556. doi:10.1016/j.jhep.2023.06.003

Ross, R., Neeland, I. J., Yamashita, S., Shai, I., Seidell, J., Magni, P., Santos, R. D., Arsenault, B., Cuevas, A., Hu, F. B., et al. (2020). Waist circumference as a vital sign in clinical practice: A consensus statement from the IAS and ICCR Working Group on visceral obesity. *Nature Reviews Endocrinology, 16*(3), 177–189. doi:10.1038/s41574-019-0310-7

Sanyal, A. J., Castéra, L., & Wong, V. W. (2023). Noninvasive assessment of liver fibrosis in NAFLD. *Clinical Gastroenterology and Hepatology, 21*(8), 2026–2039. doi:10.1016/j.cgh.2023.03.042

Sanyal, A. J., Newsome, P. N., Kliers, I., Østergaard, L. H., Long, M. T., Kjær, M. S., Cali, A. M. G., Bugianesi, E., Rinella, M. E., Roden, M., et al. (2025). Phase 3 trial of semaglutide in metabolic dysfunction-associated steatohepatitis. *The New England Journal of Medicine, 392*(21), 2089–2099. doi:10.1056/NEJMoa2413258

Sanyal, A. J., Van Natta, M. L., Clark, J., Neuschwander-Tetri, B. A., Diehl, A., Dasarathy, S., Loomba, R., Chalasani, N., Kowdley, K., Hameed, B., et al. (2021). Prospective study of outcomes in adults with nonalcoholic fatty liver disease. *The New England Journal of Medicine, 385*(17), 1559–1569. doi:10.1056/NEJMoa2029349

Sarkar, S., Kamle, M., Bharti, A., & Kumar, P. (2023). Antibiotic-resistant bacteria risks and challenges for human health and environment: An overview. *World Journal of Environmental Biosciences, 12*(2), 26–34. doi:10.51847/qDfN29z7ps

SeKo, Y., Lin, H., Wong, V. W., & Okanoue, T. (2025). Impact of PNPLA3 in lean individuals and in cryptogenic steatotic liver disease. *Liver International, 45*(4), e16164. doi:10.1111/liv.16164

Shaheen, M., Schrode, K. M., Pan, D., Kermah, D., Puri, V., Zarrinpar, A., Elisha, D., Najjar, S. M., & Friedman, T. C. (2021). Sex-specific differences in the association between race/ethnicity and NAFLD among the US population. *Frontiers in Medicine, 8*, 795421. doi:10.3389/fmed.2021.795421

Shaji, S., Gowda, B., Gurusiddappa, L. H., Veeresh, S. J., Kalikeri, S., & Bellari, K. (2024). Navigating the hazards: A review of pesticides and their effects on human well-being. *World Journal of Environmental Biosciences, 13*(2), 21–30. doi:10.51847/yl4o018DzR

Silva Júnior, W. S., Sposito, A. C., & Godoy-Matos, A. (2025). Should the new EASL-EASD-EASO clinical practice guidelines on MASLD recommend pioglitazone as a MASH-targeted pharmacotherapy? *Journal of Hepatology, 82*(1), e21–e22. doi:10.1016/j.jhep.2024.06.034

Son, S., & Lee, E. (2024). Studying the impact of non-surgical periodontal treatment on glycosylated hemoglobin level in non-diabetic people. *Annals of Dental Specialty, 12*(3), 1–6. doi:10.51847/UevUGpXDMF

Stefan, N., Yki-Järvinen, H., & Neuschwander-Tetri, B. A. (2025). Metabolic dysfunction-associated steatotic liver disease: Heterogeneous pathomechanisms and effectiveness of metabolism-based treatment. *The Lancet Diabetes & Endocrinology, 13*(2), 134–148. doi:10.1016/S2213-8587(24)00318-8

Targher, G., Byrne, C. D., & Tilg, H. (2024). MASLD: A systemic metabolic disorder with cardiovascular and malignant complications. *Gut, 73*(4), 691–702. doi:10.1136/gutjnl-2023-330595

Teng, M. L., Ng, C. H., Huang, D. Q., Chan, K. E., Tan, D. J., Lim, W. H., Yang, J. D., Tan, E., & Muthiah, M. D. (2023). Global incidence and prevalence of nonalcoholic fatty liver disease. *Clinical and Molecular Hepatology, 29*(Suppl), S32–S42. doi:10.3350/cmh.2022.0365

Triantafyllopoulos, G., Kavvadia, K., & Vrettou, C. (2023). Non-surgical periodontal treatment and its effects on patients' oral health-related quality of life: A pilot case series. *Asian Journal of Periodontics & Orthodontics, 3*, 87–95. doi:10.51847/yXbTop3W9w

Varoneckaitė, M., Jasinskaitė, K., Varoneckas, A., Vasiliauskas, A., & Leketas, M. (2024). Comparing root resorption in fixed vs. clear aligner orthodontics: A radiographic study. *Asian Journal of Periodontics & Orthodontics, 4*, 34–41. doi:10.51847/fl7oRw6Djo

Wang, D. (2024). An overview on the marine benthic dinoflagellates effects on human and environment. *World Journal of Environmental Biosciences, 13*(4), 7–13. doi:10.51847/QwZpooC6ZV

Wang, D., Day, E. A., Townsend, L. K., Djordjevic, D., Jørgensen, S. B., & Steinberg, G. R. (2021). GDF15: Emerging biology and therapeutic applications for obesity and cardiometabolic disease. *Nature Reviews Endocrinology, 17*(10), 592–607. doi:10.1038/s41574-021-00529-7

Wang, D., Miao, J., Zhang, L., & Zhang, L. (2025). Research advances in the diagnosis and treatment of MASLD/MASH. *Annals of Medicine, 57*(1), 2445780. doi:10.1080/07853890.2024.2445780

Wang, X., Bian, L., Feng, Z., O'Shaughnessy, K., Kwong, A. C., Ha, E. H., Wang, L., Chen, W., & Wu, X. (2026). A PNPLA3-NACC1-RIPK3 pathway mediates macrophage necroptosis and inflammation in MASLD. *Hepatology Communications, 10*(2), e0890. doi:10.1097/HC9.0000000000000890

Wong, V. W., Ekstedt, M., Wong, G. L., & Hagström, H. (2023). Changing epidemiology, global trends and implications for outcomes of NAFLD. *Journal of Hepatology, 79*(3), 842–852. doi:10.1016/j.jhep.2023.04.036

Yang, M., Geng, C. A., Liu, X., & Guan, M. (2021). Lipid disorders in NAFLD and chronic kidney disease. *Biomedicines, 9*(10), 1405. doi:10.3390/biomedicines9101405

Younossi, Z. M., Golabi, P., Paik, J. M., Henry, A., Van Dongen, C., & Henry, L. (2023). The global epidemiology of nonalcoholic fatty liver disease (NAFLD) and nonalcoholic steatohepatitis (NASH): A systematic review. *Hepatology, 77*(4), 1335–1347. doi:10.1097/HEP.0000000000000004

Younossi, Z. M., Kalligeros, M., & Henry, L. (2025). Epidemiology of metabolic dysfunction-associated steatotic liver disease. *Clinical and Molecular Hepatology, 31*(Suppl), S32–S50. doi:10.3350/cmh.2024.0431

Younossi, Z. M., Koenig, A. B., Abdelatif, D., Fazel, Y., Henry, L., & Wymer, M. (2016). Global epidemiology of nonalcoholic fatty liver disease: Meta-analytic assessment of prevalence, incidence, and outcomes. *Hepatology, 64*(1), 73–84. doi:10.1002/hep.28431

Younossi, Z., Anstee, Q. M., Marietti, M., Hardy, T., Henry, L., Eslam, M., George, J., & Bugianesi, E. (2018). Global burden of NAFLD and NASH: Trends, predictions, risk factors and prevention. *Nature Reviews Gastroenterology & Hepatology, 15*(1), 11–20. doi:10.1038/nrgastro.2017.109

Zeng, J., Fan, J. G., & Francque, S. M. (2024). Therapeutic management of metabolic dysfunction associated with steatotic liver disease. *United European Gastroenterology Journal, 12*(2), 177–186. doi:10.1002/ueg2.12525

Zhang, H., Chen, Q. F., Lip, G. Y. H., Tilg, H., Valenti, L., Somers, V. K., Byrne, C. D., Targher, G., Yang, W., Mantzoros, C. S., et al. (2026). Burden of metabolic diseases, 1990–2023, with forecasts to 2030 for the Asia-Pacific region. *Metabolism, 179*, 156575. doi:10.1016/j.metabol.2026.156575

Zheng, H., Sechi, L. A., Navarese, E. P., Casu, G., & Vidili, G. (2024). Metabolic dysfunction-associated steatotic liver disease and cardiovascular risk: A comprehensive review. *Cardiovascular Diabetology, 23*(1), 346. doi:10.1186/s12933-024-02434-5

Zheng, H., Wu, K., Zheng, H., Chen, G., Lan, Y., Chen, S., Casu, G., Sechi, L. A., Wu, S., Vidili, G., et al. (2025). High systemic inflammation response index and increased cardiovascular risk and mortality in MASLD: A prospective cohort study. *JHEP Reports, 7*(12), 101602. doi:10.1016/j.jhepr.2025.101602

Zhou, X. D., Chen, Q. F., Fan, Q. Y., Kim, S. U., Yip, T. C., Petta, S., Nakajima, A., Tsochatzis, E., Boursier, J., Bugianesi, E., et al. (2025). Cardiovascular-kidney-metabolic syndrome and the risk of liver fibrosis progression and liver-related events in MASLD. *Hepatology*. Advance online publication. doi:10.1097/HEP.0000000000001645

Zhou, X. D., Lian, L. Y., Chen, Q. F., Kim, S. U., Cheuk-Fung Yip, T., Petta, S., Nakajima, A., Tsochatzis, E., Shi, J., Chan, E. K., et al., (2026). Effect of hypertension on long-term adverse clinical outcomes and liver fibrosis progression in MASLD. *Journal of Hepatology, 84*(2), 254–265. doi:10.1016/j.jhep.2025.08.017

Zigmantavičius, J., Kilinskaitė, G., & Leketas, M. (2023). Dimensional changes of buccal bone after immediate implantation using different grafting materials: A systematic review. *Annals of Dental Specialty, 11*(1), 7–15. doi:10.51847/vykVdVPn8e

Zisis, M., Chondrogianni, M. E., Androutsakos, T., Rantos, I., Oikonomou, E., Chatzigeorgiou, A., & Kassi, E. (2025). Linking cardiovascular disease and metabolic dysfunction-associated steatotic liver disease (MASLD): The role of cardiometabolic drugs in MASLD treatment. *Biomolecules, 15*(3), 324. doi:10.3390/biom15030324

Zoncapè, M., Liguori, A., & Tsochatzis, E. A. (2024). Non-invasive testing and risk-stratification in patients with MASLD. *European Journal of Internal Medicine, 122*, 11–19. doi:10.1016/j.ejim.2024.01.013