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Modern Diets, Metabolic Inequity, and Race–Ethnic Disparities: Unraveling the Associations With MASLD and Cancer Risk

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ABSTRACT

Metabolic dysfunction–associated steatotic liver disease (MASLD) is quickly emerging as a global public health concern, primarily fueled by the rising consumption of processed and ultraprocessed foods. This global health issue, showing a 50% increase in prevalence over two decades to 38% of the global population, is now the second most common cause of end-stage liver disease and liver transplants in Europe and the United States and an emerging driver of hepatocellular carcinoma, particularly in Hispanic and non-Hispanic White populations. Its prevalence, driven by increasing consumption of ultraprocessed foods, obesity, and sedentary lifestyles, disproportionately impacts racial and ethnic minorities, exacerbating health disparities. Genetic polymorphisms contribute to interindividual and interethnic variations in disease susceptibility. However, genetics alone cannot explain the disparities; social determinants, food insecurity, and limited access to healthcare also play pivotal roles. MASLD prevalence is rising fastest among older adults and Hispanic women, particularly in low-income and rural communities. Despite advances in pharmacologic therapies, access remains inequitable. Lifestyle interventions remain essential. Integrating genetic and epigenetic insights into risk stratification and treatment can support precision medicine approaches. A comprehensive MASLD management framework must include policy reforms to address food deserts, healthcare access, and socioeconomic inequities. Culturally tailored public health programs and personalized care models are crucial for enhancing outcomes in vulnerable populations. In this narrative review, we examine the multifaceted contributors to MASLD pathogenesis and disparities, with a focus on genetic risk, social determinants, dietary patterns, and cancer risk and policy-level interventions to inform more equitable liver health strategies.

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Key Points

- Metabolic dysfunction–associated steatotic liver disease (MASLD) prevalence has risen by 50% globally in two decades, now affecting 38% of the world's population, and is the second leading cause of end-stage liver disease and liver transplants.
- Racial and ethnic disparities exist; non-Hispanic White and Hispanic individuals, especially women, show the highest increases, while non-Hispanic Black individuals have lower prevalence but worse outcomes.
- Genetic factors, such as the PNPLA3 I148M polymorphism, are more prevalent in Hispanic populations than in non-Hispanic Whites and non-Hispanic Blacks.
- Socioeconomic factors, including food insecurity, are linked to higher rates of MASLD and advanced liver fibrosis.
- Effective management requires dietary change, equitable access, and targeted policy reform.
- Integrating genetics and public health approaches can enhance the prevention and care of MASLD.

1 | Background

The rising incidence of metabolic dysfunction–associated steatotic liver disease (MASLD) should be alarming [1], as it represents a significant public health emergency, driven in part by the massive rise in human consumption of processed and ultraprocessed foods (UPFs) [2]. Diets high in UPF have been linked to obesity, diabetes, and hyperlipidemia. The change to MASLD from nonalcoholic fatty liver disease reflects the large body of literature supporting the role of metabolic dysfunction in the development of chronic liver disease. Given the increasing occurrence of MASLD, there is an urgent need for effective interventions to mitigate its complications, including progression to cirrhosis, liver failure, and hepatocellular carcinoma (HCC) [3].

Disparities in MASLD across racial and ethnic groups are exacerbated by genetic susceptibilities and socioeconomic inequities that promote nutrition insecurity and associated high intake of processed and UPF [2]. For example, the prevalence of metabolic-associated steatohepatitis (MASH), a severe form of MASLD, varies significantly by ethnicity. MASH has quickly risen as a leading cause of chronic liver disease and may progress the way for cirrhosis, liver failure, and HCC [4, 5]. A recent meta-analysis of 92 studies estimated that the prevalence of MASLD is 38% of the global population, representing a 50% increase compared to two decades ago [3]. MASLD is the second most common cause of end-stage liver disease and liver transplants in both Europe and the United States [5]. The rise in MASLD prevalence was observed across all age groups, but was most pronounced among those aged 65 and older [6]. Among racial and ethnic populations, non-Hispanic White individuals experienced the greatest increase, followed by Hispanic individuals. The trend was especially notable among Hispanic women, with non-Hispanic

White women showing the subsequent highest increase [3]. Geographic disparities were also evident, with rural populations experiencing the most significant growth compared to those in nonmetropolitan regions [3].

Variations in MASLD prevalence across racial and ethnic groups could be influenced by a combination of factors, including genetic susceptibility, socioeconomic factors, lifestyle behaviors, and dietary habits, particularly among minority populations [7, 8]. Addressing MASLD demands an integrative approach that spans public health policies, genetic research, and tailored community-based interventions to optimize screening and care delivery for high-risk groups with particular attention to preventing progression to advanced liver disease and cancer.

2 | Changing Dietary Landscape and the Rise of MASLD

Historically, human diets have primarily centered on unprocessed and whole foods rich in fiber, lean proteins, and complex carbohydrates, which support liver health by providing a balanced intake of macronutrients. Many modern diets are characterized by high consumption of UPFs rich in processed sugars, refined carbohydrates, and unhealthy (saturated) fats—factors that contribute significantly to the pathogenesis of MASLD [1–3]. Diets high in fructose and *trans* fats promote insulin resistance and place significant stress on hepatic lipid metabolism, leading to fat accumulation and subsequent liver dysfunction [1].

The liver is particularly vulnerable to fructose, a type of sugar that is metabolized exclusively in the liver [9, 10]. Excessive fructose consumption promotes *de novo* lipogenesis (fat formation within the liver), initiating the onset of steatosis. Notably, high-fructose diets are most prevalent among low-income populations due to the affordability of sugar-sweetened beverages and fast foods, underscoring the role of socioeconomic factors in MASLD risk [11]. Additionally, diets high in UPF promote insulin resistance, lipid accumulation in liver cells, and chronic inflammation, creating an environment conducive to liver damage. The prevalence of UPFs has been associated with the rise of chronic diseases, including MASLD and MASH [12].

3 | MASLD and Genetic Polymorphisms

Genetic, epigenetic, and socioeconomic factors appear to increase vulnerability among certain racial and ethnic groups. A systematic review found that the prevalence of MASH among US Hispanic adults is approximately 61%, significantly higher than in non-Hispanic adults, with a relative risk of 1.42 (95% CI, 1.04–1.93) [13–15]. National Health and Nutrition Examination Survey data indicate a significant increase in the prevalence of MASLD among Mexican Americans [16]. This highlights significant differences observed in the Hispanic population living in the United States, according to their country of origin [17]. In addition, Hispanics could exhibit a higher risk of MASH [8]. In contrast, non-Hispanic Black individuals appear to have a

lower prevalence of MASLD. Furthermore, non-Hispanic Black individuals exhibit a unique metabolic phenotype that offers protection against hepatic steatosis, even when faced with similar metabolic risk factors [3]. However, once disease is established, non-Hispanic Black patients experience the highest rates of overall and non-liver-related mortality, highlighting critical disparities in disease outcomes across ethnic groups.

Genome-Wide Association Studies have identified significant genetic variants associated with MASLD [18]. Table 1 outlines the major genetic variants associated with MASLD risk and their prevalence across various populations. Genetic susceptibility plays a key role in MASLD pathogenesis, with certain variants disproportionately affecting specific populations. For example, the PNPLA3 I148M polymorphism is associated with increased liver fat content and a higher likelihood of developing MASH [19]. This variant is highly prevalent among Hispanic individuals, with ~50% carriers, compared to 23% of non-Hispanic Whites and 17% of non-Hispanic Blacks [20]. Non-Hispanic Black, Hispanic, and Indigenous populations experience disproportionately high rates of MASLD and face more significant risks of liver-related complications, including increased risk of HCC development [21]. The frequency of this polymorphism is high in Hispanics of Mexican, Central, and South American descent, while those of Afro-Caribbean or European background show lower prevalence rates [22]. In addition to the PNPLA3 variant, other genetic polymorphisms, such as TM6SF2 and MBOAT7, contribute to individual differences in MASLD susceptibility [23–25].

Non-Hispanic Black individuals exhibit lower rates of MASLD, which may be attributed to certain genetic factors that provide protective benefits despite a higher incidence of insulin resistance and metabolic syndrome, but tend to have worse outcomes once the disease is established [26]. In addition, Non-Hispanic Black patients with MASLD experience the highest rates of overall and non-liver-related mortality, followed by Hispanic patients, whereas Asian patients tend to have the lowest risk of adverse outcomes when compared to White patients [23]. Asian populations, particularly those of South Asian and East Asian descent, also demonstrate unique MASLD patterns, with disease development at lower BMI thresholds compared to other ethnic groups [23]. These patterns highlight the intricate interaction between genetic predisposition, environmental influences, and social determinants of health in shaping MASLD-related outcomes, including cancer risk (Figure 1). Studies have highlighted the importance of these genetic determinants in disease progression and their potential for developing risk-stratification tools [27–29]. Additionally, these findings highlight the importance of utilizing precision medicine in managing MASLD [30].

4 | Interaction of Genetics, Social Determinants, and Epigenetics

While genetics plays a crucial role, genetic variance does not account for the striking disparities observed in MASLD. Ethnic minorities consistently experience compounded risks due to social determinants of health, such as limited healthcare access and food and nutrition insecurity. Non-Hispanic Black and Hispanic communities face nutrition insecurity due to the lower

cost and more access to UPF relative to more affluent communities in the United States [31].

Outcomes related to MASLD significantly differed between food-secure and food-insecure populations. Food insecurity has been linked to a higher prevalence of MASLD, along with poorer liver-related health outcomes. Individuals experiencing food insecurity are at an increased risk of developing MASLD and advanced fibrosis compared to those who are not food insecure. Food insecurity refers to having restricted or uncertain access to nutritionally adequate and safe food or facing challenges in obtaining food through socially acceptable means [32]. A study by Tamargo et al. observed a correlation between food insecurity and liver fibrosis among low-income, middle-aged adults [33]. In models adjusted for demographic, socioeconomic, and behavioral health factors, food insecurity was significantly associated with an increased likelihood of several health conditions. Individuals experiencing food insecurity had higher odds of advanced liver fibrosis (AOR, 2.20; 95% CI, 1.27–3.82), obesity (AOR, 1.32; 95% CI, 1.06–1.66), and diabetes (AOR, 1.41; 95% CI, 0.999–1.982; $p = 0.05$) when compared to those living in food-secure households [34]. Similarly, food insecurity was associated with increased all-cause mortality rates in adults with advanced fibrosis [35]. Food insecurity can initiate a complex cycle involving inadequate nutrition and compensatory overconsumption of low-quality foods, thereby elevating metabolic risk. Additionally, it contributes to heightened household stress, compels individuals to choose between purchasing food and obtaining medical care, raises the likelihood of not adhering to prescribed medications, and impairs effective management of chronic diseases, all of which are critical pathways that contribute to poorer metabolic and long-term health outcomes [36, 37].

Epigenetic modifications resulting from chronic exposure to adverse environmental factors, such as poor diet, sedentary lifestyle, and persistent psychosocial stress, may alter gene expression and influence disease susceptibility [38]. Addressing these disparities necessitates implementing targeted prevention programs. Engaging communities, particularly high-risk populations such as Hispanics and non-Hispanic Black, is important to improving prevention and early intervention strategies. These initiatives should incorporate culturally appropriate education, improved access to nutritious food, and tailored health interventions.

Integrating genetic and epigenetic research into clinical and public health strategies is crucial for enhancing the prevention and management of MASLD. For example, incorporating genetic risk assessments into routine screenings for high-risk groups can foster personalized prevention approaches [39–41]. Public health policies need to address the inequities in food access and healthcare availability, as these factors significantly impact MASLD outcomes [42].

In addition, clinical guidelines could incorporate genetic and epigenetic information to enhance patient risk stratification and inform interventions such as dietary modifications, pharmacotherapy, and monitoring for liver complications. Policymakers should consider these insights when pursuing broader healthcare reforms intended to diminish racial and ethnic disparities in MASLD prevalence and outcomes.

TABLE 1 | Genetic variants and their impact on MASLD risk by population [19–26].

Gene	Variant	Association with MASLD	Population-specific prevalence	Clinical implications
PNPLA3	I148M (rs738409)	Strongest genetic risk for hepatic steatosis, inflammation, and fibrosis progression	Hispanic (49%), non-Hispanic White (23%), non-Hispanic Black (17%)	Two times higher HCC risk in carriers
TM6SF2	E167K	Increases liver fat but may lower serum lipids; linked to fibrosis severity	Global, but less studied in ethnic-specific distributions.	Confers higher CVD risk
MBOAT7	Multiple variants	Disrupts phospholipid metabolism; associated with MASH and advanced fibrosis	Higher in Hispanic and Indigenous populations (Hispanic > White > Black)	May reduce response to dietary interventions
GCKR	rs1260326	Hyperactivation of glucokinase: increases hepatic fat storage and triglycerides. Common in metabolic syndrome associated with MASLD progression	Varied; limited ethnic-specific data (Hispanic [32%], White [28%], Black [18%])	Linked to rapid MASLD progression in T2DM patients. May require aggressive glycemic control
HSD17B13	rs72613567:TA (protective variants)	Loss-of-function variant protects against cirrhosis and HCC	Non-Hispanic Black (potential protective effect)	Potential therapeutic target

Abbreviations: CVD, cardiovascular diseases; GCKR, glucokinase regulatory protein; HCC, hepatocellular carcinoma; HSD17B13, hydroxysteroid 17-beta dehydrogenase 13; MASH, metabolic-associated steatohepatitis; MASLD, metabolic dysfunction–associated steatotic liver disease; MBOAT7, membrane-bound O-acyltransferase domain-containing 7; PNPLA3, patatin-like phospholipase domain-containing 3; T2DM, type 2 diabetes mellitus; TM6SF2, transmembrane 6 superfamily member 2.

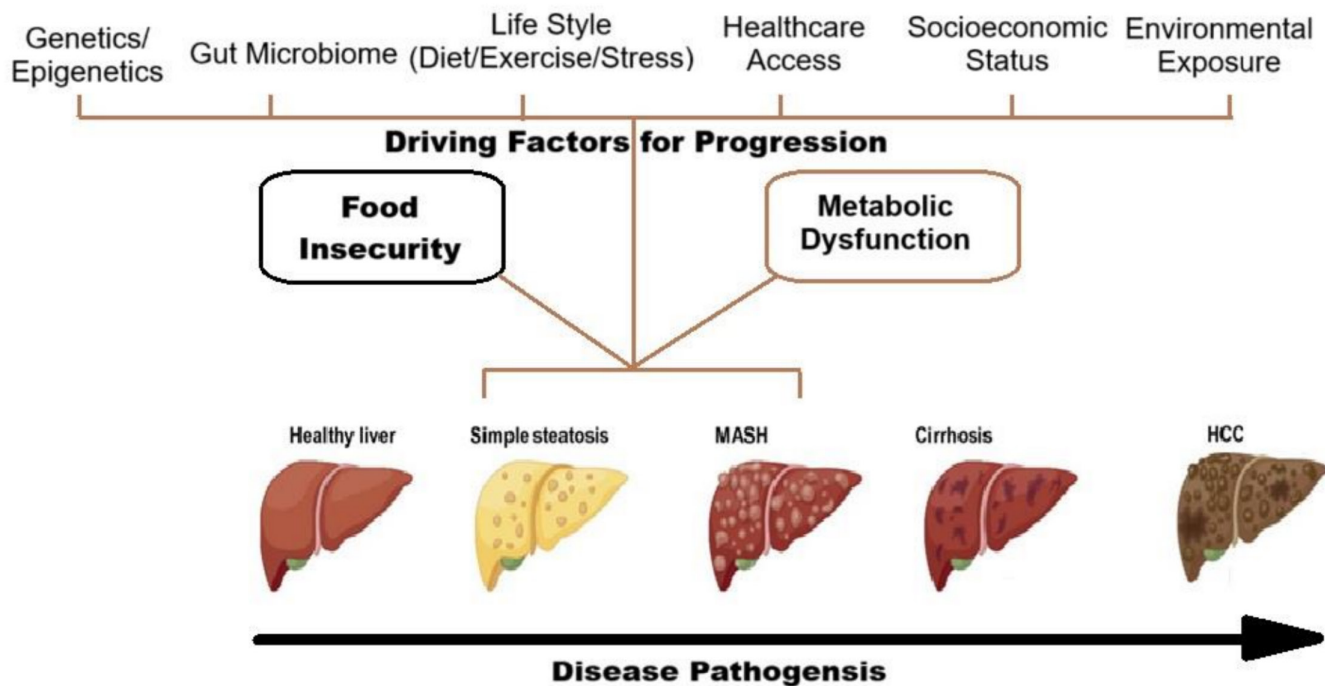


FIGURE 1 | Integrated model highlighting the multifactorial contributors in MASLD progression and associated cancer risk. The infographic illustrates how various factors, including epigenetics, genetics, environmental exposures, socioeconomic conditions, healthcare access, and lifestyle patterns, converge to influence the pathogenesis of MASLD, steatohepatitis (MASH), cirrhosis, and hepatocellular carcinoma (HCC).

5 | Health Disparities in MASLD Prevalence and Access to Care

The socioeconomic landscape further intensifies MASLD disparities. Reducing MASLD and liver cancer disparities necessitates multifaceted approaches that extend beyond individual dietary changes. Low-income communities face restricted access to fresh produce and healthier food options, especially in areas known as “food deserts,” a term used to describe neighborhoods where residents have limited access to affordable and nutritious food, typically due to the absence of grocery stores and the predominance of convenience stores and fast-food outlets [43]. This food insecurity leads to reliance on low-cost, high-calorie processed foods, which exacerbate insulin resistance and liver steatosis [44]. Given that 20%–25% of urban neighborhoods in the United States qualify as food deserts, this lack of access is a critical barrier to MASLD prevention [45].

Comprehensive policy reform is required to address these systemic barriers. This includes eliminating food deserts through incentivizing grocery stores in underserved areas, expanding the Supplemental Nutrition Assistance Program benefits for nutritious foods, implementing sugar taxes to reduce consumption of processed foods, improving healthcare infrastructure in low-income communities, and ensuring insurance coverage for preventive services and pharmacological treatments. Table 3 provides a detailed framework of these policy recommendations.

Moreover, communities disproportionately affected by MASLD are less likely to access advanced diagnostic tools and treatments [46]. In recent years, MASLD and MASH have become increasingly prevalent in communities facing socioeconomic and

healthcare disparities, leading to an alarming rise in liver cancer cases, particularly among Hispanic and Indigenous populations in the United States [12]. This trend underscores the urgency for targeted public health interventions and policy-driven solutions to address these disparities. Minority populations and individuals in lower socioeconomic strata are less likely to undergo early screening and diagnosis, often presenting with advanced MASLD and related comorbidities, such as type 2 diabetes and cardiovascular disease [47]. This inequity necessitates a public health approach prioritizing early screening, community health programs, and affordable access to therapeutic options, particularly for underserved populations. Table 2 provides an overview of recommended MASLD interventions and the significant barriers limiting their impact. Public health campaigns promoting liver health and routine screenings for MASLD among high-risk groups could also play a pivotal role in early detection, preventing the escalation of MASLD to advanced liver disease and cancer.

6 | MASLD Management: A Holistic Approach to Liver Health

Effectively managing MASLD requires a multifaceted approach that addresses diet, exercise, and access to healthcare. A diet rich in natural, unprocessed foods can dramatically reduce the burden on the liver. Reducing the consumption of processed sugars, refined carbohydrates, and unhealthy fats not only prevents the accumulation of fat in the liver but also reduces the inflammation that drives the progression to fibrosis, cirrhosis, and eventually cancer. Encouraging regular physical activity is equally important, as it improves insulin sensitivity and helps maintain a healthy weight, both

TABLE 2 | Interventions for MASLD management and associated barriers.

Intervention type	Examples	Benefits	Barriers	Implementation strategies	Target populations
Dietary	Mediterranean diet, low fructose intake	Reduces liver fat by 5%–10% in 6 months	Food deserts, the cost of fresh produce	SNAP incentives + culturally adapted nutrition education	Hispanic, low-income
Exercise	Aerobic/resistance training	Improves insulin sensitivity, reduces liver fat	Lack of safe spaces, time constraints	Community fitness programs, workplace initiatives	Urban youth
Pharmacological	GLP-1 agonists (semaglutide), resmetirom	Reduces liver fat, improves metabolic outcomes	High cost, limited insurance coverage	Advocacy for insurance mandates, patient assistance programs	Type 2 diabetes + MASLD
Policy level	Sugar taxes, subsidized healthy foods, and urban planning reforms	Population-level risk reduction, improved food access	Industry lobbying, implementation delays	Pilot programs in high-risk counties	Hispanic communities
Community based	Mobile health clinics, telemedicine, culturally tailored education	Bridges healthcare gaps, early detection (↑ Screening uptake by 40% in underserved areas)	Funding limitations, low awareness, Stigma, and low health literacy	Partner with faith-based organizations, federal grants, and train community health workers in culturally sensitive MASLD education	Hispanic, rural, low-income
Genetic screening	PNPLA3 testing for high-risk groups + GCKR panel	Early risk stratification: Identifies high-risk patients before fibrosis develops	Lack of guidelines, ethical concerns	Integrate into routine diabetes/obesity clinics; pilot Medicaid coverage	High-risk ethnic groups (Hispanic, South Asian)

Abbreviations: GCKR, glucokinase regulatory protein; GLP-1, glucagon-like peptide-1; MASLD, metabolic dysfunction–associated steatotic liver disease; PNPLA3, patatin-like phospholipase domain-containing protein 3; SNAP, Supplemental Nutrition Assistance Program.

of which are crucial for reducing liver fat and preventing the progression of MASLD [48–50]. Moreover, epigenetic mechanisms, shaped by diet and socioeconomic influences, represent another crucial yet underexplored area that could provide further insights into MASLD susceptibility and progression, particularly among populations at risk.

However, access to healthy foods and opportunities for physical activity is not evenly distributed. Fresh produce and whole foods are either too expensive or unavailable in many underserved communities. Public health initiatives must focus on eliminating food deserts and ensuring that all populations have access to affordable, nutritious food. Without such efforts, marginalized groups will continue to experience worse outcomes, including higher rates of progression to cirrhosis and HCC.

7 | Management Strategies: Diet, Exercise, and Access to Healthy Food

Modern diets also compromise the liver's role in detoxification. One might argue that the liver, with its remarkable regenerative capacity, should be able to cope with these dietary challenges. Communities with limited access to clean water and environments with high pollution levels face an added burden of environmental toxins, further straining liver function. However, the chronic nature of modern dietary habits means that the liver rarely gets a break. The continuous onslaught of unhealthy foods creates a persistent metabolic overload, preventing the liver from repairing itself effectively. Over time, this leads to inflammation, fibrosis, and, eventually, cirrhosis or liver failure. This situation is more severe in communities with limited access to healthcare, where early diagnosis and treatment of liver conditions are less likely.

Healthcare access barriers further amplify these disparities, making it difficult for minority populations to receive early diagnosis and intervention that could slow disease progression. Cultural differences, along with historical mistrust in the healthcare system, may prevent individuals from seeking early medical care, which is crucial in managing liver disease. Public health initiatives must prioritize educating individuals about the importance of liver health and the impact of modern diets, with a particular focus on underserved communities.

Socioeconomic factors, including the prevalence of food deserts in urban and underserved areas, also contribute to higher MASLD rates among racial and ethnic minorities. Limited access to fresh, affordable, and nutritious food options in these communities often leads to a reliance on processed and sugary foods. These dietary patterns increase the risk of metabolic disorders, such as MASLD, by providing excessive calories with minimal nutritional value. Hence, policies promoting access to healthy foods and discouraging the consumption of processed, sugary, and fatty foods are essential. Encouragingly, there is a growing awareness of the importance of liver health, and many are beginning to make dietary changes that support liver function. As such, there is a need for culturally informed healthcare approaches that recognize and mitigate these barriers to equitable liver disease prevention and treatment.

Addressing MASLD, we must fundamentally change our approach to diet and lifestyle. Shifting to a diet rich in natural, unprocessed foods can significantly reduce the burden on the liver [50]. Effective management requires addressing structural barriers in low-income and minority communities where food access is limited and healthcare utilization is fragmented. Policy-level interventions must ensure equitable access to healthy foods, safe spaces for physical activity, and comprehensive preventive care [51].

8 | Pharmacological and Nonpharmacological Interventions

Pharmacologic therapies, including a glucagon-like peptide-1 receptor agonist (GLP-1RA), such as semaglutide and resmetrom (a thyroid hormone receptor beta-selective agonist), have shown promise in reducing liver fat and improving metabolic outcomes; however, they remain cost-prohibitive and inaccessible to those who need them most [52, 53]. However, access to these treatments remains highly inequitable due to multiple barriers that disproportionately affect vulnerable populations. A recent study highlighted significant disparities in access to GLP-1RA medications, with socioeconomic status and insurance coverage serving as major barriers [54]. Cost-effectiveness studies have demonstrated the potential long-term benefits of various therapies for managing conditions like MASLD [55, 56]. However, the high upfront costs of these treatments pose a significant barrier for patients and healthcare systems [28].

Specific barriers include:

- Cost barriers: High out-of-pocket expenses for low-income populations without adequate insurance coverage
- Insurance restrictions: Many insurance plans, including Medicaid in several states, do not cover these medications or impose prior authorization requirements that delay access.
- Racial and ethnic disparities: Minority populations with limited insurance coverage face disproportionate barriers to accessing novel therapies.
- Geographic inequities: Patients in underserved rural and urban areas have reduced access to specialty care providers who prescribe these medications.

Improving access to these medications through policy initiatives, insurance reforms, and government-funded programs could greatly enhance outcomes for marginalized populations affected by MASLD. Additionally, current trends in insurance coverage underscore the need for broader inclusion of these therapies, as many insurance plans either do not cover them or impose high out-of-pocket expenses, thereby further restricting access for patients in need [57]. Policy advocacy is urgently needed to ensure equitable access to pharmacological interventions that can prevent progression to cirrhosis and HCC in high-risk populations.

Lifestyle interventions remain the cornerstone of MASLD management [58, 59]. Studies have shown that dietary changes (such

as decreasing the consumption of beverages high in fructose, refined carbohydrates, and *trans* fats) can effectively reverse hepatic steatosis and enhance liver function [51]. Many patients face systemic barriers to making these lifestyle changes, particularly in communities where structural issues limit access to nutritious food and safe spaces for exercise.

9 | Outcomes: The Broader Implications of MASLD

Due to the significant prevalence of obesity and metabolic dysfunction, the incidence of MASLD-related HCC is rising rapidly, emerging as a major public health concern. As MASLD progresses from steatosis to fibrosis and cirrhosis, chronic liver inflammation caused by fat accumulation sets the stage for cellular mutations and HCC development.

Notably, MASH occurs in a subset of MASLD patients, and HCC can develop even in the absence of cirrhosis, complicating screening and surveillance strategies. Screening programs developed primarily based on viral etiologies (hepatitis C virus and hepatitis B virus) of HCC need optimization, as the radiographic features of MASLD can reduce the sensitivity of ultrasound-based surveillance [60].

Ethnic disparities in HCC development are particularly pronounced in MASLD populations. Hispanic individuals, especially those carrying the PNPLA3 I148M variant, face a twofold increased risk of HCC compared to noncarriers. Indigenous populations also experience disproportionately high rates of MASLD-related HCC. These disparities underscore the urgent need for ethnic-specific risk stratification and tailored screening programs. Additionally, there appear to be variations in the efficacy of current anticancer therapeutics for HCC based on the underlying etiology (e.g., HCV-HCC vs. nonviral HCC) [61]. As such, dedicated research specific to MASLD-HCC and MASH-HCC is urgently needed to:

- Optimize screening strategies for noncirrhotic MASLD patients at high risk.
- Develop biomarkers specific to MASLD-related HCC for early detection.
- Evaluate treatment efficacy of systemic therapies in MASLD-HCC populations.
- Address disparities in HCC incidence, screening access, and treatment outcomes across racial and ethnic groups.

Beyond the liver, MASLD is closely linked to other severe comorbidities, including insulin resistance, type 2 diabetes, cardiovascular diseases, and kidney disease [62]. These comorbidities disproportionately affect racial-ethnic minorities, who often face barriers to preventive care and early diagnosis. In addition, the liver's compromised ability to detoxify harmful substances exacerbates systemic health problems, from fatigue and digestive issues to more severe conditions such as cancer. Socioeconomic factors and limited access to healthcare further compound these issues, leaving marginalized communities with fewer opportunities for early intervention and management.

10 | Care Delivery and Fragmentation: The Urgent Need for a Comprehensive Approach

Without a comprehensive approach to MASLD care, the management of this disease will become increasingly fragmented. Geolocation, physician availability, comorbidities, socioeconomic status, race and ethnicity, and food insecurity all influence care delivery today. In many cases, an individual's access to MASLD management depends on their location, healthcare provider, and insurance coverage. This unequal distribution of care risks leaving the most vulnerable populations behind.

To prevent this fragmentation, we must develop a more cohesive care delivery paradigm that addresses these disparities head-on. A one-size-fits-all approach will not work. Instead, care must be tailored to account for the unique challenges different communities face, which means expanding access to healthcare, reducing barriers to advanced pharmacological treatments like GLP-1 agonists, and ensuring that all populations have access to affordable, healthy food options.

11 | Policy Interventions: The Foundation for Tackling MASLD

Modern dietary habits play a significant role in the increasing prevalence of MASLD. Examining the historical policy decisions that have influenced today's food environment is key to understanding the current situation. In recent decades, agricultural policies have favored the mass production of corn and soy, key ingredients in many processed foods high in fructose and unhealthy fats. Subsidies for these crops have led to the widespread availability of inexpensive, calorie-dense processed foods. At the same time, healthier options, such as fresh fruits, vegetables, and lean proteins, have become relatively more expensive. Table 3 presents policy recommendations to shape the future of MASLD care

Research has identified significant disparities in the prevalence and management of MASLD. According to the clinical care pathway presented by Kanwal et al. [63], people in underserved communities, especially those with low socioeconomic status, are less likely to receive timely medical interventions and are more susceptible to reaching advanced stages of MASLD, which is mainly due to delayed diagnoses and limited healthcare access. In addition, Petroni et al. emphasized the importance of lifestyle interventions, such as dietary modifications and exercise, in managing MASLD [64]. However, systemic barriers, such as food insecurity and insufficient safe spaces for physical activity, often hinder these efforts in low-income areas.

A recent study proposed customized healthcare models for individuals with MASLD, underscoring the need for personalized care that addresses the specific needs of disadvantaged populations [65]. Without coordinated reforms, healthcare access may remain fragmented, leading to varying health outcomes that depend on geographic location and social determinants of health. The American Diabetes Association has asserted that socioeconomic status and access to quality care

TABLE 3 | Policy recommendations for shaping the future of MASLD care.

Policy area	Recommendation	Details/rationale
Food security	Eliminate food deserts	By fostering access to diverse and nutritious food sources, policies can directly address the social determinants of health that contribute to MASLD.
	Expand and improve food access programs to provide nutritious foods	Incentivizing grocery stores in underserved areas and supporting community gardens directly combat the dietary roots of MASLD by increasing the availability of fresh, healthy foods.
Healthcare access	Expand access to preventive services	Prevents financial barriers from hindering patient care and ensures all individuals have the opportunity to benefit from new therapies.
	Reduce sugar/processed food consumption	Proven strategies, such as sugar taxes and clear food labeling, can influence consumer choices, leading to healthier dietary habits and a lower prevalence of MASLD. Sugar taxes, improved food labeling, and public health campaigns on healthy eating. Taxes on sugar-sweetened beverages can reduce sales and consumption of these products.
	Expand preventive services, ensure equitable access to treatments	Subsidized diagnostics, telemedicine, and insurance coverage for pharmacological interventions
Public health education	Promote liver health awareness	Educating the public about the link between diet and liver health empowers individuals to make informed choices that help prevent MASLD
	Engage schools, workplaces, and communities	Creates widespread opportunities to foster healthy eating and exercise habits from an early age and throughout adulthood
	Invest in community-based education	Tailoring educational initiatives to high-risk populations ensures that information is culturally sensitive and effectively reaches those who need it most.
	Leverage outreach platforms	Utilizing mobile health clinics, subsidized diagnostic services, and telemedicine can overcome barriers to healthcare access, facilitating early detection and intervention, especially in remote or underserved areas.
Health equity and disparities	Address racial and ethnic disparities, consider genetic risk factors	Acknowledging genetic factors (e.g., lower MASH cirrhosis susceptibility in non-Hispanic Black individuals) is vital for personalized care strategies. This ensures all populations receive care tailored to their unique biological and social needs, preventing fragmented and unequal outcomes.
	Develop culturally sensitive campaigns (engage policymakers, healthcare providers, and community leaders)	Cross-sector partnerships to develop targeted interventions Collaboration among policymakers, healthcare providers, and community leaders is crucial for designing campaigns that resonate with diverse populations, thereby ensuring effective message delivery and engagement.

are crucial in preventing and managing MASLD, as well as its associated comorbidities, including type 2 diabetes and cardiovascular disease [66]. Without targeted policy interventions to address these challenges, MASLD rates are likely to continue increasing, particularly among vulnerable populations. Table 3 outlines key policy priorities for shaping the future of MASLD care, providing an actionable summary of these recommendations.

12 | Conclusion: The Path Forward

The rising occurrence of MASLD presents a major public health concern, primarily driven by modern dietary practices and exacerbated by socioeconomic and racial-ethnic inequalities, which highlights the pressing necessity for a comprehensive strategy for liver health that considers genetic, dietary, and economic elements, as well as cancer prevention strategies.

Addressing this issue requires thorough policy interventions. These efforts are expected to improve food security, increase access to healthcare, and reduce treatment disparities. Public health systems must adopt policies that promote healthier diets, enhance food access, and ensure equitable healthcare for vulnerable populations.

A coordinated care delivery model that accounts for geolocation, race and ethnicity, socioeconomic status, and food insecurity is crucial for delivering equitable and effective care to individuals with MASLD. By emphasizing liver health through better diet, exercise, and access to advanced therapeutic options, we can prevent the progression of MASLD to more severe outcomes, including nonliver cancer, cirrhosis, liver failure, and HCC, and enhance overall health outcomes, particularly in high-risk ethnic populations.

To mitigate the impact of MASLD and promote liver health for future generations, prioritizing prevention and addressing systemic health disparities is crucial. Policymakers should introduce supportive measures, clinicians should emphasize the importance of early detection and comprehensive care, and researchers should continue to investigate innovative approaches to MASLD, including ethnic-specific risk stratification, novel biomarkers for HCC detection in noncirrhotic MASLD, and evaluation of treatment efficacy across diverse populations. Collectively, these initiatives can lead to significant progress toward a healthier future for all communities, with reduced cancer burden and improved health equity.

Notably, food insecurity, driven by financial hardship and chronic stress, has emerged as a key independent contributor to metabolic dysfunction, including obesity, diabetes, and hypertension. Food insecurity functions as a root social determinant that operates through multiple pathways: (1) directly through stress-induced hormonal changes affecting metabolism, (2) indirectly through poor diet quality and compensatory overconsumption of low-cost processed foods, (3) via healthcare trade-offs where individuals choose between food and medical care, and (4) through medication nonadherence due to resource constraints. These cardiometabolic risk factors are strongly linked to hepatic fat accumulation and fibrosis. Research

demonstrates that food insecurity is independently associated with MASLD and advanced fibrosis, even after adjusting for diet quality and lifestyle factors, justifying its central positioning in the model alongside metabolic dysfunction as a fundamental driver rather than a mere lifestyle consequence.

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Ethics Statement

Not applicable.

Conflicts of Interest

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Data sharing not applicable to this article, as no datasets were generated or analyzed during the current study.

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