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MASLD in the Next Generation: Metabolic and Lifestyle Factors Shaping the Disease in Young Adults

Moirangthem Jeena Devi[1], Dr. Anita Malhotra[1], Dr. Piyush Ranjan[2], Dr. Manisha Sabharwal[1], Gayathri Byloppilly[2]

[1]Department of Home Science, University of Delhi, India
[2]Department of Medicine, All India Institute of Medical Sciences, New Delhi, India.

Corresponding Author: Dr. Piyush Ranjan

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ABSTRACT

Metabolic dysfunction-associated steatotic liver disease (MASLD) has become an alarming and growing health issue among young adults, closely linked to rising rates of obesity, sedentary lifestyles, and metabolic dysfunction. This narrative review comprehensively examines the multifactorial risk factors underlying MASLD development and progression in young adults, emphasizing metabolic, lifestyle, environmental, and behavioural factors. Key metabolic factors include insulin resistance, dyslipidemia, and hormonal imbalances, while lifestyle behaviours such as unhealthy diets, physical inactivity, and disrupted sleep patterns significantly exacerbate the disease risk. Additionally, developmental and reproductive influences unique to this age group further complicate the disease landscape. Recognizing these interconnected drivers is crucial for early detection and effective prevention strategies. The review also underscores the importance of tailored lifestyle interventions and public health initiatives aimed at mitigating MASLD's burden among this vulnerable population. By highlighting the complex interplay of factors shaping MASLD in young adults, this review aims to inform future research, clinical practice, and policy development focused on addressing this emerging epidemic.

Keywords: MASLD, Young Adults, Metabolic Risk Factors, Lifestyle Factors, Obesity.

INTRODUCTION

Metabolic dysfunction-associated steatotic liver disease (MASLD), a term endorsed by recent international consensus to replace "non-alcoholic fatty liver disease" (NAFLD), underscores the pivotal role of metabolic dysfunction in the pathogenesis of hepatic steatosis (1). MASLD has rapidly emerged as a significant health challenge worldwide, affecting individuals across various age groups, including young adults (2,3). Once considered a condition primarily affecting older populations, the prevalence of MASLD among young adults has surged in recent years, paralleling the global rise in obesity (4,5), sedentary lifestyles, and metabolic syndrome (6). This epidemiological shift underscores the urgent need for a comprehensive understanding of MASLD risk factors, disease progression, and long-term implications in younger populations.

The multifaceted nature of MASLD pathogenesis involves a complex interplay

of genetic predispositions, environmental influences, and lifestyle factors (7,8). From genetic polymorphisms to early-life exposures and dietary habits, various elements contribute to hepatic lipid accumulation, inflammation, and fibrosis, culminating in a spectrum of liver pathologies (9). Beyond its immediate impact on liver health, MASLD poses significant risks for cardiovascular disease, type-II diabetes, and hepatocellular carcinoma, amplifying its public health significance (10).

In the light of these challenges, this narrative review aims to provide a comprehensive synthesis of current knowledge on MASLD in young adults. Drawing from epidemiological studies, clinical observations, and mechanistic insights, this review seeks to elucidate the intricate factors driving MASLD onset and progression among this vulnerable population. Additionally, it also aims to synthesize current evidence on metabolic, lifestyle, environmental, and behavioral risk factors for MASLD in young adults, while highlighting epidemiological trends and intervention strategies.

RISK FACTORS FOR MASLD

Metabolic risk factors

Obesity: Obesity plays a central role in MASLD pathogenesis by promoting hepatic fat accumulation and insulin resistance (11). The expansion of adipose tissue, particularly visceral fat, leads to increased release of free fatty acids (FFAs) into circulation, promoting ectopic lipid deposition within the liver (12). This process fosters hepatic steatosis, inflammation, and fibrosis, which are hallmarks of MASLD progression (11).

Adipokines, cytokine-like hormones secreted predominantly by adipose tissue, also play a pivotal role in orchestrating energy homeostasis, inflammation, and fibrogenesis, positioning them as critical mediators in MASLD pathogenesis and promising therapeutic targets (13). In obesity, dysregulated adipokine profiles characterized by increased leptin and decreased adiponectin create a pro-inflammatory and metabolically imbalanced milieu. Leptin, through its profibrogenic actions, stimulates hepatic stellate cell activation and collagen deposition, driving fibrotic remodeling of the liver (14). Conversely, reduced adiponectin impairs hepatic fatty acid oxidation and enhances insulin resistance, further promoting hepatic steatosis and progression to NASH and fibrosis (13,15). Emerging evidence has elucidated the complex pathophysiological mechanisms linking obesity to MASLD, with recent studies highlighting several key contributing factors:

Role of Ectopic Fat Deposition: Bansal et. al., 2013 emphasizes the critical role of visceral and ectopic fat deposition in triggering hepatocyte lipotoxicity, which in turn drives liver inflammation and fibrosis (12).

Gut Microbiome Alterations*:* Obesity-associated gut dysbiosis is now recognized as a contributing factor to MASLD (16). A study by demonstrates that alterations in gut microbiota composition can increase gut permeability, leading to endotoxemia and hepatic inflammation.

Molecular Pathways: Advances in molecular biology have identified obesity-related upregulation of pathways such as JNK (c-Jun N-terminal kinase) and NF-κB signaling, which exacerbate insulin resistance and hepatocyte apoptosis (17).

Adipose Tissue Inflammation: Chronic low-grade inflammation in adipose tissue, characterized by macrophage infiltration, further propagates systemic inflammation and hepatic injury (18) .

Insulin Resistance (IR): Dysregulation of insulin signaling pathways plays a central role in the development and progression of MASLD among young adults (19). Insulin resistance, characterized by impaired cellular response to insulin, disrupts the balance between hepatic glucose production and peripheral glucose uptake, leading to hyperinsulinemia and compensatory hyperglycemia (20). In the context of

MASLD, hepatic insulin resistance fosters aberrant lipid metabolism, promoting de novo lipogenesis and inhibiting fatty acid oxidation (21). Consequently, hepatic lipid accumulation ensues, fueling inflammation, oxidative stress, and fibrosis (22). Furthermore, insulin resistance exacerbates systemic metabolic dysfunction, contributing to the pathogenesis of metabolic syndrome and its associated comorbidities (23).

Dyslipidemia: Abnormal lipid metabolism represents a hallmark feature of MASLD among young adults, characterized by dyslipidemia typified by elevated triglycerides and reduced high-density lipoprotein (HDL) cholesterol levels (4). Elevated circulating triglycerides reflect enhanced hepatic lipogenesis and impaired triglyceride clearance, exacerbating hepatic steatosis and inflammation (24). Concurrently, diminished HDL cholesterol levels impair reverse cholesterol transport and antioxidant capacity, further exacerbating lipid-induced hepatotoxicity (25,26). Dyslipidemia thus emerges as a critical determinant of MASLD severity and progression, amplifying the risk of adverse cardiovascular outcomes in this vulnerable population (17).

Metabolic Syndrome (MetS): The clustering of metabolic abnormalities comprising obesity, insulin resistance, dyslipidemia, and hypertension defines the metabolic syndrome, a potent precursor and comorbidity of MASLD among young adults (17,27). The synergistic interplay of these metabolic disturbances creates a pro-inflammatory and pro-fibrotic milieu within the liver, fostering the progression from simple steatosis to non-alcoholic steatohepatitis (NASH) and advanced fibrosis (28). Moreover, metabolic syndrome confers heightened risks of cardiovascular disease and type 2 diabetes, compounding the morbidity and mortality burden associated with MASLD (23,27). As such, early identification and management of metabolic syndrome components are paramount in mitigating MASLD risk and improving long term health outcomes in young adults.

Environmental Risk Factors

Air Pollution: Emerging research indicates a potential association between exposure to ambient and near-roadway air pollution and the risk of Metabolic dysfunction-associated steatotic liver disease (MASLD) in young adults (3). Airborne pollutants, such as particulate matter (PM) and nitrogen dioxide (NO_2), have been linked to increased hepatic fat deposition and higher levels of hepatic fat content (29). These pollutants may disrupt systemic lipid homeostasis, trigger inflammation, and promote oxidative stress, thereby exacerbating metabolic dysfunction and contributing to MASLD pathogenesis in susceptible individuals (30).

Lead Exposure: Environmental toxicants, including lead, pose a significant risk for hepatic damage and steatosis, particularly among young adults (3,9). Lead interferes with hepatic physiology, inducing oxidative stress, lipid peroxidation, and inflammatory responses, thereby promoting hepatic fat accumulation and liver injury (31). Common sources of lead exposure, such as lead-glazed ceramics and contaminated food products, underscore the importance of minimizing environmental exposures to mitigate MASLD risk (9,32).

Lifestyle and Behavioral Risk Factors

Dietary Factors: Diet plays a critical role in the pathogenesis of MASLD in young adults. Higher adherence to the “Western” dietary pattern characterized by elevated intakes of refined grains, sugar-sweetened beverages, red and processed meats, and fast foods has been associated with increased hepatic lipid accumulation and metabolic dysregulation (33). This dietary pattern provides excess energy and large quantities of simple sugars, such as fructose, which enhance hepatic de novo lipogenesis, impair

mitochondrial fatty acid oxidation, and promote insulin resistance, leading to steatosis and inflammatory responses (34,35). Refined carbohydrates and high-glycemic index foods further exacerbate insulin resistance and contribute to obesity-related metabolic dysfunction, creating a vicious cycle that amplifies MASLD risk (33). Although vegetable oils rich in omega-3 and omega-6 polyunsaturated fatty acids may exert beneficial effects on hepatic lipid metabolism and inflammation, these potential benefits are often attenuated by concurrent consumption of deleterious Western dietary components (36).

Sleep Disorders: Inadequate sleep duration and disturbances are associated with an increased risk of MASLD in young adults, potentially exacerbating metabolic dysfunction and liver injury (35). Sleep deprivation alters hormonal regulation, promotes appetite dysregulation, and impairs glucose metabolism, thereby contributing to insulin resistance and hepatic lipid accumulation (37). Addressing sleep-related issues and promoting healthy sleep hygiene practices are essential for mitigating MASLD risk in young adults.

Physical Inactivity: Sedentary behavior and lack of exercise contribute to metabolic disturbances, promoting hepatic fat deposition and inflammation in young adults (3,27). Physical inactivity exacerbates insulin resistance, dyslipidemia, and obesity, key drivers of MASLD pathogenesis (38). Encouraging regular physical activity and promoting a more active lifestyle are integral components of MASLD prevention and management strategies in this population.

Developmental and Reproductive Risk Factors

Early Life Factors: Low birth weight and childhood obesity are implicated in the early onset and progression of MASLD in young adults (39,40). Adverse early life experiences, such as intrauterine growth restriction and rapid weight gain during infancy, contribute to the development of metabolic dysfunction and hepatic steatosis later in life (41). Addressing developmental influences and optimizing early life nutrition are crucial for preventing MASLD and its associated complications in young adulthood.

Polycystic Ovary Syndrome (PCOS): Women with PCOS exhibit an increased risk of MASLD, driven by hormonal imbalances, insulin resistance, and dyslipidemia, particularly in young adulthood (42). PCOS-related metabolic disturbances, including hyperandrogenism and impaired glucose tolerance, predispose affected individuals to hepatic fat accumulation and inflammation (43). Early detection and comprehensive management of PCOS are essential for reducing MASLD risk and improving long-term health outcomes in young women.

Psychological Factors

Psychological stress, depression, and anxiety are increasingly recognized as contributing factors to MASLD in young adults, primarily through their effects on neuroendocrine pathways and lifestyle behaviors (44,45). Chronic psychological distress can lead to dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, increased cortisol levels, and subsequent insulin resistance and visceral fat accumulation, all of which promote hepatic steatosis (46). Moreover, mental health conditions may impair motivation for physical activity and dietary adherence, further exacerbating MASLD risk. Integrating mental health assessment and psychosocial interventions is essential in comprehensive MASLD prevention and management strategies for young adults.

Table 1. Summary of MASLD risk factors in young adults and their hepatic and clinical implications

MASLD Risk Factors and Implications in Young Adults		
Category	**Risk Factor**	**Implication**
Metabolic	Obesity	Increased hepatic fat accumulation, insulin resistance
	Insulin Resistance (IR)	Disrupted glucose metabolism, hyperglycemia
	Dyslipidemia	Elevated triglycerides, reduced HDL cholesterol
	Metabolic Syndrome (MetS)	Pro-inflammatory and pro fibrotic environment in liver
Environmental	Air Pollution	Increased hepatic fat deposition, inflammation
	Lead Exposure	Oxidative stress, lipid peroxidation, liver injury
Lifestyle & Behavioral	Dietary Factors (high fructose, sugar-sweetened beverages)	De novo lipogenesis, impaired fatty acid oxidation
	Sleep Disorders	Altered hormonal regulation, appetite dysregulation, impaired glucose metabolism
	Physical Inactivity	Insulin resistance, dyslipidemia
Developmental & Reproductive	Early Life Factors (low birth weight, childhood obesity)	Early onset and progression of MASLD
	Polycystic Ovary Syndrome (PCOS)	Hormonal imbalances, insulin resistance, dyslipidemia
Overall Implication	Increased risk of MASLD development and progression, leading to liver complications and comorbidities like cardiovascular disease and type 2 diabetes	

EPIDEMIOLOGY

Metabolic dysfunction-associated steatotic liver disease (MASLD) presents a significant health challenge worldwide, with prevalence rates varying across diverse populations and geographic regions. Studies conducted in different countries shed light on the extent of this condition's prevalence. For instance, research conducted in South Korea by Lee et al. 2022 (47) revealed a prevalence of 16.44% among males aged 18-31 years, indicating a considerable burden of MASLD in this demographic. Similarly, investigations in the USA, such as the NHANES survey by Alkhouri et al., 2022 (48), unveiled a striking prevalence of 40.04% among individuals aged 15-39 years, underlining the substantial presence of MASLD in the American populace. Moreover, studies conducted in Egypt, Mexico, Colombia, and Italy reported prevalence rates ranging from 9.4% to 31.6%, highlighting the global variability in MASLD burden (35,40,49,50). Remarkably, a retrospective cohort study in Boston by Corey et al. (2014) (51) documented an alarming prevalence of 66.7% among individuals aged 22 years or less, emphasizing the profound impact of demographic and environmental factors on MASLD prevalence. These findings underscore the need for comprehensive epidemiological research to elucidate the multifaceted determinants of MASLD and guide tailored public health strategies globally.

LIFESTYLE INTERVENTIONS FOR MASLD MANAGEMENT IN YOUNG ADULTS

Lifestyle modification remains the cornerstone of Metabolic dysfunction-associated steatotic liver disease (MASLD) management, particularly in the absence of approved pharmacological therapies. Dietary changes, regular physical activity, and sustained weight reduction are consistently endorsed as first-line strategies across major guidelines. Caloric restriction with a target weight loss of 7–10% is considered the primary goal, with evidence demonstrating that even modest reductions (3–5%) in body weight can improve hepatic steatosis, while greater reductions can reverse steatohepatitis and promote fibrosis regression (52,53). Importantly, early intervention during young adulthood may offer a critical window to halt disease progression and mitigate long-term hepatic and metabolic sequelae. Recent meta-analyses confirm that combined lifestyle interventions incorporating dietary

modification and exercise result in superior reductions in liver fat, aminotransferase levels, and insulin resistance compared to either strategy alone (54,55).

Dietary Modification

Nutritional interventions play a central role in the management of MASLD by reducing hepatic lipogenesis, improving insulin sensitivity, and attenuating inflammation. Diets emphasizing unsaturated fats, whole grains, and dietary fiber such as the Mediterranean diet have demonstrated superior efficacy in reducing intrahepatic fat and improving metabolic parameters. In a randomized crossover trial, a Mediterranean-style diet characterized by high monounsaturated fat and low added sugars led to significant reductions in hepatic steatosis and enhanced insulin sensitivity, as assessed by the euglycemic clamp, compared to a low-fat diet (55). Similarly, a 12-week intervention in obese adolescents showed that both Mediterranean and conventional low-fat diets significantly reduced BMI, hepatic fat, liver enzymes, and HOMA-IR, supporting their utility in younger populations (56).

Notably limiting simple sugars, particularly fructose, is crucial given its role in promoting de novo lipogenesis and visceral adiposity. Short-term fructose restriction in obese youth resulted in rapid reductions in hepatic fat, lipogenesis, and insulin resistance (57). Meta-analyses indicate that both low-fat and low-carbohydrate hypocaloric diets achieve comparable outcomes for weight reduction and liver fat loss, suggesting that individualized dietary approaches may be effective as long as they induce an energy deficit (58). Overall, current evidence supports adopting calorie-restricted, nutrient-dense diets prioritizing whole foods while limiting refined carbohydrates and saturated fats to slow MASLD progression (59,60).

Physical Activity

Regular physical activity independently benefits hepatic and metabolic health, even in the absence of weight loss (61). Aerobic and resistance training have both demonstrated efficacy in reducing hepatic steatosis and improving insulin sensitivity by enhancing fatty acid oxidation and glucose utilization (62). A meta-analysis of exercise interventions in adults reported significant reductions in liver fat with structured aerobic exercise, regardless of weight change (63). Similarly, resistance training improved liver fat and muscle strength, albeit with slightly less pronounced hepatic benefits than aerobic exercise (64).

In obese adolescents, a 12-week aerobic program (150 minutes per week at ≥70% VO_2 max) significantly decreased hepatic and visceral fat and improved insulin resistance (65). Current guidelines recommend a minimum of 150 minutes per week of moderate-to-vigorous aerobic activity, supplemented by resistance training at least twice weekly (66). Emerging evidence suggests young adults may derive greater hepatic improvements per unit of physical activity compared to older populations, underscoring the importance of early adoption of active lifestyles (63). Reducing sedentary time is also critical, as prolonged sitting has been associated with worsened hepatic outcomes independent of exercise levels.

Weight Loss as a Primary Target

Achieving and sustaining weight loss remains a central therapeutic objective for MASLD management. Clinical studies demonstrate that a 7–10% reduction in body weight is associated with histological improvements, including resolution of steatohepatitis and regression of fibrosis in a substantial proportion of patients (53). Even a 5% weight loss significantly reduces intrahepatic triglycerides and aminotransferase levels (67). Lifestyle intervention programs combining dietary counselling and exercise have shown robust metabolic benefits in obese adolescents, improving insulin sensitivity, fasting

glucose, HOMA-IR, triglycerides, and BMI(68).

Mechanistically, weight reduction decreases free fatty acid flux from adipose tissue to the liver, improves adipokine profiles (increased adiponectin, reduced leptin), and attenuates systemic inflammation (69,70). However, sustained weight loss is often hindered by adaptive metabolic responses, such as reductions in resting energy expenditure and compensatory hormonal changes that promote weight regain (71,72). Consequently, guidelines advocate for gradual, sustained weight loss supported by behavioral interventions, with a target of at least 10% in overweight or obese individuals to achieve durable hepatic improvements (66,67).

Behavioral and Digital Interventions

Behavioral strategies are critical for supporting adherence to lifestyle modifications and maintaining long-term weight loss. Techniques such as motivational interviewing, cognitive-behavioral therapy (CBT), and structured goal setting have demonstrated efficacy in enhancing lifestyle adherence and improving clinical outcomes (67,73). Cognitive-behavioral therapy is widely used to facilitate adherence to dietary strategies and improve emotional regulation in obesity and related conditions like MASLD (74). For example, CBT-based interventions combined with dietary counselling significantly reduced hepatic fat and improved dietary compliance over six months compared to standard advice (75).

Digital health tools, including smartphone applications, wearable devices, and telehealth platforms, offer scalable solutions to extend the reach of lifestyle interventions. A randomized controlled trial demonstrated that an MASLD -specific app delivering lifestyle education and automated reminders significantly improved weight loss, self-care behaviors, and quality of life over six months (76). Meta-analyses further confirm that eHealth interventions reduce BMI and liver enzymes (AST, ALT) compared to usual care (77). During the COVID-19 pandemic, telemedicine emerged as a viable alternative, achieving comparable reductions in body weight and BMI in patients with metabolic dysfunction-associated fatty liver disease (MAFLD) (76). These digital approaches may be particularly effective in young adults, offering accessible, engaging, and cost-effective means to support sustained lifestyle change (78).

MECHANISTIC INSIGHTS

Lifestyle interventions modulate the pathophysiology of MASLD through multiple interconnected biological pathways (Figure 1). Caloric restriction and dietary modification, particularly reductions in refined sugars and excessive caloric intake, lower circulating insulin and glucose levels, thereby suppressing hepatic de novo lipogenesis (79). This metabolic shift activates AMP-activated protein kinase (AMPK), enhances mitochondrial biogenesis, and promotes autophagy in hepatocytes, facilitating lipid oxidation and reducing hepatic fat accumulation (80). Physical activity further augments these effects by increasing energy expenditure and stimulating fatty acid oxidation in hepatic and skeletal muscle tissues (65). Myokines and hepatokines are signalling proteins secreted by skeletal muscle and the liver, respectively, that regulate glucose, lipid metabolism, and inflammation (81,82). Exercise stimulates myokines such as irisin and IL-6, which promote adipose tissue browning, enhance fatty acid oxidation, and improve insulin sensitivity (81). Simultaneously, hepatokines like FGF21 are upregulated, supporting glucose uptake and reducing hepatic lipogenesis, while fetuin-A levels decrease, attenuating pro-inflammatory signalling (81,83). These adaptations collectively mitigate hepatic steatosis and improve metabolic homeostasis in MASLD (81). Weight loss also contributes to the amelioration of MASLD by reducing adipose tissue mass and free fatty acid flux to the liver, while

improving adipokine profiles characterized by increased adiponectin and reduced leptin levels and mitigating oxidative stress and chronic low-grade inflammation (84). Furthermore, behavioural strategies and digital interventions reinforce these physiological benefits by promoting adherence to dietary and physical activity regimens (85). Sustained implementation of these lifestyle changes is critical for the long-term resolution of hepatic steatosis, improvement in insulin sensitivity, and reduction of fibrosis progression in individuals with MASLD.

Table 2. Lifestyle Interventions for MASLD with their metabolic targets and effects.

Type of Intervention	Primary Metabolic Target	Effect on MASLD	Supporting studies
Dietary changes (e.g. Mediterranean or low-fructose diet)	↓ Hepatic de novo lipogenesis; ↑ Insulin sensitivity	↓ Hepatic steatosis and Transaminases	(55–57)
Aerobic exercise (moderate-vigorous)	↑ Skeletal muscle fat oxidation; ↑ Hepatic insulin sensitivity	↓ Liver fat content; ↓ Insulin resistance; Improved AST/ALT	(63–65)
Resistance training	↑ Muscle mass; ↑ Resting metabolic rate	↓ Liver fat; ↑ Insulin sensitivity	(63,64)
Structured weight loss (≥7–10%)	↓ Adiposity; ↓ Lipotoxicity; ↑ Adiponectin	NASH resolution; Fibrosis regression; ↓ Steatosis	(53,68)
Behavioral Support	↑ Motivation; ↑ Adherence to diet/exercise	Greater weight loss; ↓ Hepatic steatosis	(75,76)
Digital Tools (Apps, Telehealth)	↑ Self-monitoring; ↑ Accountability	↑ Lifestyle adherence; ↓ Weight, BMI; Improve liver enzyme	(78,86)

Abbreviations: ALT; Alanine Aminotransferase, AST; Aspartate Aminotransferase, NASH; Non-Alcoholic Steatohepatitis, CBT; Cognitive Behavioral Therapy, MI; Motivational Interviewing, BMI; Body Mass Index, ↑; Increase, ↓; Decrease.

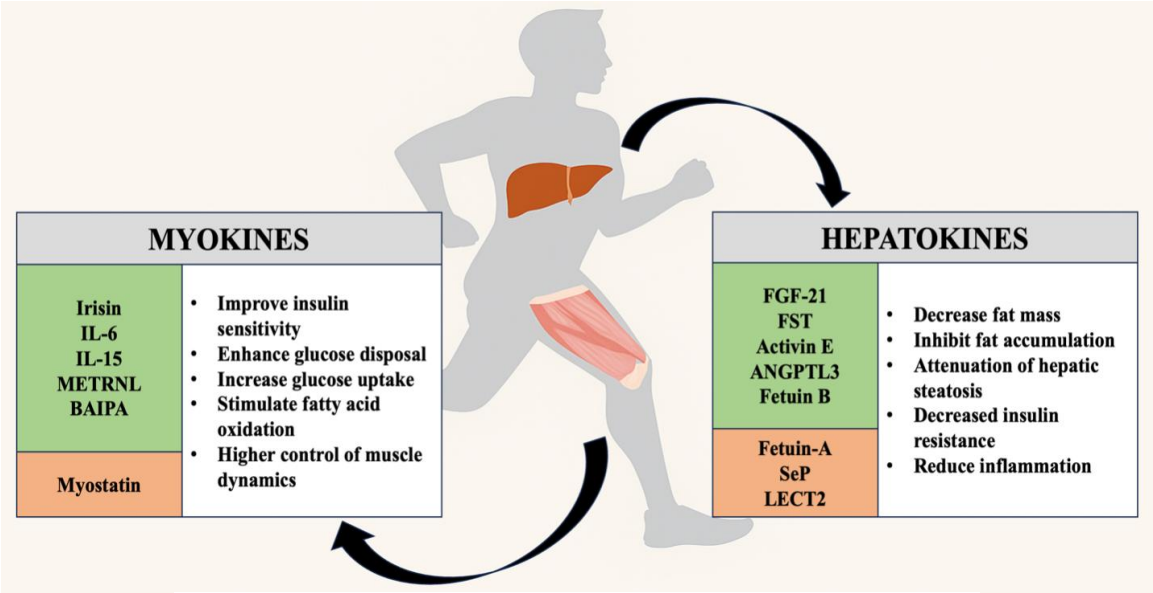

Figure 1: The conceptual model illustrates how physical activity-induced myokines and hepatokines improve metabolism, reduce liver fat, and attenuate MASLD progression.

Anti-Steatotic hepatokines/myokines are those mediators that protect against steatosis by enhancing fatty acid oxidation and improving insulin sensitivity. Exercise upregulates these beneficial mediators, promoting hepatic lipid clearance and metabolic health (81–83).

Pro-Steatotic hepatokines/myokines are those mediators that promote hepatic lipid accumulation and MASLD progression by impairing insulin sensitivity and lipogenesis. Exercise reduces these mediators, mitigating their harmful effect (81–83). (**Abbreviations:** IL6; Interleukin-6, IL5; Interleukin-5, METRNL; Meteorin-like, BAIPA; Beta-aminoisobutyric acid, FGF21; Fibroblast Growth Factor 2, FST; Folli statin, ANGPL3; Angiopoietin-like 3, SeP; Selenoprotein P, LECT2; Leukocyte cell-derived chemotaxin 2)

CONCLUSION

Metabolic dysfunction-associated steatotic liver disease (MASLD) is an increasingly prevalent and concerning public health issue, disproportionately affecting young adults worldwide. This narrative review explored the multifactorial risk factors contributing to MASLD, including metabolic, environmental, lifestyle, and developmental determinants. By categorizing these factors, it provides a comprehensive framework for understanding the complex etiology of MASLD in this population. The substantial geographic variability in MASLD prevalence highlights the necessity for region-specific interventions tailored to local population characteristics. Moreover, limitations in current diagnostic approaches for young adults underscore the urgent need for improved, age-specific screening tools.

Future research should focus on unravelling the mechanisms linking various risk factors to MASLD development and progression in young adults. Concurrently, public health initiatives must emphasize the promotion of healthy behaviors, such as balanced nutrition, regular physical activity, and sufficient sleep, as foundational strategies for MASLD prevention.

The interplay between obesity and MASLD underscores the importance of early identification and interventions targeting weight reduction, lifestyle modifications, and metabolic health to mitigate the disease burden, making it crucial for preventing severe complications like cardiovascular disease and type 2 diabetes. Success in mitigating the burden of MASLD will depend on collaborative efforts among healthcare providers, researchers, policymakers, and educators to raise awareness and empower young adults to adopt sustainable healthy lifestyles. Further research is warranted to explore personalized therapeutic approaches addressing the heterogeneity of obesity-related MASLD. Through these efforts, we can effectively combat the growing MASLD epidemic and ensure improved long-term health outcomes for the younger generation.

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