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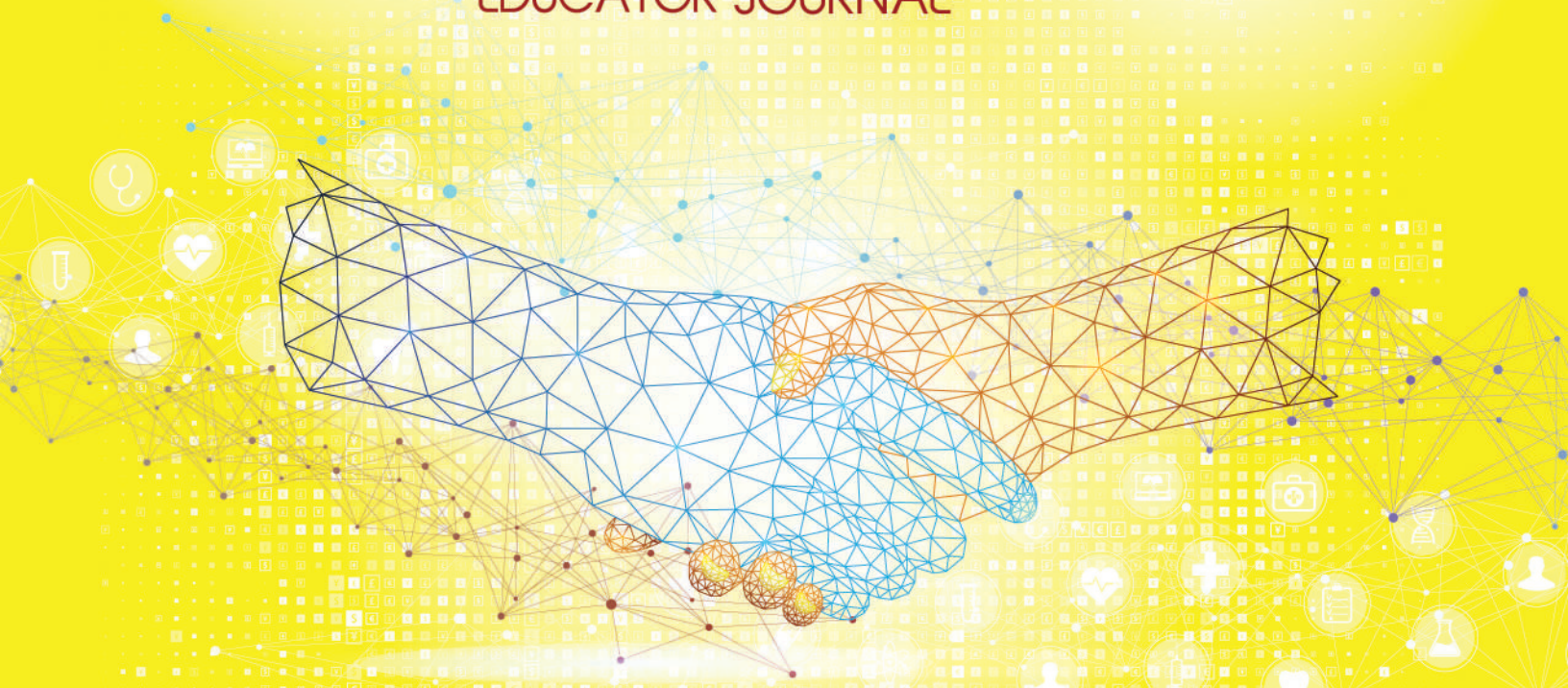

Theme of the Month

Integrated Liver and Metabolic Care

To keep Members Diabetes Care team abreast about
DSME /DSMS - (Diabetes Self management Education / Support) Concepts

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*DSME: Diabetes Self-Management Education, DSMS: Diabetes Self-Management Support

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FOREWORD

Research Society for the Study of Diabetes in India (RSSDI) remains at the forefront of diabetes education and research, creating platforms that encourage scientific discourse, knowledge dissemination, and the ongoing development of healthcare professionals. With the support of its active state chapters, RSSDI continues to promote scientific learning and community engagement across the country. The Mid-Term Conference of RSSDI Odisha conducted in May was one of the highlights, which brought together experts to discuss recent advances in diabetes and metabolic health. Another notable achievement was the Maharashtra Chapter's first academic webinar on the latest lipid guidelines, offering practical insights for clinical practice. Alongside these initiatives, multiple free health screening camps and awareness activities were conducted, reflecting RSSDI's continued focus on preventive healthcare, early detection, and making quality diabetes care more accessible. RSSDI is pleased to collaborate with USV in the collective vision of establishing India as a global benchmark in diabetes and metabolic care.

The Indian Diabetes and Metabolic Educators Journal (IDMEJ), previously known as the Indian Diabetes Educator Journal (IDEJ), is India's first and longest-running monthly publication dedicated to diabetes education since its establishment in April 2015. IDMEJ has developed over time into a useful scholarly forum for raising awareness, disseminating evidence-based knowledge, and assisting medical professionals in the all-encompassing treatment of diabetes and metabolic health. IDMEJ, which is published under the auspices of RSSDI, keeps advancing Diabetes Self-Management Education and Support (DSME/S) while expanding its scope to encompass a broader range of metabolic illnesses. Over 53,000 physicians and 29,000 diabetes educators in India are actively involved with the journal, which has a robust and expanding online presence.

The July edition of the IDMEJ focuses on the theme “Integrated Liver and Metabolic Care.” Growing evidence highlights the close relationship between metabolic disorders and liver health, particularly the rising burden of metabolic dysfunction-associated steatotic liver disease (MASLD) among individuals with obesity, type 2 diabetes, and insulin resistance. This issue provides diabetes educators with practical, evidence-based insights on early identification, risk assessment, lifestyle and nutritional interventions, weight management, and emerging therapeutic approaches. By promoting a multidisciplinary and patient-centred approach, this edition aims to support integrated care strategies that improve both metabolic outcomes and liver health, ultimately enhancing overall quality of life.

We sincerely acknowledge the invaluable contributions of all the authors and experts who have enriched this issue. This edition is dedicated to the tireless commitment of healthcare professionals whose steadfast pursuit of excellence is helping shape India into a global beacon of leadership in diabetes care.

Warm regards,

Dr. K. M Prasanna Kumar
MBBS, MD (Medicine), DM (Endocrinology)
Consultant Endocrinologist, Founder and Head of Centre for Diabetes and Endocrine Care, Bengaluru
Patron of RSSDI, Organising chairperson, RSSDI 2026

*DSME: Diabetes Self-Management Education, DSMS: Diabetes Self-Management Support

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CHIEF EDITOR'S NOTE

As Chief Editor, I am delighted to present this issue of the Indian Diabetes & Metabolic Educators Journal, dedicated to the theme "Integrated Liver and Metabolic Care." The increasing recognition of Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) as an important part of metabolic disease highlights the need to consider liver health as a key component of diabetes and overall cardiometabolic care.

This edition explores emerging concepts in MASLD, advances in non-invasive assessment, the role of nutrition and meal timing, the gut-liver axis, and the current treatment approaches. It also highlights the value of effective patient communication and behavior change strategies in achieving meaningful and sustained outcomes.

As the burden of metabolic liver disease continues to increase alongside diabetes, there is a growing need for a collaborative and patient-centered approach to care. Through this issue, we aim to help healthcare professionals, including diabetes educators, with practical, evidence-based knowledge that can be applied in daily practice to support better metabolic and liver health outcomes.

My sincere thanks to all contributors and the RSSDI community for their continued dedication to advancing metabolic health awareness, education, and clinical excellence in India.

Sincere Regards,

Dr Sunil Gupta
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Former National President DIPSI,
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Article: Insulin Resistance and Hepatic Steatosis: Bidirectional Relationship Between T2DM and MASLD

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Article: High Carbohydrate Intake and Liver Health

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Article: Beyond Liver Enzymes: Non-invasive Diagnosis of MASLD

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Issue No. 136, July 2026

Table of Content

Cover story: From NAFLD to MASLD: Redefining Metabolic Liver Disease — *Dr. G Vijaya Kumar* — 01

Insulin Resistance and Hepatic Steatosis: Bidirectional relationship between T2DM and MASLD — *Dr. R. K. C Mishra* — 04

High Carbohydrate Intake and Liver Health — *Dr. Harshal Ekatpure* — 06

Beyond Liver Enzymes: Non-invasive Diagnosis of MASLD — *Dr. Rowin Kumar* — 08

Interview of the Month with Dr. Gaurav Patil — *Dr. Gaurav Patil* — 10

Drug Spotlight: SGLT2 Inhibitors — *Dr. Pavithra Thamizharasan* — 13

Trending Diets: Time-restricted Eating in MASLD — *Dr. Mohit Mohan Singh* — 15

Gut–liver Axis in Metabolic Liver Disease — *Dr. Omkar Ramesh Patil* — 17

Diabetes Educator Toolkit: Skill of the Month: Motivational interviewing — *Dr. A. K. Gupta* — 19

Frequently Asked Questions on Integrated Liver and Metabolic Care — *Dr. Ravi Kumar* — 20

Ginger: A Monsoon Superfood for Immunity — 22

Role Play — 23

Cover Story: From Non-alcoholic Fatty Liver Disease (NAFLD) to Metabolic Dysfunction-associated Steatotic Liver Disease (MASLD): Redefining Metabolic Liver Disease

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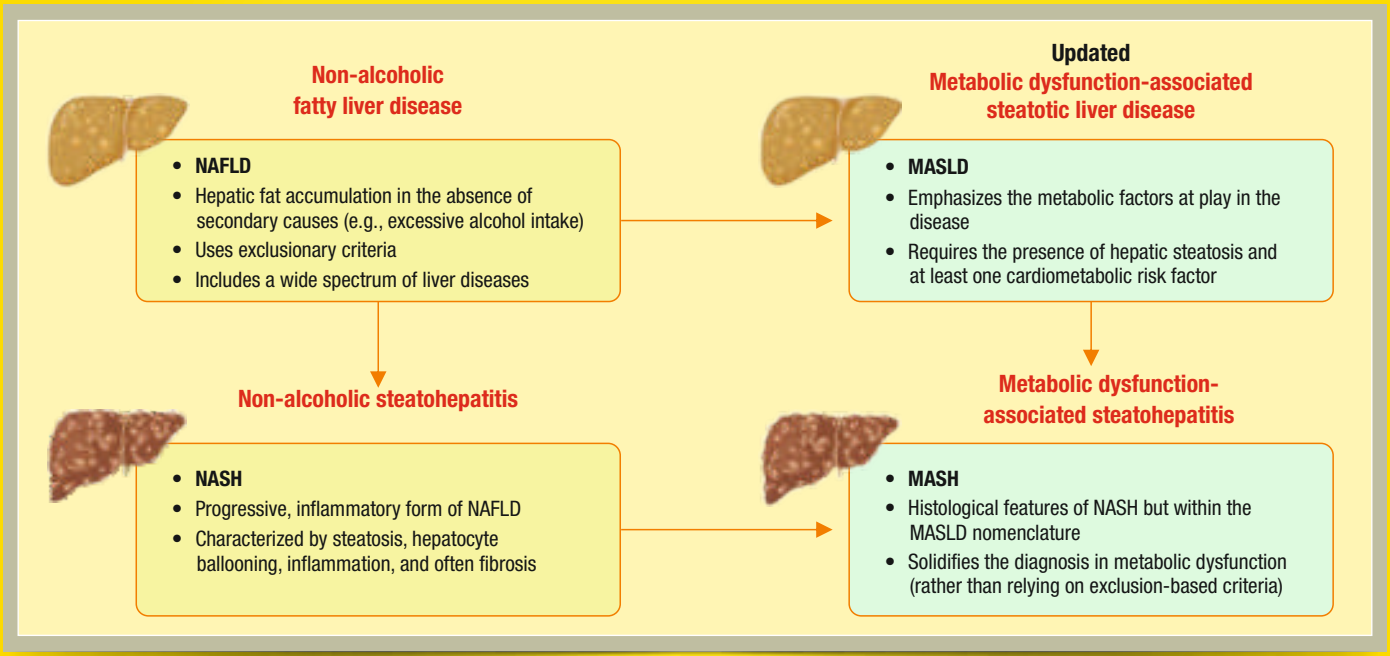
NAFLD was originally described in 1980 as a liver disease resembling alcoholic liver injury in individuals who consumed little or no alcohol. However, recent advances in scientific understanding have led to a major shift in terminology and disease definition. NAFLD is now increasingly being replaced by the term MASLD.

Why was there a need for change?

The diagnosis of NAFLD depended largely on excluding significant alcohol intake and other causes of liver disease. Researchers argued that the name failed to describe the true pathophysiology of the disorder.

In many cultural and religious settings, discussing alcohol use created discomfort and sometimes affected the doctor–patient relationship. Many patients also misunderstood the disease and believed that simply avoiding alcohol could prevent progression. Experts felt that changing the terminology could improve awareness, research interest, and patient management.



The evolution from NAFLD to MASLD

In 2020, experts proposed replacing NAFLD with metabolic dysfunction-associated fatty liver disease (MAFLD). In 2023, a global delphi consensus further renamed it MASLD. The new terminology highlights the strong link between fatty liver disease and metabolic risk factors rather than simply the absence of alcohol intake.

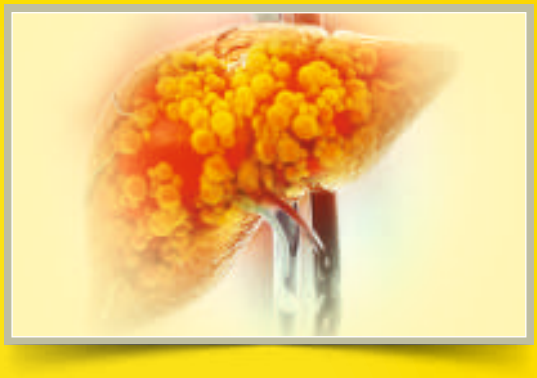
What is MASLD?

MASLD is defined as the presence of hepatic steatosis along with at least one cardiometabolic risk factor such as: obesity or increased waist circumference, type 2 diabetes mellitus (T2DM), hypertension, elevated triglycerides, low high-density lipoprotein (HDL) cholesterol and insulin resistance

The disease spectrum ranges from simple steatosis to metabolic dysfunction-associated steatohepatitis (MASH). MASH is the more severe inflammatory form and can progress to fibrosis, cirrhosis, liver failure, and hepatocellular carcinoma.

MASLD and metabolic syndrome: A strong connection

Metabolic liver disease has emerged as one of the most important global health concerns of the modern era. Over the last few decades, the prevalence of T2DM has increased rapidly due to sedentary lifestyle, and unhealthy dietary habits. MASLD is now recognized as a multisystem metabolic disorder rather than an isolated liver disease. It is closely associated with obesity, insulin resistance, metabolic syndrome, cardiovascular disease, and T2DM. Importantly, cardiovascular disease remains the leading cause of death in individuals with MASLD, highlighting the need for comprehensive metabolic risk management.



Global burden of MASLD

MASLD has become the most common chronic liver disease worldwide. Current evidence suggests that MASLD affects approximately 30% to 40% of the general adult population, including approximately 60% to 70% of individuals with T2DM and approximately 70% to 80% of those with obesity.

Changes in diagnosis and screening

The transition from NAFLD to MASLD has changed the approach to diagnosis and screening. Since many patients remain asymptomatic until advanced liver disease develops, early identification is essential. Current guidelines recommend screening high-risk groups such as individuals with obesity, T2DM, hypertension, or metabolic syndrome using non-invasive tests like: fibrosis-4 index (FIB-4), liver stiffness measurement or transient elastography.

Management of MASLD

Lifestyle modification remains the cornerstone of MASLD management. Weight loss through calorie restriction, healthy dietary habits, and regular physical activity can significantly improve liver fat, inflammation, and fibrosis, while pharmacological therapy, bariatric surgery, or liver transplantation may be considered in advanced stages of the disease.

Conclusion

The shift from NAFLD to MASLD represents much more than a simple name change. It reflects a major shift in understanding fatty liver disease as a metabolic and systemic disorder rather than merely a diagnosis of exclusion. The new terminology is more scientifically accurate, less stigmatizing, and more patient centred.

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Insulin Resistance and Hepatic Steatosis: The Bidirectional Relationship Between Type 2 Diabetes Mellitus (T2DM) and Metabolic Dysfunction-associated Steatotic Liver Disease (MASLD)

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T2DM and MASLD are among the most prevalent metabolic disorders worldwide and frequently coexist. These conditions share a bidirectional relationship, with insulin resistance acting as the central pathogenic mechanism. This contributes to metabolic dysfunction, disease progression, and an increased risk of long-term complications.

Insulin resistance and the development of MASLD

Insulin resistance impairs glucose uptake, leading to hyperglycemia and compensatory hyperinsulinemia. This promotes adipose tissue lipolysis, increasing the release of free fatty acids into circulation. The excess free fatty acids accumulate in the liver as triglycerides, causing hepatic steatosis, the hallmark feature of MASLD. Insulin resistance also stimulates hepatic de novo lipogenesis, further promoting liver fat accumulation.

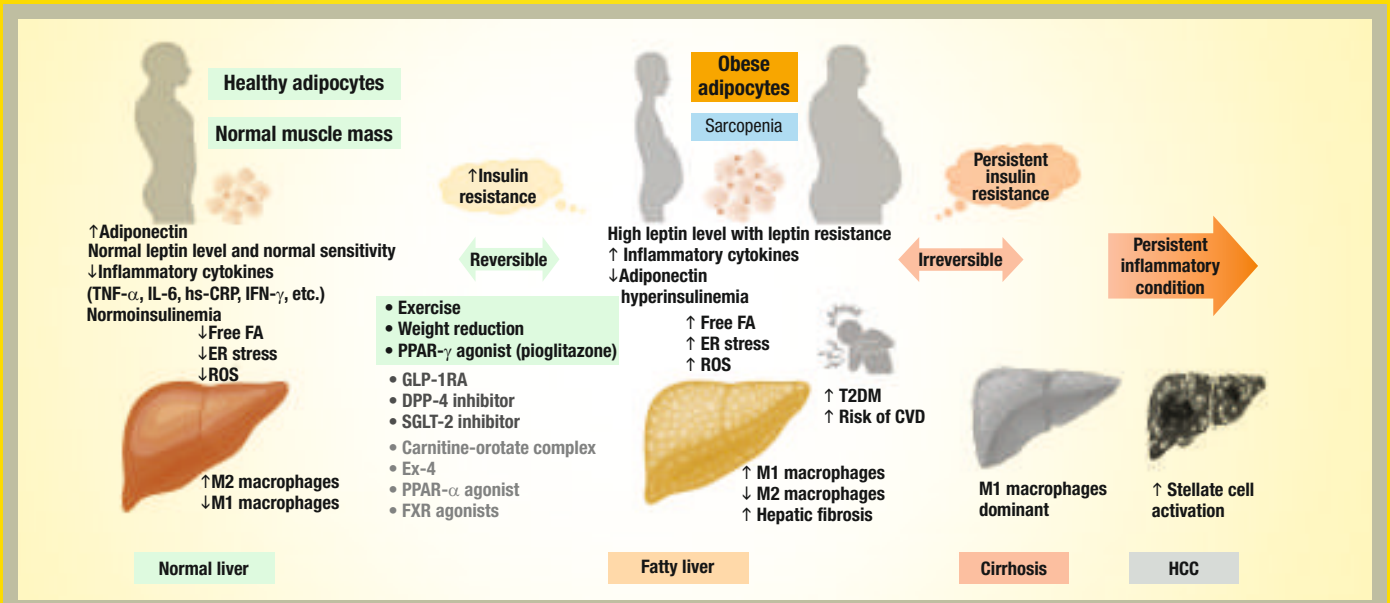


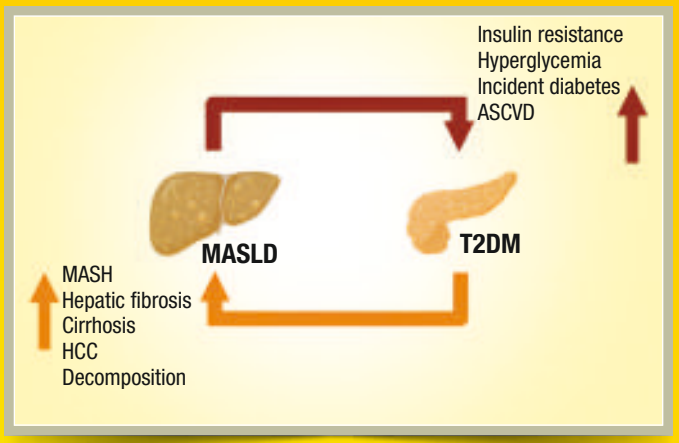
Fig. 1. Pathophysiology of insulin resistance in the progression of MASLD and T2DM (Ref: Jung I, Koo DJ, Lee WY; 2024)

Impact of MASLD on T2DM

Hepatic fat accumulation further worsens insulin resistance through inflammation, oxidative stress, mitochondrial dysfunction, and lipotoxicity. These alterations impair insulin signalling and increase hepatic glucose production, worsening hyperglycemia and poor glycemic control.

Clinical significance

MASLD is associated with an increased risk of developing T2DM, while T2DM promotes progression from steatosis to metabolic dysfunction-associated steatohepatitis (MASH), fibrosis, cirrhosis, and hepatocellular carcinoma. The prevalence of MASLD among individuals with T2DM ranges from 60% to 86%, highlighting the strong association between these disorders.



Prevention and management

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Lifestyle interventions remain the cornerstone of management. Sustained weight reduction, regular physical activity, and a balanced diet low in sugars and saturated fats can improve insulin sensitivity and reduce hepatic fat accumulation. Exercise also helps enhance glucose utilization, improve metabolic flexibility, and support glycemic control. Early identification and comprehensive management of both conditions are essential for reducing complications and improving long-term outcomes.

Overall, insulin resistance serves as the key link between T2DM and MASLD, driving the development and progression of both disorders. Addressing insulin resistance through lifestyle modification and appropriate therapeutic interventions remains fundamental to the prevention and management of both T2DM and MASLD.

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High Carbohydrate Intake and Liver Health

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Metabolic dysfunction–associated steatotic liver disease (MASLD) has emerged as one of the most common chronic liver disorders worldwide commonly associated with obesity, insulin resistance, and type 2 diabetes. MASLD ranges from simple fat accumulation in the liver to more severe forms characterized by inflammation, fibrosis, and cirrhosis. Dietary factors, particularly carbohydrate quality, play a significant role in its development and progression.

Fructose metabolism and the liver

Fructose is a sugar present naturally in fruits, honey, and some vegetables. It is also widely used in processed foods and sugar-sweetened beverages in the form of sucrose and high-fructose corn syrup. Unlike glucose, fructose is primarily metabolized in the liver and bypasses several regulatory steps that normally control carbohydrate metabolism.

When consumed in excess, fructose promotes de novo lipogenesis, a process through which carbohydrates are converted into fatty acids. This leads to the accumulation of triglycerides within liver cells, contributing to hepatic steatosis. Excess fructose intake has also been associated with oxidative stress, inflammation, insulin resistance, and increased production of uric acid, all of which can accelerate liver damage

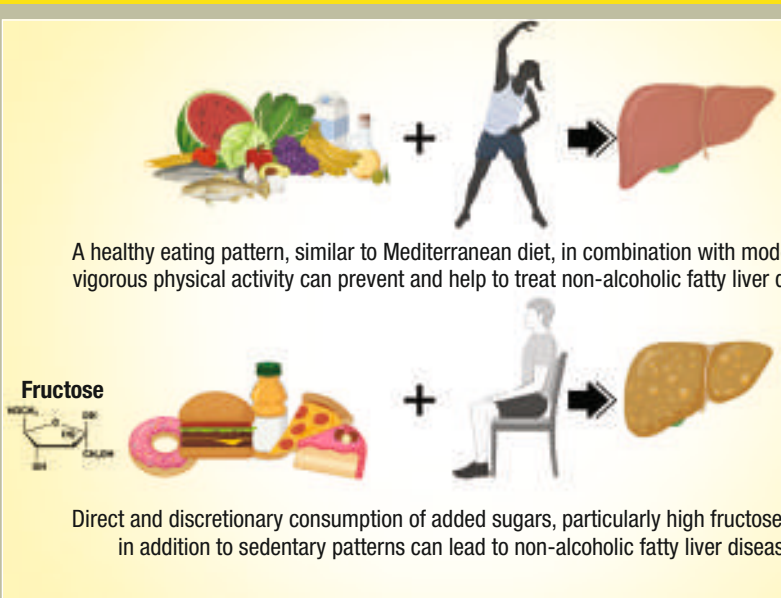

Fig 2: Dietary patterns, fructose intake, and liver health
(Ref: Lujan LML et al.,2025)

Fructose and disease progression

Fructose is an important contributor to the development of MASLD. Persistent liver fat accumulation, combined with oxidative stress and inflammatory pathways, may promote fibrosis, cirrhosis, and in advanced cases, hepatocellular carcinoma. Recent prospective cohort studies in individuals with cirrhosis reported that higher intakes of total carbohydrates, fructose, glucose, and sucrose were associated with an increased risk of mortality, highlighting the potential impact of carbohydrate quality on liver-related outcomes.

Dietary considerations

While carbohydrates remain an essential component of a healthy diet, the quality of carbohydrate intake plays an important role in liver health. Emphasis should be placed on whole grains, legumes, fruits, vegetables, and other fibre-rich sources, while limiting added sugars, sugar-sweetened beverages, and highly processed foods. The widespread availability of fructose-rich processed foods and sweetened beverages has further increased exposure to these metabolic risks.

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Beyond Liver Enzymes: Non-invasive Diagnosis of Metabolic Dysfunction-associated Steatotic Liver Disease (MASLD)

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MASLD affects nearly 30% of adults worldwide and is strongly linked to obesity, type 2 diabetes mellitus (T2DM), and insulin resistance. Early detection and fibrosis staging are essential to prevent progression to liver failure and hepatocellular carcinoma (HCC). Non-invasive tests (NITs) are now widely used as safer, accessible, cost effective and repeatable alternatives to liver biopsy for their use in MASLD diagnosis and monitoring.

Fibrosis-4 (FIB-4): It is a widely used blood-based marker for MASLD that estimates fibrosis risk using age, aspartate aminotransferase (AST), alanine aminotransferase (ALT), and platelet count. With a high negative predictive value for advanced fibrosis, it is recommended as an initial screening tool, especially in individuals with obesity and T2DM.

Enhanced liver fibrosis (ELF) test: The ELF test measures direct fibrosis markers such as hyaluronic acid, amino-terminal propeptide of type III procollagen (PIIINP), and tissue inhibitor of metalloproteinases 1 (TIMP-1), providing a more accurate assessment of advanced fibrosis than indirect scores like FIB-4. It is commonly used when initial non-invasive tests are indeterminate, although reduced specificity in low-prevalence settings may lead to false positives.

Vibration-controlled transient elastography (VCTE): VCTE/Fibro scan is a non-invasive imaging tool that measures liver stiffness and steatosis, making it valuable for fibrosis assessment in MASLD. Recommended by the American Association for the Study of Liver Diseases and European Association for the Study of the Liver, it has high accuracy for advanced fibrosis, though its performance may be limited in obesity or ascites.

Magnetic resonance elastography (MRE) is the most accurate non-invasive imaging technique for assessing liver fibrosis, particularly early-stage disease and fibrosis in individuals with obesity. Although superior to VCTE, its high cost and limited availability restrict routine use, and the American Association for the Study of Liver Diseases and European Association for the Study of the Liver recommend it mainly for inconclusive cases.

NITs play a central role in MASLD management by enabling early fibrosis detection, risk stratification, and monitoring of disease progression and treatment response. Combining serum biomarkers with imaging techniques improves diagnostic accuracy, reduces unnecessary biopsies, and supports timely, personalized patient care.

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In T2DM with Comorbidities,

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Abridged Prescribing Information

Indication: It is indicated as an adjunct to diet and exercise to improve glycaemic control in adults with type 2 diabetes mellitus.
Dosage and Administration: The recommended dose is one tablet daily. Each tablet contains a fixed dose of dapagliflozin, Sitagliptin and Metformin Hydrochloride.
Adverse Reactions: Most common adverse reactions reported are: Dapagliflozin- Female genital mycotic infections, nasopharyngitis, and urinary tract infections. Sitagliptin- Upper respiratory tract infection, nasopharyngitis and headache. Metformin- Diarrhea, nausea/vomiting, flatulence, asthenia, indigestion, abdominal discomfort, and headache.
Warnings and Precautions: Dapagliflozin: Volume depletion; Ketoacidosis in Patients with Diabetes Mellitus; Urosepsis and Pyelonephritis; Hypoglycaemia; Genital Mycotic Infections
Sitagliptin: General- Sitagliptin should not be used in patients with type 1 diabetes or for the treatment of diabetic ketoacidosis. Acute pancreatitis: Hypoglycaemia when used in combination with other anti-hyperglycaemic medicinal product; Renal impairment; Hypersensitivity reactions including anaphylaxis, angioedema, and exfoliative skin conditions- Stevens-Johnson syndrome; Bullous pemphigoid. Metformin Hydrochloride: Lactic acidosis; In case of dehydration (severe diarrhoea or vomiting, fever or reduced fluid intake), metformin should be temporarily discontinued and contact with a healthcare professional is recommended.
Contraindications: Hypersensitivity to the active substance of Dapagliflozin, Sitagliptin & Metformin or to any of the excipients listed. Any type of acute metabolic acidosis (such as lactic acidosis, diabetic ketoacidosis). Diabetic pre-coma; Severe renal failure (eGFR<30ml/min); Acute conditions with the potential to alter renal function such as: Dehydration, Severe Infection, Shock; Acute or chronic disease which may cause tissue hypoxia such as: Cardiac or respiratory failure, Recent myocardial Infarction, Shock, Hepatic Impairment, Acute Alcohol Intoxication, alcoholism
Use in a special population: Pregnant Women: Due to lack of human data, drug should not be used during pregnancy. Lactating Women: It should not be used during breastfeeding. Paediatric Patients: The safety and efficacy of drug has not yet been established. No data are available. Geriatric Patients: In Patients > 65 years, it should be used with caution as age increases.

Additional information is available on request.
Last updated: March 2026

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Corvette Team

Interview of the Month with Dr. Gaurav Patil

Dr. Gaurav Patil

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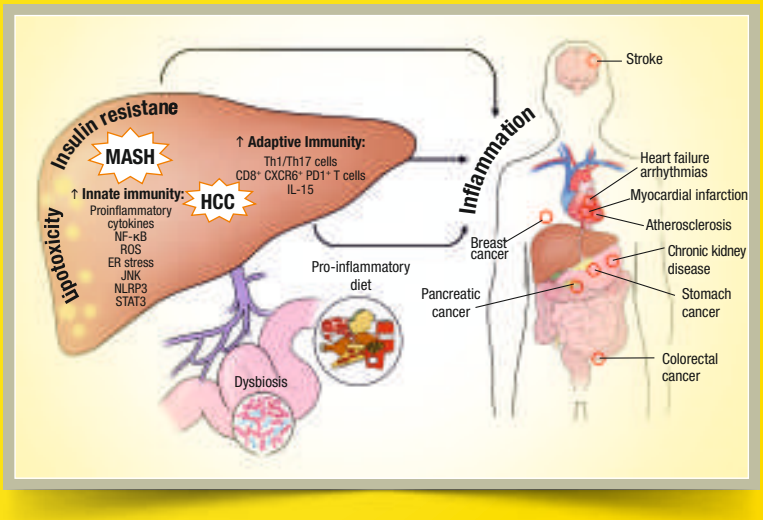
Dr. Gaurav Patil is the Director of Gastroenterology and Advanced Endoscopy at Nanavati Max Super Speciality Hospital, Mumbai, and is widely recognized for his expertise in the diagnosis and treatment of complex gastrointestinal, liver, pancreatic, and biliary disorders. With extensive experience in advanced therapeutic endoscopy, he specializes in minimally invasive procedures that offer precise diagnosis and effective treatment with faster recovery. Dr. Patil is known for his patient-centric approach, combining clinical excellence with compassionate care to deliver the best possible outcomes. He is actively involved in academic and research activities and is committed to advancing the field of gastroenterology through innovation, education, and the adoption of cutting-edge technologies in digestive healthcare.

Integrated Liver and Metabolic Care

1. Why should metabolic dysfunction-associated steatotic liver disease (MASLD) be considered a metabolic disease rather than just a liver disease?



MASLD is far more than a hepatic condition, it is a systemic metabolic disorder that reflects deep-rooted dysregulation of insulin signalling, lipid metabolism, and inflammatory pathways throughout the body. The liver is, in many ways, the mirror of metabolic health. When we see fat accumulating in hepatocytes, it is a visible manifestation of years of metabolic stress — insulin resistance, visceral adiposity, dyslipidemia, and chronic low-grade inflammation.

The renaming from NAFLD to MASLD in 2023 was not merely a semantic shift; it was a conceptual realignment. It acknowledged that the condition is almost always embedded within a cluster of cardiometabolic risk factors. Patients with MASLD carry a significantly elevated risk of type 2 diabetes, cardiovascular disease, chronic kidney disease, and even certain malignancies — risks that extend well beyond the liver. Treating MASLD in isolation, without addressing the underlying metabolic milieu, leads to

incomplete and often disappointing outcomes. A metabolic lens, therefore, is not optional it is essential for meaningful clinical management and long-term patient benefit.

2. How can early identification of MASLD be improved in routine practice?

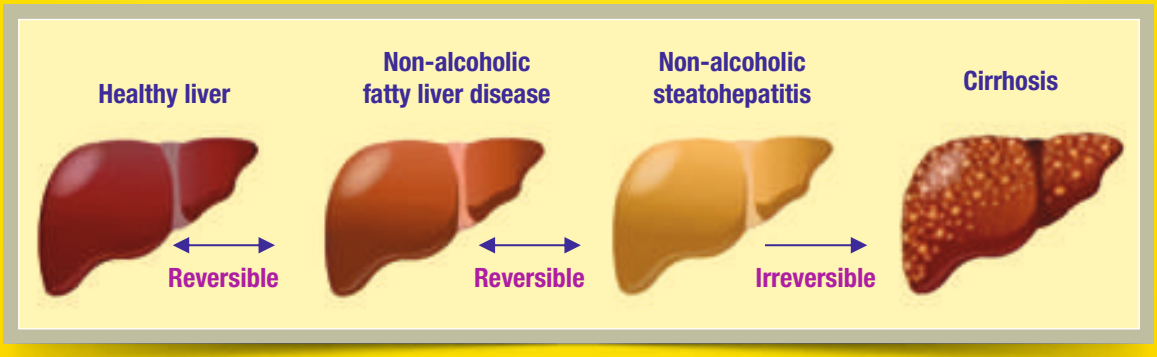
The greatest challenge with MASLD is its silent nature. Most patients are asymptomatic until the disease has progressed to an advanced fibrotic stage, by which point therapeutic options are more limited. Early identification must therefore be proactive and systematic, not reactive.

In routine practice, the first step is raising clinical suspicion in all patients presenting with obesity, type 2 diabetes, hypertension, or dyslipidemia — the metabolic quartet that almost invariably accompanies MASLD. A simple abdominal ultrasound, combined with liver function tests, can serve as a first-line screening tool. However, we must go further. Non-invasive fibrosis scores such as the FIB-4 index and the NAFLD fibrosis score are underutilised, inexpensive, and easily integrated into routine consultations. Incorporating FIB-4 into standard metabolic workups for high-risk patients could substantially improve early case detection.

Beyond tools and technology, the key lies in educating primary care physicians, diabetologists, and endocrinologists to think of the liver as part of their remit. A paradigm shifts from organ-specific to systems-based care is the cornerstone of earlier diagnosis.

3. Is fatty liver reversible? If yes, upto which stage and how?

This is one of the most encouraging aspects of MASLD. Yes, it is reversible, and the extent of reversibility depends on the stage at which intervention begins. Simple hepatic steatosis and even early steatohepatitis (MASH) are significantly reversible with lifestyle modification and metabolic optimisation. Clinical studies have demonstrated that a sustained weight loss of 7–10% of body weight can lead to meaningful histological improvement, including reduction of steatosis, resolution of inflammation, and even regression of early fibrosis. Fibrosis up to stage F2 (moderate fibrosis) is considered substantially reversible with consistent lifestyle and pharmacological interventions. Even F3 (advanced fibrosis) may show partial regression with aggressive management. However, cirrhosis (F4) is largely irreversible, though progression can be halted and portal hypertension-related complications can be mitigated. The how is equally important — it encompasses structured dietary change (Mediterranean diet has the strongest evidence), regular aerobic and resistance exercise, alcohol cessation, treatment of comorbid diabetes and dyslipidemia, and increasingly, pharmacotherapy.



4. What are the emerging therapies in improving liver and metabolic health?

The therapeutic journey for MASH has evolved meaningfully — from early agents to transformative, multi-dimensional therapies. Saroglitazar, a dual PPAR-α/γ agonist developed in India, was a significant early step — addressing both hepatic and metabolic dimensions by improving insulin sensitivity, reducing hepatic fat, and favorably modulating dyslipidemia. It remains clinically relevant, particularly in patients with coexisting hypertriglyceridemia and insulin resistance. Resmetirom, the first FDA-approved drug for MASH with fibrosis (2024), selectively enhances hepatic fat oxidation without systemic thyroid effects, achieving both MASH resolution and fibrosis improvement in the MAESTRO-NASH trial. GLP-1 receptor agonists such as semaglutide have demonstrated compelling data not only in weight reduction and glycemic control but also in reducing hepatic steatosis and inflammation. The ESSENCE trial data for semaglutide in MASH has generated considerable optimism. GLP-1/GIP dual agonists like tirzepatide are showing even more pronounced metabolic and hepatic benefits in emerging trial data. FGF21 analogues and pan-PPAR agonists like lanifibranor are demonstrating compelling anti-fibrotic effects in phase 3 trials. The trajectory is clear — from Saroglitazar's integrated approach to today's agents that simultaneously target weight, glycemia, dyslipidemia, inflammation, and fibrosis. The future lies in combination therapy strategies, and for the first time, genuine optimism in MASH management is warranted.

5. What are the most important priorities for improving liver and metabolic health at a population level?

Addressing MASLD at a population level demands a multi-pronged, intersectoral strategy — one that goes beyond the clinic and into policy, education, and the built environment. Firstly, metabolic health must be integrated into national health programmes. MASLD screening should be embedded within existing platforms such as diabetes and cardiovascular prevention programmes. In India, where the dual burden of diabetes and MASLD is particularly pronounced, this integration is both logical and urgent. Additionally, public health literacy around the liver-metabolism connection needs significant strengthening. Most people are unaware that a fatty liver is a metabolic warning sign. Targeted awareness campaigns — especially in urban and peri-urban populations with high rates of sedentary behavior and processed food consumption — can enable earlier self-referral and diagnosis. Further dietary transitions must be addressed at a structural level. Policies that incentivise consumption of whole grains, vegetables, legumes, and healthy fats and disincentivise ultra-processed foods and sugar-sweetened beverages are essential. The food environment shapes metabolic health in ways that individual counselling alone cannot correct. Also physical activity infrastructure must be democratised. Access to safe walking spaces, parks, and affordable fitness facilities must be a public health priority, not a luxury. Finally, training healthcare providers across specialities from general practitioners to diabetologists — to screen for and co-manage MASLD will be transformative. Metabolic health is a shared responsibility across medicine, and liver health must find its rightful place at the centre of that conversation.

Drug Spotlight: SGLT2 Inhibitors

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An essential family of medications for the treatment of diabetes, sodium-glucose cotransporter-2 (SGLT2) inhibitors provide both cardiovascular and renal protection in addition to glycemic control.

Name of the drug

Empagliflozin, Dapagliflozin, and Canagliflozin.

Action mechanism

Approximately 90% of the filtered glucose in the kidneys is reabsorbed by SGLT2 transporters. Inhibition of SGLT2 transporters in the proximal renal tubules reduces renal glucose reabsorption, thereby increasing urinary glucose excretion. This leads to lower blood glucose levels and promotes natriuresis and also osmotic diuresis. As a result, SGLT2 inhibitors help reduce blood pressure, promote weight loss, and improve glycemic control.

Various forms

SGLT2 inhibitors come in the following forms:

- Oral medications
- Fixed dose combos with dipeptidyl peptidase-4 (DPP-4) inhibitors, metformin or other anti-hyperglycemic medications

Advantages	Things to reconsider
0.5%-1.0% decrease in glycated hemoglobin (HbA1c) and reduced systolic blood pressure	Genital mycotic infection risk
Support weight loss	Euglycemic diabetic ketoacidosis is an uncommon danger.
Lowering the risk of cardiovascular disease events and decrease in heart failure hospitalisations	Depletion of volume in vulnerable people
Reduces albuminuria and the rate at which chronic kidney disease advances	Postural hypotension in elderly

Drug snapshot

Drug group	Specific drug	Effects	Adverse effects	Contraindications
SGLT2 inhibitors	Gliflozin	Glycemic control Renal protective Cardio protective	Euglycemic diabetic ketoacidosis Genital mycotic infection risk	Safe prescription requires careful customisation based on renal status

Nutrition-drug related key takeaways

- Encourage adequate hydration while using SGLT2 inhibitors.
- Nutritional counselling should include sick-day management education.
- Moderate carbohydrate intake supports safe glycemic management during therapy.

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Trending Diets: Time-restricted Eating in Metabolic Dysfunction-associated Steatotic Liver Disease (MASLD)

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Lifestyle modification remains the cornerstone of MASLD management, with weight loss being the primary therapeutic target. Among the various dietary approaches gaining popularity, time-restricted eating (TRE), has emerged as a promising strategy.

TRE focuses on limiting food intake to a specific daily eating window (typically 6–10 hours) while maintaining a prolonged fasting period during the remaining hours. Unlike traditional calorie-restricted diets, TRE emphasizes when food is consumed rather than what is consumed.

How does TRE benefit MASLD?

Several mechanisms may explain the beneficial effects of TRE in MASLD:

- Promotes weight loss and reduction in visceral adiposity.
- Improves insulin sensitivity and reduces hyperinsulinemia.
- Enhances hepatic fat oxidation and reduces de novo lipogenesis.
- Aligns food intake with circadian rhythms, optimizing metabolic regulation.
- May reduce hepatic steatosis

What does the evidence show?

Recent evidence suggests that TRE can significantly improve liver fat content in individuals with MASLD. A study by *Sharma S et.al.* (2025), reported that time-restricted eating over 12 weeks led to greater improvement in insulin resistance compared to hypocaloric diet alone in individuals with MASLD. A systematic review and network meta-analysis of randomized controlled trials further reported that intermittent fasting strategies, including time-restricted feeding, can improve hepatic steatosis, body weight and low-density lipoprotein (LDL)-cholesterol levels in individuals with MASLD.

Practical recommendations

For individuals with MASLD, TRE may be considered as a strategy to support liver health. Common approaches include:

- 8-hour eating window (e.g., 10 am–6 pm)
- 10-hour eating window (e.g., 8 am–6 pm)
- Avoiding late-night eating
- Prioritizing nutrient-dense and healthy foods during eating periods
- Maintaining adequate protein intake and hydration

Key takeaway

Time-restricted feeding is an emerging dietary strategy that may help reduce liver fat, improve metabolic health, and support weight management in people with MASLD. Current evidence suggests that its benefits are comparable to traditional calorie restriction, making it a practical and potentially sustainable option for long-term lifestyle management. However, dietary quality and overall caloric intake remain important determinants of success.

Resources:

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Gut–liver Axis in Metabolic Liver Disease

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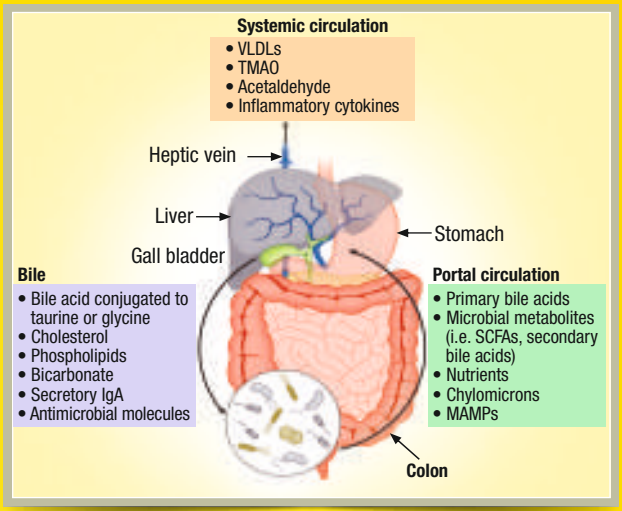
The gut–liver axis refers to the two-way communication network between the gut (including its microbiota) and the liver, shaped by signals influenced by diet, genetics, and environmental factors. Disruption of the intestinal barrier allows bacterial products to reach the liver via the portal circulation, contributing to the development or progression of liver diseases. The gut and liver communicate through the portal vein, which carries gut products to the liver, and through bile acids and antibodies that flow back to the intestine. Bile acids also help regulate the gut microbiota. This interaction is controlled by the gut lining, which acts as a barrier between microbes and the internal system.



The role of gut microbes in liver diseases such as alcohol-related liver disease (ALD) and metabolic dysfunction-associated steatotic liver disease (MASLD) has been recognized for a long time. Chronic alcohol consumption disrupts the gut microbiome, reducing microbial diversity and altering the abundance of key bacterial and fungal species. These changes promote the translocation of microbial products into the circulation, triggering immune activation and contributing to the development and progression of alcohol-related liver disease. The disruption of the gut–liver axis contributes to liver disease progression through several interconnected mechanisms.

Impaired intestinal barrier

In liver conditions, damage to the gut barrier increases intestinal permeability, allowing bacterial components like lipopolysaccharide (LPS) (endotoxin) and other microbial metabolites to enter the liver through the portal circulation. This triggers inflammation that worsens liver injury. The number of gut bacteria influences how many of these inflammatory signals reach the liver, affecting disease severity. As chronic liver disease progresses, increasing damage to gut defences further weakens the barrier and aggravates the condition.

Hyperactivation of immune cells

In the liver, LPS activates immune receptors which triggers inflammatory pathways and the release of cytokines that drive liver injury and fibrosis. Beyond the liver, this endotoxin-driven signalling also extends to peripheral tissues, amplifying systemic inflammation. Similar inflammatory effects are also seen in adipose tissue, linking gut-derived endotoxins to obesity-related inflammation. Overall, this gut-driven immune activation plays a key role in the development and progression of fatty liver disease.

Metabolites derived from the gut microbiome

Gut microbiome metabolites are small molecules produced during microbial activity. They come from bacterial processes, food breakdown, or changes to host molecules like bile acids. In dysbiosis, these metabolites can disturb immune balance, affect metabolism, and weaken gut barrier function, leading to loss of normal gut homeostasis.

Bile acid signalling disruption

Bile acids are made from cholesterol in the liver and released into the intestine to help digest fats. Beyond digestion, they also act as important messengers between the gut and liver, helping control metabolism and inflammation. They work mainly through two receptors which help regulate bile acid production, sugar and fat metabolism, and immune responses. When this system is disturbed, it is linked to conditions like fatty liver disease, diabetes, and inflammatory liver diseases. Gut bacteria also modify bile acids into different forms, which can either reduce or increase inflammation, showing how closely diet, microbes, and liver health are connected.

Dietary implications

Diet plays a central role in modulating the gut–liver axis in MASLD, primarily by shaping gut microbiota composition, intestinal barrier integrity, and microbial metabolite production. Diets high in refined carbohydrates, saturated fats, and ultra-processed foods promote dysbiosis, increased intestinal permeability, and greater translocation of microbial products such as LPS into the portal circulation, thereby amplifying hepatic inflammation. In contrast, dietary patterns rich in fibre, polyphenols, and unsaturated fats support a balanced microbiome, enhance short-chain fatty acid (SCFA) production, and strengthen gut barrier function, collectively reducing metabolic inflammation. Thus, the gut–liver axis represents a central pathway in the development and progression of metabolic liver diseases. Overall, disruption of the gut–liver axis plays a central role in the pathogenesis and progression of metabolic liver diseases through interconnected microbial, immune, and metabolic pathways.

Resources:

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Diabetes Educator Toolkit:
Skill of the Month: Motivational interviewing (MI)

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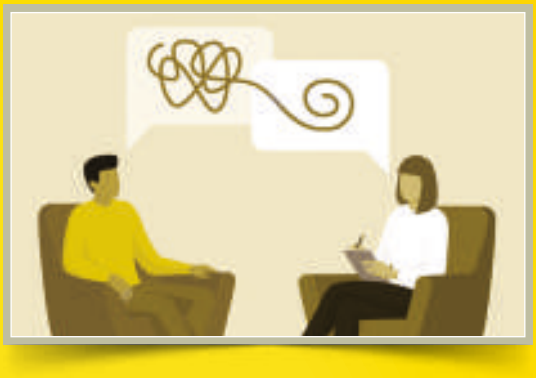
Effective diabetes management requires more than providing information. It involves supporting individuals in making and sustaining behavior changes. MI is an evidence-based counselling approach that helps diabetes educators engage individuals in meaningful conversations about their health. By strengthening intrinsic motivation and self-efficacy, MI can improve adherence to lifestyle modifications, medication regimens, and overall diabetes self-care. MI is based on the following key principles that enhance individual's motivation and adherence to treatment regimen.



1. **Empathy:** MI emphasizes creating a supportive, non-judgmental environment. Through reflective listening and acknowledgment of individual's feelings and experiences, healthcare professionals build trust, rapport, and a therapeutic partnership.
2. **Developing discrepancy:** This principle helps individuals recognize the gap between their current behaviors and their health goals. By increasing awareness of this discrepancy, MI encourages motivation for positive behavior change and improved diabetes management.
3. **Supporting self-efficacy:** MI strengthens an individual's confidence in their ability to successfully perform self-care behaviors. By fostering self-belief and highlighting past successes, healthcare professionals can promote sustained adherence to treatment recommendations.
4. **Rolling with resistance:** Rather than confronting resistance directly, MI encourages healthcare professionals to accept and explore it as a natural part of the change process. This approach helps guide individuals toward self-motivated solutions without judgment or pressure.
5. **Promoting autonomy:** MI respects an individual's right and capacity to make their own decisions. By supporting autonomy and shared decision-making, healthcare professionals empower people with diabetes to take ownership of their health and treatment plans.

Resources:

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Frequently Asked Questions on Integrated Liver and Metabolic Care

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1. I am a 42-year-old businessman with metabolic dysfunction-associated steatotic liver disease (MASLD). I hardly eat oily food, so I don't understand why my liver is affected. Could my daily habits still be causing fatty liver?

Answer: Fatty liver is not caused only by oily or fried foods, but excess sugar and refined carbohydrates can also play a major role in fat build-up in the liver. Frequent intake of sugary foods, refined carbohydrates, sweetened beverages, bakery products, and processed snacks increases glucose levels in the body. When excess sugar is not used for energy, the liver converts it into fat and stores it within the liver cells over time.

Some important factors that contribute to fatty liver include:

- Sugary foods and refined carbohydrates: Excess sugar gets converted into fat and stored in the liver.
- Long sitting hours and low physical activity: These worsen insulin resistance and increase fat accumulation.
- Poor sleep and chronic stress: Both affect metabolism and promote abdominal and liver fat storage.
- Many people with fatty liver may not eat very oily food but still develop MASLD because of unhealthy metabolic and lifestyle habits.

Note: The given image is AI generated and is used for illustrative purpose only

Reducing sugary and processed foods, improving sleep, and increasing daily activity can significantly improve liver health and help reverse early fatty liver.

2. I am a 50-year-old man with central obesity, high triglycerides, and prediabetes. I have been recently diagnosed with grade I fatty liver, but I feel completely normal. Is fatty liver really serious enough to worry about?

Answer: Fatty liver is often called a "silent" condition because many people do not experience symptoms in the early stages. However, even without symptoms, it can be a warning sign that the body is developing metabolic problems such as insulin resistance, diabetes, and increased cardiovascular risk.

- Central obesity, high triglycerides, and prediabetes are all closely connected with fatty liver.
- When excess fat starts getting stored around the abdomen and inside the liver, it affects how insulin works in the body.
- Over time, this can progress from simple fat accumulation to liver inflammation, scarring, and higher risk of heart disease if left untreated.

Long sitting hours, skipping breakfast, and eating large late-night meals further worsen insulin resistance and fat storage in the liver. However, fatty liver at an early stage can often be reversed with timely lifestyle changes and weight management.

3. I am a 40-year-old bank employee with MASLD. Because of my irregular work schedule and stress-related eating habits, I find lifestyle changes difficult. Can medication alone cure fatty liver?

Answer: Medication alone is usually not enough to reverse fatty liver. The most effective treatment for MASLD is lifestyle modification because the condition is strongly linked to eating patterns, physical inactivity, stress, poor sleep, and weight gain. Medicines may help manage associated conditions like diabetes, obesity, or high cholesterol, but they work best along with lifestyle changes. Small and practical steps are more sustainable such as:

- Try to avoid long gaps between meals, as this often leads to overeating later in the day.
- Keeping simple healthy snacks like nuts, roasted chana, fruit, or buttermilk at work can help reduce stress eating and dependence on packaged foods.
- Even short periods of movement during office hours, such as 5-10 minutes of walking after meals or in between calls, taking stairs instead of elevators can be helpful.
- Meal timing also matters; late-night heavy dinners should be reduced as much as possible.
- Stress and sleep should not be ignored, as both directly affect appetite, cravings, and fat storage.

The goal should be gradual, realistic, and consistent changes rather than strict diets. With steady efforts, fatty liver can be reversed.

Note: The given image is AI generated and is used for illustrative purpose only

Ginger: A Monsoon Superfood for Immunity

The monsoon season brings welcome relief from the summer heat but also increases the risk of respiratory infections, common colds, flu-like illnesses, and digestive disturbances. Ginger (Zingiber officinale) is a traditional medicinal spice that has long been valued for its immune-supportive properties and is considered a valuable addition to the diet during this season.

Antioxidant, anti-inflammatory, and immunomodulatory effects: Ginger contains several bioactive compounds, including gingerols, shogaols, paradols, and zingerone, which exhibit potent antioxidant, anti-inflammatory, and immuno-modulatory effects. These compounds help combat oxidative stress and regulate inflammatory pathways that can weaken immune defences.

Anti-microbial effects: Fresh ginger has also demonstrated antimicrobial and antiviral properties in experimental studies. Its active constituents may help inhibit the growth of certain pathogens and support respiratory health, making it particularly useful during the monsoon season when respiratory infections are more common. Additionally, ginger's warming properties may help relieve sore throat, nasal congestion, and cough symptoms often associated with seasonal illnesses.

Digestive health: Beyond immunity, ginger supports digestive health by reducing nausea, improving gastric motility, and alleviating gastrointestinal discomfort. Since monsoon-related infections can affect both respiratory and digestive systems, regular consumption of ginger tea, ginger-infused water, adding ginger to soups, can provide multiple health benefits.

Incorporating ginger as part of a balanced diet may therefore serve as a simple, natural strategy to support immune resilience and overall well-being during the monsoon season. While ginger is not a substitute for medical treatment, its scientifically recognized antioxidant and immuno-modulatory properties make it a true monsoon superfood.

Resources:

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Role Play

Scenario: Ms. ABC, a 38-year-old school teacher, comes to the clinic feeling confused and anxious after being diagnosed with metabolic dysfunction-associated steatotic liver disease (MASLD) during a routine health check-up. She wants to understand that when she hasn't been drinking alcohol then how she can develop fatty liver. She has a sedentary lifestyle, irregular eating habits, central obesity, and recently diagnosed with prediabetes.

Ms. ABC: Hello, doctor! I'm surprised and feel worried with my recent diagnosis of MASLD. I don't consume alcohol then how can I have fatty liver?

Diabetes educator (DE): I understand your concerns. But MASLD can occur even with no alcohol consumption. Fat builds up in your liver due to excess body weight, particularly, central obesity, unhealthy eating habits, elevated blood glucose levels and lack of physical activity.

Ms. ABC: Okay. Can you explain how prediabetes and central obesity are related with MASLD?

DE: Sure. Both prediabetes and increased waist circumference (abdominal obesity) are linked with MASLD through insulin resistance. In prediabetes, the body's cells do not respond effectively to insulin, which can cause increased fat accumulation in the liver. Central/ abdominal obesity, particularly excess visceral fat around the abdomen region, promotes inflammation and worsens insulin resistance. These factors together increase accumulation of fat in the liver and contribute to the development and progression of MASLD.

Ms. ABC: Alright. What dietary modifications do I need to follow to manage MASLD?

DE: Achieving or maintaining a healthy weight and eating a balanced diet at regular intervals is important in managing MASLD. Opt for a healthy plate by filling half of your plate with non-starchy vegetables, one-fourth with lean protein and one-fourth with whole grains and cereals. Avoid fried and processed foods, refined carbohydrates and sugary drinks, as these foods may worsen fat accumulation. Opt for healthy fats like nuts, seeds, avocados and fatty fish in the diet. In addition to eating healthy, engaging in daily physical activity is essential in decreasing abdominal fat and managing MASLD.

Ms. ABC: Understood. What physical activities do you suggest?

DE: Regular physical activity can help you manage your weight, help reduce liver fat and manage conditions associated with MASLD, such as prediabetes, by improving insulin sensitivity. For better results, opt for moderate intensity physical activity like brisk walking 30 minutes, 5 days a week. Any movement counts, walking on the school premises or taking the stairs can also make a significant contribution.

Ms. ABC: Thank you for the detailed insight.

DE: You're welcome.

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Metformin Hydrochloride 500 mg SR + Glimepiride 1 mg

Glycomet®-GP 2

Metformin Hydrochloride 500 mg SR + Glimepiride 2 mg

Glycomet®-GP 2/850

Metformin Hydrochloride 850 mg SR + Glimepiride 2 mg

Glycomet®-GP 3/850

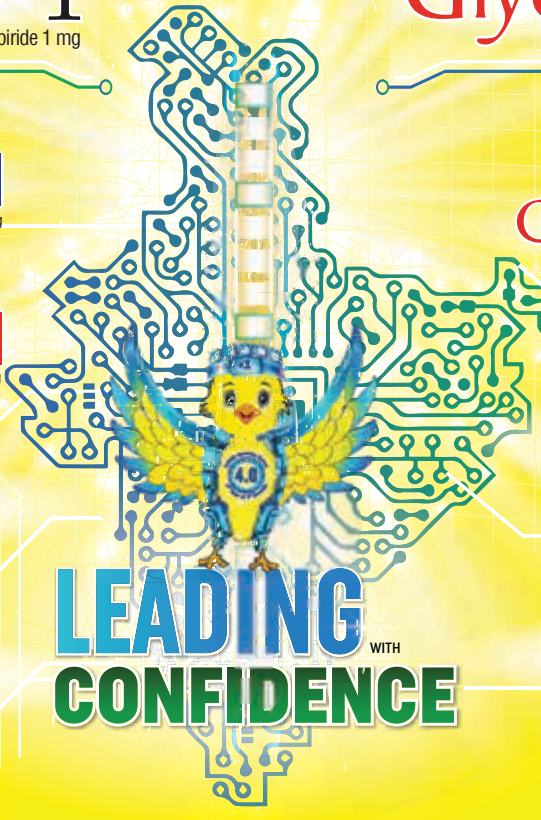
Metformin Hydrochloride 850 mg SR + Glimepiride 3 mg

Glycomet®-GP 3 FORTE

Metformin Hydrochloride 1000 mg SR + Glimepiride 3 mg

Glycomet®-GP 4 FORTE

Metformin Hydrochloride 1000 mg SR + Glimepiride 4 mg



LEADING WITH CONFIDENCE

Glycomet®-GP 0.5 FORTE

Metformin Hydrochloride 1000 mg SR + Glimepiride 0.5 mg

Glycomet®-GP 1 FORTE

Metformin Hydrochloride 1000 mg SR + Glimepiride 1 mg

Glycomet®-GP 2 FORTE

Metformin Hydrochloride 1000 mg SR + Glimepiride 2 mg

Abridged Prescribing Information

Active Ingredients: Metformin hydrochloride (as sustained release) and glimepiride tablets Indication: For the management of patients with type 2 diabetes mellitus when diet, exercise and single agent (glimepiride or metformin alone) do not result in adequate glycaemic control. Dosage and Administration: The recommended dose is one tablet daily during breakfast or the first main meal. Each tablet contains a fixed dose of glimepiride and Metformin Hydrochloride. The highest recommended dose per day should be 8 mg of glimepiride and 2000mg of metformin. Due to prolonged release formulation, the tablet must be swallowed whole and not crushed or chewed. Adverse Reactions: For Glimepiride: hypoglycaemia may occur, which may sometimes be prolonged. Occasionally, gastrointestinal (GI) symptoms such as nausea, vomiting, sensations of pressure or fullness in the epigastrium, abdominal pain and diarrhea may occur. Hepatitis, elevation of liver enzymes, cholestasis and jaundice may occur; allergic reactions or pseudo allergic reactions may occur occasionally. For Metformin: GI symptoms such as nausea, vomiting, diarrhea, abdominal pain, and loss of appetite are common during initiation of therapy and may resolve spontaneously in most cases. Metallic taste, mild erythema, decrease in Vit B12 absorption, very rarely lactic acidosis, Hemolytic anemia, Reduction of thyrotropin level in patients with hypothyroidism, Hypomagnesemia in the context of diarrhea, Encephalopathy, Photosensitivity, hepatobiliary disorders. Warnings and Precautions:: For Glimepiride: Patient should be advised to report promptly exceptional stress situations (e.g., trauma, surgery, febrile infections), blood glucose regulation may deteriorate, and a temporary change to insulin may be necessary to maintain good metabolic control. Metformin Hydrochloride may lead to Lactic acidosis; in such cases metformin should be temporarily discontinued and contact with a healthcare professional is recommended. Sulfonylureas have an increased risk of hypoglycaemia. Long-term treatment with metformin may lead to peripheral neuropathy because of decrease in vitamin B12 serum levels. Monitoring of the vitamin B12 level is recommended. Overweight patients should continue their energy-restricted diet, usual laboratory tests for diabetes monitoring should be performed regularly. Contraindications: Hypersensitivity to the active substance of glimepiride & Metformin or to any of the excipients listed. Any type of acute metabolic acidosis (such as lactic acidosis, diabetic ketoacidosis, diabetic pre-coma). Severe renal failure (GFR<30ml/min). In pregnant women. In lactating women.Acute conditions with the potential to alter renal function (dehydration, severe infection, shock, intravascular administration of iodinated contrast agents); acute or chronic disease which may cause tissue hypoxia (cardiac or respiratory failure, recent myocardial infarction, shock); hepatic insufficiency; acute alcohol intoxication; alcoholism. Use in a special population: Pregnant Women: Due to a lack of human data, drugs should not be used during pregnancy. Lactating Women: It should not be used during breastfeeding. Pediatric Patients: The safety and efficacy of drugs has not yet been established. Renal impairment: A GFR should be assessed before initiation of treatment with metformin containing products and at least annually thereafter. In patients at increased risk of further progression of renal impairment and in the elderly, renal function should be assessed more frequently, e.g. every 3-6 months. Additional information is available on request.
Last updated: March 13, 2023

In case of any adverse events, kindly contact: pv@usv.in For the use of registered medical practitioner, hospital or laboratory.

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Corvette Team

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