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Review Article

Gluten-free Diet and Metabolic Dysfunction-associated Steatotic Liver Disease: Mechanisms and Therapeutic Potential

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Abstract

Metabolic dysfunction-associated steatotic liver disease (MASLD) is a chronic liver disease associated with obesity, insulin resistance, and metabolic syndrome and has become a major global health challenge. Current management relies primarily on comprehensive lifestyle interventions, including dietary modification, regular physical activity, weight loss, management of metabolic risk factors, and emerging pharmacotherapies. The gluten-free diet (GFD) was originally developed for the treatment of celiac disease and has attracted attention in recent years for its potential metabolic benefits. Existing studies suggest that gluten- or wheat-targeted dietary interventions may affect MASLD-related pathways, including the gut microbiota, inflammation, insulin sensitivity, and liver fat accumulation. In patients with concurrent celiac disease and MASLD, a GFD is indicated for celiac disease, but its hepatic and metabolic effects are variable. However, clinical and mechanistic evidence for its potential effects in patients with non-celiac MASLD is limited and heterogeneous, relying largely on indirect evidence from animal models; furthermore, long-term, unsupervised adherence to a GFD may adversely affect metabolic health. This narrative review critically evaluates the therapeutic boundaries of gluten-targeted interventions, compares the gluten-free diet with established dietary patterns, and outlines challenges related to nutritional adequacy, cost, and adherence. Future research should focus on high-quality randomized controlled trials to identify the specific benefits and risks of gluten removal in patients with non-celiac MASLD and to inform more precise dietary guidance.

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Introduction

Metabolic dysfunction-associated steatotic liver disease (MASLD) is characterized by excessive fat accumulation in the liver and has become the most common chronic liver disease worldwide, affecting about one-third of adults.[1–6] Its prevalence varies across regions and is associated with obesity rates, metabolic syndrome, and lifestyle factors.[6–14] As shown in Figure 1, MASLD is a complex disease influenced by genetic factors, environmental exposures, and metabolic disorders such as type 2 diabetes and dyslipidemia.[13] MASLD is fundamentally a multisystem disorder. In addition to driving steatohepatitis and cirrhosis, it is frequently associated with extrahepatic complications such as cardiovascular disease, type 2 diabetes, and chronic kidney disease.[15–18] Management therefore requires a combination of lifestyle modifications—diet, exercise, and weight loss—metabolic risk-factor control, and emerging pharmacotherapies.[19–22] In this review, MASLD is used as the preferred term; older terms such as non-alcoholic fatty liver disease (NAFLD) and metabolic dysfunction-associated fatty liver disease (MAFLD) are retained only when describing studies that used those terms at the time of publication.

The association between diet and MASLD is increasingly well characterized. Epidemiological studies indicate that dietary quality is associated with MASLD risk. This relationship has been evaluated using indices such as the Dietary Inflammatory Index and the Mediterranean Diet Score.[23,24] Specific nutrients, such as polyunsaturated fatty acids, vitamin E, and

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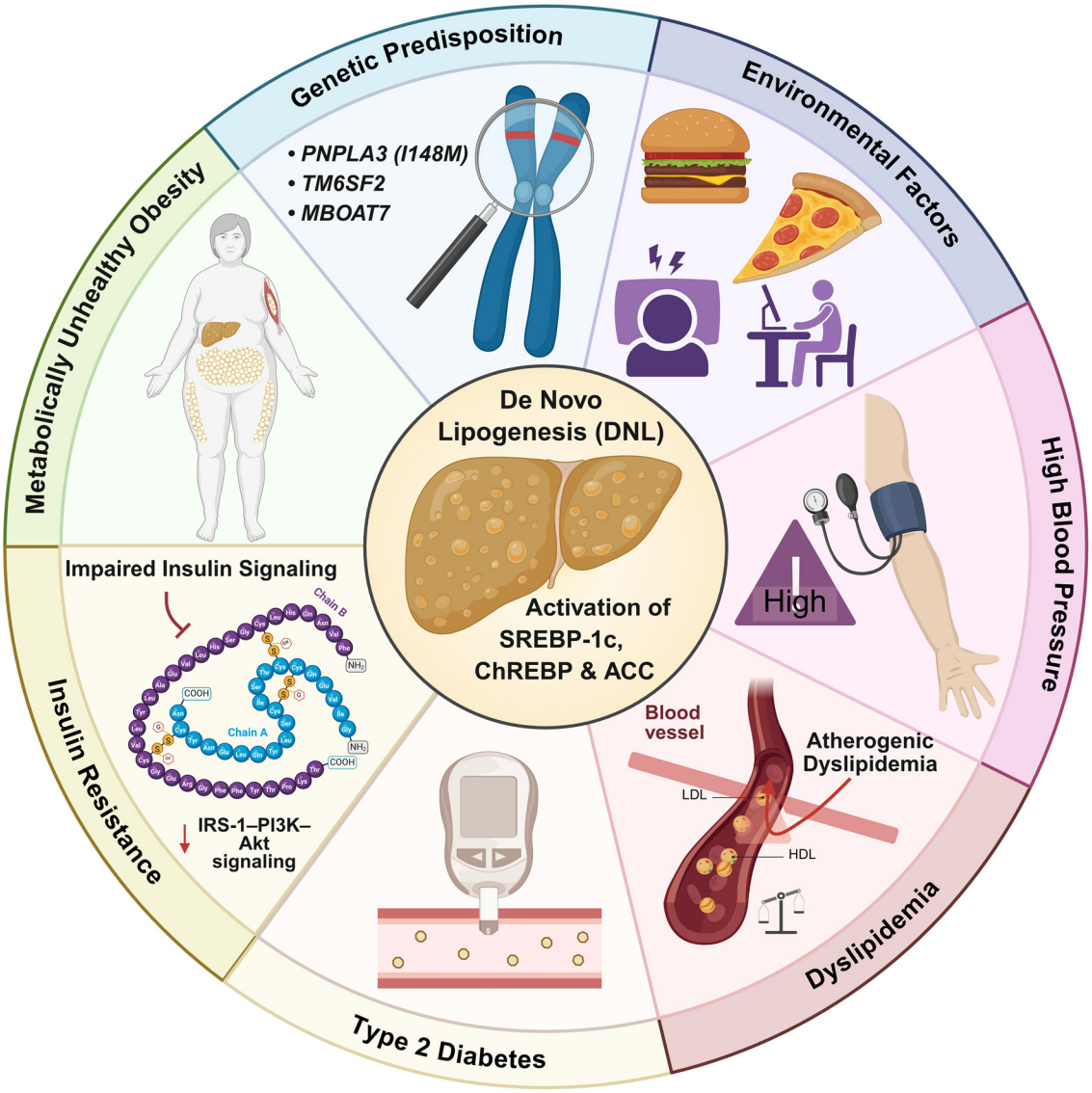


Fig. 1. Major genetic, environmental, and metabolic drivers of MASLD. Genetic susceptibility involving *PNPLA3*, *TM6SF2*, and *MBOAT7* interacts with excess energy intake, physical inactivity, obesity, type 2 diabetes, dyslipidemia, and hypertension. Impaired IRS-1–PI3K–Akt signaling contributes to insulin resistance. Together with the activation of SREBP-1c, ChREBP, and ACC, these metabolic alterations enhance de novo lipogenesis and triglyceride accumulation, thereby contributing to hepatic steatosis. ACC, acetyl-CoA carboxylase; Akt, protein kinase B; ChREBP, carbohydrate response element-binding protein; DNL, de novo lipogenesis; FFA, free fatty acid; HDL, high-density lipoprotein; IR, insulin resistance; IRS-1, insulin receptor substrate 1; LDL, low-density lipoprotein; MASLD, metabolic dysfunction-associated steatotic liver disease; *MBOAT7*, membrane-bound O-acyltransferase domain-containing 7; PI3K, phosphoinositide 3-kinase; *PNPLA3*, patatin-like phospholipase domain-containing 3; SREBP-1c, sterol regulatory element-binding protein 1c; T2DM, type 2 diabetes mellitus; TG, triglyceride; *TM6SF2*, transmembrane 6 superfamily member 2.

iron, may also influence disease development.[25–28] The gluten-free diet (GFD) is the cornerstone of treatment for celiac disease (CD) and requires avoidance of gluten from wheat, barley, and rye.[29] Whether gluten exclusion itself can provide metabolic benefits in patients with MASLD who do not have CD remains uncertain because the available evidence is limited and often confounded by changes in overall diet quality, caloric intake, fiber intake, and the use of processed gluten-free substitutes.[30–33]

Recent reviews, including those by Cazac *et al.* and Valvano *et al.*, have summarized the epidemiological overlap, shared genetic background, and liver-related manifestations of celiac disease and metabolic liver disorders.[30,34] A key question is whether gluten-free, wheat-protein-restricted, or related dietary strategies have therapeutic value for MASLD in the absence of active celiac autoimmunity. Nutritional and metabolic effects may also differ among a GFD, a low-gluten diet, an amylase-trypsin inhibitor (ATI)-free diet, a wheat-free diet, and a low-FODMAP diet. These interventions should not be considered interchangeable.

The purpose of this review is to critically evaluate the therapeutic boundaries of gluten-targeted interventions for MASLD. We distinguish between lifelong GFD use in patients with celiac disease and targeted dietary adjustments (such as reducing gluten or limiting ATI) in patients with non-celiac MASLD. Because a GFD is widely used as a lifestyle or weight-loss strategy, its potential benefits, nutritional limitations, economic burden, adherence, and associated meta-

bolic risks should be evaluated objectively.[35,36] Although this narrative review uses predefined retrieval criteria, its conclusions remain limited by potential selection bias and the lack of high-quality randomized controlled trials in the non-celiac MASLD population.

A structured literature search was conducted in PubMed, Embase, and the Cochrane Library from database inception through June 2026. Controlled vocabulary and free-text terms related to gluten- or wheat-targeted dietary interventions, including "gluten-free diet," "gluten restriction," "wheat-free diet," "amylase-trypsin inhibitors," and "ATI-free diet," were combined with terms for "MASLD," "NAFLD," "MAFLD," "hepatic steatosis," and related hepatic and metabolic outcomes. Additional studies were identified from the reference lists of relevant reviews, clinical guidelines, and primary articles. Human clinical and observational studies, experimental studies, reviews, and guidelines were included if they addressed gluten- or wheat-targeted diets in relation to hepatic steatosis, metabolic outcomes, or the gut–liver axis. Studies were excluded if they did not clearly define the intervention, the population or model, or the relevant outcomes. As this was a narrative review, studies were selected based on relevance and the strength of the evidence and then summarized by population, intervention, outcome, and whether the evidence was direct or indirect. Key clinical studies are summarized in Table 1.[37–42]

Clinical evidence of gluten- and wheat-targeted interventions in MASLD

Effects on hepatic steatosis

The effects of gluten-targeted diets on hepatic steatosis vary substantially and depend mainly on the patient cohort and the specific dietary pattern. In patients with concurrent celiac disease and hepatic steatosis, strict adherence to a GFD is essential for promoting intestinal mucosal healing. However, clinical evidence regarding the long-term effects of a GFD on liver fat accumulation remains conflicting. A cohort study by Ciccone *et al.* showed that the prevalence of hepatic steatosis increased from 1.7% to 11.1% after a GFD ($P < 0.001$), while the prevalence of metabolic syndrome increased from 3.24% to 14.59% ($P < 0.001$).[37] Worsening hepatic steatosis may be related to the consumption of ultra-processed gluten-free products. Because these commercial substitutes rely heavily on refined carbohydrate matrices and saturated fats, a high intake of these foods may increase peripheral insulin resistance and accelerate hepatic de novo lipogenesis.[38,43–45]

For patients with non-celiac MASLD, a lifelong GFD is not recommended. Gluten reduction or ATI-free diets should not be used routinely in non-celiac MASLD outside a defined gluten- or wheat-related disorder or a clinical trial. Unlike the gluten-driven autoimmune response in celiac disease, ATIs directly stimulate innate immunity by binding to Toll-like receptor 4. In a 6-week open-label randomized controlled trial by Armandi *et al.*, a wheat/ATI-targeted intervention reduced the controlled attenuation parameter by 30.5 dB/m compared with the control ($P = 0.039$), with significant changes in the homeostatic model assessment of insulin resistance ($P = 0.042$) and body mass index (BMI; $P = 0.018$).[39] However, the intervention simultaneously reduced exposure to wheat foods containing ATIs and gluten, and the observed effects cannot be attributed specifically to reduced ATI exposure.

Impact on liver function biomarkers

The effect of a GFD on liver biomarkers varies among patient groups. In patients with CD, a GFD typically reduces elevated levels of alanine aminotransferase and aspartate aminotransferase.[40] This improvement is generally associated with reduced intestinal inflammation and improved nutritional status.[41,46] In patients with MASLD, particularly those with coexisting CD, a GFD may affect liver function by altering bile acid metabolism. Manka *et al.* reported reduced fibroblast growth factor 19 (FGF19) levels in patients with CD, with lower levels associated with greater steatosis, particularly in those with elevated anti-tissue transglutaminase (anti-tTG) antibodies.[47] Activated by the farnesoid X receptor, the enterokine FGF19 represses hepatic bile acid synthesis via CYP7A1 inhibition and simultaneously coordinates glucose and lipid homeostasis.[48] FGF19 deficiency is associated with increased liver fat and markers of fibrosis.

Furthermore, metabolic effects of dietary modifications have been reported in unselected cohorts. For instance, a dietary intervention study by Popović *et al.* involving healthy women reported that a modified polyphenol-enriched GFD (supplemented with blueberry and raspberry pomace) improved systemic biomarkers.[49] However, because this cohort lacked both celiac disease and the underlying metabolic dysfunction characteristic of MASLD, these findings represent indirect evidence and should not be interpreted as evidence of the therapeutic efficacy of a standard GFD in non-celiac liver disease.

Effects on metabolic parameters

Reported metabolic effects of gluten- and wheat-targeted dietary interventions are heterogeneous. In the short term, a 6-week wheat/ATI-targeted intervention may reduce insulin resistance.[39] In contrast, longer-term GFD use in CD cohorts has been associated with adverse metabolic changes. In patients with CD, the prevalence of metabolic syndrome increased from 11.4% before treatment to 18.2% after initiation of a GFD, accompanied by a 40% reduction in dietary fiber intake and an increase in consumption of simple sugars.[42] In children, processed gluten-free foods may increase cholesterol levels, whereas naturally gluten-free foods may not have this effect.[50,51]

Crucially, these apparently contradictory metabolic outcomes may partly reflect a failure to distinguish between commercially processed gluten-free products and a naturally gluten-free whole-food diet. Ultra-processed gluten-free foods (e.g., packaged breads and baked goods) often rely on refined starches—such as potato, rice, or tapioca flour—combined with fats and sugars to mimic the texture of gluten-containing foods.[35,52] Consequently, these products often have a higher glycemic index, higher energy density, and lower fiber and micronutrient content. This nutritional imbalance may exacerbate insulin resistance and accelerate hepatic de novo lipogenesis in patients with MASLD.[30,42] Conversely, a naturally gluten-free, whole-food diet incorporating quinoa, tartary buckwheat, legumes, vegetables, and lean proteins provides ample fiber, polyphenols, and essential micronutrients.[53,54] This whole-food pattern may support intestinal barrier function and short-chain fatty acid production, but these effects likely reflect its overall dietary composition rather than gluten exclusion itself.[55,56] In addition, host genetic background may influence metabolic responses. For example, the *PNPLA3* rs738409 variant may influence metabolic outcomes, and carriers of this risk allele exhibit an increased propensity for hepatic fat accumulation after certain dietary interventions.[57]

Effects on MASLD subtypes

Obese MASLD: Obesity is a major modifier of MASLD. Excess energy intake, poor dietary quality, and the consump-

Table 1. Summary of key clinical studies evaluating gluten-free or gluten-related dietary interventions in patients with celiac disease or non-celiac MASLD

Study	Design	Sample size (*n*)	Population characteristics	Celiac disease status	Intervention	Duration	Control/comparison	MASLD assessment	Liver outcomes	Metabolic outcomes	Limitations
Ciccone *et al.* 2019[37]	Prospective cohort	185	Adults with CD	Confirmed CD	GFD	From baseline to post-GFD	Self-controlled (baseline)	Abdominal ultrasound	The prevalence of hepatic steatosis increased significantly from 1.7% at baseline to 11.1% post-GFD ($P < 0.001$)	The prevalence of metabolic syndrome increased from 3.24% to 14.59% ($P < 0.001$); significant post-GFD increases in WC, blood pressure, hyperglycemia, and BMI > 25	No non-CD control group; quantitative data on caloric and nutrient intake were not collected
Rispo *et al.* 2021[38]	Prospective observational study	221	Patients with newly diagnosed CD	Confirmed CD	GFD	2 years	Longitudinal follow-up (baseline vs. 2 years)	Abdominal ultrasound and non-invasive fibrosis scores (APRI, FIB-4, NFS)	At baseline, 29.4% had NAFLD and 14.5% had MAFLD. After 2 years on GFD, NAFLD rose to 46.6% and MAFLD rose to 32.6%	Participants with MAFLD after 2 years had significantly higher HOMA-IR, transaminase levels, LDL-C, and non-invasive fibrosis scores	No matched healthy non-celiac control group; adherence to naturally gluten-free versus processed gluten-free foods was not differentiated
Armandi *et al.* 2024[39]	Randomized controlled trial (RCT)	45 (36 completed)	Adults with biopsy-confirmed MASLD	Non-celiac	ATI-free diet (ATI/gluten-containing wheat-food reduction)	6 weeks	Gluten-containing balanced diet (DGE guidelines)	Controlled attenuation parameter (CAP) via FibroScan	Significant reduction in CAP values at week 6; mean CAP difference compared with the control group: 30.5 dB/m ($P = 0.039$)	Significant improvements in HOMA-IR ($P = 0.042$) and BMI ($P = 0.018$) observed at 6 weeks	Open-label design; short duration; small sample size; BMI changed during the intervention; ATI-specific effects cannot be separated from changes in gluten exposure, energy intake, diet quality, or weight

(continued)

Table 1. *(continued)*

Study	Design	Sample size (*n*)	Population characteristics	Celiac disease status	Intervention	Duration	Control/comparison	MASLD assessment	Liver outcomes	Metabolic outcomes	Limitations
Castillo *et al.* 2015[40]	Retrospective cross-sectional study	463	Adults with CD and available baseline transaminase data	Confirmed CD	GFD	Mean 1.5 ± 1.5 years (follow-up ranged up to several years)	Self-controlled (baseline)	Serum transaminases (ALT/AST)	Elevated serum transaminase levels were present in 40.6% at CD diagnosis; 24.2% remained elevated after GFD. Normalization occurred in 78.6% of those with baseline elevation	Not reported	Retrospective medical records; no direct liver imaging (ultrasound/MRI) to quantify fat content
Seidita *et al.* 2024[41]	Retrospective cohort	160	Pediatric patients (aged 1–14 years) with newly diagnosed CD	Confirmed CD	GFD	12 months	Self-controlled (baseline)	Liver ultrasound and serum transaminases	At 12 months, among patients with baseline abnormalities, hypertransaminasemia persisted in 53.8% (baseline prevalence: 8.1%) and hepatic steatosis persisted in 50.0% (baseline prevalence: 6.1%)	Not reported	Retrospective design; small sample size; selection bias related to tertiary referral; limited follow-up (12 months); lack of biomarkers for mechanistic pathways
Agarwal *et al.* 2021[42]	Cohort study (prospective and retrospective)	Group 1: 54; Group 2: 130	Adults with CD	Confirmed CD	GFD	Group 1: 1 year; Group 2: median ~4 years	Group 1: self-controlled; Group 2: cross-sectional data	FibroScan CAP (cutoff > 263 dB)	In Group 1, the prevalence of fatty liver increased from 14.3% at baseline to 29.5% after 1 year on a GFD ($P = 0.002$)	In Group 1, the prevalence of metabolic syndrome increased from 11.4% to 18.2%; mean BMI (+1.6 kg/m^2) and waist circumference (+3.2 cm) increased ($P < 0.01$)	Short prospective follow-up (1 year); single-center baseline data; detailed nutrient intake data were unavailable

Data are presented as reported in the primary publications. Rispo *et al.*[38] evaluated a longitudinal cohort of patients with newly diagnosed celiac disease rather than a separate non-celiac control group. Statistical significance: *P* values indicate comparisons between baseline and post-intervention follow-up unless otherwise specified. ALT, alanine aminotransferase; APRI, aspartate aminotransferase-to-platelet ratio index; AST, aspartate aminotransferase; ATI, amylase-trypsin inhibitor; BMI, body mass index; CAP, controlled attenuation parameter; CD, celiac disease; DGE, Deutsche Gesellschaft für Ernährung (German Nutrition Society); FIB-4, fibrosis-4 index; GFD, gluten-free diet; HDL, high-density lipoprotein; HOMA-IR, homeostatic model assessment of insulin resistance; LDL-C, low-density lipoprotein cholesterol; LFT, liver function test; MAFLD, metabolic dysfunction-associated fatty liver disease; MASLD, metabolic dysfunction-associated steatotic liver disease; MRI, magnetic resonance imaging; MS, metabolic syndrome; NAFLD, non-alcoholic fatty liver disease; NFS, NAFLD fibrosis score; RCT, randomized controlled trial; WC, waist circumference.

tion of ultra-processed foods contribute to insulin resistance and hepatic fat accumulation.[38] In patients with obesity and MASLD, replacing gluten-containing foods with commercially processed gluten-free products may increase the intake of refined starches, added sugars, and saturated fats, thereby promoting weight gain and potentially worsening metabolic dysfunction. Therefore, a GFD should not be recommended solely because of obesity or MASLD.[45] When gluten restriction is clinically indicated because of coexisting celiac disease or non-celiac gluten sensitivity (NCGS), dietary management should emphasize naturally gluten-free whole foods, adequate fiber intake, control of energy intake, and dietitian supervision. Direct evidence on the effects of a GFD specifically in patients with obesity and MASLD remains limited.

Lean MASLD: Lean MASLD occurs in individuals with normal body weight despite hepatic fat accumulation. Although the effects of a GFD in this population have not been adequately studied, evidence suggests that gut inflammation plays an important role in disease development.[58,59] A GFD may affect the gut microbiota and intestinal inflammation in gluten-related disorders; however, direct evidence of benefit in lean MASLD is lacking.[60] In fact, hepatic steatosis may improve in some patients with celiac disease after 12 months of a gluten-free diet, whereas others may develop new metabolic complications.[41] This highlights the importance of individualized nutritional therapy.

MASLD with celiac disease: In patients with both CD and MASLD, a GFD is mandatory for the treatment of CD, but its metabolic and hepatic effects are not uniformly favorable. In a longitudinal cohort of patients with newly diagnosed CD, NAFLD prevalence increased from 29.4% at baseline to 46.6% after 2 years of a GFD, while MAFLD prevalence increased from 14.5% to 32.6%.[38] Other studies have reported improvements in liver enzyme levels after a GFD, whereas changes in BMI and waist circumference depend on baseline nutritional status and diet quality.[30] Furthermore, genetic factors such as HLA-DQ2 and HLA-DQ8 are associated with CD risk and may influence MASLD development, suggesting that genetic information may be useful for personalized treatment.[61,62]

Potential mechanisms linking gluten- and wheat-targeted interventions to MASLD

Multiple factors contribute to the development of MASLD, including insulin resistance, mitochondrial dysfunction, endoplasmic reticulum stress, and oxidative stress.[13,63–65] The gut-liver axis plays a central role in MASLD pathophysiology, as gut dysbiosis can increase endotoxin absorption and activate inflammatory pathways in the liver.[65–68] Furthermore, bile acid signaling abnormalities (e.g., reduced FGF19) and genetic polymorphisms (e.g., *PNPLA3* rs738409) may contribute to differences in disease phenotype.[69–72] As shown in Figure 2, gluten- or wheat-targeted interventions may theoretically influence MASLD through four broad pathways: modulation of gut microbiota, regulation of inflammatory responses, effects on insulin sensitivity, and changes in oxidative stress and lipid metabolism. For each mechanism, however, the source and level of evidence should be carefully distinguished.

Modulation of gut microbiota

Evidence tier: primarily human CD studies and indirect inference; direct GFD intervention data in MASLD are lacking. A GFD can alter gut microbiota composition in patients with CD.[73] For example, a GFD has been reported to reduce the Firmicutes-to-Bacteroidetes ratio while increasing the relative abundance of Ruminococcaceae and Bacteroidetes, changes that have been associated with improved cholesterol metabolism.[74–76] However, these observations cannot be assumed to apply directly to non-celiac MASLD. In some CD cohorts, a GFD has been associated with reduced short-chain fatty acid production and increased intestinal permeability, potentially promoting hepatic fat accumulation.[76–78] Gluten exclusion may reduce gluten-induced intestinal inflammation and barrier dysfunction in gluten-sensitive individuals, but direct evidence that a GFD restores a MASLD-specific microbiome signature is currently insufficient.[79,80] Because nutritional therapy alone may not fully restore intestinal homeostasis, combining a GFD with probiotics or prebiotics may provide additional benefits, although this strategy requires dedicated MASLD trials.[81]

Regulation of inflammatory responses

Evidence that a GFD reduces systemic inflammation in non-celiac MASLD remains indirect. Evidence tier: primarily CD-related immune data, animal studies, and indirect gut-liver axis inference. Gluten intake may activate innate immunity, causing intestinal endoplasmic reticulum stress and the release of pro-inflammatory cytokines (e.g., interferon-gamma and interleukin–2), which may exacerbate hepatic steatosis.[33,82–84] These pathways may contribute to systemic inflammation and hepatic steatosis, but their relevance to non-celiac MASLD remains uncertain. In the study by Manka *et al.*, patients with higher anti-tTG levels showed a trend toward greater steatosis; FGF19 levels did not differ between anti-tTG groups, but FGF19 was inversely associated with the controlled attenuation parameter only in the higher anti-tTG subgroup.[47] Probiotic supplementation during a GFD may further reduce intestinal inflammation through modulation of the unfolded protein response, but evidence of downstream liver benefits remains indirect.[85]

Effects on insulin sensitivity

The effect of a GFD on insulin sensitivity may be bidirectional. Evidence tier: predominantly animal models; human translation unverified. High-sugar and high-fat gluten-free substitutes may promote obesity and insulin resistance.[30,33] Although a GFD has been associated with improved insulin signaling in preclinical studies, its relevance to non-celiac MASLD remains uncertain.[30,33] Mechanistic insights into how gluten restriction alters insulin signaling are largely limited to basic research and indirect animal models, warranting substantial caution when extrapolating these findings to humans. For instance, in a non-obese diabetic mouse model, maternal GFD transgenerationally improved offspring pancreatic β-cell function, independent of microbiota changes, suggesting an immunomodulatory effect of gluten exclusion.[86] Similarly, a gluten-free diet has reduced visceral fat and improved glucose homeostasis in murine models of obesity.[33] However, these animal experiments were conducted under highly controlled conditions and do not reflect the heterogeneity of human MASLD in the real world. In humans, the disease is influenced by many factors, including diet, physical activity, obesity, medications, and genetics.[26,38,57,87,88]

Oxidative stress and lipid metabolism

Evidence linking gluten-derived or gluten-free compounds to hepatic oxidative stress and lipid metabolism remains indirect. Evidence tier: in vitro and animal studies; indirect inference. Inferences regarding mitophagy activation and lipid clearance are derived mainly from isolated plant-derived compounds tested in basic models. For example, corn pep-

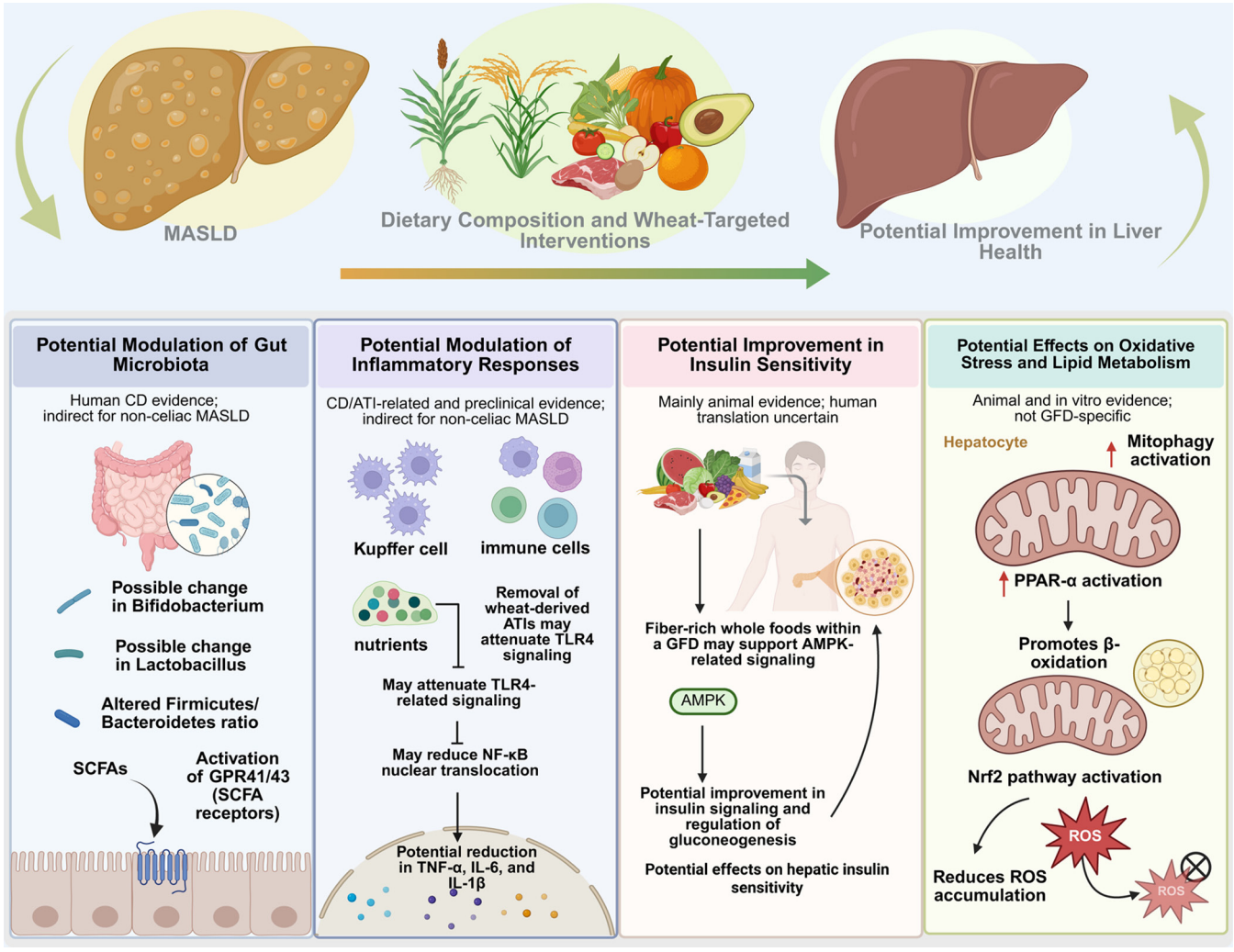


Fig. 2. Potential molecular pathways linking dietary composition and wheat-targeted interventions to MASLD. The schematic shows potential effects on gut microbiota, TLR4–NF-κB-mediated inflammation, insulin sensitivity, and oxidative stress/lipid metabolism. Evidence is derived mainly from celiac disease studies, exploratory clinical research, and preclinical models and remains limited in non-celiac MASLD. ATI-targeted interventions should not be considered equivalent to a standard GFD. AMPK, AMP-activated protein kinase; ATI, amylase-trypsin inhibitor; GFD, gluten-free diet; GPR41/43, G protein-coupled receptors 41 and 43; IL-1β, interleukin-1 beta; IL-6, interleukin-6; MASLD, metabolic dysfunction-associated steatotic liver disease; NF-κB, nuclear factor kappa B; Nrf2, nuclear factor erythroid 2-related factor 2; PPAR-α, peroxisome proliferator-activated receptor alpha; ROS, reactive oxygen species; SCFA, short-chain fatty acid; TLR4, Toll-like receptor 4; TNF-α, tumor necrosis factor alpha.

tides evaluated in high-fat-fed animal models activated the PINK1/Parkin-mediated mitophagy pathway.[89] Similarly, antioxidant and anti-inflammatory properties of pigmented corn polyphenols have been demonstrated *in vitro* using Caco-2 cell lines.[90] These findings are mechanistically informative but do not demonstrate that a commercial GFD in humans activates mitochondrial quality-control pathways or reduces hepatic oxidative stress in patients with MASLD.

However, long-term adherence to a GFD may lead to trace-element deficiencies (such as copper), thereby exacerbating oxidative stress.[91,92] Although some nutritional interventions (such as quercetin supplementation) may reduce liver damage by scavenging free radicals and modulating the immune response, these strategies should be distinguished from a standard GFD.[79] A GFD may also influence bile acid signaling; for example, FGF19 inhibition may alter hepatic lipid synthesis, while long-term adherence to a low-fiber GFD may disrupt bile acid pool homeostasis.[47] Overall, although the proposed mechanisms involving oxidative stress and lipid metabolism are biologically plausible, they have not been fully verified in the human MASLD population.

Comparison of GFD with other dietary patterns

Dietary intervention is an important component of MASLD management, but dietary patterns differ in the strength of supporting evidence, metabolic efficacy, nutritional adequacy, adherence, and long-term safety. To date, no clinical trials have directly compared a GFD with other established dietary patterns in non-celiac populations. Table 2 provides a comparison based on the relevant literature,[30,39,43,52,60,93–111] including treatment outcomes, insulin sensitivity, gut microbiota, and adherence.

Mediterranean diet

The Mediterranean diet is rich in fruits, vegetables, whole

Table 2. Comparative matrix of GFD and other dietary approaches for MASLD

Dietary pattern	Efficacy in reducing hepatic steatosis	Impact on insulin sensitivity	Gut microbiota modulation	Practical feasibility and adherence	Nutritional adequacy/risks	Level of evidence (non-celiac MASLD) (Non-Celiac MASLD)	Primary role in MASLD management
Mediterranean diet	Associated with reduced hepatic steatosis and improved metabolic health	May improve insulin sensitivity	May promote microbial diversity and SCFA-producing bacteria	Generally feasible; adherence varies; culturally integrated	Generally nutrient-dense when appropriately implemented	Moderate; supported by MASLD-focused and broader metabolic evidence[93,94,98]	Evidence-supported dietary option for MASLD management
Low-carbohydrate diet (LCD)	Short-term reductions in hepatic fat have been reported	May improve short-term glycemic control and reduce postprandial glucose and insulin excursions	May alter gut microbial composition; MASLD-specific effects remain uncertain	May be challenging to sustain, particularly with stricter carbohydrate restriction	Risk of nutritional imbalances, including inadequate fiber and excessive saturated fat	Low; evidence includes preclinical MAFLD data and short-term metabolic or hepatic findings in overweight or obese adults[95–98]	Potential short-term option for weight and glycemic management
High-protein diet (HPD)	Potential reduction in hepatic fat; evidence remains limited	Potential indirect improvement, often associated with weight loss	Effects vary by protein source and overall diet; MASLD-specific evidence is limited	Feasibility depends on dietary habits and protein-source choices	Variable; some animal-protein sources may increase gout or nephrolithiasis risk[104,105]	Low; MASLD-specific human evidence is limited, with additional preclinical and general nutritional evidence[99–103]	Adjunctive option in selected patients, particularly when preservation of lean mass is a goal
Intermittent fasting (IF; including TRE)	Short-term reductions in hepatic steatosis have been reported in some studies	Effects on fasting insulin and insulin sensitivity vary by fasting protocol and population	Potential effects on circadian and gut microbial rhythms; MASLD-specific evidence remains limited	Requires adherence to defined fasting or eating windows; long-term adherence varies	Does not inherently restrict specific food groups; nutritional adequacy depends on food quality during eating periods	Moderate; includes NAFLD-specific clinical evidence, supplemented by general metabolic and preclinical studies[106–110]	Alternative timing-based dietary strategy for selected patients
Gluten-free diet (GFD)	Potential benefit in selected contexts. A short-term wheat/ATI-targeted trial reduced CAP,[39] providing indirect proof-of-concept for wheat-protein-targeted strategies, whereas direct evidence for a standard GFD in non-celiac MASLD remains limited	No established GFD-specific effect in non-celiac MASLD; a short-term wheat/ATI-targeted intervention improved HOMA-IR[39]	Evidence is mainly derived from gluten-related disorders; direct evidence in non-celiac MASLD remains limited	Requires dietary counseling to maintain fiber intake and avoid excessive reliance on refined gluten-free substitutes	Generally adequate when based on naturally gluten-free whole foods; may be nutritionally poor when dominated by ultra-processed commercial products[43,52,111]	Insufficient for a standard GFD in non-celiac MASLD; evidence is mainly indirect, with preliminary data from a short-term wheat/ATI-targeted trial[30,39,60]	Indicated for CD; not routinely recommended for non-celiac MASLD

Evidence levels were defined as follows: High, consistent MASLD-specific clinical evidence; Moderate, direct but limited or heterogeneous clinical evidence; Low, mainly limited, indirect, general metabolic, or preclinical evidence; Insufficient, no adequate direct evidence in non-celiac MASLD. These categories are descriptive and do not represent formal GRADE ratings. References in the Level of Evidence column support the overall evidence for each dietary pattern; citations in other cells are provided only for specific study findings or safety-related statements. GFD, gluten-reduced, wheat-free, and ATI-free interventions are not interchangeable. TRE is considered a form of IF. AMPK, AMP-activated protein kinase; ATI, amylase-trypsin inhibitor; CAP, controlled attenuation parameter; CD, celiac disease; DNL, de novo lipogenesis; GFD, gluten-free diet; GRADE, Grading of Recommendations Assessment, Development and Evaluation; HOMA-IR, homeostatic model assessment of insulin resistance; HPD, high-protein diet; IF, intermittent fasting; LCD, low-carbohydrate diet; MAFLD, metabolic dysfunction-associated fatty liver disease; MASLD, metabolic dysfunction-associated steatotic liver disease; MUFA, monounsaturated fatty acid; NAFLD, non-alcoholic fatty liver disease; NCGS, non-celiac gluten sensitivity; PPAR-α, peroxisome proliferator-activated receptor alpha; RCT, randomized controlled trial; SCFA, short-chain fatty acid; TRE, time-restricted eating.

grains, nuts, legumes, and olive oil that is well established for reducing hepatic steatosis and improving metabolic health.[93,94] A standard GFD does not necessarily provide these nutritional benefits, particularly when it relies on refined gluten-free products. However, integrating Mediterranean elements, such as extra virgin olive oil, avocados, legumes, nuts, fruits, vegetables, and naturally gluten-free whole grains, can improve the nutritional quality of a GFD by increasing monounsaturated fatty acids, fiber, antioxidants, and micronutrients. Importantly, the Mediterranean diet, caloric restriction, and structured lifestyle weight loss have substantially stronger clinical support for MASLD management than any form of gluten restriction. Therefore, a GFD should be used when clinically indicated for confirmed CD, whereas gluten-reduced or other wheat-targeted strategies should be individualized for defined gluten- or wheat-related disorders rather than broadly applied to MASLD.

Low-carbohydrate diet

A low-carbohydrate diet reduces carbohydrate intake and may improve hepatic fat and metabolic parameters. Preclinical MAFLD evidence supports effects on hepatic fat,[95] while clinical evidence primarily supports weight loss and metabolic benefits.[96–98] Low-carbohydrate diets should not be considered synonymous with ketogenic diets. A conventional GFD may rely heavily on refined carbohydrates, which can worsen metabolic parameters. Replacing refined carbohydrates with naturally gluten-free whole foods and reducing refined sugar intake may mitigate this risk.

High-protein diet

A high-protein diet may promote satiety and fatty acid β-oxidation while suppressing lipogenesis, thereby reducing hepatic fat deposition.[99–102] It may also help maintain lean body mass during weight loss.[103] Increasing the proportion of high-quality protein in a GFD may help preserve lean body mass during weight loss. However, high intakes of some animal protein sources may increase the risks of gout and nephrolithiasis.[104,105]

Intermittent fasting (IF), including time-restricted eating (TRE)

IF, including TRE, may improve metabolic health by promoting hepatic lipolysis and gluconeogenesis and by optimizing circadian rhythms.[106] TRE has been associated with reductions in hepatic steatosis and improvements in metabolic parameters in some studies.[106,107] A randomized controlled trial showed that alternate-day fasting combined with aerobic exercise improved hepatic steatosis and metabolic outcomes.[108] A GFD may also be combined with IF/TRE, but this approach has not yet been directly studied in MASLD. However, IF should be used cautiously in patients with diabetes because of potential glycemic variability.[109] The optimal time window for TRE remains under investigation.[107,110]

Challenges and future directions

Limitations of current research

The current clinical evidence has both strengths and limitations. Longitudinal CD cohorts allow assessment of long-term hepatic and metabolic changes, while the Armandi *et al.* trial provides randomized proof-of-concept evidence in non-celiac MASLD.[37–39] Current studies on GFD and MASLD are largely observational and heterogeneous, limiting causal inference and generalizability.[37,38,40–42] Patient responses to a GFD are highly heterogeneous, with reports of potential short-term benefits as well as long-term risks such as nutrient deficiencies, gut dysbiosis, and metabolic deterioration.[39,76,112–114] Most evidence comes from CD populations; data on patients with non-celiac MASLD are scarce.[34,52,111,112] The Armandi trial was also limited by its open-label design, small sample size (45 randomized; 36 completed), and short follow-up.[39] Clinically, a GFD remains indicated for confirmed CD, but current evidence does not support routine GFD use for non-celiac MASLD; findings from wheat/ATI-targeted interventions require confirmation before routine clinical use. Future research should prioritize large-scale, long-term randomized controlled trials in diverse populations.

Risks of unsupervised adoption of a gluten-free diet in non-celiac individuals

Driven by aggressive marketing and contemporary lifestyle trends, GFD adoption has increased substantially among people without celiac disease or a confirmed wheat allergy.[36] Unsupervised adoption of a GFD for weight management or general health may carry metabolic risks that can be overlooked during routine clinical assessment.[115] The relationship between NCGS, response to a GFD, and metabolic outcomes is complex.[116] Some patients with NCGS may benefit from dietary modification; however, in individuals without CD or NCGS, complete gluten removal has no established benefit and may carry nutritional, metabolic, financial, and psychosocial risks.

Commercial gluten-free products commonly use refined starches to mimic the texture of wheat-based foods. Over time, this may lead to a shift toward a dietary pattern that is "low in fiber, high in refined carbohydrates, and high in saturated fat".[43,52] This dietary shift may fail to improve metabolic health and may promote liver fat accumulation and insulin resistance, thereby increasing MASLD risk.[30,42] In addition to these metabolic concerns, real-world data indicate that GFD adherence varies widely, ranging from 42% to 91%, depending on cultural and lifestyle factors.[117,118] Poor adherence may contribute to micronutrient deficiencies and increase the financial burden and psychological stress, thereby reducing quality of life.[43,52] Therefore, in the absence of clear clinical indications (such as confirmed CD or NCGS), clinicians should not recommend gluten restriction.

Nutritional deficiencies and solutions

A GFD may be deficient in fiber, B vitamins, iron, zinc, and magnesium.[43,119] Naturally gluten-free foods such as quinoa and buckwheat provide greater nutritional value than processed gluten-free products, which are often high in sugar and saturated fat.[120–123] Increasing whole-food intake, improving food fortification, and preparing meals at home can help reduce nutritional deficiencies.[124–126]

Socioeconomic and cultural considerations

Commercial gluten-free products can be expensive, making long-term dietary adherence difficult.[127,128] Preparing meals at home with naturally gluten-free staples, such as rice, corn, and legumes, can reduce costs and increase dietary variety.[125,126] Cultural dietary habits also influence GFD adoption. Individuals whose diets are based on rice and corn may find it relatively easy to adopt a GFD, whereas those in regions with strong wheat-based dietary traditions may face greater difficulty.[129,130]

Toward personalized dietary strategies

Individualized dietary approaches are important for optimiz-

ing GFD outcomes. Developing a personalized dietary plan requires consideration of metabolic status, BMI, genetic background, and nutritional status.[47,131–136] Biomarkers such as anti-tTG antibodies may help define therapeutic strategies.[45] However, anti-tTG antibodies are useful only for assessing CD or CD-MASLD overlap and should not be used to stratify patients with non-celiac MASLD. If a GFD is needed, patients can improve dietary quality by consuming naturally gluten-free whole foods, ensuring adequate protein and fiber intake, monitoring micronutrient levels, and working with a dietitian. Combining a GFD with therapies such as probiotics may improve metabolic outcomes.[137,138] Future research should explore novel approaches, such as enzymatic treatment of gluten-containing foods and gluten detoxification, to improve safety and effectiveness.[89,139–141]

Conclusions

Although gluten- or wheat-targeted interventions may influence the gut microbiota, systemic inflammation, and insulin sensitivity, these effects are not necessarily attributable to gluten exclusion itself. Current clinical evidence does not support the routine use of a standard GFD in the broader MASLD population unless patients have confirmed celiac disease or another gluten-related disorder.

A short-term wheat/ATI-targeted intervention may improve metabolic parameters, whereas long-term use of low-quality, commercially processed gluten-free substitutes may adversely affect a patient's metabolic profile. The high glycemic index and fat content of these processed gluten-free substitutes may worsen insulin resistance and contribute to de novo lipogenesis in patients with MASLD. In addition, multiyear adherence to a GFD may increase the risk of deficiencies in dietary fiber, vitamins, and trace elements (especially iron, zinc, magnesium, and copper). These potential nutritional risks warrant careful assessment of baseline metabolic indicators and celiac serology before implementing any form of wheat or gluten restriction. Future research should use high-quality, long-term randomized controlled trials in diverse, non-celiac MASLD cohorts to clarify the therapeutic window of wheat-restricted diets rather than relying solely on preliminary pilot data.

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Conflict of interest

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Author contributions

Conceptualization (DM, JS); literature investigation and analysis (DM, YY, MY, SZ, ZJ, MJ); re-evaluation of clinical evidence, reconstruction of Tables 1 and 2, major revision of the clinical-evidence and mechanistic sections, and drafting of sections of the response-to-reviewers document (ZJ); drafting the manuscript (DM); reviewing and editing the manuscript (YY, MY, SZ, ZJ, MJ, JL, JS); visualization (DM); supervision (JS, JL); project administration (JL); funding acquisition (JS). All authors read and approved the final manuscript.

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