Lifestyle, Dietary Interventions, and Biochemical Markers in the Management of Chronic Hepatobiliary and Gastric Diseases

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Abstract

Introduction: Chronic hepatobiliary diseases — including metabolic dysfunction-associated steatotic liver disease (MASLD), chronic viral hepatitis, cirrhosis, and cholelithiasis — and chronic gastric diseases — including gastroesophageal reflux disease (GERD), peptic ulcer disease (PUD), and Helicobacter pylori-associated gastritis — share modifiable risk factors centered on diet, body weight, alcohol, tobacco, and sedentary lifestyle. Despite expanding pharmacotherapy, lifestyle and dietary interventions remain first-line for many conditions and adjunctive across all of them. Objective: To synthesize current evidence on lifestyle and dietary interventions, supported by biochemical markers, in the management of chronic hepatobiliary and gastric diseases in adults, and to propose an integrated nutrology-informed framework. Methods: A structured narrative review was conducted using PubMed/MEDLINE, Scopus, Web of Science, and the Cochrane Library for studies published between January 2000 and December 2025. Search terms combined "diet," "lifestyle," "Mediterranean diet," "weight loss," "physical activity" with "MASLD," "chronic hepatitis," "cirrhosis," "gallstones," "GERD," "peptic ulcer," and "Helicobacter pylori," together with "biochemical markers," "FIB-4," "ALT," and "AST." Randomized controlled trials, cohort studies, systematic reviews, and authoritative guidelines published in English were eligible. Results: In MASLD, weight loss of ≥5% reduces hepatic steatosis, ≥7% improves necroinflammation, and ≥10% stabilizes or reverses fibrosis; the Mediterranean diet reduces hepatic fat by approximately 39% over 12 weeks. In cirrhosis, individualized protein-energy nutrition, branched-chain amino acid supplementation, and sarcopenia prevention improve survival. In gallstone disease, gradual weight loss, high-fiber, low-refined-carbohydrate diets, coffee, and physical activity reduce incidence, whereas crash dieting precipitates stones. In GERD and PUD, weight reduction, smoking cessation, alcohol moderation, and avoidance of trigger foods complement acid suppression.

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In H. pylori-associated gastritis, polyphenol-rich diets (broccoli sprouts, green tea, cranberry) and probiotics improve eradication and reduce gastric inflammation. Across all conditions, simple biochemical and non-invasive markers (ALT/AST, GGT, FIB-4, lipid panel, HbA1c, hsCRP) provide objective monitoring. Conclusion: Lifestyle and dietary interventions — Mediterranean-style nutrition, gradual weight management, physical activity, alcohol moderation, and tobacco cessation — combined with monitoring of accessible biochemical markers, form a unifying nutrological strategy across the hepatobiliary–gastric disease spectrum and should be systematically integrated into routine practice.

Keywords: Lifestyle; Diet, Mediterranean; Hepatobiliary Diseases; Gastric Diseases; Biomarkers; Nutritional Status.

Introduction

Chronic hepatobiliary and gastric diseases account for a substantial share of the global non-communicable disease burden. Metabolic dysfunction-associated steatotic liver disease (MASLD) affects approximately 25–30% of adults worldwide and has overtaken viral hepatitis as the leading cause of chronic liver disease in many regions [1,2]. Chronic hepatitis B and C together affect more than 300 million people and continue to drive cirrhosis and hepatocellular carcinoma (HCC) [3]. Gallstone disease (cholelithiasis) is one of the most common digestive disorders, with a lifetime prevalence of 10–20% in adults and rising rates linked to obesity and sedentary lifestyles [4]. In parallel, gastric diseases — gastroesophageal reflux disease (GERD), peptic ulcer disease (PUD), and *Helicobacter pylori*-associated gastritis — affect billions of individuals globally and confer substantial morbidity, healthcare cost, and risk of gastric cancer [5,6].

Despite distinct anatomical and microbiological substrates, these conditions share a remarkably overlapping set of modifiable risk factors. Excess caloric intake, ultra-processed foods, refined carbohydrates, sugar-sweetened beverages, alcohol misuse, tobacco use, sedentary behavior, and obesity each contribute simultaneously to hepatic steatosis, biliary lithogenicity, esophageal acid exposure, gastric mucosal injury, and dysbiosis of the gut microbiome [1,2,7,8]. Conversely, a small set of evidence-based interventions — Mediterranean-style dietary patterns, gradual weight management, structured physical activity, alcohol moderation, and smoking cessation — yield benefits across this entire disease spectrum [1,7,9].

From a nutrological perspective, this convergence offers an opportunity to design unified, patient-centered, lifestyle-anchored management strategies rather than disease-by-disease prescriptions. Simultaneously, the routine availability of inexpensive biochemical markers — alanine and aspartate aminotransferases (ALT, AST), gamma-glutamyl transpeptidase (GGT), bilirubin, alkaline phosphatase, lipid panel, glycated hemoglobin (HbA1c), high-sensitivity C-reactive protein (hsCRP), and the FIB-4 composite — enables objective tracking of intervention effects without invasive testing [10,11]. These markers serve both as motivational tools for patients and as objective endpoints for clinicians.

Despite a growing body of literature, the translational evidence is heterogeneous and frequently siloed by organ system. Few reviews synthesize lifestyle and dietary interventions across both hepatobiliary

and gastric domains, despite their pathophysiological and behavioral interconnection through the gut–liver axis. The present narrative review aims to provide a critical, mechanism-oriented synthesis of current evidence on lifestyle and dietary interventions, supported by biochemical markers, in the management of chronic hepatobiliary and gastric diseases in adults, and to outline practical implications for integrated nutrological care.

Materials and Methods

Study design

This is a structured narrative review designed to map and critically appraise the current evidence on lifestyle and dietary interventions, supported by biochemical markers, in the management of chronic hepatobiliary and gastric diseases in adults. The review was conducted following the general principles of the SANRA (Scale for the Assessment of Narrative Review Articles) framework [12] and incorporated key recommendations from the PRISMA 2020 statement [13] regarding transparency of search strategy and study selection, although a formal systematic review protocol was not registered.

Information sources and search strategy

Electronic searches were conducted in PubMed/MEDLINE, Scopus, Web of Science, the Cochrane Central Register of Controlled Trials, and Google Scholar for studies published between January 2000 and December 2025. The search strategy combined Medical Subject Headings (MeSH) and free-text terms in four blocks: (i) interventions — "diet," "dietary pattern," "Mediterranean diet," "weight loss," "calorie restriction," "physical activity," "exercise," "smoking cessation," "alcohol moderation," "probiotics"; (ii) hepatobiliary conditions — "MASLD," "NAFLD," "NASH," "chronic hepatitis B," "chronic hepatitis C," "cirrhosis," "cholelithiasis," "gallstones"; (iii) gastric conditions — "GERD," "gastroesophageal reflux," "peptic ulcer," "Helicobacter pylori," "chronic gastritis"; and (iv) outcomes/markers — "ALT," "AST," "GGT," "FIB-4," "liver stiffness," "HbA1c," "lipid profile," "CRP," "hepatic steatosis," "eradication rate." Boolean operators "AND" and "OR" combined blocks.

Eligibility criteria

Studies were eligible if they (i) involved adult populations (≥18 years) with at least one chronic hepatobiliary or gastric disease; (ii) evaluated a defined lifestyle or dietary intervention (alone or as adjunct to pharmacotherapy); and (iii) reported clinical, histological, imaging, or biochemical outcomes. Randomized controlled trials, controlled clinical trials, prospective and retrospective cohort studies, systematic reviews and meta-analyses, and authoritative clinical guidelines published in English were considered. Studies in exclusively pediatric populations, in acute disease without chronic follow-up, or addressing surgical interventions without lifestyle components were excluded.

Study selection and data extraction

Titles and abstracts retrieved from each database were screened for relevance. Full texts of potentially eligible articles were retrieved and assessed against the eligibility criteria. Data extracted from included studies covered: study design and population, intervention (type, intensity, duration), comparator, primary and secondary outcomes, biochemical markers monitored, follow-up duration, and adverse events.

Synthesis of evidence

Given the heterogeneity of study designs and outcome measures, evidence was synthesized narratively, organized by disease group (hepatobiliary, gastric) and by intervention type (dietary patterns, weight management, physical activity, behavioral risk factors, monitoring with biochemical markers). Risk of bias was considered when interpreting clinical evidence using domains adapted from the Cochrane RoB 2 tool [14]. No formal meta-analysis was conducted because of the descriptive scope of the review.

Results

Overview: common modifiable risk factors across the disease spectrum

Across chronic hepatobiliary and gastric diseases, five modifiable risk factors recur as principal drivers of incidence and progression: excess caloric intake and visceral adiposity; ultra-processed foods and refined carbohydrates; excess alcohol intake; tobacco use; and physical inactivity [1,2,4,7,15]. Pathophysiologically, these factors converge on three shared mechanisms — chronic low-grade systemic inflammation, dysbiosis of the gut microbiome with disruption of the gut–liver axis, and metabolic dysregulation (insulin resistance, dyslipidemia) — that in turn potentiate hepatic steatosis and fibrosis, biliary lithogenicity, and mucosal injury throughout the upper gastrointestinal tract [1,2,7,16]. Table 1 summarizes the principal lifestyle and dietary interventions and their relevance across this spectrum.

Table 1. Principal lifestyle and dietary interventions in the management of chronic hepatobiliary and gastric diseases.

Intervention	Principal mechanism	Most relevant conditions	Trackable biochemical markers
Mediterranean diet	Anti-inflammatory; insulin sensitization; gut microbiome modulation	MASLD, chronic viral hepatitis, gallstones, GERD	ALT, AST, FIB-4, lipid panel, HbA1c, hsCRP
Gradual weight loss (≥5–10%)	Reduces visceral adiposity, hepatic fat, biliary cholesterol saturation	MASLD, cholelithiasis prevention, GERD, PUD	ALT, AST, GGT, FIB-4, fasting glucose, HbA1c
Physical activity (≥150 min/wk)	Improves insulin sensitivity; counters sarcopenia; promotes weight maintenance	MASLD, cirrhosis, gallstones, gastric motility	ALT, HbA1c, lipid panel, body composition
Alcohol moderation / abstinence	Reduces hepatotoxicity, gastric acid stimulation, mucosal injury	All hepatobiliary and gastric conditions	GGT, MCV, AST/ALT ratio
Tobacco cessation	Reduces oxidative stress, acid secretion, vasoconstriction	GERD, PUD, H. pylori-associated disease, MASLD	hsCRP, cotinine, lipid panel
High dietary fiber,	Improves bile	Gallstones, MASLD,	Lipid panel, HbA1c,

Intervention	Principal mechanism	Most relevant conditions	Trackable biochemical markers
low refined carbohydrate	composition; reduces glycemic load; promotes microbiome diversity	GERD	fasting glucose
Probiotics / functional foods	Modulates gut microbiome; reduces gastric inflammation	H. pylori gastritis, MASLD, IBS overlap	Eradication rate; hsCRP; stool antigen
Branched-chain amino acids (BCAA)	Supports muscle protein synthesis; reduces ammonia	Cirrhosis, hepatic encephalopathy, sarcopenia	Albumin, ammonia, anthropometry
Trigger-food avoidance (acid, fat, caffeine, spicy)	Reduces lower esophageal sphincter relaxation; gastric irritation	GERD, PUD, functional dyspepsia	Symptom score; PPI requirement
Coffee (moderate)	Antioxidant; modulates bile acid and hepatic fat metabolism	MASLD, gallstones, cirrhosis progression	ALT, AST, GGT

Note: *MASLD, metabolic dysfunction-associated steatotic liver disease; GERD, gastroesophageal reflux disease; PUD, peptic ulcer disease; ALT, alanine aminotransferase; AST, aspartate aminotransferase; GGT, gamma-glutamyl transpeptidase; FIB-4, fibrosis-4 index; HbA1c, glycated hemoglobin; hsCRP, high-sensitivity C-reactive protein; MCV, mean corpuscular volume; PPI, proton pump inhibitor; BCAA, branched-chain amino acids; IBS, irritable bowel syndrome.*

Lifestyle and dietary interventions in hepatobiliary disease

Metabolic dysfunction-associated steatotic liver disease (MASLD/NASH*)*

MASLD, formerly known as non-alcoholic fatty liver disease (NAFLD), is now the leading global cause of chronic liver disease and is fundamentally driven by metabolic and lifestyle factors [1,2]. Lifestyle modification remains first-line therapy. A dose-response relationship is well established: weight loss of ≥5% reduces hepatic steatosis, ≥7% improves necroinflammation, and ≥10% stabilizes or reverses fibrosis [1,2,17]. A randomized controlled trial (n=259) showed that a 12-week Mediterranean diet reduced hepatic steatosis by 39% and significantly improved insulin sensitivity, while a 52-week calorie-restricted lifestyle program (n=196) reduced liver fat by 25% [2,17].

Specific dietary strategies — Mediterranean, DASH, low-glycemic, and high-protein patterns — converge in emphasizing vegetables, fruits, whole grains, legumes, fish, olive oil, and nuts, with restriction of free sugars, ultra-processed foods, and saturated fat [1,17,18]. Coffee consumption (typically ≥3 cups/day) is consistently associated with lower hepatic steatosis, fibrosis, and HCC risk [19]. Physical activity — both aerobic and resistance training, totalling ≥150 minutes per week — independently reduces intrahepatic triglycerides and counters sarcopenia [1,17].

Chronic viral hepatitis (HBV, HCV)

In chronic viral hepatitis, lifestyle interventions are increasingly recognized as essential adjuncts to antiviral therapy. A randomized trial in chronic hepatitis C (n=120) demonstrated that both a normoglucidic low-calorie diet and a low-fat diet reduced HOMA-IR, BMI, hepatic steatosis, and fibrosis scores over 12 months [20]. Mediterranean-style nutrition, vitamin D repletion, and limitation of alcohol and ultra-processed foods improve metabolic profile, vaccine and treatment response, and likely fibrosis progression in HBV and HCV cohorts [21,22]. Coffee consumption is associated with lower fibrosis progression in chronic HCV [19].

Cirrhosis

In compensated and decompensated cirrhosis, the central nutritional challenges are sarcopenia, protein-energy malnutrition, and micronutrient deficiencies [23,24]. EASL and AASLD guidelines recommend energy intake of 30–35 kcal/kg/day and protein intake of 1.2–1.5 g/kg/day, with late-evening snacks to reduce overnight catabolism, and branched-chain amino acid supplementation in patients with persistent encephalopathy or inadequate intake [23,24]. Structured low-intensity exercise, where tolerated, attenuates sarcopenia and improves quality of life. Salt restriction (≤2 g sodium/day) is central to ascites management [23].

Cholelithiasis (gallstone disease)

Cholelithiasis is strongly linked to obesity, the metabolic syndrome, refined carbohydrate intake, and rapid weight loss [4,25,26]. Prospective cohort studies and prevention trials support an inverse association between gallstone incidence and intake of dietary fiber, fruits and vegetables, nuts, fish (omega-3), and moderate coffee consumption [4,25,27]. Conversely, very-low-calorie diets (<800 kcal/day) and bariatric surgery substantially increase gallstone formation, with up to 25% of patients developing stones during rapid weight loss and 50% within six months of bypass surgery [26,28]. Gradual weight loss (0.4–0.9 kg/week), high-fiber diets, and consideration of ursodeoxycholic acid prophylaxis during rapid weight reduction protocols are key preventive measures [4,28].

Lifestyle and dietary interventions in chronic gastric disease

Gastroesophageal reflux disease (GERD)

GERD prevalence is closely tied to obesity, smoking, and dietary triggers. Lifestyle interventions — weight loss in overweight patients, head-of-bed elevation, avoidance of late evening meals (<3 hours before recumbency), and smoking cessation — improve symptoms and reduce esophageal acid exposure independently of acid-suppressive medication [5,29]. Specific dietary triggers — caffeine, chocolate, peppermint, alcohol, citrus, tomato products, and high-fat meals — reduce lower esophageal sphincter pressure or delay gastric emptying and warrant individualized avoidance [5,29].

Peptic ulcer disease (PUD)

Although peptic ulcer disease is largely attributable to *Helicobacter pylori* infection and NSAID use, lifestyle factors substantially influence ulcer development, healing, and recurrence [6,30]. Smoking is independently associated with delayed ulcer healing through reduced mucosal blood flow and increased acid secretion; alcohol promotes mucosal injury; and high-stress states contribute via neuroendocrine pathways [6]. Dietary recommendations emphasize a balanced, fiber-rich diet, moderation of caffeine

and spicy foods during active disease, and avoidance of NSAID self-medication where possible [6,30].

Helicobacter pylori-associated gastritis

Helicobacter pylori infection drives the majority of chronic gastritis worldwide and is causally linked to PUD, gastric adenocarcinoma, and MALT lymphoma [6,31]. Eradication therapy combining proton pump inhibitors with antibiotics remains standard, but rising antimicrobial resistance has prompted exploration of dietary and probiotic adjuncts [31,32]. Polyphenol-rich foods — particularly broccoli sprouts (sulforaphane), green tea catechins, cranberry, garlic, honey, and certain spices — demonstrate *in vitro* and clinical activity against *H. pylori* and improve gastric inflammation [31,32]. High dietary salt intake increases gastric cancer risk, particularly in *H. pylori*-infected individuals, and salt reduction is therefore a key public health measure [31,33]. Probiotic strains (*Lactobacillus* spp., *Bifidobacterium* spp., *Saccharomyces boulardii*) improve eradication rates and reduce antibiotic-associated adverse events when used as adjuncts to triple or quadruple therapy [32].

The gut–liver axis as a unifying mechanism

The gut–liver axis — the bidirectional anatomical, microbial, immunological, and metabolic communication between gut and liver via the portal vein — provides a unifying mechanism for the apparent overlap between hepatobiliary and gastric disease [7,34]. Dysbiosis of the gut microbiome, increased intestinal permeability ("leaky gut"), and translocation of bacterial endotoxins (lipopolysaccharide) drive low-grade hepatic inflammation, fibrogenesis, and altered bile acid metabolism. Dietary patterns rich in plant fibers and fermented foods support beneficial taxa (*Faecalibacterium prausnitzii*, *Akkermansia muciniphila*) and short-chain fatty acid production, with measurable downstream benefits on hepatic and gastric outcomes [7,34,35]. Targeted probiotic and prebiotic interventions are emerging as adjuncts in MASLD, *H. pylori* eradication, and gallstone prevention [7,32,35].

Biochemical markers for monitoring intervention efficacy

Routine biochemical markers enable objective, low-cost monitoring of lifestyle and dietary intervention efficacy across the disease spectrum. ALT and AST track hepatocellular injury and decline with weight loss, Mediterranean diet, and reduced alcohol intake [1,17,20]. GGT reflects oxidative stress and is particularly sensitive to alcohol and metabolic dysfunction [10,11]. FIB-4, calculated from age, AST, ALT, and platelets, provides a composite estimate of fibrosis and tracks longitudinal change with intervention [11]. Lipid panel, fasting glucose, and HbA1c monitor metabolic risk relevant to MASLD, gallstones, and GERD [1,17]. High-sensitivity C-reactive protein (hsCRP) reflects systemic low-grade inflammation and responds to anti-inflammatory dietary patterns [36]. In gastric disease, the urea breath test and stool *H. pylori* antigen confirm eradication, while symptom scores and proton pump inhibitor requirement track GERD and PUD response [5,6,31].

Table 2. Integrated nutrology framework for chronic hepatobiliary and gastric diseases.

Condition	Primary lifestyle/dietary goals	Adjunctive measures	Recommended monitoring
MASLD / NASH	≥7–10% weight loss;	Coffee; alcohol limit;	ALT, AST, FIB-4,

Condition	Primary lifestyle/dietary goals	Adjunctive measures	Recommended monitoring
	Mediterranean diet; ≥150 min/wk exercise	vitamin E in non-diabetics	lipids, HbA1c, TE every 6–12 mo
Chronic HBV / HCV	Mediterranean diet; alcohol abstinence; weight optimization	Vitamin D repletion; zinc if deficient; coffee	ALT, AST, FIB-4, HBV DNA/HCV RNA, TE annually
Cirrhosis	30–35 kcal/kg/d; 1.2–1.5 g protein/kg/d; sodium ≤2 g/d	Late-evening snack; BCAA; light exercise; micronutrients	Albumin, bilirubin, INR, MELD, ALBI, body composition
Cholelithiasis	Gradual weight loss; high fiber; low refined carbohydrate	Coffee; omega-3; UDCA during rapid weight loss	Lipid panel, ALP, GGT, ultrasound
GERD	Weight loss; smoking cessation; avoid late meals & triggers	Head-of-bed elevation; alcohol limit	Symptom score; PPI requirement; endoscopy as indicated
Peptic ulcer disease	Smoking cessation; alcohol moderation; NSAID avoidance	H. pylori eradication; balanced diet	Symptom score; H. pylori test; CBC if bleeding suspected
H. pylori gastritis	Salt reduction; polyphenol-rich plant foods; probiotic adjunct	Broccoli sprouts; green tea; cranberry	Urea breath test or stool antigen 4 weeks post-treatment

Note: *MASLD, metabolic dysfunction-associated steatotic liver disease; NASH, non-alcoholic steatohepatitis; HBV, hepatitis B virus; HCV, hepatitis C virus; GERD, gastroesophageal reflux disease; BCAA, branched-chain amino acids; UDCA, ursodeoxycholic acid; NSAID, non-steroidal anti-inflammatory drug; PPI, proton pump inhibitor; TE, transient elastography; MELD, model for end-stage liver disease; ALBI, albumin–bilirubin; CBC, complete blood count; ALP, alkaline phosphatase; GGT, gamma-glutamyl transpeptidase.*

Behavioral support, adherence, and digital tools

The clinical effectiveness of lifestyle and dietary interventions ultimately depends on adherence, which is consistently the most challenging element of management [1,2,17]. Multidisciplinary approaches involving nutrologists, dietitians, behavioral therapists, exercise specialists, and primary care physicians produce superior long-term outcomes compared with brief counseling [17,37]. Digital health tools — smartphone applications for dietary tracking, wearable activity monitors, telehealth follow-up, and remote biochemical monitoring — improve engagement and adherence in MASLD, cirrhosis surveillance, and post-bariatric care [17,37]. Equity, cultural adaptability, and access to whole foods must be considered when designing interventions for diverse populations [38].

Safety, limitations, and special populations

Lifestyle and dietary interventions are broadly safe but not without caveats. Very-low-calorie and crash diets precipitate gallstones and electrolyte disturbances [26,28]. Aggressive exercise programs in advanced cirrhosis must be individualized to avoid hypoglycemia, hyponatremia, and falls [23]. High-fiber diets are not appropriate during acute flares of PUD, GERD, or active gastritis. Patients with chronic kidney disease, those on anticoagulants, and those with restrictive eating disorders require tailored protocols and close monitoring. Cultural, religious, and economic factors significantly influence dietary feasibility and adherence and should be addressed proactively [38].

Discussion

A unified, nutrology-informed framework

The accumulated evidence supports a unified, nutrology-informed framework in which a small set of evidence-based lifestyle and dietary interventions — Mediterranean-style nutrition, gradual weight management, structured physical activity, alcohol moderation, tobacco cessation, and avoidance of ultra-processed foods — provides broad benefit across chronic hepatobiliary and gastric diseases [1,2,7,17]. This convergence reflects shared pathophysiology centered on chronic low-grade inflammation, metabolic dysregulation, and dysbiosis of the gut microbiome, all amenable to dietary modulation [7,34,35]. From a clinical standpoint, this allows nutrologists and primary care clinicians to deliver coherent, patient-centered recommendations rather than fragmented disease-by-disease prescriptions.

Biochemical markers as the link between intervention and outcome

Routine, low-cost biochemical markers — ALT, AST, GGT, FIB-4, lipid panel, HbA1c, hsCRP — provide objective measures of intervention efficacy and serve as motivational anchors for patients [10,11]. Their longitudinal monitoring permits early identification of non-response, allowing iterative refinement of dietary and behavioral strategies. Non-invasive imaging-based markers (transient elastography in hepatobiliary disease, ultrasound for gallbladder) further refine assessment without the cost or risk of invasive procedures. In gastric disease, simple symptom-based scores combined with eradication confirmation via breath or stool antigen complete the monitoring framework [5,6,31].

Methodological limitations of current evidence

Despite the breadth of the literature, several methodological limitations temper interpretation. Randomized controlled trials of lifestyle interventions are vulnerable to performance bias, inadequate blinding, high attrition, and variable adherence [13,17]. Dietary assessment relies largely on self-report instruments with known limitations. Many studies enroll motivated volunteers, reducing generalizability to routine clinical populations. Across hepatobiliary and gastric domains, surrogate endpoints (ALT, FIB-4, symptom scores) dominate over hard clinical endpoints such as decompensation, HCC, gastric cancer, or mortality [17,31]. Heterogeneity in intervention design, intensity, and duration limits cross-study synthesis. Few trials test integrated, multi-domain interventions against standard care with long-term follow-up.

Future research priorities include (i) pragmatic, multicenter randomized trials of unified lifestyle interventions across hepatobiliary and gastric conditions; (ii) integration of multi-omics data (microbiome, metabolomics, epigenomics) to enable precision nutrology; (iii) head-to-head comparisons

of digital health-supported versus traditional counseling; (iv) economic evaluations supporting scalable implementation; and (v) culturally adapted intervention models for global applicability.

Implications for nutrology and integrative practice

Operationalizing this framework requires interprofessional collaboration among nutrologists, hepatologists, gastroenterologists, primary care physicians, dietitians, and behavioral health specialists. A practical workflow includes structured nutritional and lifestyle assessment at diagnosis, individualized goal setting based on disease stage and patient preference, baseline and longitudinal biochemical monitoring, behavioral support and digital adherence tools, and coordinated escalation to pharmacological or surgical care when indicated [1,17,23,37]. Such an integrated approach aligns with contemporary models of preventive, personalized, and value-based medicine, and offers particular benefit in low- and middle-income settings where pharmacotherapy access is constrained but lifestyle intervention is universally feasible.

Conclusion

Lifestyle and dietary interventions — Mediterranean-style nutrition, gradual weight management, structured physical activity, alcohol moderation, tobacco cessation, and trigger-food avoidance — exert broad, biologically plausible, and clinically meaningful effects across chronic hepatobiliary and gastric diseases. In MASLD, viral hepatitis, cirrhosis, gallstone disease, GERD, peptic ulcer disease, and *Helicobacter pylori*-associated gastritis, these interventions consistently improve clinical, histological, and biochemical outcomes, often with effect sizes comparable to or complementing pharmacotherapy. Inexpensive, routinely available biochemical markers (ALT, AST, GGT, FIB-4, lipid panel, HbA1c, hsCRP, and eradication tests) enable objective monitoring of efficacy and individualized adjustment of intervention intensity.

Nevertheless, the field is constrained by adherence challenges, methodological heterogeneity, and the dominance of surrogate endpoints. Translating the considerable preclinical, epidemiological, and randomized evidence into routine practice requires rigorous, integrated trials, evidence-based protocols, and embedding of nutrology into multidisciplinary care. In the meantime, lifestyle and dietary interventions — supported by objective biochemical monitoring — represent a scientifically credible, patient-acceptable, and globally relevant component of contemporary management of chronic hepatobiliary and gastric diseases, fully consistent with an integrative, nutrology-informed model of care.

Acknowledgments

The authors thank the editorial team of the International Journal of Nutrology for their consideration of this manuscript, and acknowledge the broader community of researchers in hepatology, gastroenterology, and clinical nutrition whose primary work forms the basis of this review.

Funding

This review did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Ethical Approval

Not applicable. This review article is based exclusively on previously published literature and did not involve any new studies with human participants or animals performed by any of the authors.

Conflict of Interest

The authors declare that there is no conflict of interest regarding the publication of this article.

Author Contributions (CRediT)

Conceptualization, methodology, investigation, writing – original draft preparation, writing – review and editing: All authors. All authors have read and agreed to the submitted version of the manuscript.

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