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NED JOURNAL

ISSUE 09
DECEMBER 2025

Liver Health

Exploring nutrition and lifestyle interventions to support inflammatory biomarkers and liver enzymes

Diet and energy restriction may decrease inflammatory markers and liver enzymes and support liver health

High-intensity interval training reduces liver enzyme levels and improves MASLD-related biomarkers

Exploring the potential of oral butyrate, curcumin and probiotics on metabolic and inflammatory markers in MASLD

With special feature article on Liver Heath: Evidence based considerations

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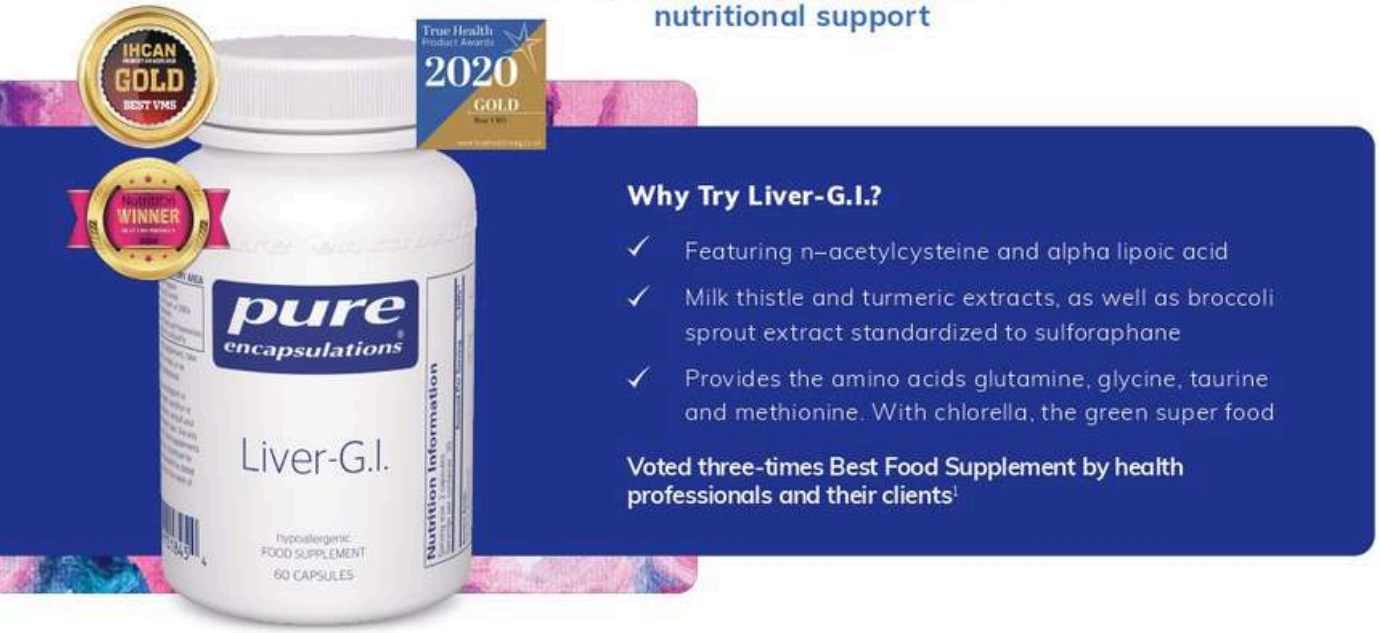

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NED is the UK's first scientific database dedicated to nutrition and lifestyle medicine research. It has been expertly curated to provide you with access to high-quality, human studies, designed to support evidence-based clinical decision making in personalised nutrition practice, and applied scientific-thinking in academic practice.

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WELCOME

Clare Grundel
Managing Editor

WELCOME TO THE NED LIVER ISSUE

Tickets now on sale!

The NED Science Forum returns. 12 May 2026 at The Royal Society of Medicine from 1pm.
This year, we will be:
"**Realising the Vision of Preventative Health - Translating Genetic and Wearable Data**".
Through a series of presentations, rooted in research and clinical practice, the NED Science Forum will demonstrate that true preventative health comes from diet and lifestyle. Embedding Registered Nutritional Therapists into primary care is the way to protect the future of the NHS and enable a population fit for life.
Tickets on sale now. Tickets available **here**
If you are a BANT member, join us for the AGM which precedes the NED Science Forum.

This 9th edition includes a **feature article on liver health**. Lead by NED Editor **Dr Michelle Barrow** and including contributions from members of the NED Expert Review Panel, it provides a narrative review of the science on nutrition, lifestyle and liver health. Involved in many processes, including metabolism, detoxification, digestion, energy regulation and nutrient storage, our livers are worth some attention when it comes to daily management of health. And that means looking at nutrition and lifestyle, the science of which NED is all about. Many thanks to Michelle for leading the team and to the expert reviewers for their valuable contributions. These feature articles are a great place to start to get an evidence-based overview of the topics.

We would like to extend our heartfelt thanks to **Pure Encapsulations** for partnering with NED as a premium sponsor. Their support allows us to develop and grow the database and we are profoundly grateful to them for believing in what we do.

If you don't have a NED account yet, **set one up here**. With an account, you get valuable extra functionality, including search history, saved searches, search alerts and tagging favourites.

With thanks to the expert reviewers who have written reviews for this edition and to the NED Editorial Board for their peer-review. Each review provides summary overviews of an article and clinical takeaways for you to apply to your own decision making with clients.

The **British Association of Nutrition and Lifestyle Medicine (BANT)** is a professional membership body for nutrition practitioners, trained in nutrition sciences and the delivery of personalised nutrition services. BANT members are reading and interpreting nutrition and lifestyle sciences such as that found in this NED Journal on a routine basis to inform their clinical decision making. You can find the BANT member practitioner listing **here**.

The Nutrition Evidence Database is one of the ways that BANT contributes to the evidence-based practice of precision nutrition. BANT is delighted to make this resource open access for all and encourages all healthcare practitioners interested in personalised healthcare to make use of the resource on a regular basis. You can sign up to an account here and subscribe to receive monthly NED alerts here.

Grab a cuppa and get into the lowdown on liver.

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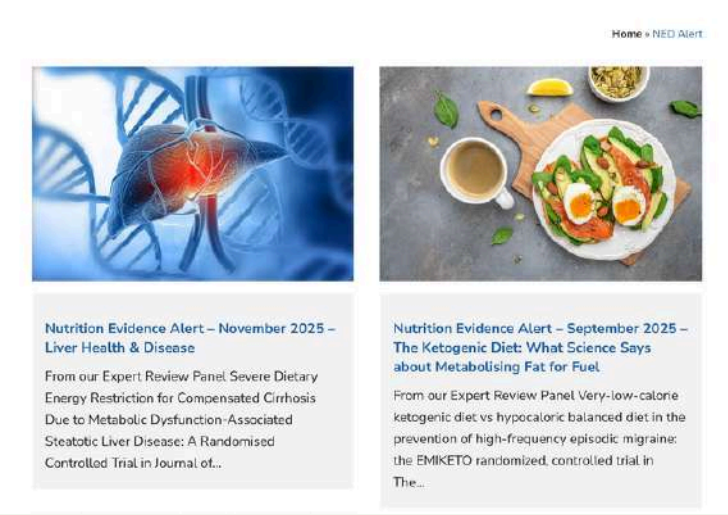
- Each month we publish a dedicated **Nutrition Evidence alert** with our editorial team's pick of the latest research and expert reviews. Sign up at here and have the science delivered straight to your inbox. Follow our socials for weekly posts on topics of interest.



If you have any questions or need support using NED, take a look at About Us and FAQs.
We'd also love to hear from you directly - you can reach us at
info@nutrition-evidence.co.uk

IN THIS ISSUE

MEET THE NED EDITORIAL BOARD

Dr Justin Roberts, Ph.D, SFHEA, FBANT **EDITOR-IN-CHIEF**

Professor Roberts is a Professor of Nutritional Physiology applied to exercise and functional health within the Cambridge Centre for Sport and Exercise Sciences, Anglia Ruskin University. He has published over 65 peer-reviewed, scientific articles and book chapters, and is a reviewer for numerous academic journals. His research focuses on nutritional strategies to promote metabolic flexibility and adaptive recovery in relation to exercise, including polyphenol and protein-targeted approaches, along with interests in pre-probiotic and food-based strategies to support gastrointestinal function.

Dr Michelle Barrow, DProf, MSc, SFHEA, FBANT, RNutr **LEAD AUTHOR**

Dr. Michelle Barrow is the Academic Team Director and Clinical Director at CNELM. Michelle also supervises PhD students at the University of Central Lancaster, Middlesex University and the University of West London. Michelle completed a Doctorate in Professional Studies (DProf) in 2019, titled "Leading transformation in Personalised Nutrition Practice". She is published in many scientific journals, including Autoimmunity Reviews, Nutrition Reviews and Current Research in Food and Nutrition.

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External lecturer at the ETH Zürich, Switzerland. Specialised in micronutrient nutrition and nutrition methodology meta-research. Widely published in the peer-reviewed, scientific press.

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Dr Kate Lawrence, BA(Hons), PhD, FHEA

Dr Lawrence is a Senior Lecturer in psychology at St Mary's University. Kate's research specialises in nutritional psychology and neurodiversity. She is widely published in scientific journals.

Clare Grundel, MANAGING EDITOR MSc, BA (Hons), MBANT

Clare is an experienced nutrition practitioner, and a regular speaker on BBC Radio Cambridge.Clare's area of clinical expertise is digestive health and chronic pain.

MEET THE NED EXPERT REVIEWERS

Our Expert Reviewers work within the nutrition industry in academia, research, clinical practice and wider healthcare, and provide unique and invaluable insights on the latest nutrition research to enable practitioners to apply the science to clinical practice.

Knowledge sharing is a key strategic pillar for the NED editorial team. Not only do the expert reviews get directly published on the NED database, they are further communicated via a series of monthly resources and across our BANT social media channels reaching in excess of 25,000 practitioners and followers.

EXPERT REVIEWERS IN THIS ISSUE (In order of appearance)

Karin Elgar, PhD, MBANT

Following the completion of a PhD in Physiology and a career in the pharmaceutical industry, Karin graduated as a nutritional therapist from the Institute of Optimum Nutrition in 2004. She has since been practising in the Greater Manchester area, specialising in women's health and autoimmunity. Karin has written a number of literature reviews and carried out a variety of research and editing projects. She has also delivered CPD seminars and webinars on various topics.

Sarah Cassar, MSc

Sarah is a Registered Nutritional Therapist with a Master's degree in Personalised Nutrition. With a strong background in education, she is committed to bridging the gap between nutrition science and practical application, empowering individuals and families to make informed dietary choices. She delivers educational sessions to children, adolescents, parents, and educators while collaborating with other healthcare professionals to promote holistic health strategies. Passionate about evidence-based nutritional practices, Sarah focuses on their impact on cognitive development, behavioural health, and overall well-being. She actively contributes to the field through research analysis, community engagement, and the indexing of scientific journals, striving to make nutrition education more accessible and impactful.

Chloe Steele, MSc, MBANT

Chloe has an MSc in Personalised Nutrition from the University of Middlesex, and specialises in cardiovascular disease, type 2 diabetes, and anxiety. Chloe started her career at BANT as a member of the Nutrition Evidence Database research team and now has over 5 years experience of research and writing. She has worked in several countries, and is currently in Australia, where she worked for Nutrition Australia and is currently the principal nutritionist for Heart Research Australia. She has published two papers in the Nutrition Medicine Journal, on gut microbiota and collagen. Chloe is a member of BANT and the Nutrition Society of Australia and sits on the editorial board for the Nutrition Medicine Institute in the UK.

Nicky Ester MSc, RNutr

Nicky received her Masters in Nutrition from University College Cork in Ireland. She also has a diploma in nutritional medicine and has trained as Natural Chef. She brings with her over 20 years' experience of working within the Health and Wellbeing sector, 10 years of which were spent in her own private clinical practice. Throughout her career she has given lectures to help increase the awareness of nutrition and its importance in relation to optimal health and well-being. She is passionate about empowering individuals to understand the role they play in their health in order to create meaningful and lasting change.

Ana-Paula Agrela, MSc

Ana is a Nutrition Consultant, and Health Coach who graduated with a BSc. (Hons) in Nutritional Science from Middlesex University and holds a Health Coaching certificate from Zest for Life. She completed her Master's degree in Holistic Health and Nutritional Education through Hawthorn University in the United States. Ana has over 20 years' experience in researching and developing health supplements for the nutraceutical industry. She also offers group education programs and private consultations to help clients make healthier food choices and lifestyle habits.

Anna Papoutsa, MSc, PGCertHE, MBANT

Anna is a Registered Nutritional Therapy Practitioner and a member of BANT, holding an MSc in Personalised Nutrition and a PGCert in Higher Education. Her dissertation delved into the intricate relationship between dietary refined carbohydrates and the onset of gout in overweight individuals, highlighting a beneficial role of magnesium in managing hyperuricaemia. Anna supports clients to optimise their health, with a particular focus on cardio-metabolic and immune function disorders. Beyond her clinical work, she teaches and lectures at the Centre for Nutrition Education & Lifestyle Management (CNELM).

Creative Editor

Claire Sambolino, MSc, ANLP, MBANT

Claire Sambolino is a PSA-accredited Registered Nutritional Therapy Practitioner with a passion for food provenance and sustainability. She completed an MSc in Personalised Nutrition in 2017, and runs online clinics in the UK and Italy specialising in metabolic health and integrative cancer nutrition. She is a certified MTIH Terrain Advocate ®, Metabolic Balance ® Consultant, NLP Coach, and Systems Approach to Cancer ® Programme enhanced cancer nutrition practitioner. Since 2007, Claire has been based in Italy with over 18 years lived-experience of the Mediterranean diet and Blue Zones.
As Creative Editor, Claire designs the layout for each NED Journal, overseeing the creative direction and realisation of the digital and printed formats, and diffusion across multi-media and social channels.

LIVER HEALTH SCIENCE TAKEAWAYS

NED INFOBITES & CLINICAL RESOURCES

Not yet discovered our one page science summaries? Our NED InfoBites are designed to provide quick overviews of some of the latest research available on particular health issues and nutrition topics. Designed as a one-page clinical handout, the NED InfoBites unite our editorial team's pick of the research and provide a plain-language summary suitable for sharing with nutrition clients. Download the latest InfoBites on Liver Health **here**.

Additionally, BANT has developed a dedicated range of resources to support practitioners and educate on common symptoms, biological processes, and dietary and lifestyle approaches to health and well-being. These are suitable to share with clients in clinical consultations and group programmes.

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Providing practitioners with health resources and client-friendly educational materials to support their clinical recommendations.

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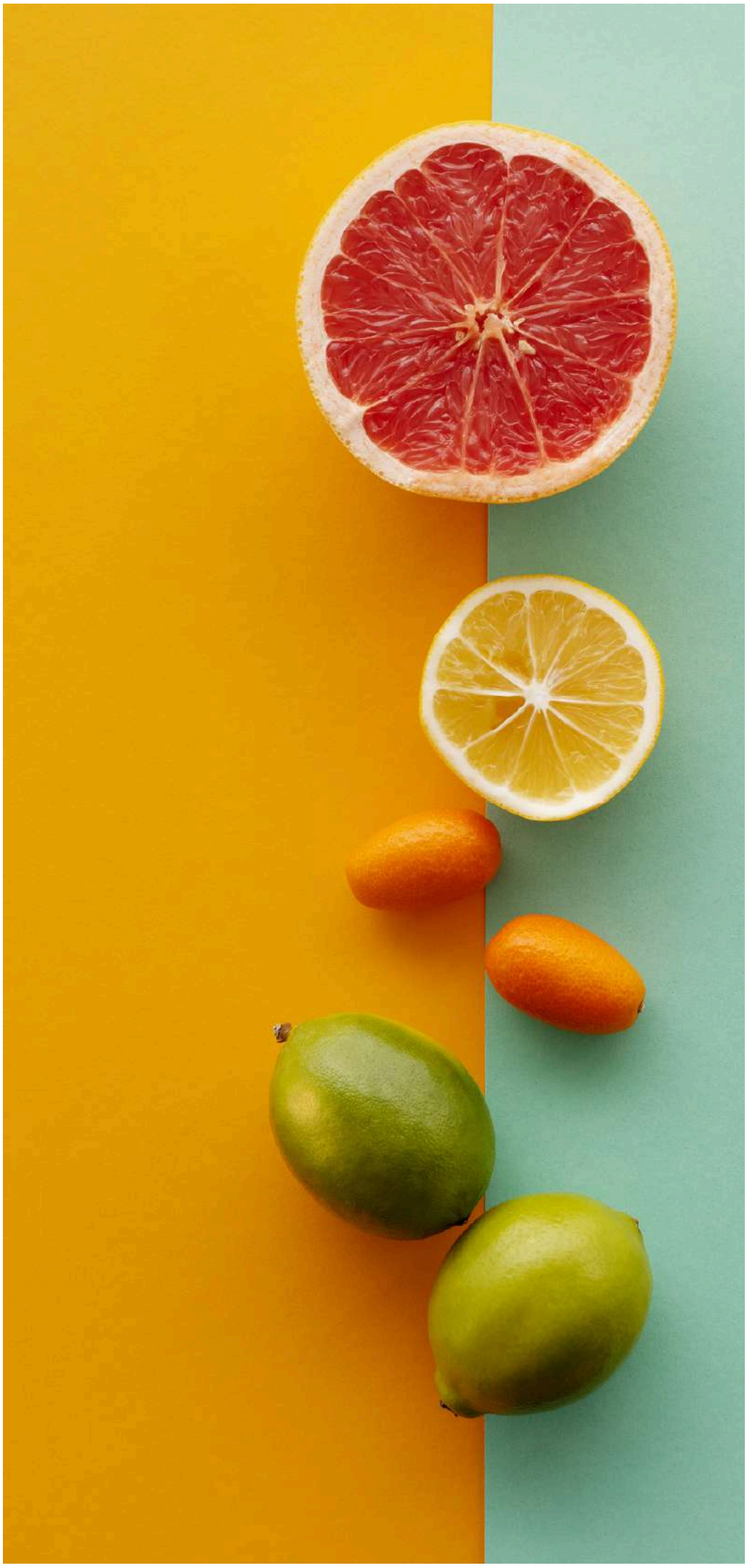
DIETARY PATTERNS & LIVER HEALTH

5 REVIEWS



Severe Dietary Energy Restriction for Compensated Cirrhosis Due to Metabolic Dysfunction-Associated Steatotic Liver Disease: A Randomised Controlled Trial

FERENC E MOZES, JEREMY F COBBOLD, FRANCESCA SAFFIOTI, ET AL.
JOURNAL OF CACHEXIA, SARCOPENIA AND MUSCLE 2025;16(3):E13783

TAKE HOME MESSAGE

Weight loss induced through low-energy meal replacement programmes may benefit patients with obesity and CC-MASLD in terms of liver fat reduction.

Read the article here

INTRODUCTION

- The aim of this study was to evaluate the signals of safety and efficacy of dietary energy restriction through meal replacements in patients with compensated cirrhosis due to metabolic dysfunction-associated steatotic liver disease (CC-MASLD).

METHOD

- Single-blind randomised controlled trial.
- 17 patients with CC-MASLD and a body mass index of ≥30 kg/m2 were enrolled.
- The intervention consisted of four meal replacements per day (880 kcal and 80g protein) for 16 weeks and gradual reintroduction of healthy food-based meals during weeks 17-22. For the 24 trial weeks participants were supported by a registered dietician.
- The control group received brief advice from their hepatologist on healthy eating.
- Primary outcomes assessed changes in liver enzymes, changes in iron-corrected T1 (cT1, a marker of fibro-inflammation) and changes in liver stiffness (magnetic resonance elastography).
- Secondary outcome measures included markers of severity of liver disease, physical performance, cardiometabolic and anthropometric parameters.

RESULTS

- 10 of 11 patients in the intervention and 5 of 6 patients in the control group completed the trial.
- There were no severe changes in liver enzymes in either group.
- At 24 weeks, the intervention group had significant improvements in cT1 (p=0.01), markers of liver fat (p=0.03) and controlled attenuation parameter (p=0.009, measure of hepatic steatosis) compared to the control group.
- There were no significant differences in liver stiffness, physical performance, liver frailty index or cardiometabolic markers between groups.
- The intervention group lost 11.9 kg more weight than the control group (CI −17.2 to −6.6). Whilst the absolute loss in fat mass was greater in the intervention group, the relative loss was lower (−3.2 (CI −6 to −0.3) and 5.4 (0.5 to 10.3), respectively).

HOW TO BRING THIS INTO YOUR CLINICAL PRACTICE

CLINICAL PRACTICE APPLICATIONS

- Low-energy meal replacement programme induced weight loss may benefit patients with obesity and CC-MASLD in terms of liver fat reduction.
- 52 people were invited to participate in this study. Of those, 35 (67%) declined with the primary reason being unwillingness to commit to the intervention - meal replacement programmes are not an easy or popular intervention.

CONSIDERATIONS FOR FUTURE RESEARCH

- Larger and longer-term clinical trials of weight loss through low-energy meal replacement programmes for patients with obesity and CC-MASLD are required to confirm the safety and efficacy of this approach.
-
- Impact on muscle mass should be assessed in future research

CONCLUSION

- Larger and longer-term clinical trials of weight loss through low-energy meal replacement programmes for patients with obesity and CC-MASLD are required to confirm the safety and efficacy of this approach.
- Impact on muscle mass should be assessed in future research.

EXPERT REVIEWER Karin Elgar PhD

CONFLICTS OF INTEREST: None
EVIDENCE CATEGORY: A: Meta-analyses, position-stands, randomized-controlled trials (RCTs)

The efficacy of DASH combined with time-restricted feeding (16/8) on metabolic associated fatty liver disease management: a randomized controlled trial

FNAVIDEH KHODADADI, HOSSEIN POUSTCHI, AMIR SADEGHI, ET AL.
JOURNAL: SCIENTIFIC REPORTS 2025;15(1):7020

INTRODUCTION

Metabolic associated fatty liver disease (MAFLD) is one of the most common chronic liver conditions. This study aimed to evaluate the effects of co-administration of dietary approaches to stop hypertension (DASH) and time-restricted feeding (TRF) on liver biomarkers (primary outcome), glucose homeostasis, lipid profile, inflammation and body composition (secondary outcomes) in patients with MAFLD.

METHOD

This was a randomised controlled trial. Patients who were diagnosed with MAFLD were stratified into groups by body mass index (BMI) and age. They were then randomly assigned to either the TRF (16/8) combined with the DASH group (n = 27) or the control group (n = 26). The control group was advised to adhere to weight loss diets.

RESULTS

After 12 weeks, the TRF + DASH group showed significantly greater reductions compared to the control group in:

Anthropometrics:

- BMI (P = .030)
- abdominal circumference (P = .005)
- **waist–hip ratio (P < .001).**

Liver health:

- alanine transaminase (ALT) (−15.23 ± 18.30 vs. −4.73 ± 13.12; P = .039)
- aspartate transaminase (AST) (−7.52 ± 8.31 vs. −3.47 ± 3.11; P = .047)
- gamma-glutamyltransferase (GGT) (−5.71 ± 12.46 vs. −1.03 ± 9.53)
- steatosis score (−68.57 ± 30.94 vs. −30.19 ± 29.49; P < .001)
- fibrosis score (−0.94 kPa; P = .001).

Lipids:

- triglycerides (171.00 ± 89.06 to 138.47 ± 92.06 mg/dL; P = .049).

Adjusted analyses confirmed that between-group differences in AST (P = .003) and fibrosis score (P = .004) remained significant after controlling for BMI and abdominal circumference.
Furthermore, there were no significant between-group changes in fasting blood sugar, HDL-C, total cholesterol, LDL-C, or hs-CRP (P > .05). Insulin and HOMA-IR improved in both groups. Most dietary and nutrient intake measures differed significantly between groups (except Selenium (P = .673) and Vitamin E (P = .545)), with the intervention group showing greater improvements in PUFA-ω6 intake (P = .013).

HOW TO BRING THIS INTO YOUR CLINICAL PRACTICE

- TRF + DASH diet may be able to reduce obesity indices and liver parameters in MAFLD patients
- The low energy density and high dietary fibre content of the DASH diet may help increase satiety and prevent overeating during eating hours.
- Simple lifestyle changes in meal timing and food quality can positively impact liver health in MAFLD patients.

Read the article here

CLINICAL PRACTICE APPLICATIONS

- Combining TRF (16/8) with a nutrient-dense DASH diet may be a more practical and sustainable dietary approach for improving liver and metabolic health in MAFLD patients.
- Improvements in liver enzymes and fibrosis markers suggest this approach could be used as part of a non-pharmacological management plan for supporting liver health.
- The intervention's impact on triglycerides highlights its potential role in addressing cardiovascular risk factors commonly associated with MAFLD.
- Nutrient density from the DASH diet (fibre, minerals, healthy fats) appears to support better satiety and adherence, addressing challenges often faced with fasting-based interventions.

CONSIDERATIONS FOR FUTURE RESEARCH

- Trials with a longer intervention period are needed to confirm the chronic effects of this type of combined diet and also to evaluate the level of continuous adherence to the TRF + DASH diet in the long term.
- Comparative studies with other intermittent fasting or dietary approaches could identify the most effective protocols.
- Adherence strategies (app-based reminders, meal planning tools) warrant testing for real-world sustainability and effectiveness.
- plain language summary logo

CONCLUSION

The authors concluded that the TRF + DASH dietary approach is more effective than a control diet in improving obesity-related measures and liver health in people with MAFLD. The DASH diet may enhance the benefits of TRF by improving satiety, reducing calorie intake, providing key nutrients, and helping to regulate fat storage in the liver.

EXPERT REVIEWER Sarah Cassar

CONFLICTS OF INTEREST: None
EVIDENCE CATEGORY: A: Meta-analyses, position-stands, randomized-controlled trials (RCTs)

The effects of intermittent fasting on antioxidant and inflammatory markers and liver enzymes in postmenopausal, overweight and obese women with rheumatoid arthritis: a randomized controlled trial

ARYAN TAVAKOLI, HASTIMANSOOREH ANSAR, ABDOLRAHMAN ROSTAMIAN, ET AL.
JOURNAL: SCIENTIFIC REPORTS 2025;15(1):2357

INTRODUCTION

The aim of this study was to evaluate the effects of intermittent fasting (IF) on inflammatory and antioxidant markers as well as liver enzymes in postmenopausal women with overweight/obesity and rheumatoid arthritis (RA).

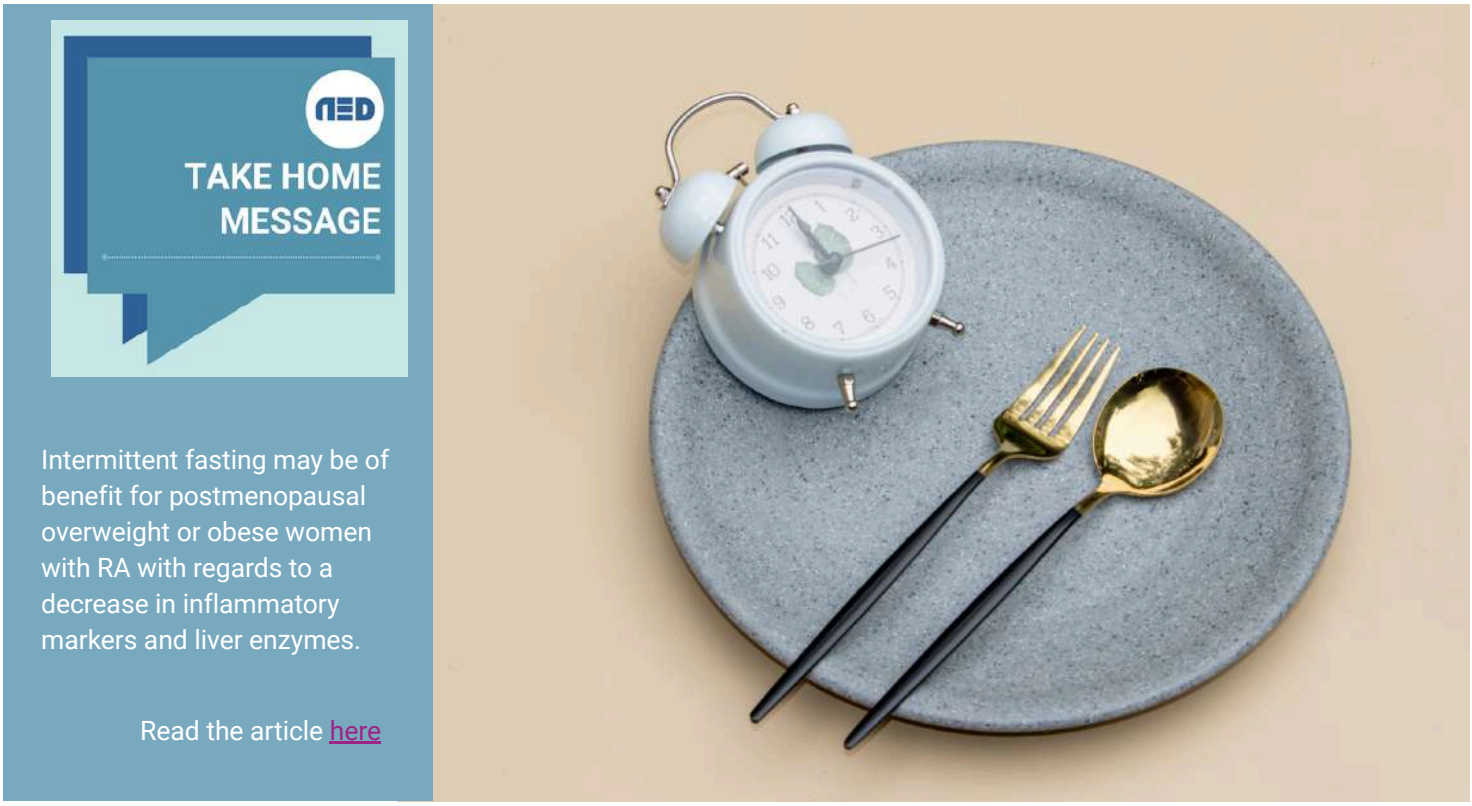
METHOD

- 8-week parallel randomised controlled trial of 44 postmenopausal women with RA and a body mass index of 25-35 kg/m_2.
- The intervention groups followed a 16:8 IF eating pattern (16 hours fasting, 8 hours eating window) with a 300 kcal per day caloric deficit. The control group followed a traditional eating pattern with three main meals and two snacks with standard healthy eating recommendations.
- Outcome measures included antioxidant and inflammatory markers (malondialdehyde (MDA), nitric oxide (NO), total antioxidant capacity (TAC), total oxidant status (TOS), catalase, myeloperoxidase, platelet/lymphocyte ratio and neutrophil to lymphocyte ratio), liver enzymes and creatinine.

RESULTS

- Three participants in each group dropped out of the trial, three in the intervention and two in the control group due to not adhering to the fasting/diet protocol, and one due to requiring surgery.
- Based on dietary food records, there were no significant differences in the intake of energy, macro- or micronutrients between the two groups over the study period (all p-values >0.05).
- After adjusting for baseline values, there were significantly greater reductions for MDA ($p<0.001$), NO ($p=0.05$) and neutrophil to lymphocyte ratio ($p=0.018$) in the IF group compared to controls and a significantly greater increase for catalase ($p=0.016$). A trend ($p=0.06$) for an increased TOS was also observed in the IF group. There were no significant differences between groups for myeloperoxidase, platelet/lymphocyte ratio or TAC.
- There were significantly greater reductions in AST and ALT ($p<0.001$ after adjustment) in the IF group, but no significant difference between groups for creatinine ($p=0.39$).

HOW TO BRING THIS INTO YOUR CLINICAL PRACTICE



Intermittent fasting may be of benefit for postmenopausal overweight or obese women with RA with regards to a decrease in inflammatory markers and liver enzymes.

Read the article here

CLINICAL PRACTICE APPLICATIONS

- Intermittent fasting/time restricted eating with a 300 kcal per day caloric deficit could be a valuable adjunct to the clinical management of postmenopausal women with RA and overweight/obesity.

CONSIDERATIONS FOR FUTURE RESEARCH

- Longer-term studies would be of value to determine the long-term efficacy and safety of, as well as compliance with, IF.
- Studies with larger sample sizes to increase statistical power would be of value.
- Such studies should also assess other outcomes such as disease severity scores and anthropometric parameters to determine clinical benefits.

CONCLUSION

The authors concluded that IF offers a benefit for this population in terms of both RA and associated metabolic complications.

EXPERT REVIEWER Karin Elgar PhD

CONFLICTS OF INTEREST: None
EVIDENCE CATEGORY: A: Meta-analyses, position-stands, randomized-controlled trials (RCTs)

Effects of a 12-Week Mediterranean-Type Time-Restricted Feeding Protocol in Patients With Metabolic Dysfunction-Associated Steatotic Liver Disease: A Randomised Controlled Trial-The 'CHRONO-NAFLD Project'

TRIADA BALI, SOFIA TSITSOU, EVANGELOS CHOLONGITAS, ET AL.
JOURNAL: ALIMENTARY PHARMACOLOGY & THERAPEUTICS 2025;61(8):1290-

TAKE HOME MESSAGE

Nutritional intervention is an effective way to help manage MASLD. • The MD may be easy to follow and an effective way to improve body weight, glucose metabolism, blood lipids, blood pressure, and liver steatosis but at the potential detriment to muscle.
Adding in eTRF or lTRF may respectively have additional benefits to glucose metabolism and blood lipid levels.

Read the article here

INTRODUCTION

Metabolic dysfunction-associated steatotic liver disease (MASLD) could be managed nutritionally through the adoption of a Mediterranean diet (MD) or the adoption of time restricted feeding (TRF).

This study aimed to determine the effects of 10-hour TRF in combination with the MD, compared to an unrestricted MD in obese and overweight individuals with MASLD and to assess whether the timing of TRF affects outcomes.

METHOD

- This was a randomised control trial of 59 individuals with MASLD and overweight/obesity split into:
- Control (n=19) – hypercaloric MD only
- Early TRF (eTRF) (n=20) – hypercaloric MD plus restricted eating between 8am-6pm.
- Late TRF (lTRF) (n=20) – hypercaloric MD plus restricted eating between 12pm-10pm.
- 12-week study.

RESULTS

- Compared to baseline:
- All groups exhibited similar body weight (7.6%–8.3%) and fat mass loss (14.8%–16.7%) by the end of the trial (P=>0.05). Muscle mass also decreased similarly across the groups.
- All groups had improved glucose metabolism. However, HOMA-IR (eTRF: P= 0.001, control: P=0.024), and Matsuda index (P=0.021, P=0.014), were not improved in the lTRF group.
- Lipids were different between the groups (P=<0.05), with control having improved low density lipoprotein (LDL) (P=0.002), and atherogenic index (P=0.046), and control and lTRF having improved total cholesterol (P=0.002 and P=0.009 respectively). These changes were not seen with eTRF.
- Systolic and diastolic blood pressure was improved in all groups (P=<0.05 for all).
- All groups showed improvements to liver steatosis (6.5%-9.5%) (P=<0.05 for all).

HOW TO BRING THIS INTO YOUR CLINICAL PRACTICE

CLINICAL PRACTICE APPLICATIONS

- The Mediterranean Diet may be an easy-to-follow diet that may benefit anthropometric and biochemical measurements in individuals with MASLD regardless of whether TRF protocols are followed.
- However, the addition of eTRF may have further benefits to glucose metabolism and lTRF may be of further benefit to blood lipid levels.

CONSIDERATIONS FOR FUTURE RESEARCH

- This study investigated individuals with MASLD and obesity. Future studies could investigate how the Mediterranean Diet and TRF affect other comorbidities regularly observed with MASLD such as type 2 diabetes, hypertension, and lipidaemia.
- Muscle loss was observed in all groups and the effects of combining the MD, TRF, and weight resistance exercise would be of interest.

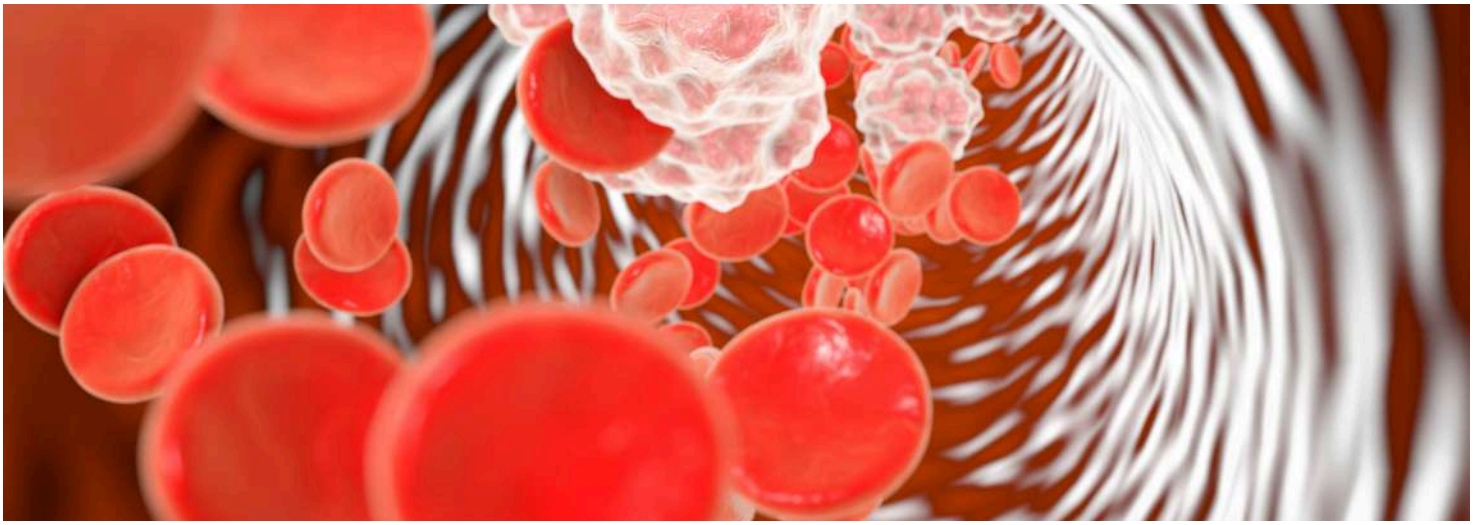
CONCLUSION

- The MD is of benefit to metabolic and anthropogenic outcomes in individuals with MASLD. Combining this with an eTRF pattern of eating could aid further improvements to glucose metabolism.

EXPERT REVIEWER Chloe Steele

CONFLICTS OF INTEREST: None
EVIDENCE CATEGORY: B: Systematic reviews including RCTs of limited number

Dietary inflammatory index and non-alcoholic fatty liver disease risk: a systematic review and meta-analysis of observational studies



AZAM DOUSTMOHAMMADIAN, BAHAREH AMIRKALALI, ALI GHOLAMI, ET AL.
JOURNAL: FRONTIERS IN NUTRITION 2025;12():1596300

INTRODUCTION

This study aimed to examine the association between the Dietary Inflammatory Index (DII) and non-alcoholic fatty liver disease (NAFLD) risk.

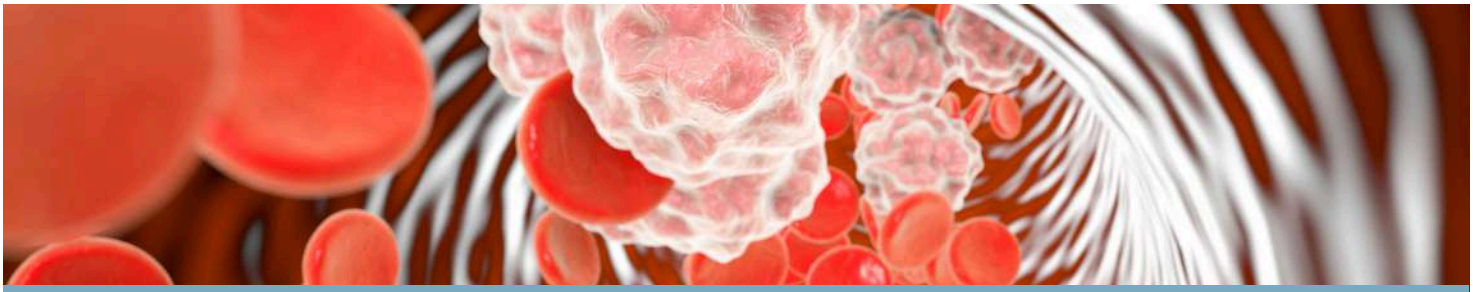
METHOD

This was a meta-analysis of eleven observational studies amongst which nine were cross-sectional (n= 82,974) and two cohort (n= 184,421) studies. It followed the PRISMA guidelines and is registered with PROSPERO.

RESULTS

- Overall study quality was acceptable, with most included studies rated as moderate to high quality, strengthening confidence in the observed associations despite inherent limitations of observational designs.
- Results showed that:
- higher DII scores were associated with significantly greater NAFLD risk in cross-sectional studies (OR = 1.56; 95% CI 1.24–1.95; $p < 0.001$; $I^2 = 86.9\%$).
- cohort studies showed a significant association between dietary inflammatory potential and NAFLD incidence (HR = 0.21; 95% CI 0.12–0.30; $p < 0.0001$), reflecting lower risk among individuals with less pro-inflammatory diets.
- subgroup analyses (body mass index, age, region, NAFLD diagnostic method, DII scoring approach) consistently supported the association between higher DII and increased NAFLD risk (all $p < 0.05$).
- certainty of evidence was rated as very low due to high heterogeneity, observational study designs, and variation in exposure and outcome measurements.

HOW TO BRING THIS INTO YOUR CLINICAL PRACTICE



TAKE HOME MESSAGE

- Diets high in pro-inflammatory foods are associated with a greater likelihood of developing NAFLD.
- Eating patterns rich in anti-inflammatory foods such as fruits, vegetables, whole grains, legumes, nuts, and olive oil, may help support liver health.
- This meta-analysis was of observational studies assessed as very low evidence certainty using the GRADE framework. It therefore has limitations and is unable to preclude causal relationships between NAFLD and DII.

Read the article here

CLINICAL PRACTICE APPLICATIONS

- Reducing the inflammatory potential of the diet, by increasing anti-inflammatory foods (e.g., fruits, vegetables, whole grains, omega-3 rich foods) and limiting pro-inflammatory items (e.g., refined carbohydrates, processed meats and added sugars), may help lower NAFLD risk in at-risk individuals.

- Incorporating DII-based dietary assessment tools into routine nutritional evaluations may support early identification of individuals consuming highly pro-inflammatory diets.

CONSIDERATIONS FOR FUTURE RESEARCH

- Longitudinal cohort studies and well-designed randomised controlled trials are needed to clarify causal relationships between dietary inflammatory potential and NAFLD risk.

- Future studies should standardise DII assessment methods and NAFLD diagnostic criteria to improve comparability across populations and research settings.

- Reducing publication bias through comprehensive reporting and inclusion of null findings will help strengthen the reliability of the overall evidence base.

CONCLUSION

- The authors concluded that higher DII scores are associated with increased NAFLD risk.

EXPERT REVIEWER Sarah Cassar

CONFLICTS OF INTEREST: None
EVIDENCE CATEGORY: A: Meta-analyses, position-stands, randomized-controlled trials (RCTs)

MEDITERRANEAN DIET

MEDITERRANEAN DIET RESOURCES

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Mediterranean Diet

A recognised healthy eating pattern for cardiometabolic health & longevity

What is the Mediterranean diet?

The Mediterranean diet is renowned for its numerous health benefits, primarily stemming from its rich composition of minimally processed plant foods, healthy fats, and moderate consumption of certain animal products. Central to this dietary pattern is the use of olive oil as the main source of fat, which is linked to a low mortality rate for cardiovascular diseases (Article: "Olive oil and cardiovascular health"). The diet's emphasis on fruits, vegetables, whole grains, legumes, and nuts, combined with moderate wine consumption, particularly resonates with its ability to reduce risks related to cardiovascular diseases, as strong evidence from large randomised controlled trials suggests (Article: "The Mediterranean diet and cardiovascular disease").

Furthermore, the Mediterranean diet is also associated with increased life expectancy and improved quality of life. This is highlighted in studies that show its ability to reduce the risk of myocardial infarction and stroke, alongside other major chronic diseases (Article: "Mediterranean diet and life expectancy: beyond olive oil, fruits, and vegetables"). The antioxidant-rich nature of this diet, bolstered by foods like wild greens that are high in flavonoids, is thought to be a contributing factor to the longer survival associated with this diet (Article: "Mediterranean diet and longevity"). Collectively, these studies underscore the Mediterranean diet as a sustainable and appealing dietary strategy with broad-ranging benefits for health and longevity.

The Mediterranean Diet (MedDiet) Pyramid is an evolving framework that translates dietary guidance into practical recommendations. It has been enriched over time to address issues such as obesity and chronic diseases by emphasising the plant-based core of the diet, frugality, and moderation. The latest version not only considers the nutritional aspect but also the cultural and lifestyle dimensions, such as conviviality, physical activity, and adequate rest, which are integral to the Mediterranean lifestyle.

The pyramid serves as a tool for deriving serving sizes and evaluating the intake of food groups. It offers guidance on protective food groups and advocates for the consumption patterns that help to mitigate noncommunicable diseases. It puts emphasis on balance and variety, and plays a significant role in improving public health outcomes and promoting longevity.

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Mediterranean Diet Pyramid

A way of living that can contribute to cardiometabolic health, healthy ageing and longevity

The Mediterranean Diet (MedDiet) Pyramid is an evolving framework that translates dietary guidance into practical recommendations. It has been adapted to reflect modern nutritional and lifestyle needs while preserving its core principles.

Mediterranean Pyramid Principles

Drink water

Moderate alcohol

Sweets
Gelati, Cakes, Desserts

Animal Proteins
Poultry, Meat
Eggs
Cheese, Yoghurt

Fish & Seafood
Fish (sea bass, salmon, swordfish, tuna, sardines, anchovies), Octopus, Mussels, Clams, Prawns, Calamari

Plant-based Whole Ingredients
Extra Virgin Olive Oil, Seasonal Fruits & Vegetables, Legumes, Whole & Antique Grains, Nuts, Seeds, Fresh Herbs, Spices

Physical Activity & Community
Lifestyle benefits of being physically active
'sense of purpose' from community

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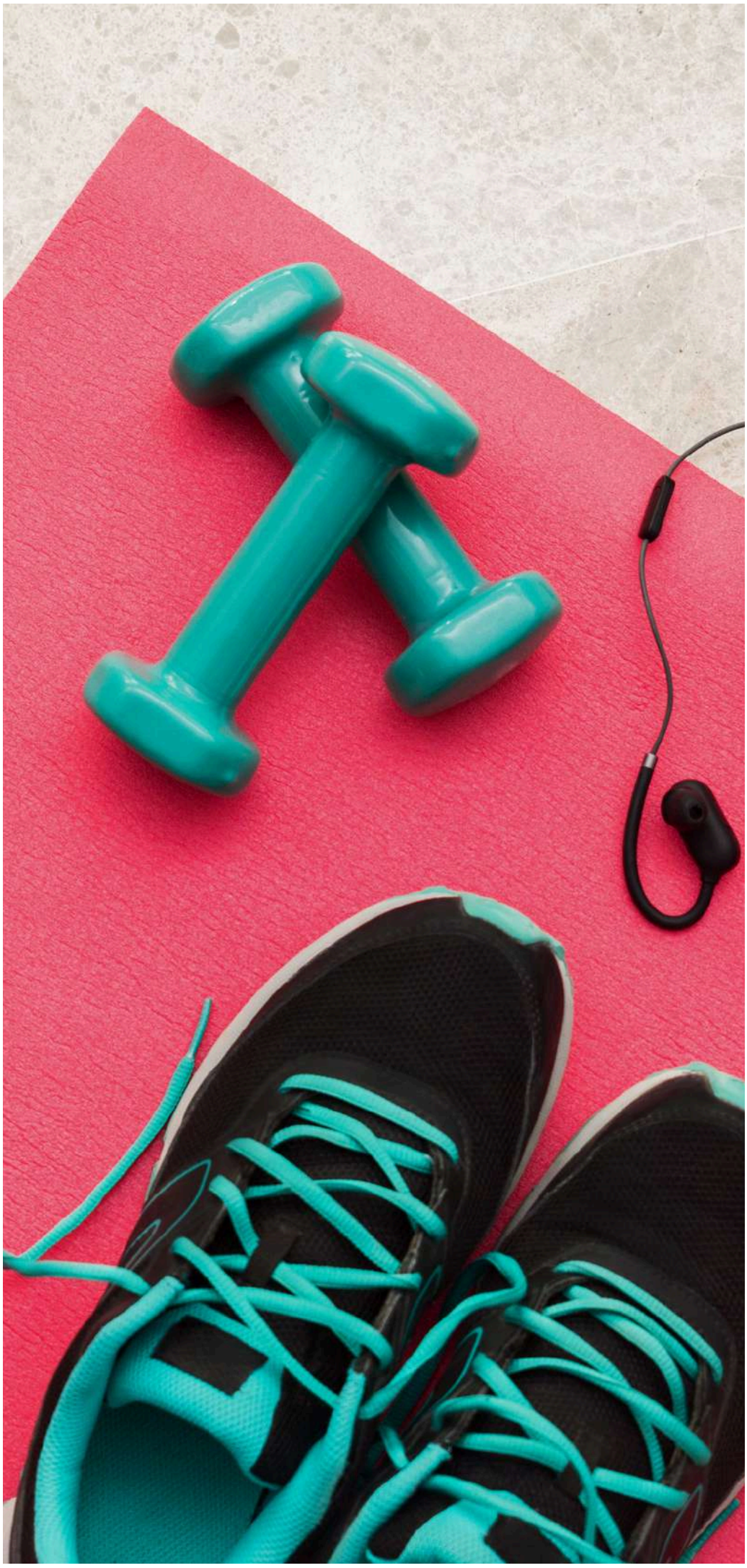
Access further resources here

EXERCISE & ITS ROLE LIVER HEALTH



2 REVIEWS

High-Intensity Interval Training Reduces Liver Enzyme Levels and Improves MASLD-Related Biomarkers in Overweight/Obese Girls

WISSAL ABASSI, NIDHAL JEBABLI, NEJMEDDINE OUERGHI, ET AL.
JOURNAL: NUTRIENTS 2025;17(1):

INTRODUCTION

Metabolic dysfunction-associated steatotic liver disease (MASLD) is characterised by the presence of hepatic steatosis with the onset of metabolic risk factors. This study aimed to evaluate the effects of high-intensity interval training (HIIT) on selected MASLD-related biomarkers in adolescents with overweight/obesity.

METHOD

This was a nine-week randomised controlled trial that enrolled thirty-six girls who were randomly assigned to the training (n = 18) or control (n = 18) groups. Girls in the control group continued their regular physical education classes but did not participate in additional training programmes.

RESULTS

- At baseline, there were no significant differences between groups.
- Anthropometric and physical outcomes (HIIT group): Significant improvements were observed in body mass ($p < .001$; ES = 0.25), BMI ($p = .001$; ES = 0.29), body fat ($p < .001$; ES = 0.52), waist circumference ($p = .003$; ES = 0.37), and systolic blood pressure ($p = .011$; ES = 0.80). Maximal aerobic speed increased ($p = .035$; ES = 0.54) and maximal heart rate improved ($p = .035$; ES = 0.73). No changes occurred in the control group.
- Between-group comparisons: The HIIT group demonstrated higher maximal aerobic speed ($p = .010$; ES = 0.99, large effect) and lower maximal heart rate ($p < .001$; ES = 1.40, large effect) versus controls.
- Biochemical outcomes (HIIT group): Significant reductions occurred in total cholesterol ($p = .003$; ES = 0.45), triglycerides ($p = .005$; ES = 0.54), low-density lipoprotein cholesterol ($p = .002$; ES = 0.60), alanine transaminase ($p = .013$; ES = 0.47), aspartate aminotransferase (AST) ($p = .012$; ES = 0.58), and HOMA-IR ($p = .010$; ES = 0.47). High-density lipoprotein cholesterol increased ($p = .013$; ES = 0.36).
- Between-group comparisons: AST was significantly lower in the HIIT group ($p = .036$; ES = 0.79, moderate effect).

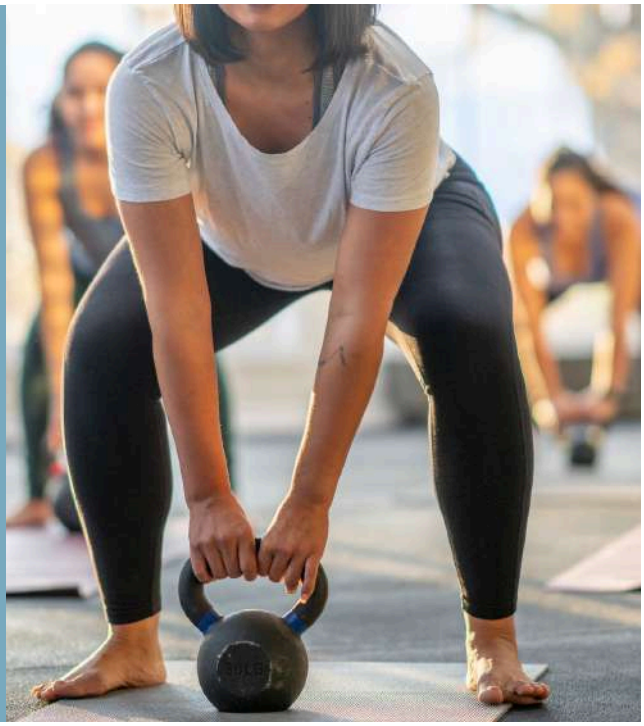
HOW TO BRING THIS INTO YOUR CLINICAL PRACTICE

- Short-term HIIT programmes may provide measurable benefits for overweight/obese adolescents, particularly in body composition and metabolic markers.
- Improvements in liver enzymes and lipids observed in this trial...

...suggest that HIIT could play a supportive role in MASLD management.

Structured exercise interventions in adolescence might help reduce longer-term risk of cardiometabolic complications.

Read the article here



CLINICAL PRACTICE APPLICATIONS

- HIIT can be considered as a structured, evidence-based exercise plan alongside dietary and lifestyle interventions for overweight/obese adolescents with MASLD.
- Incorporating short HIIT sessions may be more acceptable and feasible for young people compared to longer exercise regimens.
- Improvements in liver enzymes, blood pressure, lipids and insulin resistance highlight the importance of integrating physical activity into MASLD management plans.

CONSIDERATIONS FOR FUTURE RESEARCH

- Further research is needed to clarify the mechanisms underlying the effects of HIIT on fatty liver while focusing on the potential direct action of muscle metabolism on the liver.
- Future research should include larger, more diverse populations and both sexes to explore gender differences.
- Studies should also control for diet and energy intake, monitor adherence to HIIT, and assess pubertal hormones that may affect outcomes.

CONCLUSION

The authors concluded that a nine-week HIIT programme improves body composition and reduces liver enzymes, plasma lipids, blood pressure, and insulin resistance in over-weight/obese adolescent girls.

EXPERT REVIEWER Sarah Cassar

CONFLICTS OF INTEREST: None
EVIDENCE CATEGORY: A: Meta-analyses, position-stands, randomized-controlled trials (RCTs)

Effects of Mediterranean diet, exercise, and their combination on body composition and liver outcomes in metabolic dysfunction-associated steatotic liver disease: a systematic review and meta-analysis of randomized controlled trials

JULIETH PILAR URIZA-PINZÓN, SARA BEIGREZAEI, OSCAR H FRANCO ET AL.
JOURNAL: BMC MEDICINE 2025;23(1):502

INTRODUCTION

This study aimed to assess the effect of the Mediterranean diet (MD), exercise, and their combination on anthropometric and liver outcomes in patients with metabolic dysfunction-associated steatotic liver disease / metabolic dysfunction-associated steatohepatitis (MASLD/MASH).

METHOD

This was a systematic review and meta-analysis of thirty-seven randomised controlled trials. Among the included articles eleven assessed the MD, twenty-seven assessed exercise, and two assessed the combination of the MD and exercise.

RESULTS

In comparison to control groups:

A. MD alone:

- Weight: Significant reduction (WMD = −2.38 kg; 95% CI −4.11 to −0.66; P = 0.01).
- Body mass index: Significant decrease (WMD = −0.70 kg/m²; 95% CI −1.03 to −0.36; P < 0.001).
- Waist Circumference: Notable reduction (WMD = −1.56 cm; 95% CI −3.02 to −0.09; P = 0.04)
- Liver Enzymes: Alanine transaminase (ALT) was significantly reduced (WMD = −3.96 IU/L; 95% CI −6.54 to −1.38; P < 0.001). No significant changes on aspartate transaminase (AST) (WMD = −2.14 IU/L; 95% CI −5.63 to 1.34; P = 0.23) or gamma-glutamyl transferase (GGT) (WMD = −3.55 IU/L; 95% CI −11.57 to 4.47; P = 0.39).
- Liver fat and fibrosis: The majority of RCTs reported significant improvements in fatty liver index, liver steatosis, and fibrosis outcomes compared to control or low-fat diets.

B. Exercise interventions:

- Weight: Aerobic (WMD = −1.56 kg; 95% CI −2.31 to −0.82; P < 0.001; Power > 85%) and combined aerobic-resistance (WMD = −1.90 kg; 95% CI −3.59 to −0.22; P = 0.03) both effective.
- Liver enzymes: Resistance exercise significantly reduced ALT (WMD = −15.40 IU/L; 95% CI −28.60 to −2.20; P < 0.001); AST or GGT remained unchanged.
- Liver fat: Aerobic and combined exercise consistently decreased intrahepatic fat; resistance training effects were variable.
- Fibrosis: Mostly unchanged, except high-intensity interval training showing a significant NFS reduction (P < 0.05).

C. Combined MD + Exercise:

- Both interventions improved intrahepatic fat, though group differences were not statistically significant.

HOW TO BRING THIS INTO YOUR CLINICAL PRACTICE

TAKE HOME MESSAGE

- Lifestyle interventions remain essential in preventing and managing liver fat accumulation.
- Consistent adherence to dietary patterns and exercise protocols offers measurable metabolic and hepatic benefits.
- The MD and aerobic exercise may improve weight and liver health in people with MASLD/MASH.

Read the article here

CLINICAL PRACTICE APPLICATIONS

- The MD can be recommended as a practical dietary strategy to support weight reduction and improve liver function in patients with MASLD/MASH. Encouraging regular aerobic exercise as part of the intervention may further enhance hepatic enzyme profiles and reduce intrahepatic fat content.
- Monitoring changes in body weight, body mass index, waist circumference, and ALT levels can serve as useful clinical markers for evaluating intervention effectiveness.

CONSIDERATIONS FOR FUTURE RESEARCH

Future high-quality, large-scale RCTs employing standardised definitions of the MD, harmonised intervention protocols, and extended follow-up periods are needed to better determine the effects of lifestyle interventions on body composition and liver outcomes in MASLD.

Longer-term studies are needed to determine the sustainability of MD and exercise-induced improvements in hepatic steatosis and fibrosis.

Standardisation of liver outcome measures may enhance data comparability across trials.

Investigations into behavioural and adherence factors influencing the effectiveness of lifestyle interventions could inform tailored, patient-centred strategies.

CONCLUSION

- The authors concluded that MD and aerobic exercise independently contribute to significant weight reduction and improvements in hepatic function among patients with MASLD/MASH.

EXPERT REVIEWER Sarah Cassar

CONFLICTS OF INTEREST: None
EVIDENCE CATEGORY: A: Meta-analyses, position-stands, randomized-controlled trials (RCTs)

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SUPPLEMENTATION

3 REVIEWS

Exploring the Potential of Oral Butyrate Supplementation in Metabolic Dysfunction-Associated Steatotic Liver Disease: Subgroup Insights from an Interventional Study

MILOŠ MITROVIĆ, PETAR SVORCAN, SANJA ERCEG, ET AL.
JOURNAL: INTERNATIONAL JOURNAL OF MOLECULAR SCIENCES 2025;26(12):

TAKE HOME MESSAGE

Whilst improvements to hepatic steatosis were undetected following butyrate supplementation, biochemical markers of inflammation and liver function were improved.

Individuals with a lower BMI, higher CRP and higher HbA1c are more likely to see benefits following butyrate supplementation, however these benefits may only be small.

Read the article here

INTRODUCTION

Metabolic dysfunction-associated steatotic liver disease (MASLD) is a disease associated with the occurrence of obesity, cardiovascular disease, and diabetes mellitus. Whilst the pathogenesis is still being investigated, it has been hypothesised that gut dysbiosis may be involved. Short chain fatty acids (SCFAs) such as butyrate associated with healthy gut bacteria have been shown to act on the liver, reducing the production of lipids and decreasing hepatic steatosis. This study aimed to determine any benefits and safety of butyrate supplementation in individuals with MASLD.

METHOD

- This was a randomised control trial (RCT) of 181 individuals with MASLD and at least one of obesity, hypertension, or dyslipidaemia.
- Participants were split into 2 groups; 121 individuals received 1000mg sodium butyrate plus diet plan and 60 received 1000mg calcium butyrate plus diet plan due to the unavailability of calcium butyrate. Both groups reached statistical power.
- Study duration was 12-weeks.
- Primary endpoint was change in liver steatosis measured using the Controlled Attenuation Parameter (CAP) via FibroScan®.
- Secondary endpoints included liver stiffness, biochemical measures, hepatic steatosis and fatty liver indices, faecal calprotectin levels, stool SCFA levels, and microbiome composition.

RESULTS

- The results showed that:
- There were no significant changes in CAP (ΔCAP: sodium butyrate, 0.84; calcium butyrate, −0.23; p = 0.70).
- Sodium butyrate did improve some biochemical indicators of liver function; serum trimethylamine N-oxide (TMAO) (P=0.021) and fatty liver index (P=0.047).
- Calcium butyrate significantly improved calprotectin levels (P=0.031).
- Subgroup analysis showed that responders to butyrate were more likely to have a lower body mass index (BMI) (26.1 ± 1.7 vs 27.8 ± 1.7; P= < 0.001), higher C-reactive protein (7.7 ± 4.2 mg/L vs 4.9 ± 4.4 mg/L; P= 0.006), higher HbA1c (6.7 ± 0.4 vs. 6.4 ± 0.5, P = 0.037) and a lower abundance of the gut microbiota Subdoligranulum and higher Catenibacterium.

HOW TO BRING THIS INTO YOUR CLINICAL PRACTICE

CLINICAL PRACTICE APPLICATIONS

- Butyrate supplementation may not directly affect liver steatosis, but it may be of benefit to liver function and inflammation.
- Individuals with a lower BMI, inflammation, and higher HbA1c are likely to see the greatest benefits.

CONSIDERATIONS FOR FUTURE RESEARCH

- 12-weeks may be an insufficient study duration for effects to be seen on liver steatosis. Given that biochemical markers of liver function were improved, longer durations are warranted.
- CAP may be insufficient to detect small hepatic changes and more sensitive imaging may be requir

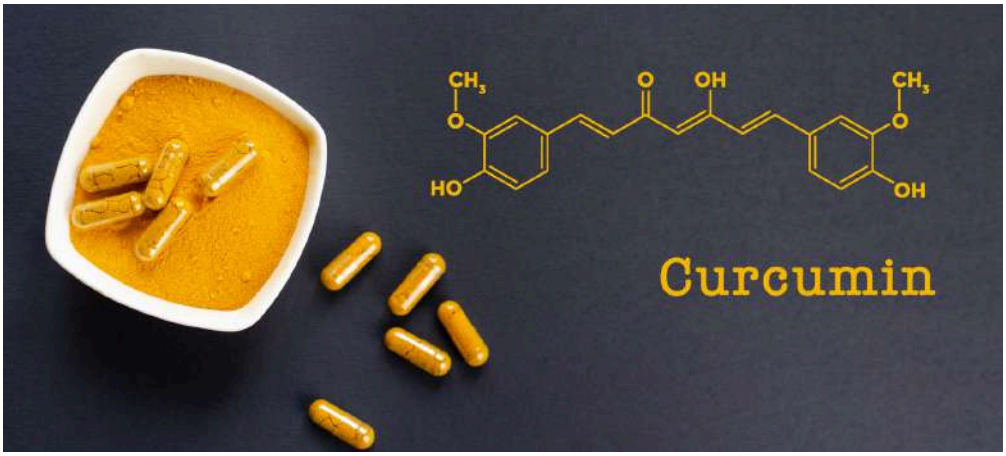
CONCLUSION

- Neither sodium nor calcium butyrate improved liver steatosis, as measured by CAP. However, the effects on metabolic and inflammatory markers indicates that specific individuals with MASLD may benefit.

EXPERT REVIEWER Chloe Steele

CONFLICTS OF INTEREST: None
EVIDENCE CATEGORY: B: Systematic reviews including RCTs of limited number

Curcumin for Inflammation Control in Individuals with Type 2 Diabetes Mellitus and Metabolic Dysfunction-Associated Steatotic Liver Disease: A Randomized Controlled Trial



METHA YAIKWAWONG, LADDAWAN JANSARIKIT, SIWANON JIRAWATNOTAI, ET AL.
JOURNAL: NUTRIENTS 2025;17(12):

TAKE HOME MESSAGE

Curcumin supplementation, alongside diet and lifestyle interventions, could be of benefit for patients with MASLD with regards to:

- Oxidative stress
- MASLD markers
- Anthropometric parameters.

Read the article here

INTRODUCTION

- The aim of this study was to evaluate the effects of curcumin supplementation on inflammatory, glycaemic and hepatic markers in patients with metabolic dysfunction-associated steatotic liver disease (MASLD).

METHOD

- Double-blind, randomised controlled trial of 78 patients with type 2 diabetes mellitus and MASLD, conducted in Thailand.
- Intervention: 750 mg curcuminoids twice a day or placebo for 12 months.
- All participants received diet and lifestyle education for 3 months prior to start of the intervention and took metformin to manage glucose levels.
- Primary outcome was tumour necrosis factor (TNF), secondary outcomes included controlled attenuated parameter (CAP), liver stiffness, anthropometric parameters, inflammatory, hepatic, glycaemic, lipid and oxidative stress markers. All outcome measures were taken every three months.
- Safety was assessed through regular kidney and liver function tests.

RESULTS

At 12 months, compared to placebo, curcumin significantly improved levels of TNF, interleukin (IL)-1β, IL-6, glutathione peroxidase, superoxide dismutase, malondialdehyde, glucose, waist circumference, fatty liver index, lipid accumulation product, liver stiffness and CAP (all $p<0.001$). Significant improvements compared to placebo were also observed for total body fat ($p=0.001$), body mass index ($p=0.002$), non-esterified fatty acids ($p=0.002$), HbA1C ($p=0.003$) and hepatic steatosis index ($p=0.01$). Most markers had significantly improved at 3 months.

- There were no differences between the groups in kidney and liver function tests, and no episodes of hypoglycaemia were reported in the curcumin group.

HOW TO BRING THIS INTO YOUR CLINICAL PRACTICE

CLINICAL PRACTICE APPLICATIONS

- Curcumin supplementation could be considered in patients with MASLD alongside diet and lifestyle interventions.

CONSIDERATIONS FOR FUTURE RESEARCH

- Similar clinical trials in other locations/with other ethnic groups would help confirm generalisability of these study results.
- Trials investigating both acute (<12 weeks) and long-term supplementation (>1 year) to increase knowledge of acute versus long-term effects.

CONCLUSION

- The authors concluded that curcumin offers a safe and effective intervention for patients with MASLD.

EXPERT REVIEWER Karin Elgar PhD

CONFLICTS OF INTEREST: None
EVIDENCE CATEGORY: A: Meta-analyses, position-stands, randomized-controlled trials (RCTs)

Meta-analysis of probiotics efficacy in the treatment of minimum hepatic encephalopathy

MARTHA

INTRODUCTION

- Minimum hepatic encephalopathy (MHE) is frequently missed as it is hard to diagnose, yet it occurs in 20-80% of cirrhosis patients.
- Without treatment the risk of cognitive decline as well as mortality rate increases by 50%.
- The study aimed to evaluate current evidence on the outcome of treating MHE with probiotics.

METHOD

- A systematic search of PubMed, China National Knowledge Infrastructure and Wanfang was conducted for RCT's up to March 2023.
- Of 361 studies, 18 RCTs (n=1226, ages 40-60) were included. Study duration ranged from one to three months.
- Search terms included "probiotics", "Lactococcus lactis", "Bacillus subtilis", "Lactobacilli", "Lactobacillus","Saccharomyces", "Bifidobacterium", "minimal hepatic encephalopathy", "overt hepatic encephalopathy (OHE)" and related terms.
- All research were RCT's investigating mild or subclinical HE where probiotics compared to a placebo or blank control.
- Exclusion criteria were non populations studies, conference articles, case reports, reviews and non-RCT's or insufficient data.
- Outcome measures: blood ammonia level, alanine aminotransferase level (ALT)) and Model for End-Stage Liver Disease (MELD) score, MHE remission rate, MHE incidence and a psychometric test, specifically number connection test A (NCT A) and digit symbol test (DST).
- Depending on heterogeneity a fixed- or random-effects model was applied.

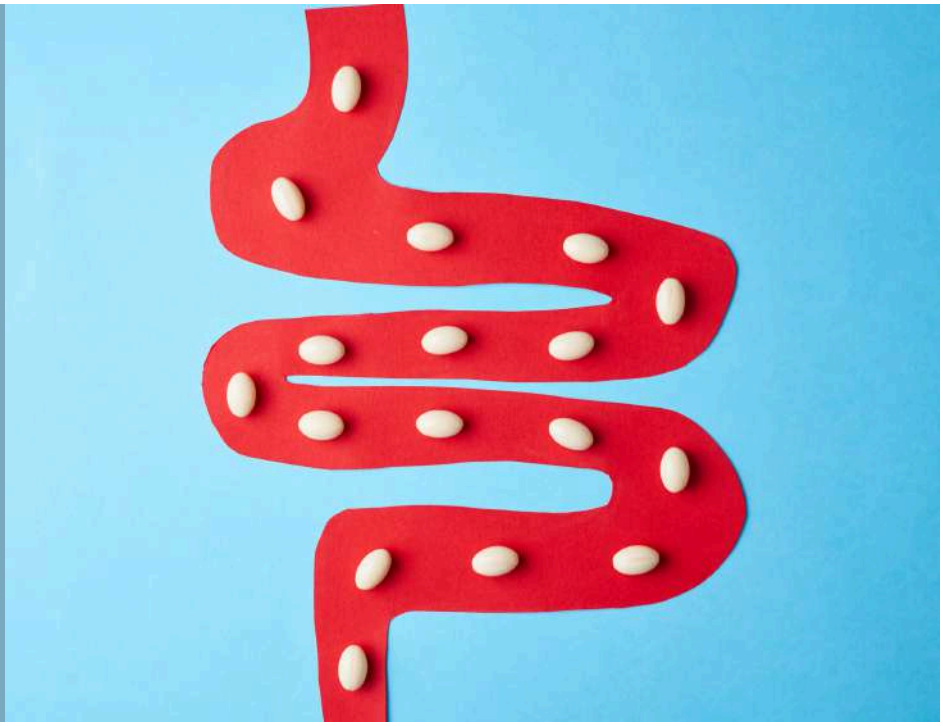
RESULTS

- Random effects-model:
- Blood ammonia (14 RCTs) was lower with probiotics (SMD = -2.68, 95% CI: - 3.90 to -1.46, p<0.0001).
- ALT (8 RCTs) was lower with probiotics (MD=-11.10, 95% CI:-16.17 to -6.03, p<0.0001).
- NCT reaction time (9 RCTs) was significantly lower with probiotics (MD=-12.54, 95% CI: -21.63 to – 3.46, p=0.007).
- DST score showed now significant difference (SMD- 0.81, 95% CI: -2.66 to 4.29, p=0.65).
- The MELD score (3 RCT's) was significantly lower (MD= -2.55, 95% CI: -3.56 to – 1.54, p<0.00001).
- MHE reversal rate was higher in the probiotic group (RR=2.79, 95% CI: 1.23 to 6.35, p=0.01).
- Fixed-effects model:
- OHE incidence (7 RCTs) was lower with probiotics (RR = 0.18, 95% CI: 0.09 to 0.34, p<0.00001).

HOW TO BRING THIS INTO YOUR CLINICAL PRACTICE

- To help support cognitive function and reduce levels of ammonia in the blood practitioners should consider recommending probiotics in patients with MHE.

Read the article here



CLINICAL PRACTICE APPLICATIONS

- Patients with a history of cirrhosis and who have been diagnosed with MHE would benefit from including probiotics in their supplement plan.
- If a patient with MHE is being treated with medications like lactulose or rifaximin, practitioners should highlight the benefits that probiotics could have on MHE incase their general practitioner needs to make adjusments to their medication.

CONSIDERATIONS FOR FUTURE RESEARCH

- The majority of studies used in the paper were trials from China (12), which may bias results pointing for future research to include more countries.
- The RCTs showed significant heterogeneity in certain areas like ALT, DST score, NCT reaction times and MHE remission rates. Additional studies would benefit from adopting standardised protocols including clearly defined probiotic strains, dosages and length of treatment.
- Including more databases to capture further RCT's missed in this study will help to increase confidence in results.
- Larger, high quality RCT's are needed to confirm the effect of probiotics on MHE.

CONCLUSION

- Probiotics are associated with improvements in MHE, including reduced blood ammonia levels and enhanced cognitive function.

EXPERT REVIEWER Nicky Ester

CONFLICTS OF INTEREST: None
EVIDENCE CATEGORY: A: Meta-analyses, position-stands, randomized-controlled trials (RCTs)

CLINICAL FACT SHEETS

FACT SHEETS

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What is Non-alcoholic fatty liver disease (NAFLD)

A condition in which fat accumulates in the liver in people who drink little or no alcohol

Non-alcoholic fatty liver disease (NAFLD) refers to a spectrum of liver damage where fat builds up in the liver, and is typically accompanied by insulin resistance. Lipid accumulation in the liver results from the imbalance between the delivery of lipids and their hepatic uptake, synthesis, oxidation, and secretion, and mitochondrial dysfunction may also play a key role in the development of advanced NAFLD. Insulin resistance in NAFLD is characterized by reduced whole-body, hepatic, and adipose tissue insulin sensitivity (1).
There are four stages of NAFLD: 1. Steatosis, where fat starts to accumulate in liver cells, 2. Non-alcoholic steatohepatitis (NASH), where the liver becomes inflamed, 3. Fibrosis, where the persistent inflammation causes scar tissues, and 4. Cirrhosis, where damage is irreversible and can lead to more serious disease.

UK Statistics

It is estimated that a third of the UK population has NAFLD, mostly in the early stage and that approximately 70%–80% of obese and diabetic patients have non-alcoholic fatty liver disease (NAFLD). It is usually diagnosed through abnormal liver function tests and/or ultrasound scans, sometimes biopsies are performed. Risk factors for NAFLD include being overweight and obese, metabolic syndrome and Type 2 Diabetes, making it part of the cardiometabolic dysregulation cluster (2)

Diet & Nutrition

At present there are no drugs available to treat NAFLD. Diet therefore plays an important role in preventing and managing the risk factors that lead to NAFLD.
BANT nutrition practitioners assess and identify potential nutritional imbalances to understand how these may contribute to an individual's symptoms and health concerns.
Practitioners consider each individual to be unique and recommend personalised nutrition and lifestyle programmes rather than a 'one size fits all' approach.

FOOD FOR YOUR HEALTH

1. Bugianesi E, Moscatiello S, Ciaravella MF, Marchesini G. Insulin resistance in nonalcoholic fatty liver disease. Curr Pharm Des. 2010 Jun;16(17):1941-51. doi: 10.2174/138161210791208875. PMID: 20370677.
2. NHS https://www.nhs.uk/conditions/non-alcoholic-fatty-liver-disease/ (2018).

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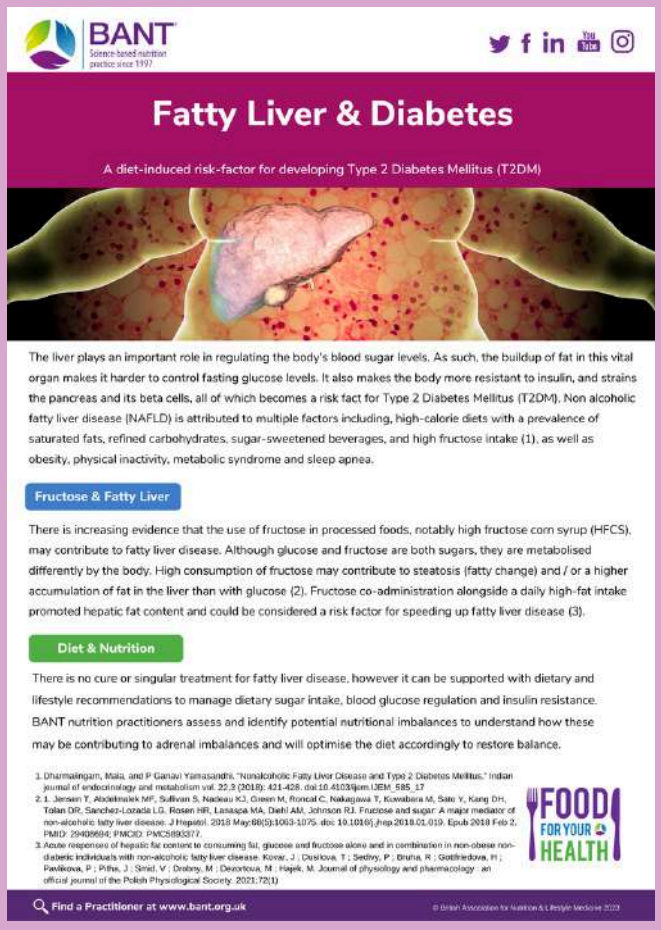

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Fatty Liver & Diabetes

A diet-induced risk-factor for developing Type 2 Diabetes Mellitus (T2DM)

The liver plays an important role in regulating the body's blood sugar levels. As such, the buildup of fat in this vital organ makes it harder to control fasting glucose levels. It also makes the body more resistant to insulin, and strains the pancreas and its beta cells, all of which becomes a risk fact for Type 2 Diabetes Mellitus (T2DM). Non alcoholic fatty liver disease (NAFLD) is attributed to multiple factors including, high-calorie diets with a prevalence of saturated fats, refined carbohydrates, sugar-sweetened beverages, and high fructose intake (1), as well as obesity, physical inactivity, metabolic syndrome and sleep apnea.

Fructose & Fatty Liver

There is increasing evidence that the use of fructose in processed foods, notably high fructose corn syrup (HFCS), may contribute to fatty liver disease. Although glucose and fructose are both sugars, they are metabolised differently by the body. High consumption of fructose may contribute to steatosis (fatty change) and / or a higher accumulation of fat in the liver than with glucose (2). Fructose co-administration alongside a daily high-fat intake promoted hepatic fat content and could be considered a risk factor for speeding up fatty liver disease (3).

Diet & Nutrition

There is no cure or singular treatment for fatty liver disease, however it can be supported with dietary and lifestyle recommendations to manage dietary sugar intake, blood glucose regulation and insulin resistance. BANT nutrition practitioners assess and identify potential nutritional imbalances to understand how these may be contributing to adrenal imbalances and will optimise the diet accordingly to restore balance.

1. Dharmalingam, Mala, and P Ganavi Yamasandhi. "Nonalcoholic Fatty Liver Disease and Type 2 Diabetes Mellitus." Indian journal of endocrinology and metabolism vol. 22,3 (2018): 421-428. doi:10.4103/ijem.IJEM_585_17
2. 1. Jensen T, Abdelmalek MF, Sullivan S, Nadeau KJ, Green M, Roncal C, Nakagawa T, Kuwabara M, Sato Y, Kang DH, Tolan DR, Sanchez-Lozada LG, Rosen HR, Lanaspa MA, Diehl AM, Johnson RJ. Fructose and sugar: A major mediator of non-alcoholic fatty liver disease. J Hepatol. 2018 May;68(5):1063-1075. doi: 10.1016/j.jhep.2018.01.019. Epub 2018 Feb 2. PMID: 29408694; PMCID: PMC5893377.
3. Acute responses of hepatic fat content to consuming fat, glucose and fructose alone and in combination in non-obese non-diabetic individuals with non-alcoholic fatty liver disease. Kovar, J ; Duskova, T ; Sedivy, P ; Bruha, R ; Gotfriedova, H ; Pavlikova, P ; Pitha, J ; Smid, V ; Drabsy, M ; Dezortova, M ; Hajek, M. Journal of physiology and pharmacology : an official journal of the Polish Physiological Society. 2021;72(1)

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BANT

Detoxification Pathways

the continual mechanism of ridding the body of environmental toxins and endogenous waste

What is the process of detoxification in the body?

Our bodies are exposed to thousands of environmental biochemicals and toxins daily, on top of all the metabolic waste products we naturally produce. These toxins are continually broken down into less harmful substances through a series of detoxification processes that take part in the liver, and are then transported to the kidneys, colon, lungs and skin for elimination as urine, stool, breath and sweat. Detoxification is a continuous process - not a singular event - without which the human body would not be able to survive. Toxic burden - an accumulation of toxins in your body - affects both physical and mental health. Most toxic chemicals are fat-soluble and not easily eliminated from the body. Many toxins are also obesogenic, meaning they can disrupt your metabolic pathways and lead to weight gain - such as phthalates (found in plastics) and BPA (used in food packaging). Fat cells in the body are used as depositories for these toxins and the more that accumulate, the greater the risk of weight gain. Conversely during weight loss these chemicals are released back into the bloodstream to be processed by the liver and eliminated from the body.

Why is the liver so important to this process?

The liver is the organ at the centre of much of the body's detoxification processes. To clear your body of toxins and wastes, it pumps <1.5 litres of blood through itself every minute and conducts three key phases of detoxification using three sets of enzymes or proteins, called the Phase I (functionalisation) enzymes, Phase II (conjugation) enzymes, and the Phase III (elimination) proteins.

- Phase I: oxidisation of toxins to neutralise and transform them into intermediary metabolites.
- Phase II: conjugation or further break down of the metabolites into water-soluble substances.
- Phase III: transport and elimination from your body through urine and stool.

How to support detoxification processes?

When your liver gets overloaded it cannot work as efficiently. Reducing exposure to environmental toxins and eating a diet rich in liver-supporting nutrients can form part of a protocol to support detoxification pathways.

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Providing practitioners with health resources to support their clinical recommendations.


BANT

DASH Eating Plan

A flexible and balanced eating plan that helps create a heart-healthy eating style for life

What is the DASH Eating Plan or Diet?

DASH, stands for dietary approaches to stop hypertension, and is a flexible and balanced eating plan promoted by the U.S.-based National Heart, Lung, and Blood Institute to prevent and control hypertension. DASH puts an emphasis on portion size, eating a healthy variety of different foods, and making sure you get the right amount of nutrients The DASH eating plan encourages reducing the sodium in your diet and to eat a variety of foods rich in nutrients that help lower blood pressure, such as potassium, calcium and magnesium. The protocol is based on daily calorific needs, as determined by age and physical activity levels, and is delivered via daily and weekly nutritional goals based on a series of public-health healthy eating principles (the equivalent to the UK Eatwell plan). To benefit from the DASH eating plan, it is deemed important to limit daily sodium levels to 2,300 mg, or 1,500 mg if desired, and to consume the appropriate amount of calories to maintain a healthy weight or lose weight if needed. DASH is promoted as a lifelong commitment to healthy living.

Personalised nutrition and the DASH eating plan

The nutritional benefits of the diet are based on the following three principals: choosing foods that are low in saturated and trans fats, rich in potassium, calcium, magnesium, fibre, and protein and low / lower in sodium. Four NHLBI-funded studies tested the health benefits of the DASH diet by comparing the DASH diet with the typical American diet or by comparing different variations of the DASH diet. Another NHLBI-funded study, the PREMIER clinical trial, measured the health benefits of following the DASH diet and increasing physical activity. The results of these studies showed that the DASH diet lowers blood pressure and LDL cholesterol in the blood and shaped the NHLBI's DASH eating plan recommendations, which includes following a DASH diet with reduced sodium intake. There is no equivalent in the UK.

Optimising diet to support blood pressure

1. Eating vegetables, fruits, and whole grains.
2. Including fat-free or low-fat dairy products, fish, poultry, beans, nuts, and vegetable oils.
3. Limiting foods that are high in saturated fat, such as fatty meats, full-fat dairy products, and tropical oils such as coconut, palm kernel, and palm oils.
4. Limiting sugar-sweetened beverages and sweets.
5. Limiting alcohol.

1. https://www.nhlbi.nih.gov/health-topics/dash-eating-plan

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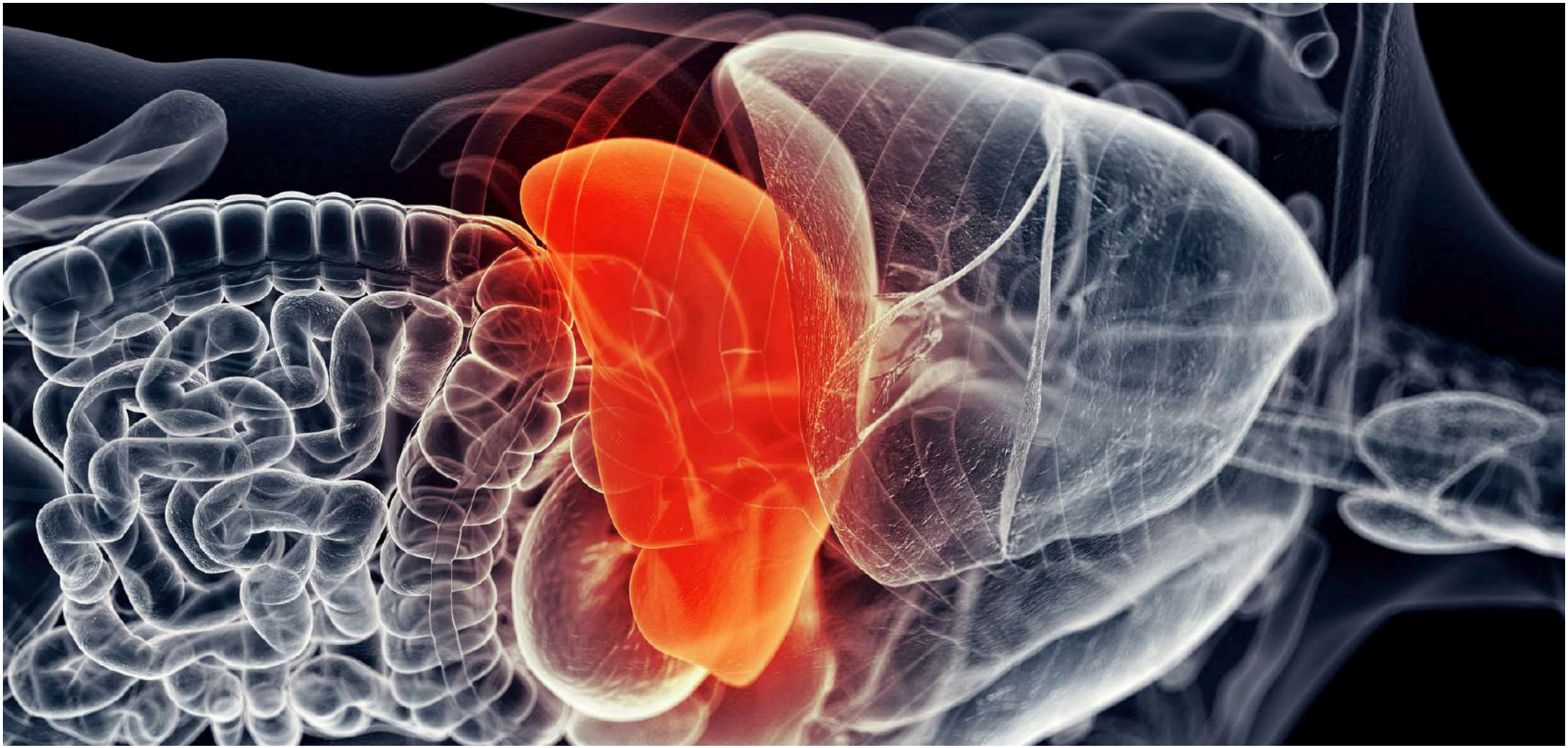
DR MICHELLE BARROW ET AL.

FEATURE ARTICLE

LIVER HEALTH:

EVIDENCE BASED CONSIDERATIONS FOR NUTRITIONAL THERAPY AND PERSONALISED LIFESTYLE SUPPORT



LIVER HEALTH:

EVIDENCE-BASED CONSIDERATIONS FOR NUTRITIONAL THERAPY AND PERSONALISED LIFESTYLE SUPPORT

Authors: Michelle Barrow, Anna Papoutsa, Nicky Ester, Ana-Paula Agrela, Chloe Steele, Sarah Cassar, Kate Lawrence

ABOUT LIVE HEALTH

ABSTRACT

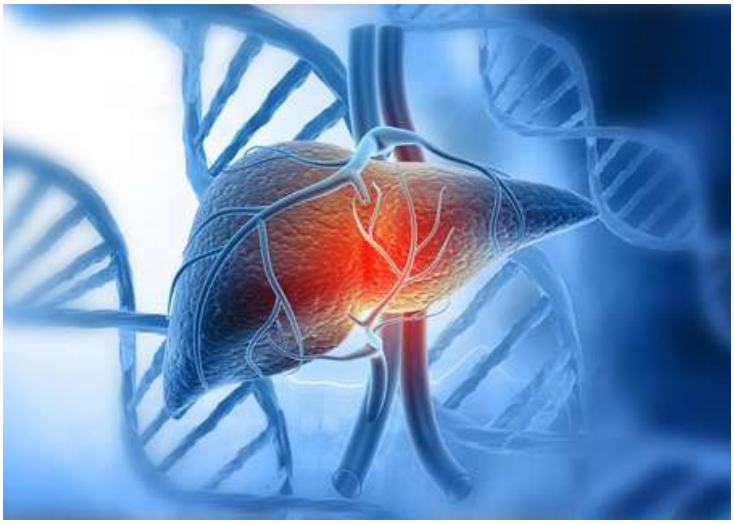
Liver health is essential for a variety of physiological processes, including transport and storage of many micronutrients. Nutritional factors, including nutrient deficiency, calorie excess and excess alcohol intake can contribute to the development of a range of liver disorders. Metabolic Associated Steatotic Liver Disease (MASLD) affects an estimated 25% of the global population and the prevalence of liver disorders is predicted to increase substantially.

This narrative review aims to provide a useful summary of common liver disorders, as well as a summary of the evidence on the role of key macro and micronutrients, dietary and lifestyle factors and the role of the microbiome in supporting liver health. It also includes a brief review of the utility of laboratory assessments for evaluating different aspects of liver health. It aims to guide evidence-based decision-making by nutrition and healthcare practitioners when recommending personalised nutrition interventions to support liver health.

INTRODUCTION

The liver is a primary metabolic organ, located in the upper right quadrant of the abdomen, responsible for critical functions including detoxification, nutrient metabolism, bile synthesis and vitamin and mineral storage. It also contributes to the regulation of immune responses, energy homeostasis, and hormonal balance. The liver's function is closely integrated with that of the gastrointestinal system, pancreas, and gallbladder.

A wide range of liver diseases and conditions can arise from diverse causes, as reviewed in the following section. For example, insufficient choline intake may cause hepatic steatosis, while other liver issues can be the result of drugs or excess alcohol intake. Metabolic Associated Steatotic Liver Disease (MAFLD) previously known as Non-alcoholic fatty liver disease (NAFLD) affects an estimated 25% of the global population and is the most prevalent chronic liver condition worldwide (1). In this review the term MAFLD will be used instead of NAFLD, even when cited research uses the latter, as NAFLD was renamed MAFLD to accurately acknowledge the role of metabolic dysfunction.



MAFLD comprises a spectrum ranging from simple hepatic steatosis to non-alcoholic steatohepatitis (NASH), fibrosis, cirrhosis, and eventually hepatocellular carcinoma (2). MAFLD is driven by metabolic syndrome and associated with obesity and diabetes (1). Pathophysiology is complex and mechanisms include hepatic lipid accumulation and insulin resistance (3) as well as oxidative stress, dysbiosis, chronic inflammation, cytokine imbalance and genetic factors (4).

Diets high in calories, refined sugar, fructose, saturated fat, trans fats, and Omega- 6 polyunsaturated fats (PUFAs) promote hepatic lipid deposition and inflammation, whereas fibre, low glycaemic-index carbohydrates, Omega 3 PUFAs, and micronutrients could have protective effects (5).

There is currently no specific pharmaceutical intervention for MAFLD. Calorie restriction, improved dietary quality, exercise, and lifestyle adjustments may all help to reduce liver fat and improve health outcomes (3).

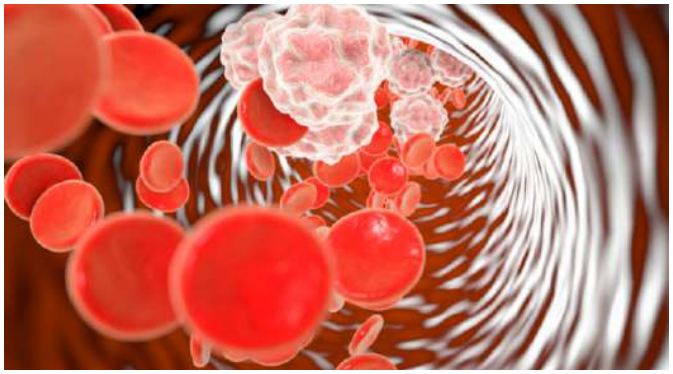
Given the modifiable nature of dietary intake, exercise and weight management, it is essential that healthcare practitioners consider nutritional status and lifestyle factors when evaluating patients with known or suspected liver dysfunction. Early identification and evidence-based dietary intervention may offer a cost-effective approach for preventing or managing liver diseases, including MAFLD.

This narrative review therefore seeks to provide a useful overview of the current evidence of the role of nutrition in liver health as well as support evidence based decision-making by nutrition and healthcare practitioners for recommending personalised nutrition interventions for liver health.

COMMON LIVER DISORDERS

WHAT ARE THE COMMON LIVER DISORDERS RELEVANT TO NUTRITION PRACTICE?

Nutritional factors, such as nutrient deficiency, excess, or toxin exposure, may contribute to a range of liver disorders including hepatic fat accumulation (steatosis). Persistent damage from these factors may provoke inflammation, hepatocellular injury, and fibrotic scarring, which can ultimately progress to cirrhosis, an irreversible stage characterised by structural alteration and loss of hepatic function (6,7).



Steatotic disorders include MASLD, alcohol-associated liver disease (ALD), and protein-deficiency–related hepatic steatosis. Non-steatotic conditions include intestinal failure–associated liver disease (IFALD), cholestatic diseases (PBC/PSC), haemochromatosis, choline-deficiency steatosis (CDS), nutritional hepatotoxicity, coeliac hepatitis and Wilson disease. Wilson disease is rare and involves impaired copper excretion, leading to hepatic copper accumulation (8).

MASLD involves hepatic steatosis, inflammation, and hepatocellular injury, and may advance to fibrosis (9). Excess energy intake, particularly from processed simple carbohydrates, promotes de novo lipogenesis (DNL), converting carbohydrates into fatty acids resulting in hepatic fat accumulation (10, 11). In a double-blind RCT (N = 94), fructose or sucrose drinks increased basal hepatic lipid secretion rates more than twofold compared with controls (median FSR %/day: sucrose 20.8 ($p = 0.0015$); fructose 19.7 ($p = 0.013$); control 9.1) (12). Increased DNL suppresses fatty acid oxidation, causing lipotoxicity, inflammation, and fibrogenesis (13). Conversely, complex carbohydrates, fibre, and wholegrains appear to be protective (14, 15).

MASLD reflects systemic metabolic dysfunction, elevating risks of fibrosis, cirrhosis, cardiovascular, renal disease, and type 2 diabetes (16). MASLD is typically asymptomatic, though upper-right abdominal discomfort and raised ALT or AST can sometimes be indicators; advanced stages can manifest with jaundice, oedema, fatigue, and abdominal distension (17, 18). Another common cause of hepatic steatosis is excessive alcohol intake in ALD, where increased NADH production inhibits fatty acid oxidation and enhances lipogenesis (19).

In choline-deficiency steatosis insufficient choline intake can cause hepatic steatosis independently of metabolic dysfunction. Choline is required for phosphatidylcholine synthesis both directly through the CDP–choline pathway and indirectly by supporting methylation via betaine. Phosphatidylcholine is essential for hepatic VLDL assembly and export; with deficiency causing intrahepatic lipid retention (20;21). Clinically, CDS resembles MASLD but occurs in lean individuals with low VLDL and plasma triglycerides, unlike the hypertriglyceridaemia and insulin resistance that are typical of MASLD (2, 22).

Severe protein malnutrition impairs apolipoprotein B and VLDL synthesis, leading to hepatic triglyceride accumulation. Subsequent refeeding, may aggravate steatosis through hyperinsulinaemia and lipogenesis, with oedema, muscle wasting, and abdominal distension (23, 24, 25). In cholestatic disease, impaired bile formation leads to hepatic and systemic retention of bile acids and bilirubin. Deficiencies in choline, taurine, or essential fatty acids may precipitate cholestasis (26). As a result, fat malabsorption leads to weight loss and fat-soluble vitamin deficiencies (27). In IFALD, prolonged parenteral nutrition induces steatosis and cholestasis through nutrient intake imbalance including excess glucose and omega-6 fatty acids, resulting in impaired VLDL export and bile metabolism (28). Hepatic inflammation and raised liver enzymes may also arise from untreated coeliac disease (29). Lastly, excess hepatic iron due to haemochromatosis or excessive vitamin A retinol intake can provoke hepatic oxidative injury and fibrosis (30, 31). Clinical management should therefore prioritise elucidating the underlying pathophysiological mechanisms and nutritional determinants, enabling the development of personalised intervention strategies that specifically address these factors.

MACRONUTRIENTS

WHAT ARE THE KEY MACRONUTRIENTS FACTORS INFLUENCING LIVER HEALTH?

The health of the liver is influenced by adequate protein intake, which supports liver repair and regeneration, immune function, metabolism regulation and maintenance of cellular homeostasis. While imbalances in macronutrient intake have been shown to impair liver health, the most effective nutritional strategies for preventing or managing conditions such as MAFLD remain to be clearly established (32).

Part of the reason for the predicted rise in liver disorders (10) may be due to the rise of obesity and the increased consumption of macronutrients, specifically saturated fat and fructose, together with insufficient protein intake and a reduction in fibre and nutrient content of the diet (33). Carbohydrates, in particular fructose added to beverages as excess calories, upregulate de novo hepatic lipogenesis. According to Lee et al., (10) total fructose-containing sugars resulted in a large increase in intrahepatocellular lipid (IHCL) in trials where excess energy from sugars was added to diets compared to the same diets without the excess sugars (13 trials; SMD: 1.72; 95% CI: 1.08, 2.36, PSMD < 0.001; no heterogeneity, I2 = 0.00%, PQ = 0.943). It was not clear if this includes fructose found in fruit, fruit juice, dried fruit and desserts.

The quality of fat in the diet also impacts liver health and disease progression. In a randomised controlled trial which investigated the impact of overfeeding on 38 participants (BMI 31± 1kg/m2, liver fat 4.7± 0/9%), overeating by 1000 kcal/day, either saturated or unsaturated fat (MUFA and PUFA) or simple sugars, showed that saturated fat increased intrahepatic triglycerides by about 55% (4.9 ± 6.6 vs. 7.6 ± 8.8%, P < 0.001) compared to 15% for unsaturated fats (4.8 ± 4.9 vs. 5.5 ± 4.8%, P < 0.02) and 33% for simple sugars (4.3 ± 4.7 vs. 5.7 ± 5.4%, P < 0.02) (34).

Using data from the NutriAct clinical trial (n = 502), Pletsch-Borba et al. (35) found that among middle-aged and older adults at risk for age-related diseases, a diet high in plant-based protein (15–25% of total energy) and unsaturated fats (15–20% of energy from MUFA and 15% from PUFA) was associated with beneficial effects on (IHCL). The improvements in liver fat and visceral abdominal fat were primarily attributed to the increased intake of unsaturated fatty acids, and these effects occurred independently of major weight loss, highlighting the importance of dietary quality.

In relation to protein, The European Association for the Study of the Liver (EASL) (36) provides clinical practice guidelines for intake, recommending 1.2-1.5 g/kg/day of protein intake for patients diagnosed with liver cirrhosis. However, a small proportion of these patients have hepatic encephalopathy, which may reduce their tolerance to animal protein (meat) and therefore vegetable and dairy proteins should be recommended. Other clinical nutrition guidance, however, is currently lacking. Recommended calorie intake for non-obese patients with cirrhosis is 35–40 kcal/kg/day; however, there are currently no recommendations regarding specific fatty acid or carbohydrate intake (32). Similarly, no specific macronutrient targets have been established for MAFLD (37).

To inform the use of macronutrients in nutritional protocols, particularly for MAFLD where pharmacological treatments remain limited, further research is needed to understand how macronutrient manipulation supports liver health. Current evidence suggests that diets high in protein and unsaturated fat, low in carbohydrate, and designed to promote weight loss are most beneficial (32, 38).

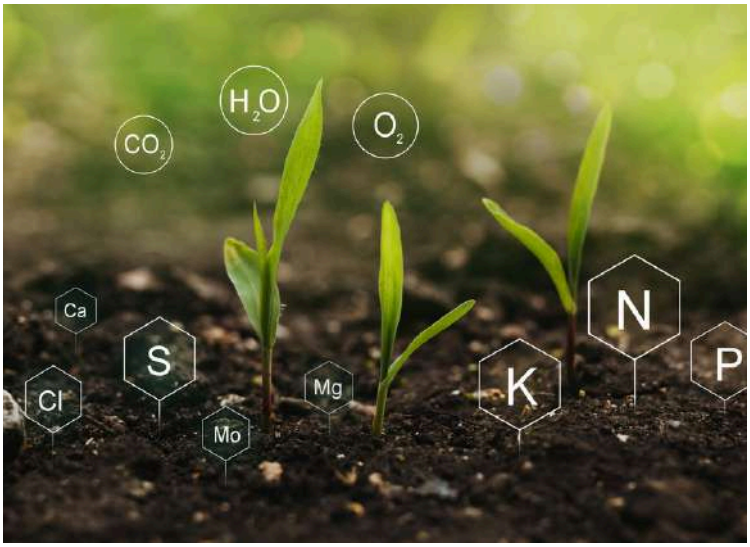
MICRONUTRIENTS

WHAT ARE THE KEY MICRONUTRIENTS FACTORS INFLUENCING LIVER HEALTH?

The liver plays a central role in micronutrient storage and homeostatic regulation, so impaired hepatic function can disrupt micronutrient metabolism and thereby contribute to disease progression (39). The liver is a key site for transport and storage of several micronutrients, including vitamin A, vitamin B12 and copper (39). Liver disease frequently leads to malnutrition due to a combination of reduced dietary intake, impaired digestion or absorption, and increased or altered metabolic demands (40). Deficiencies of fat-soluble vitamins (A, D, E and K), B-vitamins and trace elements such as zinc, magnesium and selenium are well documented in people with liver disease (41, 42), although the prevalence and pattern of deficiencies vary with the underlying diagnosis and disease severity (42).

Vitamin A is integral in liver function through its involvement in lipid metabolism, antioxidant defence, anti-inflammatory processes, and insulin sensitivity. The majority of the body's vitamin A is stored in hepatic stellate cells, underscoring the liver's role in vitamin A homeostasis (39). Liu et al. reported that reduced serum vitamin A levels were correlated with more advanced hepatic fibrosis in patients with MAFLD (43), reinforcing its protective role in liver health. Vitamin D plays multiple roles in liver health, including direct effects on liver cells, immunomodulation, antifibrotic mechanisms, and maintenance of metabolic and immune homeostasis (44). Vitamin D deficiency is consistently associated with greater disease severity. Observational studies show inverse associations between serum vitamin D and MAFLD and fibrosis (45, 46, 47). Mechanistically, vitamin D modulates hepatic inflammation and fibrogenesis (44, 48). Vitamin E, a potent lipid-soluble antioxidant, has well-documented hepatoprotective effects (49). Clinical guidelines currently only recommend vitamin E supplementation for patients with MASLD due to its ability to reduce oxidative stress and hepatic inflammation (43).

Zinc deficiency has been associated with impaired antioxidant defense, increased fibrosis risk (50, 51). Zinc contributes to hepatic protection by reducing oxidative stress, modulating inflammatory responses, and improving liver enzyme profiles in patients with chronic liver disease and MAFLD (50, 51).



Magnesium deficiency has been associated with profibrogenic activity and decreased antioxidant capacity, promoting lipid peroxidation and cytokine activation which are key pathways involved in steatohepatitis and fibrosis (52, 53). Selenium exhibits a dose-dependent relationship with liver health (43). Lower blood selenium levels were associated with an increased incidence of advanced fibrosis, whereas higher levels (>130 µg/L) were positively correlated with MAFLD and ghrelin levels, indicating a narrow optimal range for hepatoprotective benefit (43). Combined micronutrient support may therefore enhance liver health through several mechanisms.

A UK Biobank cohort analysis in individuals (n=402,476) with MAFLD, which utilised inverse probability of treatment weighting (IPTW) to account for potential confounding factors, found that habitual multivitamin use was associated with a significant lower all-cause mortality risk before (HR: 0.88, 95% CI 0.81–0.95, P = 0.034) and after (HR: 0.94, 95% CI 0.88–1.00, P = 0.037) IPTW adjustment (54). However, a meta-analysis which pooled 202 randomised trials of nutritional supplements in MAFLD found no convincing effects and concluded the evidence indicates considerable uncertainty about the effect of supplementation on clinical outcomes (55). A nuanced, personalised assessment of micronutrient status, alongside an understanding of the disease-specific mechanisms driving deficiency, is essential to clarify their contribution to liver pathophysiology and to design safe, targeted nutritional interventions to prevent or attenuate liver disease.

WHAT ARE THE KEY DIETARY FACTORS INFLUENCING LIVER HEALTH?

Excess caloric intake and insulin resistance can drive hepatic fat accumulation affecting liver health. Fat accumulation and inflammation can drive hepatic insulin resistance increasing the risk for the development of metabolic dysfunction, which is associated with MASLD (56,57).

As highlighted, increased consumption of dietary sugars, with fructose and sucrose, promotes hepatic de novo lipogenesis. In one RCT of 94 healthy men, sugar sweetened beverage consumption with moderate amounts of sucrose or fructose (80g/day) in addition to usual diet for 7 weeks resulted in a two-fold increase in hepatic fatty acid production compared to the control (median FSR %/day sucrose: 20.8; (P=0.0015), fructose: 19.7; (P=0.013), control: 9.1) (12). Glucose sweetened beverages did not have the same effect on hepatic VLDL production (median FSR %/day 11.0; (n.s)). Furthermore, low-sugar diets have been shown in one RCT of 40 adolescent boys with MASLD to result in hepatic steatosis improvements when compared to normal diet (-6.23%; 95% CI, -9.45% to -3.02%; P < .001) (58).

Fibre may act on satiety, reducing energy and fat intake (59). Fibre intake has also been shown to modulate the gut microbiota, altering bile acid production, restoring the gut barrier and inhibiting lipid absorption (60). One RCT has shown that the adoption of a high fibre (amount not given), low carbohydrate (20-25%) diet alongside dietary education amongst 44 individuals with MASLD for 2 months improved abdominal circumference, visceral fat, and body fat (P=<0.05 for all) when compared to education alone (61). Fatty infiltration of the liver was also improved (P=0.015). However, muscle mass was shown to be decreased, which could have other health implications (P=<0.05).

Liver function decline as a result of excess lipid accumulation may be mediated by mitochondrial dysfunction and the reduced capacity of the liver to transport excess lipids into circulation and increased lipid peroxidation (62).

Targeting mitochondrial function may be of benefit and diets high in polyphenols and antioxidants have been shown to benefit liver health. One RCT of 50 individuals with overweight reported that adoption of the Mediterranean diet (MD) for six months resulted in improved anthropometrics (weight, body mass index, waist and hip circumference P=0.0001 for all), lipids (cholesterol: P=0.0001, low density lipoprotein-cholesterol: P=0.005 and triglycerides: P=0.0001), hepatic fat accumulation (P=0.002), and liver stiffness (P=0.0001) compared to control (63). However, when an antioxidant supplement was introduced (silymarin 120 mg, chlorogenic acid 7.5 mg, protopine 0.04 mg, L-methionine 150 mg, and L-glutathione 10 mg), further improvements were seen on insulin sensitivity (HOMA-IR: P=0.0001) and a consistent reduction in anthropometrics was observed (waist circumference P=0.030), which may further benefit liver health. In an 18-month RCT of 294 individuals with abdominal obesity/dyslipidaemia a MD has been shown to result in intrahepatic fat loss compared to standard healthy diet (-12.2%; P=<0.001), which was doubled when enriched with polyphenols (mankai, green tea, and walnuts) and reduced red and processed meat intake (green-MD:-38.9% vs MD: -19.6%; P=0.035) (64).

A low sugar diet that is high in fibre, polyphenols, and antioxidants appears to be of benefit to liver health. However, it is also important to maintain muscle mass through exercise, which may decline with weight loss (65).

DIETARY RECOMMENDATIONS

WHAT ARE THE CURRENT DIETARY RECOMMENDATIONS FOR LIVER HEALTH?

In the UK, there are no official liver-specific dietary guidelines. The British Liver Trust and the NHS recommend a healthy, well-balanced diet for those with and for those wanting to prevent liver disease (66, 67). Their recommendations focus on maintaining a healthy body weight, optimising blood lipids, improving insulin sensitivity, and reducing inflammation which may be involved in the pathogenesis of liver disease.

As previously discussed, evidence from Abenavoli et al (68) and Meir et al (64), suggest that the adoption of a Mediterranean Diet (MD) may benefit liver health. This diet is high in monounsaturated fats, omega-3 fatty acids, fibre, and antioxidants, and may be of benefit regardless of the macronutrient content, making it an easy-to-follow pattern of eating. In one intervention study of 63 individuals with MASLD, a typical MD, low-carbohydrate MD, and low-fat MD all showed that liver enzymes and fatty liver index were improved from baseline (P=<0.05 for all), with no differences between the diets (69). Red meat and processed foods are also limited with the MD, which aligns with the NHS UK healthy eating guidelines (70).

Concerns have been raised over the suitability of diets high in saturated fat for individuals with poor liver health (71). The high saturated fat content of a ketogenic diet (KD) may seem counterintuitive, however, in the short-term, the KD has been shown to have a similar effect on liver fat content as the low-fat diet and may particularly benefit those with already established MASLD. In one RCT of 28 individuals with overweight or obesity it has been shown that the adoption of a KD for 6 weeks resulted in liver fat reductions from baseline (P=0.004) similar to individuals on a low-fat diet (P=>0.05) (72). Individuals with MASLD were shown to have more than 5% reduction in liver fat content. For individuals who are concerned, a well-planned KD, avoiding high dietary intakes of fat is possible and may still benefit liver health. In one RCT of 39 individuals with obesity, a KD high in protein (0.8-1.2g/kg ideal body weight) and low in fat (10g/day) has been shown to result in greater visceral adipose tissue loss (−21.47%± 19.1% vs. −6.33%± 28.9%; p = 0.06) and liver fat reduction (mean ± SD = −4.77 ± 4.26 vs. −0.79 ± 1.76 p = 0.0006; mean relative change = −38.5 vs. −2.7%; P= < 0.0001) compared to a standard low calorie diet (73). It is important to understand that KD success may be more apparent in men and may be affected by menopausal status in females, with benefits more apparent postmenopausally (74).

UK NHS dietary recommendations for health (70) include an emphasis on a range of fruits and vegetables. A low-fat vegan diet has been shown in one 16-week RCT of 244 individuals to result in 34.4% decreased hepatocellular lipids mean (SD) of 3.2% (2.9%) to 2.4% (2.2%) (P = .002) and 10.4% decreased myocellular lipids mean (SD) of 1.6 (1.1) to1.5 (1.0)

t(P = .03) from baseline, whilst no changes were observed in the control group with no diet changes (75). UK NHS recommendations (67) emphasise a balanced diet to support liver health, with evidence showing that MD, ketogenic, and plant-based diets may reduce liver fat and improve metabolic markers.

THE GUT MICROBIOME

WHAT IS THE ROLE OF THE MICROBIOME IN LIVER HEALTH?

The human microbiome is an ever-changing ecosystem, with significant interindividual as well as intraindividual variation (76, 77). The gut-liver axis represents a bidirectional pathway between the gut microbiota and the liver (78, 79). The liver, via the gut-liver axis, is exposed to microbial products, bile acids and nutrients making the microbiome a powerful regulator of liver health and disease (79). Three key mechanisms explain this relationship. First, the intestinal barrier acts as a first line of defence. When barrier integrity is lost, microbial components such as lipopolysaccharides (LPS) can enter circulation and travel directly to the liver, triggering immune responses which lead to inflammation. Chronic inflammation may then result in fibrogenesis (78, 80, 81). Second, microbial metabolites such as short-chain fatty acids, support the integrity of the gut lining and reduce systemic inflammation, a key factor for liver repair (82). Ammonia, ethanol and secondary bile acids elevate oxidative stress levels, damage hepatocytes and promote the progression of MASLD (78, 79). Third, immune modulation occurs when gut-derived microbial products activate hepatic pattern recognition receptors (PRR) which initiate the production of pro-inflammatory cytokines. Persistent stimulation of pro-inflammatory pathways contributes to tissue damage increasing the risk of fibrosis (79, 80).

Disruption of intestinal homeostasis and alterations in the microbiome, through these interconnected mechanisms, collectively drive the pathogenesis of liver disease, from initial fibrotic changes to end-stage cirrhosis (80, 83). Compromise of the intestinal barrier integrity and translocation of endotoxins are linked to the development of cirrhosis and hepatic encephalopathy (HE) (80, 84), where systemic inflammation and elevated ammonia levels may worsen clinical outcomes (83, 84). Disruptions in the gut microbiota can lead to overproduction of microbial metabolites including ethanol and secondary bile acids. These imbalances are key drivers in alcoholic liver disease and progression of MASLD (78, 80, 84). In parallel, sustained activation of the immune system particularly via hepatic PRR signalling results in persistent inflammation and ongoing fibrosis (80). These features are characteristics of MASLD/metabolic steatohepatitis and cholestatic liver diseases (83).

Taken together, dysbiosis emerges as a unifying factor across hepatic disorders highlighting the therapeutic potential of microbiome-targeted nutrition strategies. For instance, Ahn et al. (85) conducted a 12 week RCT in 68 obese MAFLD patients and found that a multispecies probiotic mixture significantly reduced intrahepatic fat fraction from 16.3 ± 15.0% to 14.1 ± 7.7% (p = 0.032) versus baseline. Similarly, in a meta-analysis of 15 RCTs (n = 772), probiotic-based intervention reduced serum alanine aminotransferase by a mean difference of −11.76 U/L (95 % CI −16.06 to −7.46) (86). Furthermore, in a phase II double-blind RCT of 60 patients with cirrhosis and recurrent HE, faecal microbiota transplantation significantly reduced HE recurrence (40% vs 9% in placebo, OR 0.15, 95% CI 0.04-0.64; p = 0.035) and improved gut microbial engraftment (76). These interventions can improve hepatic outcomes by correcting microbial imbalances, reducing systemic inflammation, and improving metabolic parameters. Despite promising results, variability in microbiome responses and limited long-term safety data necessitate further clinical validation (68,76;86).

The gut microbiome exerts significant influence over liver function, shaping metabolic, immune, and barrier activities along the gut–liver axis. Recognising these interactions can inform personalised nutrition interventions for the prevention and management of liver dysfunction.

HOW DO LIFESTYLE FACTORS INFLUENCE LIVER HEALTH?

Unfavourable lifestyle factors including excessive alcohol intake, smoking, physical inactivity, and adiposity are central determinants of liver health (87, 88, 89, 90, 91).

Excessive alcohol consumption strains hepatic function due to the metabolites and toxic by-products generated during alcohol metabolism (92). Alcohol-related liver disease is one of the most common and serious complications of chronic alcohol intake, with progression determined by both quantity and duration of consumption. In a Finnish cohort (n=12,368), Nivukoski et al. (89) showed that individuals who both smoked and consumed alcohol at hazardous levels had substantially higher fatty liver index scores than non-smokers who consumed alcohol, indicating additive harm to hepatic fat accumulation. Cigarette smoke promotes oxidative stress, tissue hypoxia, and systemic inflammation, all of which can accelerate hepatocyte injury and fibrosis (93, 94). A cross-sectional cohort study (n=225) by Ou et al. (95) found that smokers (n=98) had significantly higher liver stiffness on Fibroscan (10.12 ± 10.38 kPa vs. 7.26 ± 6.42 kPa, P=0.013) and smoking was independently associated with fibrosis (OR = 1.29, p = 0.015). Similarly, Jang et al. (96) observed that current male smokers had higher odds of MASLD compared with nonsmokers (OR = 1.38, 95% CI: 1.08–1.76). Moreover, participants with 10–20 pack-years had OR = 1.39 (95% CI: 1.04–1.86), and those with >20 pack-years had OR = 1.51 (95% CI: 1.14–2.00), indicating a dose-dependent relationship between cigarette exposure and MAFLD risk.

Regular exercise mitigates metabolic disturbances driving hepatic fat accumulation and fibrosis. In a 12 week randomised trial (n=18), Charatcharoenwitthaya et al. (97), demonstrated that both moderate-intensity aerobic and resistance training reduced hepatic fat content by 10.3–12.6% (95% CI) and improved insulin sensitivity independent of weight loss. Similarly, Houghton et al. (98) reported that supervised aerobic and resistance training over 12 weeks (n=24) led to a 16% relative reduction in hepatic triglyceride content and improvements in ALT compared with standard care. Exercise also lowers systemic inflammation and oxidative stress, both key contributors to fibrosis progression (98). In contrast, sedentary behaviour independently increases the risk of MASLD (100). In their randomised crossover trial, Duvivier et al. (101) found that replacing sitting with brief bouts of light walking, for just 3 minutes every 30 minutes, significantly improved postprandial glucose and triglyceride responses in a crossover study of 19 participants with type 2 diabetes.

Emerging research highlights the role of sleep quality, stress regulation and circadian rhythm alignment in maintaining liver health. Poor or insufficient sleep adversely affects insulin sensitivity and inflammatory activity, independently increasing the risk of developing MASLD (102). Disrupted circadian rhythms, common among long-term shift workers or individuals with irregular or late-night eating patterns, can alter hepatic gene expression governing glucose and lipid metabolism, promoting steatosis, oxidative stress, and inflammation (103,104). Additionally, chronic psychological stress activates the hypothalamic–pituitary–adrenal axis, leading to elevated glucocorticoid levels that impair glucose and lipid metabolism, promote hepatic fat accumulation, and exacerbate oxidative stress and inflammation (105, 106). Consistent with this, large-scale cross-sectional research in 171,321 adults reported that higher perceived stress levels were significantly associated with increased prevalence of MASLD (OR = 1.17, 95% CI), independent of body mass index and lifestyle factors (107).

Adopting a health-promoting lifestyle remains the cornerstone of liver health. Evidence consistently shows that regular physical activity, adequate sleep, and avoidance of alcohol and tobacco significantly

reduce the incidence and progression of liver disease. Promoting these behaviours may protect hepatic function, improve quality of life, and reduce the long-term risk of fibrosis, cirrhosis, and related metabolic complications.

LIVER TESTING

WHAT TESTS ARE AVAILABLE TO ASSESS LIVER FUNCTION?

Liver function tests (LFTs) are used to evaluate different aspects of liver health, including hepatocellular injury, cholestasis, and the liver's ability to synthesise and excrete essential substances. To assess these functions, several biochemical markers are measured. The enzymes alanine aminotransferase (ALT) and aspartate aminotransferase (AST) primarily reflect hepatocellular injury, as they are released into the bloodstream when liver cells are impaired. Alkaline phosphatase (ALP) and gamma-glutamyl transferase (GGT) are markers of cholestasis and indicate obstruction or impairment of bile flow. Total and direct bilirubin levels evaluate the liver's excretory function, while albumin and total protein concentrations provide information about its capacity to synthesise essential proteins such as albumin and clotting factors (108).

Reference intervals for LFTs vary based on population, methodology (109) and the intended purpose, such as diagnosis or health optimisation; while variables including age, sex and ethnicity influence the partitioning and the interpretation of those intervals (110, 111, 112). Each laboratory sets and verifies its own reference intervals based on their methods, instruments, population, and calibration. Thus, there can be differences among laboratories in upper or lower limits for the same analyte (113), including public and private NHS laboratories and this is standard practice in laboratory medicine (114).

NHS laboratories reference intervals appear to be attributed to the Pathology Harmony initiative (115, 116) aiming to reduce variation and adopt common, evidence-based reference intervals across labs drawing on multicentre datasets, such as NORIP and IFCC-traceable methods. Private UK providers generally align with the same principles and intervals (115, 117) as they must be accredited by UKAS, which requires the use of validated and traceable reference intervals consistent with national or international standards (such as those developed under IFCC or NORIP).

An improved strategy for identifying abnormal liver test results (iLFT) using a laboratory algorithm was first introduced in NHS Tayside, Scotland (118). This same approach was later applied in a population-based study (119), that found that 33% of individuals with MASLD and advanced fibrosis had ALT between 31 and 54 U/L, who would not have been all identified using the NHS standard ALT range of <40 U/L (120). This reflects later evidence indicating that higher ALT values are associated with an increased risk of hepatic steatosis and metabolic syndrome, thereby setting the upper healthy limit at <22 U/L for women and <34 U/L for men (121, 122).

The reference intervals used by the NHS, along with the ranges commonly applied by private laboratories for each biomarker in the standard LFT panels, are shown in Table 1.

It is worth noting that, nutrition and lifestyle medicine practitioners use laboratory testing for the purpose of health optimisation (123, 124) while NHS assessments mainly focus on excluding disease (113). In the context of health optimisation, the focus is placed on identifying subclinical dysfunction, often using additional markers that are not liver function measures per se. Additional markers are used to infer broader metabolic or "functional" liver health and these may include high sensitivity CRP, ferritin, oxidative stress markers, homocysteine, glutathione and others.

Assessing liver function through LFTs, alongside metabolic markers, provides valuable contextual

TABLE 1

Reference intervals for adults, used by the NHS and public labs (120) and ranges of intervals used by private laboratories (122).

Marker	*NHS/ public laboratories*	*Private laboratories/ literature*
ALT	0 – 40 U/L	19 U/L (women) and 30 U/L (men)
AST	0–32 U/L (women), 0–40 U/L (men)	13–37 U/L (women) and ~14–45 U/L (men)
ALP	30 – 130 U/L	ALP 37–106 U/L
GGT	0–50 U/L(women), 0–90 U/L (men)	<77 U/L (women <40); <64 U/L (women ≥40); <78 U/L (men <40) and <114 U/L (men ≥40)
Total and Direct (Conjugated) Bilirubin	Total 2 – 21 umol/L; Direct <4 umol/L	5–24 µmol/L
Albumin	35 – 50 g/L	37–48 g/L (<40 y), 37–46 g/L (40–70 y), and 35–46 g/L (>70 y)
Total Protein	60 – 80 g/L	62–78 g/L

information in nutrition practice. Liver function markers may reveal patterns to help identify hepatic inflammation, metabolic dysfunction, steatotic tendencies or impaired bile flow. These insights can support more precise nutritional recommendations and indicate when medical referral is appropriate. By evaluating LFTs alongside clinical history and diet-lifestyle assessment, practitioners can identify suitable interventions, such as modulating dietary carbohydrate and fat quality, adjusting protein intake or supporting weight management, to support metabolic health and enhance liver function and detoxification capacity.

SUMMARY

THE ROLE OF NUTRITION PRACTITIONERS IN SUPPORTING LIVER HEALTH

Nutritional practitioners are well positioned to support individuals' health through personalised nutrition and lifestyle interventions, particularly when these interventions target the specific pathophysiological mechanisms most relevant to the individual. Nutritional excesses, deficiencies, and toxin exposures can contribute to multiple liver disorders by inducing hepatic steatosis, inflammation, and fibrosis. Nutrition practitioners can therefore play a key role in supporting individuals with liver conditions by

applying evidence-based dietary and lifestyle strategies that modulate the mechanisms underpinning these disorders.

Pathophysiological mechanisms differ across non-steatotic liver diseases, reflecting heterogeneous aetiologies, whereas steatotic disorders such as MASLD are primarily driven by metabolic dysfunction, insulin resistance, inflammation, increased de novo lipogenesis, oxidative stress, mitochondrial dysfunction, and progressive hepatocellular injury. Macronutrient intake directly influences these mechanisms; for example, excessive consumption of simple sugars (particularly fructose) promotes steatosis, and high saturated fat intake can elevate intrahepatic triglyceride levels. In contrast, adequate intake of protein, fibre, omega-3 polyunsaturated fatty acids, and antioxidant-rich foods appears protective. Vitamins D, A, and E, as well as zinc, selenium, and magnesium, also contribute to liver health. A personalised assessment of micronutrient status is therefore essential for understanding its impact on liver function and guiding targeted nutritional interventions.

Although the UK does not have liver-specific dietary guidelines, NHS recommendations emphasise a balanced diet to support liver health (67). Beyond macronutrient quality, nutrition practitioners should consider evidence for dietary patterns such as the Mediterranean diet, ketogenic diet, and plant-based diets, which can reduce hepatic fat and improve metabolic markers. Total caloric intake should also be addressed, particularly when obesity is a comorbidity. The gut microbiome strongly influences liver health through regulation of intestinal barrier integrity, production of shortchain fatty acids, and modulation of the immune system. Dysbiosis is increasingly recognised as a shared mechanism across liver diseases, highlighting the therapeutic potential of nutritional strategies that restore gut–liver axis homeostasis and reduce hepatic injury.

Lifestyle factors, including regular physical activity, adequate sleep, and avoiding alcohol and tobacco, are consistently shown to reduce liver disease risk and slow disease progression. Engagement with these behaviours can help mitigate fibrosis, cirrhosis, and other damaging pathophysiological processes. By integrating tailored nutritional and lifestyle interventions, practitioners can provide comprehensive support for individuals with liver conditions. A range of liver function tests can offer insight into hepatic inflammation, steatosis, metabolic dysfunction, and bile flow, enabling nutritional practitioners to refine personalised interventions and determine when medical referral is warranted.

KEY PRACTICE POINTS FOR NUTRITIONAL THERAPISTS

- **Personalised support:** Target dietary interventions, with a food first approach which aims to ameliorate key mechanisms of pathophysiology, while considering individual needs and preferences.
- **Address macro and micronutrient intake:** ensure balance of macro and micronutrients nutrients with specific focus on sufficient dietary intake of protein, fibre, Omega 3 PUFA, antioxidants and vitamin D.
- **Address gut health:** as well as liver health consider the gut and promote a healthy gut microbiome, enhance gut barrier function and support immune function.
- **Address lifestyle factors:** promote exercise, weight loss, sleep hygiene and the removal of alcohol and tobacco.
- **Work collaboratively:** Collaborate closely with the multidisciplinary team to integrate nutrition care with medical management, monitor for complications such as hepatic encephalopathy, and adjust nutrition plans accordingly.

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MB conceived of the review, wrote the abstract, introduction and summary, contributed to all sections of the article, provided comments to authors and revised the content. AP, CS and SC each researched and wrote two sections of the article. NE & APA each researched and wrote one section of the article. KL contributed to planning, design and revising the final content. All authors reviewed and accepted the final manuscript.

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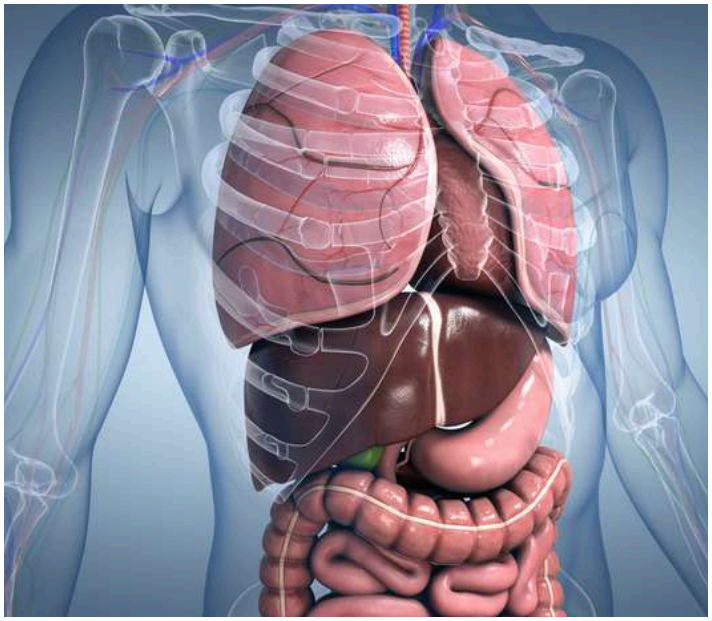

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