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NED JOURNAL

ISSUE 08
OCTOBER 2025

Ketogenic Diet

Exploring the evidence supporting a low-carbohydrate, high-fat, and moderate-protein diet

A healthy ketogenic diet may support weight loss and improve metabolic profiles in healthy subjects

A low-calorie ketogenic diet may help reduce frequency in patients with high-frequency episodic migraine

A Keto diet may be sufficient to reduce IGF-1/insulin levels for preventative and adjunct therapy medical interventions

With special feature article on Neurodivergence, Nutrition and Lifestyle

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1 In the U.K. and Ireland., as voted by health professionals and their patients. IHCAN Magazine© 2023; Nutrition i-Mag© 2023; True Health Magazine© 2023; Health Food Business Magazine© 2023. 2 According to health care professionals in the U.S., among brands surveyed. Nutritional Business Journal® 2016, 2020, 2023; Kaiser Associates 2014

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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 NEW LOOK NED JOURNAL

NED has had a significant upgrade and is now faster, smarter and more interactive, delivering clinically relevant sciences at lightning speeds. If you haven't had a chance to take a look, don't delay. Our core business remains the same - providing value-added nutrition and lifestyle science to support clinical decision making in personalised nutrition practice. Check out the AskAI function and **set up an account** to access the full functionality.

The expert reviews in this edition focus on ketogenic diets. The range of peer-reviewed articles included in this issue demonstrate how far reaching dietary change can be. We've got expert reviews on body composition and weight management, metabolic syndrome and Type 2 Diabetes, as well as inflammation, brain health, migraine and mitochondrial function - all of which assess the impact of ketogenic diets.

We close this edition with a feature article from NED Editor Dr Kate Lawrence, who lead a team of NED reviewers to answer key questions related to neurodivergence, nutrition and lifestyle medicine. Backed by research, it provides a narrative review of the science on this topic. With many thanks to Kate for leading the team and to the expert reviewers for their invaluable contributions.

SAVE THE DATE - 12 May 2026 for the NED Science Forum.

We are well into our planning for 2026 and are already looking forward to hosting the NED Science Forum at The Royal Society of Medicine in London on 12 May 2026. The details are still to be ironed out, but as at previous forums, the focus will be on cutting edge research, engaging sessions and lively debate. Ticket announcements to come.

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.

Read previous copies of the NED Journal here which BANT produces and makes available open access to all. BANT aims to bring good nutrition and lifestyle sciences to the forefront of healthcare and is able to do this through its ambition, careful management and the support of sponsors and advertisers. Thanks to the organisations who have supported this edition - **Pure Encapsulations**.

Dive in for the facts on fats.

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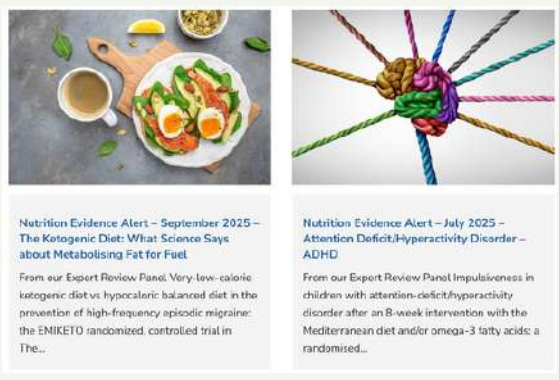
- Each month we publish a dedicated **Nutrition Evidence alert** with our editorial team's pick of the latest research and expert reviews. Sign up at here and have the science delivered straight to your inbox. Follow our socials for weekly posts on topics of interest.



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 Kate Lawrence, BA(Hons), PhD, FHEA **LEAD AUTHOR**

Dr Lawrence is a Senior Lecturer in psychology at St Mary's University, Twickenham. Kate's research specialises in nutritional psychology and neurodiversity, with a particular focus on dietary and microbiome influences on mental health and cognitive function. She is published in many scientific journals, including Frontiers in Psychology, Neuropsychology, Neuropsychologia, and BMJ Open.
Dr Lawrence is the lead author in this edition's feature article on neurodivergence.

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Dr Jessica Rigutto MPharm, MPH, Dr. sc. ETH Zurich, DipION, MBANT
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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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)

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.

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.

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.

Wilma Kirsten, MSc, INLPTA, MBANT

Wilma has been in clinical practice since 2005. The topic for her MSc dissertation project was “The impact of Coenzyme Q10 deficiency in late-onset Alzheimer’s disease in patients who use cholesterol lowering medication”. She furthermore obtained two honours science degrees, one in Nutritional Therapy and the other in Molecular Cell Biology and Health Sciences. Wilma specialises in digestive disorders (IBS and IBD), female hormonal well-being (PMS and menopause), and mental health. She has successfully helped hundreds of patients address symptoms of ill health in her clinic. Wilma is also the author of the popular science book, “Ideal Plate Composition - Choose Food to Help You Be Your Best Self”.

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.

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.

Miranda Harris, MSc, SFHEA, MBANT

Miranda is a member of BANT and a CNHC Registered Nutritional Therapy Practitioner with over 10 years clinical experience, specialising in endurance sport. She is a senior lecturer (SFHEA) focusing on research methods, dissertation supervision and sports nutrition on the Nutrition and Lifestyle Medicine MSc course at the University of Worcester. She has recently published in the European Journal of Integrated Medicine and the Journal of Nutrition and Health and is working towards a PhD by publication. She is a keen triathlete training for Ironman.

Creative Editor

Claire Sambolino, MSc, ANLP, MBANT

Claire is a CNHC Registered Nutritional Therapy Practitioner with an MSc in Personalised Nutrition and specialises in metabolic health and integrative cancer nutrition. She is a certified MTIH Terrain Advocate®, Metabolic Balance® Consultant, and NLP Coach with online clinics in the UK and Italy. She previously spent over 20 years in MarComms for multi-national food brands and combines consulting with clinical practice. 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.

KETOGENIC DIET 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 Ketogenic Diet **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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BODY COMPOSITION & WEIGHT MANAGEMENT

3 REVIEWS

Effects of a Ketogenic Diet on Body Composition in Healthy, Young, Normal-Weight Women: A Randomized Controlled Feeding Trial

YJONAS BURÉN, MICHAEL SVENSSON, PER LIV, ANNA SJÖDIN
JOURNAL: NUTRIENTS 2024;16(13):2030

TAKE HOME MESSAGE

The low carbohydrate and high fat (LCHF) ketogenic diet may be effective for weight loss in the short-term, however there may be a disproportionate loss of lean muscle in healthy, young women.

As a result, it is unclear as to the long-term effects of the diet on metabolism and weight.

Read the article here

INTRODUCTION

The ketogenic diet has been extensively studied amongst individuals with chronic diseases but dietary studies of the effects of the ketogenic diet on young, healthy, normal weight women are lacking.
This study aimed to determine the effects of a 4-week non-energy-restricted ketogenic, low carbohydrate and high fat diet (LCHF) on body composition in this group of individuals.

METHOD

- This was an unblinded randomised control cross-over trial of 17 women comparing ketogenic diet with control.
- The study ran for 4 weeks with a 15 week washout period between treatment cross-over.
- Body composition was measured using dual-energy x-ray absorptiometry.
- Women were aged 18-30 years with a body mass index of 18.5-25 kg/m-2.
- Ketogenic diets consisted of 19% daily energy intake from protein, 4% carbohydrates, and 77% fat (33% saturated fat).
- Control diet consisted of 44% carbohydrates, 33% fat and 19% protein.
- No supplements or sweeteners were consumed during the study.
- Physical activity remained constant amongst the treatment periods.

RESULTS

- Compared to control, LCHF, ketogenic diet decreased total fat mass (−0.66 kg, 95% confidence interval (CI): [−1.00, −0.32], $p < 0.001$), total lean mass (−1.45 kg, 95% confidence interval (CI): [−1.90, −1.00], $p < 0.001$), and appendicular lean mass (−0.60 kg, 95% confidence interval (CI): [−0.78, −0.42], $p < 0.001$).

HOW TO BRING THIS INTO YOUR CLINICAL PRACTICE

CLINICAL PRACTICE APPLICATIONS

- Healthy young women who are trying to lose weight may find a low carbohydrate and high fat (LCHF) ketogenic diet effective for weight loss.
- However, clinical practitioners and Nutritional therapists may like to undertake further research on ways to limit lean muscle loss when undergoing a LCHF, ketogenic diet.

CONSIDERATIONS FOR FUTURE RESEARCH

- Future research may like to consider adding a trial arm where women undergo resistance, strength training to see effects on lean muscle.

CONCLUSION

- It was concluded that LCHF, ketogenic diet is effective for weight loss in healthy, young women.
- However, there was a disproportionate loss of lean muscle mass to fat mass.

EXPERT REVIEWER Chloe Steele

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

Development and Pragmatic Randomized Controlled Trial of Healthy Ketogenic Diet Versus Energy-Restricted Diet on Weight Loss in Adults with Obesity

SU LIN LIM, MELISSA TAY, SIEW MIN ANG, SHU NING WAI, ET AL.
JOURNAL: NUTRIENTS 2025;16(24):

INTRODUCTION

This randomised controlled trial investigated the effect of a newly developed Healthy Ketogenic Diet (HKD) compared to a standard Energy-Restricted Diet (ERD) on weight loss and metabolic outcomes in adults with obesity.

METHOD

Asian adults (n = 80) with obesity (BMI ≥ 27.5 kg/m2) were randomised into either HKD (N=41) or ERD (n=39) for 6 months.

Both groups followed energy-restricted diets. The HKD also had:

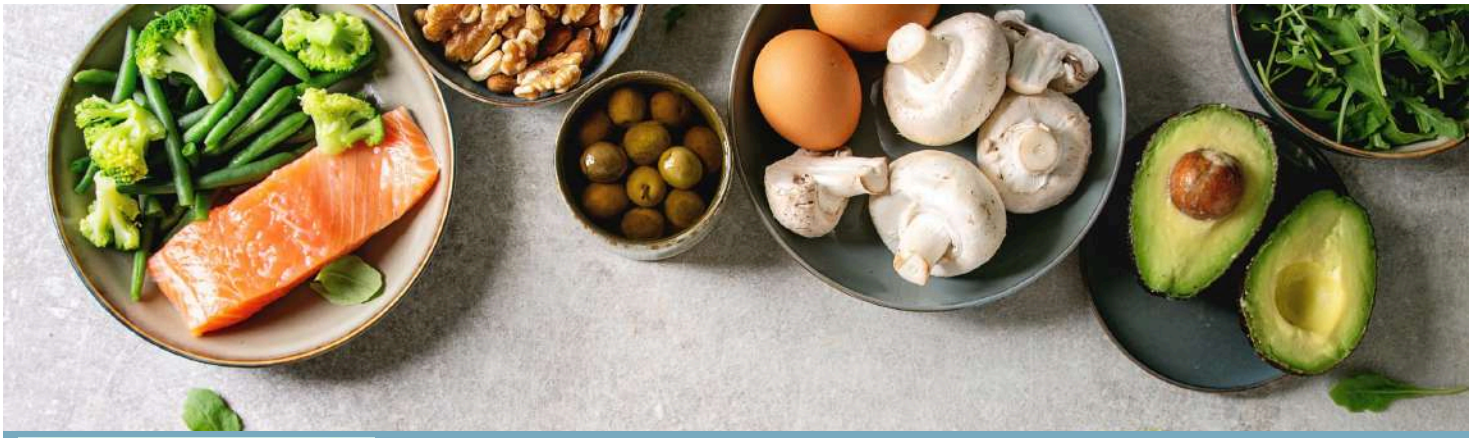
- Limited net carbohydrates to ≤50g/day
- Calorie deficit of at least 500 kcal daily
- Total fat within 50% of energy intak
- Low saturated and trans fat intake
- 25–30% of energy intake from protein (1.0–1.2 g/kg body weight)
- 20–30 g/day of fibre
- At least 2 L of fluid a day
- Micronutrient supplementation.

- Primary outcome was mean weight change from the baseline at 6 months and changes in metabolic profiles including total cholesterol, triglycerides, and LDL-cholesterol, HDL-cholesterol levels, glycated hemoglobin (HbA1c), fasting blood glucose (FBG), alanine transaminase (ALT), aspartate transaminase (AST), systolic blood pressure (SBP), and diastolic blood pressure (DBP).

RESULTS

- HKD group achieved significantly greater mean weight loss at 6 months than the ERD group (−7.8 ± 5.2 kg vs. −4.2 ± 5.6 kg, p = 0.01).
- HKD resulted in significant improvements in metabolic profiles, including reductions in HbA1c (−0.3±0.3%vs. −0.1±0.2%,p=0.008), SBP (−7.7 ± 8.9 mmHg vs. −2.6 ± 12.2 mmHg, p =0.005), and AST (−7.6 ± 15.5 IU/L vs. 0.6 ± 11.5 IU/L, p = 0.01), with no increase in LDL-cholesterol (−0.12 ± 0.60 mmol/L vs. −0.04 ± 0.56 mmol/L, p =0.97) observed in either group.
- HKD group achieved greater reductions in HbA1c, liver enzymes, SBP, total cholesterol, and triglycerides.

HOW TO BRING THIS INTO YOUR CLINICAL PRACTICE



TAKE HOME MESSAGE

- A healthy ketogenic diet may support weight loss and improvements in metabolic profiles through a number of mechanisms.
- Benefits of a ketogenic diet may be achieved even with a net carbohydrate intake of ≤50g/day.
- This study suggests the negative impact of ketogenic diets on LDL cholesterol may be overcome with additional dietary changes, including the reduction of saturated and trans fats, 20-30g fibre daily, 2 L of fluid a day and micronutrient supplementation.

Read the article here

CLINICAL PRACTICE APPLICATIONS

- The goal of a ketogenic diet is ketosis - without analysis of blood and/or urinary ketones it is not known if ketosis was achieved in these participants.
- A ketogenic diet has been defined to include 1.0g/kg body weight, 10–15g carbohydrates per day, and the remaining calories from fat. This HKD included a slightly higher net carbohydrate intake of ≤50g/day which may be preferred by some individuals.
- The negative impact of ketogenic diets on LDL cholesterol may be overcome with the additional dietary changes outlined in this study, including micronutrient supplementation.

CONSIDERATIONS FOR FUTURE RESEARCH

- Repeat studies are required to overcome reporting bias and estimation errors arising from self-reported food diaries.
- This study would benefit from a more controlled clinical setting to better understand physiological effects.
- Future research should include blood and/or urinary ketone analysis.
- Each additional HKD intervention could be independently assessed to determine impact of each.

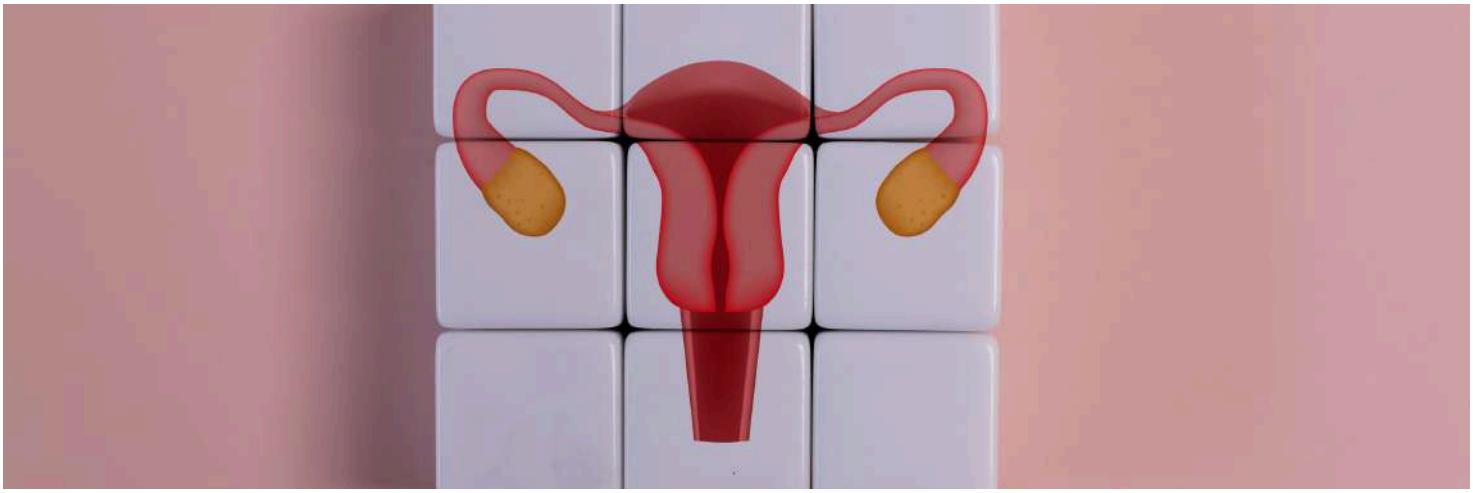
CONCLUSION

- The HKD was more effective than the ERD in promoting weight loss and improving cardiometabolic outcomes in individuals with obesity within a short time frame.

EXPERT REVIEWER Dr. Michelle Barrow

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

Effects of Ketogenic Diet on Weight Loss Parameters among Obese or Overweight Patients with Polycystic Ovary Syndrome: a Systematic Review and Meta-Analysis of Randomized Controlled Trials



FANG REN, HUI YANG, NAN-NAN XING
JOURNAL: FOOD & NUTRITION RESEARCH 2024;68():

INTRODUCTION

Polycystic ovary syndrome (PCOS) is a hormonal and metabolic condition in which patients often experience insulin resistance, dyslipidaemia and difficulty with weight management. This study aimed to evaluate the efficacy of ketogenic diets (KDs) on weight loss (WL) parameters, including weight, waist circumference (WC), body mass index (BMI), and fat mass (FM), in overweight or obese women with PCOS.

METHOD

This was a systematic review and meta-analysis of eleven articles (including twelve trials) involving a total of 426 women with PCOS. Among the included articles, three were randomised, double-blind, placebo-controlled trials comparing ketogenic diets with alternative dietary interventions (such as hypocaloric or balanced diets). Whereas the remaining eight were before–after studies where participants acted as their own controls. The duration of the intervention across the included studies varied from 6 to 24 weeks.

RESULTS

The pooled results of the meta-analysis showed that patients with PCOS following a KD, experienced statistically significant reductions in:

- weight (WMD = −9.13 kg; 95% CI, −11.88 to −6.39; P < 0.01; I 2 : 87.23%)
- BMI (WMD = −2.93 kg/m2; 95% CI, −3.65 to −2.21; P < 0.01; I 2: 78.81%)
- WC (WMD = −7.62 cm; 95% CI, −10.73 to −4.50; P < 0.01; I 2: 89.17%)
- FM (WMD = −6.62 kg; 95% CI, −8.44 to −4.80; P < 0.01; I 2: 53.92%)

HOW TO BRING THIS INTO YOUR CLINICAL PRACTICE

- PCOS can make weight loss more challenging due to its effects on hormonal and metabolic imbalances.
- A KD, which is characterised by low-carbohydrate and high-fat intake, may be a potential dietary regime for promoting...

...reductions in body weight, WC, BMI and FM in overweight or obese women with PCOS.

- Short-term dietary interventions can lead to observable improvements in body measurements for overweight or obese women with PCOS.

Read the article here

CLINICAL PRACTICE APPLICATIONS

- A nutrient-dense low-calorie diet may be an effective nutritional strategy for managing PCOS.
- KDs may be part of a dietary intervention to support WL, BMI reduction, and improved body composition in overweight or obese women with PCOS.
- KDs with very low-carbohydrate intake (<20 g/day) may help improve insulin sensitivity and metabolic outcomes in overweight or obese women with PCOS.
- Personalised protein intake as part of a KD may help maintain lean muscle mass during WL interventions.

CONSIDERATIONS FOR FUTURE RESEARCH

- Long-term follow-up studies are needed to determine whether the benefits of KDs in women afflicted with PCOS can be maintained with less restrictive and more sustainable dietary patterns.
- Future studies should assess the durability of WL and metabolic improvements over a long period of time compared to short-term outcomes reported in current trials.
- Additional larger and well-designed randomised controlled trials are needed to strengthen the current evidence and help determine which dietary patterns are more effective.

CONCLUSION

- The authors concluded that based on KDs potential in decreasing body weight, BMI, WC, and FM in obese or overweight women with PCOS, dietary modifications can play a significant role in supporting weight management in this population.

EXPERT REVIEWER Sarah Cassar

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

KETOGENIC DIET GUIDES

KETO DIET RESOURCES

BANT has developed a dedicated range of resources to complement the personalised nutrition and lifestyle advice given by practitioners in a clinical setting. These resources are open access on our website bant.org.uk and aid further comprehension of nutrition science and clinical interventions.

BANT

Ketogenic Diet

The ketogenic diet (KD) offers a wide array of potential benefits, stemming from its unique ability to transform the body's metabolic state

What is a Ketogenic diet?

A ketogenic diet (commonly referred to as a "keto" diet) is primarily focused on reducing carbohydrate intake and replacing it with healthy fats. This dietary approach forces the body into a metabolic state called ketosis, where the liver converts fats into ketone bodies, which are then used as the primary source of energy instead of glucose.

The recommended carbohydrate intake is typically around 30g/day. Blood glucose levels are kept at a low but healthy level which encourages the body to break down fat into ketones. The aim of ketogenic diets is to promote the metabolic state of ketosis, the burning of these ketones as energy, and for that state to be maintained by the body for a therapeutic period. For ketosis to happen, insulin levels in the body also need to be low.

Keto-adaption or fat-adapted is the name given when the body changes from relying on glucose as its main source of energy to relying on ketones from fat burning.

Personalised Nutrition and the Ketogenic Diet

The keto diet's benefits go beyond just weight loss. Nutritional ketosis can improve various metabolic markers such as;

- lipid profiles, insulin resistance, and blood glucose levels, promoting better cardiovascular health and reducing the risk of non-alcoholic fatty liver disease.

It is important to note that individual responses to the ketogenic diet may vary due to unique genetic and physical factors. Some individuals might encounter difficulties adapting to this dietary regime. Overall, the ketogenic diet is seen as a promising option for those seeking dietary approaches to managing metabolic conditions, in cancer, in epilepsy, and neurological conditions.

Variations of the Ketogenic Diet

- *Standard ketogenic diet (SKD):* This is a very low carb, moderate protein and high fat diet. It typically contains 75% fat, 20% protein, and 5 % carbohydrate.
- *Cyclical ketogenic diet (CKD):* This diet involves periods of higher carb refeeds, such as 5 ketogenic days followed by 2 high carb days.
- *Targeted ketogenic diet (TKD):* This allows you to add carbs around workouts.
- *High protein ketogenic diet:* This is similar to a standard ketogenic diet, but includes more protein. The ratio is often 60% fat, 35% protein, and 5% carbs.

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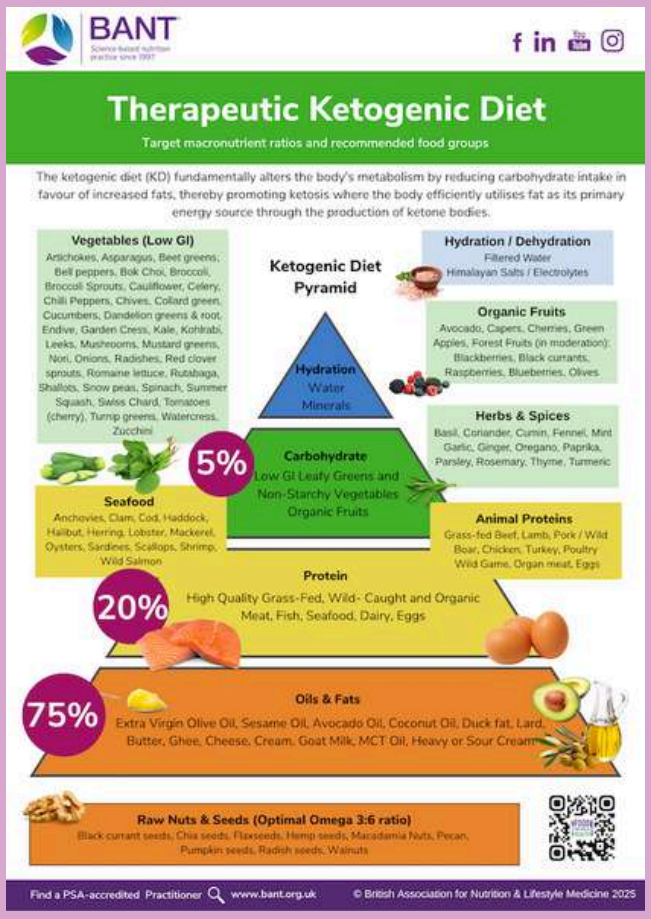

BANT

Therapeutic Ketogenic Diet

Target macronutrient ratios and recommended food groups

The ketogenic diet (KD) fundamentally alters the body's metabolism by reducing carbohydrate intake in favour of increased fats, thereby promoting ketosis where the body efficiently utilises fat as its primary energy source through the production of ketone bodies.

Vegetables (Low GI)
Artichokes, Asparagus, Beet greens, Bell peppers, Bok Choi, Broccoli, Broccoli Sprouts, Cauliflower, Celery, Chilli Peppers, Chives, Collard green, Cucumbers, Dandelion greens & root, Endive, Garden Cress, Kale, Kohlrabi, Leeks, Mushrooms, Mustard greens, Nori, Onions, Radishes, Red clover sprouts, Romaine lettuce, Rutabaga, Shallots, Snow peas, Spinach, Summer Squash, Swiss Chard, Tomatoes (cherry), Turnip greens, Watercress, Zucchini

Ketogenic Diet Pyramid

Hydration / Dehydration
Filtered Water
Himalayan Salts / Electrolytes

Organic Fruits
Avocado, Capers, Cherries, Green Apples, Forest Fruits (in moderation): Blackberries, Black currants, Raspberries, Blueberries, Olives

Hydration
Water
Minerals

Herbs & Spices
Basil, Coriander, Cumin, Fennel, Mint Garlic, Ginger, Oregano, Paprika, Parsley, Rosemary, Thyme, Turmeric

5%
Carbohydrate
Low GI Leafy Greens and Non-Starchy Vegetables
Organic Fruits

Seafood
Anchovies, Clam, Cod, Haddock, Halibut, Herring, Lobster, Mackerel, Oysters, Sardines, Scallops, Shrimp, Wild Salmon

Animal Proteins
Grass-fed Beef, Lamb, Pork / Wild Boar, Chicken, Turkey, Poultry Wild Game, Organ meat, Eggs

20%
Protein
High Quality Grass-Fed, Wild- Caught and Organic Meat, Fish, Seafood, Dairy, Eggs

75%
Oils & Fats
Extra Virgin Olive Oil, Sesame Oil, Avocado Oil, Coconut Oil, Duck fat, Lard, Butter, Ghee, Cheese, Cream, Goat Milk, MCT Oil, Heavy or Sour Cream

Raw Nuts & Seeds (Optimal Omega 3:6 ratio)
Black currant seeds, Chia seeds, Flaxseeds, Hemp seeds, Macadamia Nuts, Pecan, Pumpkin seeds, Radish seeds, Walnuts

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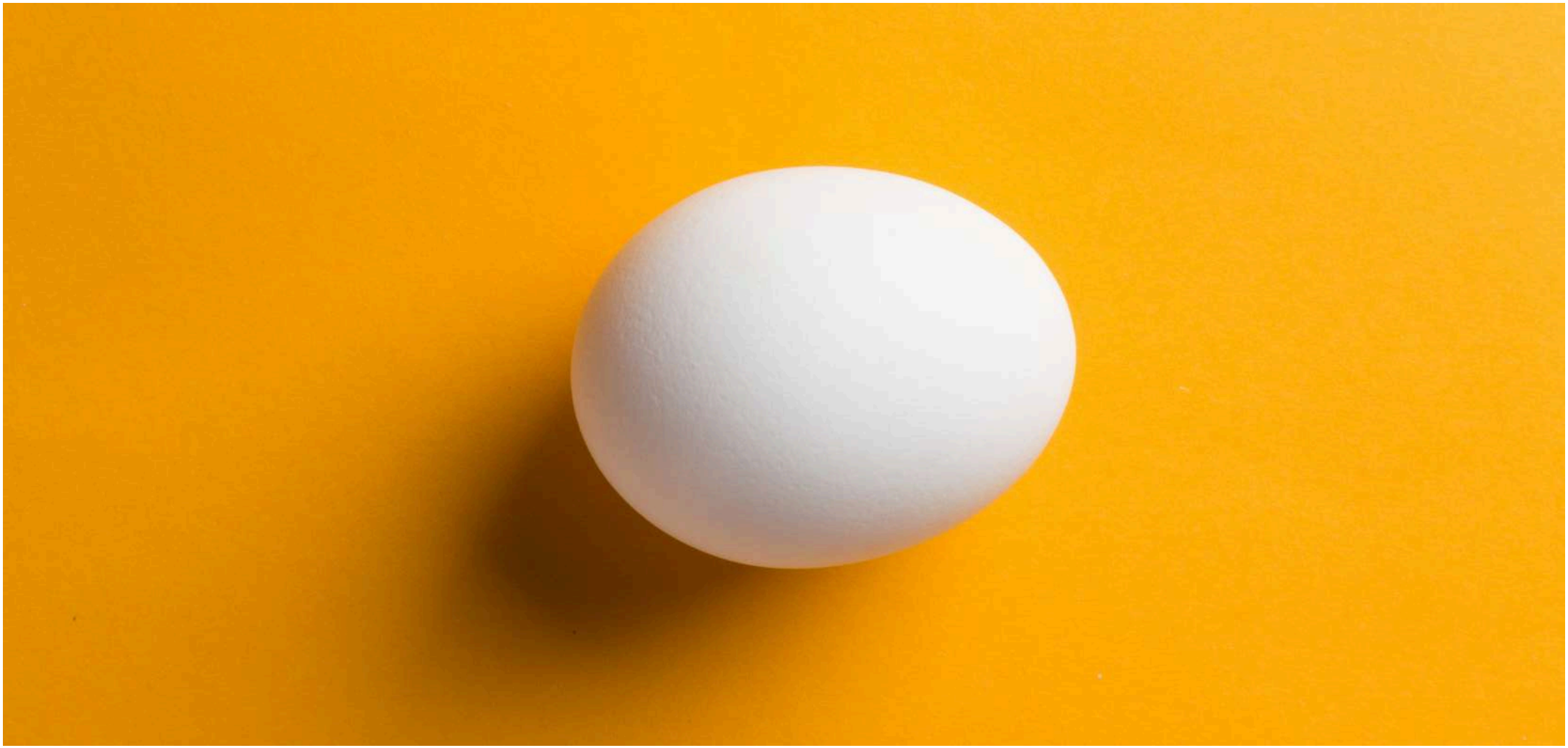
2 REVIEWS

BRAIN HEALTH & MIGRAINE



Effects of Ketogenic Diet on Cognitive Function of Patients with Alzheimer's Disease: a Systematic Review and Meta-Analysis

LIYANG RONG, YATING PENG, QI SHEN, KEYING CHEN, ET AL.
JOURNAL: THE JOURNAL OF NUTRITION, HEALTH & AGING 2024;28(8):100306

TAKE HOME MESSAGE

A Ketogenic Diet may support cognitive function and mental state in patients with Alzheimer's Disease but may also have a negative effect on blood lipids.

Read the article here

INTRODUCTION

- The aim of this review was to evaluate the potential benefits of a ketogenic diet (KD) for cognitive function in patients with Alzheimer's disease (AD).

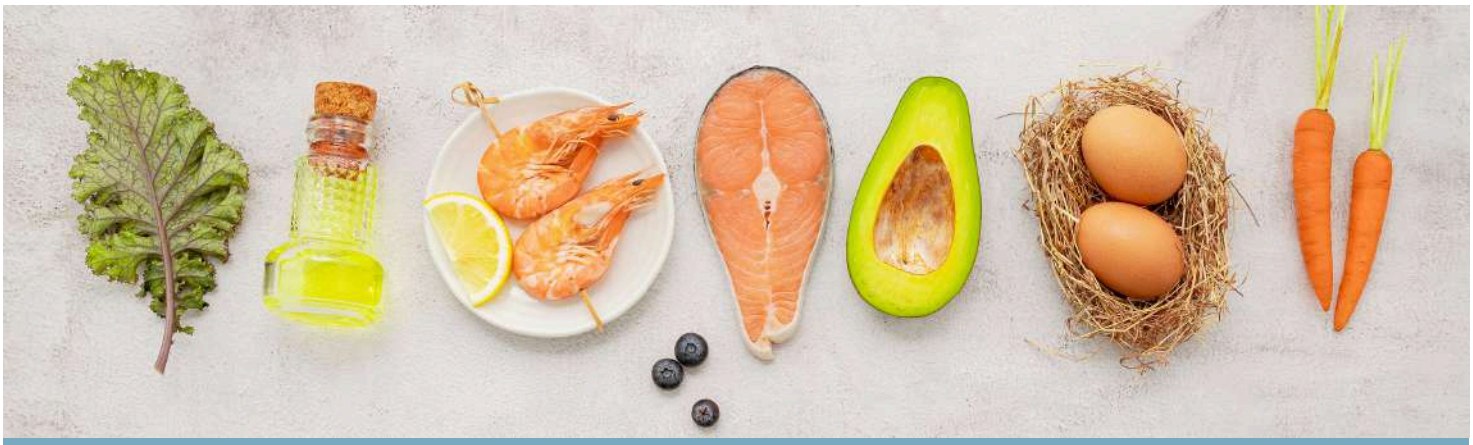
METHOD

- Systematic review and meta-analysis, searching PubMed, Cochrane Library, and Embase.
- Registered on PROSPERO and adhering to PRISMA Guidelines.
- Inclusion criteria: Randomised controlled trials (RCTs), patients with AD (other types of dementia were excluded) and studies using the following outcome measures -Mini mental status examination (MMSE), Alzheimer's Disease Assessment Scale-Cognitive Subscale (ADAS-Cog), daily living activities (ADL), and the Nishimura Geriatric Rating Scale for Mental Status (NM).
- Cochrane Risk of Bias tool was used to assess study quality.

RESULTS

- 10 RCTs with a total of 691 AD patients were included. Duration of intervention ranged from 12-60 weeks.
- None of the studies had an overall high risk of bias.
- Improvements were seen in MMSE (p=0.003, n=5), ADAS-cog (p=0.002, n=3) and NM (p=0.003, n=2) but not in ADL (p=0.95, n=2).
- 4 of the studies evaluated blood lipids and meta-analysis showed an increase in triglycerides (p=0.02) and LDL-cholesterol (p=0.03), with no change in total or HDL cholesterol (p=0.09 and p=0.63, respectively).

HOW TO BRING THIS INTO YOUR CLINICAL PRACTICE



CLINICAL PRACTICE APPLICATIONS

- A ketogenic diet could be considered to support cognitive function and mental state in patients with Alzheimer's Disease.
- When advising on a ketogenic diet, blood chemistry, including blood lipids, should be monitored.
- The impacts of long-term KD use in relation to the gut microbiome and dietary compliance should be considered when recommending KD as a dietary strategy for Alzheimer's Disease.

CONSIDERATIONS FOR FUTURE RESEARCH

- Research to determine the optimal type of ketogenic diet/diet composition to support cognitive function.
- Clinical trials of a KD in other types of dementia.

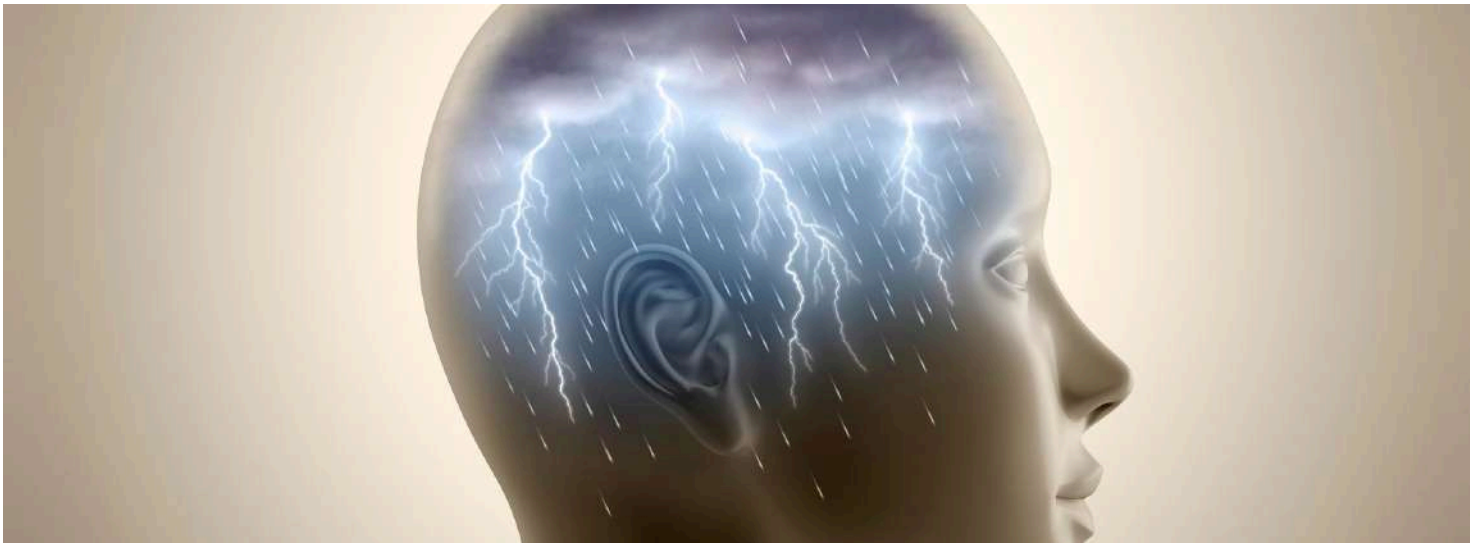
CONCLUSION

- The authors conclude that there is "compelling evidence" for the potential of a KD in patients with AD whilst also highlighting the potential negative effects on blood lipids.

EXPERT REVIEWER Karin Elgar PhD

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

Very-Low-Calorie Ketogenic Diet vs Hypocaloric Balanced Diet in the Prevention of High-Frequency Episodic Migraine: the EMIKETO Randomized, Controlled Trial.



MASSIMILIANO CAPRIO, ELEONORA MORICONI, ELISABETTA CAMAJANI, ET AL
JOURNAL: JOURNAL OF TRANSLATIONAL MEDICINE 2023;21(1):692

INTRODUCTION

- The aim of this study was to compare the effects of a very low-calorie ketogenic diet (VLCKD) with a hypocaloric balanced diet (HBD) on migraine prevention in adults with high-frequency episodic migraine (HFEM) and overweight/obesity.

METHOD

- 24-week randomised controlled trial, including 57 adults aged 18-65 years with HFEM and a body mass index between 25-35kg/m2.
- Patients on the VLCKD diet plan received a diet containing less than 800 kcal per day (75-105g protein, 30-50g carbohydrate (CHO), 20g fat, 25g fibre) for the first 4 weeks. This was followed by a low-calorie diet for 4 weeks during which CHO were gradually reintroduced. From week 12 the group followed the HBD.
- Participants in the control group followed the HBD with a calorie intake of 1500-1600kcal per day (30% fat, 55% CHO, protein approximately 0.8-1.5g/kg ideal bodyweight) for the duration of the study.
- The primary outcome measure was monthly migraine days (MMD). Secondary outcomes included anthropometric parameters, blood lipids, glycaemic and inflammatory parameters, electrolytes, liver and kidney function tests.

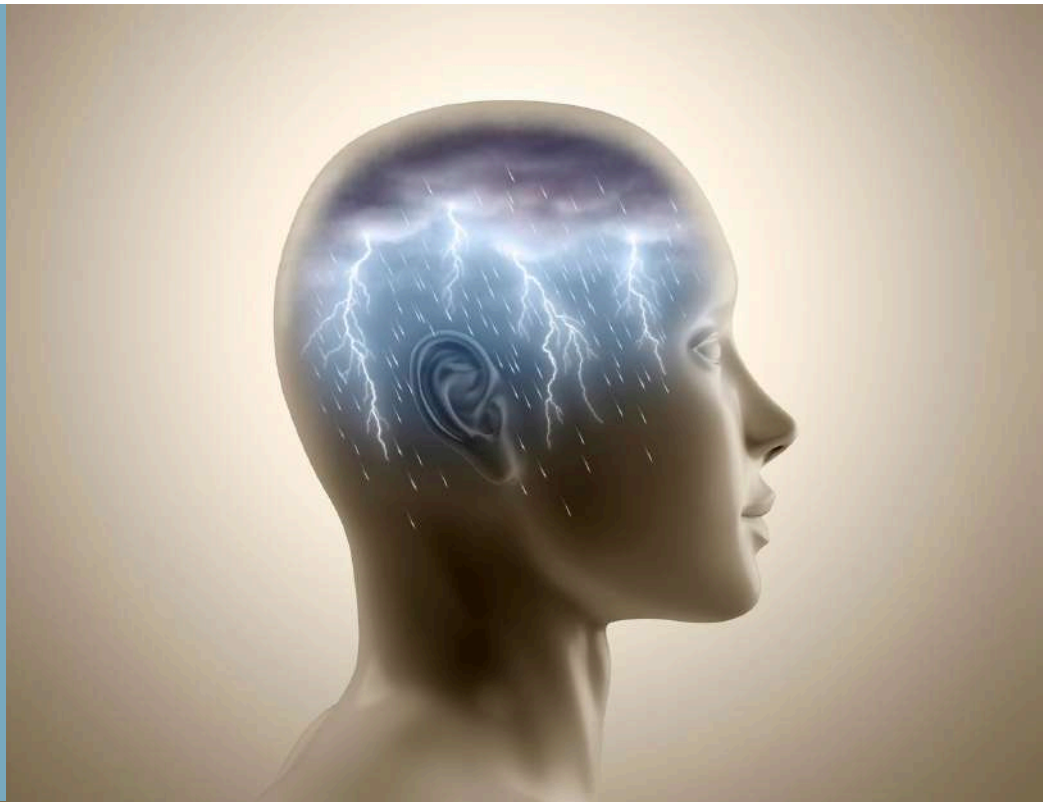
RESULTS

- 13.8% of participants on the VLCKD diet and 50% on the HBD dropped out of the study, although no attrition bias was seen in sensitivity analysis.
- Patients on the VLCKD had a statistically significant reduction in MMD after 8 weeks (− 6.4±4.8 vs − 2.2±5.0, p=0.008). The reduction remained greater in the VLCDK group after reintroducing CHO and switching to a HBD (week 12: − 7.2±5.42 vs − 3.13±3.58, p=0.007; week 24: − 6.8±6.42 vs − 3.6±3.3, p=0.042).
- Weight loss was greater in the VLCKD compared to the control group (week 8: − 8.2±4.5 vs − 4.3±2.9, p=0.002; week 12: − 9.1±6.4 vs − 4.9±2.7, p=0.020; 24 weeks: − 9.1±6.4 vs − 4.3±2.9, no p-value given).
- At week 8, adherence to the different dietary protocols was higher in the VLCKD than in the HBD group (p=0.028).
- There was a greater reduction in glucose levels in the VLCKD group at week 12 (p=0.003) as well as a greater reduction in AST (p=0.046). There were no significant differences in other blood tests.

HOW TO BRING THIS INTO YOUR CLINICAL PRACTICE

- A very low calorie ketogenic diet (VLCKD) may help reduce migraine frequency in patients with HFEM and overweight/obesity.

Read the article here



CLINICAL PRACTICE APPLICATIONS

- A short-term VLCKD can be considered in patients with HFEM and overweight/obesity to help reduce migraine frequency and weight.

CONSIDERATIONS FOR FUTURE RESEARCH

- Clinical trials on a ketogenic diet (not low calorie) in normal weight migraineurs, to establish whether benefits are mediated via weight.
- Clinical trials of ketogenic diets in patients with other types of migraines.
- Larger studies with consideration for factors that led to high drop-out rates on HBD.

CONCLUSION

- The authors conclude that a VLCKD is an effective treatment or adjuvant therapy for patients with HFEM.

EXPERT REVIEWER Karin Elgar PhD

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

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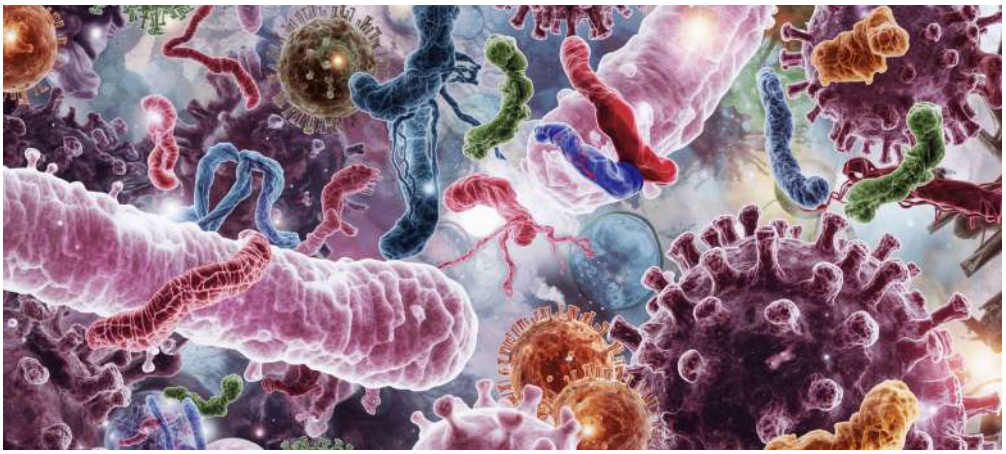
INFLAMMATION

2 REVIEWS

Ketogenic Diet Induced Shifts in the Gut Microbiome Associate with Changes to Inflammatory Cytokines and Brain-Related miRNAs in Children with Autism Spectrum Disorder



NINA P ALLAN, BRENNAN Y YAMAMOTO, BRADEN P KUNIHIRO, ET AL.
JOURNAL: NUTRIENTS 2024;16(10):1401

TAKE HOME MESSAGE

- Given the impact of neuro-inflammation on metabolic pathways, gut microbiome and ASD patients' social behaviour, an anti-inflammatory diet is an important consideration for this cohort.
- Supplementation options such as butyric acid might also be considered given its effect on inflammation, gut dysbiosis, and BDNF.

Read the article here

INTRODUCTION

This children's interventional pilot study evaluated the change in inflammatory markers that has consistently been shown to be elevated in individuals within the autism spectrum disorder (ASD) following a ketogenic dietary (KD) intervention. The study furthermore assessed gut microbiome composition given that gastrointestinal problems have additionally been shown to be notable in this cohort.

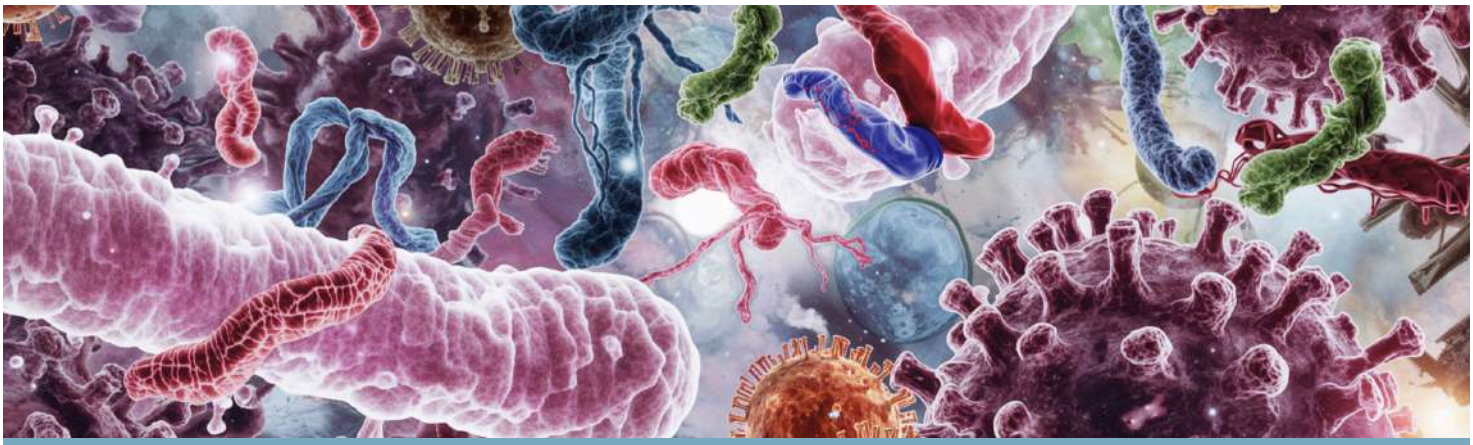
METHOD

- The initial cohort included 47 children (aged 7-19 years). Analysis was based on 11 children's non-fasting blood samples and 7 children's at-home collected stool samples. Samples were collected pre- and post a 4-month KD intervention that included daily medium-chain triglyceride (MCT) oil supplementation with carbohydrate intake restricted to 20-25 g/day and protein intake based on weight and age of each participant.
- There was no clearly defined targeted level of ketosis to be maintained. Blood plasma was used both in ketone body- and 14-plex cytokine Luminex assays. Stool samples were subjected to DNA and RNA isolation using 16S sequencing and BUK/BAC qPCR assays.

RESULTS

- The authors noted a significant increase in concentrations of acetoacetic acid (by 0.97 nM ± 0.66; p = 0.02) and hydroxybutyric acid (by 0.63 nM ± 0.38; p = 0.02) ketone bodies.
- Significant bacterial changes included Lactobacillales order (p = 0.02), Bacteroidaceae family (p = 0.03), Oscillospiraceae family (p = 0.04), Ruminococcus genus (p = 0.04), Bacteroides genus (p = 0.03), Ruminococcus gnavus (p = 0.03), and Clostridium cocleatum (p = 0.04).
- Additional observations included significantly decreased plasma levels of brain-derived neurotrophic factor (BDNF). Observed changes in miRNAs that have been linked with BDNF regulation in the brain included significant reduction of miR-134 (p = 0.008) and miR-132 (p = 0.027).

HOW TO BRING THIS INTO YOUR CLINICAL PRACTICE



CLINICAL PRACTICE APPLICATIONS

- A comprehensive stool analysis should be considered for any ASD suggested dietary intervention protocol to assess the specific needs of the individual in terms of gut microbiome status.
- A carefully balanced KD or at least a gluten-free diet should be considered for ASD individuals to address inflammation and gut microbiome dysbiosis shown to be elevated in ASD patients.

CONSIDERATIONS FOR FUTURE RESEARCH

- Larger studies are needed to elicit whether a KD should form part of a standardised treatment protocol for ASD individuals.
- Cohorts need to include an even distribution between age and sex to determine the effect of a KD on both neuro-inflammatory and gut microbiome markers for the individual.
- The direct impact on improvement of social behaviour in ASD individuals following a KD requires further investigation.

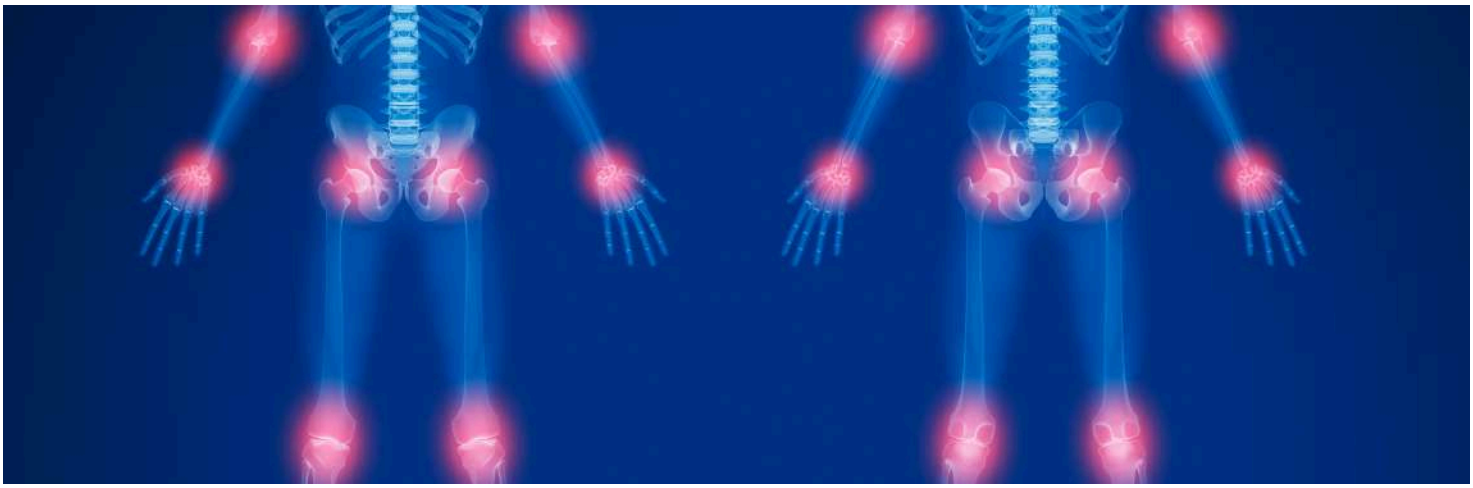
CONCLUSION

A KD may directly target brain activity via its effect on metabolic pathways, particularly those that impact on sociability in ASD patients. This children's study showed that a short KD intervention can reduce certain inflammatory markers and furthermore improve gut microbiome composition whilst also increasing the production of butyrate. Butyrate is shown to reduce inflammation through its effect on cytokines, neurotransmitters, and miRNAs associated with BDNF.

EXPERT REVIEWER Wilma Kirsten

CONFLICTS OF INTEREST: None
EVIDENCE CATEGORY: C: Non-randomized trials, observational studies, narrative reviews

The Effect of a Ketogenic Diet versus Mediterranean Diet on Clinical and Biochemical Markers of Inflammation in Patients with Obesity and Psoriatic Arthritis: A Randomized Crossover Trial



VAIA LAMBADIARI, PELAGIA KATSIMBRI, AIKATERINI KOUNTOURI, ET AL.
JOURNAL: INTERNATIONAL JOURNAL OF MOLECULAR SCIENCES 2024;25(5):2475

INTRODUCTION

Introduction: A randomised cross-over trial was conducted to evaluate the effectiveness of the Mediterranean Diet (MD) with an isocaloric Ketogenic Diet (KD), in patients with obesity and psoriasis along with psoriatic arthritis.

METHOD

- Twenty-six participants, with a mean age of 52, were randomly assigned to either the MD or KD group for eight weeks. After a six-week washout interval, the two groups were switched to the other type of diet for eight weeks. Sixteen out of the 23 participants (69%) completed the trial.
- The primary outcomes were to investigate changes in clinical scores of disease severity - Psoriasis Area and Severity Index (PASI) and Disease Activity Index for Psoriatic Arthritis (DAPSA) - after an 8-week KD intervention compared to an 8-week MD intervention.

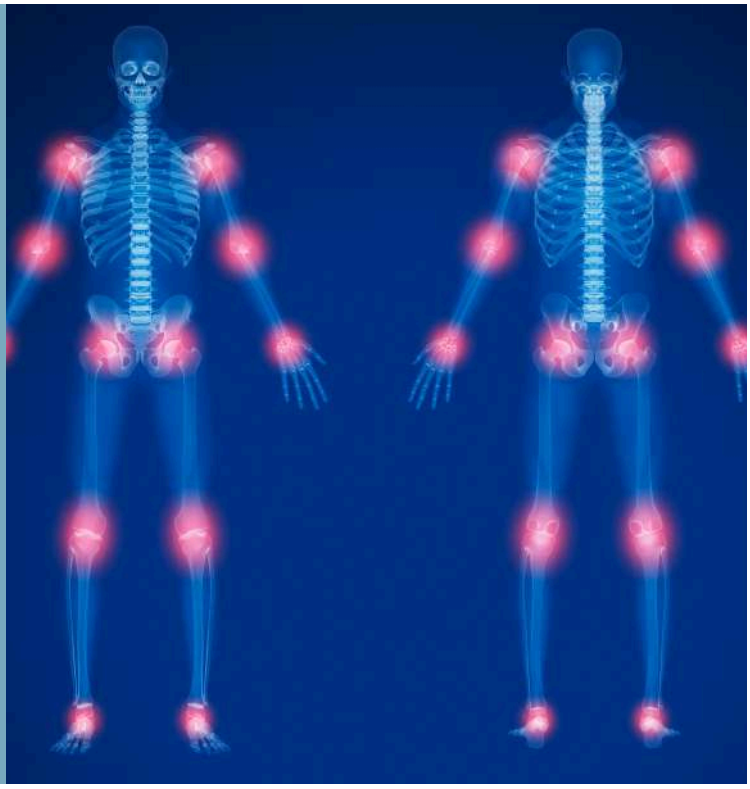
RESULTS

- After KD patients displayed a reduction in triglycerides (TGs) compared with baseline (p=0.022). No changes in TGs were observed after MD.
- KD patients displayed a significant reduction in PSAI (p=0.04) compared to baseline, showing a 2-fold higher reduction compared to the MD (-61.58% vs -31.24%, p=0.038).
- Similarly, KD resulted in a significant reduction in DAPSA (p=0.004) compared to baseline, showing a 3-fold higher reduction compared to the MD (-98.62% vs. -32.64%, p=0.034).
- KD patients displayed a significant reduction in IL-6 (p=0.047) compared to baseline, showing a 2-fold higher reduction compared to MD (-55.6% vs. -20.56%, p=0.041).
- Additionally, KD patients showed a reduction in IL-17 (p=0.042) and IL-23 (p=0.037), whereas no significant differences were observed in the inflammatory markers in MD.

HOW TO BRING THIS INTO YOUR CLINICAL PRACTICE

Read the article here

- A short-term ketogenic diet is reported to reduce inflammation and disease activity in patients with obesity and psoriatic arthritis, performing better than a Mediterranean diet in clinical and biochemical outcomes.
- A Ketogenic diet may be considered as an adjunct in the comprehensive management of psoriatic arthritis, especially in obese patients.



CLINICAL PRACTICE APPLICATIONS

- This cross-over clinical trial demonstrates that a short-term ketogenic diet (KD) may help reduce disease activity and reduce inflammatory markers in patients with obesity and psoriatic arthritis, more effectively than a Mediterranean diet (MD).
- KD is reported to have improvements in PASI and DAPSA scores, as well as reductions in IL-6, IL-17, and IL-23.
- Clinicians may consider offering a ketogenic diet as adjunctive to pharmacological treatment for managing inflammation and disease severity in psoriatic conditions in obese patients.

CONSIDERATIONS FOR FUTURE RESEARCH

- The sample size of this study was small, therefore large and more diverse patient population groups are needed for future studies.
- The findings of this study identify an association between dietary interventions and auto-immune disorders and thus emphasise a need for more interventional trials to compare different dietary patterns.
- Further investigations are needed to explore the molecular and immunological mechanism by which ketogenic diets modulate specific cytokines and pathways involved in psoriatic disease.

CONCLUSION

Conclusion: This cross-over trial concluded that patients on a Ketogenic diet experienced greater reductions in clinical scores of disease activity and inflammatory markers in obese patients with psoriasis and psoriatic arthritis than those on a Mediterranean diet.

EXPERT REVIEWER Ana-Paula Agrela

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

CLINICAL GUIDES

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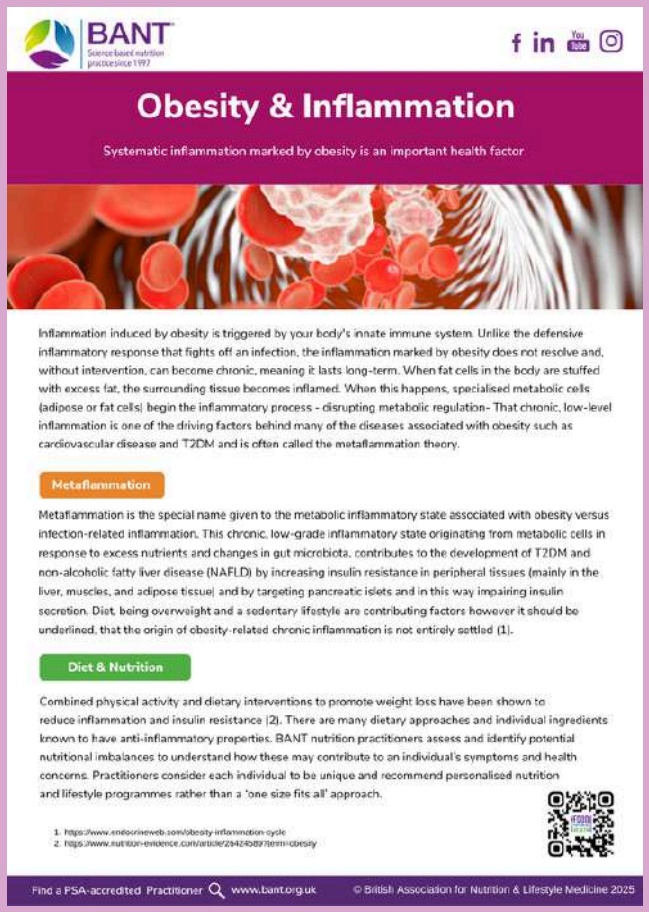
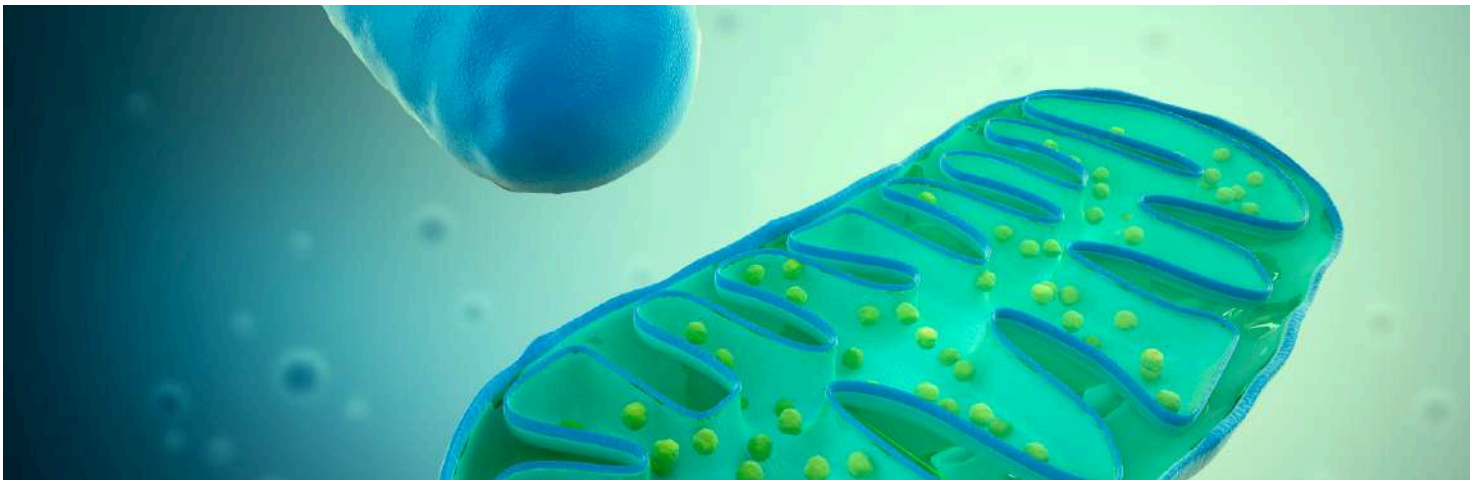


Access further resources here

MITOCHONDRIAL FUNCTION

1 REVIEW

Intermittent Fasting, Calorie Restriction, and a Ketogenic Diet improve Mitochondrial Function by Reducing Lipopolysaccharide Signaling in Monocytes during Obesity: A Randomized Clinical Trial.



MARTHA GUEVARA-CRUZ, KARLA G HERNÁNDEZ-GÓMEZ, CITLALLY CONDADO-HUERTA, ET AL.
JOURNAL: CLINICAL NUTRITION (EDINBURGH, SCOTLAND) 2024;43(8):1914-1928

INTRODUCTION

- Mitochondrial dysfunction in monocytes is linked to inflammation in obesity.
- This study assessed the impact of calorie restriction (CR), intermittent fasting (IF), and a ketogenic diet (KD) and ad libitum habitual diet (AL) on mitochondrial function in monocytes.
- In addition, it evaluated how changes in the gut microbiota, resulting from these dietary interventions and treatment with rifaximin, a broad-spectrum, non-absorbed rifamycin antibiotic, impacted monocyte function.

METHOD

- The study was an open randomised controlled clinical trial conducted in Mexico City (July 2022-March 2023).
- Participants were aged between 18-60 with a BMI between 30- 50kg/m2.
- 44 of 58 participants completed the 8 week long study, and were randomly assigned to each group, CR, IF, KD or AL; 11 per group.
- Participants followed the diet for 1 month, then 7 days of rifixamin was administered.
- Mitochondrial function parameters included several markers oxygen consumption rate (OCR), ATP linked respiration, proton leakage, bioenergetic health index (BHI), nonmitochondrial respiration, spare capacity and glycolysis, amongst others.
- Lipopolysaccharide (LPS) was used as a marker of inflammation.
- Gut microbiota diversity was measured using the Shannon diversity index.

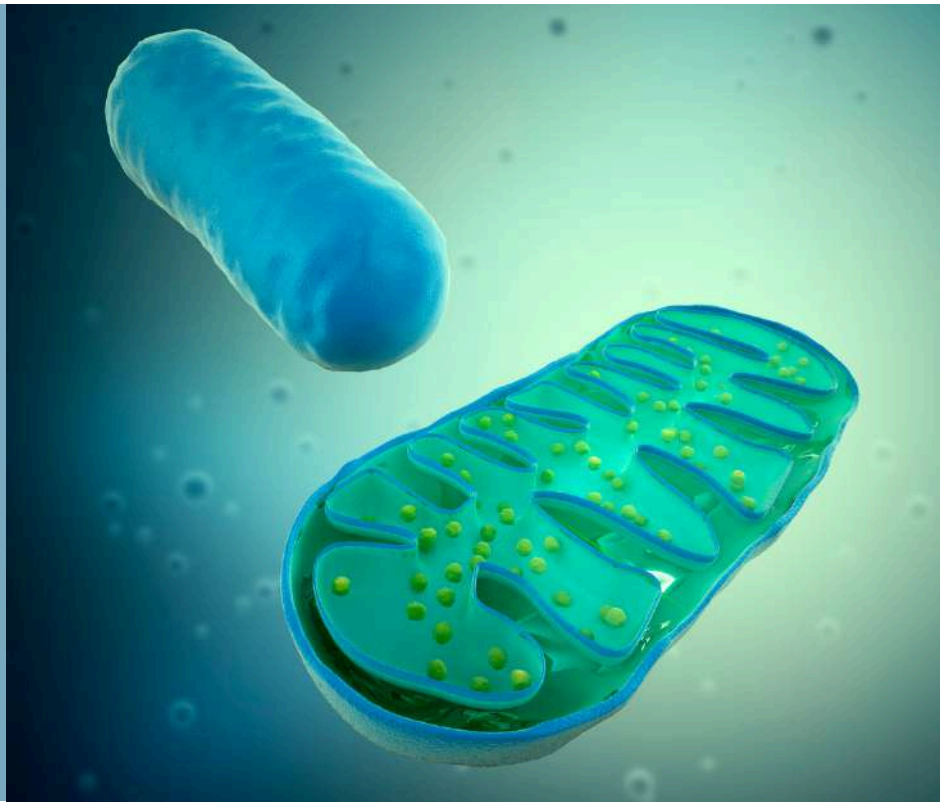
RESULTS

- Maximal respiration significantly improved comparing all three groups to AL ($p = 0.025$, $n2p = 0.159$, CI 95% = 0.05,0.27)
- BHI increased in all three dietary interventions by week 8 ($p = 0.0003$, $n2p = 0.203$, 95% CI = 0.09,0.32).
- Serum LPS decreased in CR ($p = 0.001$), IF ($p = 0.0001$), KD ($p = 0.004$) and was inversely correlated with mitochondrial health (MR: $r2 = -0.4$, $p = 0.02$; BHI: $r2 = -0.3$, $p = 0.057$).
- Gut microbiota diversity increased in IF and KD at 8 weeks ($p=0.0001$).
- Beneficial bacteria increased: Phascolarctobacterium faecium (CR; $p = 0.019$) and Ruminococcus bromii (CR and KD; $p = 0.02$).

HOW TO BRING THIS INTO YOUR CLINICAL PRACTICE

- In obese individuals following dietary interventions, specifically CR, IF and KD, may help to improve mitochondrial efficiency due to their positive impact on the microbiome and inflammation improving metabolic resilience in these individuals.

Read the article here



CLINICAL PRACTICE APPLICATIONS

- Measuring mitochondrial function might help to provide evidence of the effectiveness of dietary interventions including CR, IF and KD where it can be used as an indicator of metabolic and inflammatory status.

CONSIDERATIONS FOR FUTURE RESEARCH

- As there are few studies to report the use of this analysis in dietary intervention studies more research is needed to support these findings.
- A larger sample size with balanced gender representation would reduce result variability and provide more robust data to support new interventions and guide future research.
- Including metabolomic analysis in future research could help to identify if there are any mediators which contribute to mitochondrial dysfunction.
- To understand if the observations found stem from dietary intervention, rifaximin administration or both, further studies would benefit from including a placebo alongside the rifaximin administration.

CONCLUSION

- In individuals with obesity, the following dietary interventions CR, IF and KD improved mitochondrial bioenergetic marker levels.
- Decreased serum LPS and gut modulation may play a role in the changes observed in the monocyte energetic profile.

EXPERT REVIEWER Nicky Ester

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

MITOCHONDRIA GUIDES

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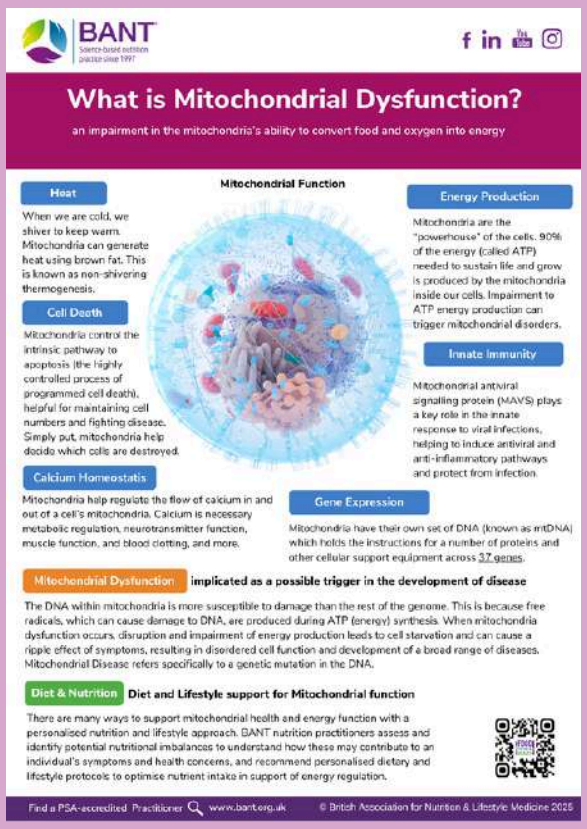
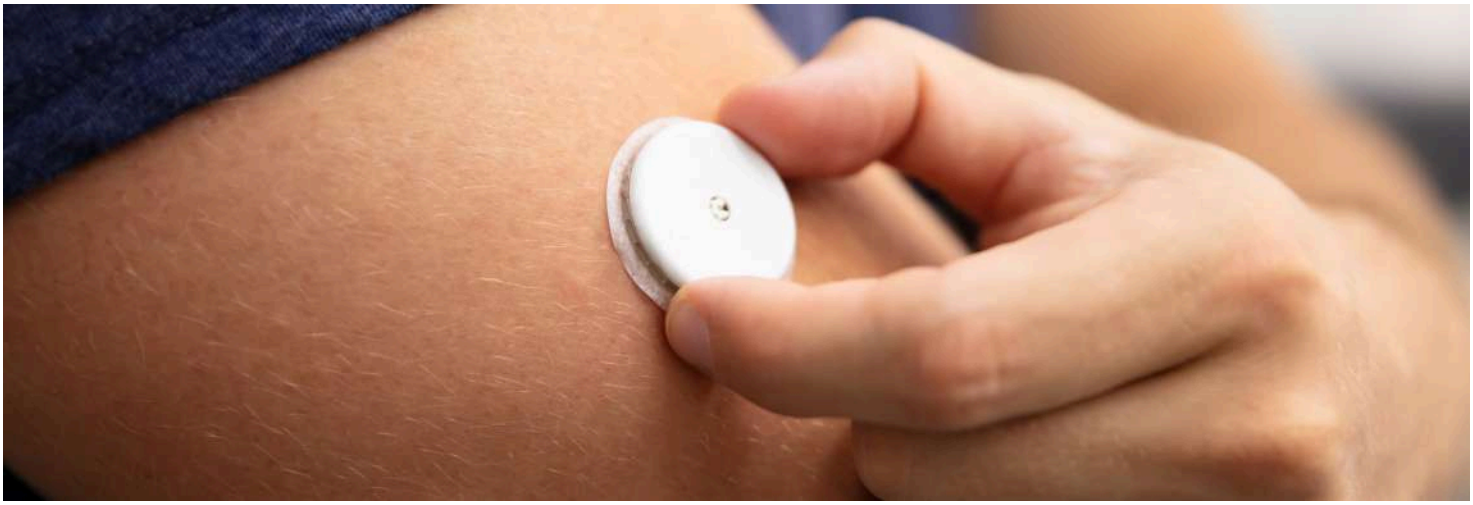


Access further resources here

TYPE 2 DIABETES MELLITUS

7 REVIEWS

Impact of Continuous Glucose Monitoring Versus Blood Glucose Monitoring to Support a Carbohydrate-Restricted Nutrition Intervention in People with Type 2 Diabetes



CAROLINE G P ROBERTS, BRITTANIE M VOLK, HOLLY J WILLIS,
JOURNAL: DIABETES TECHNOLOGY & THERAPEUTICS 2025;27(5):341-356

INTRODUCTION

A randomised, two-arm, parallel-group study was conducted in adults with type 2 diabetes (T2D) to compare changes in time in range (TIR) using continuous glucose monitoring (CGM) versus blood glucose monitoring (BGM) over three months.

METHOD

A total of 163 participants with T2D (mean age: 52 years, mean HbA1c: 8.1%) and on glucose-lowering medication were randomly assigned to either the CGM or BGM group for 12 weeks.

All participants followed a medically supervised ketogenic diet (MSKD) and were instructed to consume no more than 30 grams of carbohydrates per day.

CGM participants used the FreeStyle Libre device, while all participants performed two daily fingerstick tests to measure blood ketone levels.
In the CGM arm, data from the Libre 2 system was accessible to the study team but not to the participants or the care team, whereas in the BGM arm, both participants and the care team were blinded to the continuous glucose monitoring (CGM) data from the Freestyle Libre Pro system.

The primary outcome was the change in CGM-derived TIR (% time with glucose 70–180 mg/dL; 3.9-10 mmol/L) from baseline to M3.

The secondary outcomes included: (1) change in other CGM-derived metrics from baseline to month 1 (M1) and M3. (2) Percent of participants reaching CGM-derived consensus targets at M3. (3) change in HbA1c from baseline to M3. (4) 90-day average blood-beta-hydroxybutyrate (BHB) levels.

RESULTS

- TIR improved significantly in both groups: by 28% in the CGM group (from 61% to 89%) and by 22% in the BGM group (from 63% to 85%) ($p<0.001$); no significant difference between arms ($p=0.26$).
- A greater percentage of CGM users reached the combined glycaemic targets of TIR >70% and time below range (TBR) <4%, by M3 compared to BGM users ($p = 0.04$).
- Medication use was reduced in both groups between baseline and M3 ($p < 0.001$), with no differences between CGM and BGM groups ($p = 0.79$).
- HbA1c decreased by 1.6% (8.1%–6.5%) in the CGM group and 1.5% (8.1%–6.6%) in the BGM group ($p < 0.001$). A smaller reduction was reported in the BGM group.
- Both groups maintained a lowered-end of nutritional ketosis, with 90-day mean BHB levels of 0.8 mmol/L in the CGM group and 0.7 mmol/L in the BGM group ($p = 0.07$).

HOW TO BRING THIS INTO YOUR CLINICAL PRACTICE

- A medically supervised ketogenic diet may result in substantial improvements in glycaemic control, medication reduction, and weight loss in individuals with T2D.
- Both CGM and BGM are effective tools for supporting a ketogenic dietary intervention; however, CGM may offer added benefits of patient confidence and attainment of glycaemic targets.
- The personalisation of glucose monitoring strategies based on a patient's individual needs, preferences, and resources remains essential in optimizing diabetes care.

Read the article here

CLINICAL PRACTICE APPLICATIONS

- Medically supervised ketogenic diet programs (MSKDP) have been shown to improve glucose control,whether patients use continuous glucose monitoring (CGM) or blood glucose monitoring (BGM).
- Both the CGM and BGM groups saw a reduction in the need for glucose-lowering medication, highlighting the effectiveness of close monitoring during these dietary changes.
- Despite metabolic improvements, both groups showed inadequate fiber intake and poor overall diet quality, emphasizing the need for ongoing nutritional counseling to prevent long-term health issues.

CONSIDERATIONS FOR FUTURE RESEARCH

- The study sample was predominantly white, middle-aged, and already enrolled in a digital MSKDP. Future studies should include more diverse populations to enhance generalisability.
- Long-term studies are needed to assess the durability of glycaemic and weight improvements, adherence to carbohydrate-restricted diets, and long-term safety of such interventions.
- Additional research is required to better understand the clinical significance of glucose levels <70 mg/dL, but >54 mg/dL, particularly in individuals at a low risk for hypoglycaemia, including those with prediabetes and people with T2D who are not using insulin. To potentially refine glucose management guidelines and improve patient outcomes in this overlooked range.

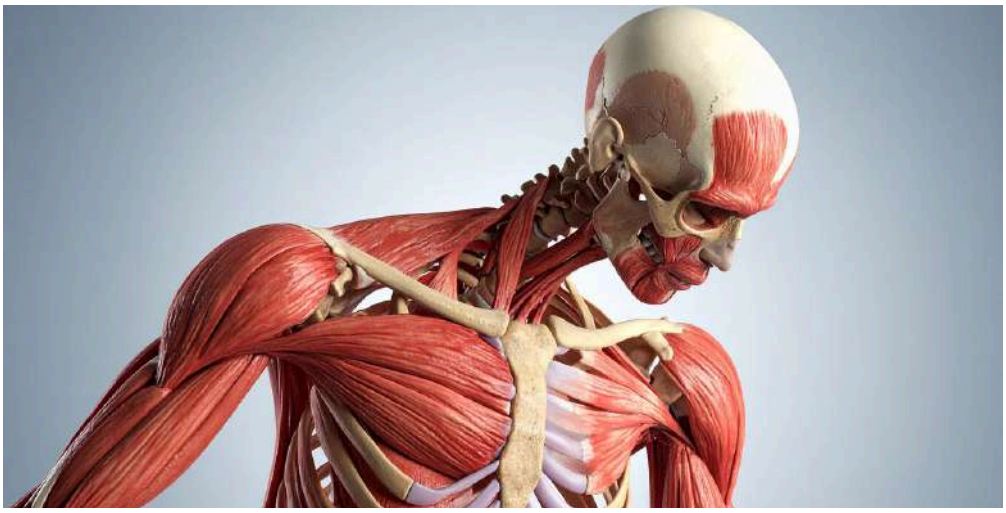
CONCLUSION

This randomised controlled trial demonstrated that both CGM and BGM, when combined with a ketogenic diet and remote care, significantly improved glycaemic outcomes (TIR, TAR, and HbA1c). However, there were no statistically significant differences between the two monitoring methods.

EXPERT REVIEWER Ana Paula Agrela

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

A 3-Week Ketogenic Diet Increases Skeletal Muscle Insulin Sensitivity in Individuals With Obesity: A Randomized Controlled Crossover Trial



THIEN VINH LUONG, METTE GLAVIND BÜLOW PEDERSEN, CAROLINE BRUUN ABILD, ET AL. JOURNAL: DIABETES 2024;73(10):1631-1640

TAKE HOME MESSAGE

- In the short-term, the KD may kickstart blood-sugar control and weight loss in individuals with obesity through reduced circulating insulin levels and improved muscle insulin sensitivity.
- However, effects are unclear in the long-term.

Read the article here

INTRODUCTION

- The ketogenic diet (KD) has been shown in previous research to improve glycaemic control and decrease the reliance on glucose-lowering drugs in individuals with type 2 diabetes (T2D). However,mechanisms behind this are unclear.
- This study aimed to determine whether improved glycaemic control associated with KD was because of improved insulin sensitivity in skeletal muscle, liver and adipose tissue in individuals with obesity and insulin resistance (IR).

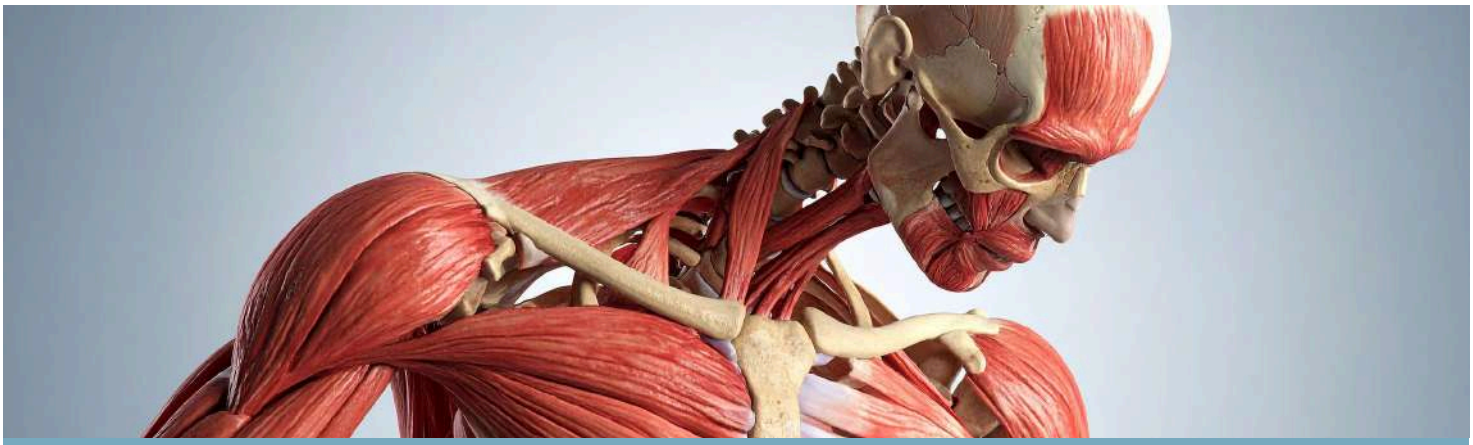
METHOD

- This was a randomised control crossover trial of 11 individuals who were given a 3-week ketogenic diet and a 3-week standard diet, separated by a 1-week washout period.
- The KD was isocaloric with 5% energy from carbohydrates, 20% energy from protein, and 75% energy from fat.
- Participants were responsible for cooking meals and adhering to the diet at home.
- To ensure adherence, the ketone beta-hydroxybutyrate was measured and individuals were excluded if they did not reach ketosis.
- Body chemistry and composition were measured.
- Insulin sensitivity was assessed using a hyperinsulinaemic-euglycaemic clamp.

RESULTS

- When individuals were eating a KD, their weight, body mass index, fat mass, lean mass, and triglycerides were all significantly reduced compared to standard diet (P=<0.05 for all).
- Blood glucose levels were also significantly reduced with KD compared to standard diet (P= <0.001).
- Compared to the standard diet, there was an increase in the rate of glucose uptake by skeletal muscle after the KD (ΔR_d 2.0 [1.7–3.4] vs. 1.6 [0.9–2.9] mg/kg/min; P= < 0.05).
- No improvements in hepatic insulin sensitivity were seen with the KD. (ΔEGP% 71 ± 31% vs. 78 ± 15%; P = 0.53).
- There was a reduction in lipolysis during KD, which indicated that adipose tissue insulin sensitivity was reduced (62 ± 16 vs.76±8%; P= < 0.05).

HOW TO BRING THIS INTO YOUR CLINICAL PRACTICE



CLINICAL PRACTICE APPLICATIONS

- In the short-term, a KD may reduce blood glucose levels in obese individuals and induce weight loss.This may be due to decreased circulating insulin levels and associated increased muscle insulin sensitivity.
- However, given that liver insulin sensitivity remained unchanged and adipose tissue became less sensitive to insulin, the long-term effects of the KD on blood glucose management is unclear.
- Clinicians should closely monitor blood glucose in individuals with obesity who undertake a KD.

CONSIDERATIONS FOR FUTURE RESEARCH

- Future research should focus on the long-term effects of KD on blood glucose management.
- Other mechanisms of action could also be investigated such as whether KD affects insulin-mediated hepatic glucose production.

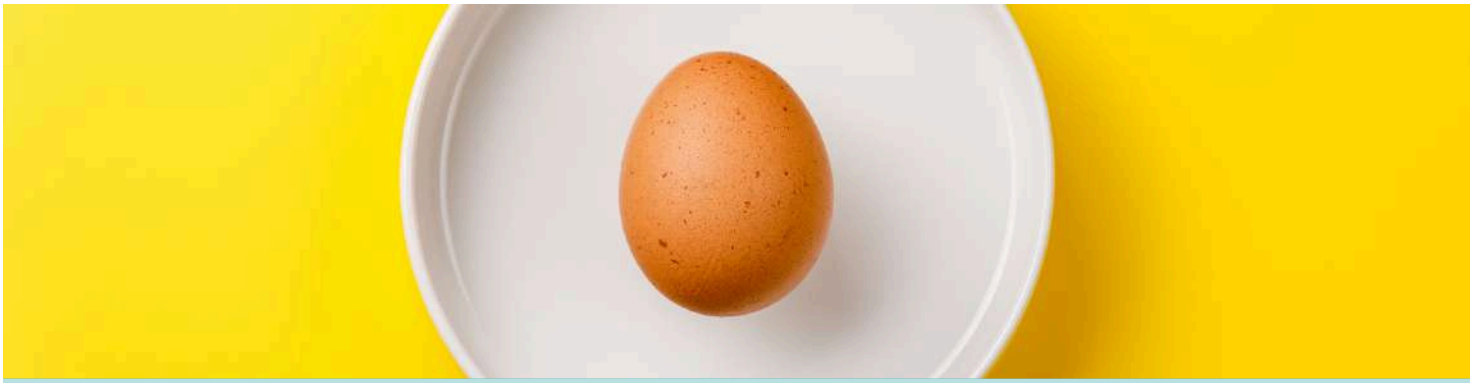
CONCLUSION

- A 3-week KD improved glycaemic control, however in this small study, there were conflicting effects on insulin sensitivity in various organ systems.
- A positive association was observed between muscle insulin sensitivity and KD, however no improvements were found in hepatic insulin sensitivity and a worsening of adipose tissue insulin sensitivity.

EXPERT REVIEWER Chloe Steele

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

Asian Low-Carbohydrate Diet with Increased Whole Egg Consumption Improves Metabolic Outcomes in Metabolic Syndrome: A 52-Week Intervention Study



BONGGOCHPASS PINSAWAS, APINYA SURAWIT, PICHANUN MONGKOLSUCHARITKUL 1, JOURNAL: THE JOURNAL OF NUTRITION 2024;154(11):3331-3

INTRODUCTION

This study aimed to evaluate the effects of a balanced low-calorie diet (BLC) compared to an Asian ketogenic diet (AKD) on metabolic markers on individuals with metabolic syndrome (MetS).

METHOD

This was a 52-week, open-label, randomised controlled trial. Participants (n=76) were randomly assigned to 3 groups: 1) BLC, 2) egg white Asian KD (White-AKD), and 3) egg yolk Asian KD (Yolk-AKD).

RESULTS

- Both AKD groups had significant within-condition reductions in waist circumference (WC) ($P < 0.05$), with no differences between them. At week 12, reductions were -2.1 cm (BLC) (95% CI: 6.7, 2.5 cm), -3.0 cm (White-AKD) (95% CI: 7.2, 1.1 cm), and -4.0 cm (Yolk-AKD) (95% CI: 8.0, 0.1 cm).
- At week 35, the White-AKD group showed a significant reduction in systolic blood pressure (BP) from 131.1 ± 3.0 mmHg at baseline to 121.9 ± 2.2 mmHg, compared to BLC (132.2 ± 2.4 mmHg) and Yolk-AKD (126.3 ± 2.1 mmHg) ($P < 0.05$). Diastolic BP also decreased significantly in the White-AKD group from 86.8 ± 1.8 mmHg at baseline to 82.3 ± 1.2 mmHg at week 52, with differences observed relative to both BLC (91.7 ± 1.3 mmHg) and Yolk-AKD (87.6 ± 1.1 mmHg) ($P < 0.05$).
- After 1 year, body weight decreased significantly for both AKD groups ($P < 0.05$): Yolk-AKD - 4.1 kg (95% CI: 6.8, 4.4 kg) and White-AKD - 4.0 kg (95% CI: 9.4, 1.5 kg) compared to BLC - 0.4 kg (95% CI: 9.0, 1.5 kg).
- At 1 year, White-AKD showed higher glucose (90 minutes) and lower insulin (60 minutes) whereas, Yolk-AKD had lower glucose (120 minutes), higher insulin (30 minutes), and lower insulin (60 and 120 minutes) compared to BLC ($P < 0.05$) during a glucose tolerance test.
- In both AKD, total and low-density lipoprotein (LDL) cholesterol increased ($P < 0.05$) whereas BLC did not show significant changes ($P < 0.05$). Triglycerides decreased by −30.5 ± 12.4 mg/dL in White-AKD (wk 35) and by −33.3 ± 13.3 mg/dL in Yolk-AKD (wk 12). High-density lipoprotein (HDL) increased by 8.5 ± 2.8 mg/dLin White-AKD (wks 35–52) and by 4.8 ± 2.2 mg/dL in Yolk-AKD (wk 52) ($P < 0.05$).
- Aspartate aminotransferase (AST) and alanine aminotransferase (ALT) levels decreased in BLC and Yolk-AKD at week 35 compared to baseline (AST BLC: mean change -4.2 U/L, 95% CI [-6.8, -1.6], $P = 0.003$; AST Yolk-AKD: -5.1 U/L, 95% CI [-7.5, -2.7], $P = 0.001$; ALT BLC: -6.3 U/L, 95% CI [-9.4, -3.2], $P = 0.002$; ALT Yolk-AKD: -7.5 U/L, 95% CI [-10.1, -4.9], $P = 0.001$). While ALT increased from week 35 to 52 in the BLC group (mean change +1.8 U/L, 95% CI [-0.5, +4.1]), this change was not statistically significant ($P = 0.12$). In contrast, the White-AKD group showed a significant within-group reduction in ALT at week 52 (mean change -5.6 U/L, 95% CI [-8.9, -2.3], $P < 0.01$).
- BLC showed increased levels of interleukin-6 (IL-6 wk 35). Both AKD groups had reductions in hormones associated with insulin sensitivity and appetite including C-peptide (wks 12-35) and leptin ($P < 0.05$); the Yolk-AKD also showed decreases in inflammation-related hormones IL-6, PYY, and tumour necrosis factor-α (wk 35) ($P < 0.05$).
- Simple Method for Quantifying MetS (siMS) score decreased in all groups after week 12 ($P < 0.05$).

HOW TO BRING THIS INTO YOUR CLINICAL PRACTICE

TAKE HOME MESSAGE

- An AKD that includes whole eggs may positively influence metabolic health.
- AKDs are associated with improved metabolic markers in individuals with MetS including reductions in triglycerides, BP, and blood glucose. While total cholesterol increased, this was accompanied by favourable shifts in lipid subtypes such as increased HDL cholesterol and decreased triglycerides.
- Long-term adherence and safety of ketogenic dietary approaches may be influenced by individual and lifestyle factors.

Read the article here

CLINICAL PRACTICE APPLICATIONS

- AKD interventions, with or without eggs, may contribute to reductions in body weight and WC. Individuals with MetS may experience more pronounced improvements in their sIMS scores, exceeding the benefits typically observed from simply consuming a balanced low-calorie diet.
- Egg yolk-enriched AKDs could help improve postprandial glucose and insulin responses as well as have a positive impact on inflammatory markers.
- AKD-based interventions may increase LDL and total cholesterol levels. Thus, regular lipid profile monitoring should be part of the intervention.
- AKD-based interventions may support liver function in individuals with MetS.

CONSIDERATIONS FOR FUTURE RESEARCH

- Further research is needed to examine possible negative outcomes associated with maintaining an AKD over time. It is important to identify and understand financial limitations, cultural influences, and practical obstacles that may prevent sustained adherence to an AKD.
- It is unclear to what extent individuals with MetS are able to follow an AKD over a long period of time. Investigating variables that affect adherence such as cultural eating habits and lifestyle factors may help improve long-term compliance.
- Given that AKDs have been associated with rises in LDL and total cholesterol, it is important for future studies to examine their long-term impact on cardiovascular health across broader and more diverse populations.

CONCLUSION

- Both AKD interventions were more effective than BLC for promoting weight loss, sustaining WC reduction, lowering sIMS scores and improving metabolic outcomes, blood lipid profiles and liver function in individuals with MetS. By emphasising a low-saturated fat diet while not restricting dietary cholesterol, this approach shows promise for the management of MetS and obesity.

EXPERT REVIEWER Sarah Cassar

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

Replacing Dietary Carbohydrate with Protein and Fat improves Lipoprotein Subclass Profile and Liver Fat in Type 2 Diabetes independent of Body Weight: evidence from 2 RCTs

MADS N THOMSEN, MADS J SKYTTE, AMIRSALAR SAMKANI ET AL.
JOURNAL: THE AMERICAN JOURNAL OF CLINICAL NUTRITION 2025;121(2):224-231

TAKE HOME MESSAGE

- A reduced carbohydrate/high protein diet (30% CHO, 30% protein) may benefit patients with T2DM with improved lipid profiles and liver health.

Read the article here

INTRODUCTION

The aim was to evaluate the effect of a 6-week carbohydrate-reduced high-protein diet (CRHP, 30% carbohydrates, 30% protein, 40% fat) on lipid profiles, with an emphasis of lipoprotein density subclasses, in patients with type 2 diabetes mellitus (T2DM) compared to a conventional diabetes diet (CD, 50%, 17% and 33%, respectively).

METHOD

- Secondary analysis of two open-label, randomised controlled trials (RCT) in adults with T2DM:
- 1) isoenergetic diet crossover RCT in 30 participants who had a CRHP diet and a CD diet.
- 2) hypocaloric diet parallel RCT of 72 participants split into two groups, where one group followed the CRHP diet the other followed the CD diet. The weight loss goal was 6%.
- In both studies, all meals were provided to participants.
- Outcome measures included: triacylglycerol (TAC), total cholesterol, HDL and LDL cholesterol and density profiles of lipoproteins.

RESULTS

- There was no difference between groups in weight maintenance or weight loss in either study.
- In the isocaloric study, patients on CRHP improved significantly more than the CD group in various subclasses of lipoproteins : TAG-rich lipoproteins (TRL, mean -33%, 95% CI (-48%, -14%), LDL5 (-16%; (-26%, -4%)), HDL3 particles (-8%; (-14%, -0.7%)), HDL2/HDL3 ratio (10%; (0%, 22%)), and LDL1 (-20%; (-35%, -2%)).
- In the hypocaloric study, patients on the CRHP improved significantly more in LDL5 (-13%; (-22%, -3%) and HDL2/HDL3 ratio (11%; (0.7%, 22%)), with a trend to reduced TRL (-16%; (-30%, 1%).
- Intrahepatic TAG (IHTG) content reduced significantly more in the CRHP group than the CD group in both studies (-55%; (-74%, -22%) in the isocaloric study; -26% (-45%, 0%) in the hypocaloric study). Changes in IHTG correlated with changes in lipoprotein subclasses.
- In both studies, glycaemic and lipid markers improved significantly more in the CRHP compared to the CD group.

HOW TO BRING THIS INTO YOUR CLINICAL PRACTICE

CLINICAL PRACTICE APPLICATIONS

- A reduced carbohydrate/high protein diet (30% CHO, 30% protein) could be considered for patients with T2DM to improve lipid profiles and liver health.

CONSIDERATIONS FOR FUTURE RESEARCH

- Longer-term studies in a real-life situation (meals not provided) are needed to confirm the long-term safety and efficacy of as well as compliance with a reduced carbohydrate diet.

CONCLUSION

A CRHP diet may improve metabolic dysfunction–associated steatotic liver disease and dyslipidaemia in patients with T2DM independent of weight loss, resulting in reduced atherogenicity.

EXPERT REVIEWER Karin Elgar PhD

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

Ketogenic Diet but not Free-sugar Restriction alters Glucose Tolerance, Lipid Metabolism, Peripheral Tissue Phenotype, and Gut Microbiome: RCT

AARON HENGIST, RUSSELL G DAVIES, JEAN-PHILIPPE WALHIN, ET AL.
JOURNAL: CELL REPORTS. MEDICINE 2024;5(8):101667

INTRODUCTION

A randomised controlled trial was conducted to evaluate the effect of restricted dietary sugars (LOWSUG), or restricted carbohydrates (Ketogenic diet; LOWCHO), on free-living physical activity energy expenditure (PAEE) over 12 weeks in healthy adults. These two diets were each compared to a control diet with moderate sugar and carbohydrates (MODSUG).

METHOD

- Fifty-three participants were randomly assigned to one of the three dietary groups - LOWSUG, LOWCHO or MODSUG - for 12 weeks. Circulating biochemical profiles, diurnal glycemia, and muscle glycogen concentrations were assessed at baseline, 4 and 12 weeks.
- Participants in the LOWSUG group consumed less than 5% of their total energy intake from sugars, while those in the LOWCHO group consumed less than 8% of their energy from carbohydrates. Of the 60 participants initially enrolled, 53 (88%) completed the trial.

RESULTS

Impact of LOWSUG

- Body mass was significantly reduced at both weeks 4 and 12 compared to the MODSUG group (p= 0.05; p=0.04, respectively).
- Total cholesterol was lowered at week 12 in the LOWSUG group compared to MODSUG (p= 0.001).
- By week 12, there was an 18% reduction in Akt (protein kinase B) compared to MODSUG (p=0.02), which coincided with increased glycogen concentrations at both weeks 4 and 12 (p=0.03; p= 0.004, respectively).
- LOWCHO altered gut microbiota beta diversity (p=0.04) and reduced the abundances of Bifidobacterium at weeks 4 and 12 (p=0.04;p= 0.01), while also changing metabolic pathway abundances at weeks 4 and 12 (p=0.02;p=0.04), and decreasing serum propionate at week 4 (p=0.03), with no changes in gut permeability markers or overall microbiome alpha diversity.

Impact of LOWCHO

- Body mass was significantly reduced at both weeks 4 and 12 compared to the MODSUG group (p< 0.001 for both time points).
- Increases were observed in low-density lipoproteins (LDLs), and very-low-density lipoproteins VLDLs, along with a reduction in total lipid high-density lipoprotein (HDL) particles (all p<0.05 vs. MODSUG).
- The ketogenic diet increased apolipoprotein B (apoB) concentrations (p=0.006) and fasting triacylglycerol concentrations at week 4 (p = 0.04) compared to MODSUG.
- Respiratory expenditure rate (RER) was significantly reduced at weeks 4 and 12 in the fasted state (p=0.004; p=0.04), postprandial state (p =0.005; p=0.01), and during exercise (p< 0.001; p= 0.001) compared to MODSUG.
- Skeletal muscle pyruvate dehydrogenase kinase 4 (PDK4) protein levels were elevated at week 4 (p= 0.04), while insulin receptor (INSR), adenosine monophosphate-activated protein kinase (AMPK), glucose transporter 4 (GLUT4), and perilipin 1 (PLIN) were decreased at week 12 (p= 0.006, p =0.02, p=0.03, and p=0.02, respectively), compared to MODSUG.
- Gut microbial beta diversity was altered, with a significant reduction in the abundance of bifidobacterium observed at both weeks 4 and 12 (p=0.04).

HOW TO BRING THIS INTO YOUR CLINICAL PRACTICE

TAKE HOME MESSAGE

- Neither sugar nor carbohydrate-restricted diets were found to significantly affect energy expenditure or physical activity.
- Both 12-week sugar and carbohydrate-restricted diets resulted in reductions in body mass and cholesterol.
- Ketogenic carbohydrate restriction may not deliver the expected cardiometabolic health benefits typically associated with weight loss. Instead, reducing free sugar intake could be a more effective dietary approach for improving overall cardiometabolic health in many individuals.
- A carbohydrate-restricted diet providing less than 8% of total energy intake negatively influenced glucose tolerance, lipid metabolism, peripheral tissue phenotype, and gut microbiome composition.

Read the article here

CLINICAL PRACTICE APPLICATIONS

- This study found that both sugar and carbohydrate-restricted diets reduce body mass and cholesterol levels – an important consideration for targeted strategies by clinicians.
- A ketogenic diet, restricting carbohydrates to below 8% of energy intake, affected glucose tolerance, increased apolipoprotein levels and reduced bifidobacterium abundance. As such, clinicians should be aware that a ketogenic diet may not be appropriate for all clients. Monitoring lipid panels, glucose intolerance and gastrointestinal symptoms may therefore be useful.

CONSIDERATIONS FOR FUTURE RESEARCH

- This study demonstrated that carbohydrate restriction can positively influence certain cardiometabolic markers. However, further research is warranted to explore the effects of ketogenic diets in populations with prediabetes, metabolic syndrome and cardiovascular disease.
- Carbohydrate restriction was associated with reduced GLUT4, AMPK, and insulin receptors. Consequently, additional research is needed to examine how ketogenic diets may influence exercise-induced adaptations, particularly those related to insulin sensitivity and metabolic flexibility.

CONCLUSION

- This RCT demonstrated that sugar restriction led to weight loss, improved cholesterol profiles, and beneficial metabolic changes, including reduced Akt levels and increased glycogen storage. In contrast, carbohydrate restriction also reduced weight but negatively impacted lipid markers, metabolic proteins, and gut microbiota composition.

EXPERT REVIEWER Ana-Paula Agrela

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

Effect of Weight-maintaining Ketogenic Diet on Glycemic Control and Insulin Sensitivity in Obese T2D subjects.

AURORA MEROVCI, ANDREA HANSIS-DIARTE, EUGENIO CERSOSIMO, ET AL.
JOURNAL: BMJ OPEN DIABETES RESEARCH & CARE 2024;12(5):

INTRODUCTION

Obesity and type 2 diabetes (T2D) are related comorbidities. Recently hypocaloric, low carbohydrate, ketogenic diets have been shown to promote weight loss and improve glycaemic control in individuals with T2D. However, it is unclear as to whether improved glucose control seen with the ketogenic diet is independent of weight loss. This study aimed to determine the effect of the ketogenic diet on glucose metabolism in individuals with T2D in the absence of weight loss.

METHOD

- 29 overweight/obese subjects with T2D were given either 1) standard diet (SD), 2) low carbohydrate, ketogenic diet (KD) or 3) low carbohydrate, ketogenic diet with keto ester supplementation (KD+) for 10 days. All diets were designed to be weight-maintaining.
- All meals were made by a certified dietitian and participants were only allowed to eat the prepared food and drink water.

RESULTS

- Body weight, fat content and body fat percentage did not change significantly in any of the groups.
- The KD and KD+ diets were shown to induce metabolic changes, with carbohydrate oxidation decreasing (KD p=0.019; KD+ p=0.003) and fat oxidation increasing (KD p=0.020; KD+ p=0.002) whereas the standard diet did not.
- Fasting plasma glucose modestly declined in all three groups but failed to reach significance (SD p=0.465; KD p=0.525; KD+ p=0.137).
- HbA1c, insulin resistance, muscle insulin sensitivity, insulin clearance rate, diastolic blood pressure, heart rate, total cholesterol, LDL cholesterol, HDL cholesterol, and triglycerides were unchanged in all groups.

HOW TO BRING THIS INTO YOUR CLINICAL PRACTICE

- In the absence of weight loss, a ketogenic diet did not improve glucose metabolism in individuals with obesity and T2D in the absence of weight loss.
- However, it is unclear whether other diet types may have similar outcomes.

Read the article here

CLINICAL PRACTICE APPLICATIONS

- Weight loss is important for improving blood sugar levels in individuals with obesity and T2D.
- In the absence of weight loss, a ketogenic diet may have very little benefit for individuals who are trying to improve blood glucose control.

CONSIDERATIONS FOR FUTURE RESEARCH

- Future research could analyse other diets in the absence of weight loss on blood glucose levels. This would aid understanding of the level of importance of weight loss compared to diet type on blood glucose control.

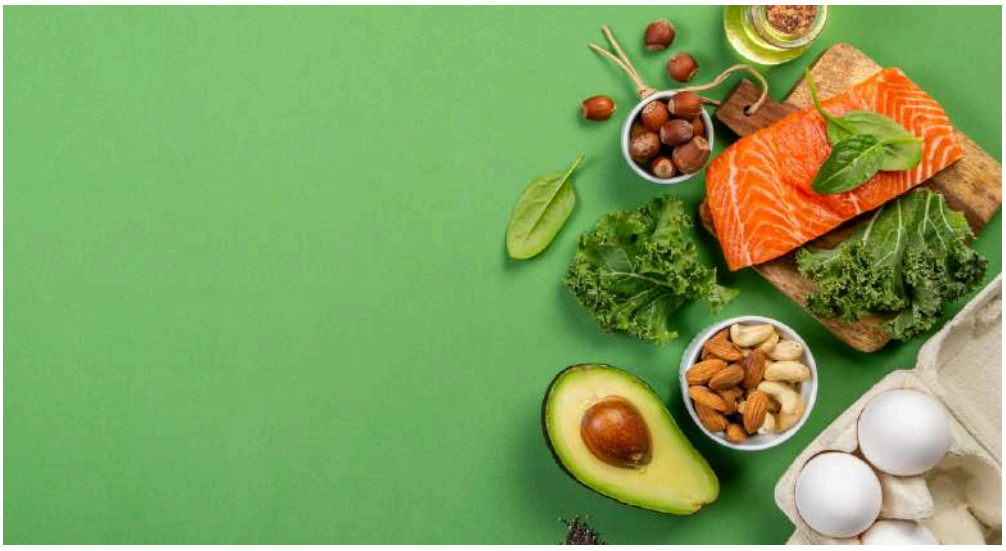
CONCLUSION

A weight maintaining 10-day ketogenic diet with or without ketone supplementation did not improve glucose control, insulin sensitivity, blood lipids, or blood pressure in individuals with obesity and T2D.

EXPERT REVIEWER Chloe Steele

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

Effect of Ketogenic Diets on Insulin-like Growth Factor (IGF)-1 in humans: A systematic review and meta-analysis.



TIAN WANG, LUIGI FONTANA, GAYATHIRI RAJAKUMAR, ET AL.
JOURNAL: AGEING RESEARCH REVIEWS 2024;102():102531

TAKE HOME MESSAGE

- KD's alone without moderate to severe energy restriction may be sufficient to reduce IGF-1/insulin levels offering a more sustainable and safer alternative for both preventative and adjunct therapy alongside other medical interventions for age-related health conditions including cancer and dementia.

Read the article here

INTRODUCTION

- Excessive nutrient intake may stimulate Insulin Growth factor (IGF)-1 through different signalling pathways, whereas moderate food reduction including fasting may lower IGF-1, which has been correlated with the probability of decreased cognitive impairment and mortality, and a reduced cancer risk.
- A more sustainable and safer strategy to fasting may be achieved by adapting macronutrient ratios of high fat (>55%), moderate protein and low carbohydrate (CHO) (<50g/day), known as a ketogenic diet (KD).
- This Systematic Review and Meta-analysis aimed to evaluate ad libitum KD with at least 65% fat and <50g/day CHO.

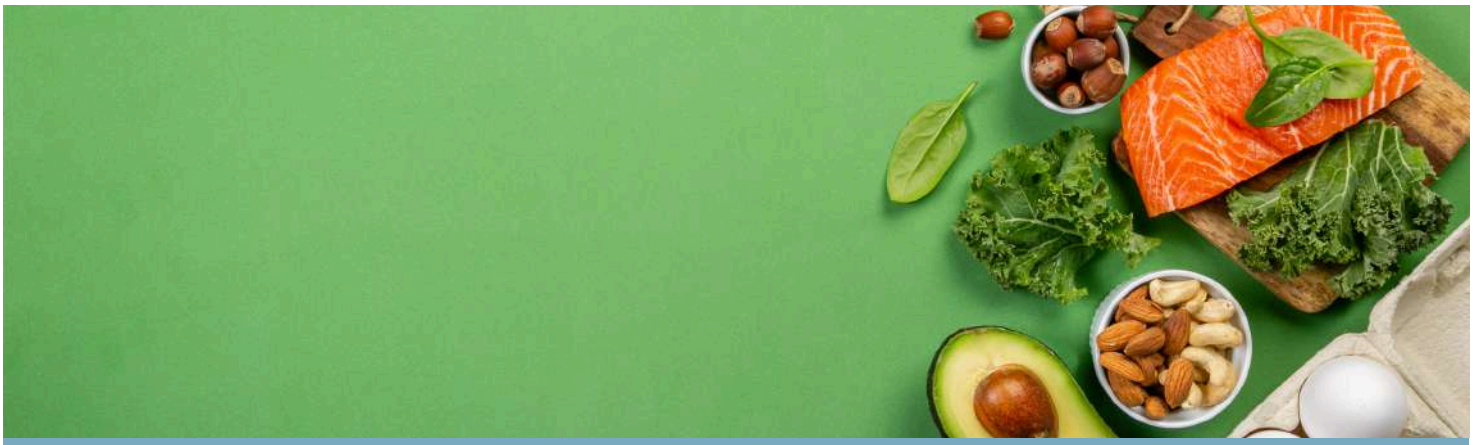
METHOD

- Article selection for inclusion and exclusion used PICO; the review followed PRISMA guidelines and was registered on PROSPERO.
- Literature searching resulted in 11 interventional studies using non-randomised and randomised clinical trials published between 2012-2023, which measured circulating IGF-1, glucose and insulin levels. Control conditions mainly used no intervention other than a standard or Western diet.
- Meta-analysis used a random-effect model to account for demographic and clinical heterogeneity. Sub-group analysis included cancer, sex, randomisation and dietary duration.
- 522 male and female individuals, mean age 50.5 ± 10.9,with 262 individuals were selected for the KD intervention and 236 completed KD's of between 1 and 20 weeks.
- Bias was rated low to moderate in the IGF-1 measurements.

RESULTS

- Serum IGF-1: pooled results from 10 studies observed significant reductions post-intervention (MD: 24.9 ng/mL (95 % CI, − 31.7 to − 18.1) p<0.0001), an average 19.7 % reduction across all participants.
- Fasting Glucose: Nine out of ten studies were associated with post-intervention reductions (MD: 7.3 mg/dL (95 % CI, − 11.62 to − 2.98) p=0.0009) with six studies showing statistically significance. The overall reductions were 6%.
- Insulin levels: Nine studies showed a reduction (MD − 2.57 mU/L, (95 % CI − 4.41 to − 0.74), p=0.006) indicating an average decrease of 29 %.
- None of the sub-group analysis showed any significant differences in reduction of serum IGF-1, blood glucose or insulin.

HOW TO BRING THIS INTO YOUR CLINICAL PRACTICE



CLINICAL PRACTICE APPLICATIONS

- Since high IGF-1 levels are associated with many age-related conditions including cancer, cardiovascular disease, diabetes, dementia and mortality, short term KD's may be considered an efficacious strategy for modulating aging and offering other metabolic effects including reduced insulin and fasting glucose.
- Short term KD's may offer a safer and more sustainable dietary strategy for older adults than prolonged fasting or moderate to severe calorie restriction, highlighting a potential option for reducing cancer risk and other age-related conditions through the IGF-1 pathway.

CONSIDERATIONS FOR FUTURE RESEARCH

- Further research into different populations is needed to contribute to what is already known about the impact of different durations of ad libitum KD's on IGF-1, blood glucose and insulin levels.
- Further research is needed with standardised KD interventions and macronutrient content.

CONCLUSION

- An ad libitum KD dietary strategy with >55% fat induces ketosis and can lower serum levels of IGF-1, blood glucose and insulin without fasting or moderate/severe calorific reduction irrespective of sex, cancer type and dietary duration.

EXPERT REVIEWER Miranda Harris

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

MACRONUTRIENT CLINICAL GUIDES

MACRONUTRIENT RESOURCES

BANT has developed a dedicated range of resources to complement the personalised nutrition and lifestyle advice given by practitioners in a clinical setting. These resources are open access on our website bant.org.uk and aid further comprehension of nutrition science and clinical interventions.

CLIENT-FRIENDLY GUIDES:

Providing practitioners with health resources to support their clinical recommendations.

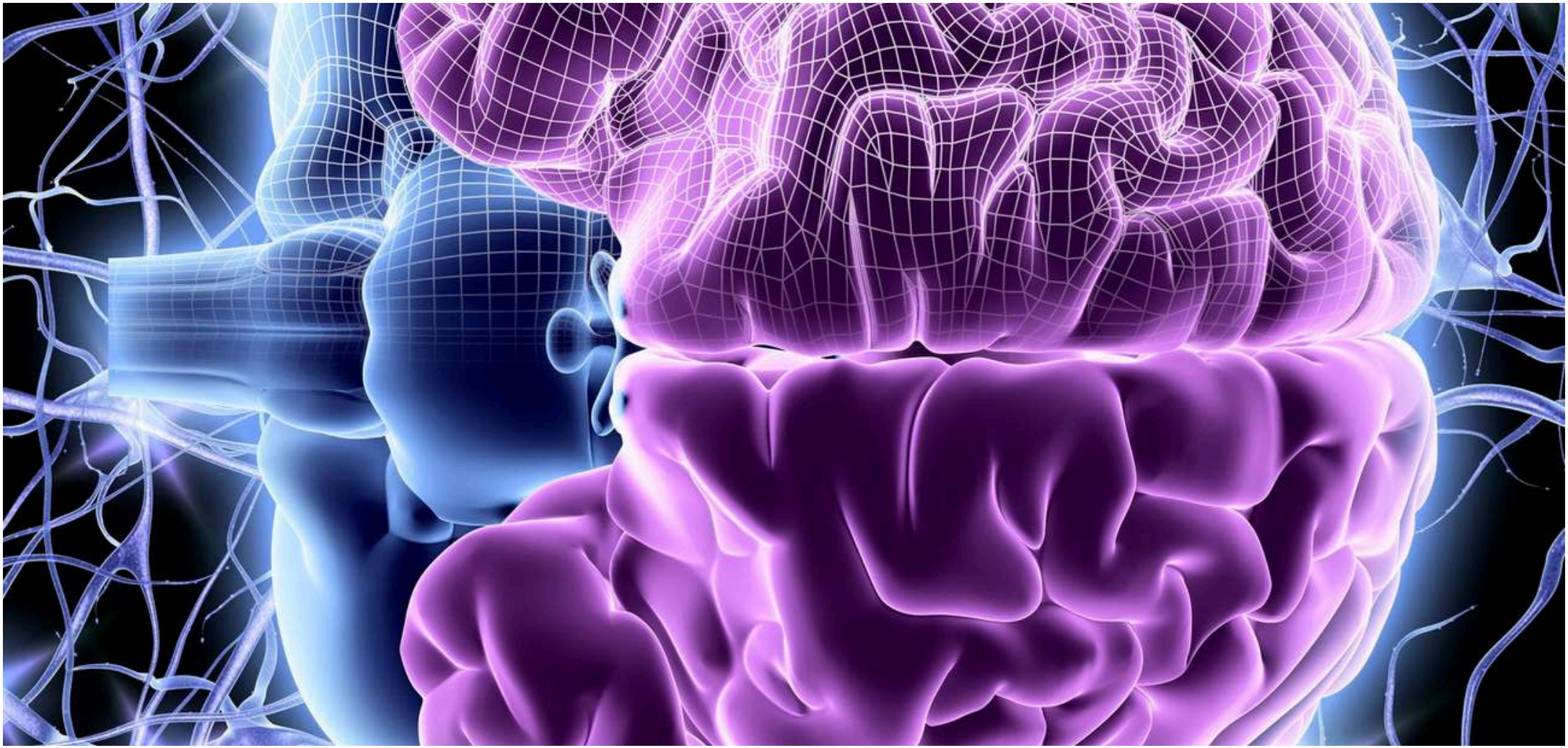
Access further resources here

DR KATE LAWRENCE ET AL.

FEATURE ARTICLE

NEURODIVERGENCE:

EVIDENCE-BASED CONSIDERATIONS FOR NUTRITIONAL THERAPY AND PERSONALISED LIFESTYLE SUPPORT



NEURODIVERGENCE:

EVIDENCE-BASED CONSIDERATIONS FOR NUTRITIONAL THERAPY AND PERSONALISED LIFESTYLE SUPPORT

Authors: Kate Lawrence, Gail Brady, Chloe Steele, Miranda Harris, Sarah Cassar, Wilma Kirsten, Kirsty Baxter, Michelle Barrow

ABOUT NEURODIVERGENCE

ABSTRACT

Neurodivergent individuals (including autistic individuals and those with ADHD) can sometimes present with gastrointestinal symptoms, nutritional deficiencies and selective eating. Dietary and lifestyle factors may play a role in supporting these individuals. Additionally, various macro and micronutrients, including essential fatty acids, key minerals such as iron, magnesium, and zinc, and vitamins including A, D, and B-complex may be deficient in certain individuals.

Emerging evidence suggests that supplementation, alongside dietary and lifestyle strategies, may support symptom management and wellbeing. Optimising gut health and microbiome balance, alongside personalised lifestyle strategies, may further enhance support. This narrative review summarises current evidence on the role of nutrition in supporting neurodivergent individuals, with the aim of guiding evidence-based therapeutic decision-making in clinical practice.

INTRODUCTION

Neurodivergence refers to a neurocognitive profile that diverges from the majority (1) and encompasses conditions traditionally classified as neurodevelopmental disorders such as attention deficit hyperactivity disorder (ADHD), autism, dyslexia, and developmental coordination disorder (DCD). Globally, prevalence estimates are approximately 1% for autism (3), 6% for ADHD (4), 7% for dyslexia (5), and 5% for DCD (6), indicating that neurodivergence affects a substantial proportion of the population. While these conditions are heterogeneous in presentation, impact, and intervention requirements, they nonetheless demonstrate considerable overlap, frequent co-occurrence, and shared traits (6,7). This supports the value of a broad, integrative approach, particularly in exploring the potential role of nutritional and lifestyle interventions. Terminology used throughout this paper is non-pathologising and non-stigmatising, aligning with recommendations from Dwyer et al. (8) to adopt identity first language (i.e. autistic person) in relation to autism.

Gastrointestinal problems are common in autistic individuals (9), with estimates ranging widely from 9-91% (10,11). Evidence also suggests a higher prevalence of gastrointestinal issues in individuals with ADHD (12) while research on this topic in DCD and dyslexia remains limited. Furthermore, evidence is gathering that the gut microbiota of autistic individuals and those with ADHD may differ to that of typically developing individuals (13–15), suggesting a potential role for the gut/brain axis in these conditions. Nutritional deficiencies have also been reported, across these conditions, in nutrients such as iron and omega-3 fatty acids which play a key role in brain health and development (16,17). Addressing gastrointestinal symptoms and nutritional deficiencies in neurodivergent individuals may help enhance quality of life and wellbeing.

NICE guidelines (17,18) for neurodivergent individuals encourage early-interventions and holistic family-centred care including psychosocial interventions, behavioural therapies, educational support and pharmaceutical interventions with some recognition of the role of healthy dietary choices and individual sensitivities for those with ADHD. Families and individuals often seek integrative ways to support themselves and their children, with the use of nutritional therapy and lifestyle support being increasingly utilised.

It is of critical importance that such interventions are guided by a strong scientific evidence base. Nutritional programmes must be 'personalised and tailored to an individual patient' (18) and it is essential that practitioners have access to rigorous, up-to-date scientific reviews to inform safe and effective care. This narrative review provides an overview of the current evidence for nutritional and lifestyle support and interventions, aimed at supporting nutrition practitioners in making recommendations for neurodivergent clients.

DIETARY INTERVENTIONS

WHAT ARE THE POTENTIAL BENEFITS OF DIETARY INTERVENTIONS FOR NEURODIVERGENT INDIVIDUALS?

Neurodivergent children, particularly those who are autistic or diagnosed with ADHD, may exhibit atypical responses to dietary components such as gluten (found in wheat, barley, oats, and rye), casein (from mammalian dairy), and soy (19).

Adams and colleagues (2018) report higher rates of food hypersensitivity in autistic children compared to neurotypical peers, with gastrointestinal issues, deficiencies in digestive enzymes, limited diets, and food sensitivities significantly correlating with autism severity ($r = 0.59$, $p < .001$) (20). Although some studies suggest an association between ADHD and coeliac disease, findings remain inconsistent (21).

Evidence for the effectiveness of elimination diets remains mixed. A six-week gluten-free (GF) dietary intervention in Iran, involving 76 autistic children, resulted in significant reductions in gastrointestinal symptoms and stereotyped behaviours, as measured by the Gilliam Autism Rating Scale–Second Edition (80.03 ± 14.07 vs. 75.82 ± 15.37; $p < .05$) (22). However, seven other studies of gluten- and casein-free (GFCF) diets in children aged 2–12 years reported no statistically significant effects (23–29). Some research has nevertheless reported improvements in language development, hyperactivity, tantrums (30), and core autistic traits (31). Hafid & Ahami (2018) suggested that positive responses may be limited to children with both elevated urinary peptide levels and gastrointestinal issues. It may therefore be important to identify subgroups most likely to benefit (25). Additionally, GFCF diets have been associated with adverse gastrointestinal effects (31).

The Few Foods Diet (FFD), or oligoantigenic diet, eliminates commonly consumed, potentially reactive foods (e.g., gluten, dairy, citrus), and allows only a limited selection of tolerated foods. Designed primarily as a diagnostic tool rather than a long-term treatment, the FFD has shown potential in small-scale trials. A five-week Randomised Controlled Trial (RCT) involving 24 children aged three–eight years reported a significantly greater reduction in physical and sleep complaints in

the diet group (77%) compared to controls (17%) (p = 0.001)(32). Similarly, an oligoantigenic intervention in 16 children aged 7–13 years reported significant reductions in Autism Rating Scale scores following a four-week elimination phase (33). A meta-analysis of five RCTs concluded that the FFD significantly reduced ADHD symptoms, with larger effect sizes than other dietary interventions (34). However, the FFD is highly restrictive, difficult to sustain, and its mechanisms remain poorly understood. Less restrictive dietary strategies designed to target the gut microbiome may offer a more feasible and sustainable long-term approach (35).

Ketogenic (KT) diets have also shown promise, with a three-month modified GF KT diet (high-fat, appropriate-protein, and low-carbohydrate), supplemented with medium-chain triglyceride oil, improving core autistic characteristics in 15 children aged 2–17 years (36), while a six-month KT pilot study involving 30 children aged 4-10 years reported significant improvements in the Childhood Autism Rating Scale, of 4.77 (SE 0.89) p < .001) (37). Despite promising results, findings for dietary interventions remain heterogeneous and larger, well-controlled trials are needed to determine their therapeutic potential and identify which individuals are most likely to benefit.

Whilst evidence reports behavioural and gastrointestinal symptoms may be alleviated through the removal of specific dietary components, practitioners are reminded of other regimes, which may already be familiar in practice, such as the Mediterranean, low FODMAP and Glycaemic Load Diets (38) as well as the introduction of fermented foods (39) and foods rich in polyphenols. Personalised use of such dietary approaches may be helpful for symptom management and wellbeing for neurodivergent individuals.

NUTRITIONAL SUPPORT

HOW CAN WE SUPPORT NEURODIVERGENT INDIVIDUALS WITH RESTRICTED DIETS AND...

...AVOIDANT/RESTRICTIVE FOOD INTAKE DISORDER (ARFID)?

Avoidant/Restrictive Food Intake Disorder (ARFID) is a feeding and eating disorder that significantly affects nutritional status, weight, and psychosocial functioning (40–44). In contrast to other eating disorders, individuals with ARFID are not concerned about their body shape or weight (41), but their food avoidance typically stems from three primary factors: lack of interest in eating, sensory sensitivity to the characteristics of food and fear of aversive consequences such as vomiting or choking (40–42,45).

Autism and ARFID demonstrate a notable coexistence as autistic individuals frequently display behavioural and cognitive patterns including restrictive eating habits resulting from sensory sensitivities (41,42,45). Mealtime behaviour and eating patterns for neurodivergent individuals are deeply affected by how their sensory processing alters their perception of smell, taste and texture (46,47). These sensory challenges often lead to food selectivity, neophobia, and increased distress during mealtimes which can limit dietary variety and nutritional intake (Nimbley et al., 2022; Cobbaert et al., 2024).

When food selectivity is present, neurodivergent individuals may benefit from adapted support aimed at enhancing their intrinsic motivation and sensory functioning, as well as optimising the physical and social environment, to make their mealtimes more enjoyable (47). A tailored sensory intervention, based on a thorough sensory assessment, may be an effective way to support atypical eating behaviours in neurodivergent individuals (46).

Recent intervention studies, including those evaluating sensory-based therapies, have demonstrated positive outcomes in enhancing oral motor skills, increasing food acceptance, and decreasing distressed behaviours during mealtimes (47). Additionally, cognitive behavioural therapy for ARFID (CBT-AR), especially exposure-based approaches, has shown promising results in reducing food avoidance among neurodivergent children and adolescents by targeting core mechanisms such as sensory sensitivity and fear of aversive consequences (43,44,49).

While CBT-AR can help neurodivergent individuals increase food intake, their parents/caregivers' role is equally important. Their involvement during interventions helps them to better manage their child's behaviour at home, promoting continued progress and encouraging generalisation of learned abilities and the inclusion of new food (44). Interventions that are both sensory-based and family-centred appear to offer the most sustainable support in addressing restrictive eating and broadening food repertoires (50).

Increasing research supports the gut microbiota's involvement in food selectivity and gastrointestinal issues which frequently occur in neurodivergent individuals (51,52). Autistic children often experience gut dysbiosis which can lead to inflammation along with sensory sensitivities and restrictive eating habits (12). Prebiotic and probiotic supplements along with foods abundant in polyphenols and flavonoids demonstrate effective outcomes in inflammation reduction and oxidative stress protection while simultaneously promoting gut health, reducing gastrointestinal issues and enhancing behavioural outcomes (51). Thus, approaches that support the gut–brain axis may act as complementary measures to CBT and parent/caregiver-led interventions when addressing ARFID and related feeding problems (53).

A multidisciplinary approach that addresses ARFID as a multi-faceted eating disorder can produce effective support for food selectivity issues in autistic children (54). Healthcare professionals need to conduct combined evaluations of nutritional status and sensorimotor capabilities to devise tailored interventions which recognise both sensory preference drivers in children and their family environment. Structured desensitisation methods including gradual exposure techniques alongside texture fading and supportive mealtime environments, which are part of multicomponent strategies, may help improve family wellbeing together with nutritional outcomes.

WHAT IMPACT DO ARTIFICIAL ADDITIVES AND SUGAR HAVE ON NEURODIVERGENT INDIVIDUALS?

Research on the influence of food additives on neurodivergence dates to the 1970's when Feingold reported that hyperactive children consume larger than typical amounts of food additives (55). This led to the development of the Feingold diet, which excluded the consumption of salicylates (56). Since then, dietary exclusion of food additives for individuals with ADHD has become controversial. Whilst some research has shown positive outcomes with exclusion, others have not. One systematic review and meta-analysis of 54 randomised control trials (RCTs) reported that eight trials looked at an artificial food colour exclusion diet (AFC), which involved adoption of the Feingold diet, or the exclusion of tartrazine, or the exclusion of specific food colours (57). The results showed that positive treatment effects were seen on ADHD symptom severity with the AFC diet (SMD:0.32, 95% CI:0.06, 0.58, p = 0.02). However, more recently a systematic review of six meta-analyses of double-blind placebo-controlled trials looked at diets eliminating AFC, amongst others (58). It reported that although the exclusion of food colours did affect ADHD symptoms, the effect sizes were too small to warrant giving parents and caregivers a list of additives they should eliminate from their child's diet (effect size range 0.08 to 0.44). Yet they are large enough to warrant further research, and any effect may be enough for some parents or caregivers to consider eliminating food additives.

Whilst additive removal may have a small effect, added sugars in foods may have an impact on children. Sugar-sweetened beverage (SSB) consumption before the age of two-years has been shown in one cohort study, to be associated with an increased probability of ADHD development (59). The cohort included 25,305 children from Korea at 24 months of age, who had been exposed to high SSB's from juices and soft drinks (≥200 mL) and 339,931 who had not (<200 mL). Over a mean follow-up period of 9.2 years the incidence of ADHD was higher in those given SSB's compared to the reference group (29.6 and 23.8 per 10,000 person-years) and these individuals had an increased likelihood of developing ADHD (adjusted hazard ratio (HR): 1.17, 95% confidence interval [CI] 1.08–1.27).

This was sustained from the ages of two to three years (HR: 1.14 95% CI: 1.05–1.23). Amongst adults, it has also been shown that SSB's may exacerbate ADHD symptoms (60). This cross-sectional study of 441 medical students reported that daily added sugar intake from beverages higher than 25g/day had a 1.8 times higher probability of ADHD symptoms than students who reported lower consumption (adjusted odds ratio: 1.80, 95% CI: 1.15 to 2.84, p = 0.011). Although these data do not establish causality, they warrant further investigation. It is interesting to note that both trials included beverages that have added sugars and other additives and preservatives and a combination of these may be responsible for the heightened response.

Practitioners should be aware that the involvement of food additives in the development and exacerbation of neurodivergent symptoms may be small when excluded in isolation. Foods containing additives are often also high in added sugars and when combined may have a cumulative effect on ADHD development and symptoms. Instead of focusing on specific additives, eliminating or reducing ultra-processed foods may support the reduction of additives and sugar for adults and children with ADHD.

WHAT DOES THE EVIDENCE SAY ABOUT THE ROLE OF PROBIOTICS AND PREBIOTICS IN SUPPORTING NEURODIVERGENT INDIVIDUALS?

The gut-brain axis has been cited as a potential pathway through which the gut can communicate with the brain. Dysbiosis of the gut microbiota has been seen in autistic children and those with attention deficit hyperactivity disorder (ADHD) and may be associated with the development of neurodivergence. One systematic review and meta-analysis of 18 studies including 493 autistic children and 404 controls showed that autistic children have a microbiota composition dominated by species in the Bacteroidetes, Firmicutes, and Actinobacteria phyla compared to controls ($p = 0.002$, $p < 0.001$, and $p < 0.001$ respectively) (61). This was reflected in higher abundances of Bacteroides ($p < 0.001$), Parabacteroides ($p < 0.001$), Clostridium ($p < 0.01$), Faecalibacterium ($p = 0.04$), and Phascolarctobacterium ($p = 0.02$), and a lower abundance of Coprococcus ($p = 0.004$) and Bifidobacterium ($p < 0.001$). Children with ADHD have shown similar gut dysbiosis to autistic children. In one randomised control trial (RCT) of 75 infants who were followed until adolescence, it was shown that there was a lower abundance of Bifidobacterium species in the first 6 months of life of children who were later in life diagnosed with ADHD (8.26 (1.24) log cells/g than in neurotypical children (9.12 (0.64) log cells/g; $p = 0.03$) (62).

Whilst gut dysbiosis may be present in those with ADHD and autistic children, the use of probiotics to support autistic individuals may be dependent upon whether they are single or multi-strained. One systematic review and meta-analysis of six RCTs with 302 children showed that the use of probiotics had no effect on autistic behaviours as measured by the Aberrant Behaviour Checklist and the Child Behaviour Checklist (63). However, it did improve gastrointestinal symptoms (GI) (MD=-0.59, 95%CI [-1.02,-0.17], $p = 0.006$). Similar results were seen in a second systematic review and meta-analysis of 10 child studies, with no effect of probiotic supplementation on autistic behaviours. However, this study reported that unlike single-strain

probiotics, multi-strain probiotics may reduce autistic behaviours (pooled SMD: −0.42, 95% CI: −0.83 to −0.02, p = 0.04). Multi-strain probiotics (8x109 CFU of each of L. helveticus, B. animalis ssp. lactis, Enterococcus faecium, B. longum and Bacillus subtilis) have also been shown in one three-month RCT of 60 college students to decrease hyperactivity (p = 0.012) but have no effect on attention, timing, or impulsivity (64). Higher academic grades were associated with lower levels of hyperactivity and impulsivity in those who received probiotics (rs = 0.554, p = 0.005 and rs = 0.520, p = 0.009, respectively).

Timing of probiotics may be important and if given early, probiotics may reduce the likelihood of developing neurodivergence. Pärtty et al. (2015) also showed in their trial that giving a probiotic in early life may decrease the likelihood for the development of ADHD and autism. The 75 infants in this trial were given Lactobacillus rhamnosus GG (1x1010 colony-forming units) or placebo during the first six months of life and at the age of 13, ADHD or autism was diagnosed in 17.1% of children given placebo but in none of the children given probiotics (p = 0.008)(62).

To date, prebiotics have also shown limited effects on autistic behaviours. In a recent RCT of 41 autistic children the supplementation of glucooligosaccharides (GOS) (2.4g/day) compared to placebo did not affect social and mealtime behaviour and only marginally affected GI symptoms, despite an increase in Bifidobacterium levels (1.4–5.9%, p = 0.001) (66). In contrast, the addition of prebiotics to probiotics has been shown to be beneficial for the management of ADHD symptoms. In one 10-week RCT of 231 adults with ADHD and/or borderline personality disorder (BPD), the supplementation of a synbiotic containing 400 billion lactic acid bacteria/dose in combination with fermentable fibres (2.5g/dose) was shown to improve irritability compared to placebo (OR: 0.2, 95% CI: -6.8 to -0.3; p = 0.03) (67). Emotional dysregulation (−3.6, 95 % CI:-6.8 to −0.3; p = 0.03), emotional symptoms (−0.6, 95 % CI:-1.2 to −0.05; p = 0.03), inattention (−1.8, 95 % CI: −3.2 to −0.4; p = 0.01), functioning (−2.7, 95 % CI: −5.2 to −0.2; p = 0.03), and perceived stress (−0.6, 95 % CI: −1.2 to −0.05; p = 0.03) were also all improved compared to placebo. However, improvements with synbiotics may be limited to individuals with elevated soluble vascular cell adhesion molecule-1 (sVCAM-1), which is a marker for vascular inflammation. In a second RCT of 182 adults and children with ADHD given a lactic acid bacteria (4x1011 CFU) and fermentable fibre (2.5g) synbiotic, no improvements were seen in ADHD symptoms compared to placebo (68). However, in children with elevated sVCAM-1 (>519519.7 pg/mL) at baseline, synbiotics reduced autism characteristicss (95% CI: −0.083, −0.001, p = 0.044), and restrictive, repetitive, and stereotyped behaviours (95% CI: −0.199, −0.006, p = 0.039). In adults with elevated sVCAM-1 improvements to emotional regulation were observed (95% CI: −17.0, −2.40, p = 0.011) especially in the domains of clarity (95% CI: −2.56, −0.088, p = 0.037), goals (95% CI: −4.37, −0.291, p = 0.027), strategies (95% CI: −5.51, −0.457, p = 0.002), and nonacceptance (95% CI: −3.39, −0.125, p = 0.036) of the Difficulties of Emotional Regulation Scale-16.

Practitioners may like to consider that gut dysbiosis and lower numbers of Bifidobacterium may be associated with neurodivergence. However, despite this, the supplementation of single strain probiotics may have limited effects on autistic and ADHD behaviours. To better manage hyperactivity, multi-strain probiotics may be beneficial. Given that timing of supplementation may be important and with neurodivergence having a genetic element, neurodivergent parents may like to consider multi-strain probiotics in early life to reduce the likelihood of the development of autistic and ADHD behaviours in children. Synbiotics may be an overall better choice for some adults and children with benefits to emotional regulation. Assessment of sVCAM-1 levels may help to understand who would best benefit from synbiotic supplementation; however further research is required before it is considered a reliable biomarker.

ESSENTIAL FATTY ACIDS

CAN ESSENTIAL FATTY ACIDS SUPPORT THE WELLBEING OF NEURODIVERGENT INDIVIDUALS?

Polyunsaturated omega-3 fatty acids (n-3 PUFAs), including eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), are essential nutrients

that the body converts inefficiently and must therefore be obtained through diet (69). DHA, the most abundant omega-3 in the brain, is critical for the development and function of the cerebral cortex and hippocampus, regions essential for learning and memory (70,71). In an RCT, 401 five-year-olds whose mothers received 400 mg/day DHA during pregnancy demonstrated better cognitive outcomes than 397 controls, with lower omission T-scores on the Conners' Kiddie Continuous Performance Test ($p < .01$) (72). Specifically, children in the DHA group had better performance on the Conners' Kiddie Continuous Performance Test, with lower omission T-scores (DHA: 47.6 ± 10.3; placebo: 49.6 ± 11.2; $p < .01$), indicating enhanced attention.

Deficiencies in n-3 PUFAs have been implicated in attention-deficit/hyperactivity disorder (ADHD). Children and adolescents with ADHD show significantly lower red blood cell (RBC) n-3 PUFA levels compared to controls (73,74), with lower levels linked to more severe symptoms (51). A systematic review of seven studies involving 590 children (aged 8–12) reported that 500 mg/day EPA significantly improved inattention ($p < .0001$), and six studies ($n = 551$) found improvements in hyperactivity ($p = 0.04$) (76). However, a recent meta-analysis of 37 trials found only low-certainty evidence for PUFA benefits, with high-certainty evidence showing no effect on parent-rated symptoms (74).

Similar deficiencies have been observed in autism. In a study of 121 autistic children (aged 3–17) and 110 controls, autistic participants had significantly lower RBC levels of arachidonic acid (AA) and DHA (77). An RCT involving 31 preterm autistic infants (18–38 months) showed that supplementation with EPA (338 mg), DHA (225 mg), and γ-linolenic acid (83 mg) led to significant behavioural improvements on the Brief Infant-Toddler Social and Emotional Assessment compared to canola oil controls (difference in change = −2.1 points; 95% CI: −4.1, −0.2) (78).

There may be a potential role for omega-3 fatty acids in the development of reading abilities. In a study of 42 children (25 with dyslexia) positive associations were found between omega-3 blood concentrations and reading performance (79). A similar study in adults (32 with dyslexia and 20 controls) demonstrated higher levels of word reading were associated with higher total omega-3 concentrations across groups, although there were no significant absolute differences in membrane fatty acid levels between those with dyslexia and controls (80). However, a clinical trial assessing supplementation with omega-3 fatty acid (ethyl-EPA, 500 mg/day) and carnosine (400 mg/day) in children with dyslexia, found no benefits compared to placebo (81). In one study, children with DCD ($n = 117$) received daily omega-3 (558 mg EPA, 174 mg DHA), omega-6 (60 mg γ-linolenic acid), and vitamin E. After three months, the treatment group showed improvements in reading ($p < 0.004$), spelling ($p < 0.001$), and behaviour ($p < 0.01$), but not motor skills (82), suggesting that the combination of acids may be important for maximising benefits.

Practitioners should be aware that evidence is mixed with regard to the role of dietary supplementation with n-3 PUFAs for neurodivergent individuals. However, supplementation with EPA and DHA, may offer safe and potentially beneficial support for neural development and function in some individuals with developmental conditions. Further research is needed to determine optimal dosages and fatty acid combinations and to explore whether benefits extend to adults.

MICRONUTRIENTS

HOW CAN MICRONUTRIENT SUPPLEMENTATION WITH MINERALS SUPPORT THE WELLBEING OF NEURODIVERGENT INDIVIDUALS?

Mineral deficiencies may be an influencing factor in the aetiology and progression of ADHD (83). Autistic children may also have an increased need for minerals due to a range of metabolic issues including increased

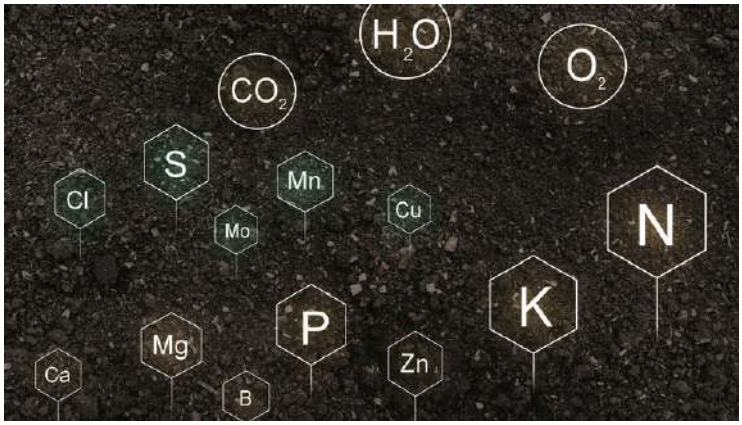
oxidative stress, mitochondrial disorders, methylation issues and substrate deficiencies (sulfate and lithium) (84). Additionally, feeding issues and ARFID are associated with autism (60), and this may perhaps be reflected in mineral deficiencies often seen in autistic individuals (86).

Iron (Fe)

Iron deficiencies have been noted in patients with hyperactivity (87). Selective eating habits may also lead to deficiencies in autistic children (88). Adequate dietary intake of Fe is essential for neurotransmitter function. It is required for dopamine synthesis which takes place in the prefrontal cortex, a key area of the brain structure that has also been associated with ADHD. Additionally, a reduction in dopamine transporter activity has been associated with the progression of ADHD (87). It has been hypothesised that Fe supplementation could improve characteristics of hyperactivity, impulsiveness and inattentiveness in individuals with ADHD (83). These results were reflected in a 2017 RCT of N=42 non-anaemic children with low serum ferritin diagnosed with ADHD (87). Ferrous sulfate was supplemented as an adjuvant therapy to methylphenidate at a dose of 5mg/kg/day for 2 months. Results showed improved symptoms of inattention, hyperactivity and impulsiveness as reported by parents.

Magnesium (Mg)

It has been reported that a Mg deficiency could be a predictive factor in neurological conditions such as ADHD and autism (89), and individuals with ADHD may have an elevated requirement for magnesium, however, no causal relationships has been established (90). A deficiency in Mg has been associated with cognitive impairment, lack of concentration, mood swings, nervousness and fatigue. Some studies have reported lower levels of Mg in individuals with a diagnosis of ADHD and autism, whilst others have not (91). Research into Mg supplementation has produced mixed results (92). However, an RCT into the combined benefits of supplemental Mg (6mg/kg/day) and vitamin D (50000 IU/wk) reported improvements in behavioural and emotional characteristics including emotional problems, peer problems, total difficulties and internalising ($p = 0.001$) in adolescents with ADHD and low serum baseline levels of vitamin D and magnesium (93).



Zinc (Zn)

Zn is required for cognitive and mental development Zn is involved in the dopaminergic and adrenergic pathways that help regulate attention, memory, mood, stress response (94). Low levels can play a role in depression and anxiety disorders. Serum levels of Zn may be reduced in autistic individuals (95). Deficiencies in zinc have also been found in patients with hyperactivity (83). It has been hypothesised that this may be due to higher levels of Zn wasting as urinary levels have been found to be higher in children with ADHD compared to neurotypical controls (94). Higher levels of pro-inflammatory cytokines often seen in ADHD, could also be due to lower levels of Zn. Zn may act as dopamine reuptake inhibitor and be beneficial as an adjuvant therapy to the stimulant drug methylphenidate used to manage ADHD, and which may contribute to decreased levels of Zn (83). An RCT researching supplementation of 150mg/day of Zn for 12 weeks was found to improve symptoms of impulsivity, hyperactivity and socialisation in 400 patients with ADHD (96).

VITAMINS

WHAT ROLE DO VITAMINS PLAY IN SUPPORTING THE WELLBEING OF NEURODIVERGENT INDIVIDUALS?

Vitamins and multinutrients are required for the optimisation of metabolic health including the methylation pathways and krebs cycle. They are also required as co-factors in multiple enzymatic pathways (97). ADHD has been associated with vitamin deficiencies (93) and in some instances, excess (94). Research has shown that changes in diet and improvement in vitamin status could improve the

efficacy of ADHD interventions (83). Additionally, it has been reported that autistic individuals are more susceptible to nutrient deficiencies often due to self-limiting diets, as well as potential increased needs for specific nutrients (84).

Vitamin A

Vitamin A has been found to be low in autistic children compared to neurotypical controls (98). Vitamin A may play a role in the regulation and synthesis of serotonin, a pathway that is often found to be dysregulated in autistic people (95). Deficiencies have been reported to exacerbate autistic characteristics (86). In several studies, supplementation with vitamin A has been shown to improve autistic characteristics, including a study of 33 children diagnosed with ASD which found doses of 0.06mg/kg vitamin A to be effective for improving autistic characteristics (86).

B Complex Vitamins

B complex vitamins (B1, B6, B9, B12) play a vital role in the central nervous system (CNS) function (95). Vitamins B9 and B12 are key in the synthesis of neurotransmitters that affect mood and emotions, such as dopamine (99). Deficiencies in vitamin B12 are also known to contribute to neurological issues including cognitive impairment, motor disturbances, abnormal balance and sensory and memory loss (86). Some studies have associated deficiencies in B12 and B9 (folate) with ADHD, whilst others have not (99). Lower levels have been found in children with ADHD compared to neurotypical controls, and supplementation may improve ADHD characteristics. Conversely, elevated levels of vitamin B6 have been reported in autistic individuals. This may be due to insufficient levels of the enzyme, pyridoxal kinase, required to metabolise vitamin B6 (86).

Vitamin D

Multiple studies have shown a connection between deficiencies in vitamin D and ADHD (100). The relationship between vitamin D and autism is however unclear (101). Vitamin D is needed for neurodevelopment, mental health and CNS function (68). It also has anti-inflammatory properties (95). A recent meta-analysis found that autistic individuals had lower levels of vitamin D compared to neurotypical control groups in 16/19 studies (101). In addition, one study found that that supplementation with vitamin D at a dose of 0.049 mg/kg/day can improve symptoms of irritability and hyperactivity in autistic children (86). Another meta-analysis of 3 studies reported that vitamin D supplementation may be beneficial for hyperactivity but not core autistic characteristics such as sensory issues (102).

Multi-Nutrient Formulas

A recent systematic review and meta-analysis of 16 RCTs reported that multi-nutrient formulas may be more effective for improving characteristics of autism and ADHD than single nutrients alone (97). Practitioners should be aware that vitamin and mineral deficiencies may exacerbate the symptoms of ADHD, autism and neurodivergence but have not been shown to be causative. The correction of identified nutrient deficiencies through diet and supplementation may be beneficial and support management of symptoms.

LIFESTYLE FACTORS

WHICH MODIFIABLE LIFESTYLE FACTORS MAY HELP SUPPORT WELLBEING FOR INDIVIDUALS WITH NEURODIVERGENCE?

Modifiable lifestyle factors, particularly physical activity and sleep are increasingly recognised as effective non-pharmacological strategies for supporting wellbeing in neurodivergent individuals. Physical activity interventions have been associated with improvements in quality of life and sleep outcomes. In autistic children aged 3–18 years, different physical activities significantly improved social functioning (103). A similar study, in autistic children aged 3-18, reported that exercise significantly improved executive function

functioning (104) with ping pong (70 minutes 2x/week for 12 weeks) and cycling (60 minutes 5x/week for 2 weeks) showing the greatest benefits. A systematic review of 21 studies in children with ADHD found that physical activity, including ball games, racket sports, mindfulness, and yoga, improved executive function, attention, inhibition, and broader neurobehavioural outcomes, with sessions of at least 20 minutes yielding positive effects (105).

Moderate to intense exercise reduced ADHD characteristics and enhanced neurophysiological function. Tailoring activities to individual preferences (e.g., martial arts or team sports) may enhance adherence and social engagement (105). In children and adolescents with DCD, a systematic review of 13 studies (n = 453) found that high-intensity interval training (HIIT) significantly improved mental health, mood, quality of life, social behaviour, inhibitory control, and physical fitness (106).

In contrast, poor sleep quality is associated with reduced wellbeing in neurodivergent populations. In autism, sleep issues correlate with core traits and behavioural difficulties in children under 18 (107,108). Adolescents with ADHD also show a bidirectional relationship between sleep disturbances and symptom severity (109). A meta-analysis found that autistic adults and those with ADHD experienced poorer sleep than neurotypical controls, including longer sleep latency, reduced efficiency, more awakenings, and worse overall quality (110). Pooled prevalence estimates for insomnia and sleep disorders were 59% and 51% in autism, and 63% and 52% in ADHD, respectively.

Physical activity may mitigate sleep problems. In a meta-analysis of 388 participants with ADHD (ages 6–54), exercise was associated with self-reported sleep quality (111). While objective measures in a smaller subsample (n = 131) showed a non-significant trend toward increased sleep duration, overall findings suggest potential exercise benefits. Possible mechanisms explaining the effect include melatonin regulation, thermoregulation, and vagal nerve function. Interventions ranged from jogging and HIIT to Tai Chi. Although heterogeneity limits standardised recommendations, sessions of 30–50 minutes, 2–3×/week were generally effective.

Liang et al. (2023) identified significantly lower physical activity in autistic children (n = 679; 59 min/day) compared to controls (n = 1573; 77 min/day) (112). Total sleep time was significantly lower for autistic children (461.00 min; SD 34.05) compared to controls (474.54 min; SD 48.76). These findings highlight that many autistic individuals fall short of WHO's recommended 60 minutes/day of moderate-to-vigorous physical activity (113).

Practitioners should be aware of these WHO recommendations and embrace a collective approach to promote exercise in neurodivergent children and adults (112). As physical activity and sleep are intrinsically linked as part of a 24-hour movement framework (114), the 'take home' message is, a wide range of personalised activities should be considered to support sleep, wellbeing and quality of life. Sleep hygiene practices (including sleeping environment, routines, caffeine and alcohol use, and technology habits) should also be encouraged, particularly in adolescents with ADHD (115).

SUMMARY

THE ROLE OF NUTRITION PRACTITIONERS IN SUPPORTING NEURODIVERGENT INDIVIDUALS

Nutrition practitioners are uniquely positioned to support the wellbeing of neurodivergent individuals through food-based strategies that address both physiological needs and lifestyle factors. This review provides an evidence-base for evaluating different interventions aimed at supporting safe and effective care. Research suggests that dietary patterns and

nutrient intake can influence brain development and behaviour, highlighting the importance of a personalised, whole-food approach to care. Nutritional therapists can play a key role in identifying and addressing dietary insufficiencies, supporting digestive health, and helping individuals and families navigate selective eating patterns or ARFID whilst reducing the intake of ultra-processed food.

Evidence points to the potential benefits of optimising intake of essential fatty acids, vitamins, and minerals in supporting neurodiverse individuals. While much research has explored the impact of supplementation and this could certainly be useful for some individuals, practitioners may also wish to encourage nutrient-dense food choices that supply omega-3 fatty acids, magnesium, zinc, iron, and B vitamins. Emerging evidence highlights that autistic individuals and those with ADHD show differences in gut microbiota compared to typically developing individuals. These differences may account for the higher prevalence of gastrointestinal issues but could equally arise as a consequence of them, and therefore recommendations to mitigate this difference need to be carefully considered. It is also important to acknowledge the role of lifestyle factors, particularly physical activity and sleep, which are increasingly linked to improvements in executive function, emotional wellbeing, and quality of life in neurodivergent individuals.

By integrating nutritional and lifestyle interventions into a personalised plan, practitioners can support neurodivergent individuals in ways that complement existing medical, psychological and therapeutic care. A collaborative, strengths-based approach, focusing on both symptom management and overall health, allows nutrition practitioners the chance to contribute meaningfully to the wellbeing and daily functioning of this diverse population.

KEY PRACTICE POINTS FOR NUTRITIONAL THERAPISTS

- **Personalise support:** Tailor dietary advice to individual needs, preferences, and sensory sensitivities common in neurodivergence.
- **Focus on whole foods:** Encourage nutrient-dense sources of omega-3s, B vitamins, magnesium, zinc, and iron, while being mindful of selective eating and ARFID.
- **Address lifestyle factors:** Promote physical activity and good sleep hygiene as part of a 24-hour movement and recovery framework.
- **Work collaboratively:** Integrate nutritional support with existing medical, psychological and therapeutic interventions for a holistic approach.
- **Support families and carers:** Provide practical strategies to improve diet quality and reduce stress around mealtimes.

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

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