

REVIEW

[View Article Online](#)
[View Journal](#) | [View Issue](#)
Cite this: *Food Funct.*, 2025, **16**, 2622Received 4th December 2024,
Accepted 4th March 2025

DOI: 10.1039/d4fo06011c

rsc.li/food-function

Dietary therapy to halt the progression of diabetes to diabetic kidney disease†

Hongtu Hu,^{a,b} Guohua Ding^{*a,b} and Wei Liang ^{*a,b}

Diabetic Kidney Disease (DKD) is a common and serious complication of diabetes, particularly Type 2 Diabetes Mellitus (T2DM), which significantly contributes to patient morbidity and mortality. The limitations of traditional treatments like ACE inhibitors and ARBs in managing DKD progression highlight the need for innovative therapeutic strategies. This review examines the impact of various dietary patterns, such as the Mediterranean diet, ketogenic diet, intermittent fasting, DASH diet, and vegetarian diet, on the management of DKD. Evidence suggests these diets can halt the progression of DKD, although further research is needed to confirm their long-term effectiveness and safety. Personalized dietary approaches tailored to individual needs may enhance outcomes for DKD patients.

1 Introduction

Over the past century, non-communicable diseases (NCDs),¹ including cardiovascular disease, cancer, and diabetes,² have emerged as the leading causes of mortality and morbidity worldwide. Among these, diabetes, particularly Type 2 Diabetes Mellitus (T2DM), has become an epidemic, contributing significantly to the global healthcare burden.³ Diabetic Kidney Disease (DKD) is a common and severe complication of T2DM, characterized by progressive kidney damage that increases the risk of kidney failure and premature death. DKD is not only a major cause of end-stage renal disease (ESRD), but it also exacerbates the comorbidities associated with diabetes, such as cardiovascular diseases, creating a vicious cycle of deterioration in patient health and quality of life.⁴

Despite advancements in treatment options, conventional therapies, such as Angiotensin-Converting Enzyme (ACE) inhibitors and Angiotensin II Receptor Blockers (ARBs), have shown limited efficacy in halting or reversing the progression of DKD. These therapies primarily aim to control blood pressure and proteinuria but do not address the root causes or mitigate the long-term impacts of DKD progression. As a result, there remains a critical gap in effective treatments that can prevent the worsening of kidney function in diabetic patients.

Emerging evidence suggests that dietary interventions may offer a promising alternative or adjunct to pharmacological treatments in managing DKD. While various dietary patterns,

including the Mediterranean diet, ketogenic diet, intermittent fasting, Dietary Approaches to Stop Hypertension (DASH) diet, and vegetarian diet, have been explored for their potential benefits in managing DKD, the evidence is still inconclusive, particularly regarding their long-term efficacy, safety, and mechanisms of action. This highlights a significant research gap in understanding how different dietary patterns affect DKD progression, and whether these dietary approaches can provide sustained benefits over time. Therefore, this review aims to critically evaluate the current state of knowledge on dietary therapies for DKD, examining their potential to improve patient outcomes and slow disease progression, while identifying areas where further research is urgently needed.

2 Literature search method

To identify relevant studies, we screened publications to date that discussed dietary therapies, such as Mediterranean diet, Ketogenic Diet, and DASH diet, and their role in preventing the progression of diabetes to DKD. We conducted searches in databases including Elsevier, Wiley, Taylor, Francis Online, and Web of Science using keywords such as “diabetes”, “diabetic kidney disease”, “Mediterranean diet”, “Ketogenic Diet”, “fasting”, “Ketogenic Diet”, “DASH diet” and “vegetarian diet”. A total of 281 articles were identified, of which 133 were actually cited.

3 T2DM and diet

Diabetes mellitus (DM) is a prevalent group of NCDs, with over 500 million individuals affected globally, and more than 90%

^aDivision of Nephrology, Renmin Hospital of Wuhan University, Wuhan, China.E-mail: ghxding@whu.edu.cn, dr.liangwei@whu.edu.cn^bNephrology and Urology Research Institute of Wuhan University, Wuhan, China†Electronic supplementary information (ESI) available. See DOI: <https://doi.org/10.1039/d4fo06011c>

of these cases are T2DM.⁵ It is well established that weight gain and obesity are associated with the development of T2DM. Additionally, the development of T2DM is accompanied by obesity and insulin resistance, primarily due to the phosphorylation of the insulin receptor substrate (IRS) by receptor tyrosine kinases (RTK), which further contributes to elevated glycemia. Both factors are directly related to food intake.³

The etiology of T2DM remains complex and not fully understood. However, evidence indicates that T2DM is characterized by pathophysiological changes, including pancreatic β -cell dysfunction, insulin resistance, and chronic inflammation, which collectively impede glycemic control.⁶ Furthermore, T2DM promotes increased cellular and chemokine levels, such as TNF- α and IL-6, and activates pro-fibrotic signaling pathways, including TGF- β and NF- κ B, ultimately leading to complications.⁷

Recent studies have highlighted that an unhealthy diet significantly contributes to T2DM development.⁸ Diets high in calories, fat, and sugar are associated with weight gain and

obesity, particularly central obesity (abdominal fat accumulation), leading to reduced insulin sensitivity and the development of insulin resistance. This impairs the entry of blood glucose into cells for energy production, resulting in elevated blood glucose levels and the onset of T2DM.⁹ Additionally, a diet high in fat, particularly saturated fatty acids, has been linked to fat accumulation in visceral organs, such as the liver. This ectopic fat accumulation interferes with insulin signaling, leading to chronic inflammation, insulin resistance, and elevated blood glucose levels.¹⁰

In addition, diet significantly influences the composition and functionality of the gut microbiome. Disturbed gut microbes have been linked to exacerbated metabolic disorders and chronic inflammation, reducing insulin sensitivity.¹¹ Therefore, dietary habits play a crucial role in T2DM development. Diets high in calories, fat, and sugar increase the risk of T2DM (Fig. 1). Consequently, adopting a healthy and balanced dietary structure and appropriate nutritional management is paramount in preventing and controlling T2DM.

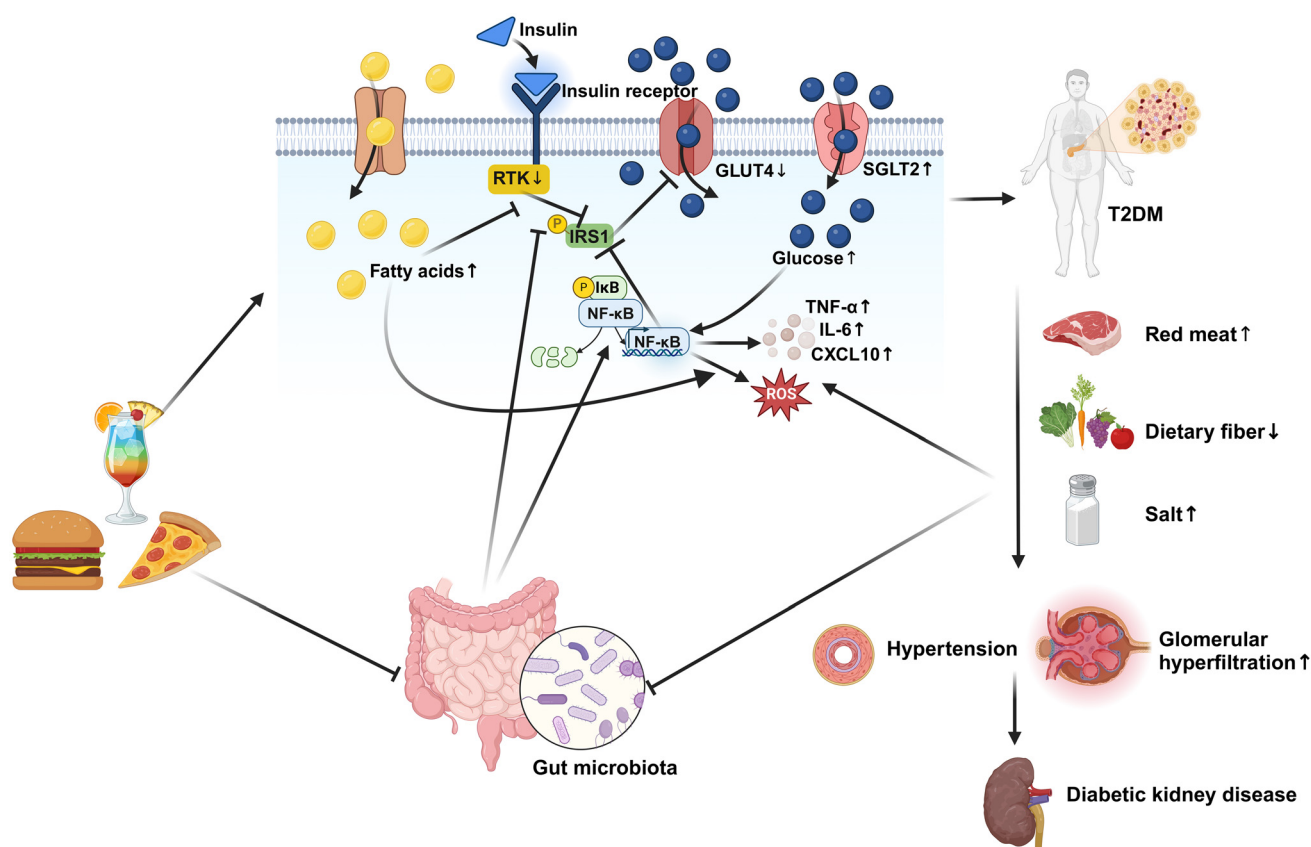


Fig. 1 The role of diet in type 2 diabetes (T2DM) and diabetic kidney disease (DKD). A high-calorie, high-fat, and high-sugar diet is linked to weight gain and central obesity, disrupting insulin signaling, triggering chronic inflammation, and causing insulin resistance. This diet also alters gut microbiota, leading to metabolic disorders and reduced insulin sensitivity, ultimately causing type 2 diabetes (T2DM). In T2DM patients, continued intake of such a diet elevates blood glucose, damages glomerular filtration membranes, and accelerates diabetic kidney disease (DKD). Western diets high in salt and fat increase hypertension risk, worsening glomerular damage and promoting DKD. Excessive red and processed meat consumption burdens kidneys, accelerating dysfunction, while moderate vegetable protein intake offers protection. Low dietary fiber disrupts gut flora, leading to metabolic disorders and inflammation, accelerating DKD. Created with BioRender.com.

5 Role of dietary therapy in DKD

5.1 DKD and mediterranean diet (MED)

The traditional MED originates from the coastal areas of the Mediterranean Sea and is characterized by high consumption of vegetables, fruits, nuts, and olive oil.²² The MED has garnered considerable research interest in recent years due to its potential to prevent various chronic diseases, including cardiovascular disease, T2DM, metabolic syndrome, certain cancers, and chronic kidney disease.^{23,24} A *meta*-analysis of 2 217 404 participants showed that adherence to the MED significantly reduces the risk of developing colorectal cancer.²⁵ Another *meta*-analysis demonstrated that the MED reduces liver enzymes and liver fat content in patients with non-alcoholic fatty liver disease (NAFLD).²⁶ Additionally, recent studies have associated the MED with notable improvements in overall cognition and gastrointestinal symptoms.²⁷

In diabetes treatment, the MED has yielded noteworthy outcomes. Prospective studies have shown that adherence to the MED is associated with a reduced risk of developing diabetes,^{28,29} and significant reductions in glycated hemoglobin levels³⁰ and inflammation³¹ in patients with prediabetes and T2DM. Furthermore, the MED has been shown to diminish the incidence of cardiovascular complications,³² carotid atherosclerosis,³³ non-alcoholic fatty liver disease,³⁴ and mortality in patients with T2DM.³⁵ A recent *post-hoc* analysis from the San Carlos Gestational Prevention Study demonstrated that a nutritional regimen based on the MED before the twelfth week of gestation reduces the incidence of gestational diabetes mellitus at three years postpartum.³⁶ Moreover, a prospective cohort study found that the MED significantly reduces the risk of DKD in patients with hyperglycemia,³⁷ with a dose-dependent effect.³⁸ However, adding extra virgin olive oil to the MED does not prevent DKD in the cohort study.³⁹ Numerous studies have also substantiated that adherence to the MED is not markedly correlated with cardiovascular risk factors and renal function.^{40,41}

From a mechanical perspective, the components of the MED are effective in reversing the progression of T2DM to DKD. The abundance of antioxidants, such as ascorbic acid and β -carotene in MED foods significantly reduces oxidative stress, chronic inflammation, and platelet aggregation.^{42,43} Furthermore, adherence to the MED has been linked to lower levels of C-reactive protein (CRP),⁴⁴ which correlates with a reduced impact of elevated blood glucose on renal cellular function, thus lowering the risk of developing DKD from T2DM. Recent clinical studies have demonstrated that glucagon-like peptide-1 (GLP-1) can attenuate the progression of T2DM⁴⁵ and DKD⁴⁶ by promoting insulin production and secretion. Notably, the MED has been shown to promote GLP-1 secretion,⁴⁷ enhance endothelial function, and delay DKD progression in T2DM patients.⁴⁸

Moreover, evidence indicates that the MED can correct the imbalance of gut microbial homeostasis in patients with T2DM and DKD.⁴⁹ Gut microbes utilize the fiber in the MED to synthesize short-chain fatty acids (SCFAs),⁵⁰ which regulate

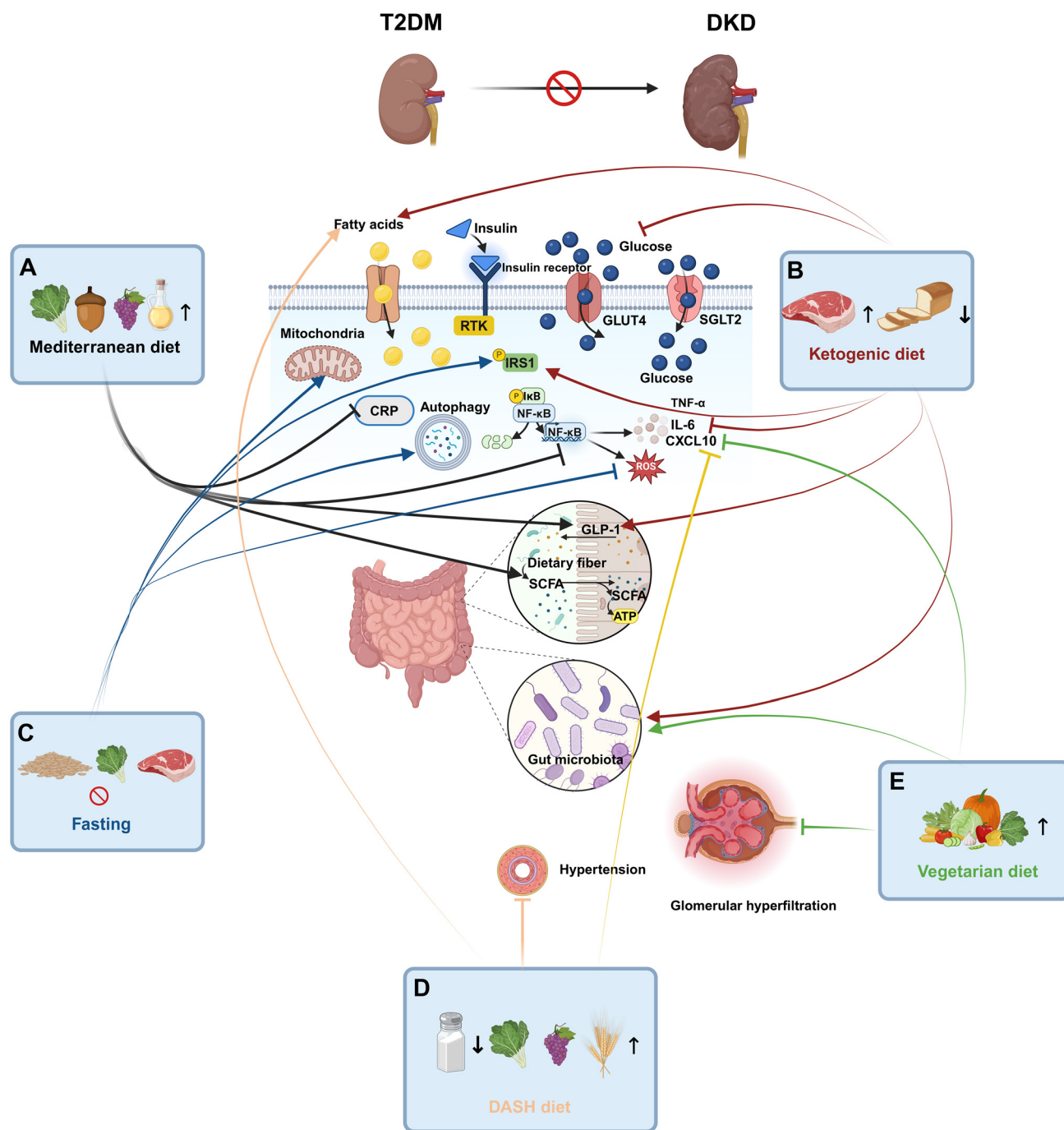


Fig. 2 Molecular mechanisms of dietary therapy and their metabolites in diabetic kidney disease. (A) The Mediterranean diet (MED) has been demonstrated to impede the progression of Type 2 diabetes mellitus (T2DM) to diabetic kidney disease (DKD) through the reduction of inflammatory responses and oxidative stress, the stimulation of glucagon-like peptide 1 (GLP-1) secretion, and the modulation of the gut microbiota. (B) The ketogenic diet (KD) has been demonstrated to impede the progression of T2DM to DKD through the regulation of the body's metabolic state, the influence of gut microbiota homeostasis, the promotion of GLP-1 secretion, and the exertion of anti-inflammatory and antioxidant effects. (C) Fasting has been demonstrated to delay renal cell damage and loss of function. This is achieved by improving insulin sensitivity, reducing inflammation and oxidative stress, and promoting mitochondrial function and autophagy processes. (D) The DASH diet has been demonstrated to safeguard kidney function by regulating blood pressure and reducing inflammation, lipid levels and kidney burden. (E) The vegetarian diet has been demonstrated to impede the progression of T2DM to DKD through a number of mechanisms. These include antioxidant and anti-inflammatory effects, modulation of gut microbiota, reduction of proteinuria and improved nutritional status. Created with BioRender.com.



5.4 DKD and DASH diet

The DASH diet has demonstrated considerable success in managing hypertension. A recent randomized controlled trial (RCT) (IRCT20180201038585N12) involving 60 subjects with

5.5 DKD and vegetarian diet

A vegetarian diet is based on plant-based foods, excluding all animal products, including red and white meat and fish. The classification of vegetarian diets includes strict vegetarian, egg-lacto-vegetarian, lacto-vegetarian, egg-vegetarian, and fish-vegetarian diets.¹¹⁴ Prospective epidemiological studies indicate that a vegetarian diet may have a preventive effect against obesity,¹¹⁵ hypertension,¹¹⁶ cancer,¹¹⁷ and cognitive decline.¹¹⁸ A recent meta-analysis indicates that adherence to a plant-based diet for a minimum of 14 days is associated with improved insulin resistance in individuals with a BMI above the healthy range.¹¹⁹ Furthermore, a vegetarian diet has been demonstrated to diminish the likelihood of developing T2DM.¹²⁰

Food & Function

In conclusion, a vegetarian diet may benefit individuals with DKD, but it must be planned and managed appropriately to ensure adequate nutritional intake and avoid potential risks.

Different dietary patterns exhibit varying degrees of risk. In MED, the consumption of certain foods, such as full-fat

Implementing these dietary patterns in managing DKD must consider the individual's health status, nutritional requirements, and personal preferences (Table 1). Therefore, it is recommended that dietary plans be developed under the guidance of healthcare professionals to ensure the safety and efficacy of the dietary regimen. Several clinical trials (NCT05495451, NCT05984459, NCT05589467, and NCT06152588) aim to improve outcomes for T2DM or DKD patients by combining dietary therapy (Table S1†).

7.1.4 Integration with medication. It is imperative to enhance the integration of dietary therapies with existing pharmacological treatments for DKD. While certain dietary components may augment medication efficacy, others may impede drug metabolism or exacerbate adverse effects. A com-

Table 1 Comparison of different dietary therapies in DKD

Dietary pattern	Advantages	Disadvantages	Research conclusions
Mediterranean diet	Rich in healthy fats (olive oil), antioxidants (fruits and vegetables), and fiber (whole grains); associated with reduced inflammation, improved glycemic control, and cardiovascular health benefits. ^{7,31–43}	May be difficult to adhere to in the long term due to dietary restrictions and availability of specific foods; can be more expensive compared to standard diets. ^{60,61,133}	Shows potential in reducing the risk of DKD progression, improving glycemic control, reducing cardiovascular risk, and enhancing overall health outcomes. Widely supported by numerous studies. ^{31–43}
Ketogenic diet	Low carbohydrate intake forces the body to use fat as a primary energy source, which can improve glycemic control, promote weight loss, and potentially improve insulin sensitivity. ^{38,62–71}	May lead to nutrient deficiencies (fiber, vitamins, and minerals) and is not suitable for everyone, especially those with kidney issues due to the high protein and fat content. ^{88–90}	Mixed results; some studies show benefits in weight loss and glycemic control, while others highlight potential risks, especially for kidney health. Long-term safety and effects need further research. ^{38,62–71}
Fasting	Flexible approach with various fasting schedules; can promote weight loss, improve insulin sensitivity, and reduce oxidative stress and inflammation. ^{91–105}	Can be difficult to maintain; potential for overeating during feeding periods, which might negate the benefits; initial adaptation phase can be challenging.	Emerging evidence suggests benefits for metabolic health, weight loss, and reduction in inflammation and oxidative stress. Long-term effects and optimal fasting protocols require further investigation. ^{91–105}
DASH diet	Emphasizes fruits, vegetables, whole grains, and low-fat dairy; associated with lower blood pressure, improved heart health, reduced risk of cardiovascular diseases, and better glycemic control. ^{109–121}	May be difficult to adhere to due to dietary restrictions and the need for significant changes in eating habits; requires careful planning to meet nutritional needs. ¹²¹	Associated with improved kidney function, lower blood pressure, and reduced cardiovascular risk. Adherence can be challenging, but the health benefits are well-supported by research. ^{109–121}
Vegetarian diet	High in fiber (fruits, vegetables, legumes) and low in saturated fats; associated with reduced risk of chronic diseases (heart disease, hypertension), improved kidney function, and potential weight management benefits. ^{122–132}	Requires careful planning to ensure adequate nutrient intake, especially protein, vitamin B12, iron, and omega-3 fatty acids; potential for nutrient deficiencies if not well-planned.	Generally beneficial for kidney function and overall health; reduces risk of chronic diseases and supports weight management. Nutrient deficiencies need to be managed through careful dietary planning. ^{122–132}

prehensive understanding of these interactions is essential to optimize treatment regimens.

7.1.5 Impact of comorbidities. A significant proportion of individuals with DKD also present with comorbidities such as cardiovascular disease or hypertension. This necessitates considering additional dietary factors. Managing DKD through diet is further complicated by the need to balance the dietary demands of multiple conditions.

7.2 Strategies to overcome challenges and improve clinical translation

7.2.1 Personalized nutrition. Advances in nutrigenomics and metabolomics offer the potential for developing personalized nutrition plans based on an individual's genetic and metabolic profile. By understanding how specific nutrients interact with a patient's unique biological makeup, healthcare providers can design more effective dietary interventions.

7.2.2 Long-term clinical trials. Large-scale, long-term clinical trials are imperative to assess the efficacy and safety of various dietary patterns in treating DKD. These studies must encompass diverse populations to account for genetic and environmental differences and aim to elucidate the mechanisms by which diet affects kidney function.

7.2.3 Role of the gut microbiota. Recent studies have demonstrated that the gut microbiota plays a pivotal role in mediating the effects of dietary interventions in DKD. The underlying mechanism involves multifaceted interactions, including the production of short-chain fatty acids (SCFAs,

e.g., butyric acid, propionic acid) by GM fermentation of dietary fiber. These SCFAs have been shown to reduce renal inflammation and maintain intestinal barrier integrity by inhibiting histone deacetylase (HDAC) and activating G-protein-coupled receptors (e.g., GPR43/41), which can reduce the intestinal-derived accumulation of uremic toxins (e.g., indophenol sulphate, paracresol) and alleviate renal tubulointerstitial fibrosis.¹³⁰ Secondly, intestinal-derived lipopolysaccharide (LPS) translocation caused by GM dysregulation can activate the Toll-like receptor 4 (TLR4) signaling pathway, promote the release of inflammatory factors (e.g., IL-6, TNF- α) from glomerular mesangial cells, and accelerate the progression of DKD.¹³¹ Clinical studies have further revealed a direct association between GM composition and renal function indices. A cohort study of 432 DM patients found that the relative abundance of butyric acid-producing bacteria in the gut (e.g., *Faecalibacterium prausnitzii*, *Roseburia* spp.) was positively correlated with estimated glomerular filtration rate (eGFR) ($\beta = 0.34$, $P < 0.01$). Furthermore, the annual rate of decline in eGFR increased by 0.82 mL min^{-1} per 1.73 m^2 for each log unit decrease in its abundance.¹³² Furthermore, elevated levels of the GM metabolite trimethylamine-N-oxide (TMAO) induced apoptosis in renal tubular epithelial cells, which was independently associated with the urinary albumin/creatinine ratio (UACR) in DKD patients (OR = 1.52, 95%CI 1.18–1.96).¹³³ The aforementioned mechanisms provide the foundation for dietary modification strategies. The first strategy involves the implementation of a high-fiber/resistant starch diet ($\geq 30 \text{ g per}$



day) to enhance uremic toxin clearance through the enrichment of SCFAs-producing bacteria. The second strategy focuses on the incorporation of Mediterranean dietary polyphenols (e.g., olive oil hydroxytyrosol) to selectively promote *Akkermansia muciniphila* and other beneficial bacteria and inhibit the overgrowth of pathogenic Proteobacteria. In addition, prebiotic/prebiotic combination interventions (e.g., oligofructose + *bifidobacteria*) restored GM diversity and lowered TMAO levels. Future studies must further elucidate the profile of flora characteristics and personalized dietary targets at different DKD stages to optimize the precise GM-based nutritional treatment strategy.

7.2.4 Develop comprehensive guidelines. Researchers, clinicians, and policymakers must collaborate to develop comprehensive dietary guidelines for DKD. These guidelines must be evidence-based, culturally sensitive, and adaptable to patients' individual needs.

7.2.5 Patient education and support. Enhanced patient education and support are essential for improving adherence to dietary interventions. This may involve creating digital tools and resources, such as mobile apps or online platforms, to provide personalized dietary advice, track progress, and offer motivational support.

7.2.6 Integration with digital health technologies. Integrating diet management with digital health technologies, such as wearables and mobile health apps, can provide real-time feedback and support to patients, enhancing adherence and treatment outcomes. These technologies can also enable remote monitoring and counseling, improving the accessibility of diet management.

7.2.7 Interdisciplinary research. Encouraging interdisciplinary research and drawing insights from nutritional sciences, nephrology, genomics, and behavioral sciences can facilitate a more comprehensive approach to managing DKD. Collaborative research programs can foster innovation and accelerate the translation of research findings into clinical practice.

BMI	Body mass index
CKD	Chronic kidney disease
CRP	C-reactive protein
DASH	Dietary approaches to stop hypertension
DKD	Diabetic kidney disease
DPP-4	Dipeptidyl peptidase 4
eGFR	Estimated glomerular filtration rate
ESRD	End-stage renal disease
GLP-1	Glucagon-like peptide 1
HbA1c	Hemoglobin A1c
HDAC	Histone deacetylase
IL-6	Interleukin 6
LPS	Lipopolysaccharide
KD	Ketogenic diet
MED	Mediterranean diet
MODY	Maturity onset diabetes of the young
NAFLD	Non-alcoholic fatty liver disease
NCD	Non-communicable disease
RCT	Randomized controlled trial
RAAS	Renin-angiotensin-aldosterone system
SCFA	Short-chain fatty acid
SGLT2	Sodium-glucose co-transporter 2
SMBG	Self-monitoring of blood glucose
T2DM	Type 2 diabetes mellitus
TNF- α	Tumor necrosis factor alpha
TMAO	Trimethylamine-N-oxide
UACR	Urinary albumin/creatinine ratio

Author contributions

Hongtu Hu and Wei Liang were involved in the conception and design. Hongtu Hu and Wei Liang drafted of the manuscript. Wei Liang and Guohua Ding revised the manuscript critically for intellectual content. Hongtu Hu, Wei Liang and Guohua Ding approved the final version to be published. All authors agree to be accountable for all aspects of work ensuring integrity and accuracy.

Data availability

No datasets were generated or analyzed during the current study.

Conflicts of interest

The authors declare no competing interests.

Acknowledgements

This study was supported by the National Natural Science Foundation of China (82070713 to G. Ding, 81970631 to W. Liang). Fig. 1 and 2 were created with BioRender.com. We extend our apologies to the authors whose contributions

8 Conclusion

In summary, dietary therapies show great promise in treating DKD. However, their use must be individualized and closely monitored to maximize benefits and minimize risks. Future research should focus on large-scale, long-term clinical trials to further elucidate the efficacy and safety of these dietary approaches in different DKD patient populations. Combining dietary therapies with pharmacological treatments could pave the way for a comprehensive and effective DKD management strategy, ultimately improving patients' prognosis and quality of life.

Abbreviations

ACEI	Angiotensin-converting enzyme inhibitor
ARB	Angiotensin II receptor blocker



References

References

- preceded this work, as we regretfully acknowledge our inability to cite them in this review article due to the word limit stipulated by the journal's requirements.
- ## References
- 1 C. P. Benziger, G. A. Roth and A. E. Moran, The Global Burden of Disease Study and the Preventable Burden of NCD, *Glob. Heart*, 2016, **11**, 393–397.
 - 2 A. H. Mokdad, L. Dwyer-Lindgren, C. Fitzmaurice, R. W. Stubbs, A. Bertozzi-Villa, C. Morozoff, R. Charara, C. Allen, M. Naghavi and C. J. Murray, Trends and Patterns of Disparities in Cancer Mortality Among US Counties, 1980–2014, *J. Am. Med. Assoc.*, 2017, **317**, 388–406.
 - 3 N. G. Forouhi, Embracing complexity: making sense of diet, nutrition, obesity and type 2 diabetes, *Diabetologia*, 2023, **66**, 786–799.
 - 4 M. C. Thomas, M. Brownlee, K. Susztak, K. Sharma, K. A. Jandeleit-Dahm, S. Zoungas, P. Rossing, P. H. Groop and M. E. Cooper, Diabetic kidney disease, *Nat. Rev. Dis. Primers*, 2015, **1**, 15018.
 - 5 D. Tomic, J. E. Shaw and D. J. Magliano, The burden and risks of emerging complications of diabetes mellitus, *Nat. Rev. Endocrinol.*, 2022, **18**, 525–539.
 - 6 A. L. Gloyn and D. J. Drucker, Precision medicine in the management of type 2 diabetes, *Lancet Diabetes Endocrinol.*, 2018, **6**, 891–900.
 - 7 S. Rovira-Llopis, C. Banuls, N. Diaz-Morales, A. Hernandez-Mijares, M. Rocha and V. M. Victor, Mitochondrial dynamics in type 2 diabetes: Pathophysiological implications, *Redox Biol.*, 2017, **11**, 637–645.
 - 8 R. Hariharan, E. N. Odjidja, D. Scott, N. Shivappa, J. R. Hebert, A. Hodge and B. de Courten, The dietary inflammatory index, obesity, type 2 diabetes, and cardiovascular risk factors and diseases, *Obes. Rev.*, 2022, **23**, e13349.
 - 9 M. Bluher, M. Aras, L. J. Aronne, R. L. Batterham, F. Giorgino, L. Ji, K. H. Pietilainen, O. Schnell, E. Tonchevska and J. P. H. Wilding, New insights into the treatment of obesity, *Diabetes, Obes. Metab.*, 2023, **25**, 2058–2072.
 - 10 M. E. Piche, A. Tchernof and J. P. Despres, Obesity Phenotypes, Diabetes, and Cardiovascular Diseases, *Circ. Res.*, 2020, **126**, 1477–1500.
 - 11 W. M. de Vos, H. Tilg, M. Van Hul and P. D. Cani, Gut microbiome and health: mechanistic insights, *Gut*, 2022, **71**, 1020–1032.
 - 12 A. Ceriello and F. Prattichizzo, Variability of risk factors and diabetes complications, *Cardiovasc. Diabetol.*, 2021, **20**, 101.
 - 13 A. Odermatt, The Western-style diet: a major risk factor for impaired kidney function and chronic kidney disease, *Am. J. Physiol.: Cell Physiol.*, 2011, **301**, F919–F931.
 - 14 A. Mirzababaei, F. Abaj, Z. Roumi, R. A. Khosroshahi, Y. Aali, C. C. T. Clark, M. Radmehr and K. Mirzaei, Consumption of red, white, and processed meat and odds of developing kidney damage and diabetic nephropathy (DN) in women: a case control study, *Sci. Rep.*, 2024, **14**, 10344.
 - 15 R. N. Moorthi, C. J. Vorland and K. M. Hill Gallant, Diet and Diabetic Kidney Disease: Plant Versus Animal Protein, *Curr. Diabetes Rep.*, 2017, **17**, 15.
 - 16 M. M. Adeva-Andany, C. Fernandez-Fernandez, N. Carneiro-Freire, M. Vila-Altesor and E. Ameneiros-Rodriguez, The differential effect of animal versus vegetable dietary protein on the clinical manifestations of diabetic kidney disease in humans, *Clin. Nutr. ESPEN*, 2022, **48**, 21–35.
 - 17 W. L. Lau, T. Tran, C. M. Rhee, K. Kalantar-Zadeh and N. D. Vaziri, Diabetes and the Gut Microbiome, *Semin. Nephrol.*, 2021, **41**, 104–113.
 - 18 Y. Lytvyn, P. Bjornstad, D. H. van Raalte, H. L. Heerspink and D. Z. I. Cherney, The New Biology of Diabetic Kidney Disease-Mechanisms and Therapeutic Implications, *Endocr. Rev.*, 2020, **41**, 202–231.
 - 19 K. R. Tuttle, R. Agarwal, C. E. Alpers, G. L. Bakris, F. C. Brosius, P. Kolkhof and J. Uribarri, Molecular mechanisms and therapeutic targets for diabetic kidney disease, *Kidney Int.*, 2022, **102**, 248–260.
 - 20 S. Jiang, J. Fang and W. Li, Protein restriction for diabetic kidney disease, *Cochrane Database Syst. Rev.*, 2023, **1**, CD014906.
 - 21 S. Castro-Barquero, A. M. Ruiz-Leon, M. Sierra-Perez, R. Estruch and R. Casas, Dietary Strategies for Metabolic Syndrome: A Comprehensive Review, *Nutrients*, 2020, **12**, 2983.
 - 22 C. Davis, J. Bryan, J. Hodgson and K. Murphy, Definition of the Mediterranean Diet; a Literature Review, *Nutrients*, 2015, **7**, 9139–9153.
 - 23 L. J. Dominguez, G. Di Bella, N. Veronese and M. Barbagallo, Impact of Mediterranean Diet on Chronic Non-Communicable Diseases and Longevity, *Nutrients*, 2021, **13**, 2028.
 - 24 E. A. Hu, L. M. Steffen, M. E. Grams, D. C. Crews, J. Coresh, L. J. Appel and C. M. Rebholz, Dietary patterns and risk of incident chronic kidney disease: the Atherosclerosis Risk in Communities study, *Am. J. Clin. Nutr.*, 2019, **110**, 713–721.
 - 25 Z. Ungvari, M. Fekete, J. T. Fekete, G. Grosso, A. Ungvari and B. Györfy, Adherence to the Mediterranean diet and its protective effects against colorectal cancer: a meta-analysis of 26 studies with 2,217,404 participants, *Geroscience*, 2025, **47**, 1105–1121.
 - 26 Y. Xiong, X. Shi, X. Xiong, S. Li, H. Zhao, H. Song, J. Wang, L. Zhang, S. You, G. Ji, B. Liu and N. Wu, A systematic review and meta-analysis of randomized controlled trials: effects of mediterranean diet and low-fat diet on liver enzymes and liver fat content of NAFLD, *Food Funct.*, 2024, **15**, 8248–8257.

- 27 B. A. Seelarbokus, E. Menozzi, A. H. V. Schapira, A. Z. Kalea and J. Macnaughtan, Mediterranean Diet Adherence, Gut Microbiota and Parkinson's Disease: A Systematic Review, *Nutrients*, 2024, **16**, 2181.
- 28 F. Jannasch, J. Kroger and M. B. Schulze, Dietary Patterns and Type 2 Diabetes: A Systematic Literature Review and Meta-Analysis of Prospective Studies, *J. Nutr.*, 2017, **147**, 1174–1182.
- 29 J. G. Sobiecki, F. Imamura, C. R. Davis, S. J. Sharp, A. Koulman, J. M. Hodgson, M. Guevara, M. B. Schulze, J. S. Zheng, C. Agnoli, C. Bonet, S. M. Colorado-Yohar, G. Fagherazzi, P. W. Franks, T. E. Gundersen, F. Jannasch, R. Kaaks, V. Katzke, E. Molina-Montes, P. M. Nilsson, D. Palli, S. Panico, K. Papier, O. Rolandsson, C. Sacerdote, A. Tjonneland, T. Y. N. Tong, Y. T. van der Schouw, J. Danesh, A. S. Butterworth, E. Riboli, K. J. Murphy, N. J. Wareham and N. G. Forouhi, A nutritional biomarker score of the Mediterranean diet and incident type 2 diabetes: Integrated analysis of data from the MedLey randomised controlled trial and the EPIC-InterAct case-cohort study, *PLoS Med.*, 2023, **20**, e1004221.
- 30 C. D. Gardner, M. J. Landry, D. Perelman, C. Petlura, L. R. Durand, L. Aronica, A. Crimarco, K. M. Cunanan, A. Chang, C. C. Dant, J. L. Robinson and S. H. Kim, Effect of a ketogenic diet versus Mediterranean diet on glycated hemoglobin in individuals with prediabetes and type 2 diabetes mellitus: The interventional Keto-Med randomized crossover trial, *Am. J. Clin. Nutr.*, 2022, **116**, 640–652.
- 31 H. A. Al-Aubaidy, A. Dayan, M. A. Deseo, C. Itsiopoulos, D. Jamil, N. R. Hadi and C. J. Thomas, Twelve-Week Mediterranean Diet Intervention Increases Citrus Bioflavonoid Levels and Reduces Inflammation in People with Type 2 Diabetes Mellitus, *Nutrients*, 2021, **13**, 1133.
- 32 M. Vitale, M. Masulli, I. Calabrese, A. A. Rivelles, E. Bonora, S. Signorini, G. Perriello, S. Squatrito, R. Buzzetti, G. Sartore, A. C. Babini, G. Gregori, C. Giordano, G. Clemente, S. Grioni, P. Dolce, G. Riccardi, O. Vaccaro and T. I. S. Group, Impact of a Mediterranean Dietary Pattern and Its Components on Cardiovascular Risk Factors, Glucose Control, and Body Weight in People with Type 2 Diabetes: A Real-Life Study, *Nutrients*, 2018, **10**, 1067.
- 33 T. Seres-Noriega, C. Vinals, V. Perea, A. Mesa, L. Boswell, K. Mariaca, J. Blanco, I. Vinagre, A. Pane, C. Milad, C. Sola, E. Esmatjes, I. Conget, M. Gimenez and A. J. Amor, Adherence to an energy-restricted Mediterranean diet is associated with the presence and burden of carotid atherosclerosis in people with type 1 diabetes, *Diabetes Metab. Res. Rev.*, 2024, **40**, e3783.
- 34 S. Montemayor, C. M. Mascaro, L. Ugarriza, M. Casares, I. Llompant, I. Abete, M. A. Zulet, J. A. Martinez, J. A. Tur and C. Bouzas, Adherence to Mediterranean Diet and NAFLD in Patients with Metabolic Syndrome: The FLIPAN Study, *Nutrients*, 2022, **14**, 3186.
- 35 N. Becerra-Tomas, S. Blanco Mejia, E. Viguiliouk, T. Khan, C. W. C. Kendall, H. Kahleova, D. Rahelic, J. L. Sievenpiper and J. Salas-Salvado, Mediterranean diet, cardiovascular disease and mortality in diabetes: A systematic review and meta-analysis of prospective cohort studies and randomized clinical trials, *Crit. Rev. Food Sci. Nutr.*, 2020, **60**, 1207–1227.
- 36 R. Martin-O'Connor, A. Ramos-Levi, V. Melero, M. Arnoriaga-Rodriguez, A. Barabash, J. Valerio, L. Del Valle, P. de Miguel, A. Diaz, C. Familiar, I. Moraga, A. Duran, M. Cuesta, M. J. Torrejon, M. Martinez-Novillo, C. Marcuello, M. Pazos, M. A. Rubio, P. Matia Marin and A. L. Calle-Pascual, Early Mediterranean-Based Nutritional Intervention Reduces the Rate of Gestational Diabetes in Overweight and Obese Pregnant Women: A Post-Hoc Analysis of the San Carlos Gestational Prevention Study, *Nutrients*, 2024, **16**, 2206.
- 37 C. Qu, J. Zhao, J. Lai, X. Wu, P. Huang, T. Zhu, Y. Li, T. Liu, J. Yuan, N. Wang, M. P. Peppelenbosch, H. Chen, B. Xia and J. Qin, Adherence to a Mediterranean diet is associated with a lower risk of diabetic kidney disease among individuals with hyperglycemia: a prospective cohort study, *BMC Med.*, 2024, **22**, 224.
- 38 A. Jayedi, K. Mirzaei, A. Rashidy-Pour, M. S. Yekaninejad, M. S. Zargar and M. R. Akbari Eidgahi, Dietary approaches to stop hypertension, mediterranean dietary pattern, and diabetic nephropathy in women with type 2 diabetes: A case-control study, *Clin. Nutr. ESPEN*, 2019, **33**, 164–170.
- 39 A. Diaz-Lopez, N. Babio, M. A. Martinez-Gonzalez, D. Corella, A. J. Amor, M. Fito, R. Estruch, F. Aros, E. Gomez-Gracia, M. Fiol, J. Lapetra, L. Serra-Majem, J. Basora, F. J. Basterra-Gortari, V. Zanon-Moreno, M. A. Munoz, J. Salas-Salvado and P. S. Investigators, Mediterranean Diet, Retinopathy, Nephropathy, and Microvascular Diabetes Complications: A Post Hoc Analysis of a Randomized Trial, *Diabetes Care*, 2015, **38**, 2134–2141.
- 40 M. Moradi, E. Daneshzad, M. M. Najafabadi, N. Bellissimo, K. Suitor and L. Azadbakht, Association between adherence to the Mediterranean diet and renal function biomarkers and cardiovascular risk factors among diabetic patients with nephropathy, *Clin. Nutr. ESPEN*, 2020, **40**, 156–163.
- 41 A. De Mauri, D. Carrera, M. Vidali, M. Bagnati, R. Rolla, S. Riso, D. Chiarinotti and M. Torreggiani, Does Mediterranean Adequacy Index Correlate with Cardiovascular Events in Patients with Advanced Chronic Kidney Disease? An Exploratory Study, *Nutrients*, 2022, **14**, 1687.
- 42 R. Zamora-Ros, M. Serafini, R. Estruch, R. M. Lamuela-Raventos, M. A. Martinez-Gonzalez, J. Salas-Salvado, M. Fiol, J. Lapetra, F. Aros, M. I. Covas, C. Andres-Lacueva and P. S. Investigators, Mediterranean diet and non enzymatic antioxidant capacity in the PREDIMED study: evidence for a mechanism of antioxidant tuning, *Nutr., Metab. Cardiovasc. Dis.*, 2013, **23**, 1167–1174.
- 43 V. Tosti, B. Bertozzi and L. Fontana, Health Benefits of the Mediterranean Diet: Metabolic and Molecular Mechanisms, *J. Gerontol., Ser. A*, 2018, **73**, 318–326.



- 44 M. I. Maiorino, G. Bellastella, M. Petrizzo, L. Scappaticcio, D. Giugliano and K. Esposito, Mediterranean diet cools down the inflammatory milieu in type 2 diabetes: the MEDITA randomized controlled trial, *Endocrine*, 2016, **54**, 634–641.
- 45 A. Ceriello, K. Esposito, L. La Sala, G. Pujadas, V. De Nigris, R. Testa, L. Bucciarelli, M. Rondinelli and S. Genovese, The protective effect of the Mediterranean diet on endothelial resistance to GLP-1 in type 2 diabetes: a preliminary report, *Cardiovasc. Diabetol.*, 2014, **13**, 140.
- 46 S. C. Naaman and G. L. Bakris, Diabetic Nephropathy: Update on Pillars of Therapy Slowing Progression, *Diabetes Care*, 2023, **46**, 1574–1586.
- 47 H. Oeseburg, R. A. de Boer, H. Buikema, P. van der Harst, W. H. van Gilst and H. H. Sillje, Glucagon-like peptide 1 prevents reactive oxygen species-induced endothelial cell senescence through the activation of protein kinase A, *Arterioscler., Thromb., Vasc. Biol.*, 2010, **30**, 1407–1414.
- 48 A. Ceriello, K. Esposito, R. Testa, A. R. Bonfigli, M. Marra and D. Giugliano, The possible protective role of glucagon-like peptide 1 on endothelium during the meal and evidence for an “endothelial resistance” to glucagon-like peptide 1 in diabetes, *Diabetes Care*, 2011, **34**, 697–702.
- 49 L. Zhao, F. Zhang, X. Ding, G. Wu, Y. Y. Lam, X. Wang, H. Fu, X. Xue, C. Lu, J. Ma, L. Yu, C. Xu, Z. Ren, Y. Xu, S. Xu, H. Shen, X. Zhu, Y. Shi, Q. Shen, W. Dong, R. Liu, Y. Ling, Y. Zeng, X. Wang, Q. Zhang, J. Wang, L. Wang, Y. Wu, B. Zeng, H. Wei, M. Zhang, Y. Peng and C. Zhang, Gut bacteria selectively promoted by dietary fibers alleviate type 2 diabetes, *Science*, 2018, **359**, 1151–1156.
- 50 D. K. Mandaliya and S. Seshadri, Short Chain Fatty Acids, pancreatic dysfunction and type 2 diabetes, *Pancreatol.*, 2019, **19**, 280–284.
- 51 A. Puddu, R. Sanguineti, F. Montecucco and G. L. Viviani, Evidence for the gut microbiota short-chain fatty acids as key pathophysiological molecules improving diabetes, *Mediators Inflammation*, 2014, **2014**, 162021.
- 52 P. Mirtschink, C. Jang, Z. Arany and W. Krek, Fructose metabolism, cardiometabolic risk, and the epidemic of coronary artery disease, *Eur. Heart J.*, 2018, **39**, 2497–2505.
- 53 F. Chicco, S. Magri, A. Cingolani, D. Paduano, M. Pesenti, F. Zara, F. Tumbarello, E. Urru, A. Melis, L. Casula, M. C. Fantini and P. Usai, Multidimensional Impact of Mediterranean Diet on IBD Patients, *Inflammatory Bowel Dis.*, 2021, **27**, 1–9.
- 54 D. Dynka, K. Kowalcze and A. Paziewska, The Role of Ketogenic Diet in the Treatment of Neurological Diseases, *Nutrients*, 2022, **14**, 5003.
- 55 D. Barry, S. Ellul, L. Watters, D. Lee, R. Haluska Jr. and R. White, The ketogenic diet in disease and development, *Int. J. Dev. Neurosci.*, 2018, **68**, 53–58.
- 56 L. Crosby, B. Davis, S. Joshi, M. Jardine, J. Paul, M. Neola and N. D. Barnard, Ketogenic Diets and Chronic Disease: Weighing the Benefits Against the Risks, *Front. Nutr.*, 2021, **8**, 702802.
- 57 M. N. Roberts, M. A. Wallace, A. A. Tomilov, Z. Zhou, G. R. Marcotte, D. Tran, G. Perez, E. Gutierrez-Casado, S. Koike, T. A. Knotts, D. M. Imai, S. M. Griffey, K. Kim, K. Hagopian, M. Z. McMackin, F. G. Haj, K. Baar, G. A. Cortopassi, J. J. Ramsey and J. A. Lopez-Dominguez, A Ketogenic Diet Extends Longevity and Healthspan in Adult Mice, *Cell Metab.*, 2017, **26**, 539–546.
- 58 J. C. Newman, A. J. Covarrubias, M. Zhao, X. Yu, P. Gut, C. P. Ng, Y. Huang, S. Haldar and E. Verdin, Ketogenic Diet Reduces Midlife Mortality and Improves Memory in Aging Mice, *Cell Metab.*, 2017, **26**, 547–557.
- 59 M. Ferrer, N. Mourikis, E. E. Davidson, S. O. Kleeman, M. Zaccaria, J. Habel, R. Rubino, Q. Gao, T. R. Flint, L. Young, C. M. Connell, M. J. Lukey, M. D. Goncalves, E. P. White, A. R. Venkitaraman and T. Janowitz, Ketogenic diet promotes tumor ferroptosis but induces relative corticosterone deficiency that accelerates cachexia, *Cell Metab.*, 2023, **35**, 1147–1162.
- 60 P. Puchalska and P. A. Crawford, Multi-dimensional Roles of Ketone Bodies in Fuel Metabolism, Signaling, and Therapeutics, *Cell Metab.*, 2017, **25**, 262–284.
- 61 J. A. Torres, S. L. Kruger, C. Broderick, T. AmaralKhagva, S. Agrawal, J. R. Dodam, M. Mrug, L. A. Lyons and T. Weimbs, Ketosis Ameliorates Renal Cyst Growth in Polycystic Kidney Disease, *Cell Metab.*, 2019, **30**, 1007–1023.
- 62 S. Kumar, T. Behl, M. Sachdeva, A. Sehgal, S. Kumari, A. Kumar, G. Kaur, H. N. Yadav and S. Bungau, Implicating the effect of ketogenic diet as a preventive measure to obesity and diabetes mellitus, *Life Sci.*, 2021, **264**, 118661.
- 63 C. Zhou, M. Wang, J. Liang, G. He and N. Chen, Ketogenic Diet Benefits to Weight Loss, Glycemic Control, and Lipid Profiles in Overweight Patients with Type 2 Diabetes Mellitus: A Meta-Analysis of Randomized Controlled Trials, *Int. J. Environ. Res. Public Health*, 2022, **19**, 10429.
- 64 S. Li, G. Lin, J. Chen, Z. Chen, F. Xu, F. Zhu, J. Zhang and S. Yuan, The effect of periodic ketogenic diet on newly diagnosed overweight or obese patients with type 2 diabetes, *BMC Endocr. Disord.*, 2022, **22**, 34.
- 65 A. Basolo, S. Magno, F. Santini and G. Ceccarini, Ketogenic Diet and Weight Loss: Is There an Effect on Energy Expenditure?, *Nutrients*, 2022, **14**, 1814.
- 66 F. Brouns, Overweight and diabetes prevention: is a low-carbohydrate-high-fat diet recommendable?, *Eur. J. Nutr.*, 2018, **57**, 1301–1312.
- 67 American Diabetes Association, Standards of medical care in diabetes–2013, *Diabetes Care*, 2013, **36**(Suppl 1), S11–S66.
- 68 A. Paoli, L. Mancin, A. Bianco, E. Thomas, J. F. Mota and F. Piccini, Ketogenic Diet and Microbiota: Friends or Enemies?, *Genes*, 2019, **10**, 534.
- 69 A. Koliada, G. Syzenko, V. Moseiko, L. Budovska, K. Puchkov, V. Perederiy, Y. Gavalko, A. Dorofeyev, M. Romanenko, S. Tkach, L. Sineok, O. Lushchak and A. Vaiserman, Association between body mass index and



- Firmicutes/Bacteroidetes ratio in an adult Ukrainian population, *BMC Microbiol.*, 2017, **17**, 120.
- 70 C. E. Forsythe, S. D. Phinney, M. L. Fernandez, E. E. Quann, R. J. Wood, D. M. Bibus, W. J. Kraemer, R. D. Feinman and J. S. Volek, Comparison of low fat and low carbohydrate diets on circulating fatty acid composition and markers of inflammation, *Lipids*, 2008, **43**, 65–77.
 - 71 I. Tomita, S. Kume, S. Sugahara, N. Osawa, K. Yamahara, M. Yasuda-Yamahara, N. Takeda, M. Chin-Kanasaki, T. Kaneko, E. Mayoux, M. Mark, M. Yanagita, H. Ogita, S. I. Araki and H. Maegawa, SGLT2 Inhibition Mediates Protection from Diabetic Kidney Disease by Promoting Ketone Body-Induced mTORC1 Inhibition, *Cell Metab.*, 2020, **32**, 404–419.
 - 72 T. Greco, T. C. Glenn, D. A. Hovda and M. L. Prins, Ketogenic diet decreases oxidative stress and improves mitochondrial respiratory complex activity, *J. Cereb. Blood Flow Metab.*, 2016, **36**, 1603–1613.
 - 73 V. J. Miller, R. A. LaFountain, E. Barnhart, T. S. Sapper, J. Short, W. D. Arnold, P. N. Hyde, C. D. Crabtree, M. L. Kackley, W. J. Kraemer, F. A. Villamena and J. S. Volek, A ketogenic diet combined with exercise alters mitochondrial function in human skeletal muscle while improving metabolic health, *Am. J. Physiol. Endocrinol. Metab.*, 2020, **319**, E995–E1007.
 - 74 W. S. Yancy, Jr., M. Foy, A. M. Chalecki, M. C. Vernon and E. C. Westman, A low-carbohydrate, ketogenic diet to treat type 2 diabetes, *Nutr. Metab.*, 2005, **2**, 34.
 - 75 Z. Guo, F. Zhong, M. Hou, J. Xie, A. Z. Zhang, X. Li, Y. Li, B. Chang and J. Yang, Key enzyme in charge of ketone reabsorption of renal tubular SMCT1 may be a new target in diabetic kidney disease, *Nephrol., Dial., Transplant.*, 2023, **38**, 2754–2766.
 - 76 M. M. Poplawski, J. W. Mastaitis, F. Isoda, F. Grosjean, F. Zheng and C. V. Mobbs, Reversal of diabetic nephropathy by a ketogenic diet, *PLoS One*, 2011, **6**, e18604.
 - 77 S. R. Wan, F. Y. Teng, W. Fan, B. T. Xu, X. Y. Li, X. Z. Tan, M. Guo, C. L. Gao, C. X. Zhang, Z. Z. Jiang and Y. Xu, BDH1-mediated betaOHB metabolism ameliorates diabetic kidney disease by activation of NRF2-mediated anti-oxidative pathway, *Aging*, 2023, **15**, 13384–13410.
 - 78 A. N. Friedman, M. Chambers, L. M. Kamendulis and J. Temmerman, Short-term changes after a weight reduction intervention in advanced diabetic nephropathy, *Clin. J. Am. Soc. Nephrol.*, 2013, **8**, 1892–1898.
 - 79 G. J. Ko, C. M. Rhee, K. Kalantar-Zadeh and S. Joshi, The Effects of High-Protein Diets on Kidney Health and Longevity, *J. Am. Soc. Nephrol.*, 2020, **31**, 1667–1679.
 - 80 X. Tong, Y. Deng, L. Liu, X. Tang, T. Yu, J. Gan, Q. Cai, R. Luo and N. Xiao, Clinical implementation of ketogenic diet in children with drug-resistant epilepsy: Advantages, disadvantages, and difficulties, *Seizure*, 2022, **99**, 75–81.
 - 81 B. G. Allen, S. K. Bhatia, C. M. Anderson, J. M. Eichenberger-Gilmore, Z. A. Sibenaller, K. A. Mapuskar, J. D. Schoenfeld, J. M. Buatti, D. R. Spitz and M. A. Fath, Ketogenic diets as an adjuvant cancer therapy: History and potential mechanism, *Redox Biol.*, 2014, **2**, 963–970.
 - 82 D. Tinguely, J. Gross and C. Kosinski, Efficacy of Ketogenic Diets on Type 2 Diabetes: a Systematic Review, *Curr. Diabetes Rep.*, 2021, **21**, 32.
 - 83 D. Tang, Q. Tang, W. Huang, Y. Zhang, Y. Tian and X. Fu, Fasting: From Physiology to Pathology, *Adv. Sci.*, 2023, **10**, e2204487.
 - 84 K. A. Varady, S. Cienfuegos, M. Ezpeleta and K. Gabel, Clinical application of intermittent fasting for weight loss: progress and future directions, *Nat. Rev. Endocrinol.*, 2022, **18**, 309–321.
 - 85 H. Kord-Varkaneh, A. Salehi-Sahlabadi, G. M. Tinsley, H. O. Santos and A. Hekmatdoost, Effects of time-restricted feeding (16/8) combined with a low-sugar diet on the management of non-alcoholic fatty liver disease: A randomized controlled trial, *Nutrition*, 2023, **105**, 111847.
 - 86 S. Gallage, A. Ali, J. E. Barragan Avila, N. Seymen, P. Ramadori, V. Joerke, L. Zizmare, D. Aicher, I. K. Gopalsamy, W. Fong, J. Kosla, E. Focaccia, X. Li, S. Yousuf, T. Sijmonsma, M. Rahbari, K. S. Kommos, A. Billeter, S. Prokosch, U. Rothermel, F. Mueller, J. Hetzer, D. Heide, B. Schinkel, T. Machauer, B. Pichler, N. P. Malek, T. Longerich, S. Roth, A. J. Rose, J. Schwenck, C. Trautwein, M. M. Karimi and M. Heikenwalder, A 5 : 2 intermittent fasting regimen ameliorates NASH and fibrosis and blunts HCC development via hepatic PPARalpha and PCK1, *Cell Metab.*, 2024, **36**, 1371–1393.
 - 87 J. Kang, X. Shi, J. Fu, H. Li, E. Ma and W. Chen, Effects of an Intermittent Fasting 5 : 2 Plus Program on Body Weight in Chinese Adults with Overweight or Obesity: A Pilot Study, *Nutrients*, 2022, **14**, 4734.
 - 88 E. Duregon, M. E. Fernandez, J. Martinez Romero, C. Di Germanio, M. Cabassa, R. Voloshchuk, M. R. Ehrlich-Mora, J. M. Moats, S. Wong, O. Bosomptra, A. Rudderow, C. H. Morrell, S. Camandola, N. L. Price, M. A. Aon, M. Bernier and R. de Cabo, Prolonged fasting times reap greater geroprotective effects when combined with caloric restriction in adult female mice, *Cell Metab.*, 2023, **35**, 1179–1194.
 - 89 O. Strilbytska, S. Klishch, K. B. Storey, A. Koliada and O. Lushchak, Intermittent fasting and longevity: From animal models to implication for humans, *Ageing Res. Rev.*, 2024, **96**, 102274.
 - 90 S. Y. Chair, H. Cai, X. Cao, Y. Qin, H. Y. Cheng and M. T. Ng, Intermittent Fasting in Weight Loss and Cardiometabolic Risk Reduction: A Randomized Controlled Trial, *J. Nurs. Res.*, 2022, **30**, e185.
 - 91 A. Parvaresh, R. Razavi, B. Abbasi, K. Yaghoobloo, A. Hassanzadeh, N. Mohammadifard, S. M. Safavi, A. Hadi and C. C. T. Clark, Modified alternate-day fasting vs. calorie restriction in the treatment of patients with metabolic syndrome: A randomized clinical trial, *Complement. Ther. Med.*, 2019, **47**, 102187.



- Food Funct., 2025, 16, 2622–2636 | 2635

- and K. Mirzaei, The association of dietary approaches to stop hypertension (DASH) with the odds of diabetic nephropathy and metabolic markers in women: a case-control study, *BMC Women's Health*, 2023, **23**, 63.
- 114 S. M. Hargreaves, D. L. Rosenfeld, A. V. B. Moreira and R. P. Zandonadi, Plant-based and vegetarian diets: an overview and definition of these dietary patterns, *Eur. J. Nutr.*, 2023, **62**, 1109–1121.
 - 115 C. Agnoli, L. Baroni, I. Bertini, S. Ciappellano, A. Fabbri, S. Goggi, D. Metro, M. Papa, R. Sbarbati, M. L. Scarino, N. Pellegrini and S. Sieri, A comprehensive review of healthy effects of vegetarian diets, *Nutr., Metab. Cardiovasc. Dis.*, 2023, **33**, 1308–1315.
 - 116 P. D. Lopez, E. H. Cativo, S. A. Atlas and C. Rosendorff, The Effect of Vegan Diets on Blood Pressure in Adults: A Meta-Analysis of Randomized Controlled Trials, *Am. J. Med.*, 2019, **132**, 875–883.
 - 117 L. Hardt, Y. Mahamat-Saleh, D. Aune and S. Schlesinger, Plant-Based Diets and Cancer Prognosis: a Review of Recent Research, *Curr. Nutr. Rep.*, 2022, **11**, 695–716.
 - 118 F. Liang, J. Fu, G. Turner-McGrievy, Y. Wang, N. Qiu, K. Ding, J. Zeng, J. B. Moore and R. Li, Association of Body Mass Index and Plant-Based Diet with Cognitive Impairment among Older Chinese Adults: A Prospective, Nationwide Cohort Study, *Nutrients*, 2022, **14**, 3132.
 - 119 A. D. Termannsen, C. S. Sondergaard, K. Faerch, T. H. Andersen, A. Raben and J. S. Quist, Effects of Plant-Based Diets on Markers of Insulin Sensitivity: A Systematic Review and Meta-Analysis of Randomised Controlled Trials, *Nutrients*, 2024, **16**, 2110.
 - 120 S. Tonstad, K. Stewart, K. Oda, M. Batech, R. P. Herring and G. E. Fraser, Vegetarian diets and incidence of diabetes in the Adventist Health Study-2, *Nutr., Metab. Cardiovasc. Dis.*, 2013, **23**, 292–299.
 - 121 H. Kahleova, K. F. Petersen, G. I. Shulman, J. Alwarith, E. Rembert, A. Tura, M. Hill, R. Holubkov and N. D. Barnard, Effect of a Low-Fat Vegan Diet on Body Weight, Insulin Sensitivity, Postprandial Metabolism, and Intramyocellular and Hepatocellular Lipid Levels in Overweight Adults: A Randomized Clinical Trial, *JAMA Netw. Open*, 2020, **3**, e2025454.
 - 122 H. Kahleova, M. Matoulek, H. Malinska, O. Oliyarnik, L. Kazdova, T. Neskudla, A. Skoch, M. Hajek, M. Hill, M. Kahle and T. Pelikanova, Vegetarian diet improves insulin resistance and oxidative stress markers more than conventional diet in subjects with Type 2 diabetes, *Diabetic Med.*, 2011, **28**, 549–559.
 - 123 C. A. Mocanu, T. P. Simionescu, A. E. Mocanu and L. Garneata, Plant-Based versus Animal-Based Low Protein Diets in the Management of Chronic Kidney Disease, *Nutrients*, 2021, **13**, 3721.
 - 124 M. M. Jibani, L. L. Bloodworth, E. Foden, K. D. Griffiths and O. P. Galpin, Predominantly vegetarian diet in patients with incipient and early clinical diabetic nephropathy: effects on albumin excretion rate and nutritional status, *Diabetic Med.*, 1991, **8**, 949–953.
 - 125 A. Perez-Torres, A. Caverni-Munoz and E. Gonzalez Garcia, Mediterranean Diet and Chronic Kidney Disease (CKD): A Practical Approach, *Nutrients*, 2022, **15**, 97.
 - 126 S. Kundu, K. S. Hossain, A. Moni, M. S. Zahan, M. M. Rahman and M. J. Uddin, Potentials of ketogenic diet against chronic kidney diseases: pharmacological insights and therapeutic prospects, *Mol. Biol. Rep.*, 2022, **49**, 9749–9758.
 - 127 M. Charkviani, C. Thongprayoon, S. Tangpanithandee, P. Krisanapan, J. Miao, M. A. Mao and W. Cheungpasitporn, Effects of Mediterranean Diet, DASH Diet, and Plant-Based Diet on Outcomes among End Stage Kidney Disease Patients: A Systematic Review and Meta-Analysis, *Clin. Pract.*, 2022, **13**, 41–51.
 - 128 A. K. Bello, J. Kurzawa, M. A. Osman, M. E. Olah, A. Lloyd, N. Wiebe, S. Habib, U. Qarni, S. Shojai and R. P. Pauly, Impact of Ramadan fasting on kidney function and related outcomes in patients with chronic kidney disease: a systematic review protocol, *BMJ Open*, 2019, **9**, e022710.
 - 129 A. Gluba-Brzozka, B. Franczyk and J. Rysz, Vegetarian Diet in Chronic Kidney Disease-A Friend or Foe, *Nutrients*, 2017, **9**, 374.
 - 130 M. Deng, X. Li, W. Li, J. Gong, X. Zhang, S. Ge and L. Zhao, Short-Chain Fatty Acids Alleviate Hepatocyte Apoptosis Induced by Gut-Derived Protein-Bound Uremic Toxins, *Front. Nutr.*, 2021, **8**, 756730.
 - 131 F. Wang, C. Liu, L. Ren, Y. Li, H. Yang, Y. Yu and W. Xu, Sanziguben polysaccharides improve diabetic nephropathy in mice by regulating gut microbiota to inhibit the TLR4/NF-kappaB/NLRP3 signalling pathway, *Pharm. Biol.*, 2023, **61**, 427–436.
 - 132 P. Angoorani, H. S. Ejtahed, S. Hasani-Ranjbar, S. D. Siadat, A. R. Soroush and B. Larijani, Gut microbiota modulation as a possible mediating mechanism for fasting-induced alleviation of metabolic complications: a systematic review, *Nutr. Metab.*, 2021, **18**, 105.
 - 133 J. Lee, J. Lee, K. Kim, J. Lee, Y. Jung, J. S. Hyeon, A. Seo, W. Jin, B. Weon, N. Shin, S. Kim, C. S. Lim, Y. S. Kim, J. P. Lee, G. S. Hwang and S. H. Yang, Antibiotic-induced intestinal microbiota depletion can attenuate the acute kidney injury to chronic kidney disease transition via NADPH oxidase 2 and trimethylamine-N-oxide inhibition, *Kidney Int.*, 2024, **105**, 1239–1253.

