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Regulation of Specific Dietary Patterns on Inflammatory and Oxidative Pathways in Type 2 Diabetes Mellitus

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Abstract. In the past few decades, type 2 diabetes mellitus (T2DM) has developed into a health burden to the global population. Current evidence shows that hyperglycemia alone is insufficient to explain the development of diabetic complications. Chronic inflammation and oxidative stress also play an important role in exacerbating insulin resistance and damaging target-organs. This review discusses the role of inflammation and oxidative stress in T2DM pathogenesis and the impacts of dietary patterns and components on those pathways, including the use of biomarkers in clinical dietary intervention studies. The pathogenic network of dysregulation of immunity in adipose tissue, intercellular communication mediated by small extracellular vesicles, the imbalance of the adipokine network, and impaired insulin signaling constitutes the pathogenic network driving the disease progression. Dietary intervention strategies, such as the Mediterranean diet, n-6 polyunsaturated fatty acids, dietary fibers, short-chain acids, oat β-glucan, L-arabinose, and polyphenols, work together in unique but complementary ways to regulate the network. At the same time, numerous clinical studies support the advantages of this network in managing glycemic levels, weight, and cardiovascular risks. Collectively, these findings provide the foundation for targeted dietary intervention and provide practical advice for individualized strategies tailoring specific inflammatory and oxidative profiles of T2DM patients.

Keywords: mediterranean diet, oxidative stress, type 2 diabetes mellitus, IFN-γ

1. Introduction

In the current era, diabetes has become increasingly prevalent globally. The estimated number of people with diabetes rose from approximately 151 million in 2000 to over 589 million in 2025 (according to International Diabetes Federation Atlas 1st editions) [1]. Type 2 diabetes mellitus (T2DM) accounted for 90% of all cases, and it experienced the greatest increase in number. This trend is closely related to the global transition to a sedentary lifestyle and the consumption of ultra-processed foods in daily life. T2DM, characterized by hyperglycemia induced by insulin resistance, often results in damage to the heart and kidneys, and these injuries are difficult to control effectively. Although the glycated hemoglobin level can be reduced by glucose-lowering therapy, evidence from the real world shows limitations of this strategy [2]. Even when glycemic targets are achieved, T2DM patients still experience significant residual risks. These risks include both macrovascular

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events (myocardial infarction, stroke) and microvascular complications (nephrosis, retinopathy). Strict glycemic control has not only failed to achieve the anticipated amelioration in all-cause mortality and cardiovascular outcomes but also faced difficulties in preventing disease progression into the terminal stage. These clinical issues indicate that hyperglycemia should not be considered the sole driver of progression of T2DM complications. Therefore, purely glucocentric strategies which focus on glycemic control are insufficient to prevent malignant disease progression. It further highlights how necessary it is to incorporate the regulation of inflammation and oxidative stress into the management framework. However, currently, a comprehensive disease-intervention strategy remains absent.

At the same time, a growing body of evidence indicates that a chronic inflammation state and oxidative stress have independent and essential roles in insulin resistance exacerbation and target-organ damage. Nevertheless, these pathways are not sensitive to conventional glucose-lowering medicine. Therefore, a method that integrates precise dietary modulation with oxidative and inflammatory biomarkers should be established, to develop an individualized preventive framework for patients.

Considering these unresolved challenges and new opportunities, this review first systematically elucidates the mechanisms of inflammation and oxidative stress in T2DM. Second, it synthesizes evidence on the effects of various dietary patterns and specific nutrients in regulating these pathways. Last, it summarizes the biomarkers and indicators employed in clinical studies of dietary interventions. Accordingly, a reference for establishing individualized prevention strategies is provided.

2. Pathophysiological mechanisms of diabetes inflammation and oxidative stress

Present research has investigated different aspects of inflammation and oxidative stress in diabetes mellitus. However, there are no comprehensive reviews of these studies focused on the pathways of inflammation and oxidative stress in T2DM, and the relationship between various pathways has not been elucidated clearly. This chapter aims to outline the main pathogenic aspects of inflammation and oxidative stress by focusing on adipose tissue immune microenvironment alteration, small extracellular vesicles' role in communication, the pathological impact of key adipokines, and dysregulation of insulin signaling pathways. This overview intends to provide essential background for subsequent discussions on screening for biomarkers and establishing intervention strategies.

Visceral adipose tissue (VAT), once regarded as an energy reservoir, is now considered a pivotal part of the T2DM inflammation pathway. As pointed out by Wensveen et al., an essential cause of T2DM progression and insulin resistance is the accumulation of pro-inflammatory cells in VAT [3]. Under healthy conditions, regulatory cells (type 2 innate lymphocytes, eosinophils) constitute the main population of lean adipose tissue. Through secretion of type 2 cytokines (interleukin-4 (IL-4), interleukin-5 (IL-5), interleukin-13 (IL-13)), they maintain adipose tissue macrophages (ATMs) in an M2-like state, an anti-inflammatory state. This effect maintains tissue homeostasis. However, high-fat diet-induced adipocyte hypertrophy triggers cellular stress. This stress activates cells' ligands to produce interferon-γ (IFN-γ), a characteristic of the development of type 1 inflammatory responses in VAT, and initiates inflammation. Such a phenomenon is frequently found in diet-induced obesity. This suggests that VAT inflammation processes targeting strategy could be a future promising method for T2DM progression prevention.

Beyond the impact of changes in the microenvironment of VAT, according to the review by Chen et al., small extracellular vesicles (sEVs) that originate from adipose tissue for intercellular communications are also involved in T2DM pathogenesis [4]. In T2DM progression and

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management, these molecules play a complex role. Through extraction and testing of sEVs from distinct sources, the researchers concluded that VAT-derived sEVs contribute significantly to the progression and induction of insulin resistance. Meanwhile, sEVs from adipose-derived stem cells (ADSCs) enhance insulin sensitivity through their bioactive components such as microRNAs. This finding proves their therapeutic potential in treating diabetes.

Apart from this, adipokine network dysregulation is another important pathogenic component. The review by Yuan et al. focuses on asprosin, a kind of glucogenic protein produced by white adipose tissue when fasting [5]. This protein plays complicated roles in different body parts, while it is also responsible for metabolic diseases. For example, it promotes hepatic glucose output through activation of the cyclic adenosine monophosphate (cAMP)–protein kinase A (PKA) pathway in the liver. It also triggers endoplasmic reticulum stress and inflammation through the protein kinase Cδ (PKCδ)/sarco/endoplasmic reticulum Ca^{2+}-ATPase 2 (SERCA2) axis in skeletal muscle. Notably, in patients with T2DM and obesity, elevated circulating asprosin levels have been discovered. This elevation also has a positive association with homeostatic model assessment for insulin resistance (HOMA-IR), an index for insulin resistance that is negatively related to homeostasis model assessment for β-cell function. This suggests that asprosin can be used as a biomarker connecting inflammation, oxidative stress and T2DM.

Insulin resistance itself is another critical factor in the development of T2DM. Since insulin resistance is an important pathological characteristic of T2DM, treating it has proved to be a way to prevent and reverse T2DM. Nonetheless, hyperinsulinemia, a situation in which there is an abnormal rise in plasma insulin levels, brings adverse effects. These adverse effects include impaired insulin action, reduced ability to lower blood glucose levels, yet preserved capability for lipid synthesis and lipoprotein secretion. These results prove that solving insulin sensitization problems is necessary. Selective inhibition and activation of certain factors, as explained by Accili et al., result in selective insulin sensitization. This finding provides insights into the treatment of T2DM [6].

In conclusion, inflammatory and oxidative stress-based pathways responsible for the development of T2DM are not isolated processes. In fact, this complicated network consists of factors from multiple layers. These factors include local immune dysregulation of adipose tissue, long-range communication through small extracellular vesicles, imbalance of the adipokine network, and transduction of impaired insulin signaling. Through their interaction, they continuously intensify insulin resistance and induce vascular endothelial injury and target organ dysfunction. The analysis of key nodes in the network is useful for searching for new biomarkers with potential clinical value. At the same time, this review serves as a specific foundation for future personalized nutrition intervention strategies based on inflammation and oxidative stress.

3. Dietary nutrition regulation of inflammation and oxidative stress in diabetes

Dietary patterns and nutrient composition are the key regulatory factors of inflammation and oxidative stress. They are indispensable to T2DM initiation and progression. Although previous research has concentrated on the direct effect of specific nutrients or foods on glycemic fluctuations, a systematic and integrated understanding of how various dietary patterns and nutrients intervene in disease progression through inflammatory and oxidative stress pathways remains insufficient, with findings of research being fragmented. Therefore, this chapter aims to explore two perspectives: dietary patterns and dietary components. By reviewing the anti-inflammatory and anti-oxidative molecular mechanisms of dietary patterns and specific bioactive dietary components including unsaturated fatty acids, dietary fibers, short chain fatty acids, β-glucan, and polyphenols, this review

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provides a mechanistic theoretical foundation for the precise dietary intervention strategies for T2DM.

In a review, Scaglione et al. synthesized numerous influences of the Mediterranean diet on the metabolic syndrome components and related cardiovascular risks [7]. The Mediterranean diet, as noted in the review, is mainly characterized by the abundance of olive oil. It also includes rich plant-based foods and fish. Ample polyphenols (such as oleuropein, resveratrol, and naringenin), monounsaturated fatty acids (particularly oleic acid), ω-3 polyunsaturated fatty acids and dietary fibers inhibit the nuclear factor κB signaling, reduce pro-inflammatory adipocytokines (tumor necrosis factor-α [TNF-α], interleukin-6 [IL-6]), and reduce reactive oxygen species generation and lipid peroxidation, in order to alleviate the oxidative stress of pancreatic β-cells and improve peripheral insulin sensitivity. Moreover, the review presents numerous clinical studies. The PREDIMED trial showed that elderly individuals at high risk of cardiovascular problems experienced a 52% reduction in diabetes risk when following the Mediterranean diet compared with those following a low-fat diet. The ATTIVA study found that in participants with and without diabetes following the Mediterranean diet, basal glucose and insulin levels were reduced by 15%, while the HOMA-IR was improved by 27%. This review integrates both molecular mechanisms and clinical evidence, providing a scientific foundation demonstrating how the Mediterranean diet improves T2DM-related metabolic disorders through multiple effects including anti-inflammatory effects, antioxidant activity, and insulin sensitization.

The CORDIOPREV trial provided another piece of robust clinical evidence. Conducted by Delgado-Lista et al, this trial was single-centered and randomized controlled, lasting for 7 years. The researchers aimed to find the difference in how the Mediterranean diet and a low-fat diet prevent recurrent cardiovascular problems [8]. 1002 patients were enrolled in the trial. They were assigned to the Mediterranean diet group and the low-fat diet group in relatively equal numbers (502 and 500). High-intensity dietary intervention, regular contact, and group sessions were provided to both groups by dietitians. The primary outcomes were major cardiovascular events. The results indicated the primary endpoint was observed in 87 patients in the Mediterranean diet group, compared with 111 patients in the low-fat diet group. The multivariable-adjusted hazard ratios ranged from 0.719 to 0.753 across different models, all favoring the Mediterranean diet. In the male subgroup, the superiority of the Mediterranean diet was even clearer, with primary endpoint event rates of 16.2% versus 22.8%, whereas in the female subgroup there was no statistically significant difference. Through the large sample size, long follow-up duration, and intense intervention design, the trial demonstrated in a secondary prevention population that the Mediterranean diet provided an approximately 26% reduction in the hazard ratio of major cardiovascular events compared with a low-fat diet. This finding further proves the previous point that, through the synergistic anti-inflammatory and antioxidant mechanisms arising from its rich content of polyphenolic compounds, monounsaturated fatty acids, and dietary fibers, the Mediterranean diet can ameliorate the progression of T2DM.

Apart from dietary patterns, individual dietary components' roles in regulating inflammation and oxidative stress have also been extensively explored. A 10-week randomized parallel trial was conducted by Bjermo et al., which aimed to compare how n-6 polyunsaturated fatty acids (PUFAs) and saturated fatty acids (SFAs) influence the liver fat content and systemic inflammation in individuals with abdominal obesity [9]. The trial enrolled 67 abdominally obese individuals, with 15% having T2DM. They were randomly assigned to either a diet group rich in vegetable-derived n-6 PUFAs or a diet group with butter as the main SFA source. Both diets were isocaloric and consistent in macronutrient intake proportions. The primary outcome was the difference in hepatic

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fat content assessed by magnetic resonance imaging (MRI) and magnetic resonance spectroscopy (MRS). Secondary outcomes included serum proprotein convertase subtilisin/kexin type 9 (PCSK9) concentrations, lipid profiles, insulin sensitivity, and multiple inflammatory markers. The results demonstrated that, compared with patients in the SFA diet group, patients in the n-6 PUFA diet intervention group experienced significantly greater reductions in liver fat content. The difference between groups in relative change was 16% by MRI and 34% by MRS. Regarding inflammation markers, serum PCSK9 concentrations, TNF receptor-2 concentrations, and IL-1 receptor antagonist concentrations were lower in the PUFA patients than in the SFA patients. Among compliant subjects, significantly lower insulin, total cholesterol to high-density lipoprotein (HDL) cholesterol ratio, low-density lipoprotein (LDL) cholesterol, and triglycerides were observed in the PUFA group than in the SFA group, while adipose tissue expression between the two groups was not significantly different. Through a rigorous randomized controlled design and highly precise imaging assessment, the trial was the first to reveal in humans that, even without weight loss, n-6 PUFA intake effectively reduces liver fat content and improves systemic inflammation status. Simultaneously, the trial provided counterevidence against the pro-inflammatory effects of n-6 PUFAs hypothesized previously, offering high-quality clinical evidence for the modulation of T2DM-related inflammatory pathways by dietary fatty acid composition.

Ferrer et al. underscored the potential of dietary fibers and their downstream metabolites in regulating inflammation and oxidative stress [10]. The researchers performed an in vitro experiment using human colonic epithelial Caco-2/TC7 cells, aiming to investigate the protective effect of short chain fatty acids (SCFAs) against oxidative stress induced by TNF-α in the intestine. SCFAs are byproducts of fermentation of dietary fibers by gut microbiota [10]. During the experiment, fully differentiated Caco-2/TC7 cells were treated with 1 mM of three kinds of SCFAs (acetate, butyrate, propionate) or a mixture of the three (at a 60:25:15 ratio to simulate physiological conditions) for 24 hours, with 1 hour of 5 ng/mL TNF-α pretreatment to mimic inflammation. The primary outcome indicators were lipid peroxidation levels (malondialdehyde (MDA) and 4-hydroxyalkenals (4-HAD)), protein oxidation levels (carbonylated species concentrations), and antioxidant enzyme activities (catalase (CAT), superoxide dismutase (SOD), glutathione peroxidase (GPx)). None of the SCFA treatments significantly affected oxidative stress levels or antioxidant enzyme activities under basal conditions, according to the results. However, under an inflammatory condition induced by TNF-α, the level of lipid peroxidation was almost double that in the control group, and both CAT and GPx activities were significantly inhibited, while protein oxidation and SOD activity remained unchanged. The levels of lipid peroxidation in the acetate, propionate, butyrate and combined-treatment groups were reduced to near-control levels. Both CAT and GPx activities were increased in all treatment groups, but only propionate significantly enhanced the recovery of CAT activity. The above results indicate that reduced production of SCFAs due to gut microbiota dysbiosis may be a cause of oxidative damage. Therefore, the role of SCFAs in mitigating intestinal oxidative stress and regulating oxidative stress-related diseases has been revealed

The synergistic effect of dietary components was also studied. Wang et al. have provided some experimental data on this effect [11]. The researchers first screened different groups of insulin resistant HepG2 cell models, each with a distinct ratio (ranging from 1:10 to 10:1) of oat β-glucan (OBG) to L-arabinose (LA). They found that the 1:1 ratio group had the most pronounced synergistic effect on glucose uptake. This group was then selected for in vivo experiments. In db/db mice, the combined intervention group showed a significant reduction in fasting blood glucose (27.14 vs 33.73 mmol/L, $P<0.01$) compared to the model group, and the area under the curve of the oral glucose tolerance test was also reduced significantly ($P<0.05$). Lower HOMA-IR was found in

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the combined intervention group than in either of the single-intervention groups. This finding revealed that the combined intervention had additional benefits in improving glucose metabolism and insulin sensitivity. In terms of mechanisms, the combined intervention significantly increased the messenger RNA (mRNA) and protein expression levels of insulin receptor substrate 1 (IRS1), phosphoinositide 3-kinase (PI3K), protein kinase B (AKT), and glucose transporter type 4 (GLUT4). At the same time p-IRS1 levels were reduced and p-AKT levels were increased. This indicated that the combination may promote positive modulation of the PI3K/AKT signaling pathway to alleviate hepatic insulin resistance. In addition, the combination of the two components was more effective than either one alone in improving lipid levels and reducing hepatic steatosis and inflammation. In short, this study has shown that the combination of the two components is effective and has identified some molecular reasons for this synergy. The above results provided a theoretical basis for the combination of OBG and LA in functional foods and offered a new approach to precise matching of dietary components in dietary intervention for T2DM.

In another comprehensive review, Bellanti et al. further explored the role of dietary polyphenols in inflammation and oxidative modulation through regulation of redox-sensitive signaling pathways [12]. The review synthesized clinical evidence that indicated polyphenolic compounds (curcumin and resveratrol) exert their anti-inflammatory and anti-oxidative effects mainly through nuclear factor erythroid 2-related factor 2 (Nrf2) activation and nuclear factor kappa B (NF-κB) inhibition. Regarding the mechanisms, polyphenols promote the nuclear translocation of Nrf2, which leads to increased transcription of cytoprotective genes such as heme oxygenase-1 (HO-1), nicotinamide adenine dinucleotide phosphate (NAD(P)H) quinone dehydrogenase 1 (NQO1), and glutamate-cysteine ligase catalytic subunit (GCLC), thus enhancing antioxidant defenses. At the same time, NF-kB activation was suppressed, which caused pro-inflammatory cytokine (TNF-α, interleukin-1β (IL-1β), IL-6) expression to be reduced. The authors also pointed out that regularly consuming green tea rich in epigallocatechin gallate was shown to be associated with reduced inflammation and improved cardiovascular health. Some spice combinations including curcumin and black pepper showed the potential to improve bioavailability. The review also highlighted that although there are many health benefits associated with dietary polyphenols, their effectiveness might be affected by bioavailability and individual metabolic responses. Therefore, an individualized dietary intervention could be beneficial in improving therapeutic results.

In summary, dietary nutrition is highly significant in controlling and improving T2DM-related inflammation and oxidative stress. Holistic dietary patterns combine different nutrients to suppress inflammatory pathways and control oxidative damage systematically, whereas different dietary components suppress insulin resistance and improve target organ function through synergistic effects and multiple molecular pathways. Identification of major nodes in this network of diets not only helps to identify dietary targets but also lays a practical foundation for creating nutritional strategies for T2DM patients based on their specific inflammatory and oxidative profile (Table 1).

Table 1. Summary of dietary components and their mechanisms in T2DM

Component Name	Key Mechanism	Key outcomes
n-6 PUFAs	Downregulate PCSK9 Suppress TNF-α and IL-1β pathways	Reduce liver fat Improve lipid profile
Dietary Fibers and SFAs	Upregulate CAT and GPx activity Restore redox balance	Reverse lipid peroxidation

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Table 1. (continued)

Oat β-glucan and L-arabinos	Activate PI3K/AKT pathway Upregulate IRS1, PI3K, AKT, GLUT4 Suppress IRS1 phosphorylation	Improve insulin resistance Reduce hepatic steatosis Reduce hepatic inflammation
Polyphenols	Activate Nrf2 Inhibit NF-κB	Anti-inflammation Antioxidation

4. Clinical research evidence

Preceding chapters have laid out the molecular and mechanistic foundation for regulation of T2DM inflammation and oxidative stress through diet. However, whether these mechanisms translate into concrete metabolic benefits in clinical practice still requires population-based research to verify. In this chapter, data from two randomized controlled trials of intermittent fasting and the Mediterranean diet will be synthesized. These trials focus on the effect of diet intervention on weight management, glycemic control, and cardiovascular risk factors, and summarize dietary intervention biomarkers, thereby providing quantifiable evidence-based support for clinical application of dietary interventions.

Borgundvaag et al. conducted a systematic review and a meta-analysis. They compared the impact that intermittent fasting (IF) and a standard diet have on weight and glycemic control in T2DM patients [13]. Seven studies involving a total of 338 participants with an average body mass index (BMI) of 35.65 kg/m^2 and an average baseline glycated hemoglobin (HbA1c) of 8.8% met the inclusion criteria. The pooled estimates of body weight and HbA1c differences were calculated by a random-effects model. The results demonstrated that, compared with a standard diet, IF induced a greater reduction in weight of 1.89kg (95% confidence interval (CI) -2.91 to -0.86 kg). Subgroup analyses indicated that IF-induced extra weight loss was more obvious in participants with baseline BMI greater than 36 kg/m^2 (-3.24 kg, 95% CI -5.72 to -1.15 kg) and in studies with durations shorter than or equal to 4 months. However, compared with a standard diet, no increase in the reduction of HbA1c was observed under IF, and there was no additional benefit from IF in fasting glucose, lipid profiles, or blood pressure. By systematically integrating data from randomized controlled trials, this review was the first to show the advantage of IF over a standard diet in weight loss in numbers among a large group of T2DM patients. It also showed the effects of IF and the standard diet on blood sugar control were different. Based on these findings, IF may be suitable for weight loss in people with T2DM. However, more trials are needed to verify the long-term safety and metabolic benefits of IF [13].

Another meta-analysis was conducted by Zheng et al. to evaluate the clinical effects of the Mediterranean diet in patients with T2DM [14]. Seven randomized controlled trials involving 1371 participants were included in this study. All seven trials compared the Mediterranean diet to a control diet. The researchers found that the Mediterranean diet has significant advantages in glycemic control. It reduced HbA1c by 0.39% and reduced fasting plasma glucose by 15.12 mg/dL. This suggests that the benefits of the Mediterranean diet for glucose metabolism in T2DM patients are practical. In terms of blood pressure management, both systolic and diastolic blood pressure were reduced under the Mediterranean diet. There were no significant changes in the lipid profile of HDL, LDL, and total cholesterol (TC). However, figures related to weight, including BMI and waist circumference, showed obvious improvements. By combining high quality randomized controlled trials from both Chinese and English databases, this meta-analysis clarified the clinical benefits in

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T2DM patients. At the same time, it provided strong evidence-based support for the Mediterranean diet as a good all-round treatment for T2DM. However, it has not been clarified whether the Mediterranean diet affects the lipid profile. Therefore, new research should be conducted with more rigorous design and longer follow-up durations to further clarify the different benefits of the Mediterranean diet across different subpopulations with T2DM [14].

In summary, both intermittent fasting and the Mediterranean diet have a large body of randomized trial data for people with T2DM. Intermittent fasting has the advantage in weight management, but it does not improve glycemic control over the normal diet. The Mediterranean diet showed advantages in controlling blood pressure, blood glucose, and weight, but its effect on lipids remained a question. Clarifying the characteristics of the target population and the benefit scope of these intervention strategies may help develop more tailored dietary strategies for people with T2DM.

5. Conclusion

In conclusion, no single pathway can account for the processes of inflammation and oxidative stress associated with T2DM. The development of T2DM involves a complex network of numerous factors at multiple levels. These include local immune dysregulation in adipose tissue, long-range signaling through small extracellular vesicles, adipokine network disruption, and impaired insulin signal transduction. Diet affects the network at numerous levels. Dietary patterns, such as the Mediterranean diet, exert an anti-inflammatory effect while also alleviating oxidative damage by the combined effort of multiple dietary components. Polyunsaturated fatty acids (n-6) reduce the amount of fat in the liver and improve the inflammatory state. Fibers and the short-chain fatty acids generated by gut microbiota fermentation show antioxidant effects. By combining oat β-glucan with L-arabinose, insulin resistance in the liver is reduced while activating the PI3K/AKT signaling pathway. Polyphenolic compounds have anti-inflammatory and antioxidant effects by regulating the Nrf2/NF-kB pathway in both directions. In addition, randomized controlled trials have provided clinical evidence for the translation of these mechanisms. Based on the evidence, intermittent fasting has advantages in weight management, and the Mediterranean diet is beneficial for glycemic control, blood pressure regulation, and weight loss. Through the joint effect of these links, insulin resistance and target organ function are constantly enhanced. Identifying key nodes in this network can help to determine the targets for dietary intervention and thus provide a practical basis for implementing personalized nutritional strategies based on the disease states of patients. Although the current evidence suggests that dietary intervention may be beneficial to the metabolic disorders linked to T2DM, future research should further determine whether there are different effects of various diets in people at different disease states. In addition, future research should explore the optimal combination ratio of multiple components, and combine technologies to identify new biomarkers, to translate mechanistic insights into precise nutrition strategies. Moreover, long-term follow-up and real-world studies will help verify the effectiveness and safety of dietary interventions, thereby providing more solid theoretical support for clinical application.

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