The Impact of Lifestyle and Dietary Antioxidants on Diabetes: A Comprehensive Review of Prevention and Management Strategies

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Abstract. Diabetes mellitus is a pervasive global health crisis characterised by chronic hyperglycemia, leading to severe microvascular and macrovascular complications. Oxidative stress, an imbalance between free radicals and antioxidants, is a central pathogenic mechanism, impairing insulin production and exacerbating insulin resistance. This comprehensive review synthesises evidence on the pivotal role of lifestyle modifications and dietary antioxidants in diabetes prevention and management. Findings reveal that integrated lifestyle interventions are fundamental, encompassing balanced nutrition, regular physical activity, weight management, stress reduction, and optimised sleep. Moreover, dietary antioxidants, including Vitamins C, E, carotenoids, and polyphenols, sourced from whole foods, demonstrate protective effects against diabetes risk and improve metabolic parameters. Notably, whole food consumption is superior to isolated antioxidant supplements, which have shown inconsistent efficacy and potential prooxidant effects at high doses. The most effective strategy for mitigating diabetes and its complications involves a synergistic approach, combining comprehensive lifestyle changes with antioxidant-rich diets. Future research should focus on personalised nutrition and the intricate interplay between diet, the gut microbiome, and metabolism.

Keywords: Diabetes; Lifestyle; Antioxidants; Oxidative Stress; Prevention; Management.

INTRODUCTION

Diabetes mellitus constitutes a chronic metabolic disorder marked by consistently elevated blood glucose concentrations. As time progresses, this enduring hyperglycemia culminates in profound and extensive damage to various organ systems, encompassing the cardiovascular system, vasculature, ocular structures, renal function, and neural pathways [1]. The widespread nature of this detriment accentuates the imperative for efficacious preventive measures and management protocols [2].

The worldwide prevalence of diabetes signifies a substantial public health emergency. At present, it is estimated that 589 million adults between the ages of 20 and 79 years globally have diabetes, with projections indicating an increase to 853 million by the year 2050 [3, 4]. A particularly disconcerting facet of this public health challenge is the considerable fraction of undiagnosed cases, which comprises approximately 252 million individuals worldwide who remain oblivious to their condition [5]. This lack of awareness considerably heightens their susceptibility to developing severe complications and facing premature mortality [5, 6]. The predominant proportion of adults diagnosed with diabetes, specifically three-quarters, inhabit low- and middle-income countries (LMICs), thereby underscoring the disproportionate repercussions of this disease in these geographical locales [7, 8]. The affliction accounts for more than 3.4 million fatalities each year, and its economic implications are substantial, with global healthcare expenditures attributable to diabetes exceeding one trillion US dollars for the first time in 2024 [7, 9, 10].

The alarming prevalence of undiagnosed cases on a global scale, particularly the nearly 79% in Nigeria, underscores a formidable systemic challenge to disease identification [5, 7]. This phenomenon transcends mere statistical significance; it signifies a profound inadequacy in public health infrastructure and the efficacy of awareness initiatives. When individuals remain unrecognised as diabetic, they are deprived of timely interventions and management strategies, which directly exacerbate the onset and severity of diabetes-related complications. Such delays in healthcare invariably culminate in adverse health outcomes and impose an even greater economic strain over time, as the management of advanced complications is markedly more costly than the proactive management of the disease in its nascent stages [5]. This trend indicates that public health initiatives must urgently prioritise comprehensive screening and educational endeavours to transition from a reactive model focused on managing severe complications to a proactive framework centred on prevention and early detection [5, 11].

Prediabetes, also known as Impaired Glucose Tolerance (IGT) or Impaired Fasting Glucose (IFG), is a condition where blood glucose levels exceed normative values but do not reach the thresholds for a definitive diagnosis of diabetes. This condition often presents without symptoms, leading to a lack of awareness among affected individuals [5]. However, prediabetes does not necessarily lead to Type 2 diabetes; it presents an opportunity for intervention [3, 5]. Implementing lifestyle modifications, such as a modest reduction in body weight and 150 minutes of moderate aerobic physical activity weekly, can significantly reduce the likelihood of developing Type 2 diabetes by nearly 60% over three years [4]. This underscores the public health importance of screening for prediabetes among at-risk demographics and advocating for lifestyle modifications [4]. Understanding the various types of diabetes, which are broadly classified based on their foundational causes and mechanisms, is crucial for a proactive prevention strategy.

Classification of Diabetes

Type 1 diabetes constitutes 5% to 10% of all diabetes cases [7]. It represents the predominant type diagnosed in individuals under the age of 20, although its onset may occur at any stage of life, with an average diagnosis age of 24 years [10, 11]. The symptoms typically emerge abruptly. This condition is characterised by the chronic and progressive loss of insulin-producing beta cells within the pancreatic islets [2]. This cellular destruction is attributed to a T cell-mediated autoimmune response, wherein the immune system erroneously targets and obliterates these essential cells [12]. The resultant significant deficiency of insulin implies that glucose cannot enter the body's cells efficiently, culminating in its accumulation within the bloodstream and consequently resulting in hyperglycemia [13]. The specific instigator of this autoimmune reaction remains ambiguous; however, genetic predispositions, particularly certain HLA genotypes, alongside environmental factors, such as viral infections, are posited to contribute to the phenomenon. At present, prevention of Type 1 diabetes is not feasible and requires the administration of exogenous insulin daily to ensure survival and maintain glycemic control [10].

Type 2 diabetes is a widespread variant affecting adults, characterised by progressive symptoms and a complex pathophysiological underpinning [2]. Its pathophysiological underpinnings involve beta cell dysfunction and insulin resistance, which are two primary pathological states [1]. Beta cell dysfunction involves impaired insulin secretion, while insulin resistance is an aberration in insulin signalling within glucose-absorbing tissues like muscle, liver, and adipose tissue [3, 14]. Both pathological states contribute to sustained hyperglycemia and exacerbate the disease's progression [1]. Beta cell dysfunction is often a pivotal determinant and may precede insulin resistance. Factors contributing to beta cell dysfunction include inflammation, oxidative stress, endoplasmic reticulum stress, and mitochondrial dysfunction [1]. Insulin resistance is significantly modulated by obesity, which cultivates chronic low-grade inflammation and increases proinflammatory cytokines, such as TNF-α and IL-6, which desensitise glucose-absorbing organs to insulin, disrupting the standard signalling cascade for glucose uptake [3, 4].

Gestational Diabetes (GDM) is a form of glucose intolerance that arises during pregnancy, affecting approximately 12.4% of live births globally [9, 15]. The pathophysiology of GDM arises from an inadequate compensatory response of pancreatic β-cells to the physiological insulin resistance induced by pregnancy [16]. In GDM, β-cells are unable to satisfy the increased demand, resulting in maternal hyperglycemia [16]. The normal insulin signalling pathway is disrupted, resulting in impaired glucose uptake. Hormones like human placental lactogen (hPL) and cortisol, along with proinflammatory cytokines like TNF-α and IL-6, contribute significantly to this physiological disruption [15]. These factors facilitate the aberrant phosphorylation of insulin receptor substrates (IRS-1) at serine residues, obstructing the proper activation of downstream signalling molecules [15]. This results in GLUT4 vesicles remaining sequestered within the cytoplasm, leading to elevated blood glucose concentrations [15]. The dysfunction of β-cells in GDM arises from a complex interplay of hormonal, cytokine-mediated, metabolic, and genetic factors, culminating in impaired insulin production and secretion, ultimately leading to β-cell apoptosis [15, 16]. GDM is a robust predictor of subsequent Type 2 diabetes for the mother, suggesting that it is not merely a transient complication of pregnancy but serves as a critical early indicator of long-term metabolic health risks [16].

Specific Types of Diabetes Due to Other Causes can be categorised into three types: Pancreatic Diabetes, Monogenic Diabetes, and Drug-Induced Diabetes [17]. Pancreatic diabetes is caused by damage to the pancreas, impairing insulin production [4, 18]. Cystic Fibrosis-Related Diabetes (CFRD) is prevalent among cystic fibrosis patients, requiring insulin therapy and a high-calorie diet [4, 19]. Post-Pancreatitis Diabetes, also known as "Type 3c diabetes," occurs after pancreatitis, leading to insulin-producing cell destruction. Post-Transplantation Diabetes (NODAT) arises in organ transplant recipients due to immunosuppressants impairing insulin secretion or increasing resistance [20]. Monogenic diabetes is caused by mutations in a single gene, often showing familial patterns, and making genetic testing pivotal [19]. Neonatal diabetes is a rare type that appears within the first six months due to a single gene mutation impacting insulin production or utilisation [19, 21]. Maturity-Onset Diabetes of the Young (MODY) is hereditary diabetes manifesting before age 25, often with notable familial prevalence [21]. Genetic testing is crucial for accurate diagnosis and treatment direction [21–23]. Drug-induced diabetes is triggered by certain pharmaceuticals, such as glucocorticoids, antipsychotics, and immunosuppressants [22]. Glucocorticoids augment hepatic glucose production and promote insulin resistance in peripheral tissues, leading to significant hyperglycemia [22]. Antipsychotics, such as olanzapine and clozapine, correlate with heightened Type 2 diabetes risk, potentially involving weight gain, increased insulin resistance, and direct pancreatic beta cell toxicity [24, 25]. Immunosuppressants, like calcineurin inhibitors and corticosteroids, can either directly damage pancreatic beta cells or induce insulin resistance [8].

Type 5 diabetes, also known as Severe Insulin-Deficient Diabetes (SIDD) or malnutrition-related diabetes, is a newly recognised form of diabetes estimated to affect 20 to 25 million people globally, predominantly in Asia and Africa [26]. This discovery is crucial for understanding how diabetes impacts lean and malnourished teens and young adults in low- and middle-income countries (LMICs). Unlike Type 1 diabetes, which is an autoimmune condition, or Type 2 diabetes, characterised by insulin resistance, Type 5 diabetes is unique. It's primarily caused by chronic undernutrition, especially during childhood or adolescence, leading to impaired pancreatic development and significant insulin deficiency [17]. Though observed for over 70 years, Type 5 diabetes was largely ignored and often misdiagnosed as Type 1 or Type 2. Recent research has confirmed its distinct metabolic profile: individuals with Type 5 diabetes are insulin deficient but not insulin resistant [26]. This distinction is vital because, unlike other forms, many people with Type 5 diabetes may be able to manage their condition with oral medication instead of insulin injections. This cost-effective treatment option offers a significant advantage in resource-limited regions where the condition is most prevalent [26].

Diagnostic Criteria. The diagnosis of diabetes and prediabetes is based on specific blood glucose measurements, with confirmatory testing typically required on a separate day, unless initial glucose levels are exceptionally high or classic symptoms are overtly present. The American Diabetes Association (ADA) and the World Health Organisation (WHO) provide standardised criteria for these diagnoses, as outlined in Table 1.

Table 1 – ADA Diagnostic Criteria for Diabetes and Prediabetes [2]

Test	Normal Range	Prediabetes Range	Diabetes Range
A1C Test	< 5.7%	5.7% - 6.4%	≥ 6.5%
Fasting Plasma Glucose (FPG) Test	< 100 mg/dL	100 mg/dL - 125 mg/dL	≥ 126 mg/dL
Oral Glucose Tolerance Test (OGTT)	< 140 mg/dL	140 mg/dL - 199 mg/dL	≥ 200 mg/dL
Random (Casual) Plasma Glucose Test	N/A (used with symptoms)	N/A	≥ 200 mg/dL

Complications of Diabetes. Diabetes is associated with a wide array of complications, categorised into microvascular (affecting small blood vessels) and macrovascular (affecting large blood vessels) diseases. These complications lead to chronic damage, dysfunction, and eventual failure of various organs throughout the body [14]. Microvascular complications include diabetic retinopathy (DR), diabetic nephropathy (DN), and diabetic neuropathy (PN), which cause visual impairment and blindness, kidney damage, and nerve damage [27]. Macrovascular complications involve damage to large blood vessels, increasing the risk of major cardiovascular events, such as cardiovascular disease (CVD), cerebrovascular disease (CeVD), and peripheral artery disease (PAD). Chronic hyperglycemia plays a central role in initiating these complications through a cascade of metabolic and structural derangements [27]. Key mechanisms contributing to widespread damage include advanced glycation end products (AGEs), oxidative stress, inflammation, polyol pathway activation, protein kinase C (PKC) pathway activation, hexosamine pathway flux, endothelial dysfunction, atherosclerosis, hypercoagulability, and impaired fibrinolysis [3, 27, 28]. The underlying mechanisms of complications include chronic hyperglycemia, oxidative stress, inflammation, polyol pathway activation, hexosamine pathway flux, endothelial dysfunction, atherosclerosis, hypercoagulability, and impaired fibrinolysis [29]. These complications can lead to vascular damage, dysfunction, and eventual failure of various organs throughout the body [27, 29].

RESULTS AND DISCUSSION

The Pivotal Role of Lifestyle in Diabetes Prevention and Management

Lifestyle modifications are cornerstones in both the prevention and effective management of diabetes, offering profound benefits that extend beyond glycemic control to overall health and well-being.

Dietary Interventions. Healthy eating is crucial for managing blood glucose levels and improving overall health in individuals with or at risk of diabetes. The principles of healthy eating emphasise a shift towards nutrient-dense foods and away from highly processed options. This includes prioritising non-starchy vegetables, lean proteins, plant-based protein sources, quality carbohydrates, and healthy fats [30, 31]. The "Diabetes Plate Method" offers a simplified visual guide for meal planning, focusing on half of a 9-inch plate filled with non-starchy vegetables, one-quarter with lean protein, and the remaining quarter with quality carbohydrates [32]. This Diabetes Plate Method simplifies meal planning by visually balancing macronutrients and encouraging portion control [32–34]. Understanding how different foods affect blood glucose is also critical, as foods high in simple carbohydrates, unhealthy fats, and excessive calories have a more significant impact [34, 35]. Consuming carbohydrates alongside protein, fat, or fibre can slow down the rate at which blood sugar rises, helping to prevent sharp spikes. Planning for regular, balanced meals with consistent carbohydrate amounts is a key strategy for avoiding erratic blood sugar fluctuations [33]. Portion control is especially important when dining out, where serving sizes tend to be much larger than needed. Practical tools like hand guides can help estimate appropriate portion sizes [30]. For personalised guidance and comprehensive support, consulting a doctor or registered dietitian for diabetes self-management education is highly recommended [30]. The current dietary guidelines represent a significant evolution from traditional, often restrictive, diabetes diets, moving beyond merely listing forbidden foods to empowering individuals with a framework for balanced nutrition and mindful eating [34]. The emphasis on non-starchy vegetables, lean proteins, and quality carbohydrates is not solely about limiting glucose intake but about optimising nutrient density, promoting sustained satiety, and fostering a healthier relationship with food [31, 34].

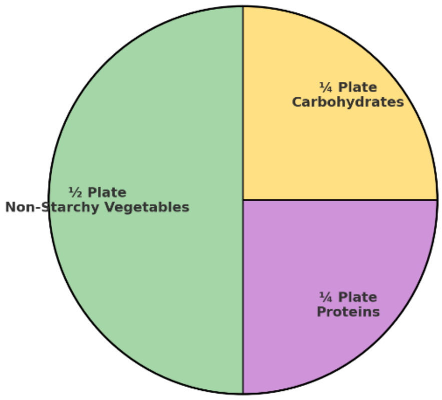

Figure 1 – The Diabetes Plate Method, recreated by the authors based on [32]

Physical Activity Guidelines. Regular physical activity is crucial for diabetes prevention and management, offering numerous benefits such as weight loss, reduced blood sugar levels, enhanced insulin sensitivity, and improved aerobic fitness [36]. The American Diabetes Association and the World Health Organisation provide guidelines for adults living with diabetes, suggesting 150-300 minutes per week of moderate-intensity aerobic physical activity or 75-150 minutes per week of vigorous-intensity aerobic activity [30, 37]. Strength training sessions should be included to enhance strength, balance, and support an active lifestyle.

Research indicates that the optimal dose of physical activity for reducing HbA1c, a key marker of long-term blood glucose control, is approximately 1,100 METs-min/week across various glycemic categories. Specific modalities and timing of exercise can further optimise outcomes [36].

Table 2 – Physical Activity Guidelines for Adults with Type 2 Diabetes

Activity Type	Recommended Duration / Frequency	Approximate Equivalent (METs-min/week)
Moderate-intensity aerobic activity	150–300 min/week	244 min/week (optimal for 1100 METs-min/week)
Vigorous-intensity aerobic activity	75–150 min/week	157 min/week (optimal for 1100 METs-min/week)
Strength training (all major muscle groups)	≥ 2 sessions/week	314 min/week (moderate), 183 min/week (vigorous)
Combined aerobic and resistance training	Greater reduction in A1C than either alone	Varies, but often more effective
Breaking sedentary time	3 min of activity every 30 min	Improves glycemic control and insulin sensitivity

Combining aerobic and resistance training has been shown to yield a greater reduction in A1C levels than either type of exercise performed alone [38]. High-intensity interval training (HIIT) offers a time-efficient approach to achieving significant glycemic control. However, it may carry a higher risk of musculoskeletal injury and transient post-exercise hyperglycemia for some individuals [38]. The specific timing of exercise, particularly after meals, is crucial for optimal glycemic control and insulin sensitivity [31, 39, 40]. The guidelines demonstrate a sophisticated understanding of exercise physiology in diabetes management, recognising that exercise is not a monolithic intervention but a highly adaptable tool that can be personalised for maximal glycemic benefits and overall health [39].

Weight management is crucial for diabetes prevention and control, as excess adiposity is linked to metabolic dysfunction [24, 31, 35]. Obesity is a complex medical condition influenced by behavioural, environmental, and genetic factors [40]. Being overweight or obese increases the risk of developing Type 2 diabetes, hypertension, high cholesterol, and blood clots, all major risk factors for cardiovascular disease [40, 41]. Losing just 5% to 10% of body weight can significantly lower the risk of developing Type 2 diabetes, improve overall diabetes management, and increase energy levels [42]. Effective weight management strategies involve a sustainable combination of healthy eating, regular physical activity, and mindful portion control. Medical advancements, such as anti-obesity medications like Liraglutide, Semaglutide, and Tirzepatide, can help reduce hunger and promote satiety and may also improve blood glucose management. Bariatric surgeries can induce significant weight loss in severe obesity [42, 43].

The reclassification of obesity as a disease transforms the approach to its treatment, moving beyond simple diet and exercise recommendations to a more comprehensive medical model. This shift in the medical paradigm acknowledges that lifestyle changes alone may not be sufficient due to complex biological and genetic predispositions influencing metabolism and weight regulation [43]. Effective weight management in diabetes requires a multidisciplinary approach that integrates nutritional and exercise counselling, pharmacological and surgical options, and psychological support. This nuanced understanding of obesity as a treatable disease is critical for improving patient outcomes and reducing the overall burden of diabetes [42, 43].

Stress Management. Stress can significantly impact blood sugar levels and diabetes management. Stress hormones like cortisol and adrenaline, part of the "fight or flight" response, make it harder for insulin to function effectively, leading to increased insulin resistance and a rise in blood sugar levels [25, 44]. Chronic or prolonged stress can maintain elevated blood sugar, increasing the risk of developing diabetes complications. The bidirectional relationship between stress and diabetes is significant, leading to a vicious cycle of neglecting self-care routines, unhealthy habits, and missing medical appointments [8, 25].

Effective stress reduction techniques are essential for comprehensive diabetes management. Cognitive strategies involve challenging negative thoughts, focusing on the present moment, and using positive self-talk [45]. Mind-body practices like deep breathing, visualisation exercises, and progressive muscle relaxation can calm the nervous system and steady the heart rate [46]. Physical activity can boost mood and contribute to blood glucose regulation. Seeking social and emotional support, connecting with understanding friends, dedicating time for self-care, practising gratitude, and consulting a mental health counsellor when overwhelmed are also crucial [39, 46].

The direct physiological impact of stress hormones on insulin sensitivity and the psychological burden of managing a chronic condition like diabetes create a complex psychosomatic loop 1]. Effective diabetes care must integrate mental health support and stress reduction techniques as core components. Recognising emotional well-being is as critical as dietary adherence, and physical activity is crucial for achieving optimal glycemic control and preventing long-term complications [25, 29].

Optimising Sleep Quality. Sleep quality significantly impacts metabolic health and diabetes risk. Insufficient sleep duration, poor sleep quality, and sleep disorders like insomnia and sleep apnea are strongly associated with an increased risk of diabetes [47]. A U-shaped relationship exists between sleep duration and the risk of Type 2 diabetes, with the lowest risk observed at 7-8 hours of sleep per day [37, 48]. Sleep deprivation impairs the body's ability to utilise insulin effectively, leading to reduced insulin sensitivity and higher blood sugar levels [47]. Sleep deprivation also disrupts the balance of hunger-regulating hormones, increasing caloric intake and weight gain [37]. Poor sleep quality is linked to increased systemic inflammation and oxidative stress. Behaviorally, insufficient sleep can impair decision-making and increase the likelihood of engaging in other risk factors for diabetes, such as smoking, sedentary behaviour, and excessive alcohol consumption [48]. To optimise sleep quality for diabetes management, several sleep hygiene practices are recommended. Establishing a consistent sleep schedule, creating a relaxing bedtime routine, avoiding screens, monitoring blood sugar levels before bedtime, limiting stimulants close to bedtime, ensuring a sleep-friendly environment, and engaging in regular physical activity can improve sleep quality [47]. The U-shaped relationship between sleep duration and diabetes risk, along with its direct impact on insulin sensitivity and hunger hormones, makes sleep a crucial metabolic regulator [48].

Dietary Antioxidants: A Mechanistic and Clinical Perspective

Understanding Oxidative Stress and Antioxidants in Diabetes Pathogenesis. Oxidative stress is a fundamental imbalance in the body, where the production of reactive oxygen species (ROS) and reactive nitrogen species (RNS) overwhelms the body's capacity to neutralise them with antioxidants [49, 50]. These unstable molecules can damage vital cellular components, leading to cellular dysfunction, injury, and even death [50]. Oxidative damage is implicated in the initiation and progression of chronic diseases like atherosclerosis, ischemic heart disease, liver disease, and various forms of cancer [49]. In diabetes, oxidative stress is a pivotal contributor to the disease's onset and progression, impairing insulin production, exacerbating insulin resistance, perpetuating hyperglycemic memory, and inducing systemic inflammation [49]. Hyperglycemia induces excessive free radical generation through mechanisms like auto-oxidation of glucose, glycated proteins formation, and increased NADPH oxidase activity [18]. Antioxidants function by counteracting free radicals, primarily by being oxidised themselves, and can also exert indirect effects by inhibiting enzymes that generate free radicals or by enhancing intracellular antioxidant enzymes [18]. Oxidative stress is not just a symptom of the disease but a direct driver of its progression, suggesting that therapeutic and preventive strategies should directly target the reduction of oxidative stress, making dietary antioxidants and other redox-modulating interventions highly relevant for comprehensive diabetes management [50].

Key Dietary Antioxidants: Sources and Mechanisms of Action. Living cells possess a sophisticated defence system against oxidative damage, comprising two major groups of antioxidants: enzymatic and non-enzymatic. Enzymatic antioxidants include crucial enzymes like glutathione peroxidase, catalase, and glutathione reductase, which directly neutralise free radicals or support other antioxidant processes. Non-enzymatic antioxidants, which are largely obtained through diet, encompass a diverse array of compounds, including vitamins (A, E, C), enzyme cofactors (Q10), minerals (zinc, selenium), peptides (glutathione), phenolic acids, and nitrogen compounds (uric acid) [51].

Table 3 – Major Dietary Antioxidants: Sources and Mechanisms of Action

Antioxidant Type	Primary Dietary Sources	Key Mechanisms of Action
Vitamin C	Citrus fruits, berries, tomatoes, potatoes, green leafy vegetables	Neutralises free radicals, regenerates Vitamin E, a cofactor for collagen synthesis, and attenuates inflammation
Vitamin E	Vegetable oils (wheat germ, sunflower, olive), nuts (almonds), seeds (sunflower), green leafy vegetables (spinach, broccoli)	Scavenges free radicals, protects cell membranes from lipid peroxidation, and inhibits platelet aggregation.
Carotenoids	Yams, kale, spinach, watermelon, cantaloupe, bell peppers, tomatoes, carrots, mangoes, oranges (yellow, red, orange fruits/veg)	Act as antioxidants, reduce inflammation, protect eyes (absorb blue light), and may protect against cancer.
Flavonoids & Polyphenols	Grains, cereals, pulses, vegetables (onions, celery, broccoli), fruits (apples, berries, citrus, grapes), spices, chocolate, tea, coffee, wine	Antioxidative, anti-inflammatory, improve insulin secretion, reduce oxidative stress, control gluconeogenesis, and regulate gut hormones

Vitamin C, also known as ascorbic acid, is a crucial water-soluble vitamin essential for human health. Its primary function is to neutralise free radicals and reduce inflammatory responses [52]. Vitamin C is also a crucial cofactor for biological processes like collagen biosynthesis, carnitine and catecholamine metabolism, and iron absorption [53]. Its antioxidant function is particularly significant in its ability to regenerate vitamin E from its oxidised form, demonstrating a synergistic interaction between these two vitamins [54].

The effectiveness of one antioxidant depends on the availability and activity of another. Vitamin C, a water-soluble antioxidant, can restore the function of Vitamin E, a fat-soluble antioxidant, indicating that these compounds work together across different cellular compartments [54]. Focusing on a single antioxidant may be less effective than ensuring a balanced intake of multiple antioxidants that support the body's overall redox balance. A "whole food" approach to nutrition is recommended, as it naturally provides these complex and interdependent antioxidant systems [52, 53].

Vitamin E, also known as tocopherol, is a fat-soluble vitamin that is essential for protecting cellular integrity, particularly cell membranes. It is found in vegetable oils, nuts, seeds, and green leafy vegetables. Its primary function is as a potent scavenger of free radicals, embedding within lipid bilayers to prevent the peroxidation of polyunsaturated fatty acids in cell membranes [20]. Vitamin E also contributes to vascular health by inhibiting the aggregation of platelets and the adhesion of monocytes, which are linked to cardiovascular diseases [30]. Its fat-soluble nature allows it to directly integrate into fatty environments, acting as a first line of defence against lipid peroxidation [45, 55]. This is particularly relevant in conditions like diabetes, where cell membranes are under constant oxidative assault, impacting cell integrity and signalling. Different antioxidants have specialised roles, highlighting the importance of consuming a diverse range of foods to ensure the intake of various antioxidants, each contributing to comprehensive cellular protection in distinct ways [56].

Carotenoids are pigments found in plants, algae, and photosynthetic bacteria, responsible for the vibrant yellow, red, and orange colours in fruits and vegetables. Common dietary sources include yams, kale, spinach, watermelon, cantaloupe, bell peppers, tomatoes, carrots, mangoes, and oranges. For optimal absorption, carotenoids are best consumed with a source of fat [57]. There are over 600 different types of carotenoids, broadly classified into xanthophylls and carotenes. Xanthophylls, like lutein and zeaxanthin, impart yellow hues and are associated with eye health due to their ability to absorb harmful blue light. Carotenes, like alpha-carotene, beta-carotene, and lycopene, typically contribute red and orange colours. Some, like beta-carotene, can be converted into Vitamin A in the body [23, 57]. Carotenoids not only contribute to antioxidant activity but also lower systemic inflammation, offering protection against heart disease by preventing blockages in arterial walls [57, 58]. Specific carotenoids, like lycopene, accumulate in tissues like the testis, mitigating oxidative damage. The strong correlation between fruits and vegetables' vibrant colours and their carotenoid content provides a practical strategy for ensuring a broad intake of diverse antioxidants [57].

Flavonoids, a subgroup of polyphenols, are found in various foods and beverages, including grains, cereals, pulses, vegetables, fruits, spices, chocolates, and beverages. These compounds have numerous biological activities, including antioxidative, anti-inflammatory, anti-mutagenic, and anticarcinogenic properties. They can also regulate key cellular enzyme functions, particularly in the context of diabetes [28]. Flavonoids can improve insulin sensitivity by suppressing protein tyrosine phosphatase 1B and inhibiting inflammatory pathways. They can enhance insulin secretion by activating calcium-calmodulin-dependent protein kinase type 2 in pancreatic β-cells and upregulating glucose transporter 2 (GLUT2) expression [58, 59]. They can reduce oxidative stress by enhancing mitochondrial membrane potential and protecting β-cells from inflammation. They can control gluconeogenesis by inhibiting key enzymes and activating pathways like AMP-activated protein kinase (AMPK) [20, 30]. Flavonoids also regulate gut hormones, such as GLP-1, PYY, CCK, and ghrelin, which are essential for appetite regulation, satiety, and systemic glucose metabolism [59]. This suggests that dietary polyphenols do not merely act as simple antioxidants but actively modulate the "gut-hormone-metabolism axis." This suggests that a polyphenol-rich diet extends beyond direct cellular protection to influence systemic metabolic control through interactions originating in the gut, potentially involving the gut microbiota [28, 59]. This represents a significant advancement in understanding the holistic impact of diet on diabetes prevention and management [28].

Whole Foods vs. Supplements: Efficacy and Safety Considerations. The debate surrounding the efficacy of antioxidants often centres on whether they are best obtained from whole food sources or through dietary supplements. Scientific evidence overwhelmingly favours the former, with significant implications for diabetes prevention and management.

Plant-based foods are the best sources of antioxidants, providing key vitamins and phytochemicals like flavonoids, isothiocyanates, and phenolic acids. These compounds are more effective when consumed within the intricate matrix of whole foods, as they exhibit synergistic effects. Healthy fats in whole foods can enhance the absorption of fat-soluble antioxidants like carotenoids and Vitamin E [60]. Furthermore, foods rich in these compounds offer other health advantages, such as being high in fibre, low in saturated fat and cholesterol, and providing essential vitamins and minerals. These additional benefits contribute to overall health and aid in diabetes management. A well-balanced diet that emphasises a variety of colourful fruits, vegetables, nuts, seeds, and whole grains is the most effective strategy for ensuring a diverse and adequate intake of antioxidants [60]. The scientific finding that whole foods are superior to isolated supplements for antioxidant benefits reflects the fundamental principle about how nutrients function within biological systems [20]. Biological systems have evolved to process nutrients within complex food matrices, where various compounds interact synergistically to enhance absorption, bioavailability, and overall protective effects. Isolating a single compound disrupts this natural synergy, leading to reduced efficacy or adverse effects [58]. Therefore, nutritional science and public health recommendations should prioritise dietary patterns and the consumption of whole foods over the reductionist approach of single-nutrient supplementation, especially for chronic disease prevention and management [58].

High-dose antioxidant supplements can have detrimental effects, sometimes acting as prooxidants rather than protective agents. This prooxidant effect can occur depending on factors such as the presence of metal ions, the concentration of the antioxidant, and its specific redox potential within the biological environment [61]. For example, Vitamin C can exhibit prooxidant activity at very high concentrations, particularly when combined with transition metals like iron or copper [57, 62]. Alpha-tocopherol (Vitamin E) and beta-carotene can lose their effectiveness or become prooxidants at high oxygen tension or in the absence of co-antioxidants like Vitamin C for their regeneration [55]. Flavonoids and phenolics have also been reported to act as prooxidants in systems containing transition metals [61].

High doses of antioxidants may also interfere with normal biological processes, potentially reducing the body's natural response to free radicals and creating an environment more susceptible to oxidation [63]. There is concern that they could enhance malignancy by allowing genetically damaged cells to survive and proliferate. This counterintuitive finding challenges the common perception that "more is always better" for health, as excessive amounts of certain compounds can disrupt the delicate balance of redox homeostasis within the body. It highlights the need for extreme caution with antioxidant supplementation, particularly in the general population or without a clear medical indication, and careful monitoring. A balanced diet of whole foods is the safest and most effective approach, as it provides these compounds in physiologically appropriate and synergistic concentrations [62, 63].

Integrated Strategies for Diabetes Prevention and Management

Synergistic Effects of Lifestyle Modifications and Antioxidant-Rich Diets. Diabetes pathophysiology and interventions suggest that an integrated strategy is the most effective approach for preventing and managing diabetes. This strategy involves multiple lifestyle modifications and a diet rich in antioxidants, which address distinct diabetes pathways, such as insulin resistance, beta-cell dysfunction, chronic inflammation, and overall metabolic dysregulation [24, 54].

An antioxidant-rich diet combats oxidative stress, a central underlying mechanism for diabetes development and its complications [64]. Regular physical activity improves insulin sensitivity and reduces systemic inflammation, while maintaining a healthy weight reduces the metabolic burden on pancreatic beta cells [38]. These physiological improvements create a more favourable internal environment, potentially enhancing the bioavailability and efficacy of dietary antioxidants.

The interconnectedness of various lifestyle factors and the underlying molecular mechanisms of diabetes leads to the conclusion that a truly effective strategy cannot be piecemeal but must be holistic. Each lifestyle factor, including diet, exercise, weight, stress, and sleep, independently influences diabetes but also interacts with and amplifies each other's effects. Oxidative stress and inflammation are common underlying mechanisms for diabetes development and complications, and are profoundly influenced by all lifestyle factors and dietary antioxidants. Optimal diabetes prevention and management necessitate a comprehensive, synergistic strategy that leverages the combined power of improved diet, regular physical activity, healthy weight maintenance, stress reduction, and adequate sleep [24, 38, 58]. This integrated approach creates a powerful defence against the disease's pathogenesis, leading to more profound and lasting improvements than any single intervention alone.

Practical Recommendations. Based on the comprehensive review, the following practical recommendations are crucial for individuals seeking to prevent or manage diabetes:

Dietary Focus: Prioritise a diverse, predominantly plant-based diet rich in whole foods. Emphasise non-starchy vegetables as the foundation of meals, complemented by lean proteins, quality carbohydrates from whole grains and fruits, and healthy fats. Utilise practical meal planning tools, such as the Diabetes Plate Method, and practice mindful portion control. Seeking personalised guidance from a registered dietitian or nutritionist is highly recommended to tailor dietary strategies to individual needs and preferences.

Physical Activity: Adhere to established guidelines, aiming for at least 150 minutes per week of moderate-intensity aerobic activity, or 75 minutes of vigorous-intensity activity, supplemented by two or more strength training sessions per week. Incorporate regular movement breaks throughout the day to mitigate the effects of prolonged sitting. Consider combined training approaches or high-intensity interval training (HIIT) where appropriate and safe, and prioritise exercising after meals for optimal glycemic control.

Weight Management: Strive to achieve and maintain a healthy body weight. Recognising obesity as a complex medical condition, individuals may benefit from a multi-faceted approach that includes lifestyle modifications and potentially medical or surgical interventions, under professional guidance. The focus should be on sustainable habits and seeking emotional support to navigate the challenges of weight management.

Stress Reduction: Integrate stress management techniques into daily life. Practices such as deep breathing exercises, mindfulness, engaging in regular physical activity, and seeking mental health support when feeling overwhelmed are vital. Acknowledge and actively address "diabetes distress," the emotional burden associated with managing the condition.

Sleep Optimisation: Establish and adhere to a consistent sleep schedule, going to bed and waking up at the same time daily. Cultivate a relaxing bedtime routine, ensure a conducive sleep environment (dark, quiet, cool), and manage blood sugar levels effectively to minimise nocturnal disruptions.

Prioritise Whole Foods over Supplements: Emphasise obtaining antioxidants from a wide variety of colourful fruits, vegetables, nuts, seeds, and whole grains. Exercise caution against high-dose antioxidant supplements, as they may have prooxidant effects and have not consistently demonstrated clinical benefits comparable to whole food sources. Consult a healthcare provider before considering any supplementation.

Early Detection and Monitoring: Promote and engage in regular screening for prediabetes, especially for individuals with risk factors, to enable timely intervention. For those diagnosed with diabetes, consistent medical check-ups, adherence to prescribed treatments, and ongoing diabetes self-management education are crucial for preventing complications and improving long-term health outcomes.

CONCLUSIONS

Diabetes mellitus is a global health challenge with complex pathophysiological mechanisms including insulin resistance, pancreatic beta-cell dysfunction, oxidative stress, and chronic inflammation. Lifestyle factors play a crucial role in its development and management. A holistic approach combining balanced nutrition, regular physical activity, weight management, stress reduction, and optimal sleep with a diet rich in antioxidants offers the most sustainable strategy for prevention and improved outcomes. The synergistic benefits of these combined interventions outweigh the limited and sometimes risky effects of isolated antioxidant supplements. Further high-quality, long-term randomised controlled trials are essential to determine the effects of specific dietary antioxidants and their optimal combinations within diverse populations. The development of personalised nutrition strategies tailored to individual genetic profiles and metabolic responses is a promising frontier for optimising diabetes care. Public health education and accessible screening programs are crucial to address the global burden of undiagnosed diabetes, particularly in vulnerable populations.

Conflict of Interest

The authors declared no conflict of interest.

Data Availability Statement

Data Sharing does not apply to this article, as no data sets were generated or analysed during the current study.

Author's Contribution

Abdulgafar Ahmad Onikoko: Conceptualisation, writing of the original draft, editing, proofreading, and compilation of the manuscript. Adesanya Mariam Opeyemi: Conceptualisation, writing of the original draft, proofreading, editing, and revising the manuscript. Fajimi Sefiu Remi: Writing of the original draft, proofreading, editing, and revising the manuscript. Oluwaseun Chimdindu Benson: Writing of the original draft, proofreading, editing, and revising of the manuscript. Olaniyi Samson Adebayo: Writing of the original draft, proofreading, editing, and revising of the manuscript. Nummi Atiku: Writing of the original draft, proofreading, editing, and revising of the manuscript. Obiageri Ihuarulam Okeoma: Writing of the original draft, proofreading, editing, and revising of the manuscript. Adeleke Shalome Oluwaferanmi: Writing of the original draft, proofreading, editing, and revising the manuscript.

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