BIELAS, Kinga, CHODKOWSKA , Ewa, DOMINICZAK, Kinga, SZAFRAŃSKA, Katarzyna, MUCHA, Aleksandra, SZEPS, Aleksander, ZABRZEŃSKI, Bartosz and SCHENDZIELORZ, Jessika. The Effect of a Ketogenic Diet on Human Health and Diseases: A Literature Review. Quality in Sport. 2025;43:61246. eISSN 2450-3118.
https://doi.org/10.12775/QS.2025.43.61246
https://apcz.umk.pl/QS/article/view/61246

The journal has been awarded 20 points in the parametric evaluation by the Ministry of Higher Education and Science of Poland. This is according to the Annex to the announcement of the Minister of Higher Education and Science dated 05.01.2024, No. 32553. The journal has a Unique Identifier: 201398. Scientific disciplines assigned: Economics and Finance (Field of Social Sciences); Management and Quality Sciences (Field of Social Sciences).
Punkty Ministerialne z 2019 - aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).
The authors declare that there is no conflict of interest regarding the publication of this paper.
Received: 20.05.2025. Revised: 05.07.2025. Accepted: 05.07.2025. Published: 07.07.2025.

The Effect of a Ketogenic Diet on Human Health and Diseases: A Literature Review

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Abstract

Introduction and Objective. The ketogenic diet (KD), characterized by a high intake of fats coupled with a significant reduction in carbohydrates, induces a state of ketosis that leads to significant metabolic changes in the body. Although its use in the treatment of epilepsy has been known for over a century, in recent years, this diet has gained popularity in the treatment of other diseases, including metabolic, cardiovascular, cancerous, and neurodegenerative disorders. Despite the growing body of research confirming its potential therapeutic benefits, many of the mechanisms of action remain not fully understood. The aim of this study is to review the current literature regarding the effects of the ketogenic diet on the human body, its applications in the treatment of various conditions, and potential adverse effects.

Review methods. A literature review was conducted using the PubMed and Google Scholar databases. The search included publications from the last 5 years, with a particular focus on clinical trials, randomized controlled trials, meta-analyses, and systematic reviews. The main

keywords used were "ketogenic diet," "ketogenic diet and health," and "ketogenic diet mechanisms."

State of Knowledge. The ketogenic diet has a beneficial effect on weight reduction and improved glycemic control in individuals with type 2 diabetes, and in the case of type 1 diabetes, it may lead to a reduction in insulin requirements, although careful monitoring is required due to the risk of hypoglycemia and diabetic ketoacidosis. Changes in the lipid profile observed during the diet are inconclusive, complicating the clear assessment of its impact on cardiovascular risk. Furthermore, the ketogenic diet may assist in the treatment of cancer by limiting glucose availability to cancer cells and lowering insulin levels. In neurodegenerative diseases, such as drug-resistant epilepsy, Alzheimer's disease, and Parkinson's disease, it demonstrates neuroprotective effects by providing an alternative energy source for neurons and modulating oxidative stress and inflammation. Despite its potential benefits, the diet is associated with adverse effects, including constipation, hypoglycemia, an increased risk of kidney stones, and lipid disturbances.

Summary. The ketogenic diet holds therapeutic potential in the treatment of various diseases, but its use should be monitored, especially in the case of long-term application, due to the possibility of adverse effects. Further research is needed, particularly in the context of long-term outcomes and the safety of its use in different patient populations. Additional studies should also be conducted to assess the effectiveness of the ketogenic diet in the treatment of neurodegenerative diseases and cancer, in order to confirm its full therapeutic potential.

Keywords: ketogenic diet, ketogenic diet and health, ketogenic diet mechanisms, ketogenic diet adverse effects

Introduction and objective

The ketogenic diet (KD) is a specialized dietary regimen characterized by a high fat content, very low carbohydrate intake, and a protein amount adjusted to the individual's requirements. This nutritional model induces a state of ketosis due to an increased production of ketone bodies, resulting in significant changes in protein and fat metabolism [1,2]. The history of using a low-carbohydrate diet as a therapy for epilepsy spans over 100 years. The first documented positive effects of fasting, manifested as a reduction in the frequency of

epileptic seizures, were observed in 1911 by Parisian physicians Gulep and Marie. In 1921, Dr. Rollin Woodyatt observed that under conditions of starvation or a diet high in fats and low in carbohydrates, ketone bodies such as acetone and beta-hydroxybutyric acid are produced. The term "ketogenic diet" was introduced by Dr. Russell Wilder in 1923, who developed and implemented this therapeutic method at the Mayo Clinic for children with drug-resistant epilepsy [3,4]. Currently, the ketogenic diet is gaining increasing popularity. The mechanisms of its beneficial effects remain poorly understood in many aspects and require further analysis. There is substantial evidence for its positive use in treating metabolic diseases such as obesity and type 2 diabetes. Studies suggest that it can significantly reduce cardiovascular disease risk factors [5]. The ketogenic diet is also of interest for its supportive role in treating various types of cancer and neurodegenerative diseases [2]. The immunomodulatory properties of ketone bodies offer potential applications in inflammatory diseases, such as inflammatory bowel disease [6]. However, it is important to remember that despite its many benefits, the ketogenic diet also has contraindications and potential adverse effects [1]. The objective of this review is to examine the literature on the mechanisms of action of the ketogenic diet on the human organism, its clinical applications in the treatment of various disorders, and the potential side effects associated with its use.

Review methods

A literature review was conducted using the PubMed and Google Scholar databases. The primary search terms included "ketogenic diet", "ketogenic diet and health", "ketogenic diet mechanisms" and "ketogenic diet adverse effects". The review primarily focused on studies published in the past five years, with particular emphasis on clinical trials, double-blind randomized controlled trials, meta-analyses, reviews, and systematic reviews.

State of knowledge

Foundational concepts of the ketogenic diet

An officially established classification of ketogenic diets that clearly defines the macronutrient composition does not exist. Up to the present, four primary forms of the ketogenic diet have been identified. The first, known as the classical ketogenic diet, is characterized by a fat-to-protein and carbohydrate ratio of 3:1 or 4:1 (in grams). This results in 87% and 90% of the daily caloric intake being provided by fats, predominantly derived from long-chain triglycerides (LCTs). This dietary approach must be gradually introduced to allow the body to adapt to utilizing fatty acids as the primary energy source, with no necessity for restrictions on energy intake or fluid consumption [1,7]. The second variant is the Medium-Chain Triglyceride (MCT) diet, which is based on medium-chain triglycerides. This

diet should provide 73% of total energy from fats, with MCTs contributing 30-60% of the total caloric intake. The advantage of this version of the ketogenic diet lies in the enhanced efficiency of ketone body production, which allows for an increase in carbohydrate and protein intake, thus reducing the proportion of fats. This significantly facilitates adherence to the diet [2]. The third variant is the Modified Atkins Diet (MAD), wherein fats account for 65% of the total caloric intake, and the ratio of fats to protein and carbohydrates may range from 1:1 to 2:1 (in grams). This flexible approach to macronutrient distribution, combined with the absence of restrictions on protein intake, fluid consumption, and total caloric intake, allows for more effective control over the diet [7]. The final main form of the ketogenic diet, the low glycemic index (GI) diet, emphasizes the use of foods with a glycemic index below 50 and should provide 60% of the daily caloric requirement from fats. This classification does not include numerous novel variations of the ketogenic diet that have emerged in response to the growing interest in this form of nutrition, such as the Mediterranean ketogenic diet [1].

The fundamental principle of the ketogenic diet is the replacement of carbohydrates with high-fat components. Glucose no longer serves as the primary energy source, glycolysis is inhibited, and the process of ketogenesis is upregulated [2]. The decrease in blood glucose and insulin levels activates hormone-sensitive lipase, leading to the release of free fatty acids (FFAs) from adipocytes. These are transported to the liver, where they undergo β-oxidation in the mitochondria of hepatocytes, resulting in the production of acetyl-CoA. In the absence of sufficient carbohydrates, due to a deficit in oxaloacetate used in gluconeogenesis, acetyl-CoA cannot enter the Krebs cycle and is instead converted into ketone bodies, including acetoacetate, β-hydroxybutyrate, and acetone. These ketone bodies are utilized as alternative energy sources, particularly for the brain, skeletal muscles, and heart. Furthermore, ketone body production may exhibit additional roles as signaling mediators or modulators of inflammation and oxidative stress [8]. During the ketogenic diet, ketone body concentrations typically range from 0.5 to 3 mM. In contrast, on a standard high-carbohydrate diet, this concentration usually does not exceed 0.3 mM. Nutritional ketosis differs from ketoacidosis, a condition where the body's buffering capacity is overwhelmed, and uncontrolled ketone body levels exceeding 10 mM lead to a reduction in blood pH and disruption of systemic homeostasis [9].

The impact of a ketogenic diet on body weight reduction.

The ketogenic diet may offer several advantages in terms of weight loss compared to other dietary interventions. Studies have shown that the total daily energy expenditure in individuals adhering to a KD increases by approximately 400–600 kcal/day, which is

attributed to the enhanced rate of gluconeogenesis under ketogenic conditions [2,10]. Furthermore, the KD facilitates weight reduction by decreasing hunger and enhancing satiety, even during a negative energy balance. The mechanism of appetite suppression is multifactorial and involves the direct effects of ketone bodies, elevated levels of circulating free fatty acids (FFAs), and alterations in anorexigenic and orexigenic hormones and neuropeptides. During adherence to the KD, the secretion of cholecystokinin (CCK) and glucagon-like peptide-1 (GLP-1)—known as satiety peptides—increases, while the production of ghrelin, a hunger-stimulating hormone, decreases [2,10,11]. This represents a significant advantage over traditional low-calorie diets. Notably, the initial phase of a high-fat diet often leads to substantial weight reduction. Within the first two weeks, a body weight decrease of up to 4.5 kg may be observed. This is primarily due to two mechanisms. Firstly, reduced carbohydrate intake leads to lower insulin concentrations, which in turn diminishes sodium retention in the kidneys. As a result, increased sodium excretion leads to greater water loss. Secondly, the depletion of glycogen stores in the liver and muscles under carbohydrate-restricted conditions results in further fluid loss, as glycogen is stored with water. These rapid initial effects may have psychological benefits, potentially motivating patients to maintain dietary adherence. Evidence also suggests that KD may reduce binge-eating behaviors and food addiction symptoms, as well as improve mood and cognitive function—factors of particular importance for individuals struggling with obesity and excess body weight [12]. Compared to carbohydrate-based meals, ketogenic meals cause minimal fluctuations in blood glucose and insulin levels, thereby improving overall glycemic control and reducing insulin resistance—a common obstacle to weight loss in obese individuals. Lower insulin levels, due to its anabolic nature, allow for more efficient utilization of fat as an energy source by promoting the release of fatty acids from adipose tissue and their subsequent oxidation [5,10]. The most effective weight loss and fat mass reduction outcomes have been observed with hypocaloric ketogenic diets, as demonstrated by numerous randomized controlled trials. These studies consistently show superiority over other dietary strategies, even when caloric intake is comparable between groups or when participants are allowed to eat ad libitum [1,12].

Ketogenic dietary intervention in the treatment of type 2 and type 1 diabetes

The basis of treatment for type 1 diabetes before the discovery of insulin was a low-carbohydrate starvation diet, which gradually lost its significance over time. Currently, the ketogenic diet is gaining interest as an adjunctive therapy for the treatment of type 2 diabetes. The benefits of ketogenic meals are multifaceted. Observed effects in numerous studies include weight loss, stabilization of blood glucose and insulin levels, reduction in glycated

hemoglobin (HbA1c) concentration, and increased tissue sensitivity to insulin, as well as a reduction in the HOMA-IR index [5,13,14]. Simply reducing carbohydrate intake in the diet yields beneficial results, but it seems that the full therapeutic effects can only be achieved by leading the body into a state of ketosis. An important issue arises with the potential for reducing the doses of antidiabetic medications or even discontinuing them entirely. However, this requires confirmation through carefully planned studies [15,16]. Scientific evidence shows that the positive impact of the ketogenic diet on glucose-insulin metabolism diminishes over time. This can be explained by the difficulty in maintaining such strict dietary restrictions long-term, which leads to a decline in the effectiveness of the intervention [17]. Studies indicate that concerns about potential side effects of the ketogenic diet in patients with T2DM are actually disproportionate compared to the number of observed cases. Proper monitoring of the patient's health and appropriate dietary adjustments minimize the risk of negative effects [13]. The use of a ketogenic diet in the treatment of type 1 diabetes requires further research. Limited literature suggests possible benefits, including stabilization of blood glucose levels, reduction of HbA1c concentrations, a positive impact on pancreatic function, and anti-inflammatory effects. A low-carbohydrate diet may reduce insulin doses and improve the quality of life in patients. However, the risk of severe hypoglycemia and diabetic ketoacidosis in this patient group should be considered, and appropriate medical supervision must be ensured [13,18].

Influence of ketogenic dietary patterns on cardiovascular health

Cardiovascular diseases are the leading cause of death worldwide and represent a major health issue. It has long been believed that a high-fat diet rich in saturated fats is unhealthy and contributes to the development of diseases by increasing LDL levels, leading to an accelerated process of atherosclerosis. This view directly informs dietary recommendations by physicians, who advocate for low-fat diets that limit saturated fat intake to less than 10%, providing over 60% of daily energy intake from carbohydrates [5]. However, the effect of the ketogenic diet on risk factors is not so straightforward and may be more beneficial than previously thought. Randomized controlled trials indicate that a single marker like LDL cannot fully predict cardiovascular risk, and a more holistic approach is required, considering other biomarkers and metabolic interactions. Furthermore, scientific evidence suggests that despite observing an increase in total cholesterol and LDL fractions, the LDL particle profile improves. The amount of small dense LDL, which has the greatest atherogenic potential, decreases in favor of larger particles. Some studies show that the ketogenic diet may positively influence the lipid profile by lowering triglycerides and raising HDL levels, without

significantly affecting LDL [3,5]. Scientific facts clearly confirm that individuals with overweight or obesity and type 2 diabetes benefit greatly in terms of improving cardiovascular risk factors. Ketogenic meals lead to significant improvements in the lipid profile in this group of patients. Furthermore, the reduction of excessive body weight, lowering of blood pressure, and stabilization of glucose-insulin metabolism are also noteworthy. There are reports suggesting that maintaining a low-carbohydrate diet long-term may be challenging, and its positive effect on HDL and triglycerides may diminish over time. Further well-designed studies on the long-term effects of the ketogenic diet on lipid profiles are necessary [19,20,21]. One potential advantage of the ketogenic diet, contributing to its cardioprotective properties, is its anti-inflammatory effect. Inflammation is a key factor in the early stages of atherosclerosis development. It has been proven that increased levels of pro-inflammatory cytokines correlate directly with an increased risk of cardiovascular disease. Several mechanisms of the anti-inflammatory effect of a low-carbohydrate diet can be identified. First, the state of nutritional ketosis induces a systemic immunomodulatory effect that is anti-inflammatory due to a series of biochemical and physiological changes. Secondly, the removal of simple sugars from the diet, which exacerbate inflammatory responses, plays a crucial role. Moreover, the high intake of unsaturated omega-3 fatty acids, whose cardioprotective and anti-inflammatory properties are well-documented, significantly supports the reduction of cardiovascular disease risk. The pleiotropic effect of ketone bodies also seems to extend to their impact on the endothelium. They modulate endothelial cells, improving their function and delaying their aging [22,23]. Current scientific evidence indicates a promising potential for the ketogenic diet in preventing and treating cardiovascular diseases, justifying the need for further research in this field. These results may contribute to improved health and a reduced risk of death worldwide.

Therapeutic Mechanisms of the Ketogenic Diet in Cancer Treatment

Cancer is the second leading cause of death worldwide, following cardiovascular diseases. Interventions aimed at reducing the risk of cancer occurrence and supporting its therapeutic process are the subject of numerous studies. The ketogenic diet appears to possess potential anticancer properties by limiting glucose availability, the primary energy source for cancer cells. The Warburg effect, characterized by the preference for glycolysis as the main pathway for ATP production, is a hallmark of cancer metabolism. Carbohydrate deficiency induces metabolic stress in cancer cells, drastically reducing glucose levels and forcing them to switch to ketone body metabolism. The reduced ability of cancer cells to adapt to ketosis makes them more susceptible to energy deficits. Chronic metabolic stress induced by a low-

carbohydrate diet inhibits cell proliferation and enhances apoptosis, ultimately slowing tumor development [5,24]. Limiting carbohydrate intake may also inhibit cancer progression by weakening insulin and IGF-1 signaling. Elevated levels of these hormones are associated with increased tumorigenesis and more aggressive disease progression. The low glucose availability in the ketogenic diet reduces insulin and IGF-1 levels, disrupting signaling pathways that regulate cancer cell growth and division [19,25]. Additionally, high-fat meals, through their influence on glucose and lipid metabolism, intensify oxidative stress in cancer cells. The ketogenic diet improves mitochondrial oxidative phosphorylation, but due to defects in the electron transport chain in cancer cells, it results in increased formation of superoxide anion radicals and reactive oxygen species. Due to a lack of protective mechanisms against oxidative stress, vulnerable cancer cells undergo damage and apoptosis, potentially inhibiting tumor growth [19,24,26]. Chronic inflammation promotes cancer development by hindering the immune system's ability to detect cancer cells. Available data suggest that the ketogenic diet may exhibit anti-inflammatory effects by regulating signaling cascades and cytokine levels. Beta-hydroxybutyrate (βHB), one of the ketone bodies, has strong anti-inflammatory properties and plays a significant role in several signaling pathways such as mTOR, TNFα, IGF, and PI3K. Observations have shown that βHB can limit tumor growth by inhibiting epithelial cell proliferation. Furthermore, ketosis increases the activity of NK cells and T lymphocytes while weakening the effect of immunosuppressive factors within tumors, facilitating immune system detection of cancer cells [24,27]. Preclinical animal model studies confirm that the ketogenic diet, due to its pleiotropic effects, has the potential to reduce cancer risk and even slow tumor growth, reverse cancer cachexia, and extend survival. Human data comes from small clinical trials, observational studies, and case studies, so their reliability is limited. There is a need for further well-designed advanced clinical trials in larger patient groups to unequivocally determine the effect of the ketogenic diet on cancer development, identify patients who may benefit the most, and define which dietary strategies are most effective [5,28,29].

Therapeutic Potential of the Ketogenic Diet in Neurological Diseases

The first paper on the positive impact of the ketogenic diet on the treatment of epilepsy in children was published by Dr. Russell Wilder nearly 100 years ago. Until the 1940s, this dietary approach was the dominant method for treating epilepsy in children and adults, leading to a reduction in seizure frequency by approximately 50%. The widespread use of anticonvulsant drugs caused the diet to lose significance, and it began to be considered only as adjunctive or alternative therapy. In the 1990s, its popularity surged again, especially for

treating drug-resistant epilepsy [2,30]. The mechanisms of the antiepileptic effect of low-carbohydrate meals have not been fully understood, although many potential pathways seem to exist. Ketone bodies, as an additional energy source, may improve mitochondrial function through increased ATP production, leading to stabilization of synaptic activity. Ketones, by hyperpolarizing the cell membrane, reducing the release of excitatory neurotransmitters, and increasing the production of inhibitory neurotransmitters, as well as activating K2P potassium channels, can reduce neuronal excitability. Additionally, during the ketogenic diet, there is an observed increase in neurotrophic factors, such as BDNF, GDNF, and NT-3, alongside a reduction in pro-apoptotic and pro-inflammatory factors. The state of ketosis also exhibits neuroprotective effects by increasing levels of calbindin and glutathione, reducing oxidative stress, and inhibiting mTOR and Nrf2 pathways [2,31]. Many studies confirm the efficacy of the ketogenic diet in reducing seizures in children and adolescents with drug-resistant epilepsy. However, data on adults are scarce. For ethical reasons, the ketogenic diet is always introduced as an adjunctive therapy, and during its use, there is the potential for reducing or even discontinuing anticonvulsant medications. Critics of this approach emphasize the need for a randomized, double-blind, placebo-controlled clinical trial to confirm its effectiveness [31,32].

Alzheimer's disease (AD) is a chronic neurodegenerative disorder and the most common form of dementia. It is characterized by the deposition of β-amyloid and tau proteins, which aggregate to form neurofibrillary tangles that damage brain cells, leading to cognitive dysfunction [2]. The ketogenic diet may have a beneficial effect on AD, whose pathogenesis is linked to brain insulin resistance and glucose metabolism dysfunction, hence the term "type 3 diabetes." Ketosis provides an alternative energy source for the brain in the form of ketone bodies, compensating for glucose deficiency. The diet may also reduce insulin resistance and influence neurodegenerative processes by reducing amyloid plaque deposition, improving mitochondrial function, lowering inflammation, and supporting neurogenesis. It has been observed that supplementation with MCT oil further supports ketone body production and may improve cognitive functions. Clinical studies have shown improved intellectual functions, daily functioning, and quality of life in AD patients following the ketogenic diet. Side effects were mild, and some patients continued the diet after the studies concluded [7,30,33].

Parkinson's disease (PD) is a progressive neurodegenerative disorder with an increasing prevalence, particularly in the elderly population. PD symptoms result from the loss of dopaminergic neurons in the substantia nigra, including tremor, bradykinesia, and neuropsychiatric disorders. The pathogenesis of the disease is not fully understood but

involves mechanisms such as chronic inflammation, mitochondrial dysfunction, oxidative stress, and accumulation of α-synuclein [33]. The ketogenic diet, as a source of ketone bodies, may counteract these processes through neuroprotective and anti-inflammatory effects. Studies in animal models and humans have shown that the diet may improve both motor and non-motor functions, reduce anxiety and depression symptoms, improve metabolic parameters, and positively influence the gut microbiome. Some studies also observed improvements in memory and voice quality. Although the ketogenic diet does not replace pharmacotherapy, it can support its effects, including enhancing the bioavailability of levodopa. Side effects are usually mild. Given the promising results of preclinical and clinical studies indicating the multifaceted benefits of the ketogenic diet in PD patients, there is an urgent need for further, well-designed clinical trials in larger patient groups to fully assess the efficacy, safety, and long-term effects of such a dietary intervention [2,30,33].

Potential Adverse Health Impacts of the Ketogenic Diet

Despite documented therapeutic benefits, the ketogenic diet is associated with the risk of adverse effects, particularly with long-term use or improper dietary balancing. One of the most common transient side effects is a phenomenon known as “ketogenic flu.” It is characterized by symptoms such as headaches, dizziness, fatigue, irritability, and sleep disturbances, which persist for the first few days or weeks of following the high-fat diet. Although these symptoms may be uncomfortable, the complication does not pose a threat to health and subsides after the body adapts to the new dietary regimen [35,36]. Gastrointestinal disturbances are commonly observed during the ketogenic diet, including nausea, vomiting, abdominal pain, diarrhea, and constipation. These result from excessive fat intake but also from fiber deficiency, which plays a crucial role in proper bowel function [7,33]. The restrictive nature of the low-carbohydrate diet carries the risk of vitamin and mineral deficiencies during long-term adherence. Lack of professional dietary support and proper food choices can lead to inadequate growth rates in children and osteopenia, associated with vitamin D deficiency and insufficient calcium intake. The use of the ketogenic diet should be supported by appropriately chosen supplementation with sugar-free vitamins from the B group, whose intake is limited on this diet. To ensure proper metabolic balance, supplementation with long-chain polyunsaturated omega-3 fatty acids and certain trace elements, such as selenium, zinc, calcium, and L-carnitine, is also recommended [1,9]. Research indicates that the use of the ketogenic diet may also carry the risk of kidney stones in children, suggesting the need for preventive measures. The use of potassium citrate may reduce this risk and should be considered as part of standard supportive care [7]. Nutritional

ketosis reduces appetite due to its impact on hunger and satiety centers through hormonal and neuronal mechanisms. Additionally, the characteristic monotony and low sensory appeal of the ketogenic diet may increase reluctance to eat meals, reducing motivation to continue the diet, especially in its classic form. In the context of treating obesity, this phenomenon is beneficial, promoting an energy deficit. However, in patients with malnutrition, particularly those suffering from cancer or neurodegenerative diseases, this effect may be undesirable and lead to further deterioration of nutritional status [1,9]. Furthermore, the ketogenic diet may predispose individuals to hypoglycemia and acid-base imbalances, leading to metabolic acidosis. Cases of pancreatitis and liver inflammation have also been reported. Biochemical profiles may show hyperuricemia, hypomagnesemia, hyponatremia, and hyperlipidemia. However, the definitive impact of ketogenic meals on lipid profiles requires further research. Some researchers suggest that in the case of significant increases in total cholesterol and triglycerides, atorvastatin should be included to inhibit endogenous cholesterol production and reduce fat intake to 20-25% [7,9]. Although the ketogenic diet demonstrates therapeutic potential for many conditions, its use is not recommended for all patients. The introduction of this nutritional model should be preceded by a thorough health evaluation and consultation with a physician and dietitian, especially in the presence of concurrent cardiovascular, liver, kidney, or endocrine disorders. Proper dietary balancing is also critical to prevent nutrient deficiencies and minimize the risks of adverse effects from long-term use of this dietary approach.

Summary

The ketogenic diet demonstrates a broad therapeutic spectrum. In metabolic diseases such as obesity and type 2 diabetes, it leads to significant weight reduction, stabilization of glycemia, and decreased insulin resistance. In type 1 diabetes, it may allow for a reduction in insulin doses, although it requires strict monitoring for the risk of hypoglycemia and ketoacidosis. In cardiovascular diseases, improvements in the lipid profile (reduced triglycerides, increased HDL) are observed, although changes in LDL levels may be varied, highlighting the need for a comprehensive risk assessment. In oncology, the ketogenic diet may inhibit tumor cell growth by reducing glucose availability and insulin levels, as well as increasing tumor sensitivity to cytotoxic therapies. In neurodegenerative diseases — including drug-resistant epilepsy, Alzheimer’s disease, and Parkinson’s disease — ketones serve as an alternative fuel for neurons and exhibit neuroprotective, anti-inflammatory, and antioxidant effects. However, long-term or improperly balanced use of the ketogenic diet may lead to adverse effects, such as "ketogenic flu," gastrointestinal disturbances, kidney stone risks, and

deficiencies in vitamins and minerals. Difficulties in maintaining the diet long-term may limit its effectiveness in treating chronic conditions. Therefore, the introduction of the ketogenic diet should be under strict medical and dietary supervision, with appropriate monitoring of metabolic parameters, supplementation, and individualized dietary adjustments. Further, well-designed clinical trials are necessary to assess the long-term benefits and risks and optimize dietary strategies for different diseases.

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All authors have read and agreed with the published version of the manuscript.

Conflict of interest
The authors report no conflict of interest.
Financial disclosure
The study did not receive any funding.
Institutional Review Board Statement
Not applicable.
Informed Consent Statement
Not applicable.
Data Availability Statement
Not applicable

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