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Role of ketogenic and low-carbohydrate diets in the pathogenesis and treatment of obesity and diabetes mellitus type 1 and 2 - A Literature Review

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ABSTRACT

Introduction: Ketogenic and low-carbohydrate diets are characterized by a significant reduction in daily carbohydrate intake, resulting in ketone bodies becoming an alternative energy source to glucose. Studies over the past decade have confirmed the therapeutic potential of the ketogenic diet in such disease entities as diabetes, polycystic ovary syndrome, acne, cancer, neurological diseases. In recent years, there has been a marked increase in patients with obesity and diabetes mellitus type 2, which has increased the number of studies on the impact of a carbohydrate-restricted diet on the pathogenesis and therapeutic process in this disease. This paper comprehensively analyzes the pathogenesis of obesity, diabetes mellitus type 1 and 2, and the effect of carbohydrate-restricted diet on these disease.

Aim of the Study: The purpose of this study is to examine the effects of low-carbohydrate and ketogenic diets on the pathogenesis and treatment of obesity, type 1 and 2 diabetes mellitus.

Materials and Methods: An analysis of papers available in PubMed was performed.

The following keywords were used : non-carbohydrate diet, ketogenic diet, obesity, diabeletes mellutis type 1, diabetes mellutis type 2

Basic results: The ketogenic diet has a proven effective effect in reducing body weight, and thus shows a preventive effect on the development of type 2 diabetes. Low-carbohydrate diets improve glucose control and lower HbA1c levels. In addition, such a dietary pattern can help reduce the dose of hypoglycemic medications.

Conclusion: According to the recommendations, there is no single optimal nutritional model in patients with diabetes, while the use of low-carbohydrate and ketogenic diets can lead to the desired results and achieve therapeutic goals in patients with diabetes.

Keywords: low-carbohydrate diet, ketogenic diet, obesity, diabeletes mellutis type 1, diabetes mellutis type 2

Introduction

Diabetes is a chronic metabolic disease associated with hyperglycemia that poses a major public health challenge. There has been a significant increase in the number of patients in recent decades, and IDF projections indicate that by 2045 about 783 million people, or one in eight adults will have diabetes. In Europe, more than one-third of adults (36%) with diabetes, do not have a diagnosis [1]. More than 90% of diabetes cases are type 2 diabetes, where preventive measures are possible to prevent the disease. The main reasons for the increase in the number of patients with diabetis are urbanization, an aging population, a decrease in physical activity levels and an increase in the prevalence of overweight and obesity [2]. Growing at a very fast rate, the prevalence of overweight and obesity is responsible for many health consequences such as cardiovascular disease, diabetes, cancer, and negative psychological and social effects. The primary strategy for treating type 2 diabetes is weight loss, and the resulting benefits are not limited to glycemic control - there is also a reduction in other modifiable risk factors for developing cardiovascular disease[1,2,3].

A large number of scientific studies confirm the effectiveness of low-carbohydrate and ketogenic diets in reducing body weight. In medicine and dietetics since 1960, these diets have become very popular as treatments for overweight and obesity. In 1920, the ketogenic diet began to be used in patients with drug-resistant epilepsy, and the effectiveness of such treatment has been confirmed by numerous studies and is included in current standards for the treatment

of this disease entity.

In addition studies over the past decade have confirmed the therapeutic potential of the ketogenic diet in such disease entities as diabetes mellitus, polycystic ovarian syndrome, acne, cancer and neurological diseases. The premise of the ketogenic diet is to strictly limit carbohydrate intake to 10-15 grams per day, provide one gram of protein per kilogram of body weight and cover the remaining energy needs from fat. Low-carbohydrate diets provide less than 45% of daily caloric needs from carbohydrates, 50-60% from fats and about 20-30% from protein[4,5].

Type of diet	Carbohydrate	Fat	Protein
Ketogenic diet	10-15g per day	cover the remaining energy needs	1g per kilogram of body weight
Low-carbohydrate diet	Less than 45%	50-60%	20-30%

Pathogenesis of obesity

There are two models to explain the pathogenesis of obesity. The most popular energy balance model is based on the principle of thermodynamics: when energy intake exceeds energy expenditure, weight gain occurs. In this model, according to the scientific statement of the endocrine society, “a calorie is a calorie”, so consuming excessive amounts of high-calorie food leads to weight gain, regardless of the percentage distribution of macronutrients. However, the type of food consumed affects the total number of calories consumed. Diets rich in simple sugars and processed foods tend to be more caloric than those based on whole and minimally processed foods. Diets with plenty of fiber-rich fruits and vegetables reduce total caloric intake due to feelings of satiety. The process of “programming” body weight to a higher value is also involved in the pathogenesis of obesity, which explains why lost weight tends to return after a certain period of time - the so-called yo-yo effect. This mechanism causes increased body weight to become protected by the body, and therapeutic methods targeting this pathological process have the potential to successfully treat obesity [6].

The second carbohydrate-insulin model is an alternative view, which posits that a high-carbohydrate, low-fat diet causes postprandial hyperinsulinemia. Hyperinsulinemia leads to increased glucose uptake by cells and fat deposition. As a result, the availability of energy for the body is reduced, the person feels hungry which leads to overeating and consumption of

excessive calories. In this model, overeating is a consequence of increasing obesity, not the main cause [7]. According to this theory, food with different percentages of macronutrients has different effects on hormones, metabolic pathways, gene expression and the gut microbiome which can potentially affect fat storage.

State of ketosis

Under physiological conditions, carbohydrates are the main source for energy production. After a few days of drastically reducing carbohydrate intake, glucose stored in the form of glycogen is depleted. As a result, the body is forced to produce energy from fats, which are broken down into free fatty acids. Free fatty acids are unable to cross the blood-brain barrier as a result of which they do not deliver energy to the brain [8]. Energy must therefore be supplied by ketone bodies, which are produced from fatty acids in the mitochondria of liver cells. The main ketone body produced in the liver is acetoacetate, and the main circulating ketone in the blood is Beta-hydroxybutyrate. Under normal conditions, the concentration of ketone bodies is very low <0.3mmol/l compared to the glucose concentration of about 4mmol/l. When the concentration of ketone bodies reaches about 4mmol/l they are used as an energy source, since glucose and ketone bodies have a similar Michaelis constant for transport to the brain [9]. Ketone bodies are capable of producing more energy compared to glucose.

Ketosis is a physiological mechanism, there is no change in blood pH during it, which distinguishes it from pathological uncontrolled diabetic ketoacidosis, in which there is a drop in pH. Although there is a drop in glycemia in ketosis, it is maintained within physiological limits of about 65-80 mg/dl [9].

Blood levels	Normal diet	Ketogenic diet	Diabetic ketoacidosis
Glucose (mg/dl)	80-120	65-80	>200
pH	7,35-7,45	7,35-7,45	<7,3

Effect of ketogenic diet on weight loss

Numerous scientific studies conducted provide strong and irrefutable scientific evidence regarding the effectiveness of ketogenic diets in weight loss. The mechanism that leads to

weight reduction is not clearly accepted and explained by scientists. Some scientists believe that there are no metabolic benefits of low-carbohydrate diets, and that weight loss is caused by a reduction in caloric intake resulting from increased protein intake and its satiety effect. Consuming more protein raises the levels of appetite-suppressing hormones GLP-1, peptide YY and cholecystokinin, while at the same time there is also a decrease in levels of ghrelin, known as the hunger hormone.

Other researchers believe that the ketogenic diet is directly responsible for suppressing hunger. One study involved seventeen obese men who were randomly assigned to two different groups. The first group of men followed a high-protein-ketogenic diet with 4% carbohydrate content, and the second group followed a high-protein non-ketogenic diet with 35% carbohydrate content. The study lasted 4 weeks, and in the short term, the high-protein, low-carbohydrate ketogenic diet reduced hunger and significantly reduced food intake. As a result, weight reduction in men following the ketogenic diet was greater by 2kg [10].

Low-carbohydrate diets lower insulin levels consequently leading to increased lipolysis and breakdown of triglycerides in adipose tissue into fatty acids and glycerol. Plasma triglyceride levels are reduced with a long-term low-carbohydrate, high-fat diet, compared to a diet with a standard macronutrient distribution, as studies have shown. [11]. A study was conducted that compared the effects of a very low-carbohydrate (VLC) and low-fat (LF) diet on lipid profile and metabolism in 15 obese men (BMI 34 kg/m^2) in a randomized crossover study (2 × 6 weeks). Metabolic syndrome markers TAG, TAG/HDL ratio and glucose: significantly decreased only after the low-carbohydrate diet (-44%, -42%, -6%, respectively). In addition, weight reduction was greater with the low-carbohydrate diet [12].

In the initial phase of very low-carbohydrate diets, the brain must obtain glucose from gluconeogenesis, because the human body needs about 60-65 g of glucose per day, which is not covered from carbohydrate intake. The energy cost of gluconeogenesis is about 400-600 kcal per day, and if the protein needed for gluconeogenesis comes from the body then the energy cost increases even more due to the protein resynthesis taking place. Based on the results of the study, the researchers hypothesized that running gluconeogenesis generates means energy expenditure and this is the advantage of low-carbohydrate diets in the process of weight loss. However, further research is needed to clarify whether gluconeogenesis relies primarily on food-derived protein or an endogenous source of protein[13].

Another study comparing a very-low-carbohydrate ketogenic diet with a balanced, low-calorie mediterranean diet showed that both diets had the same effect on resting energy expenditure. In contrast, the very low-carbohydrate ketogenic diet led to a lower respiratory ratio and increased fat oxidation at rest, and the effect was long-lasting. In addition, the percentage decrease in body weight and body fat was significantly greater in the ketogenic diet group [14]. The popularity of the ketogenic diet, due to its effectiveness in weight reduction, continues to grow. However, the mechanism responsible for weight reduction has not been fully elucidated, and most likely results from several interacting processes.

The relationship between obesity and diabetes mellitus type 2

Increased body weight is associated with both the development and progression diabetes mellitus type 2 . Type 2 diabetes mellitus is a metabolic disease in which there is simultaneously a decreased response of the body to insulin (development of insulin resistance) and abnormal production of this hormone by the β cells of the pancreas.

The strongest independent factor in the development of type 2 diabetes is insulin resistance. According to studies, visceral adipose tissue is an independent risk factor for the development of insulin resistance and other elements of the metabolic syndrome [15,16]. Visceral adipose tissue releases free fatty acids, and their elevated levels reduce insulin-stimulated glucose uptake throughout the body. Skeletal muscle is 80% affected by this process, which demonstrates that the development of muscle insulin resistance occurs [17]. A long-term increase in FFA levels impairs insulin's ability to inhibit glycogenolysis and increases gluconeogenesis which further raises blood sugar levels and exacerbates insulin resistance.

Obesity is a disease in which there is chronic inflammation, leading to increased release of pro-inflammatory cytokines. Some of these substances impair peripheral tissue insulin sensitivity, they include: adipin, resistin, TNF -α, Il-6, MCP-1, PAI-1, angiotensinogen [18].

The development of insulin resistance results in greater insulin production - hyperinsulinemia develops. Excess insulin inhibits lipolysis, promoting further weight gain, and further exacerbates insulin resistance as receptors become less sensitive to consistently high levels of insulin. A vicious cycle of insulin resistance and obesity is created.

The reverse concept is also worth noting. It shows that insulin resistance in people with type 2 diabetes occurs primarily in the skeletal muscles of lean people predisposed to diabetes, even before the development of obesity. According to this theory, insulin resistance is not an effect, but a cause of excessive fat accumulation [19].

Type 2 diabetes is led to by pancreatic β-cell dysfunction and the resulting impairment of insulin secretion, which develops gradually. The initial impairment that becomes apparent during the course of the disease is the disappearance of the first phase of insulin secretion. There is also impaired pulsatile insulin secretion, which accounts for 75% of the total daily secretion of the hormone. It has been shown that pulsatile administration of exogenous insulin causes a greater hypoglycemic effect than continuous administration, which proves that the described disorder has pathogenic significance. Another disorder is the excessive production of proinsulin and degradation intermediates, whose biological effects are weak. The hypothesis, which is strongly supported by scientific research, is that hyperproinsulinemia is associated with increased stress on pancreatic beta cells under hyperglycemic conditions. Stimulation of the beta-cell secretory pathway by high glucose concentrations causes proinsulin to be secreted in an immature form before it is converted to insulin [20,21].

Low-carbohydrate and ketogenic diets in the treatment of diabetes mellitus type 2

Lifestyle modification by increasing physical activity to 150 minutes per week and an optimal diet are the cornerstones of type 2 diabetes treatment. Low-carbohydrate and ketogenic diets provide less glucose, consequently lower insulin levels and benefit the healing process of type 2 diabetes.

A systematic review including all known randomized trials of low-carbohydrate diets versus low-fat/high-carbohydrate diets between 2000 and 2007 found that low-carbohydrate and high-protein diets were more effective in reducing weight at 6 months and reducing the risk of cardiovascular disease [22].

In a 2020 review, the effect of the ketogenic diet on glycemic control, insulin resistance and lipid metabolism in patients with type 2 diabetes was studied. The meta-analysis performed included 13 studies and analyzed 567 patients. Following a ketogenic diet resulted in a mean decrease in fasting blood glucose of 1.29 mmol/L and a decrease in HbA1c levels by an average of 1.07% in patients. Such a decrease in HbA1c levels is comparable to the effect of blood glucose-lowering drugs, demonstrating the high effectiveness of the ketogenic diet in long-term glycemic control.In addition, a decrease in triglycerides, LDL cholesterol with an increase in HDL cholesterol could be observed in patients after following the ketogenic diet [23].

Another large review analyzed nine studies involving 734 patients with diabetes. The aim of the study was to compare the effectiveness of a low-carbohydrate diet with a normal or high-carbohydrate diet. The results of the meta-analysis showed that a low-carbohydrate diet

improves glucose control, lowers HbA1c, and lowers triglycerides and raises HDL cholesterol [24].

The 2022 meta-analysis included 8 studies that collectively included 611 participants with type 2 diabetes. 331 participants followed a ketogenic diet with an intake of less than 50g of carbohydrates per day, and the duration of the studies ranged from 3 months to 2 years. Seven studies evaluating key indicators of carbohydrate metabolism were included in the analysis: fasting glucose, HbA1c, fasting insulin and HOMA-IR. A significant decrease in HbA1c levels was observed in the ketogenic diet group compared to the control group, while there were no significant differences in fasting glucose levels or the insulin resistance index. The reduction in fasting insulin levels approached statistical significance suggesting a potential benefit in improving insulin sensitivity, but there is a need for further research to confirm this hypothesis [25].

A study comparing the efficacy of a novel digitally monitored continuous care intervention (CCI) with standard recommendations in the management of type 2 diabetes was also conducted. 262 patients were enrolled in the CCI, in which the goal was to achieve and maintain nutritional ketosis by initially limiting carbohydrate intake below 30g per day fill and providing 1.5g of protein per kilogram of body weight. The standard care group included 87 patients and received the American Diabetes Association's standard recommendations for nutrition, lifestyle and diabetes management. The following parameters improved more in the CCI group: HbA1c level, fasting glucose, fasting insulin, body weight, systolic blood pressure, diastolic blood pressure, triglycerides and hepatic alanine aminotransferase. There was a particularly positive effect in the CCI group on fasting glucose and insulin levels, as there was a reduction in patients' use of hypoglycemic drugs. There was a 62% reduction in insulin use, and patients were able to discontinue sulfonylurea derivative drugs altogether [26].

Other studies have been conducted showing that following a ketogenic diet can help reduce the need for hypoglycemic drugs. The 34 adults were divided into 2 groups: one group followed a very low-carbohydrate ketogenic diet (LCK) and the other a moderate carbohydrate, calorie - restricted, low-fat diet (MCCR). In the LCK group, all who were using a sulfonylurea or DPP-4 inhibitor discontinued these medications by 12 months after the start of the study, and 3 study participants discontinued metformin treatment. None of the study participants in the MCCR group discontinued the aforementioned medications. The LCK group also showed greater weight reduction and a greater decrease in HbA1c levels [27].

A smaller study examined the effects of a ketogenic diet in patients with newly diagnosed type 2 diabetes who were overweight or obese. The study included 60 patients who were divided into two equal groups. The study lasted 12 weeks and one of the groups followed the ketogenic diet and the other the classic diet used in type 2 diabetes. In the group following the ketogenic diet, as many as 9 patients achieved normal blood glucose levels compared to 2 patients in the control group (following the standard diet). It is noteworthy that 6 patients in the ketogenic diet group had to withdraw from the study because the diet proved to be too strict. At the end of the study, it was also checked how willing the patients were to continue their current eating habits. It turned out that participants in the ketogenic diet group were less willing to continue adhering to the diet compared to patients in the control group following a diabetic diet. Most of those in the ketogenic group complained that meals low in carbohydrates were unappetizing [28].

Type of diet	Carbohydrate	Fat	Protein
Ketogenic diet	30-50 g	130g	60g
Routine diet for diabetes	250-280g	20g	60g

In summary, all the studies cited above show that there is solid evidence supporting the effectiveness of the ketogenic diet in treatment type 2 diabetes [22,23,24,25,26,27,28]. First of all, the ketogenic diet has a proven effective effect in reducing body weight, and thus affects one of the main causes of the development of type 2 diabetes. In addition, the ketogenic diet has a beneficial effect by reducing fasting blood glucose and significantly decreasing HbA1c. In some patients, this diet can reduce the need for blood glucose-lowering drugs.

It is important to note that for some patients with type 2 diabetes, pregnant and breastfeeding women, patients with kidney disease, this dietary model may be contraindicated.[29] Another serious contraindication to this diet is if patients are taking SGLT-2 inhibitors, as this is associated with an increased risk of developing diabetic ketoacidosis.[6] An increased risk of diabetic ketoacidosis and hypoglycemic episodes also occurs in patients taking GLP-1 drugs, so patients taking this group of drugs should be closely monitored by physicians [30].

Diabetes mellitus type 1

In diabetes mellitus type 1, there is autoimmune damage to the β-cells of the pancreas which leads to absolute insulin deficiency. Autoantibodies appear several months before the clinical manifestation of diabetes. The process of destruction of pancreatic islets can be initiated by

some environmental factor, such as an infectious agent which in genetically predisposed people initiates the development of diabetes. The American Diabetes Association (ADA) has also included latent autoimmune diabetes in adults (LADA) among forms of type 1 diabetes.
The number of studies on the use of the ketogenic diet in patients with type 1 diabetes is small. One reason for the limited number of scientific studies is the fear of developing ketoacidosis in patients with type 1 diabetes after following a low-carbohydrate diet. In addition, articles have appeared confirming an increased risk of hypoglycemia when following a regimented carbohydrate-restricted diet [30,31].

Effects of the ketogenic diet on the pathogenesis of type 1 diabetes mellitus

The ketogenic diet may influence the development of type 1 diabetes, particularly affecting intestinal homeostasis and the process of autoimmunity. The process of destroying pancreatic beta cells may be associated with disruption of the intestinal microbiota, including a decrease in butyrate-producing bacteria and an increase in Bacteroides. The ketogenic diet increases levels of β-hydroxybutyrate, which has anti-inflammatory effects and may modulate immune responses, such as by reducing pro-inflammatory Th17 cells. In addition, butyrate has been suggested to have a protective effect on pancreatic cell autoimmunity and to act to support the integrity of the intestinal barier [5,30].

Low-carbohydrate and ketogenic diets in the treatment of diabetes mellitus type 1

The recommended treatment for type 1 diabetes is functional intensive insulin therapy (FIT), which should be started from the moment the disease is diagnosed. Prior to the discovery of insulin by surgeon Frederick Banting and medical student Charles Best in 1921, the only treatment option of diabetes mellutis type 1 was a diet with strict carbohydrate restriction of less than 10g per day. The development of devices to help control glycemia is very large. The rtCGM- real time continuous glucose monitoring and isCGM- intermittently scanned continuous glucose monitoring systems are increasingly used by patients. Despite the development of technology to assist patients in the treatment process, a common problem is the lack of achieved therapeutic goals. Current treatment standards place great emphasis on calibrating the insulin dose to the amount of food taken in by the patient, so diet is still a very important part of the treatment process.

A study was conducted in which ten patients with type 1 diabetes were randomly divided into two groups: one group followed a standard diet without carbohydrate restriction, and in the other group patients took in 75g of carbohydrates per day. The patients' HbA1c levels and daily insulin requirements were compared. In the group with reduced carbohydrate intake, there was a significant reduction in HbA1c levels-an average of 8.9-8.2%- and a reduction in daily insulin requirements - from 64.4 to 44.2 units per day [32].

A similar study analyzed 11 patients with type 1 diabetes who followed a ketogenic diet with less than 55g of carbohydrates per day. Patients showed a decrease in HbA1c levels and reduced glycemic fluctuations. Unfortunately, the incidence of hypoglycemia increased at the same time [33].

A major problem in the use of a low-carbohydrate diet in patients with type 1 diabetes is the restriction of certain foods which results in some patients being unable to adhere to the recommendations for long periods of time. The long-term effects and outcomes of the ketogenic diet in patients with type 1 diabetes are unknown, so patients should be under the continuous care of physicians. There is limited research-supported knowledge on the use of the ketogenic diet in type 1 diabetes, and further research is needed on the efficacy and safety of this dietary strategy.

Conclusions

In recent years, lifestyle diseases such as obesity and type 2 diabetes have led to disability in an increasing number of the population. The ketogenic diet has a proven effective effect in reducing body weight, and thus shows a preventive effect in the development of type 2 diabetes. Low-carbohydrate diets improve glucose control, lower HbA1c and triglyceride levels, and raise HDL cholesterol levels. In addition, such a dietary model can help reduce the dosage of hypoglycemic drugs, and in some patients it will be possible to stop taking medicaments. The number of scientific studies proving the benefit of a low-carbohydrate diet in patients with type 1 diabetes is limited. Patients with type 1 diabetes using this dietary strategy show a decrease in HbA1c levels and less glycemic fluctuation. Unfortunately, some studies show that patients may experience more frequent hypoglycemic episodes. According to the recommendations, there is no single optimal dietary model in patients with diabetes. The use of low-carbohydrate and ketogenic diets can lead to the desired results and achieve therapeutic goals for patients with diabetes.

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