

Popular Article

Ketogenic Diet: Its Impact on Lifestyle and Health

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Abstract

The ketogenic diet, characterized by high fat and low carbohydrate intake, induces ketosis. Initially proposed in the 1920s for diabetes and epilepsy, it emphasizes specific fat-to-carbohydrate ratios. Ketogenic diets have demonstrated efficacy in weight loss, impacting metabolism and insulin sensitivity. Glycerol from triglycerides generates glucose, benefiting central nervous system function. Ketone bodies, produced during fat metabolism, serve as a "super fuel" with potential benefits for various conditions. Ketogenic diet, coupled with exercise, shows mixed effects on cholesterol and cortisol levels. Ketogenic diets exhibit promises in addressing epilepsy by modulating nerve cell excitation. In cancer, the diet's glucose-limiting effects create an unfavorable environment for cancer cells, suggesting a potential adjuvant therapy. Despite its therapeutic potential, controversies persist regarding hormonal changes and outcomes. The ketogenic diet emerges as a multifaceted approach with diverse applications, warranting further research and clinical exploration.

Introduction

The ketogenic diet is a dieting plan with a high fat and low-carbohydrate content. It induces ketosis, where the body uses fat as its primary fuel source by significantly reducing carbohydrates and increasing fat and protein intake. Ketogenic diet was initially proposed in the 1920s as a therapeutic approach for diabetes and epilepsy. It underscores a specific ratio of fat to carbohydrate and protein, commonly in proportions like 4:1 or 3:1. When on a ketogenic diet, a significant quantity of fatty acids undergoes metabolism in the liver to generate ketones, such as β -hydroxybutyrate. These ketones enter the bloodstream, causing a state known as ketosis, characterized by a serum beta-hydroxybutyrate level exceeding 3 mmol/L. While in healthy individuals, insulin levels are adequate to curb ketone production, conditions such as diabetes (both type 1 and type 2), alcoholism, periods of hunger or extreme dieting, and certain metabolic disorders can result in an accumulation of excess ketones, leading to ketoacidosis.



Principles of ketogenic diet

The ketogenic diet typically consists of high-fat foods, moderate protein intake, and very low carbohydrate consumption, forcing the body to enter a metabolic state that promotes fatburning. The primary goal of the ketogenic diet is to induce a state of ketosis, where the body shifts from using glucose as its primary energy source to using ketones, which are produced from the breakdown of fats.

The ketogenic diet aims to decrease body fat and enhance metabolic health, showing potential benefits in diseases like diabetes and heart disease. The macronutrient distribution is typically 55-60% fat, 30-35% protein, and 5-10% carbohydrate. Limiting carbohydrate intake induces a shift in the body's energy production from glucose metabolism to ketosis. This leads to the utilization of ketone bodies derived from fat metabolism, establishing a metabolic state in which the body predominantly relies on fat as its primary source of fuel. Ketone bodies can be utilized as a source of energy by organs, including the brain, heart, and muscles. Production depends on factors like basal metabolic rate, and body fat percentage. Ketone bodies considered a "super fuel," produce more ATP than glucose, allowing the body to maintain efficient fuel production during a caloric deficit. Additionally, ketone bodies can reduce free radical damage and enhance antioxidant capacity. When carbohydrate intake is restricted, the body undergoes gluconeogenesis and ketogenesis. Gluconeogenesis produces glucose internally when glucose availability is low, and ketogenesis produces ketone bodies as an alternate energy source.

In the ketogenic diet, glycerol produced through the hydrolysis of triglycerides in the liver can generate 16–60% glucose, providing an initial energy source for the central nervous system. This process proves beneficial in lowering blood glucose and insulin levels while enhancing insulin sensitivity. Additionally, the ketogenic diet elevates ketone bodies, intermediate products derived from liver fat oxidation, directly suppressing appetite, and reducing calorie intake. Consequently, ketogenic diet is advocated for weight loss and as a treatment for obesity-related conditions like hypertension, cardiovascular disease, cancer, and sleep apnea.

Ketogenic diet and lifestyle

The ketogenic diet has demonstrated greater efficacy in weight loss compared to a standard diet with similar caloric deficits in trials spanning from three months to two years. The initial rapid weight loss upon starting the diet may result from the body's efforts to utilize glycerol from triglycerides in response to a shortage of dietary carbohydrates. This leads to a reduction in adipose fat stores and, consequently, weight loss. The conversion of glycerol into glucose through the gluconeogenesis pathway is a highly energy-demanding process, causing the body to expend



excess energy before adapting to using ketone bodies as its primary energy source. low carbohydrate and high fat content is

In conjunction with dietary changes, exercise serves as a crucial weight-loss strategy by increasing energy expenditure and reducing body fat accumulation. Limited evidence suggests that the ketogenic diet on its own or when combined with exercise intervention in individuals who are overweight or obese may lead to elevated levels of both LDL (low-density lipoprotein) and HDL (high-density lipoprotein), along with reduced serum concentrations of triglycerides compared to a conventional carbohydrate and fat intake. Elevated levels of HDL are linked to a decreased risk of cardiovascular disease, whereas heightened levels of LDL and triglycerides are significant risk factors for cardiovascular disease.

The pairing of a ketogenic diet with exercise has been observed to sustain elevated cortisol levels in men within two hours post-exercise. However, ketogenic diet by itself does not seem to influence cortisol levels, suggesting that this may be a typical physiological response to exercise. On the other hand, adopting a ketogenic diet without exercise intervention can result in a reduction in testosterone levels during periods of rest in men. Research indicates that men with lower total testosterone levels face a 44% higher risk of mortality and an increased likelihood of specific cardiovascular and respiratory diseases by more than 38%. Therefore, it is crucial to remain vigilant regarding potential changes in endocrine hormone levels associated with low-carbohydrate diets.

Ketogenic diet and diabetes

Studies have demonstrated that the ketogenic diet can enhance and potentially reverse insulin resistance in individuals with type 2 diabetes or those at risk of developing diabetes. Elevated insulin resistance has been linked to an increased conversion of glucose into triglycerides, which poses a risk for heart disease. Low low-carbohydrate diet proves effective with type 2 diabetes and enhances glycemic control. However, there is currently no evidence supporting the notion that a ketogenic diet can delay or prevent the onset of type 1 diabetes.

Ketogenic diet and epilepsy

Ketogenic diets have demonstrated greater efficacy in addressing generalized seizures in comparison to partial seizures. Epilepsy is linked to abnormal excitation of nerve cells, and one potential mechanism through which ketogenic diets may treat epilepsy is by enhancing the depolarization state of nerve cells. The injection of ketone bodies has been found to decrease susceptibility to seizures in mice, evidenced by reduced rates of spontaneous firing in histological sections. Moreover, ketone bodies can directly impede vesicular glutamate transport. The



inhibitory neurotransmitter γ -aminobutyric acid (GABA), derived from glutamate, plays a role in reducing membrane excitability. Numerous human and animal studies indicate that ketogenic diets elevate GABA levels and diminish glutamate levels in the brain.

Ketogenic diet and cancer

Cancer cells require substantial energy for their proliferation, exhibiting an increased uptake of glucose compared to normal cells. They convert this glucose into lactate through glycolysis, a process known as the Warburg effect. Laboratory studies have indicated that the absence of glucose can induce apoptosis in cancerous cells. The ketogenic diet, by limiting glucose intake, has the potential to hinder cancer cells from generating energy via glycolysis, offering a potential therapeutic approach for cancer treatment. Moreover, the decreased glucose intake promotes the production of ketone bodies. Some cancer cells exhibit a reduced ability to utilize ketone bodies as an energy source due to the decreased activity of ketolysis enzymes. Consequently, this results in a reduction in insulin and insulin-like growth factors, known drivers of cancer cell proliferation. In essence, the implementation of a ketogenic diet creates an unfavorable environment for cancer cells, potentially augmenting the antitumor effect.

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