



Muscle Metabolism and the Ketogenic Diet: Effects in Health and Disease

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ABSTRACT

Widespread therapeutic uses of dietary intervention include everything from the treatment of neurological conditions to efforts to lengthen life. The alteration in energy metabolism is the most significant result of different diets. Due to the fact that muscles account for 40% of the body's total mass and are one of the primary locations for absorbing glucose and energy, different diets predominantly affect their metabolism, resulting in both favorable and unfavorable changes to physiology and signaling pathways. In this review, we explore the potential effects of fasting, exogenous ketone body administration, and low-carbohydrate, high-fat diets (KD) on the energy metabolism of muscles. These interventions are all prospective therapeutic modalities for the management of a variety of disorders. The main effects of KD on muscle are seen in modifications to energy metabolism, specifically reduced carbohydrate and enhanced fat oxidation.

Keywords: Fasting Heart, Ketogenic Diet, Ketone Bodies, Muscle, Muscle Metabolism.

INTRODUCTION

Widespread therapeutic uses of dietary intervention include everything from the treatment of neurological conditions to efforts to lengthen life. The alteration in energy metabolism is the most significant result of different diets. Due to the fact that muscles account for 40% of the body's total mass and are one of the primary locations for absorbing glucose and energy, different diet predominantly affect their metabolism, resulting in both favorable and unfavorable changes to physiology and signaling pathways. In this review, we explore the potential effects of fasting, exogenous ketone body administration, and low-carbohydrate, high-fat diets (KD) on the energy metabolism of muscles. These interventions are all prospective therapeutic modalities for the management of a variety of disorders. The main effects of KD on muscle are seen in modifications to energy metabolism, specifically reduced carbohydrate and enhanced fat oxidation. This has an impact on the number of mitochondria, oxidative metabolism, antioxidant capacity, and enzyme function. The benefits of KD for muscles are still debatable, which may be due to its differing effects on distinct fiber types, particularly the ratio of different fiber types in muscles. Although the effects of KD or its mimics are generally positive, they can occasionally have negative effects such heart fibrosis. The KD has long been recognized as a remarkably effective dietary approach for the treatment of intractable epilepsy, and in the past ten years, it has attracted increasing research interest due to mounting evidence of its promising therapeutic potential for a number of diseases, including obesity and malignancies. We describe the experimental and/or clinical evidence of the effectiveness and safety of the KD in treating various diseases in this review, and we also address potential mechanisms of action based on recent developments in our knowledge of the impact of the KD at the cellular and molecular levels. We stress that several processes, some of which need additional explanation, may be used by the KD to function. As ketogenesis takes place in this process, keto bodies are formed as a result, and keto bodies are the best alternatives for the body's energy supply. Because consuming less carbohydrates when following this diet reduces the Amount of glucose produced by the metabolism of carbohydrates. Thus, the body begins using keto bodies as fuel, and B-hydroxybutyrate produces ATPs, our body's energy packets. Adipose tissue sequences single -cell RNA, causing gamma and T lymphocytes to become active and lessen adipose tissue inflammation. Thus, as a result of a rise in the mechanism of ketogenesis, inflammation does not develop in adipose tissue.

Various forms of the ketogenic diet:

The traditional long-chain triglyceride (LCT) KD: The most common variety of KD is the standard LCT, which has a 4:1 ratio of fat (in grams) to protein + carbohydrate. It is the most traditional type of KD and is frequently employed in the therapeutic Environment (in grams). A 3:1 or lower ratio may be used as fat makes up 90% of calories and is primarily sourced from food. Additionally

While older kids may benefit more from starting KD with a 4:1 ratio, then a lower ratio, low ratios are appropriate for commencement of KD in infants.

Medium-chain triglyceride (MCT) KD: The MCT KD offers more flexibility in diet ratios than the LCT KD, and the calories consumed are determined by the proportion of energy that comes from MCT. Additionally, clinical evidence supports the MCT and LCT KD's equal efficacy. The MCT KD, however, frequently causes gastrointestinal adverse effects.

Modified Atkins diet (MAD): The MAD is based on the Atkins diet, a popular weight management plan that has many of the same food options as the traditional KD but does not require exact ingredient measurements. No protein, fluid, or calorie restrictions apply to the MAD.

Low glycemic index treatment: The low glycemic index approach is based on the idea that the protective effect of KD depends on glucose levels remaining stable, but it uses a more flexible regimen with a low-carbohydrate composition to reduce glycemic increases.

Metabolic effects of the ketogenic diet:

During KD, blood lipid metabolism is frequently of concern. The majority of cellular energy is produced in the presence of oxygen through the process of glycolysis from pyruvate, which is produced when glucose is digested, and oxidative phosphorylation in the mitochondria. The breakdown of fatty acids generates cellular energy in the absence of glucose. Low-density lipoprotein (LDL), cholesterol, and triglyceride (TG) concentrations may rise as a result of a diet high in protein, low in carbohydrates, and low in fat.

In humans, glycerol, which is produced through TG lysis, and glycogenic amino acids are the two sources of glucose. The latter source becomes more crucial when someone is in ketosis. The primary source of glucose during the initial stages of the KD is glycogenesis produced from amino acids. A decrease in amino acid contribution is followed by an increase in glucose produced from glycerol. In contrast to the 60% glucose produced in the liver during several days of total fasting, TG-hydrolysis-induced glycerol during KD can actually generate more than 16% glucose.