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Research Article

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The Effects of Low Carbohydrates/Ketogenic Diets (Lc/Kd) On Anaerobic Training and Post-Workout Recovery

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ABSTRACT

Background: Athletes typically require balanced diets that are 50 % carbohydrates, 20 % protein, and 30 % fat. Many athletes believe eating low-carbohydrate and high-fat diets can help them perform better and support their competitive activities. The relationship between low-carbohydrate diets and anaerobic sports activities has received little attention. This study aimed to look at how a low-carbohydrate/ Ketogenic diet (LC/KD) affected anaerobic exercise, hormonal profiles, and metabolic responses.

Participants and Methods: Twelve Healthy athletes took part in this study and underwent the Wingate test twice: once after the normal diet ND (50 percent carbohydrate, 20% protein, 30% fat) and once after 4 weeks of the LC/KD (10 percent carbohydrate, 25 percent protein, 65 percent fat). Venous blood samples were taken before and after the workout to measure insulin, testosterone, GH, cortisol, blood lactate, β hydroxybutyrate, Adrenaline, Noradrenaline, and glucose concentrations.

Results: The LC/KD diet significantly increased testosterone and growth hormone (GH) concentrations (0.14 vs 0.25 ng/mL) and (495.3 vs 661 nmol/L), while insulin and cortisol levels decreased (5.21 vs 3.78 IU/mL) and (17.01 vs 16.31 g/dL). The LC/KD diet also reduced blood lactate levels at rest and after exercise (8.95 vs 7.03 mmol/L) while increasing β hydroxybutyrate (0.05 vs 0.173 mmol/L). Glucose levels were lower after consuming LC/KD compared to ND (97.1 vs 81.1 mg/dL). This study found that a CHO deficiency in the diet can cause hormonal and cellular changes that favor the utilization of non-esterified fatty acids and, to a lesser extent, amino acids. Increased Testosterone, Growth Hormone, and Insulin all have metabolic and anabolic effects.

Conclusions: Reducing CHO intake for three days increases many hormones such as growth hormone and testosterone whereas decreased insulin and other products. The concept of vasodilating muscle arterioles by modifying eicosanoid production. Eicosanoid, which is reportedly produced in the L- CHO diet, may be responsible for improved muscle oxygenation, which may benefit even the most competitive athletes.

Keywords: Low Carbohydrates Diet, Ketogenic Diet, Athletes, β Hydroxybuterate, Insulin, Glucose, Testosterone, Cortisol

Introduction

A change in diet has been shown to alter physical performance and the pattern of substrate utilization during exercise. Animal and human studies on the effect of a low-carbohydrate (L-CHO), fat-rich diet have revealed reduced carbohydrate stores and increased fat utilization by working muscles, as well as a decreased contribution of muscle glycogen to energy yield [1]. LCDs have recently made their way into the athletic arena and have piqued the interest of a few researchers as a potential mechanism for improving endurance performance. It has recently been debated what type of diet is best for an athletic population, and one recent approach in the scientific community has been to investigate the effects of LCDs, also known as high-fat diets, on performance parameters in athletes. This strategy has primarily targeted endurance athletes such as competitive cyclists and distance runners [2].

A common misconception among athletes is that a low-carbohydrate, high-fat diet will impair exercise performance. LCD proponents argue that this dietary practice provides a large amount of lipid as a substrate for ATP synthesis, potentially reducing reliance on limited muscle glycogen stores and, ultimately, delaying muscle glycogen breakdown during exercise [3].

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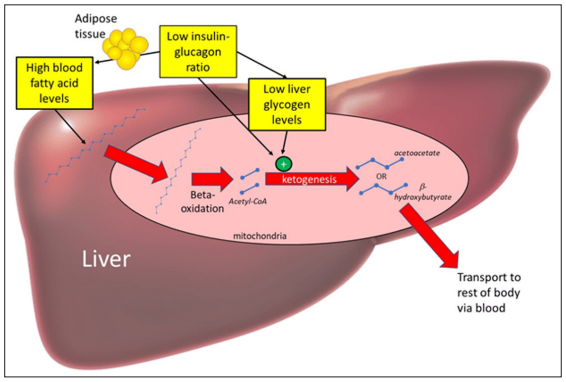
Endogenous carbohydrate stores from glycogen in skeletal muscle and the liver are considered finite, whereas fat from adipose tissue is considered a steady supply of energy. Elevated ketone bodies produced by the KD may serve as an alternative or supplemental fuel source to sustain endurance exercise [4]. Several studies have been conducted in the last decade to investigate the effect of low carbohydrate (LC) or KD (LC/KD) diets on endurance exercise performance in humans. The vast majority of the studies concentrated on endurance-trained individuals, primarily male athletes [5].

It is commonly assumed that endurance athletes require a carbohydrate intake of 7 to 10 g/kg/d to replenish muscle and liver glycogen stores and ensure adequate glucose availability for skeletal muscle contraction during endurance-type aerobic exercise [6,7]. Earlier research has shown that consuming a high-carbohydrate diet increases carbohydrate oxidation and muscle glycogenolysis during exercise]. Consuming a lot of carbs may provide enough substrate to fuel your daily training and competition needs, as well as potentially increasing the relatively low amount of carbohydrate stored in your body as glycogen [8].

During rest and exercise, the primary substrates for skeletal muscle metabolism are carbohydrates and fat. Their contribution to total oxidative metabolism is determined by several factors, including exercise intensity, duration, diet, and other variables such as training status [9]. LCDs cause metabolic and hormonal changes in exercising skeletal muscle that may improve fat oxidation and promote glycogen sparing. On an LCD, there is a shift toward a greater reliance on fat oxidation for fuel at rest and during exercise, similar to endurance training adaptations. This could be due to a combination of increased oxidative enzymes, increased mitochondrial density, increased intramuscular triglyceride storage and utilization, and enhanced muscular uptake of plasma-free fatty acids [10]. LCDs have also been shown to increase resting human skeletal muscle pyruvate dehydrogenase (PDH) kinase activity while decreasing the amount of active PDH, which reduces carbohydrate oxidation [11]. These combined mechanisms would reduce muscle glycogenolysis and carbohydrate oxidation while increasing the utilization of free fatty acids during exercise.
Low-carbohydrate diet affects performance during a 30-second Wingate test. This test estimates in addition to the performance indices measured in this study, blood lactate (LA), β-hydroxybutyrate (β-HB), and glucose concentrations were measured before and after exercise to understand better the mechanisms linking dietary changes to subsequent exercise performance [12].

Low-carbohydrate, high-fat diets may increase the use of fat as fuel during exercise, resulting in a glycogen-sparing effect and potentially improving endurance exercise capacity. The human body's low glycogen storage capacity, which is estimated to be 300 to 400 g in skeletal muscle and 70 to 100 g in the liver, limits the ability to maintain a high-power output during prolonged endurance exercise [13]. It has been suggested that an LCD may cause a decrease in pre-exercise muscle glycogen content, particularly in untrained individuals, which may defeat the purpose of creating the glycogen-sparing effect in the first place [14].

Exogenous fatty acid oxidation is a significant source of energy during low-to-moderate intensity exercise. The contribution of fatty acids to oxidative metabolism increases as the duration of the exercise bout increases during moderate-intensity exercise. Skeletal muscle requires a constant supply of exogenous substrates to fuel contraction during exercise. The liver produces glucose and ketone bodies through the processes of gluconeogenesis and ketogenesis, respectively. Lipolysis of adipose tissue maintains serum fatty acid concentrations [15].



The ketogenesis process

The contribution of fatty acids to oxidative metabolism varies depending on the intensity and duration of exercise. Exogenous fatty acid oxidation is a significant source of energy during low-to-moderate intensity exercise. The contribution of fatty acids to oxidative metabolism increases as the duration of the exercise bout increases during moderate-intensity exercise. In this regard, strategies that increase fatty acid availability may be critical to optimizing endurance exercise performance. The KD may be beneficial, particularly for aerobic endurance exercise, by encouraging the use of fat as fuel rather than carbohydrates [16].

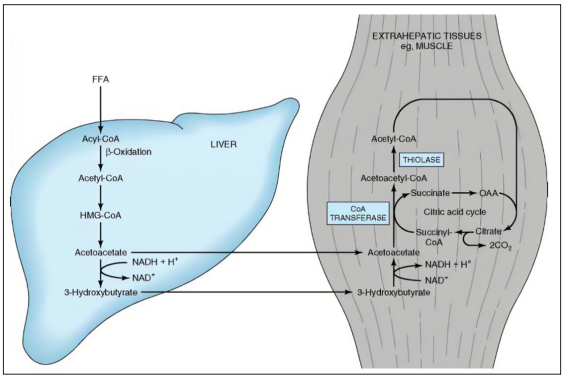
Despite the performance enhancement found in a few studies, some have shown that LCDs do not affect exercise performance in trained or untrained individuals [17].

On the other hand, demonstrated enhanced endurance capacity in endurance-trained individuals on an LCD despite lower pre-exercise glycogen levels [18]. The metabolic adaptations associated with a short-term LCD combined with an already enhanced oxidative system, including up-regulation of mitochondrial oxidative enzymes and increased mitochondrial density; suggest that endurance athletes may still be able to perform similar amounts of physical work on an LCD despite potentially more difficult perceived effort.

Its Influence on Aerobic Exercise Anaerobic exercise is a type of short-duration, high-intensity exercise that lasts less than 2 minutes. The phosphagen and lactic acid systems, which rely heavily on skeletal muscle glycogen, meet energy demands. High contractile forces occur within the Nutrients 2019, 11, 2296 7 of 16 muscles during anaerobic exercise, and muscle fibers are damaged. In addition to carbohydrate replenishment during the recovery period, adequate consumption of essential amino acids is required to support the protein synthesis required to repair and rebuild the muscle [19].

In this regard, LC/KDs typically provide enough protein (about 15% of daily calories) to avoid amino acid deficiency

[20]. However, because of the low carbohydrate intake, the increased reliance on amino acids in gluconeogenesis, as well as the impairment of glycogen-store restoration, may hurt anaerobic performance. Several studies examined the effects of LC/KDs on anaerobic performance in various populations, including endurance athletes, CrossFit participants, gymnasts, and powerlifters [21,22].



Utilization of 3-Hydroxybuterate by extrahepatic tissues (muscles)

Participants and methods

12 healthy athletes have enrolled in this study. The mean of their age was 27±6.4 years and the mean of their weights was 78.3 ± 6.6 kg and their mean Body Mass Index (BMI) was 24±4.8. The 12 subjects performed two all-out 30-second Wingate tests (WT; Bar-Or 1980) on a cycle ergometer (Monark Crescent, Varberg, Sweden) in a randomized order, followed by three days on either a (ND) normal diet (50 percent carbohydrate, 30 percent fat, and 20 percent protein) or a (LCD) low carbohydrate/ ketogenic diet containing (10 percent carbohydrate, 65 percent fat, and 25 percent protein). Both diets provided the same amount of energy, with athletes receiving 35 Kcal per 1 kg of body weight per day. To avoid caffeine or tobacco consumption, food was provided for the subjects in a room under observation, and the athletes did not participate in any competitive sports before the study but continued with their normal activities. On the day of the exercise testing, the subjects arrived at the Laboratory after fasting overnight. Half an hour before the exercise, a catheter was inserted into the antecubital vein for blood sampling. Venous blood samples were taken before the exercise and then again at 3, 15, 30, and 60 minutes later to determine LA and BG, plasma β-HB, concentrations.

Blood was drawn directly from the catheter and placed in heparinized tubes. Whole-blood aliquots for LA and β-HB measurements were deproteinized with perchloric acid immediately before centrifugation at 2,000 g. The supernatant was stored at 5°C and analyzed the same day or the next. Additional whole blood aliquots were deproteinized with uranyl acetate (URAC, Boehringer, Manheim, Germany) and centrifuged at 2,000 g for 10 minutes for BG determination.

The supernatant was kept at 5°C until the following day's analysis. The remaining heparinized blood was collected for IRI analysis of plasma. It was centrifuged for 10 minutes at 4°C at 2,000 g, and the plasma was kept frozen at (70°C until it was analyzed. Blood samples were transferred to ice-cold tubes containing an anticoagulant and antioxidant for catecholamine determination (ethyleneglycol-bis Coxonitrilo tetra acetate and glutathione).

The plasma was separated using refrigerated centrifugation, and samples were stored at (70°C) until analyzed. The plasma β-HB concentration was determined enzymatically. The levels of LA and BG in the blood were measured enzymatically using commercial kits (Boehringer, Mannheim, Germany). The data were collected for both diets at the beginning and at the end of the study. Means and percentages of changes were calculated and statistical analysis was performed using SAS/STAT software.

Results

The diets consumed by the athletes during this study were isocloric. The difference between the normal diet (ND) and low-carb diet was the percentage of carbohydrates and fat in both of them as shown in Table 1.

Table 1: Nutrient percentage of low carbohydrate and normal diet

Diet	Normal Diet (ND)	Low Carb Diet (LCD)
% Carbohydrates	50	10
% Protein	30	25
% Fat	20	65
Total Calories (Kcal)	2730±123.4	2718±112.2

The L-CHO / ketogenic diet significantly impacted the levels of many hormones. It increased testosterone and growth hormone (GH) concentrations (0.14 ±0.01vs 0.25±0.2 ng/mL) and (495.3±75.1 vs 661±88.7 nmol/L) and both hormones improved by 78.8 % and 33.5 % respectively and the difference was significant ($p < 0.05$). The data are presented in Table 2.

Table 2: Testosterone and growth hormone levels in athletes after consuming normal and low-carb diets

Hormone	Normal Diet (ND)	Low-carb Diet (LCD)	% change
Testosterone ng/mL	0.14±0.01	0.25±0.2*	78.8
Growth hormone nmol/L	495.3±75.1	661±88.7**	33.5

*,**The difference was significant ($p<0.05$)

Table 3, shows the results of this study which showed that the Low Carb Diet (LCD) had a clear effect on insulin and cortisol levels compared with the Normal Diet or ND.

Insulin levels plummeted by 37.8 % (5.21±1.1 vs 3.78±0.7 IU/mL) and the difference was significant ($p<0.05$). In contrast, cortisol levels decreased by only 4.6 % (17.01±2.9 vs 16.31±2.1 g/dL) compared with normal diet and the difference was not significant at $p<0.05$.

Table 3: The effect of normal and low carbohydrate diet on Insulin and cortisol levels

Hormone	Normal Diet (ND)	Low Carb Diet (LCD)	% change
Insulin IU/mL	5.21±1.1	3.78±0.7*	37.8
Cortisol g/dL	17.01±2.9	16.31±2.1**	4.6

*Significant difference ($p<0.05$)

**Non-significant difference ($p<0.05$)

The L-CHO diet also reduced blood lactate levels by 21.5% (8.95±2.3 vs 7.03±1.5 mmol/L) while the increase in hydroxybutyrate level was huge and it was around 250 % (0.05±0.01 vs 0.173±0.1 mmol/L). Glucose levels were lower after consuming L- CHO compared to ND (97.1±11.3 vs 81.1±7.6 mg/dL) and the decrease in glucose level was about 16.5 %. The differences in lactate, hydroxybuterate, and glucose levels were significant as shown in Table 4.

Table 4: The effect of normal and low carbohydrate diet on Lactate, Hydroxybuterate, and glucose levels

Product	Normal Diet (ND)	Low Carb Diet (LCD)	% changes
Lactate mmol/L	8.95±2.3	7.03±1.5*	21.5
Hydroxybutyrate mmol/L	0.05±0.01	0.173±0.1*	250
Glucose mg/dL	97.1±11.3	81.1±7.6*	16.5

*Significant difference $p<0.05$

Conclusion

A low carbohydrate or Ketogenic diet increases the levels of Testosterone and growth hormone. This increase can have a favorable effect on athletes' performance compared with a normal diet.

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