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Effects of a low carbohydrate diet on sports performance

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Abstract

Although high carbohydrate intake (>60%) is generally recommended for athletes, nowadays many experiments involve a low carbohydrate diet. Carbohydrate restriction leads to significant hormonal changes as well as reduced glucose utilization and increased utilization of free fatty acids and ketone bodies as energy sources. This narrative review aimed to discuss the physiological basis of low carbohydrate ketogenic diets (LCKD) and their both positive and negative effects on body composition, power, strength, aerobic capacity and anaerobic performance of athletes and physically active subjects. We searched and analyzed earlier and recently published papers on the subject. Research results showed that LCKD facilitates a reduction of body mass and fat mass while promoting maintenance of lean body mass (LBM). However, compared to a diet with a high carbohydrate content, it is challenging to increase LBM. Despite significant metabolic changes and increased fat oxidation LCKD did not show clear and convincing effects on endurance ability. While LCKD can preserve endurance performance in sports where intensity does not exceed 65-70% VO_2max, it is not superior compared to a diet high in carbohydrates. Also negative effects on aerobic capacity can be manifested, especially in women, which may be related to a lower status and transport of iron and due to the difference in fat oxidation between genders. Reduced availability of glucose, decreased glycolytic enzyme activity and metabolic inefficiency (higher oxygen consumption for fat oxidation compared to glucose oxidation) might impair anaerobic performance where the intensity exceeds 70-80%. It seems that LCKD has no particular effects on maximum strength, power and anaerobic lactate abilities because they depend on the phosphagen energy system.

KEYWORDS: sports performance, low carbohydrate diet, body composition, keto diet.

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Introduction

The application of a properly programmed diet in athletes is an extremely important factor that contributes to the achievement of sports results, because it greatly affects physical abilities, body composition, speed of recovery, replenishment of spent energy depots, electrolytes, etc. According to previous recommendations, the largest part of an athlete's diet should contain carbohydrates (CHO), which account for more than 60% of daily calorie intake [17]. Carbohydrates stored in the liver and muscles as glycogen are a readily available source of energy for prolonged moderate intensity physical activities and the primary source of energy for high-intensity physical activities (>80% of maximum oxygen consumption – VO_2max) [27]. In addition, carbohydrates lead to an increased level in the anabolic hormone insulin and thus contribute to muscle protein synthesis and prevent muscle protein breakdown [1]. On the other hand, in athletes fat intake should not be higher than 30%, while the general recommendation for protein intake is 1.4-1.8 g/kg/d [17]. However, regardless of

the recommendations above some researchers have postulated that in certain cases a low carbohydrate diet, with high fat and/or protein contents may also be equally or even more effective than the standard diet recommended for athletes [43]. A diet based on a low carbohydrate intake has generally been distinguished from the ketogenic diet. A low carbohydrate ketogenic diet (LCKD) leads to ketosis, i.e. increased production of ketone bodies, while a low carbohydrate diet (LCD) is any diet that reduces carbohydrate intake (usually below 200 g), but does not necessarily lead to ketosis [1]. Restriction of carbohydrate intake leads to changes in metabolism by shifting it from "glucocentric" to "adipocentric", namely, there is a reduced use of glucose as an energy source and an increased use of free fatty acids (FFA) and ketone bodies (from food fats, protein and the adipose tissue) [1]. The functioning of tissues, for which glucose is necessary (red blood cells, retina, lens, kidney medulla, central nervous system), is maintained through gluconeogenesis and glycogenolysis. Even though no carbohydrates are ingested, the body will make 200 g of glucose a day only through the liver and kidneys, mostly from amino acids (alanine and glutamine) and less from glycerol, pyruvate and lactate [1, 48]. However, when gluconeogenesis fails to keep pace with bodily needs for glucose, the production of ketone bodies from FFA in the liver increases. After fatty acids are released from the adipose tissue, they are transported to the liver, where they are transformed by the process of β-oxidation into acetyl-CoA, which can be oxidized in the citric acid cycle or used to obtain ketone bodies. By a further process and action of certain enzymes, three ketone bodies are formed, with acetic acid (AcAc) being formed first, followed by beta-hydroxybutyric acid (BHB) and acetone [1]. When BHB values exceed 0.5 mmol/L, nutritional ketosis is observed. Then ketone bodies replace glucose and become the primary source of energy for the brain and nerve cells. Also, BHB and AcAc are optimal substrates for muscle tissue and are rapidly oxidized [1]. Therefore the energy sources during LCKD are fatty acids (70% of caloric requirements from dietary fat and lipolysis of adipose tissue pools), KBs (20% of caloric requirements from lipolysis and ketogenesis adipose stores), and glucose (10% of calorie requirements from gluconeogenesis) [1]. Besides, studies have shown that LCKD results in the reduction of total carbohydrate oxidation and a 2-3-fold increase in whole-body fat oxidation and lipolysis during steady-state aerobic exercise [5, 43, 47]. This proportion of energy contribution and changes in substrate oxidation has led to assumptions that LCKD may be beneficial for prolonged endurance activities, since they rely predominantly on fat [43]. On the other hand, a decrease in the oxidation and availability of glucose may negatively affect high-intensity activities (>70/80% VO_2max), where carbohydrates are the primary source of energy [11, 50]. Also, the effect of LCKD on lean body mass (LBM) is highly questionable because ketosis is a physiological process that acts catabolically, which is manifested by increased levels of adrenaline, cortisol [19] and glucagon at a decrease in insulin levels [42, 48, 52]. In addition, LCKD inhibits the mTOR signaling pathway, which is responsible for protein synthesis and muscle mass increase [1, 28]. On the other hand, it has been shown that BHB has a proteolytic effect [40] and that LCKD leads to an increase of the levels of some anabolic hormones such as testosterone, growth hormone [25, 42] and IGF-1 [13], potentially having a positive effect on LBM. Maintaining optimal LBM values is extremely important for athletes, as its reduction could negatively affect sports performance. In view of the above, this narrative review aims to discuss the physiological basis for low carb ketogenic diets and their both positive and negative effect on body composition, power, strength, aerobic capacity and anaerobic performance in athletes and physically active subjects.

Adaptation of athletes to LCKD

When switching to LCKD, athletes should adapt to an increased use of fatty acids and ketone bodies at a reduced use of carbohydrates as a previously dominant energy source. The rate of fatty acid oxidation increases significantly during the first week and in the following weeks it reaches maximum values of up to 1.5 g/min [4]. At the same time, it usually takes 2 to 3 weeks to reach the optimal value of ketone bodies. When the organism adapts to an increased level of ketone bodies it starts utilizing them as a source of energy. In athletes constant values of beta-hydroxybutyrate were reported to exceed 0.5 mmol/L of blood, which was sufficient to achieve nutritional ketosis [34, 44], although some other authors found higher values (from 1 to 3 mmol/L) [1]. During this transition, which is extremely difficult and can last from 7 to 20 days, athletes experience a decrease in physical abilities and an increase in the subjective feeling of fatigue. Additionally, nerve cells work less efficiently, which makes it difficult to concentrate and efficiently perform high-intensity technical tasks [4, 52]. However, when athletes adapt to this diet they can function effectively without their physical abilities being impaired [4, 43].

The effect of LCKD on body composition in athletes

In order to show maximum performance and achieve a competitive result it is very important to have an optimal body composition, which in athletes in almost all sports should contain a low percentage of fat mass and high LBM. Low-fat mass is associated with higher aerobic potential [9] and high LBM with maximum strength [23], power, explosiveness, speed and other abilities [6]. Additionally, achieving an optimal body composition is crucial for participation in aesthetic sports, such as fitness and bodybuilding, but also in sports categorized according to body weight, such as combat sports [28]. Especially in these sports athletes try to reach an ideal weight within a very short time, so they often rely on some extreme methods (use of salted baths, saunas, diuretics, laxatives, etc.) and/or extremely energy-restrictive diets, mainly requiring reduced carbohydrate intake [28]. Reduction in carbohydrate intake or application of LCKD due to significant metabolic effects, manifested in increased lipolysis of adipose tissue and increased water excretion, results in a very rapid reduction of body mass and fat mass, which can lead to optimal body composition. However, in the long term when caloric intake was uniform LCKD compared to the standard diet did not show better effects on weight and fat reduction in either the general population or athletes [1]. Also, rapid changes in body composition can lead to some side effects, such as dehydration, electrolyte imbalance, cardiac arrhythmias, fatigue and psychological difficulties, etc. [1, 28]. Such effects combined with intensely strenuous training and competitions, which also have a stressful and catabolic effect on the athlete's body, can lead to a drop in LBM and physical abilities [22]. It is very difficult to maintain and even more difficult to increase muscle mass on a ketogenic diet. The state of ketosis is very similar to the state of starvation when the activity of metabolic mechanisms stimulating autophagy and catabolic processes occurs [1, 28]. Under such conditions there is an increased activity of the enzyme 5 adenosine monophosphate-activated protein kinase – AMPK. This enzyme has a catabolic effect, it inhibits the activity of the mammalian target of the rapamycin signaling pathway – mTOR, and thus prevents protein synthesis and increases muscle mass [26, 28]. Oxaloacetic acid is another limiting factor for adding muscle mass, but also for complete oxidation of fat on a ketogenic diet. In the absence of glucose there is not enough oxaloacetic acid, which is necessary to start the cascade of the Krebs cycle in interaction with acetyl CoA. To maintain the function of the citric acid cycle, oxaloacetate must be provided by deamination of glucogenic amino acids such as aspartate and asparagine, which consequently leads to protein resynthesis and reduction of muscle mass [1, 28]. Most studies, which examined the influence of LCKD on the body composition of athletes, in addition to the decrease in body mass and fat mass also recorded a decrease in muscle mass, i.e. LBM. In a study on taekwondo athletes the use of LCKD, in which a caloric deficit of 25% was observed, in addition to a decrease in body mass and fat mass led to a decrease in LBM (from 54.7 ± 3.9 to 52.5 ± 4.7 kg; $p < 0.05$) [32]. Taekwondo practitioners trained as much as 5 hours per day, of which 2 hours were strength development exercises, 1 hour of low-intensity morning exercises and 2 hours of specific exercises for taekwondo athletes. Similarly, another study on crossfitters, which showed a decrease in energy intake (~500 kcal), after 12 weeks resulted in a 1.4% decrease in LBM of leg muscles and an 8% decrease in the thickness of m. vastus lateralis [16]. The results of these studies can be attributed to the combined catabolic effects of intense and extensive exercise, ketosis and energy deficit. However, even when energy intake was uniform in highly trained powerlifters and Olympic weightlifters (9 male, 5 female), administration of LCKD for 3 months resulted in a significant decrease in body mass (–1.7 kg) and LBM (–1.7 kg), while the standard diet phase increased body mass (+1.6, difference 3.3 kg) and LBM (+0.6 kg, difference 2.3 kg) [12]. In this study the differences in LBM can be explained by a significantly lower carbohydrate intake, which consequently led to increased catabolic effects, but also to redistribution and excretion of water due to diuresis, which is increased on LCKD due to glycogen loss and ketonuria. Approximately 100 g of glycogen is stored in the liver, while 400 g is stored in the muscles, with each stored gram associated with 2 g of water [38]. Additionally, the decrease in LBM was also connected by a slightly lower protein intake, which in the above-mentioned studies ranged from 1 to 1.5 g/kg. According to Kerksick et al. (2018), in order to maintain or add muscle mass, protein intake in athletes must be 1.4-2.0 g/kg [17]. However, contrary to previous results preservation or even a slight increase in muscle mass after the application of LCKD was reported in several studies. In a study of top gymnasts regularly performing strength exercises after a month of LCKD there was a significant reduction in body weight and fat mass, but not LBM (–1.1 kg; $p > 0.05$; although the % LBM increased by 2.6%). On the other hand, in the group administered a standard diet

LBM increased by 300 g. However, it is important to keep in mind that that study was conducted on only 9 male gymnasts [29]. In a study of Volek et al. (2002), after the application of LCKD for 6 weeks a decrease in fat mass (–3.4 kg) and an increase in LBM (+1.1 kg) were reported in physically active subjects [46]. In another study cited by Volek et al., administration of LCKD in combination with resistance training for 12 weeks decreased fat mass by 7.7 kg, and increased LBM by 1 kg, while the group on the standard diet increased LBM by 1.8 kg [45]. What is common to these studies, which can explain the differences from previous results, is the application of resistance training, optimal energy intake and adequate protein intake, which was generally about 2 g/kg. The control groups from these studies had the same energy intake and an adequate protein intake, but significantly higher carbohydrate intake, which is most likely the reason for the significantly higher growth of LBM compared to subjects on LCKD. Phinney (2004) found that combined carbohydrate and protein intake preserved LBM (which was assessed by nitrogen balance), while those who consume protein alone experienced progressive loss of body nitrogen [30]. Intake of glucose and amino acids in combination (especially leucine) activates the mTOR signaling pathway and has an anabolic effect on muscle mass [28]. Therefore, athletes are generally advised to consume a meal rich in carbohydrates and proteins after training. Considering that in all the above-mentioned studies the subjects applied resistance training or strength exercises in combination with other types of physical activity, we can say that such activities themselves act catabolically and lead to degradation of muscle tissue. Whether anabolic effects on muscle mass growth are achieved during the recovery period depends on the amount of energy intake and a combination of nutrients after training. However, most studies show that despite both the application of resistance training and the fact that the respondents are athletes who compete in strength sports, they still lose a significant amount of body weight from LBM (on average 34%) [1]. In turn, the increase in LBM was observed in only two studies, which could be further explained by certain anabolic effects that LCKD promotes [45, 46]. Low glucose and insulin levels are associated with an increase in growth hormone [15, 25], while high protein intake is related to an increase in IGF 1 [13]. In terms of testosterone levels, the results of these studies are inconsistent. Although high fat intake and increased cholesterol in the LCKD were associated with an increase in testosterone levels [25, 42], Durkalec-Michalski et al. (2021) found no effects of LCKD on certain hormones [7]. Evidence is also available suggesting that beta-hydroxybutyrate activates the mTOR signaling pathway and prevents proteolysis [40]. These are some of the factors that, together with adequate nutrient and energy intake, may explain the maintenance or increase in LBM on LCKD. The LCKD diet can improve the body composition of athletes by reducing body weight and fat mass, but compared to the standard diet in an isocaloric situation do not show better results. Also, compared to a diet with high carbohydrate content, it is challenging to increase LBM and resistance training with adequate protein intake and minimal carbohydrate intake are necessary for muscle mass preservation or slightly increment.

Effects of LCKD on strength and power sports

Strength and power are physical abilities that primarily depend on the phosphagen energy system, so it is unlikely that changes in glucose and fat oxidation will have an impact on them. However, the LCKD could have an indirect effect here because it significantly affects body composition and LBM. However, although studies on this topic are few, they generally show that despite the reduction of LBM during LCKD, there is no decline in the strength and power of athletes. In crossfitters (5 males and 2 females) [16], powerlifters, and Olympic weightlifters (9 males and 5 females) [12], who applied LCKD for 3 months, despite a significant drop in LBM, no effect was observed on a vast majority of all measured parameters that assessed strength and power (the power to weight ratio, 1RM – one repetition maximum of squat, clean and jerk, snatch, push-up, chest push-up, deadlift), except for the maximum number of push-ups, where progress was recorded ($p < 0.05$). It is possible that no drop in performance may have been caused by the fact that LBM did not decrease due to degradation of muscle tissue, but due to redistribution of body fluids, as those authors reported. Also, the production of maximum force largely depends on the amount of ATP in the muscle and its resynthesis via the phosphagen rather than the glycolytic system [10]. 1RM is performed after a complete repair, which allows enough time to compensate for the oxygen consumed and resynthesize adenosine triphosphate (ATP) and creatine phosphate (CP). Furthermore, protein intake in those studies ranged from 1.1 to 1.5 g/kg, which was not in line with the general recommendations for strength athletes amounting to 1.7 g/kg [39]. On the contrary, high protein intake (2.8 g/kg) with electrolyte

supplementation maintained nitrogen balance and preserved LBM in top male gymnasts, which indirectly affected the preservation of performance that is important for gymnastics (squat jumps, countermovement jumps, reverse grip chin-ups, push-ups, legs closed barrier test, parallel bar dips) [29]. Although a majority of respondents in those studies were men, studies in women show similar results. In women (n = 21) who regularly performed strength exercises and whose protein intake was 1.9 g/kg, maintenance of LBM (–0.7 kg +/– 1.7; p = 0.202) and improvement in bench-press and squat strength were observed compared with the baseline. However, LBM (+0.7 +/– 1.1; p = 0.074) and strength gains were greater in the control group compared with the LCKD group [41]. Additionally, in a study of 11 women and 11 men, whose protein intake was 1.7 g, cross-fit-specific performance was maintained in both genders [7]. Considering that almost all the above-mentioned studies refer to athletes classified according to body weight, reduction in total mass and fat mass and preservation of performance despite the fall of LBM could help them to classify for a competition, but may hinder achieving a better result.

Effects of LCKD on performance in endurance sports

One of the reasons why LCKD advocates recommend its use in endurance sports is limited carbohydrate stores (~2200 kcal) compared to fat stores (~30,000 kcal in a person with 7-14% of fat mass) [44]. Adaptation to LCKD leads to a decrease in glucose oxidation, while at the same time leading to a 2- to 3-fold increase in FFA oxidation [5, 34, 43, 47]. Besides, compared to a high carbohydrate diet (HCD), LCKD achieves the maximum rate of fat oxidation at a relatively higher work intensity (1.5 g/min at about ~70% of maximum oxygen uptake – VO_2max vs ~0.7 g/min at 55% VO_2max [43], while resynthesis and glycogen levels after exercise are preserved and do not differ significantly from HCD [31, 43]. The higher fat intake through diet and adaptation to its increased use will allow the body to use fat more effectively and at a higher % of VO_2max, i.e. preserved carbohydrate reserves will later be included as a dominant energy source in the event of an increased work intensity. Despite such pronounced metabolic effects, most studies have shown that LCKD has no significant effect on endurance performance [11, 16, 24, 34], while even negative effects in certain studies have been reported [5, 7, 35, 51]. Interestingly, the decline in aerobic capacity is more common in females [7, 35, 51]. In a study of Durkalec-Michalski et al. (2021), consuming a LCKD led to an 10.4% decrease in peak oxygen uptake during ICT (p = 0.027) in females, while in males there was no significant effect. One possible explanation for VO_2 peak drop is that this study resulted in certain alterations in haematological parameters (haemoglobin – HGB, mean corpuscular HGB, and mean corpuscular HGB concentration) in females. These results may indicate a lower status and transport of iron. In females the daily iron intake was lower than the dietary recommendations for athletes [7]. Also, due to increased levels of interleukin 6 and hepcidin during LCKD iron transport may be impaired [27]. Iron is a key functional constituent of hemoglobin and myoglobin and it is required for oxygen uptake, transport and energy production [2]. A poor iron status reduces oxygen-carrying capacity of RBCs, leading to a decline in physical performance [27]. As women are biologically susceptible to a lower iron status, the reduced iron intake with a LCKD may impact women more than men [27]. Also women may possibly achieve lower VO_2 peak values after consuming LCKD due to the difference in fat oxidation between the genders. Namely, in a previous study by Durkalec-Michalski et al. (2019) it was shown that men have a higher rate of fat oxidation than women up to 80% VO_2 max and that they are more prone to shifts in macronutrient utilization (in favor of fat utilization) during submaximal intensity exercise [8]. On the other hand, in some studies positive results for the endurance of athletes have been recorded. In elite race walkers [5] and off road cyclist [50] an improvement in aerobic capacity was reported. However, in off road cyclists the increase in VO_2max in relative values (ml/kg/min) was due to body mass and fat mass reduction, while in elite race walkers it was not accompanied by an improvement in endurance performance. In another study on highly trained endurance athletes the application of LCKD for 12 weeks resulted in an improvement in the time achieved in a 100 km race by 4.1 min, while HCD improved the time by 1.1 min. This improvement was not statistically significant either relative to baseline time or between groups; moreover, the VO_2max, at which this time was achieved was not specified [24]. At an exercise intensity of less than 60-65% VO_2max metabolic efficiency is preserved, i.e. oxygen uptake is followed by an equal oxygen consumption, which along with the reduction in body weight observed in some studies may explain the preservation of performance at this intensity [24, 34]. However, in endurance sports it is often necessary to increase the intensity above 70% VO_2max [4]. Studies

show that with increasing intensity LCKD impairs performance by reducing work economy. Highly trained runners [34], fast walkers [5], cyclists [50] and recreationally active men [11] showed increased oxygen consumption, increased energy expenditure and reduced lactate production. Consequently, a reduction in time achieved and reduced power output throughout the race and at the end of the race were recorded at an exercise intensity greater than 70-80% VO_2max. At an exercise intensity exceeding 70% VO_2max the carbohydrate requirement is far greater; however, due to adaptation to ketosis glycolytic enzymes are inhibited, which prevents oxidation of glycogen and glucose. Thus, for the most part the body is forced to continue to rely on fat [4]. Compared to glucose oxidation, fat oxidation requires significantly more oxygen (8%) to obtain energy [4]. It was found that even after 8 months on LCKD there is no increased activity of gluconeogenesis compared to HCD at an intensity of 72% VO_2max [47]. All this together indicates that LCKD limits the availability of glucose and increases the oxygen cost, which consequently reduces the economy of work and endurance of athletes. Thus LCKD can maintain performance at exercise intensity below 70% VO_2max, but with an increase in intensity above 70-80% VO_2max a decrease is observed in metabolic efficiency and endurance of athletes.

Effects of LCKD on anaerobic performance

In sports activities where anaerobic metabolism is dominant, energy from the phosphagen energy system and glycolysis is mainly used, so it is unlikely that a reduced rate of glucose oxidation and an increased rate of fat oxidation after LCKD adaptation will have a favorable effect on performance. In a study on physically active subjects, HCD and standard diet compared to LCD achieved greater time to failure during supramaximal work intensities (HCD – 4.4 ± 0.3 min; Control – 3.7 ± 0.3 min; LCD – 3.0 ± 0.2 min) [21]. Also Langfort et al. (1997) and Wroble et al. (2019) showed that the application of LCD for 3 and 4 days in subjects regularly training at high intensity resulted in a decreased peak power and mean power during 30 s (measured by the Wingate test) [20, 49]. Although the results of these studies can be explained by the short duration and lack of adaptation, longer studies show similar results. Thus, in highly trained taekwondo athletes after 3 and active recreationists after 6 weeks a decrease was recorded in peak power and mean power during the Wingate test of 30 s [11, 32]. The above-mentioned studies applied the Wingate test, during which the largest part of glycolytic energy is used (56%) at a smaller part of phosphocreatine (28%) and aerobic energy (16%), so the results of these studies are explained by reduced availability of glycogen and glucose [36]. On the other hand, the performance of crossfitters did not decrease in the 400 m race, although during this distance most of the energy is provided from the glycolytic system. However, subjects lost a significant amount of body mass (–3.0 kg), which increased running economy (increased power to weight ratio) and thus it masked metabolic inefficiency, which occurs at an intensity above 70% VO_2max [16]. A slightly longer application of LCKD for 12 weeks increased peak power during the 6-second sprint (Wingate test) and peak power during the test, where pedaling was performed at maximum power and speed for 3 min, in subjects on LCKD compared with those on HCD [24]. Unlike previous studies, this study used HIIT (high-intensity interval training), strength training and a sufficient protein intake (1.9 g/kg/LBM) and despite the reduction in body weight LBM was preserved, which altogether had a positive effect on anaerobic performance. Additionally, high-intensity short-duration activities require total energy from the phosphagen system, which can further explain the improvement of the 6-s sprint in runners, as well as maintaining performance in the 100-m sprint in taekwondo athletes [32]. LCKD does not appear to have negative effects on anaerobic performance, which relies predominantly on the phosphagen energy system, whereas due to reduced glucose availability there may be a decline in those performances that rely heavily on the glycolytic system. Body mass and LBM play an important role in these performances because their optimal ratio can affect the economy of movement and the manifestation of power and speed. Likewise, the application of HIIT training during LCKD appears to play a significant role in anaerobic performance, which could be a topic of future research.

Cyclic ketogenic diet and carbohydrate supercompensation

The cyclic ketogenic diet is realized through a weekly cycle, during which an athlete adheres to a diet low in carbohydrates (10-20% of daily energy intake) for 4 to 5 days and then during the remaining 2-3 days of the cycle increases daily carbohydrate intake to 60-70% of total energy intake. During the LCKD phase the athlete can improve the body composition and maximize FFA oxidation, whereas during the carbohydrate loading phase the athlete should provide supercompensation of

glycogen, which will allow high-intensity work [52]. In his review Burke (2015) found that the application of LCKD (5-10 days) followed by the procedure of CHO loading (6.8-11 g/kg carbohydrates for 1-3 days) has no negative effects on endurance, but that is not superior to the standard CHO loading procedure [3]. On the other hand, in a study of Lambert et al. (2001) the group that first applied LCKD completed the 20-km race in less time compared to the group that applied the standard procedure (29.5 ± 2.9 min vs 30.9 ± 3.4, $p < 0.05$) [18]. However, the results of this study are explained by the reduced utilization of glycogen and greater reliance on FFA. Therefore, it seems that the race intensity was not high enough, so the importance of glycogen supercompensation in that study has been questioned. In turn, Michalczyk et al. (2019) reported a decrease in anaerobic performance [25], which was restored, but not improved after the CHO loading procedure. As the intensity of exercise increases, the importance of glycogen utilization and glucose oxidation increases markedly. In a study of Havemann et al. (2006) in contrast to the standard procedure the procedure of CHO loading after LCKD resulted in a decrease in anaerobic performance [14]. It has been noted that oxidation of carbohydrates is higher with the standard CHO loading procedure [3] allowing higher work intensity, while with LCKD there is a decrease in glycogenolysis and a decrease in the active form of pyruvate dehydrogenase both during rest and submaximal and maximum intensity [37]. Therefore, in comparison to the standard CHO loading procedure the combination of LCKD with the CHO loading procedure does not provide greater effects on either aerobic or anaerobic performance.

LCKD and oxidative stress

In the case of top athletes training and competition are very intensive and lead to an increase in oxidative stress and inflammation. Based on our best knowledge, only two studies by the same authors examined the antioxidant potential of the ketogenic diet in athletes. In their work, after 3 weeks of LCKD application in taekwondo athletes a significant decrease was recorded in the oxidative marker malondialdehyde. A decrease in LDL and an increase in HDL noted in that study also indicate an increase in the antioxidant capacity of the blood. Exercise-induced oxidative stress can lead to skeletal muscle cell damage, which in turn leads to an increase in LDL, while HDL is a potent antioxidant suppressing the accumulation of oxidized lipids [33]. In another study, also conducted on taekwondo athletes, the keto diet resulted in a decrease in the cytokine called tumor necrosis factor-alpha [32]. Those authors concluded that a ketogenic diet leads to reduced oxidative stress and inflammatory response, although in those studies there was no improvement in other analyzed cytokines and oxidative markers (interleukin-6, interferon-gamma, superoxide dismutase, ROS) [32, 33]. Given that there are not enough studies on this topic, additional research is needed to examine the effects of a ketogenic diet on inflammatory response and oxidative stress after exercise in highly trained athletes.

Conclusions

Despite significant metabolic changes and increased fat oxidation the administration of a low carbohydrate ketogenic diet failed to show clear and convincing effects on endurance ability. LCKD can preserve endurance performance in sports where intensity does not exceed 65-70% VO_2max, but it is not superior to a diet high in carbohydrates. Also, negative effects on aerobic capacity can be manifested especially in women, which may be related to a lower status and transport of iron and due to the difference in fat oxidation between genders. In sports activities where the intensity exceeds 70-80% VO_2max, LCKD can have negative effects on performance, because in these sports activities the requirement for carbohydrate intake is remarkably greater. On the other hand, it seems that LCKD has no particular effects on maximum strength, power and anaerobic lactate abilities such as jumps and short sprints, because they depend on the phosphagen energy system. Further, LCKD enables the reduction of body mass and fat mass and maintenance of lean body mass, which can be useful in aesthetic sports, sports classified by body weight, as well as all other situations where the goal is to establish optimal body composition. However, compared to a diet with a high carbohydrate content, it is challenging to increase LBM and resistance training with an adequate protein intake and the minimal carbohydrate intake necessary for muscle mass preservation or one slightly exceeding that minimum. Additional research is needed to examine the impact of LCKD on inflammatory response and oxidative stress after exercise in highly trained athletes.

Conflict of interests

The author declares there was no conflict of interest.

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