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Review article

Carbohydrate restriction: Friend or foe of resistance-based exercise performance?



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ABSTRACT

It is commonly accepted that adequate carbohydrate availability is necessary for optimal endurance performance. However, for strength- and physique-based athletes, sports nutrition research and recommendations have focused on protein ingestion, with far less attention given to carbohydrates. Varying resistance exercise protocols, such as differences in intensity, volume, and intraset rest prescriptions between strength-training and physique-training goals elicit different metabolic responses, which may necessitate different carbohydrate needs. The results of several acute and chronic training studies suggest that although severe carbohydrate restriction may not impair strength adaptations during a resistance training program, consuming an adequate amount of carbohydrate in the days leading up to testing may enhance maximal strength and strength–endurance performance. Although several molecular studies demonstrate no additive increases in postexercise mammalian target of rapamycin 1 phosphorylation with carbohydrate and protein compared with protein ingestion alone, the effects of chronic resistance training with carbohydrate restriction on muscle hypertrophy are conflicting and require further research to determine a minimal carbohydrate threshold necessary to optimize muscle hypertrophy. This review summarizes the current knowledge regarding carbohydrate availability and resistance training outcomes and poses new research questions that will better help guide carbohydrate recommendations for strength and physique athletes. In addition, given that success in physique sports is based on subjective appearance, and not objective physical performance, we also review the effects of subchronic carbohydrate ingestion during contest preparation on aesthetic appearance.

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Introduction

Glucose availability is important for muscular performance lasting >30s, and dietary carbohydrates are now considered the most important of the three macronutrients for fueling endurance sports [1]. However, before the late 19th century, athletes, coaches, and even some scientists believed protein was the major source of energy for muscular activity [2]. From a historical perspective, Zuntz [3,4], Frenzel and Reach [5], and Krogh and Lindhard [6] demonstrated through a series of landmark experiments that carbohydrates and lipids fuel exercise, with the varying proportions of the two macronutrients in the diet and the intensity of work determining the proportions in which they were oxidized. In 1924,

researchers from Harvard Medical School conducted experiments with runners from the Boston Marathon to investigate the role of carbohydrates on exercise performance [7]. It was observed that blood glucose levels were reduced in several Boston Marathon runners who crossed the finish line, thus establishing a relationship between blood glucose, carbohydrate consumption, and performance [7]. In the following year, many of these same athletes were supplemented with a large amount of carbohydrates the day before the race, and they were allowed to eat sugary candy before and during the event [8]. Blood glucose concentrations after completion of the marathon were sampled by researchers and they found that by normalizing blood glucose concentrations (before and during running), symptoms of fatigue and stupor were prevented and mental focus was increased [8]. This was one of the first studies to establish a causal link between carbohydrate consumption and sport performance. In the 1960s, it was demonstrated that high-carbohydrate diets improved endurance performance and carbohydrate feedings during exercise delayed fatigue [9–11]. Finally, in

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the 1970s, research revealed that manipulation of dietary carbohydrate levels from a carbohydrate-free diet, mixed diet, and high-carbohydrate diet increased muscle glycogen content and consequentially increased the time of moderate exercise to exhaustion [12]. The supra-cited effects of manipulation of carbohydrate ingestion leading to increased muscle glycogen content are currently known as *carbohydrate loading* [13,14]. Those studies were essential to paving the road for the carbohydrate saga in sports nutrition.

It is now fully appreciated that adequate carbohydrate ingestion is necessary for optimal endurance performance, with recommendations well established in sports nutrition societies [15]. However, for strength- and physique-based athletes, sports nutrition recommendations have focused more on amino acid and protein ingestion [16], with much less attention given to carbohydrate requirements. In fact, the role of dietary carbohydrates in strength training performance and adaptation are currently under debate, with some researchers questioning whether dietary carbohydrates impart any ergogenic or adaptive benefit to strength training at all [17]. For strength- and physique-based athletes, carbohydrates are consumed for the following four main purposes:

1. To maintain high muscle glycogen levels to sustain muscle contractions during high-intensity strength training sessions.
2. To enhance muscle recovery and adaptation (via increasing protein synthesis and suppressing protein breakdown) between exercise sessions [18].
3. To enhance aesthetics acutely, especially for physique competitions, whereby carbohydrate loading increases muscle glycogen and intermuscular water content leading to a more “muscular” appearance [19].
4. To improve body composition via reductions in fat mass; however, in this case, most individuals reduce carbohydrate consumption in an attempt to mobilize more fatty acids from the adipose tissue [20].

In all four situations, carbohydrates are important (either in high/adequate amounts or low amounts) for strength training and the physique athlete, and are discussed in detail after a brief explanation of carbohydrate metabolism.

Carbohydrate metabolism

Ingested carbohydrates are degraded by extensive enzymatic systems in the body, initially found in saliva (salivary amylase), then in the pancreatic juice (pancreatic amylase), and finally by intestinal microvilli enzymes (maltase, lactase and sucrase). After being digested, they are absorbed as glucose or fructose in the intestine, via glucose/fructose transporters [21]. In sports nutrition, the choice of a given source of carbohydrates is extremely important for several reasons, from palatability to digestion and absorption [15]. Because carbohydrate consumption directly effects the postprandial glycemic response, and because glucose is the major source of fuel for the central nervous system and erythrocytes (in addition to being important for high-intensity muscle contractions), maintaining blood glucose levels is fundamental for both exercise performance and survival [22].

In mammals, hepatocytes are fundamental for blood glucose regulation, and it has been hypothesized that hepatocytes have evolved to guarantee extra glucose supply for the brain [22]. In the postabsorptive period, for example, when food is no longer available and blood glucose levels begin to decrease, glucose is released by the liver to counter balance glucose utilization by the peripheral tissues [23]. This process of glucose release from the liver to the

circulatory system and peripheral tissues depends on several counterregulatory hormones such as glucagon, cortisol, epinephrine, and several others secreted by different organs [24,25]. Because hepatocytes contain the enzyme glucose-6-phosphatase, which splits off the phosphate molecule from the glucose carbon, glycogen from the liver can be released back into circulation to supply the peripheral tissues [26]. Contrarily, after a meal, glucose is stored in the liver and in peripheral tissues, in a process highly regulated by insulin [25,24]. It is interesting to note that the human body only stores ~100 g of glycogen in the liver and ~400 g or 1.5 g/100 g of glycogen in skeletal muscle (or more, depending on the amount of lean mass and training status of an individual), but humans easily store 20 kg of triacylglycerol in adipose tissue [22]. Under this perspective, glycogen is, if compared with fats, scarce in the body, and may be reduced as a result of fasting, low dietary carbohydrate intake, exercise, or a combination of all three factors. The availability of muscle glycogen has been postulated to be essential in the evolutionary process [22]. For example, performance of high-intensity muscle contractions may have been essential for survival of our ancestors to evade predators. Because muscle glycogen is an important source for high-energy anaerobic generation [23], sparing muscle glycogen would have been of ultimate relevance for survival [22]. Replenishment of muscle glycogen, not only after exercise but also after feast and famine periods would have been another important issue for survival [22,23]. In a modern view, sports nutrition for strength-training athletes would be a sum of the following factors:

- Perform high-intensity muscle contractions;
- Utilize muscle glycogen;
- Replenish muscle glycogen;
- Perform better (survive and adapt), in athletic settings.

Muscle contractions and glucose metabolism

Muscle contraction is a process dependent on intramuscular adenosine triphosphate (ATP) being replenished by bioenergetic systems [23]. During efforts lasting 30 to 180s, the ATP required for muscle contractions is mainly powered through glycolysis [27]. Glycolysis is a set of 10 step reactions generating two or three ATPs, depending on whether glucose or glycogen is the initial substrate, respectively [27]. During anaerobic exercises, when available, glycogen is the preferred source of energy [28]. Blood glucose also contributes to muscle glycolysis, increasing its contribution as muscle glycogen is used [29]. However, muscle performance decreases as muscle glycogen is depleted [12]. During anaerobic glycolysis, by-products of metabolism such as hydrogen ions and lactate are produced [30]. Hydrogen ions has been considered inhibitors of muscle contraction either by inhibiting key enzymes of glycolysis (such as phosphofructokinase) [30] or by competing with calcium for the troponin-binding site [31]. Accumulation of hydrogen ions from glycolysis also has been postulated to be partially responsible for muscle fatigue, which at first glance, would be negative for muscle performance. However, it should be considered that fatigue is an important determinant for a greater muscle fiber recruitment [32–34], which seems to be important for muscle growth [35].

Skeletal muscle tissue accounts for ~40% of total body weight, contains 50% to 75% of all body proteins, and accounts for 30% to 50% of whole-body protein turnover in humans. The composition of skeletal muscle is mainly water (75%); protein content (20%); and other products such as minerals, lipids, and carbohydrates (5%) [36]. In addition, in respect to glucose uptake and glycogen storage, skeletal muscle tissue is the main reservoir and tissue of

deposition. It has been observed in the postprandial state in humans and under euglycemic-hyperinsulinemic conditions, 80% of glucose uptake occurs in skeletal muscle. Studies using the euglycemic-hyperinsulinemic clamp and femoral artery/vein catheterization to quantify glucose uptake have shown that adipose tissue uses ~5% of an infused glucose load and bone is metabolically inert. This suggests that the majority of glucose is deposited into skeletal muscle [37,38].

In regard to the glucose transport, it has been thought that several mechanisms act collectively to maintain a constant glucose flux to the contracting muscles. For example, muscle contractions increase cytoplasmic calcium and modify the energy status of muscle cells (increasing the adenosine monophosphate-to-ATP ratio) and both are considered important triggers for an increased muscle glucose uptake response [39,40]. Reactive oxygen species and hypoxia also may be additional triggers in the muscle contraction process mediating increased glucose uptake [39,41]. The increased transport of glucose mediated by muscle contractions uses specific carrier proteins called glucose transporters. In muscle cells, the glucose transporter number 4 (GLUT-4) is the major isoform expressed [42]. The movement of this transporter from the sarcoplasm to the sarcolemma occurs by exocytosis, trafficking, docking, and fusion of GLUT4-containing storage compartment or “vesicles” into the cell-surface membranes [23]. After exercise, glycogen synthase is activated, and muscle glycogen concentrations are allowed to increase, especially in the presence of proper dietary carbohydrate consumption [11,14]. In addition, although glycogen depletion seems to be an important factor in the rate of restoration of muscle glycogen levels after exercise [43], this phenomenon has yet to be explored with the use of resistance exercise.

Metabolic demands of strength/power athlete training and physique athlete training

Resistance-training variable prescriptions (volume, intensity, rest between sets, repetition tempo, etc.) vary widely based on the desired outcome. Consequentially, metabolic demand and substrate utilization also will differ between resistance-training prescriptions. During the yearly training plan, strength and power athletes often divide their training into periods of general preparation, defined in part by performing higher volumes and lower intensities to optimize work capacity and hypertrophy, and specific preparation, defined by lower volumes and higher intensities to optimize force and power output [44]. Although debate exists over the optimal intensity range to maximize hypertrophy [45], physique athletes generally perform high volumes (15–20 sets per muscle group) with moderate to higher repetitions (8–16 repetitions), and incomplete recovery periods (~1 min) [46]. Although there is a wealth of research examining differences in metabolic demand and substrate use between various aerobic exercise protocols, research examining the difference in metabolic responses between resistance exercise protocols is lacking. For this reason, we split the following discussion into the following two outcomes: 1) training for strength outcomes, characterized by low repetitions (<6), higher intensities (>85% 1 repetition maximum [RM]), and longer rest periods (>3 min); and (2) hypertrophy outcomes, characterized by higher repetitions (>8), moderate intensities (60–80% 1 RM), and incomplete rest periods (<2 min).

Several studies have investigated the energy cost of resistance training. The results of these studies suggest that the more musculature used, especially the larger muscles of the lower body, the greater the energy expenditure. For example, Ratamess et al. [47] reported mean maximum rate of oxygen consumption (VO_2) of 19.6 and 12.5 mL·kg⁻¹·min⁻¹ when performing three sets of squats

or bench press to muscular failure at 75% 1 RM, respectively. Similarly, Reis et al. [48] reported energy costs of 25.7 and 11.41 mL·kg⁻¹·min⁻¹ for one set of leg press and bench press to muscular failure with 80% 1 RM, respectively. Although not surprising, performing multiple sets per exercise results in greater energy expenditures than performing a single set per exercise [49]. In addition, it appears that higher repetitions (10 RM) and shorter rest intervals (30–60s) results in greater energy expenditures than lower repetitions (5 RM) and longer rest periods (3–5 min) [50]. These differences in energy expenditure are both positively related to the total amount of work performed during the set [51] and the total time under tension [52]; however, energy expenditure during the recovery period may not differ between higher- and lower-repetition training and shows little to no correlation with the amount of work performed [51].

Only a few studies have investigated glycogen utilization during resistance exercise. Roy and Tarnopolsky [53] reported a 36% reduction in vastus lateralis muscle glycogen after 3 rounds of a full-body circuit that contained 3 sets of leg press and 6 sets of leg extension in addition to 6 upper body exercises each with 10 repetitions at 80% 1 RM. Pascoe et al. [54] reported a 29% reduction in glycogen after 6 sets of 6 repetitions of leg extension with 70% 1 RM. Tesch et al. [55], using a volume prescription that more closely resembles what might be performed by athletes engaged in a hypertrophy phase of resistance training, had subjects perform front squats, back squats, leg presses, and knee extensions for 5 sets of 6 to 12 repetitions to muscular failure with a work-to-rest ratio of 1:2 (providing ~60–90s rest between sets). Muscle glycogen was reduced ~26% and postexercise muscle lactate averaged 17.3 mmol/kg wet weight. Haff et al. [56] reported a 40% reduction in muscle glycogen after 3 sets of 10 repetitions of isokinetic knee extensions, back squats (65% 1 RM), speed squats (45% 1 RM), and single-leg squats (10% 1 RM). Given that resistance exercise tends to recruit more high-threshold motor units than aerobic exercise, Koopman et al. [57] investigated the differences in glycogen depletion between type I and II fibers after 8 sets of 8 repetitions at 75% 1 RM in the leg press and leg extension exercises. An average 33% reduction in muscle glycogen was found, with significant differences between type II fibers (40–44% reduction) and type I fibers (24% reduction).

Although it is clear that resistance training depends heavily on glycolytic metabolism, less is known about how varying intensities affect glycogen depletion during resistance exercise. To our knowledge, only two studies have directly accessed this question. In the first study, Tesch et al. [58] investigated the difference in glycogen depletion between 5 sets of 10 repetitions of concentric-only leg extension with either 30%, 45%, or 60% of the concentric 1 RM. All sets were separated by 2 min and each intensity protocol was separated by 45 min, such that all exercises were completed on the same day. Mixed muscle glycogen depletion was related to the percentage of type IIAX/X fibers recruited, which was dictated almost exclusively by the load used. In this study, glycogen depletion was greatest in the 60% 1 RM condition, whereas the 30% and 45% 1 RM conditions produced minimal glycogen depletion in type IIAB/B fibers. There are three major limitations to consider when interpreting the results of this study. First, exercise conditions were carried out on the same day, spaced 45 min apart. Therefore, it is possible that preexisting reductions in glycogen or fatigue from the previous sessions may have influenced the subsequent sessions. Second, muscle glycogenesis rates of up to 11 mmol·kg⁻¹·wet-wt·h⁻¹ have been reported after resistance exercise in the absence of any postexercise caloric intake [59], further confounding the differences in glycogen depletion between conditions. Finally, the predetermined number of repetitions prescribed in the lower intensity

(30 and 45% 1 RM) conditions are not representative of the common prescription of “repetitions to failure” in current low-intensity resistance training protocols that have been demonstrated to induce changes in muscle hypertrophy or strength [60]. In a later study, Robergs et al. [59] evaluated differences in glycogen depletion after 6 sets of 6 repetitions of leg extension with 70% 1 RM or 6 sets of 12 repetitions matched for total work with 35% 1 RM. Despite a twofold greater rate of glycogenolysis in the 70% 1 RM condition compared to the 35% 1 RM condition (46 versus 21 mmol·kg wet-wt·sec⁻¹, respectively), no differences in total mixed muscle glycogen depletion between conditions (~30% depletion) were reported. Therefore, it appears that the volume of work performed, when of a higher intensity of effort (approaching muscular failure), in addition to the total duration of the training session, have the most effect on glycogen use during resistance exercise.

In comparison to endurance or high-intensity interval training, studies examining substrate use during resistance exercise are scarce. Keul et al. [61] conducted one of the first studies to examine energy metabolism in relation to resistance training. In this study, competitive weight lifters performed 6 sets of 10, 5, 3, 2, 1, and 1 repetitions with a progressively increasing load and 2 min of rest in the bench press, deadlift, and squat exercise. The authors found increases in blood lactate only after the first set of 10 repetitions and concluded that for typical strength orientated training (low-repetition, high-load) the catabolism of high-energy phosphates is responsible for nearly all ATP regeneration. Using a protocol more similar to hypertrophy-oriented training, Tesch et al. [55] had bodybuilders perform five sets of front squats, back squats, leg press, and leg extension to muscular failure (resulting in ~6–10 repetitions per set) with 80% 1 RM and 60s of rest between sets. Compared with pre-exercise, postexercise phosphocreatine (PCr) and glycogen were reduced by ~49% and 26%, respectively; however, muscle biopsies were obtained 30s after the completion of the final set, and it was likely that PCr levels had increased since exercise cessation. Markers of glycolysis such as intramuscular glucose, glucose-6-phosphate, and alpha-glycero-phosphate were increased by ~1130%, 384%, and 246%, respectively. These results suggest that resistance training programs typically employed during the general preparation phase or by physique athletes have high phosphate and glycolytic requirements. It is interesting to note that resting glycogen levels in this group of bodybuilders were 50% to 100% greater than those reported in non-athletic populations. This was an interesting discovery, as it was previously thought by some coaches at the time that resistance training did not significantly enhance glycogen storage. In a pioneering study, MacDougall et al. [62] had bodybuilders perform either one or three sets of unilateral arm curls to muscular failure with 80% 1 RM and assessed changes in intramuscular ATP, PCr, glycogen, and lactate. Intramuscular PCr was reduced by 64% and 50% after one and three sets, respectively. Glycogen was reduced by 12% and 24%, and intramuscular lactate increased to 91.4 and 118 mmol/kg, after one and three sets, respectively. Based on this data, Lambert and Flynn [46] estimated that for the single set performed to muscular failure, stored ATP provided 1.6%, PCr hydrolysis provided 16.3%, and glycolysis provided 82.1% of ATP demands.

Based on the aforementioned data, we speculated that energy expenditures and rates of glycolysis are likely greater during training sessions focused on developing hypertrophy than in training sessions focused on developing maximal strength or power. In addition, PCr and glycolytic flux rates are greater when sets are taken to muscular failure than when an equal amount of work is performed but sets are ceased well short (three to five repetitions) of muscular failure [63]. Given that training to muscular failure is

more prominent during hypertrophy training, we speculate that physique athletes and strength/power athletes performing a general preparation phase catabolize more carbohydrates than strength/power athletes performing a specific preparation phase. More research, however, is necessary to explore energy expenditure and substrate utilization differences between hypertrophy and strength-oriented resistance training.

Muscle glycogen availability and dietary carbohydrate affect resistance-based muscular performance

Maintaining muscle glycogen via adequate carbohydrate consumption (4–7 g/kg daily) is recommended to optimize both acute strength performance (i.e., a power lifter who must execute three 1 RM attempts for the squat, bench press, and deadlift with 15–30 min of rest between attempts during competition) and supporting high weekly volumes of resistance training [63]. From a mechanistic perspective, it would appear that maintaining glycogen is indeed important for performance in both acute and training scenarios. Glycogen is localized to three distinct compartments within the muscle cell: subscarcolemmal, intermyofibrillar (located between the myofibrils and in close proximity to the mitochondria), and intramyofibrillar (located within the myofibrils, often in the I-band near the sarcoplasmic reticulum and t-tubule triad junction) [64]. Intramyofibrillar glycogen is theorized to provide the fuel for the sarcoplasmic reticular calcium release and calcium-transporting ATPases [65]. Rapid calcium kinetics in type II muscle fibers would likely require greater ATP to sustain contractile force, however, type II muscle fibers store ~40% to 50% less intramyofibrillar glycogen than type I fibers [66], likely because increasing intramyofibrillar concentrations push contractile filaments closer to each other and compromise shortening velocity [64]. To investigate the relationship between calcium ion (Ca²⁺) kinetics and glycogen, Nielson et al. [67] stimulated intact mouse flexor digitorum brevis muscle fibers for 0.35s every 10s for 42 contractions. Fibers were then divided into fatigued (>75% force decrements) or non-fatigued (<50% force decrements). Intramyofibrillar glycogen was reduced by 68% in the fatigued fibers whereas no significant changes occurred in the non-fatigued fibers. For all fatigued fibers in the study, a strong correlation between low intramyofibrillar glycogen and decreasing tetanic Ca²⁺ concentrations existed. In an in vitro human study, Ortenblad et al. [65] demonstrated that replenishing glycogen via carbohydrate feeding restored sarcoplasmic reticulum Ca²⁺ release 4 h postexercise, whereas in the absence of carbohydrate intake sarcoplasmic reticulum Ca²⁺ release rate remained depressed by 77%. Given that Ca²⁺ handling is also negatively affected in preparations where global ATP is held constant but intramyofibrillar glycogen is reduced [67], glycogen reductions have been suggested to reduce acute force output in an indirect fashion. For example, metabolic disturbances caused by glycogen depletion may occur in a structural nature, as enzymes associated with the glycogen particle may modulate the sarcoplasmic reticulum Ca²⁺ release channels involved in excitation-contraction coupling [68]. Collectively, this data suggests that maintaining intramuscular glycogen via adequate carbohydrate consumption is necessary to both meet the direct metabolic demands during resistance exercise and to indirectly to support rapid calcium kinetics in contracting type II muscle fibers.

Several studies have used diet and exercise to investigate how changes in glycogen may affect resistance exercise performance; however, to our knowledge, only one study directly measured glycogen levels before testing. In that study, Jacobs et al. [69] reported significant reductions in type I and II muscle fiber glycogen content after a prolonged aerobic and sprint interval exercise protocol.

Muscular strength during a single maximal effort dynamic contraction was reduced after glycogen depletion. However, because the strength assessment took place 2 h after the glycogen depletion protocol, it is unclear whether the reductions in force were the result of glycogen depletion or general fatigue because of the prolonged exercise. Leveritt and Abernethy [70] used a similar glycogen depletion protocol followed by 2 d of restricted carbohydrate intake (1.2 ± 0.5 g/kg, $19 \pm 3\%$ energy intake). Compared with a control condition, carbohydrate restriction reduced the total amount of repetitions performed during isoinertial squat with 80% 1 RM but did not affect isokinetic torque during the knee extension. Mitchell et al. [71] subjected individuals to a similar depletion protocol followed by 2 d of a high (7.6 g/kg) or low (0.34 g/kg) carbohydrate diet. In contrast to Leveritt and Abernethy [70], no differences in total repetitions of squat, leg press, or leg extensions were found between conditions. These conflicting results seem paradoxical. Participants in a study by Mitchell et al. [71] performed a higher volume of resistance exercise, likely requiring greater glycolytic energy production, and consumed $\sim 33\%$ less g/kg carbohydrate after depletion. One may assume that low muscle glycogen would have impaired resistance-exercise performance more under this protocol than Leveritt and Abernethy; however, this was not the case. Lambert and Flynn [46] suggested that as a result of shorter rest periods and each set being taken to failure, intramuscular acidosis may have been the cause of fatigue in the Mitchell et al. [71] study rather than carbohydrate availability. A second explanation may have been pre-exercise blood glucose concentrations. Participants completed the resistance exercise in Mitchell et al. [71] in the fasted state, and blood glucose concentrations were similar between conditions at baseline. Although the exercise protocol was similar to what resistance athletes perform in the field, very few athletes perform resistance training in the fasted state. Given that pre-exercise carbohydrate feeding and subsequent increases in blood glucose have been shown in several [72–76] studies to enhance resistance-exercise performance, it is interesting to speculate that had an isocaloric meal matching the respective high-carbohydrate and low-carbohydrate ratios been provided before resistance exercise, baseline blood glucose would be different between conditions and performance may have been affected. Hypothetically, if performance was affected, then perhaps pre-exercise blood glucose is more important than muscle glycogen to maintain high volumes of resistance-resistance; however, future research is necessary to investigate this hypothesis.

Studies examining the effects of pre-exercise carbohydrate feeding/supplementation on resistance-exercise performance have produced mixed results. Lambert et al. [77] reported improvements in leg extension performance with the ingestion of 1 g/kg glucose polymer pre-exercise. In this study, participants performed sets of 10 repetitions using their 10 RM with 3 min of rest for as many sets as possible until 7 repetitions could no longer be completed. Haff et al. [78] reported similar results, where pre-exercise carbohydrate ingestion improved total work and average power during 16 sets of isokinetic leg extension exercise. In another study, Haff et al. [79] investigated the effects of pre-exercise carbohydrate supplementation during twice-daily resistance training. A morning resistance-training session was completed followed by either the consumption of carbohydrate or placebo, and 4 h later, participants performed sets of squats with 10 repetitions and 55% 1 RM to fatigue. Compared with placebo, participants completed more repetitions and more sets with carbohydrate ingestion. Most recently, Krings et al. [80] reported improvements in bench press repetitions with 73% 1 RM and a trend for improved total repetitions as part of a battery of high-intensity resistance exercise, plyometrics, and sprint interval work. On the other hand, Conley et al. [81] reported

no difference in multiple sets of 10 repetition squats with 65% 1 RM to failure when carbohydrate (oats) were consumed before exercise. Vincent et al. [82] reported that pre-exercise carbohydrate ingestion did not affect isokinetic work, power, or torque after a free-weight training session consisting of eight different exercises, and Kulik et al. [83] reported no differences in total repetitions and work between carbohydrate and placebo during five sets of five repetition squats with 85% 1 RM. It is likely that the duration of the sessions in combination with the volume of work completed explains these differences in outcomes. In the studies where ergogenic effects were reported, session durations all lasted ≥ 50 min. In contrast, in three of the four studies where an ergogenic effect was not present, the exercise sessions lasted < 40 min. In the study where the exercise protocol lasted > 40 min [82], total volume completed during the free-weight resistance-training sessions between conditions was not reported. It appears that when combined with a standard diet ($> 55\%$ kcal from carbohydrate), pre-exercise carbohydrate ingestion may only provide an ergogenic benefit when the total training session is longer in duration (> 50 min), higher in volume (> 10 sets), and employs a moderate intensity (50–75% 1 RM). Given that this is the most common exercise prescription for hypertrophy outcomes, it is likely that pre-exercise carbohydrate ingestion is most beneficial to physique athletes and athletes in the general preparation phase. However, additional research is necessary to investigate the ergogenic effects of pre-exercise carbohydrate intake before a typical power lifting or specific preparation type training session (five to six exercises with five to six sets of two to six repetitions) before further recommendations can be made.

Researching examining the effects of non-acute low-carbohydrate intakes on resistance exercise performance in healthy individuals engaged in a resistance-training program are limited. Three studies reported no difference between conditions for peak knee extension/flexion torque, handgrip maximal strength [84], squat jump peak or mean power [85], or 1 RM bench press and knee flexion/extension peak torque [86] after 7 to 21 d of reduced carbohydrate intake. Given the ambiguity of carbohydrate recommendations for strength athletes [17], it is unclear whether the carbohydrate restriction in these studies was severe enough to affect performance. Carbohydrate intakes in these were 30% versus 55% for 7 d, 4.4 g/kg versus 6.5 g/kg for 4 d, and 42% versus 62% for 21 d, respectively. On the other hand, Escobar et al. [87] reported a trend toward an increase in repetitions during a 12-min workout when CrossFit athletes increased carbohydrate consumption from ~ 3 to 6 g/kg per day for 3 d. Given the low volume of work and type of tests conducted in the first three studies, it appears that moderate carbohydrate restriction (30–42% total energy intake) does not affect strength-based performance but increasing carbohydrate consumption could enhance general preparation phase performance (i.e., muscular endurance or resistance-training density).

The effects of more severe carbohydrate restriction on resistance-exercise performance are less clear. For example, Sawyer et al. [88] reported consuming ~ 30 g/d carbohydrate resulted in small, yet significant, increases in back squat 1 RM, handgrip strength, and vertical jump, but not bench press 1 RM or power. Body weight decreased by ~ 1.8 kg during the carbohydrate restriction, but not standard diet, which may have positively changed the power-to-mass ratio and thereby explain the improvement in vertical jump. In addition, the order of conditions may have influenced the results. Participants completed 7 d of standard diet, were tested, then completed 7 d of carbohydrate restriction before being tested again. Therefore, owing to lack of randomization, or baseline testing, it is possible that a learning effect took place. Using a

within-subjects crossover design, Paoli et al. [89] reported no differences in vertical jump nor body weight tests of muscular endurance after 30 d of a ketogenic or standard diet in elite gymnasts. In contrast to Sawyer et al. [88], vertical jump did not improve despite a decrease in body weight in the ketogenic group. The results of this study, however, are confounded by the supplements administered during the ketogenic, but not standard diet, conditions. For example, the sources of caffeine (guarana, coffee) and herbal antioxidant supplements taken during the ketogenic condition may have influenced the training adaptations. Wilson et al. [90] investigated the effects of a 10-wk ketogenic diet compared with a standard diet in conjunction with 8 wk of resistance training in trained men. Bench press and back squat 1 RM increased similarly in both groups. A 1-wk carbohydrate loading (~265 g/d) period was administered following week 10 post-testing, and performance and body composition tests were conducted again at the end of week 11, and the difference in performance outcomes, these results lend some evidence to our hypothesis that pre-exercise blood glucose availability may influence low-volume, high-load strength-based performance to a greater degree than muscle glycogen. The results of these studies, although limited, seem to suggest that although severe carbohydrate restriction may not impair strength adaptations during a resistance-training program, consuming an adequate amount of carbohydrate in the days leading up to testing may enhance strength-based performance (i.e., a powerlifting competition) and strength-endurance-based performance (i.e., CrossFit, physique training, or general preparation phase workouts).

Dietary carbohydrate and muscle glycogen affect postresistance exercise adaptations

Insulin and insulin growth factor 1 (IGF-1) have been shown to play a major role in skeletal muscle anabolism and potentially more importantly, restricting protein breakdown. The receptors of insulin (IR) and IGF-1 (IGF-1 R) have a 45% to 65% homology between their ligand-binding domains and 60% to 85% homology in their tyrosine kinase substrate recruitment domains. It has been suggested that their respective genes have evolved from an ancestral gene that has been highly conserved in the vertebrate family. This hormone/receptor communication initiates metabolic, cellular-growth, and differentiation responses from environmental conditions and nutrient availability [91]. The consumption of carbohydrate stimulates an insulin response, which then initiates an intramuscular cellular signaling process that promotes glucose uptake, yet also promotes muscle protein synthesis (MPS) and restricts the rate of protein breakdown (MPB). This signaling process involves the hormone insulin binding and phosphorylating the IR and insulin receptor substrate-1/2 on tyrosine residues and activates of phosphatidylinositol 3-kinase. The resulting phosphoinositol triphosphate promotes phosphorylation of protein kinase B/Akt, which then phosphorylates various substrates that orchestrate the various physiological effects. Increased glucose uptake is mediated predominantly through phosphorylation of Akt substrate of 160 kDa (AS160) and AS160, and thus facilitates the translocation of GLUT-4 vesicles to the plasma membrane and disinhibition of glycogen synthesis by phosphorylation of glycogen synthase kinase-3. The Akt promoted activation of mammalian target of rapamycin 1 (mTORC₁), and subsequent ribosomal protein p70 S6 kinase and eukaryotic translation initiation factor 4 E-binding protein 1 (4 E-BP1), is responsible for muscle protein synthesis (Fig. 1). Concurrently, Akt-mediated inhibition of forkhead transcription factors activity reduces expression of the E3 ubiquitin ligases that are principally responsible for mediating muscle

atrophy via atrogin-1/muscle atrophy F-box and muscle RING finger-1 [92–95].

It has been shown that an acute bout of resistance exercise may reduce glycogen content by ~23% to 36% [53,59]. This may influence and upregulation of 5'-adenosine monophosphate-activated protein kinase (AMPK), which is an enzyme that appears to function as a metabolic sensor in skeletal muscle because it becomes activated in response to decreased ATP levels. It inhibits ATP-consuming pathways such as MPS and activates pathways involved in carbohydrate and fatty acid catabolism to facilitate resynthesis and restoration of ATP levels. It is responsive to acute exercise and has an adaptive response to chronic exercise training [96]. In addition, AMPK directly phosphorylates at least two proteins to induce rapid suppression of mTORC₁ activity, the TSC2 tumor suppressor and the critical mTORC₁ binding subunit raptor [97], and 4 E-BP1 protein activity [98]. However, newer human model research assessing concurrent training (endurance and resistance exercise) has shown that AMPK may not influence downstream mTORC₁ protein translational activity [99]. With respect to resistance training and carbohydrate availability, Camera et al. [100] reported no difference in AMPK, mTORC₁ phosphorylation, or MPS after resistance exercise in a glycogen-depleted compared with a repleted state. Moreover, and perhaps more importantly for athletes that train in fasted state and then consume food after training, the authors reported glycogen content did not affect the mTORC₁ and MPS response to postexercise carbohydrate and protein ingestion.

Proponents of nutrient timing in the fitness industry have suggested carbohydrate ingestion is necessary to induce the anticatabolic/anabolic effects of insulin after resistance exercise. It should be noted that insulin does indeed promote muscle protein synthesis and slow muscle protein breakdown. However, recently it has been suggested that although insulin, in general, promotes the phosphorylation of intramuscular proteins related to MPS (Akt, mTORC₁, p70 S6 K, and eIF4 E-BP1), insulin concentrations from 30 to 167 IU/mL did not influence a further rise in MPS [101]. Wilkes et al. [102] supported this with data using a euglycemic clamp method showing that a minimal concentration of insulin ~15 IU/mL (3 × postabsorptive) are sufficient to maximally suppress leg MPB by 47% in the younger individuals. This was further supported by a meta-analysis that showed no significant effect of insulin on MPS. The anticatabolic effect of insulin acting on MPB was confirmed in a recent systematic review and meta-analysis of 44 human studies, which concluded insulin did not significantly affect MPS but has a crucial role in reducing MPB. This group revealed that in healthy individuals, the effect of insulin on MPS only becomes significant with an increase of essential amino acid delivery to the skeletal muscle [103]. A systematic review by Trommelen et al. [104] again supported that the effect of insulin on MPS is limited. They concluded that concurrent and exogenous insulin and amino acid administration (ingestion or infusion) effectively increase MPS, but this effect is mainly attributed to the hyperaminoacidemia rather than hyperinsulinemia. Moreover, exogenous insulin administration systemically mediates hypoaminoacidemia (amino acid uptake), which adverts any insulin stimulated effect on MPS. With respect to resistance-exercise adaptations, several studies have investigated the effects of adding carbohydrate to protein in the postexercise feeding. Koopman et al. [105] reported the addition of either 0.15 or 0.6 g/kg carbohydrate did not further enhance the MPS response to 0.3 g/kg protein during a 6-h recovery period from resistance exercise. Despite a fivefold greater insulin area under the curve and greater phosphorylation of Akt, Staples et al. [106] reported the addition of 50 g of carbohydrate did not influence the increase in MPS or reduction in MPB response to the ingestion of 25 g of protein alone. Based on these studies, it

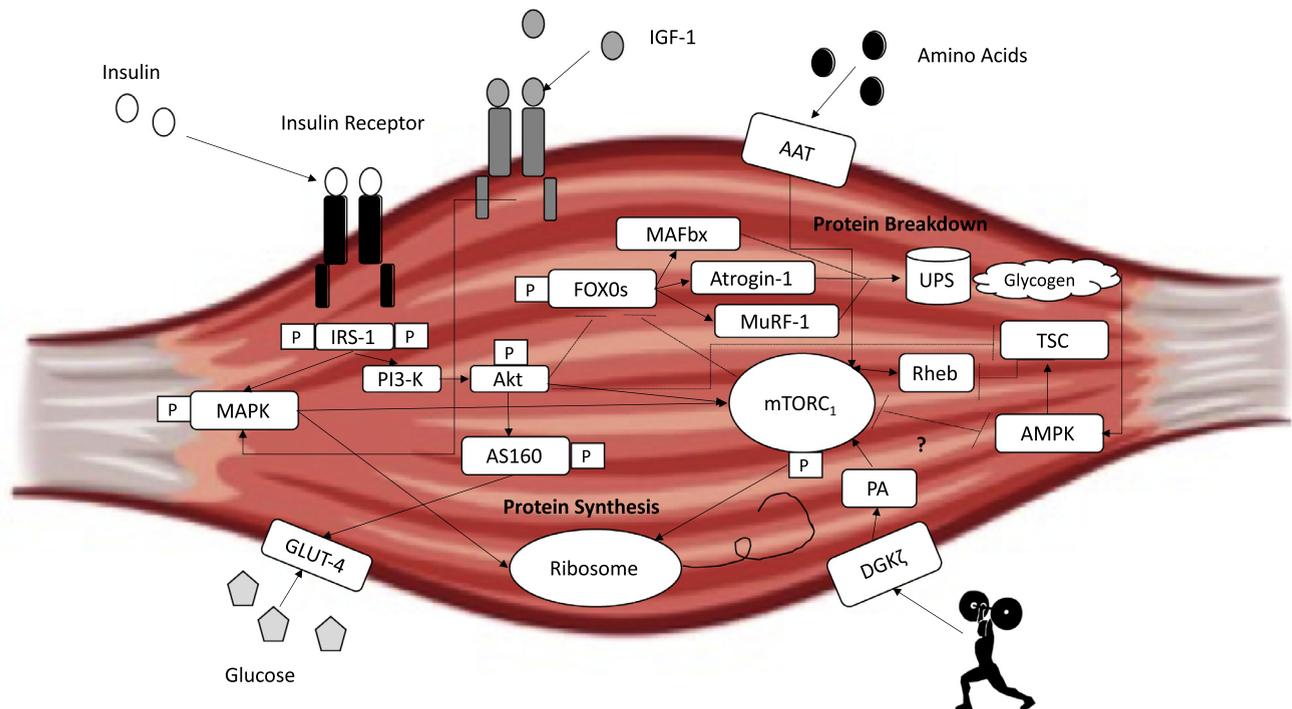


Fig. 1. The coordinated effect of resistance exercise, nutrient intake, and hormonal responses to resistance exercise that regulate and facilitate muscle protein turnover rates. Insulin receptor; Insulin receptor substrate-1 (IRS-1); insulin growth factor-1 (IGF-1); phosphorylation (P); phosphatidylinositol-3-kinase (PI3-K); protein kinase B (Akt); mitogen-activated protein kinase (MAPK); Akt substrate of 160 kDa (AS160); glucose transporter type 4 (GLUT-4); forkhead box protein family (FOXOs); muscle atrophy F-box protein (MAFbx); muscle ring factor-1 (MuRF-1); mechanistic target of rapamycin complex-1 (mTORC₁); phosphatidic acid (PA); diacylglycerol zeta (DGK ζ); amino acid transporter (AAT); Ras homolog, mTORC₁ binding (Rheb); the ubiquitin proteasome system (UPS); 5' adenosine monophosphate-activated protein kinase (AMPK); tuberous sclerosis complex (TSC). Adapted from (Cleasby et al., 2016; Escobar, VanDusseldorp, & Kerkick, 2016; Goodyear et al., 1998; Röckl, Witzack, & Goodyear, 2008).

appears that, acutely, neither glycogen content nor postexercise carbohydrate ingestion effects the molecular adaptations to resistance exercise when a sufficient dose of protein is consumed.

Effects of carbohydrate manipulation on body composition outcomes

When an organism consumes less calories than expended for a consistent period (i.e., >24–48 h) weight loss from reductions in body tissue occur; however, a variety of factors that affect eating behavior and activity will influence both the rate as well as the composition of the weight lost [107]. Given the risk for cardiovascular and metabolic diseases associated with excess adiposity, even in individuals with body mass index <25 kg/m² [108], and the importance of maintaining muscle mass to reduce the risk for sarcopenia, cardiovascular, and metabolic diseases [109], a wealth of studies have been conducted over the past decade examining how different diet compositions affect fat and lean mass changes during a period of caloric deficit. Differences in protein intake have been a major criticism of the greater reductions in fat mass observed in carbohydrate-restricted groups compared with fat-restricted groups [107]. A recent meta-analysis by Hall and Guo [110] assessed 32 studies comparing carbohydrate and fat restriction in overweight/obese individuals. Only studies where protein was matched between groups and food was provided to control for errors in reporting were included. The results of this meta-analysis demonstrated no differences in fat loss between the groups. Individuals in the fitness industry have criticized this research by claiming it does not account for genetic differences and contend that differences in genotype will determine the success of a low-carbohydrate versus low-fat diet. In a large (N=608),

long-term (12 mo) clinical trial, Gardner et al. [111] investigated the relationship between genotype pattern, diet composition, and weight loss. Overweight individuals were randomly divided into a low-carbohydrate group (30% carbohydrate, 45% fat, 23% protein) or low-fat group (48% carbohydrate, 29% fat, 21% protein) and then further stratified into either low-fat genotype (hypothesized to respond better to a low-fat diet) or a low-carbohydrate genotype (hypothesized to respond better to a low-carbohydrate diet or a neutral genotype). No differences were reported between dietary composition groups for weight loss or percent body fat reductions. In addition, there were no interactions between genotype and dietary composition, indicating that individuals mismatched for hypothesized dietary composition (i.e., a low-fat genotype consuming a low-carbohydrate diet) did not lose more fat than those correctly matched. The results of the aforementioned studies seem to suggest that when protein intake is held constant, there is no advantage to adopting a low-carbohydrate diet over a low-fat diet, or vice versa. These studies, however, were conducted in overweight/obese individuals not taking part in a resistance training regimen.

The interaction between carbohydrate-restricted diets and resistance training induced body composition outcomes in healthy individuals has not been well studied. The first study to our knowledge to investigate this phenomenon in strength-based athletes was conducted by Paoli et al. [89]. In this study, 30 d of a very low-carbohydrate diet (~22 carbohydrates daily) resulted in greater reductions in weight and fat mass than a standard Western diet in elite male gymnasts. Given the positive association between higher protein intakes and body composition outcomes, especially in lean athletes [112], these results are not unexpected and likely not attributable to restricted carbohydrates because total energy intake was lower and protein intake was ~2.4-fold greater during the

carbohydrate-restricted condition. In another study, Kephart et al. [113] assigned CrossFit athletes to either a ketogenic low-carbohydrate diet or a control diet for 3 mo. Although there were no significant differences between groups for any body composition variables, a trend was found for a decrease in fat mass and vastus lateralis muscle thickness in the ketogenic diet group. These outcomes are also not unexpected, as the ketogenic diet group consumed less self-reported calories and a trend for more protein compared with baseline measurements. Unfortunately, small sample sizes and lack of dietary reporting in the control group make it difficult to draw further inferences from these results.

As of the writing of this review, only two studies have been conducted whereby a carbohydrate-restricted diet was consumed for >2 mo in conjunction with strength training in trained individuals. In the first study, Wilson et al. [90] reported similar increases in lean mass and vastus lateralis muscle thickness between a protein-matched ketogenic diet and control diet after 8 wk of resistance training. Although individuals were placed on an eucaloric diet, similar decreases in fat mass were observed between groups. In the second study, Vargas et al. [114] had trained men consume a moderate hyperenergetic diet matched for protein (2 g/kg daily) of either 55% or <10% carbohydrate for 8 wk in conjunction with four resistance-training workouts per week. Despite being prescribed a caloric surplus, both groups lost body fat, however, the change was only significant in the ketogenic diet group (~1 kg). Conversely, there was a significant increase in lean mass only in the conventional diet group (~1.3 kg). Total body water increased in the conventional diet group and slightly, but not significantly, decreased in the ketogenic diet group. A major limitation to this study is the lack of dietary reporting in both groups. It is possible that an appetite suppressive effect took part in the ketogenic group, resulting in insufficient energy being consumed to support muscle hypertrophy. Based on these results, however, we can conclude that although consuming a severely carbohydrate-restricted diet may be a viable option for enhancing changes in body composition via reductions in body fat, consuming an adequate amount of carbohydrates in conjunction with a caloric surplus is necessary to optimize muscle hypertrophy during resistance training. More research, however, is necessary to determine a carbohydrate consumption threshold to support hypertrophy in resistance training athletes.

Effects of carbohydrate manipulation on aesthetic outcomes in competitive physique athletes

Physique athletes (i.e., bodybuilding, physique, figure, and bikini divisions) are assessed based on their presentation of an aesthetic appearance, defined by appropriate levels of muscularity, bilateral symmetry, aesthetic balance between various muscle groups, and a low body fat [115]. Ergo, although two athletes of similar stature and muscular development may present with differing body fat percentages, the athlete with the lower measured body fat percentage may not always win. As such, physique athletes must not only dramatically reduce body fat while minimizing reductions in lean mass, but may also go through extreme, sometimes dangerous, protocols in the days leading up to competition (peak week) to reduce subcutaneous extracellular water and increase intracellular water in an attempt to present a “hard” appearance. For example, physique athletes will sometimes significantly increase water intake and sodium consumption at the start of the peak week, and then restrict water and sodium in the days leading up to competition to “reduce subcutaneous water retention” [116]. However, this practice of dehydration will likely reduce intramuscular water and plasma volume [117], which may lead to

lower muscle volume and reduce the efficacy of “pumping up” before posing in competition [118].

Carbohydrate and glycogen manipulation during peak week is a popular strategy employed by physique athletes to “harden” their appearance [119]. Classically, physique athletes would severely restrict carbohydrate intake and perform several glycogen-depleting workouts at the start of the week and then carbohydrate load 1 to 3 d before competition. This approach has been modified by some, but not all contemporary coaches, whereby physique athletes taper training and increase carbohydrate intake during peak week [116]. Although never measured, it is hypothesized that physique athletes will have reduced levels of glycogen because of prolonged periods (generally 15–30 wk) of calorie- and carbohydrate-restricted diets employed during contest preparation [118]. Thus, with adequate carbohydrate loading, even without exhaustive exercise, a level of glycogen supercompensation should occur [120]. It is commonly accepted that each gram of glycogen binds 2.7 to 4 g of water [121] and increases in body water with carbohydrate loading are predominantly the result of an increase in intercellular water [122]. Therefore, carbohydrate loading during peak week should increase muscular volume, which, theoretically, would increase subcutaneous tension, thereby stretching the skin over the musculature and leading to a more muscular and leaner appearance. However, during physique competition, competitors hold isometric contractions intermittently for 30 to 60s with intermittent breaks as they present one pose after another. As a result, intramuscular blood flow may be compromised owing to higher pressures [123], leading to a dynamic exchange in fluid between intra- and extracellular fluid compartments. How varying levels of muscle glycogen affect this dynamic requires further investigation.

Studies examining the quantitative and qualitative effects of this practice are limited. Balon et al. [124] conducted the first study examining the effects of carbohydrate loading on muscle girth in recreationally resistance-trained men. Participants completed 3 d of intense resistance training followed by 3 d of taper. In the glycogen depletion–repletion protocol, participants consumed a eucaloric diet consisting of <10% calories from carbohydrate for 3 d followed by 3 d of 80% carbohydrate intake. Girth measurements of the upper and lower limbs as well as the chest were not different after glycogen repletion nor different from the training-only condition. Because there were no differences in body weight between the control diet and the carbohydrate-rich diet, it is possible that either glycogen depletion did not occur to an appreciable level during the carbohydrate-restricted period or that the carbohydrate loading did not contain enough total calories to induce glycogen supercompensation. In addition, the participants in this study, although recreational weight lifters, were not as lean as physique athletes during peak week. Participants in Balon et al. [124] had an average body fat percentage of $10\% \pm 1\%$. On the other hand, male physique athletes often have body fat percentages of 4% to 8% for competition [20,125]. Therefore, it is possible that changes in muscle volume were not detectable beneath the underlying subcutaneous tissue in this cohort. Finally, comparisons in girth were not made between depleted and repleted states, which are more reflective of the goals of physique athletes during peak week. In support of this hypothesis, Bamman et al. [126] reported a 4.9% increase in biceps thickness the day before competition with carbohydrate loading compared with 6 wk before. Although this was likely the result of carbohydrate loading, intramuscular glycogen was not directly assessed. To our knowledge, only one published study has directly assessed the relationship between carbohydrate loading and muscle volume. Nygren et al. [127] had trained men perform intense cycling for 45 min followed by 4 d of very low carbohydrate (25 g/d) feeding. For the next 4 d, participants consumed

a very high-carbohydrate, low-fat diet. Muscle glycogen in the depleted state decreased to 281 mmol·kg·dry⁻¹ weight and then increased 225% to 634 mmol·kg·dry⁻¹ weight after carbohydrate loading. Vastus muscle and thigh cross-sectional increased by 2.5 and 4 cm², respectively, compared with measures taken in the depleted state. The results of this study demonstrate that carbohydrate loading in a state of partially reduced glycogen leads to an increase in muscle volume. Given that participants in Nygren et al. [127] were untrained, resistance training increases the capacity to store glycogen [128], bodybuilders store 50% more glycogen than untrained individuals [55], and physique athletes have more muscle mass than non-resistance-trained individuals, we speculate that carbohydrate loading during peak week may result in both a greater absolute and relative increase in muscle volume, which in very lean individuals would manifest in visually detectable changes in muscularity and leanness during competition; however, more research is necessary to explore this hypothesis.

Conclusions and Future Perspectives

The metabolic response to resistance training is distinct from endurance training and differs from high-intensity interval training because of longer time under tension and more prolonged eccentric contractions [46]. Moreover, variances in resistance training variable prescription, such as the differences in volume, intensity, and inter-set rest between typical strength and hypertrophy prescriptions, results in varying metabolic responses. Research examining the effects of varying levels of carbohydrate restriction has produced conflicting results. From this body of research, it appears that low glycogen or carbohydrate availability does not negatively affect acute resistance exercise performance when the volume is less than eight sets and duration (<45 min) of exercise is low and the intensity is high (>85% 1 RM). On the other hand, increasing carbohydrates after a period of restriction may enhance both acute strength performance (i.e., 1 RM testing during a powerlifting competition) and muscular endurance (i.e., CrossFit). In addition, increasing blood glucose before acute resistance exercise via carbohydrate ingestion may result in greater work performed during longer duration (>50 min) resistance training sessions with a higher volume (>10 sets) and moderate intensity (50–75% 1 RM). Given that volume is closely related to muscle hypertrophy [129], pre-exercise carbohydrate ingestion may be especially important for hypertrophy outcomes, such as off-season physique athletes and strength athletes completing a general preparation phase. If carbohydrates are restricted to only the pre-exercise period, then it is likely that muscle glycogen will remain low during a period of higher-volume resistance exercise. More research is therefore necessary to investigate the effects of increasing blood glucose in a state of partial glycogen depletion on resistance exercise performance.

The results from a number of molecular studies reveal that reduced glycogen and blood glucose does not negatively influence the acute MPS stimulating effects of postexercise protein ingestion. However, the effects of resistance training with chronically restricted carbohydrate ingestion is conflicting. Protocols where protein ingestion was not matched between groups or varying levels of carbohydrate restriction (i.e., 0.5%, 10%, 30% total energy) have all been defined as restrictive) are likely responsible for differences in lean mass outcomes. Although studies with resistance training athletes are limited, it appears that non-severe levels of carbohydrate restriction (i.e., 30–40% total energy) do not negatively influence hypertrophy adaptations, but severe carbohydrate restriction (i.e., <10% total energy, or ketogenic diets) may compromise muscle hypertrophy during a caloric surplus. In light of these

discrepancies, more research is necessary to investigate where a minimal carbohydrate threshold exists in relation to hypertrophy outcomes during a caloric surplus.

Success in physique sports require attaining high levels of muscularity, low body fat percentages, and the ability to display an aesthetic physique on stage during competition. Based on studies in the overweight/obese populations and one study in resistance-trained individuals, in conjunction with a higher (1.8–2.2 g/kg) protein consumption, maintaining a hypocaloric condition via carbohydrate restriction seems to be a viable strategy to reduce body fat while minimizing lean mass loss. Whether carbohydrate restriction is superior or inferior to fat restriction for improving body composition in strength and physique athletes remains to be answered and necessitates further research. Finally, carbohydrate loading is a popular precompetition strategy to enhance aesthetics in physique athletes. Although increasing carbohydrate consumption after glycogen depletion increases muscle cross-sectional area, future research is necessary to address whether these increases translate to increases in whole muscle volume in very lean individuals. If so, does carbohydrate loading lead to visually noticeable improvements in physique?

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