

Chapter II Review of Literature

A variety of athletes attempt to reduce their body weight in order to achieve a weight class (e.g. wrestlers), to improve performance by decreasing energy cost of activity (e.g. runners), or to change appearance (e.g. bodybuilders). Ideally, this weight loss would occur using modest energy restriction over a long period of time. However it is not unusual for athletes to use more severe energy restriction to cause rapid weight loss.

In an effort to lose subcutaneous fat, bodybuilders usually reduce their caloric intake before a competition. Bodybuilders studied by Bamman et al. (1993) lost an average of 7.3 kg during the 12 weeks before competition. Total caloric intake decreased significantly, 25.8%, from week 6 to the competition. Research has shown that bodybuilders preparing for competition ingested 50 - 60% less calories than bodybuilders who weren't preparing for competition (Rankin 1995). This energy restriction may decrease bodybuilders' performances in the weight room. Bamman et al. (1993) reported a significant decrease in maximal isokinetic force in the dead-lift after bodybuilders dieted and lost 7.3 kg over 12 weeks. Walberg et al. (1988) reported short-term energy restriction can result in significant reductions in muscle endurance of resistance trainers. Trained resistance trainers consumed a formula diet of 18 kcal kg⁻¹ d⁻¹ for seven days and lost a significant amount of weight. The group that consumed high protein/moderate carbohydrate lost 3.6 ± 0.5 kg and the group that had moderate protein/high carbohydrate lost 4.0 ± 0.2 kg. Isometric endurance during knee extensions was significantly reduced after weight loss.

A shorter energy restriction phase has also been reported to decrease anaerobic performance. When a group of wrestlers consumed a formula diet that provided 18 Kcal kg⁻¹ day⁻¹ for 3 days and lost an average of 2.4 kg ± 1.0, their anaerobic performance was significantly lower than before weight loss (Walberg-Rankin et al. 1996).

This decrease in anaerobic performance may be associated with a decrease in muscle glycogen after energy restriction. Tarnopolsky et al. (1996) reported that weight loss was associated with a significant decrease in muscle glycogen. After a group of wrestlers lost 5% of their body weight (p<0.01), muscle glycogen was 54% lower than before weight loss (268 ± 71 versus 124 ± 92 mmol/kg dry wet, p=0.018). When another group of a wrestlers reduced their caloric intake by 66% and decreased their body weight by 2.2% in 48 hour, their muscle strength was reduced (Housten et al. 1981). This was concurrent with a significant reduction in muscle glycogen concentration (29%, p<0.05). A decrease in lean body mass was correlated to a decrease in isokinetic muscle strength when a group of obese women consumed a diet that consisted of 544 kcal d⁻¹ for four weeks (Krotkiewski et al. 1990). After the first two weeks, the women had lost an average of 6.6 kg and muscle glycogen concentration in the vastus lateralis muscle was decreased significantly (80.4 ± 3.1 to 61.5 ± 3.2 umol/g, p<0.01). The subjects also experienced a concurrent decrease in isokinetic muscle strength.

Not all research has observed an association between muscle glycogen and high intensity performance. For example, Grisdale et al. (1990) reported that muscle glycogen depletion had only a minor effect on performance of isometric contractions. Thus, although the data is not consistent, it is possible that the reduction in muscle glycogen with energy restriction may mediate impaired performance of high intensity activity.

Muscle Glycogen and Resistance Training

Keul et al. (1978) stated that ATP and CP stores were the only fuel for heavy-resistance exercise. More recent studies show that resistance exercise elicits a considerable glycogenolytic effect. Tesch et al. (1986) had nine strength trained male athletes perform five sets each of front squats, back squats, leg presses, and knee extensions. Resistance was approximately 10 RM. The subjects rested 1 minute between sets. Muscle biopsies were taken before exercise and 30 seconds after exercise. Muscle glycogen decreased 26% after the resistance exercise (160 ± 20 to 118 ± 24 mmol kg⁻¹ wet weight, $p < 0.001$).

Essen-Gustavsson and Tesch (1990) found a significant decrease in muscle glycogen after nine bodybuilders performed heavy-resistance exercise. Subjects performed five sets each of front squats, back squats, leg press, and leg extensions. Each set ranged from 6 to 12 reps and the weight was selected so that failure occurred within 12 repetitions. Subjects rested 1 minute between sets. The exercise protocol lasted approximately 30 minutes. Muscle biopsies were taken from the vastus lateralis muscle before and 30 seconds after exercise. Muscle glycogen decreased 28% (690 ± 82 to 495 ± 95 mmol kg⁻¹ dry weight, $p < 0.001$) after the resistance exercise.

MacDougall et al. (1988, abstract) also observed a decrease in muscle glycogen following resistance exercise. Eight bodybuilders performed single-arm biceps curls. Subjects performed either one set of 10 RM or three sets of 10 RM with three minutes rest between sets. Muscle biopsies were taken from the biceps brachii before and immediately following completion of the exercise test. After the single set muscle glycogen decreased by 13% (375 to 327 mmol kg⁻¹ dry weight). Muscle glycogen decreased by 25% (372 to 278 mmol kg⁻¹ dry weight) following the 3 sets biceps curls. Data regarding the total work done during the different protocols were not reported.

Another study found that when experienced resistance trainers performed leg extensions for 6 sets of 6 repetitions at 70% of 1 RM, muscle glycogen decreased 39% (120.3 ± 10.8 to 73.4 ± 8.1 mmol kg⁻¹ wet weight) (Robergs et al. 1991). On the next day, the same subjects used the opposite leg to perform six sets of leg extensions at 35% of 1 RM. The number of repetitions varied, because leg extension exercise continued in each set until force output matched the 70% of 1 RM trial. After this low intensity trial, muscle glycogen decreased a similar amount, 38% (122.4 ± 9.7 to 75.9 ± 9.2 mmol kg⁻¹ wet weight). Thus, glycogen depletion with resistance exercise in this study appeared to be related to total work done.

The above studies show that muscle glycogen is a fuel for resistance training. These studies examined the affects of resistance workouts performed by trained subjects.

Untrained subjects may experience different rates of muscle glycogen depletion during a resistance workout. These studies also show that upper and lower body workouts result in decreased muscle glycogen. If muscle glycogen is already lower following energy restriction, the additional depletion occurring during resistance training may cause premature fatigue.

Carbohydrate Ingestion and Resistance Performance

Numerous studies have shown that carbohydrate ingestion delays fatigue during endurance exercise. Mechanisms suggested to explain the ergogenic effect of carbohydrate include maintenance of blood glucose permitting high rates of carbohydrate oxidation (Coggan and Coyle 1991) and muscle glycogen sparing (Hargreaves et al. 1984). The value of carbohydrate consumption before resistance training is less clear.

Lambert et al. (1991) investigated the effect of consuming a glucose polymer solution before and during a single bout of resistance exercise. Each subject participated in a cross over design where he either consumed a 10% glucose polymer solution or a placebo beverage immediately before exercise and after the 5th, 10th, and 15th sets. The glucose polymer solution was 1g carbohydrate per kg before exercise and .17g carbohydrate per kg during exercise. The exercise protocol consisted of knee extension exercises done at 80% of 10 RM. The subjects rested three minutes between sets. When the 10th rep could no longer be extended to 170 degrees the next set was nine reps and so forth. When the subject could no longer extend the 7th rep to 170 degrees he was considered fatigued and testing ended.

Subjects were told to refrain from exercise during the 48 hours before the test. During the time before the first test, subjects recorded their food intake. This diet was then duplicated during the 48 hours preceding the second test. The subjects fasted for 10 hours and then were feed a meal that consisted of 728 calories, 51% carbohydrate, 14% protein, and 35% fat. Four hours later the exercise test began.

Performance was measured as number of sets to exhaustion. The researchers found a trend toward improved performance ($p=0.067$), but it was not significant. When performance was considered as the total number of repetitions done, another non-significant trend was observed ($p=0.056$). Approximately 15 sets of knee extensions were done per subject and the average exercise time was 56 minutes.

On the other hand, Conley et al. (1995, abstract) did not observe an increase in performance when a carbohydrate beverage versus a placebo was ingested 15 minutes before and during multiple sets of parallel squats. It was not reported how much carbohydrate was given. The subjects performed sets of 10 reps at 65% of 1 RM to failure with 3 minutes of rest between sets. Performance was measured in number of sets, number of repetitions, and total work done. In another study, consumption of a carbohydrate beverage (100g CHO) immediately before a free weight workout did not increase isokinetic performance after the workout (Vincent et al. 1993, abstract). The free weight workout consisted of multiple sets of squats, leg press, leg extensions, calf press,

barbell curl, preacher curl, and dumbbell curl. Each set consisted of 12 to 15 repetitions with 60 seconds between sets. After the free weight workout, subjects performed isokinetic knee extensions and forearm flexors for three sets of 15 repetitions. There was no significant difference observed in total work done, average power, peak torque, and work fatigue between the carbohydrate and placebo trials.

It is not clear why the effects of carbohydrate consumption on resistance exercise performance have been conflicting. However, the duration of the resistance exercise and therefore the drain on muscle glycogen may be important. For example, the performance test used by Conley et al. (1995) was 35 minutes while the test used by Lambert et al. (1991) was 56 minutes. Carbohydrate availability may not have become a limiting factor during 35 minutes of exercise, but may have become limiting during 56 minutes of exercise (Conley and Stone 1996). For all three of these studies, subjects consumed the carbohydrate very close to the time of exercise (Lambert et al. 1991, Conley et al. 1995, and Vincent et al. 1993). This may be why no benefit from consuming the carbohydrate versus placebo was observed. There may not have been enough time for the ingested carbohydrate to be digested, enter the blood and be transported into muscles prior to exercise. Studies have reported that blood glucose peaks 30 minutes after ingesting carbohydrate (Jandrain et al. 1989, Pallikarakis et al. 1986).

The maintenance of blood glucose permits high rates of carbohydrate oxidation (Coggan and Coyle 1991) and muscle glycogen sparing (Hargreaves et al. 1984) during aerobic exercise. Even though a decrease in blood glucose has not been observed during resistance exercise (Keul et al. 1978), a higher level of blood glucose at the start and during resistance exercise may be beneficial. Lambert et al. (1991) reported that blood glucose was significantly higher after the 7th set of leg extensions and at fatigue when subjects consumed a carbohydrate beverage immediately before exercise versus when a placebo was consumed. Conley et al. (1995) reported that subjects who consumed a carbohydrate beverage 15 minutes prior to exercise had higher blood glucose levels pre-exercise, immediately after exercise, and one and two hours after exercise than when a placebo was consumed. Since a significant decrease in muscle glycogen has been reported after resistance training (Tesch et al. 1986, MacDougall et al. 1988, Robergs et al. 1991), ingesting a carbohydrate beverage prior to resistance exercise may increase blood glucose availability and improve performance if muscle glycogen levels becomes limiting.

If muscle glycogen levels are reduced due to energy restriction, the additional depletion occurring during resistance exercise may cause muscle glycogen to become a limiting factor for muscle performance. Thus, carbohydrate supplementation may be of more benefit for performance when the athlete is in a negative energy balance. Ingesting carbohydrate before a resistance training session may allow some glycogen synthesis prior to exercise, delay fatigue, and increase the total work done. With carbohydrate ingestion, muscle glycogen resynthesis can occur at approximately 7-10 mmol kg⁻¹ wet weight per hour compared to only 1-2 mmol kg⁻¹ when no carbohydrates are consumed (Robergs 1991).

After a resistance exercise bout, carbohydrate ingestion has been reported to increase muscle glycogen resynthesis compared to consuming water (Pascoe et al. 1993). Subjects performed sets of 6 repetitions of one-legged leg extensions at 70% 1 RM, with 30 seconds between sets, to failure. Muscle glycogen was decreased by 30% after exercise. Subjects consumed a carbohydrate beverage (1.5g CHO/kg) or water immediately and 1 hour post exercise. Muscle glycogen content did not significantly change during the 2 hours post exercise for the water group, but there was a significant increase for the carbohydrate group (91.7 ± 11.8 to 117.6 ± 16.5 mmol kg⁻¹ wet weight, $p < 0.05$). This shows that if muscle glycogen has been reduced by exercise, muscle glycogen synthesis can occur at a rate of about 13 mmol kg⁻¹ hour⁻¹ when carbohydrates are ingested. There may be a similar rate of resynthesis for subjects who consume carbohydrates when they have reduced muscle glycogen due to energy restriction.

Resistance Exercise and Cortisol

Another potential benefit of carbohydrate consumption before resistance exercise may be a reduction in the catabolic state (e.g. muscle damage and protein breakdown) that has been observed following this activity as reflected by increases in plasma creatine kinase (Paul et al. 1989, Kraemer et al. 1993) and cortisol (Kraemer et al. 1987, Kraemer et al. 1993). Cortisol is released from the adrenal glands which sit on top of the kidneys. It is an important metabolic hormone that enables the body to resist stress by increasing blood glucose, fatty acid, and amino acid levels and enhancing blood pressure. The main metabolic effect of cortisol is gluconeogenesis, the formation of glucose from amino acids. An increase in cortisol could be a concern for athletes because cortisol may cause muscle proteins to be broken down to provide amino acids for repair, for making enzymes used in metabolic processes, or substrates for gluconeogenesis. Excessively high levels of cortisol depress activity of the immune system and inhibit the inflammatory response by stabilizing lysosomal membranes and preventing vasodilatation (Marieb 1992). This may be of importance to the athlete because after exhaustive exercise that results in glycogen depletion, it has been reported that the muscle shows signs of damage including the presence of lysosomes and vacuoles and a disruption of striation pattern (Appell et al. 1992).

Cortisol has been reported to increase during and up to 15 minutes after exercise when trained subjects performed repeated sets of 10 RM lifts (three sets of eight different exercises) with only one minute rest between sets (Kraemer et al. 1993). When the intensity and rest between sets was increased (5 RM with three minutes rest) significant increases in cortisol were not observed. The researchers concluded that cortisol release is related to the duration of force production and the length of rest periods between sets.

A significant increase in plasma cortisol levels has been reported when trained subjects rested for very short periods between resistance exercises (Kraemer et al. 1987). Subjects performed three sets of 10RM of bench press, double leg extensions, shoulder press, double leg curls, upright row, leg press, lat pull down, seated calf raises, two-arm curls, and hang cleans. They rested 10 seconds between sets and alternated 30 and 60

seconds of rest between exercises. There was a significant increase in cortisol levels from pre to 5 minutes post exercise (from about 400 to 625 nmol L⁻¹, p<0.05).

Longer rest periods between sets have not been associated with increased cortisol levels (Nieman et al. 1995, Guezennec et al. 1986). When trained subjects rested 3 minutes between sets of 10 repetitions at 65% 1RM of the parallel leg squat, no significant increase in cortisol was observed (Nieman et al. 1995). Cortisol levels only increased slightly from 691 ± 87 to 719 ± 72 nmol L⁻¹ from pre to post exercise. Additionally the small change in cortisol levels reported by Guezennec et al. (1986) after resistance exercise may also be due to the long rest periods. During this study, trained subjects performed bench press at 70% of 1RM for six sets with 3.75 minutes between sets. Thus, resistance trainers who utilize long rest periods are less likely to experience increases in cortisol levels.

Cortisol release may also be associated with the amount of muscle mass utilized in the resistance workout. After trained subjects performed sets of bench press, cortisol did not significantly change from baseline (Guezennec et al. 1986). This may be because the bench press utilizes a smaller muscle mass than squats; therefore, metabolism is not raised as high (McMillian et al. 1993). An increase in post exercise cortisol levels was observed when subjects performed multiple sets of squats (McMillian et al. 1993). Subjects performed dead-stop squats with 2.5 minutes between sets and each set consisted of 10 repetitions. Set 1 was done at 40% of 1 RM, set 2 at 50%, and sets 3, 4 and 5 at 60%. Subjects then rested for two minutes and performed ¼ squats with one minute of rest between sets and each set consisted of 10 repetitions. Set 1 was at 60% of 1 RM for the dead-stop squat, set 2 was at 75%, and sets 3, 4, and 5 were at 90%. After 2 minutes of rest, the subjects performed three sets of 10 vertical jumps with one minute of rest between sets. Five minutes post exercise, trained subjects had somewhat higher cortisol levels (486 ± 57 nmol/L) compared to non-exercising control subjects (317 ± 42 nmol/L).

Cortisol release in response to resistance exercise may be affected by exercise training. The above studies examined cortisol levels in trained subjects. When McMillan et al. (1993) looked at the cortisol response in untrained subjects five minutes after exercise, cortisol levels were significantly higher for untrained subjects (600 ± 58 nmol/L) compared to control subjects (317 ± 42 nmol/L) who did not perform resistance exercise. The untrained subjects also had higher cortisol levels five minutes post exercise compared to trained subjects levels (486 ± 57 nmol/L). This may mean that the resistance workout was less stressful for the trained subjects even though they were working at a higher absolute workload.

Increased cortisol levels after resistance exercise may affect the function of the immune system. Dohi et al. (1997, abstract) concluded that an increase in cortisol during exercise may be associated with lower lymphocyte T and B cell proliferation response. Non-strength trained women performed six sets of 10RM squats with two minutes rest between sets. Subjects who had a high cortisol response (>1000nmol L⁻¹) during exercise also had

significantly lower lymphocyte proliferation response. Those who had a low cortisol response ($<500 \text{ nmol L}^{-1}$) did not have a decrease in lymphocyte proliferation response. This suggests that a high cortisol response to resistance exercise may decrease the function of the immune system.

Cortisol has been reported to increase after resistance exercise. This increase may be related to rest intervals, muscle mass utilized, and training level of subjects. An increase in cortisol may have negative consequences for resistance trainers since cortisol may cause muscle proteins to be broken down to provide amino acids for repair, for making enzymes used in metabolic process, or substrates for gluconeogenesis. Additionally, high cortisol levels may inhibit the immune response.

Diet and Cortisol

Cortisol levels in healthy men have been shown to significantly increase during fasting (Beer et al. 1989, Veldhuis et al. 1993, Bergendahl et al. 1996). Beer et al. (1989) reported that on the first morning of fasting cortisol levels increased from $64.8 \pm 6.7 \text{ nmol/L}$ before fasting to $741 \pm 90 \text{ nmol/L}$. On the second morning, cortisol levels were $1900 \pm 39 \text{ nmol/L}$. Bergendahl et al. (1996) reported smaller changes in cortisol levels. After five days of fasting, cortisol levels increased from 284 ± 19.9 to $594 \pm 33.8 \text{ nmol/L}$. Fichter et al. (1984) reported that when healthy young women fasted and lost 8.0kg of body weight, cortisol levels were significantly increased ($7.33 \pm 4.2 \text{ ug/100mL}$ to $12.41 \pm 2.9 \text{ ug/100mL}$). Cortisol levels returned to normal when the subjects gained weight. When female subjects fasted for 24 hours they had higher cortisol levels than subjects who did not fast (Bonen et al. 1983). These results suggest that energy restriction may increase cortisol levels

The protein/carbohydrate ratio has been reported to affect cortisol levels (Anderson et al. 1987). Subjects first consumed a high protein diet and then consumed a high carbohydrate diet. The first diet consisted of 44% protein, 35% carbohydrate, and 21% fat. The second diet contained 70% carbohydrate, 10% protein, and 20% fat. The diets were isocaloric and lasted 10 days each. The calorie contents were based on diet records completed before the study and each subject received optimal energy intake for weight maintenance. Subjects maintained normal exercise levels throughout the study. Cortisol levels were significantly higher after the high protein diet than the high carbohydrate diet. These results suggest that when a higher carbohydrate diet is consumed, less cortisol is released.

However, Volek et al. (1997) reported that cortisol levels for resistance trainers were not affected by daily food intake. Trained subjects performed a bench press protocol of five sets of 10RM and a jump squat exercise protocol for five sets of 10 repetitions at 30% of the subject's 1RM squat. Subjects rested two minutes between all sets. Subjects recorded their food intake for 17 days. These records were analyzed for total calories, carbohydrate, protein, fat, saturated fatty acids, monounsaturated fatty acids, polyunsaturated fatty acids, cholesterol, and dietary fiber. There was no significant correlation between any nutritional variables and cortisol levels prior to exercise or five

minutes post exercise. Cortisol levels were not significantly increased after exercise. This may be due to the small muscle mass used during bench press (McMillian et al. 1993), the timing of the blood samples, and/or the amount of rest between sets (Kraemer et al. 1993). The researchers stated that if blood samples were taken later in recovery perhaps they would have found a correlation between dietary intake and cortisol levels. The mean carbohydrate intake was 56%, the low was 48%, and the high was 69%. These values are not as high (70%) or low (35%) as the percentages used in the study by Anderson et al. (1987). Thus, there may not have been enough variation in the macronutrient intake to see an effect on cortisol.

Tsai et al. (1993) also reported no change in cortisol levels during three days of either a carbohydrate rich diet (55% CHO, 15% protein, and 30% fat) versus a protein/fat rich diet (15% carbohydrate, 25% protein, and 60% fat). Half the subjects consumed the carbohydrate diet first, the other half consumed the protein/fat diet first. Subjects cycled at 80% of VO_2max until perceived exhaustion at 19 or 20 on Borg's scale or up to 20 minutes. After the carbohydrate rich diet all 6 subjects finished the test, but only 2 subjects were able to complete the test after the protein/fat rich diet. Despite the differences in exercise performance, there were no significant differences in cortisol levels pre or post exercise between the two diets.

Additionally, Witt et al. (1993) reported that cortisol levels were not affected by the percentage of carbohydrate in the diet. Eight trained and seven sedentary male subjects consumed three different diets that lasted five days each. The low carbohydrate diet provided 15% carbohydrate, 15% protein, and 70% fat. The medium carbohydrate diet supplied 45% carbohydrate, 15% protein, and 40% fat. The high carbohydrate diet furnished 75% carbohydrate, 15% protein, and 10% fat. The diets were given in four different orders and 36 hours separated each diet. Trained subjects did have slightly higher cortisol levels than untrained subjects, but there were no significant differences in cortisol levels following the diets.

In summary, diet may affect cortisol levels. Fasting has been reported to increase cortisol levels. (Beer et al. 1989, Comeron et al. 1991, Veldhuis et al. 1993, Bergendahl et al. 1996, Fichter et al. 1984). The amount of carbohydrate in the diet may also affect cortisol levels. Anderson et al. (1987) found that a higher carbohydrate diet resulted in lower cortisol levels than a high protein diet. Other studies have found that the percentage of macronutrients did not affect cortisol levels (Tsai et al. 1993, Volek et al. 1997, Witt et al. 1993). The explanation for different effects of carbohydrate consumption on cortisol levels observed by different research groups is not clear. At this time no definite conclusion can be drawn about the influence of macronutrients on cortisol release.

Carbohydrate supplementation and cortisol response to exercise

Ingesting carbohydrate prior to exercise may reduce the necessity for gluconeogenesis and therefore inhibit the release of cortisol. Lower post exercise cortisol levels have been reported when carbohydrate was consumed during aerobic exercise as compared to placebo (Anderson et al. 1991, Deuster et al. 1992, Mitchell et al. 1990). When subjects

consumed a water placebo during a cycling bout, they had significantly higher cortisol levels than when a carbohydrate beverage was consumed (Mitchell et al. 1990). Subjects performed four randomized cycling trials involving seven 12-minute cycling bouts at 70% of VO_2max and a final all-out self-paced 12 minute ride. In between cycling bouts, subjects rested for 3 minutes. During this time they consumed their assigned beverages. Over the four trials, subjects consumed 5.0, 6.0, and 7.5g/100ml carbohydrate solutions and a water placebo. At 85 minutes, all of the carbohydrate trials resulted in significantly lower cortisol levels than the water placebo. This suggests that carbohydrate supplementation diminished the glucose counterregulatory response of trained athletes during continued exercise.

Carbohydrate loading has been reported to decrease cortisol levels post exercise (Anderson et al. 1991). Subjects consumed either 600g or 300g of carbohydrate daily for three days. The exercise test consisted of eight sessions of 10 minutes of rest and 20 minutes of exercise on a cycle ergometer. The subjects were in 25 degree Celsius water up to their necks. The exercise output increased after the carbohydrate loading diet and cortisol levels were significantly different post exercise compared to the low carbohydrate diet (858 ± 77 vs. 1152 ± 94 nmol l^{-1}). There was no difference in pre exercise cortisol levels between diets. Exercise increased cortisol levels, but the increase was lower after the high carbohydrate diet. If carbohydrate ingestion before resistance exercise decreased cortisol response, there may be a more favorable metabolic environment for an increase in muscular mass and strength.

CK and resistance training

Plasma CK, an indicator of muscle membrane leakage and damage, has been reported to significantly increase within 12 hours after a resistance exercise bout that consisted of three sets at 70 - 80% of 1 RM for bench press, shoulder press, latissimus pulldown, arm curl, leg press, and leg curl (Paul et al. 1989). Each exercise was performed until fatigue and the subjects rested one minute between sets and two minutes between exercises. There was a significant increase in CK 12 and 24 hours post exercise for both trained (175 iu L^{-1} at baseline to 550 iu L^{-1} at 12 hours and to 400 iu L^{-1} at 24 hours) and untrained subjects (from about 125 iu L^{-1} at baseline to 650 iu L^{-1} at 12 hours and to 675 iu L^{-1} at 24 hours). However, the increase in CK was less for the trained subjects than the untrained subjects at 24 hours post exercise ($p < 0.05$). Thus, exercise training may affect the change in CK levels.

When sedentary men between the ages of 50 and 69 began a resistance training program, the increase in CK eight hours after the first session (278 ± 175 U/L) was twofold above baseline (133 ± 88 U/L, $p < 0.01$) (Hurley et al. 1995). The training sessions consisted of multiple resistance exercises with 90 seconds of rest between sets. For each exercise, only one set was performed. Each set began with four to five repetitions at a 5RM resistance then the weight was decreased just enough so the subject could perform another two or three repetitions. This continued, with no break in the cadence, until 15 repetitions were completed. At the end of the 16 week training program, the subjects were tested at the same resistance as at the beginning (absolute) and a new resistance adjusted for the

current 5RM (relative). The increase in CK after the absolute session was 47% lower ($p < 0.01$) than after the first session. The increase after the relative session was 35% lower ($p < 0.05$) than following the first session. These results suggest that there was a training effect on CK, but the marker for muscle membrane damage still increased somewhat after the training program.

Tiidus and Ianuzzo (1983) examined the effects of intensity and duration of resistance exercise performed by untrained males on CK. Each set consisted of 10 repetitions, but eventually the subjects fatigued and were unable to finish 10 reps. The subjects rested one minute between each set. When subjects performed a total of 150 knee extensions at 35% of 10RM, CK did not significantly increase after exercise, but there was a significant increase after 70% and 90% of 10RM. The 200% increase after the 90% of 10RM bout was significantly greater than after the 70% bout. No significant increase in CK was seen when subjects did 100 or 200 knee extensions at 70% of 10RM, but there was a significant increase following 300 repetitions. When the total work was kept constant, CK was elevated significantly more following the highest intensity and shortest duration (80% of 10RM for 170 reps) compared to the longer duration and lower intensity protocol (30% of 10RM for 545 reps). The peak values for CK were seen 24 hours post exercise. The results of this study suggest that higher intensity resistance exercise workouts will produce a greater rise in CK indicating greater damage done to the muscle membrane. This damage may be related to cortisol release and increased use of muscle glycogen. Kraemer et al. (1993) found that the rise in CK 24 hours post exercise was significantly correlated to the rise in cortisol 5 minutes post exercise ($r = 0.84$) for the 10 RM/1 minute rest protocol (details described above). All protocols resulted in elevated CK 24 hours post exercise. CK may be lower post exercise, if carbohydrate consumption before resistance training decreases cortisol and muscle damage.

Carbohydrate and muscle damage

Several studies have examined whether or not carbohydrate ingestion influences CK response to aerobic exercise. Cade et al (1991) reported that consumption of a carbohydrate electrolyte drink before a swim workout reduced the increase in CK in competitive swimmers. When the swimmers only consumed water before and during workouts lasting 120 minutes, CK was 315 ± 122 IU/l. For a week, swimmers either consumed a glucose-electrolyte solution (6% carbohydrate) or an equal volume of water before and during the 120 minute workout. After a week of increased training, CK increased to 500 ± 180 IU/l for the group consuming water but decreased for the group consuming the glucose-electrolyte solution (280 ± 15 IU/l). This study is flawed in that the subjects were allowed to consume ad libitum diets that were not measured. However, ingesting a glucose-electrolyte solution before and during the swim workout resulted in significantly lower CK levels versus consuming water ($p < 0.05$).

In contrast, Kirwan et al. (1990) reported that CK was not affected by the amount of carbohydrate athletes consumed for five days. For this study, ten runners exercised for around 80 minutes a day at about 80% of VO_2max for five days (days 4-8 of the study). They consumed carbohydrates estimated to be equal to (8g CHO/d) or below (3.9g

CHO/d) energy expenditure. The final three days were rest days. The five days of intense training resulted in significant increases in CK, but there was no significant difference between the two carbohydrate groups. The group that consumed 8g CHO/kg had CK values of 106 ± 18 , 296 ± 81 , and 327 ± 70 mU/ml for days 4, 7, and 9. The group that consumed 3.9g CHO/kg had values of 103 ± 13 , 257 ± 47 , and 352 ± 68 mU/ml. Thus, the effect of dietary carbohydrate on CK response to exercise is controversial.

The presence of 3-methylhistidine (3MH) in the urine is an indicator of muscle protein breakdown. Viru and Seli (1992) reported that urinary 3MH peaked 12 to 14 hours after an acute bout of strength and power training. The increase in 3MH was higher for subjects who performed at 70% of 1RM than for subjects who performed at 50% of 1RM. Roy et al. (1996) observed that when subjects consumed a carbohydrate beverage (1g CHO/kg) immediately post and one hour after resistance exercise, 3MH was significantly lower than when the same subjects consumed a placebo. This suggests that there is less protein breakdown when carbohydrate is consumed. This may be related to a decrease in cortisol when carbohydrate is consumed. A similar benefit may be seen in subjects in negative energy balance who ingest carbohydrate prior to resistance exercise.

Rate of perceived exertion and carbohydrate ingestion

A relationship between carbohydrate ingestion and lower Rate of Perceived Exertion (RPE) has been observed in studies using prolonged aerobic exercise. Wilber and Moffatt (1992) reported the RPE was significantly lower when subjects consumed a 7% carbohydrate beverage than when they consumed a placebo prior to and during running on a treadmill. Additionally, when subjects consumed an 8% glucose/electrolyte drink versus a placebo every 15 minutes during 180 minutes of cycling at 70% VO_2 max, they reported lower RPE for the legs (Burgess et al. 1991). Plasma glucose was higher during the exercise except at 15 and 90 minutes for the glucose trial. The researchers concluded that a physiological link exists between plasma glucose, carbohydrate oxidation during prolonged exercise, and rate of perceived exertion. A similar relationship may be seen in subjects in negative energy balance who ingest carbohydrate prior to resistance exercise.

Another study also found a relationship between blood glucose levels and RPE (Robertson et al. 1996). Subjects consumed a 6% glucose/sucrose solution at the rate of $0.6 \text{ g kg}^{-1} \text{ hr}^{-1}$ or an equal volume of artificially sweetened placebo every 20 minutes throughout a cycling bout at 70% VO_2 max for two hours. There was no difference in RPE values for the first 80 minutes of exercise, but there was a significant difference at 100, 120, and 140 minutes. Over-all RPE and RPE for legs was significantly lower ($p < 0.05$) during the carbohydrate trial versus the placebo trial. The researchers concluded that blood glucose provided by the carbohydrate supplement reduces the rate of perceived exertion during the later stages of prolonged exercise at a moderate intensity.

Weight loss may also affect RPE. Horswill et al. (1990) reported that when wrestlers lost approximately 6% of their body weight RPE scores were higher during a six minute bout of high intensity arm cranking than before weight loss. Subjects underwent two four day periods of energy restriction. The order of the diets was randomly assigned and a one

week washout period occurred between the two diets. The high carbohydrate diet consisted of 65.9% carbohydrate, 11.4% protein, and 22.7% fat. The low carbohydrate diet contained 41.9% carbohydrate, 11.4% protein, and 46.7% fat. There was no interaction for RPE and type of diet, but there was a significant effect for weight loss. This study demonstrates that energy restriction can increase rate of perceived exertion. A similar effect may occur with resistance trainers who undergo energy restriction.

Summary

Athletes frequently pursue ways to reduce body weight in order to improve physique, attain a weight class, or reduce the energy cost of activity. This weight loss may have negative consequences. There is evidence that athletes who reduce their caloric intake have reduced muscle glycogen and performance. Carbohydrate supplementation prior to resistance exercise may increase blood glucose availability and improve performance if muscle glycogen levels become limiting. Even though some carbohydrate beverages are marketed for improved performance during resistance training there is very little evidence to support this.

Another potential benefit of carbohydrate supplementation prior to resistance training would be a reduction in markers associated with protein catabolism and muscle damage. Resistance training has been reported to cause increases in cortisol and creatine kinase levels. The necessity for gluconeogenesis may be reduced if carbohydrate is ingested prior to resistance exercise and hence the release of cortisol would be inhibited. A carbohydrate supplement may also reduce muscle damage, thus reducing CK levels.

Additionally, ingesting carbohydrate prior to resistance exercise may reduce the rates of perceived exertion. A relationship between blood glucose levels and RPE has been reported for aerobic training. The same benefit may be found during resistance training.

These issues have not been addressed for resistance trainers who are in a negative energy balance. It would be worthwhile to determine from controlled research, if a carbohydrate supplement before resistance training has some of the positive effects as shown for aerobic exercise.