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Hormonal Response to Carbohydrate Supplementation at Rest and After Resistance Exercise

Sean R. Schumm, N. Travis Triplett, Jeffrey M. McBride, and Charles L. Dumke

This investigation examined the anabolic-hormone response to carbohydrate (CHO) supplementation at rest and after resistance exercise. Nine recreationally trained men randomly underwent 4 testing conditions: rest with placebo (RPL), rest with CHO (RCHO), resistance exercise with placebo (EPL), and resistance exercise with CHO (ECHO). The resistance-exercise protocol was four sets of Smith machine squats with a 10-repetition-maximum load, with 90-s rests between sets. Participants then consumed either a placebo or CHO (24% CHO, 1.5 g/kg) drink. Blood was taken before exercise (Pre), immediately after testing (Post), and then 15 (15P), 30 (30P), and 60 (60P) min after drink ingestion. Blood was analyzed for cortisol, glucose, insulin, and total testosterone (TTST). Cortisol did not change significantly in any condition. Glucose concentrations increased significantly from Pre to 15P and 30P during RCHO and Pre to 15P, 30P, and 60P in ECHO ($p \leq .05$). Insulin concentrations increased significantly from Pre to 15P, 30P, and 60P in the RCHO and ECHO conditions ($p \leq .05$). There were no significant changes in TTST concentrations during RPL or RCHO. Both EPL and ECHO demonstrated a significant elevation in TTST concentrations from Pre to Post ($p \leq .05$). During ECHO, TTST concentrations at 60P were significantly lower than Pre levels ($p \leq .05$), but there were no significant treatment differences in TTST concentrations at any time point during the EPL and ECHO conditions. Ingesting CHO after resistance exercise resulted in decreased TTST concentrations during recovery, although the mechanism is unclear.

Keywords: strength training, metabolism, nutrition

Resistance exercise has been associated with acute elevations in testosterone concentrations in both trained and untrained participants (Ahtiainen, Pakarinen, Alen, Kraemer, & Hakkinen, 2003; Ahtiainen, Pakarinen, Kraemer, & Hakkinen, 2003; Chandler, Byrne, Patterson, & Ivy, 1994; Kraemer et al., 1999, 1990; Kraemer, Volek, Bush, Putukian, & Sebastianelli, 1998; Linnamo, Pakarinen, Komi, Kraemer, & Hakkinen, 2005; Raastad, Bjoro, & Hallen, 2000; Smilios, Pilianidis, Karamouzis, & Tokmakidis, 2003; Tremblay, Copeland, & Van Helder, 2004).

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These acute elevations in testosterone typically persist for up to 30 min after the exercise bout (Ahtiainen, Pakarinen, Alen, et al.; Ahtiainen, Pakarinen, Kraemer, & Hakkinen; Chandler et al.; Kraemer et al., 1999, 1990). It has been theorized that repeated, acute elevations in testosterone associated with resistance exercise might affect the overall adaptation to resistance exercise (Kraemer & Ratamess, 2005). Dietary intake might also play an important role in the acute and chronic responses to resistance exercise. Carbohydrate intake after resistance exercise appears to have positive effects on postexercise protein kinetics (Borsheim et al., 2004; Roy, Tarnopolsky, MacDougall, Fowles, & Yarasheski, 1997; Smilios et al.). In previous research, however, carbohydrate ingestion has been shown to be associated with decreases in testosterone concentrations (Chandler et al.; Kraemer et al., 1998). It is unclear whether it was the carbohydrate ingestion itself that served to trigger decreases in testosterone concentrations or whether it is an interaction between resistance exercise and carbohydrate ingestion (Chandler et al.; Kraemer et al., 1998).

One study indicated increased protein synthesis when participants ingested 1 g/kg of carbohydrate after resistance exercise (Roy et al., 1997). This was corroborated in a later study by Roy, Fowles, Hill, and Tarnopolsky (2000), who found that carbohydrate supplementation increased protein synthesis and decreased protein degradation after resistance exercise. Carbohydrate supplementation also increases insulin concentrations, which is thought to enhance amino acid transport into the muscle cell and thus contribute to increased protein synthesis or decreased protein degradation (Ahtiainen, Pakarinen, Kraemer, & Hakkinen, 2003; Roy et al., 1997). These improvements in net protein balance with carbohydrate supplementation suggest that carbohydrate supplementation might augment the recovery process after resistance exercise and thus help optimize adaptations to resistance training. The available evidence regarding carbohydrate supplementation and resistance exercise suggests that there might be some benefits to carbohydrate supplementation in terms of protein synthesis.

As previously mentioned, carbohydrate ingestion might also influence serum testosterone concentrations (Ebeling, Stenman, Seppala, & Koivisto, 1995; Habito & Ball, 2001; Hjalmarsen, Aasebo, Aadvaag, & Jorde, 1996; Meikle, Stringham, Woodward, & Memurray, 1990; Pasquali, Macor, & Vicennati, 1997). The relationship between macronutrient ingestion and acute testosterone changes has been investigated more extensively at rest than in exercise situations. There is evidence that ingesting fat might decrease serum testosterone concentrations, whereas ingesting protein and carbohydrate without fat does not significantly affect testosterone concentrations (Meikle et al.). Conversely, Habito and Ball found that even low-fat meals that contained protein and carbohydrate acutely decreased testosterone concentrations. There is also evidence that carbohydrate ingestion alone might decrease serum testosterone concentrations in older men (Hjalmarsen et al.). It is difficult to interpret the existing research because of differing macronutrient feeding protocols and participant characteristics (Ebeling et al.; Habito & Ball; Hjalmarsen et al.; Meikle et al.; Pasquali et al.). Overall, the relationship between carbohydrate ingestion and testosterone concentrations at rest is not completely understood.

There are few data regarding the relationship between carbohydrate ingestion and testosterone concentrations after resistance exercise. Kraemer et al. (1998) observed an acute elevation in testosterone concentrations with resistance exercise.

They also demonstrated that testosterone concentrations dropped below baseline with carbohydrate/protein supplementation taken 2 hr before and immediately after exercise (Kraemer et al., 1998). This testosterone depression did not happen with placebo ingestion after resistance exercise (Kraemer et al., 1998). Chandler et al. (1994) found similar patterns of testosterone changes with ingestion of carbohydrate after resistance exercise. They found that testosterone concentrations dropped below preexercise levels 30 min after postexercise carbohydrate ingestion (Chandler et al.). This suggests that carbohydrate or mixed-macronutrient feeding might indeed result in decreased testosterone concentrations when done immediately after resistance exercise, although further research is needed to validate these findings. Both Kraemer et al. (1998) and Chandler et al. noted that testosterone concentrations were lowest when insulin concentrations were highest. This suggests an inverse relationship between insulin and testosterone concentrations with feeding after resistance exercise. Although insulin release can be triggered with ingestion of carbohydrate or protein or a combination of the two, amino acid availability with protein ingestion might further contribute other mechanisms of protein synthesis (Borsheim et al., 2004; Chandler et al.; Roy et al., 2000, 1997). It has been theorized that an increase in protein synthesis or decrease in protein breakdown might result in an up-regulation of androgen receptor content, resulting in a decrease in circulating testosterone concentrations (Kraemer et al., 2006). This provides a theoretical link between insulin concentrations after exercise and circulating testosterone concentrations (Kraemer et al., 2006). To study the relationship between insulin and testosterone, carbohydrate-only supplementation was necessary to prevent any confounding or redundant effects of protein intake. It is not known whether carbohydrate ingestion has the same effect on testosterone in both resting and postexercise conditions because no research has been performed with both resting and exercise conditions in the same group of participants. Thus, the effects of carbohydrate ingestion on both insulin and testosterone concentrations need to be better understood before hormonal or cellular mechanisms can be targeted.

Based on the available research, we hypothesized that carbohydrate ingestion would decrease testosterone concentrations both at rest and after resistance exercise. The primary purpose of this investigation was to examine whether carbohydrate ingestion resulted in changes in testosterone concentrations both at rest and after resistance exercise. Another important purpose was to illuminate the relationship between testosterone and insulin in resting and resistance-exercise conditions.

Methods

Study Design

Participants underwent one orientation session and one familiarization session followed by four testing sessions. The familiarization session and all exercise sessions were separated by 7–14 days. The participants were tested in random order in each of the following four test conditions: rest with placebo (RPL), rest with carbohydrate supplementation (RCHO), exercise with placebo (EPL), and exercise with carbohydrate supplementation (ECHO). Both the researchers and the participants were blinded to the supplement condition. At each session five blood draws were taken: before exercise (Pre), immediately after exercise or an equivalent rest

period (Post), and 15 (15P), 30 (30P), and 60 (60P) min after supplement ingestion (Figure 1). The participants ingested a carbohydrate supplement or placebo beverage immediately after the second blood draw and then rested quietly for 60 min until the final blood draw.

Participants

This study involved 9 recreationally weight-trained (minimum 1 year) men age 18–49 who served as participants ($M \pm SD$; age 26.2 ± 8.7 yr, height 174.5 ± 6.8 cm, body mass 84.6 ± 15.2 kg, 15.1% ± 5.35% body fat, 167.5 ± 35.4 kg 1-repetition-maximum squat, 1.98 ± 0.3 one-repetition maximum [1RM]/body weight). Eight of the nine participants were between the ages of 18 and 26, with 1 at age 49. The 1 older participant did not differ significantly from the group mean of the other 8 in any hormonal or performance variable and thus was included in the data analysis. All participants reported not having previously taken anabolic steroids, growth hormone, or related anabolic drugs. We used these criteria to eliminate variable hormonal responses because of muscle-building-drug supplementation, training status, or gender. All participants were informed of study procedures and signed consent forms before initiation of the study. The institutional review board at Appalachian State University approved the study.

Orientation, Dietary Analysis, and Body Composition

At the orientation session participants were given instructions for the study, and measurements of height, body mass, and body composition were taken. Participants were also tested for their 1RM in the Smith machine squat. Participants were provided instruction by a registered dietitian on the procedures for keeping food diaries for 3 days before each exercise session. Participants were instructed to maintain their normal dietary habits. There was no attempt made to manipulate diet because chronic dietary intake does not appear to affect the acute testosterone or cortisol responses to resistance exercise (Volek, Kraemer, Bush, Incledon, & Boetes, 1997). Food records were kept to help explain any potential differences in baseline hormone levels. There were no differences in total kilocalorie intake or macronutrient distribution between conditions (results not reported). Participants were instructed not to participate in strenuous exercise for 48 hr before exercise testing, not to eat for 12 hr before exercise testing, and not to ingest caffeine or alcohol for 24 hr before exercise testing. Body composition was determined using dual-energy X-ray absorptiometry (DEXA; Hologic QDR, Bedford, MA, USA), with the same technician performing all the scans. A total-body scan was performed for each participant. DEXA calibrations were performed daily and before each scan with a calibration block from the manufacturer. In a separate familiarization session participants were tested for their 10-repetition maximum (10 RM) in the Smith machine squat.

Strength Testing

Participants were first tested for their 1RM in the Smith machine squat during the orientation session using the protocol described by McBride, Triplett-McBride,

Blood Draws
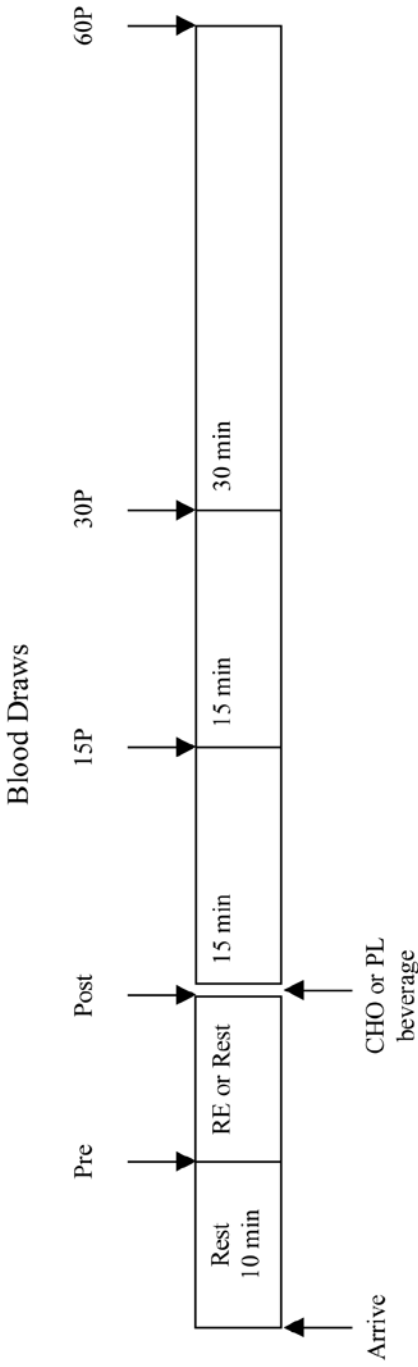
Figure 1 — Timeline of events at each testing session. Pre = preexercise or prerest period; Post = postexercise or postrest period; 15P = 15 min after beverage ingestion; 30P = 30 min after beverage ingestion; 60P = 60 min after beverage ingestion; RE = resistance exercise; CHO = carbohydrate; PL = placebo.

Davie, and Newton (2002). They were given a number of warm-up trials using 30% (8–10 repetitions), 50% (4–6 repetitions), 70% (2–4 repetitions), and 90% (1 repetition) of an estimated 1RM from the participant's recommendation. The resistance was then increased to elicit a 1RM within three or four maximal efforts. Adequate rest was allowed between all trials (3–5 min). Squats were performed to a 90° knee angle determined with a goniometer. A piece of tape visible to the participant was put on the Smith machine column such that the bottom edge of the bracket attached to the bar was even with the tape when proper depth was reached. All testing sessions were performed using the squat depth previously described and were supervised by a certified strength and conditioning specialist. During the separate familiarization session, 10RM was determined by starting with ~80% of 1RM and increasing or decreasing resistance as needed to determine 10RM. The participants also performed four sets of Smith machine squats with 90 s of rest between to establish the resistance adjustments that would be necessary to maintain sets of ~10 repetitions during the testing sessions.

Testing Sessions

Participants reported to the neuromuscular laboratory in the morning after having fasted for the previous 12 hr. This helped ensure that no acute dietary variables would affect hormone concentrations. All testing sessions for each participant were conducted at the same time of day to minimize the influence of diurnal variation on hormone concentrations. On reporting to the laboratory in the morning for each exercise session, participants handed in their food diary and were weighed. They sat quietly for 10 min before undergoing an initial 10-ml blood draw. All blood draws were taken from the antecubital forearm vein with participants in a seated position. Both right and left arms were sampled to maintain viability of blood draws. After the initial blood draw, participants either began the resistance-training protocol or rested for 10 min, which is approximately how long it took to complete the protocol. In the exercise conditions, participants performed four sets of the Smith machine squats to volitional failure. The rest period between all sets and exercises was 90 s and was closely monitored. This resistance-training protocol was selected because it has previously been shown to acutely increase serum testosterone concentrations immediately after exercise (Kraemer et al., 1998). The resistance for each set was adjusted to maintain ~10 repetitions per set. The resistance and number of repetitions were recorded for each set at every exercise session to track the total work performed in each of the resistance-exercise conditions.

Participants did not consume anything at any time during the exercise phase or equivalent rest phase of any of the four testing sessions. Immediately after the exercise protocol, or at the end of the equivalent rest period, a second blood sample was taken. Immediately after the second blood draw, the participants ingested a carbohydrate beverage or placebo beverage. The carbohydrate beverage was a 24% carbohydrate solution made with Gatorade Thirst Quencher (Gatorade, Chicago, IL, USA) with added maltodextrin (Carbo Gain, Now Foods, Bloomingdale, IL, USA) that provided 1.5 g/kg of carbohydrate. A 24% carbohydrate solution at a dose of 1.5 g/kg has previously been associated with a decrease in postexercise testosterone levels (Chandler et al., 1994). The Gatorade Thirst Quencher was mixed to the standard concentration of 6% carbohydrate, and then maltodextrin

was added to increase the carbohydrate content to 24%. The placebo beverage (Gatorade, Chicago, IL, USA) was an equal volume of fluid flavored with an artificial sweetener to match the taste of the carbohydrate beverage with identical electrolyte composition. The participants then rested quietly for 60 min after drink ingestion. During that period blood samples were taken 15, 30, and 60 min after drink ingestion (Figure 1). A 60-min rest period was chosen because changes in serum testosterone levels have been evident in previous research within or at 60 min after exercise or supplementation (Chandler et al.; Kraemer et al., 1998).

Blood Collection and Analysis

Blood was collected with a 20-gauge needle and a Vacutainer blood-collection system. The blood was allowed to clot and then centrifuged at $3,000 \times g$ for 15 min. The serum was then removed and stored in microcentrifuge tubes frozen at –80 °C until subsequent analysis. The serum was analyzed for total testosterone, cortisol, and insulin in duplicate via commercial assay kits (DRG International Inc., Mountainside, NJ, USA). Total testosterone was analyzed using DRG kit EIA-1559 with an intra-assay variation of 2.9%. Insulin was analyzed using DRG kit EIA-2935 with an intra-assay variation of 3.8%. Cortisol was analyzed using DRG kit EIA-1887 with an intra-assay variation of 2.1%. In the current investigation, each participant's samples were always analyzed within one kit to remove the interassay variability. Serum was also analyzed for glucose and lactate using a YSI 2300 STAT glucose and lactate analyzer (YSI Incorporated, Yellow Springs, OH, USA). Hormonal data were not corrected for plasma volume shifts because hormone concentrations represent exposure at the tissue level. Hydration status was not quantified, although it appeared to have a negligible effect on biochemical variables because there were not differences in baseline hormone levels between any of the conditions.

Statistical Analysis

Hormone responses were analyzed using multivariate analysis of variance with repeated measures. When significance was found, follow-up paired t tests were performed with a Holm's sequential Bonferroni procedure to adjust significance levels. Pearson's correlation coefficients were determined for biochemical variables. The significance level for all statistical tests was set at $p \leq .05$. All statistical analyses were performed using a statistical software package (SPSS, Version 12.0, SPSS Inc., Chicago, IL, USA).

Results

Testing-Session Characteristics

The mean resistance, percent of 1RM, and mean repetitions performed for each set are outlined in Table 1. Although the mean resistance did decrease for each set, it was not significantly different from the first set until the fourth set ($p < .01$). This was to ensure that the desired number of repetitions was maintained. Some

Table 1   Testing-Session Characteristics, *M* ± *SD*

|	Mass (kg)	% one-repetition maximum	Repetitions
Exercise			
Set 1	130 ± 28	78 ± 5	10.1 ± 1.5
Set 2	121 ± 28	72 ± 5	9.7 ± 1.0
Set 3	109 ± 28	65 ± 5	9.7 ± 1.0
Set 4	97 ± 26	58 ± 6*	8.7 ± 3.6
Placebo			
Set 1	127 ± 23	76 ± 6	10.0 ± 1.3
Set 2	118 ± 26	70 ± 4	9.9 ± 0.3
Set 3	109 ± 28	65 ± 6	10.1 ± 1.1
Set 4	99 ± 31	58 ± 7*	10.0 ± 0.0

*Significantly less than Set 1.

participants reported nausea and feeling ill, but all completed the protocol. There were no significant differences in any of the performance variables between the two conditions.

Total Testosterone

There were no significant differences in baseline (Pre) total testosterone concentrations between any of the four conditions. In the RPL and RCHO conditions, total testosterone concentrations did not change significantly from Pre levels in either condition (see Figure 2a). There were also no significant differences in total testosterone between RPL and RCHO at any time point. In the EPL and ECHO conditions, total testosterone was significantly elevated ($p = .007$ and $p < .001$, respectively) from Pre to Post in both conditions (see Figure 2b). With EPL, total testosterone concentrations returned to baseline by 15P and remained there for the remainder of the postexercise period. With ECHO, total testosterone concentrations dropped significantly ($p = .003$) below baseline at 60P (see Figure 2b). Despite these different patterns of change after exercise, there were no significant treatment differences in total testosterone concentrations at any time point during the EPL and ECHO conditions.
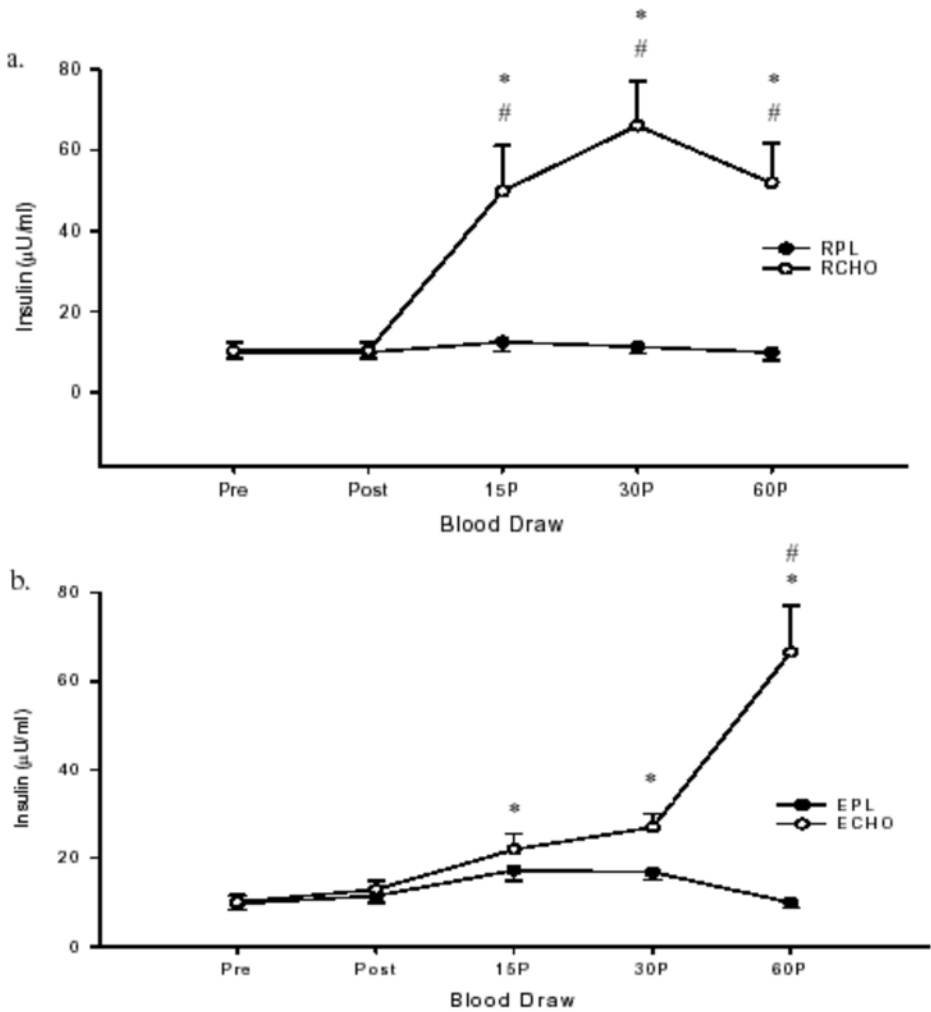
Insulin

There were no significant differences in baseline (Pre) insulin concentrations in or among any of the four conditions. There was a significant main effect ($p = .001$) of carbohydrate supplementation on insulin concentrations, with the carbohydrate conditions (RCHO and ECHO) having higher overall insulin concentrations than the placebo conditions (RPL and EPL). Insulin concentrations increased significantly ($p < .01$) above RCHO Pre concentrations at 15P ($p = .004$), 30P ($p = .001$), and 60P ($p = .002$; see Figure 3a). At the 15P, 30P, and 60P time points insulin concentrations were significantly higher ($p = .005$, $p = .001$, and $p = .002$, respectively) in the RCHO condition than in the RPL condition. With RPL, insulin concentrations
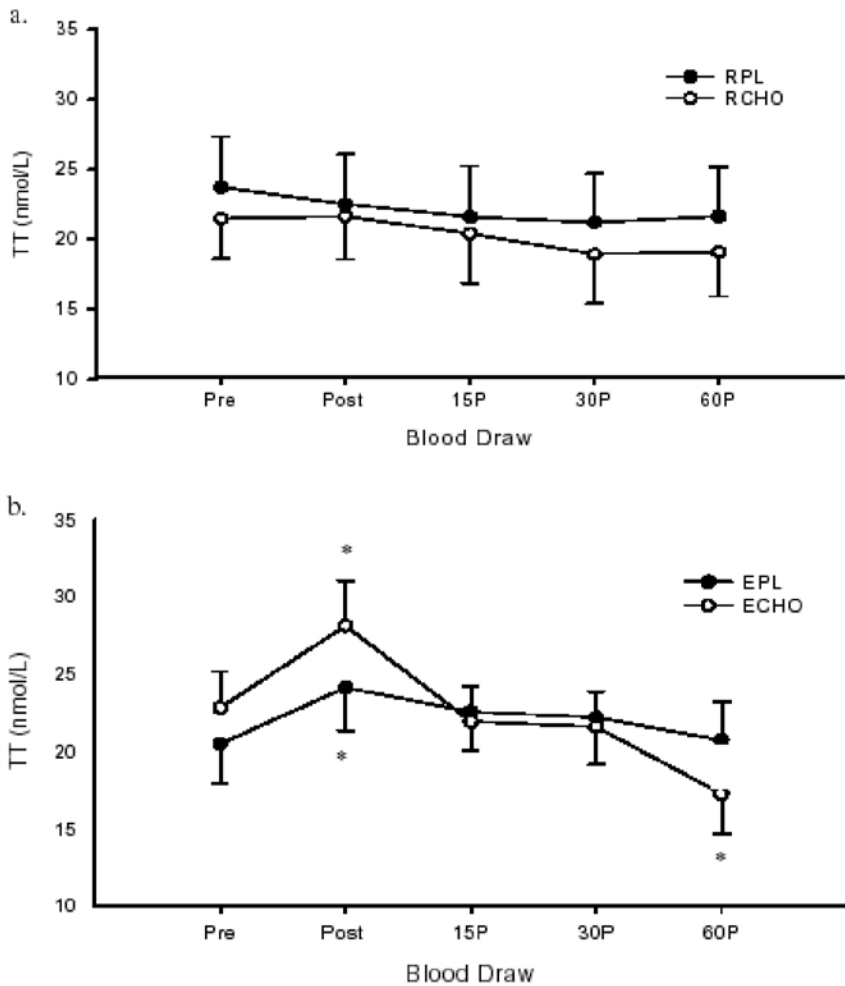
Figure 2 — Serum total testosterone (TT) (a) at rest and (b) with resistance exercise, $M \pm SE$. RPL = rest with placebo; RCHO = rest with carbohydrate; EPL = exercise with placebo; ECHO = exercise with carbohydrate. *Significantly different from corresponding Pre value.

did not change significantly. With EPL, insulin concentrations were significantly higher at 15P ($p = .002$) and 30P ($p = .001$) than preexercise baseline (see Figure 3b). With ECHO, insulin concentrations also increased above baseline at 15P ($p = .015$), 30P ($p = .001$), and 60P ($p = .001$). Insulin concentrations in ECHO were also significantly higher ($p = .001$) at 60P than in EPL. Although insulin concentrations were elevated in both RCHO and ECHO, concentrations appeared to increase more rapidly in RCHO.
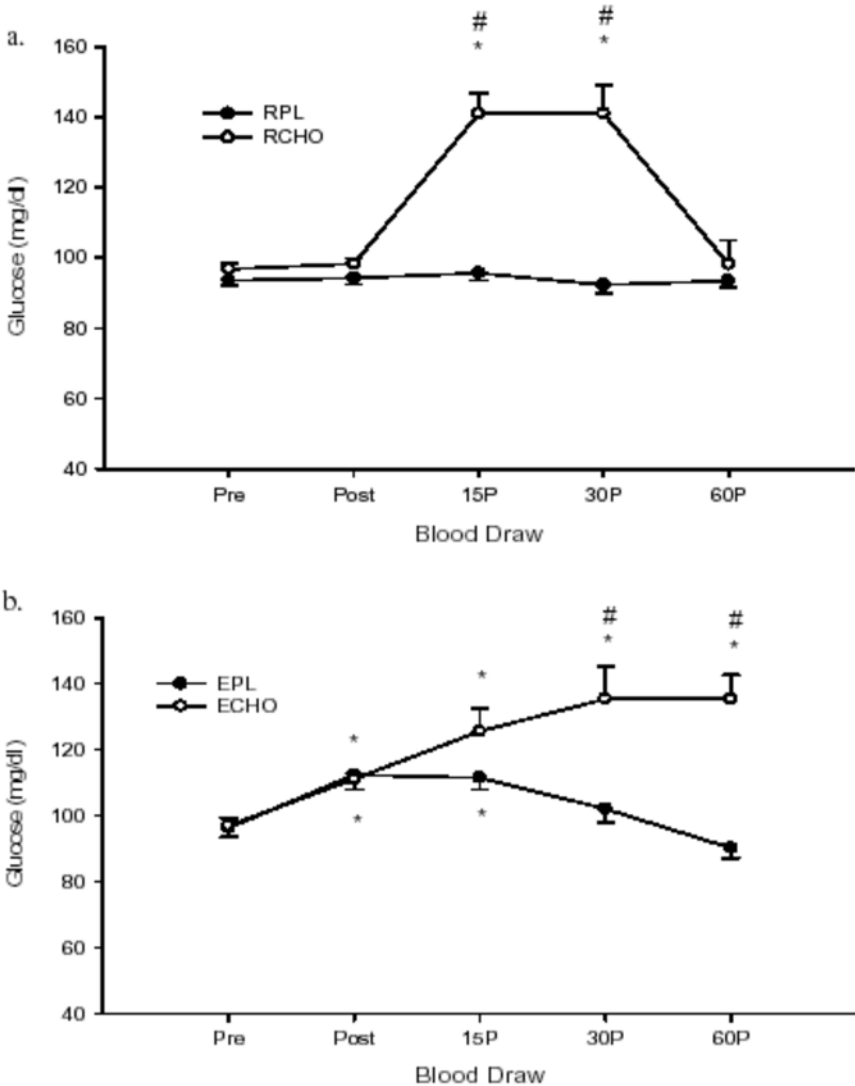
Figure 3 — Serum insulin (a) at rest and (b) with resistance exercise, *M ± SE*. RPL = rest with placebo; RCHO = rest with carbohydrate; EPL = exercise with placebo; ECHO = exercise with carbohydrate. *Significantly greater than corresponding Pre value. #Significantly greater than corresponding RPL or EPL or value.

Glucose

There were no significant differences in baseline (Pre) glucose among any of the four conditions. There were significant main effects for exercise ($p = .033$), time ($p = .027$), CHO ($p < .001$), and CHO × Time ($p = .043$) interactions with serum glucose concentrations. Serum glucose concentrations also increased significantly with carbohydrate intake both at rest and after exercise. In the RCHO, glucose

concentrations were significantly increased ($p \leq .05$) at 15P ($p < .001$) and 30P ($p = .002$) but had returned to baseline by 60P (see Figure 4a). Conversely, RPL did not result in any change in glucose concentrations. At the postexercise time point, there was a significant ($p = .001$) increase in glucose in both the EPL and ECHO conditions (see Figure 4b). With EPL, glucose remained significantly elevated ($p =$

Figure 4 — Serum glucose (a) at rest and (b) with resistance exercise, $M \pm SE$. RPL = rest with placebo; RCHO = rest with carbohydrate; EPL = exercise with placebo; ECHO = exercise with carbohydrate. *Significantly greater than corresponding Pre value. #Significantly greater than corresponding RPL or EPL value.

.001) at 15P but had returned to baseline at 30P. With ECHO, glucose concentrations continued to rise until 30P and remained significantly elevated ($p = .002$) at 60P.

Cortisol

There were no significant differences in baseline (Pre) cortisol concentrations among any of the four conditions. Cortisol concentrations showed a nonsignificant downward trend over time in the RPL and RCHO conditions (see Figure 5a). In
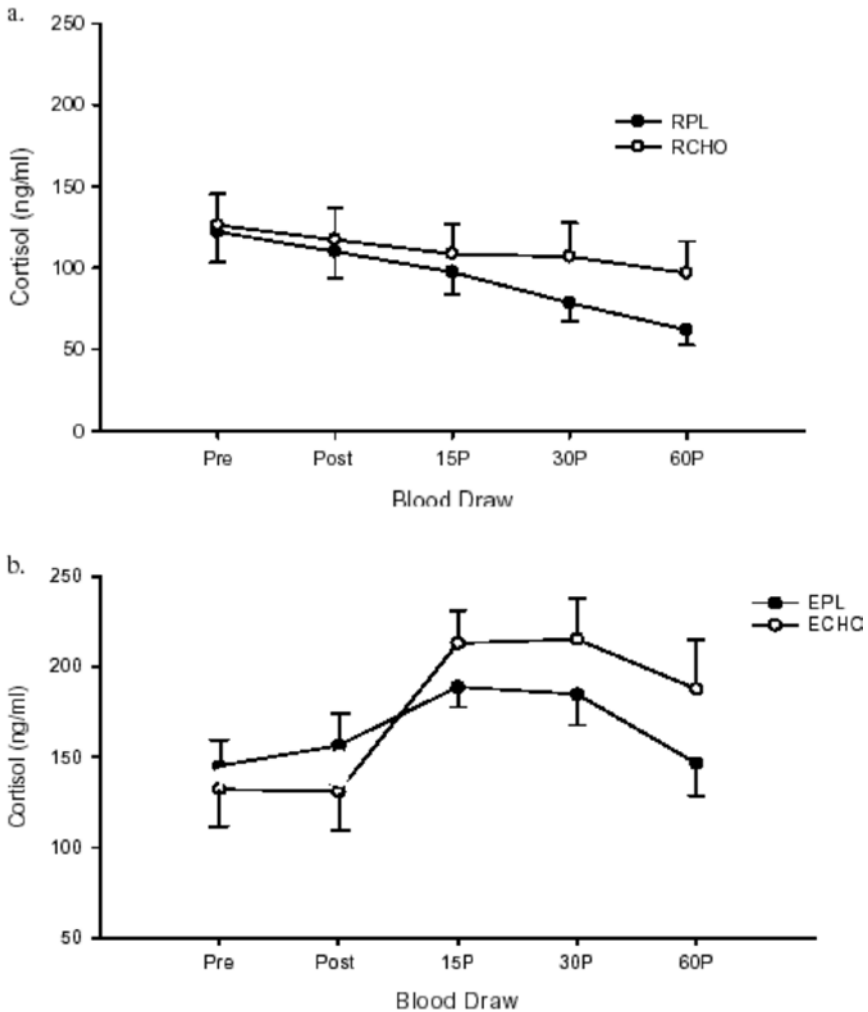

Figure 5 — Serum cortisol (a) at rest and (b) with resistance exercise, $M \pm SE$. RPL = rest with placebo; RCHO = rest with carbohydrate; EPL = exercise with placebo; ECHO = exercise with carbohydrate. No significant changes or differences in cortisol levels.

EP and ECHO conditions, cortisol tended to rise after exercise similarly at the 15P time point with both conditions, but these increases were not significant ($p = .63$; see Figure 5b). Despite these differences in the overall trends, there were no significant differences in cortisol concentrations between any of the four conditions at any time point resulting from large variability in cortisol levels and the cortisol response to exercise.
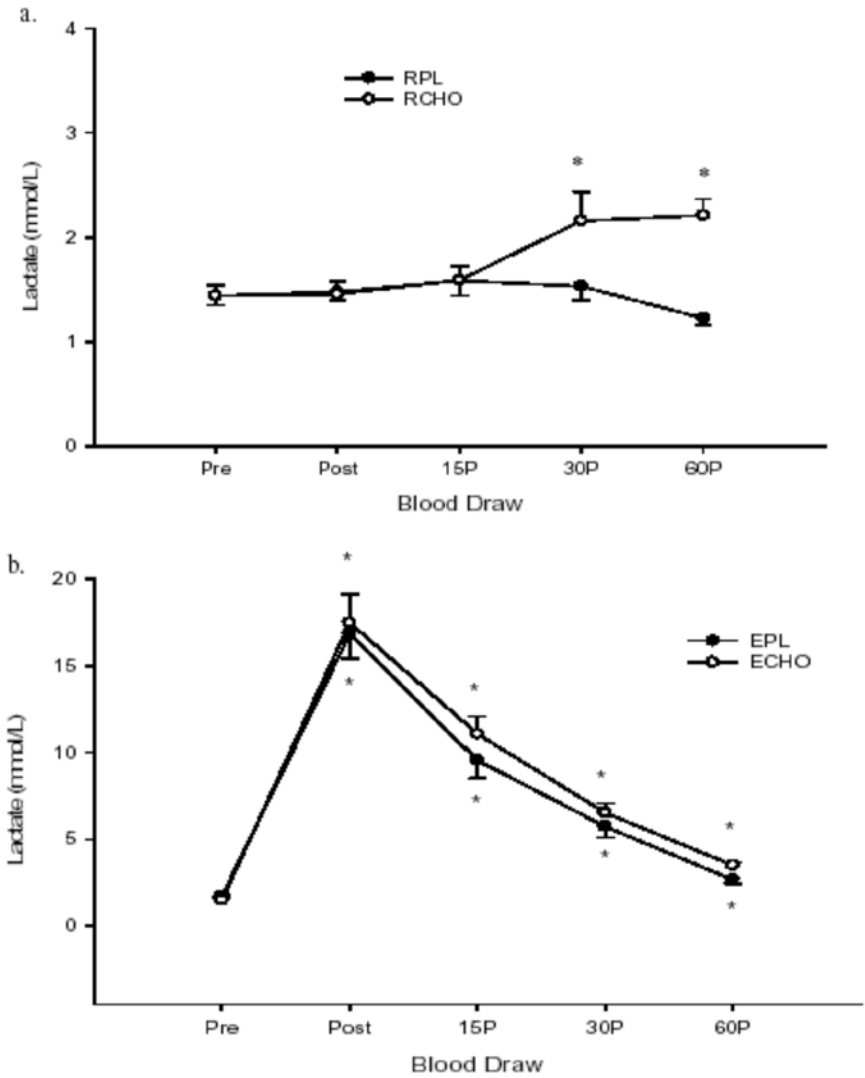
Lactate

There were no significant differences in baseline (Pre) serum lactate concentrations among any of the four conditions. Carbohydrate supplementation caused a significant increase in lactate concentrations at rest but not after exercise. In the RCHO condition, lactate concentrations were significantly ($p \leq .01$) above baseline at 30P and 60P but remained at baseline concentrations in the RPL condition (see Figure 6a). Carbohydrate ingestion did not appear to have the same effect after exercise. In both EPL and ECHO, lactate concentrations peaked immediately after exercise, followed by a decrease, although they remained significantly above baseline at 60 P (see Figure 6b). This pattern was the same with both EPL and ECHO conditions.

Correlations

There also were no significant correlations between any of the independent (exercise, rest, carbohydrate, placebo) and dependent (total testosterone, insulin, glucose, cortisol) variables. Although peak insulin concentrations occurred at the same time point as the lowest total testosterone reading in the ECHO condition, there were no significant correlations between insulin and total testosterone at any time points in any of the four conditions. Overall, total testosterone was not correlated with glucose, insulin, lactate, or cortisol.

Discussion

The primary finding of this investigation was that carbohydrate intake after resistance exercise resulted in decreased total testosterone concentrations (24% below preexercise baseline) 60 min into recovery (see Figure 2). Total testosterone will be referred to as testosterone throughout the discussion. Thus, there appeared to be some interaction between acute resistance exercise and carbohydrate supplementation that resulted in a depression of testosterone that was not as pronounced at rest in healthy young men. When comparing all time points, there was no significant correlation between insulin concentrations and testosterone concentrations, as had been theorized in some previous investigations (Chandler et al., 1994; Kraemer et al., 1998). Similar to much of the previous research regarding the hormonal responses to resistance exercise, EPL resulted in a significant elevation of testosterone of 18% and 23% in ECHO immediately after exercise (Ahtiainen, Pakarinen, Alen, et al., 2003; Ahtiainen, Pakarinen, Kraemer, & Hakkinen, 2003; Chandler et al.; Kraemer et al., 1999, 1990, 1998; Linnamo et al., 2005; Raastad et al., 2000; Smilios et al., 2003; Tremblay et al., 2004). After this acute elevation of testosterone, concentrations fell back to near baseline by 30 min post in both the ECHO and EPL conditions, which was also consistent with previous research in

Figure 6 — Serum lactate levels (a) at rest and (b) with resistance exercise, *M ± SE*. RPL = rest with placebo; RCHO = rest with carbohydrate; EPL = exercise with placebo; ECHO = exercise with carbohydrate. *Significantly greater than corresponding Pre value.

which testosterone concentrations typically returned to baseline within 30–45 min after exercise (Ahtiainen, Pakarinen, Alen, et al.; Ahtiainen, Pakarinen, Kraemer, & Hakkinen; Chandler et al.; Kraemer et al., 1999, 1990, 1998; Linnamo et al.; Raastad et al.; Smilios et al.; Tremblay et al.).

In the RPL condition, testosterone showed no significant changes over the course of the session, which would be expected given that the relatively short duration of the testing sessions would not allow for any significant diurnal variations in testosterone to take place. Carbohydrate supplementation did not appear to have an effect on testosterone at rest because there were also no significant changes in testosterone concentrations in the RCHO condition. These results are different than those of Hjalmarsen et al. (1996), who found that testosterone concentrations decreased with an oral glucose load. The most likely reason for these differences is that the participant populations are very different. Hjalmarsen et al. used older men (mean age 62 years) with chronic obstructive pulmonary disease, and the current investigation used younger, healthy men. Conversely, Meikle et al. (1990) found no difference in testosterone concentrations in young, healthy men after placebo or a low-fat protein/carbohydrate supplement (51 g protein, 146 g carbohydrate, 1 g fat) but found a significant decrease in testosterone concentrations after an isocaloric protein/carbohydrate supplement with higher fat content (17.9 g protein, 67.5 g carbohydrate, 50.4 g fat). The difficulty in interpreting these results is that both the fat and protein content differed between the two supplement conditions and thus differences in testosterone response could be a result of added fat or less protein. The results of the current research taken with the results from Meikle et al. suggest that fat is the primary mediator of acute changes in testosterone concentrations at rest because both carbohydrate and protein/carbohydrate combinations have yielded little acute impact on testosterone concentrations.

The testosterone results after the acute effect of exercise are similar to those of Kraemer et al. (1998) and Chandler et al. (1994), who found that supplementation immediately after resistance exercise resulted in testosterone concentrations that dropped below baseline 30–60 min after exercise. This effect appeared to be mediated by the ingestion of carbohydrate, protein, or a combination of the two. Kraemer et al. used a carbohydrate/protein supplement, and Chandler et al. tested protein, carbohydrate, and carbohydrate/protein supplements and reported similar findings. The unique finding in the current investigation was that these same trends were not seen in the same group of participants when they were given carbohydrate at rest. Again, this suggests some type of interaction between resistance exercise and carbohydrate that resulted in decreased testosterone concentrations in the exercise recovery period.

This leads to a question of why testosterone concentrations appear to be affected by carbohydrate ingestion after exercise but not at rest. Fluctuations in testosterone can be caused by changes in testosterone output, testosterone uptake, or shifts in plasma volume. Increases in testosterone concentration immediately after resistance exercise might be largely related to shifts in blood plasma volume (Ahmadizad & El-Sayed, 2005; Collins, Hill, Cureton, & DeMello, 1986; Kraemer, Kilgore, & Kraemer, 1993; Kraemer, Kilgore, Kraemer, & Castracane, 1992). One possibility is that the carbohydrate beverage was absorbed more quickly and increased plasma volume to a greater extent than the placebo beverage, resulting in a lower testosterone concentration at 60P. This seems unlikely because higher concentrations of carbohydrate do not facilitate gastric emptying or expansion of plasma volume, and the total volume and electrolyte content of the placebo and CHO beverages were identical (Murray, Bartoli, Eddy, & Horn, 1997). Despite this, differences in plasma volume between conditions cannot be ruled out. The

current investigation does not provide a definitive answer as to what the mechanisms are for decreases in testosterone concentrations with carbohydrate ingestion after resistance exercise. Based on previous investigations, it is difficult to tell whether decreases in testosterone are caused by decreased output.

One study showed that concentrations of luteinizing hormone (LH) did not change with supplementation after resistance exercise despite decreases in testosterone, and another investigation found a significant decrease in LH with feeding after resistance exercise (Chandler et al., 1994; Kraemer et al., 2006). It is also difficult to tell whether LH responded to testosterone concentrations or vice versa. Another possible scenario is that changes in testosterone with carbohydrate ingestion after resistance exercise were caused by increases in testosterone uptake. One potential explanation was an increase in testosterone uptake at the muscle level. One possibility was an interaction with glucose and/or insulin that caused an up-regulation in androgen-receptor expression or binding affinity, which could serve to decrease circulating testosterone concentrations. A recent study found that feeding after resistance exercise increased androgen-receptor content and decreased serum testosterone concentrations compared with a water-only condition (Kraemer et al., 2006). With feeding those researchers found that testosterone dropped below baseline starting at 20 min postexercise, whereas in the current investigation we did not see a depression of testosterone concentrations until 60 min postexercise (Kraemer et al., 2006). It should be noted that they used a mixed-fuel feeding containing carbohydrate, fat, and protein, whereas the current investigation used a carbohydrate-only feeding (Kraemer et al., 2006). This is one potential explanation for the differences in testosterone decrease between the two studies. Nonetheless, this does lend support to the idea that an up-regulation of androgen receptors takes place with feeding after resistance exercise and that this might be related to decreases in circulating testosterone concentrations (Kraemer et al., 2006). Given this evidence and the fact that carbohydrate ingestion has been shown to favorably affect protein kinetics after resistance exercise, it seems plausible that androgen-receptor expression could be favorably affected (Borsheim et al., 2004; Kraemer et al., 2006; Roy et al., 2000, 1997). It seems logical that insulin mediates the effects of carbohydrate feeding, whereas other mechanisms might be at work with protein or amino acid feeding (Borsheim et al.; Roy et al., 2000, 1997). What is not completely clear is exactly how manipulating total kilocalories or macronutrient content could affect these potential mechanisms. Despite what is known, the current investigation did not directly measure plasma volume, testosterone production, or testosterone uptake, so the mechanisms at work remain somewhat speculative.

In addition to testosterone, glucose and insulin responded to carbohydrate supplementation differently after resistance exercise than at rest (see Figures 3 and 4). Plasma glucose concentrations were significantly above baseline immediately after exercise in both the EPL and ECHO conditions. In the EPL condition, glucose stayed above baseline at 15P but then returned to baseline at 30P and 60P. With carbohydrate feeding, glucose concentrations continued to rise until 30P and remained elevated at 60P. Kraemer et al. (2006) found elevations in glucose persisting until 50 min after mixed feeding, with glucose returning to baseline at 60 min postfeeding. Chandler et al. (1994) showed significant elevations of glucose at 30 min postfeeding, but levels had returned to normal at 60 min. The pattern of glucose elevation in the ECHO condition also differed from the RCHO condition,

in which glucose levels returned to baseline by 60P (see Figure 3). It is difficult to explain this persistent elevation in glucose in the ECHO condition, especially because recent evidence suggests that resistance exercise improves glucose tolerance (Venables, Shaw, Jeukendrup, & Wagenmakers, 2007). It is possible that differences in plasma volume between the exercise and resting conditions explain some of the differences because resistance exercise can cause plasma volume shifts (Ahmadizad and El-Sayed, 2005; Kraemer et al., 1993, 1992). Based on the insulin data, it appeared that lower insulin levels in the ECHO condition at 15P and 30P than in the RCHO condition might have allowed glucose to remain in circulation longer with ECHO (see Figures 3 and 4). Insulin also responded differently to carbohydrate ingestion at rest compared with exercise, with a more rapid insulin response to carbohydrate feeding at rest (see Figure 3). Previous investigations have also found elevations in insulin 60 min after feeding after resistance exercise, although they had more pronounced elevations in insulin earlier in the postfeeding period (Bloomer, Sforzo, & Keller, 2000; Chandler et al.). It seems plausible this somewhat blunted insulin response was caused by the resistance training itself, because Bird, Hons, Tarpenning, and Marino (2006) saw insulin concentrations increase for 15–30 min after feeding during a 60-min resistance-exercise session. It is difficult to interpret what mechanisms are at work because the investigations in this area have used different resistance-exercise protocols and different feeding protocols in many cases (Bloomer et al.; Chandler et al.; Kraemer et al., 1998).

It is also interesting to note how insulin concentrations might be related to testosterone concentrations. Peak insulin concentrations occurred at the same time as the lowest testosterone reading (60P). Despite this, insulin and testosterone did not appear to have this same inverse relationship throughout the ECHO condition or any of the other conditions. The results in the resting conditions (RPL, RCHO) were in agreement with those of Ebeling et al. (1995), who found hyperinsulinemia did not result in any change in testosterone concentrations in young, sedentary men. These results suggest that insulin might only be related to testosterone concentrations in exercise recovery situations. Because there was no correlation between insulin and testosterone in either the RPL or RCHO condition, it appears that insulin does not markedly influence testosterone concentrations at rest in young, healthy men. During resistance exercise, insulin does not appear to directly mediate testosterone concentrations, because they were significantly elevated immediately after exercise with very little change in insulin concentrations, given that both would be affected by any plasma volume shifts (see Figures 2b and 3b). After exercise, though, insulin concentrations rose sharply from 30P to 60P and testosterone concentrations decreased during that same time period in the ECHO condition (Figures 2b and 3b). Although insulin concentrations did not appear to be related to testosterone concentrations overall, it is still possible that insulin is an indirect mediator of testosterone concentrations in specific circumstances, such as after resistance exercise.

There has been no research done on potential mechanisms of the interactions between insulin and testosterone. The available data from the current investigation and previous research seem to suggest an indirect link between insulin and testosterone because insulin clearly does not affect testosterone concentrations in all situations (Bloomer et al., 2000; Chandler et al., 1994; Ebeling et al., 1995). Again, a theoretical link between the two is androgen-receptor expression (Kraemer

et al., 2006). Regulation of testosterone appears to be mediated by a complex interaction of exercise state and feeding state that is not well understood. It should also be noted that these changes in blood testosterone concentrations and perhaps in androgen-receptor levels with feeding may or may not result in any physiologically significant enhancement of the overall adaptation to resistance exercise.

Another noteworthy finding was the lack of significant differential effects on cortisol concentrations with carbohydrate ingestion. The information regarding cortisol concentrations with supplementation and resistance exercise is also not entirely clear, although there is evidence that suggests that supplementation after resistance exercise might not significantly affect cortisol concentrations (Kraemer et al., 1998; Williams, Ismail, Sharma, & Jones, 2002). It is noteworthy that there was a significant exercise main effect, with cortisol concentrations being significantly higher overall in the exercise conditions. This was not unexpected because resistance exercise has been previously shown to acutely elevate cortisol concentrations (Ahtiainen, Pakarinen, Kraemer, & Hakkinen, 2003; Kraemer et al., 1999; Raastad et al., 2000; Smilios et al., 2003; Tremblay et al., 2004). There were no significant differences between time points in the ECHO and EPL conditions because of a highly variable cortisol response to exercise. In addition, because cortisol was not significantly different between the EPL and ECHO conditions, it did not appear to play a significant role in mediating testosterone concentrations after resistance exercise. Like cortisol, lactate did not appear to be related to testosterone concentrations based on the patterns of change. As would be expected, there was a main exercise effect, with higher overall lactate concentrations in the exercise conditions than the resting conditions. In the RCHO condition lactate concentrations were significantly above baseline at 30P and 60P, but lactate remained at baseline in the RP condition. This might have been the result of a shift in fuel utilization to glucose with carbohydrate ingestion, which in turn resulted in higher lactate concentrations (de Sousa, Simoes, Oshiiwa, Rogero, & Tirapegui, 2007). In the EPL and ECHO conditions, the lactate response to exercise and the decrease in lactate after exercise followed very similar patterns, with no apparent effect of carbohydrate ingestion.

The results of the current investigation overall demonstrated that carbohydrate intake after resistance exercise results in a decrease in testosterone concentrations by 60 min into recovery. Based on their patterns of change, cortisol and lactate did not appear to be related to this decreased testosterone concentration during the recovery period. Changes in serum glucose and insulin at rest did not result in any changes in testosterone. The relationship between glucose and insulin and testosterone after resistance exercise is not as clear, however. Other potential mechanisms that might influence testosterone concentrations after resistance exercise include testosterone production by the testes or adrenal cortex, hepatic or muscle blood flow and subsequent uptake of testosterone, or androgen-receptor binding, but these mechanisms can only be speculated on because they are beyond the scope of the current investigation.

In conclusion, depression of blood testosterone concentrations with feeding after resistance exercise has been demonstrated in multiple investigations including the current one (Chandler et al., 1994; Kraemer et al., 2006, 1998). Although it has been theorized that this testosterone depression after feeding with resistance exercise is related to androgen-receptor expression, insulin levels, or both, the

mechanisms at work are still uncertain (Ratamess et al., 2005; Kraemer et al., 2006). It is also uncertain precisely how each of three major macronutrients might differentially affect these mechanisms. It appears that glucose and insulin kinetics might be affected by resistance exercise followed by carbohydrate ingestion. Future research in this area should focus on further examining the mechanisms at work in the hormonal changes following feeding after resistance exercise.

Acknowledgments

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