

Dietary supplements affect the anabolic hormones after weight-training exercise

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Chandler, R. M., H. K. Byrne, J. G. Patterson, and J. L. Ivy. Dietary supplements affect the anabolic hormones after weight-training exercise. *J. Appl. Physiol.* 76(2): 839–845, 1994.—To examine the effect of carbohydrate and/or protein supplements on the hormonal state of the body after weight-training exercise, nine experienced male weight lifters were given water (Control) or an isocaloric carbohydrate (CHO; 1.5 g/kg body wt), protein (PRO; 1.38 g/kg body wt), or carbohydrate-protein (CHO/PRO; 1.06 g carbohydrate/kg body wt and 0.41 g protein/kg) supplement immediately and 2 h after a standardized weight-training workout. Venous blood samples were drawn before and immediately after exercise and during 8 h of recovery. Exercise induced elevations in lactate, glucose, testosterone, and growth hormone. CHO and CHO/PRO stimulated higher insulin concentrations than PRO and Control. CHO/PRO led to an increase in growth hormone 6 h postexercise that was greater than PRO and Control. Supplements had no effect on insulin-like growth factor I but caused a significant decline in testosterone. The decline in testosterone, however, was not associated with a decline in luteinizing hormone, suggesting an increased clearance of testosterone after supplementation. The results suggest that nutritive supplements after weight-training exercise can produce a hormonal environment during recovery that may be favorable to muscle growth by stimulating insulin and growth hormone elevations.

glucose; insulin; growth hormone; insulin-like growth factor I; testosterone; lactate; luteinizing hormone; carbohydrate; protein

THE MOLECULAR MECHANISM by which resistance exercise causes hypertrophy is not well understood. A potential stimulus for enhanced protein synthesis may be muscle contraction-activated amino acid uptake (2, 8). However, the hormonal milieu after exercise has a significant effect on the protein synthesis of contractile proteins. The hormones that can influence protein synthesis include insulin, growth hormone, testosterone, and insulin-like growth factor I (IGF-I).

During an acute bout of resistance exercise (weight-training exercise), growth hormone levels increase. Growth hormone may peak immediately postexercise or shortly thereafter (21, 22, 34). Similarly, testosterone levels (7, 21, 22, 35) have been reported to be elevated immediately after exercise; however, the effect of weight-training exercise on blood testosterone levels is not without controversy (5, 10, 20). IGF-I is most likely not affected by an acute bout of resistance exercise (20), although isolated random increases in IGF-I have been detected within 1 h after a weight-training workout (21, 22). In contrast to the responses of growth hormone and testosterone, insulin, one of the more potent anabolic hormones, does not appear to be affected by weight-training

exercise (10). However, the responses of insulin after weight-training exercise have not been sufficiently studied.

A carbohydrate load elicits a predictable increase in plasma insulin concentration within 30 min of ingestion. Protein consumption will elevate plasma insulin as well, although the increase is not comparable to that elicited by carbohydrate consumption (27). However, a carbohydrate and protein mixture has been shown to cause a significant insulin rise beyond that seen for carbohydrate alone (26, 28, 32, 39).

An increase in plasma insulin can create a cascade of stimulatory effects on the release of other anabolic hormones, such as growth hormone and IGF-I (9, 17, 31). In addition, insulin enhances the protein synthetic process and directly stimulates muscle amino acid uptake for protein synthesis and substrate uptake to support the process (16). During weight training, habitual tension overload of a muscle leads to increased protein synthesis and muscle hypertrophy. A possible mechanism of enhancing this process may be by increasing the plasma insulin concentration and other anabolic hormones after weight-training exercise. In fact, Haberson (11) determined that a high-caloric supplement in addition to resistance training will increase fat-free mass (FFM) over a control condition. Therefore the objective of this study was to determine whether a hormonal environment conducive to the enhancement of protein synthesis could be induced by carbohydrate, protein, or carbohydrate-protein supplements after weight-training exercise.

METHODS

Study 1

Subjects. Nine healthy drug-free male weight lifters with ≥ 2 yr of concurrent weight-training exercise were recruited to participate in this study. Questionnaires were used to determine health and drug-use profiles of the subjects. Their ages ranged from 21 to 35 yr with a mean age of 25.1 ± 1.5 (SE) yr. The mean weight of the subjects was 78.9 ± 2.9 kg and mean percent body fat was $11.8 \pm 1.2\%$ as determined by underwater weighing. Each subject was completely informed of the potential risks and possible benefits associated with participation in the study before he signed an informed consent document. The study was approved by the Institutional Review Board of the University of Texas.

Experimental design. There were four different treatments given on 4 separate days separated by ≥ 7 days. The four treatments consisted of three different dietary supplement treatments and a control treatment. The control treatment consisted of water only. The three nutritional supplements, provided by Shaklee US (San Francisco, CA), were comprised of carbohydrate (CHO; 55% dextrose, 41% maltodextrin, 4% vita-

mins, minerals, and artificial flavor), protein (PRO; 63% milk protein isolate, 27% whey protein isolate, 10% vitamins, minerals, artificial flavor, and artificial sweetener), or a mixture of carbohydrate and protein (CHO/PRO; 40% dextrose, 30% maltodextrin, 19% milk protein isolate, 8% whey protein isolate, 3% vitamins, minerals, and artificial flavor). For the CHO treatment each subject was administered 1.5 g/kg body wt of carbohydrate per supplement. The other two treatments, CHO/PRO and PRO, were given isocaloric to the CHO treatment. The CHO treatment amount of 1.5 g/kg body wt yielded 5.65 kcal/kg body wt. Therefore each subject received 1.06 g/kg carbohydrate and 0.41 g/kg protein for the CHO/PRO treatment and 1.38 g/kg protein for the PRO treatment. CHO, CHO/PRO, and PRO were made as 24, 31.5, and 9.6% solutions, respectively. However, because of the taste and texture of the supplements, subjects were allowed to ingest water ad libitum. On each of the experimental days the subjects performed a resistance weight-training workout. Immediately and 2 h after each workout, the subjects randomly received one of three experimental treatment beverages or water as a control treatment. Each subject received all treatments or a total of four experiments. After finishing the first supplement, subjects then showered. During the first 6 h of recovery the subjects consumed only water in addition to their supplement. Six hours postexercise the subjects consumed a standardized predominantly carbohydrate meal (~65% carbohydrate, 25% fat, and 10% protein). The experiment was concluded at 8 h postexercise. During the 8-h postexercise recovery period, the subjects rested quietly but were not allowed to sleep.

Experimental protocol. Subject percent body fat was determined by hydrostatic weighing (36). Residual volume was measured using the oxygen rebreathing technique (37).

One week before the experimental treatments, one repetition maximum (1 RM) values for each subject were determined for the eight exercises to be used in the experiment. In one session, the 1 RM was determined for each exercise in the order in which the exercises would be executed during the experimental trials. Performed in this order, these exercises included two-leg press, two-leg extension, two-leg curl, bench press, dumbbell flies, dumbbell rows, reverse close grip latissimus dorsi pull down (lat pull down), and overhead shoulder press. After several warm-up sets (number of warm-up sets was determined by the individual) of progressively decreasing repetitions and increasing intensity, subjects were given three attempts at 1 RM. The rest period between attempts was not limited as during the experimental trials.

After 1 RM testing, subjects performed two familiarization trials before the study began. Each familiarization trial was separated by ≥ 2 days. The familiarization trials were conducted to ensure that subjects could execute ≥ 8 repetitions but ≤ 10 repetitions in the second set for each exercise. The intensity needed for this criterion was ~75% of the 1 RM for each exercise (actual % of 1 RM for each exercise: 92.65 \pm 2.83% for two-leg press, 66.03 \pm 2.57% for two-leg extension, 78.10 \pm 1.75% for two-leg curl, 75.99 \pm 1.01% for bench press, 67.33 \pm 3.41% for dumbbell flies, 73.02 \pm 1.87% for dumbbell rows, 67.87 \pm 1.46% for lat pull down, and 69.57 \pm 1.18% for overhead shoulder press).

The subjects kept dietary records for the 3 days before the day of each experiment to note any unusual dietary consumption before testing. Subjects were asked to eat similar meals (similar in carbohydrate, protein, and fat content) before each trial. During these days subjects were asked to abstain from alcohol and caffeine consumption. In addition the subjects were asked to report to the laboratory after 36 h of rest and a 12-h fast. Diet analysis indicated that diets were consistent between treatments.

On the day of the experimental trials, the subjects reported

to the laboratory at 8:00 A.M. and were weighed in shorts and T-shirts. Immediately before the experimental trials, subjects performed warm-up sets for the two-leg press, bench press, lat pull down, and overhead shoulder press. During the experimental trials, subjects performed the above eight exercises that engaged large muscle masses. For each exercise, subjects performed 2 sets of 8–10 repetitions at ~75% (see above) of their 1 RM. Between sets and exercises the subjects were allowed 90 s of rest. Although an intensity of 75% of a 1 RM is classified as moderate to heavy, the experimental protocol workout was fatiguing and highly stressful for the subjects as a consequence of the limited rest intervals.

Blood collection and analyses. Approximately 5 ml of preexercise venous blood was drawn when the subjects reported to the laboratory. Immediately after the exercise bout, a postexercise sample was collected. After a 20-gauge \times 3.2-cm indwelling venous catheter (Baxter Healthcare, Deerfield, IL) was placed into an antecubital vein, blood samples were taken at 0.5, 1, 2, 3, 4, 5, 6, and 8 h postexercise. The earlier time points are typical observation points during a glucose tolerance test that have been used to assess insulin and glucose levels in other studies from our laboratory (14, 15, 29, 39). The extended time points were included to detect hormonal changes later in the recovery period [i.e., growth hormone changes in response to hypoglycemia (30)]. Hematocrits were determined in triplicate by microcentrifugation. Each blood sample was placed into 250 μ l of cold EDTA (anticoagulant) and 93 μ l aprotinin (protein stabilizer). Two hundred microliters of this whole blood were immediately added to 400 μ l perchloric acid (PCA) and centrifuged at 1,000 g for 15 min. The supernatant was removed and frozen at -20°C until later analyzed for lactic acid content according to the methods of Hohorst (12). The remaining blood was centrifuged at 1,000 g for 15 min. The plasma was removed, separated into aliquots, and frozen at -20°C for subsequent analysis. The plasma was assayed by radioimmunoassay for insulin with intra- and interassay coefficient of variability (CV) of 5.70 \pm 2.50 and 6.15 \pm 0.87%, respectively (Biomedicals, Costa Mesa, CA); testosterone with intra- and interassay CV of 6.5 \pm 1.38 and 7.5 \pm 0.95%, respectively (Biomedicals); human growth hormone with intra- and interassay CV of 6.68 \pm 1.91 and 8.85 \pm 1.48%, respectively (Biomedicals); and IGF-I with intra- and interassay CV of 2.70 \pm 0.30 and 6.80 \pm 1.60%, respectively (Nichols Institute Diagnostics, San Juan Capistrano, CA). IGF-I samples were first extracted using the acid-ethanol technique (Nichols Institute Diagnostics). When it was determined that plasma testosterone levels were affected by all nutritive supplements, human luteinizing hormone with intra- and interassay CV of 5.55 \pm 1.15 and 5.35 \pm 0.44%, respectively (Biomedicals), was assayed for CHO and Control treatments. Plasma glucose concentration was determined by a glucose analyzer (model 23A, Yellow Springs Instruments, Yellow Springs, OH). All samples were run in duplicate and corrected for postexercise hemoconcentration.

Study 2

Two hours after the meal during *study 1*, growth hormone and testosterone levels were lower than immediately before the meal. It was not certain whether these decreases were a consequence of the meal that was provided at 6 h postexercise or the unperturbed pattern of changes for these hormones. Therefore another study was undertaken to evaluate the effect of the meal on the hormonal response late in recovery. Six of the original nine subjects and one new subject volunteered to complete another exercise trial, which was identical to the CHO trial in *study 1* with the exception that the subjects did not receive a meal 6 h postexercise. The new subject underwent exercise evaluation as described in *study 1*.

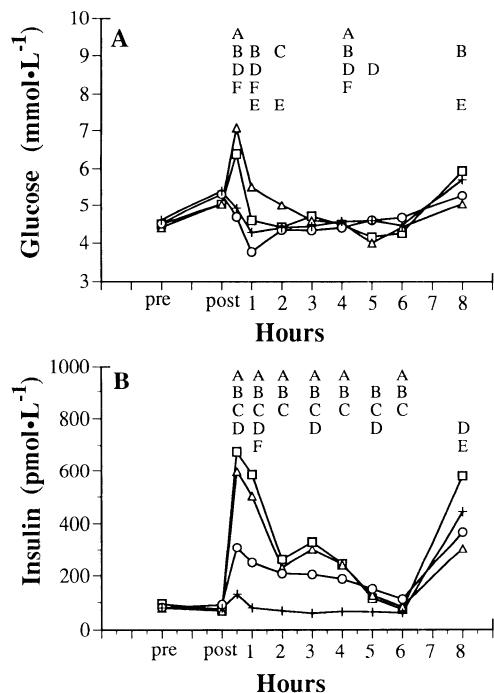


FIG. 1. Study 1. Plasma glucose (A) and insulin (B) concentrations for carbohydrate-protein (CHO/PRO; \square), CHO (Δ), PRO (\circ), and Control (+) treatments. Values are means \pm SE. Significant differences ($P \leq 0.05$): ^A CHO/PRO from Control; ^B CHO from Control; ^C PRO from Control; ^D CHO/PRO from PRO; ^E CHO/PRO from CHO; ^F CHO from PRO.

Statistical Analysis

Data from study 1 were analyzed using a two-way analysis of variance (treatment \times time) with repeated measures. With a significant F ratio, means were subjected to planned comparison analysis with every orthogonal combination between treatments to determine significant differences ($P \leq 0.05$). Sphericity was violated; therefore, the Huynh-Feldt ϵ correction was used during the F tests. Data from study 2 were analyzed using a one-way analysis of variance (time) with repeated measures with an α level of 0.05.

RESULTS

Study 1

Blood lactate concentrations were not different among the treatments before exercise (0.82 ± 0.06 , 0.81 ± 0.07 , 0.90 ± 0.08 , and 0.74 ± 0.08 mmol/l for CHO/PRO, CHO, PRO, and Control, respectively) and after exercise (11.68 ± 0.59 , 12.65 ± 0.61 , 12.61 ± 0.61 , and 11.93 ± 0.58 mmol/l for CHO/PRO, CHO, PRO, and Control, respectively) and during recovery (data not shown). This indicates that the exercise stress was equivalent among trials.

Plasma glucose levels pre- and immediately postexercise did not vary among treatments (Fig. 1). However, the effect of exercise was evident because the postexercise plasma glucose levels were elevated above preexercise concentrations. At 0.5 h after exercise and treatment ingestion, plasma glucose levels in the CHO/PRO and CHO treatments were significantly greater than those of the PRO and Control treatments. By 1 h postexercise, plasma glucose in the CHO treatment was significantly greater than in all other treatments. Plasma glucose con-

centration during the CHO/PRO treatment was greater than that during the PRO treatment but not greater than that during Control. Even though supplementation was given again after 2 h postexercise, there were no differences in plasma glucose concentrations among treatments at 3 or 4 h postexercise. At 5 h postexercise, the plasma glucose concentrations of the CHO/PRO and CHO treatments were significantly less than the PRO and Control treatment concentrations. Plasma glucose concentration during the CHO/PRO treatment was still significantly less than the PRO treatment glucose concentration after 6 h of recovery. Two hours after the meal (8 h postexercise), plasma glucose levels were elevated during all treatments, but the greatest response was seen during the CHO/PRO treatment and the least response during the CHO treatment.

The plasma insulin levels did not differ among treatments either before or immediately after exercise (Fig. 1). After the first supplement, plasma insulin concentrations were elevated above Control for all experimental treatments. CHO and CHO/PRO treatments produced significantly higher insulin concentrations than PRO. The second CHO and CHO/PRO supplements caused a second rise in insulin but not to the extent caused by the first supplement. An elevated plasma insulin concentration was not detected 1 h after the second PRO supplement. Insulin concentrations reached baseline during all treatments by 6 h postexercise. However, by 8 h postexercise and 2 h after the meal, insulin levels had risen significantly above preexercise concentrations in all treatments. At this time, the insulin level in the CHO/PRO treatment was significantly greater than levels of the PRO and Control treatments.

The acute resistance exercise protocol caused an elevation of plasma growth hormone immediately after exercise (Fig. 2). Thereafter, growth hormone quickly declined, reached baseline concentration by 2 h, and remained at that level for the next 3 h. At 6 h postexercise there was a significant rise in growth hormone during the CHO/PRO treatment above PRO and Control concentrations. At 8 h postexercise growth hormone levels had returned to baseline for all treatments.

Plasma testosterone levels before exercise were similar in all treatments (Fig. 2). Acute exercise significantly elevated testosterone levels in all treatments. Thirty minutes after supplementation, testosterone had decreased below baseline in the CHO, PRO, and CHO/PRO treatments and had returned to baseline in the Control treatment. At 1 h postexercise, testosterone in the CHO and PRO/CHO treatments was significantly lower than Control. The testosterone levels remained steady until 4 h postexercise except in the PRO treatment, in which the testosterone concentration continued to decline. During the time from 2 to 5 h postexercise, testosterone levels were significantly lower than Control in the CHO and PRO treatments. CHO/PRO testosterone levels were significantly lower than Control at 4 h postexercise. Testosterone began to increase by 5 h postexercise in the CHO and CHO/PRO treatments and by 6 h postexercise in the PRO treatment. There was no change in the Control treatment during these times. The testosterone concentration during CHO/PRO treatment was significantly

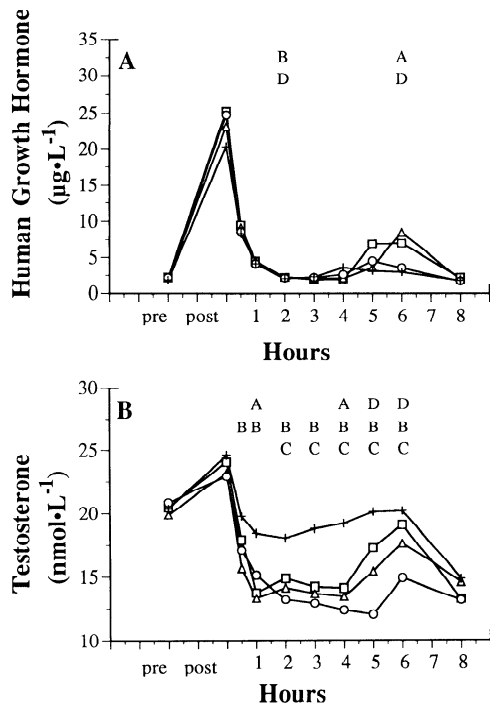


FIG. 2. Study 1. Plasma human growth hormone (A) and testosterone (B) concentrations for CHO/PRO (□), CHO (Δ), PRO (○), and Control (+) treatments. Values are means \pm SE. Significant differences ($P \leq 0.05$): ^A CHO/PRO from Control; ^B CHO from Control; ^C PRO from Control; ^D CHO/PRO from PRO.

greater than that observed during the PRO treatment but not different from Control or CHO. At 6 h postexercise, differences persisted between CHO/PRO and PRO and between CHO and Control. After the standardized meal, testosterone levels fell again to values similar to those seen after the supplements. No differences were noted among treatments.

Preexercise values of IGF-I were similar in all treatments (Table 1). Neither the supplementation nor the meal had a significant effect on IGF-I.

During recovery, the luteinizing hormone concentration oscillated (Table 1). No significant differences were detected between CHO and Control treatments.

Study 2

Exercise increased blood lactate concentration from 0.86 ± 0.11 mmol/l before exercise to 12.67 ± 0.81 mmol/l

after exercise. Most lactate had been cleared by 1 h postexercise (3.80 ± 0.52 mmol/l), yet a significant difference from the preexercise level was still noted by 4 h postexercise (1.30 ± 0.10 mmol/l). Lactate levels were not different from preexercise values at 5 h postexercise (1.22 ± 0.11 mmol/l).

The plasma glucose level rose significantly in response to exercise (Table 2). The glucose concentration was greatly increased after the first supplementation. The second supplement, given 2 h after exercise, did not elicit any notable response inasmuch as plasma glucose concentration continued to decline rapidly toward the preexercise concentration. By 4 h postexercise, glucose was not significantly different from the preexercise concentration. At 6 h postexercise, the glucose concentration was actually lower than the preexercise level. By 8 h postexercise, basal glycemia was observed.

Plasma insulin was significantly depressed after exercise (Table 2). The CHO supplement caused the plasma insulin level to rise, reaching a peak value at 1 h postexercise, after which insulin began to decrease. The second supplement increased the insulin concentration but, as in study 1, to a lesser degree than the first supplement. Insulin decreased steadily until 6 h postexercise, at which time the insulin concentration stabilized at baseline.

Exercise resulted in a significant increase in plasma growth hormone (Table 2). By 2 h postexercise, growth hormone had returned to preexercise concentration. Between 3 and 5 h postexercise, growth hormone remained lower than the preexercise level. At 6 h postexercise there was a significant elevation in growth hormone that returned to baseline by 8 h.

As in study 1, plasma testosterone levels increased after exercise (Table 2). After supplementation the testosterone level fell well below baseline. At 5 h the testosterone level began to increase, and by 6 h it had returned to the preexercise level where it remained until the end of the recovery period.

IGF-I was not affected by exercise or supplementation (Table 2) and did not change significantly throughout recovery.

DISCUSSION

Heavy-resistance exercise will lead to an increase in muscle cross-sectional area and to an increase in strength. Early strength gains for an untrained individ-

TABLE 1. Study 1. Luteinizing hormone and plasma insulin-like growth factor I concentrations

Time	Luteinizing Hormone, IU/l		Insulin-Like Growth Factor I, μ g/l			
	CHO	Control	CHO/PRO	CHO	PRO	Control
Preexercise	7.1 \pm 0.6	7.5 \pm 0.8	241.5 \pm 25.7	247.7 \pm 25.0	260.5 \pm 23.1	247.7 \pm 27.7
Postexercise	5.5 \pm 0.6	5.7 \pm 0.8	243.8 \pm 26.4	245.9 \pm 24.4	255.53 \pm 22.0	249.8 \pm 29.3
0.5	6.0 \pm 0.6	6.6 \pm 0.8	238.1 \pm 24.1	218.2 \pm 19.4	240.7 \pm 25.1	232.7 \pm 23.1
1	7.1 \pm 0.7	6.9 \pm 0.6				
2	7.4 \pm 1.0	6.7 \pm 0.5				
3	6.6 \pm 1.0	6.2 \pm 0.8	222.8 \pm 24.5	232.0 \pm 20.5	230.0 \pm 19.6	214.3 \pm 21.9
4	6.6 \pm 0.7	6.5 \pm 1.1				
5	8.5 \pm 0.6	7.2 \pm 0.8				
6	6.6 \pm 0.9	6.3 \pm 0.6	248.3 \pm 24.5	232.5 \pm 19.4	224.5 \pm 20.4	231.7 \pm 26.7
8	6.4 \pm 0.9	6.9 \pm 0.6	237.5 \pm 24.5	235.0 \pm 20.1	221.2 \pm 23.3	232.7 \pm 29.5

Values are means \pm SE. Time is preexercise, immediately postexercise, or no. of hours postexercise. CHO, carbohydrate; PRO, protein.

TABLE 2. Study 2. Hormone and metabolite concentrations

Time	Glucose, mmol/l	Insulin, pmol/l	Human Growth Hormone, $\mu\text{g/l}$	Testosterone, nmol/l	Insulin-Like Growth Factor I, $\mu\text{g/l}$
Preexercise	4.5 \pm 0.1	80.7 \pm 12.0	3.2 \pm 1.0	22.5 \pm 2.1	271.3 \pm 27.8
Postexercise	5.4 \pm 0.3*	62.3 \pm 12.5*	22.8 \pm 7.5*	25.3 \pm 1.7*	266.1 \pm 27.0
0.5	7.6 \pm 0.7*	426.1 \pm 80.9*	14.4 \pm 3.3*	20.5 \pm 1.7*	254.2 \pm 28.3
1	6.4 \pm 0.6*	491.1 \pm 97.4*	5.5 \pm 1.2*	15.6 \pm 1.7*	
2	5.4 \pm 0.2*	262.2 \pm 40.6*	2.6 \pm 0.4	14.6 \pm 2.1*	
3	4.8 \pm 0.2*	325.7 \pm 34.4*	1.9 \pm 0.2*	14.2 \pm 1.7*	261.4 \pm 27.3
4	4.2 \pm 0.3	215.6 \pm 32.3*	2.0 \pm 0.1*	15.3 \pm 1.4*	
5	4.1 \pm 0.3	156.5 \pm 30.6*	2.0 \pm 0.2*	17.0 \pm 1.4*	
6	3.4 \pm 0.1*	58.6 \pm 6.2	11.5 \pm 4.1*	21.0 \pm 1.4	280.3 \pm 41.4
8	4.3 \pm 0.1	58.1 \pm 7.8	4.0 \pm 0.8	20.1 \pm 1.7	246.2 \pm 28.3

Values are means \pm SE. Time is preexercise, immediately postexercise, or no. of hours postexercise. * Significantly different from preexercise values.

ual are a result of increased motor learning. However, after a few weeks of consistent training, strength gains become increasingly correlated to muscle cross-sectional area (25, 33). Gains in muscle mass are likely influenced not only by the volume and intensity of exercise during training but also by the hormonal environment of the trained muscles. Several anabolic hormones can affect maximal muscle growth. These include insulin, growth hormone, IGF-I, and testosterone. In particular, insulin may enhance muscular growth by stimulating amino acid uptake and net protein synthesis (16). Furthermore, insulin can also influence growth by affecting the secretion and potency of other hormones that can directly stimulate protein synthesis (17, 30). Therefore methods of supplementation after exercise that have been found to increase the plasma insulin concentration may be a sound means of enhancing exercise-induced muscle development without the introduction of exogenous natural and synthetic anabolic hormones.

In the present study, plasma glucose concentrations peaked immediately postexercise during the PRO and Control treatments and by 0.5 h after supplement ingestion during the CHO and CHO/PRO treatments. Even though the total amount of carbohydrate ingested was highest in the CHO treatment, the plasma glucose response for the CHO treatment was not significantly different from that of the CHO/PRO treatment. This was most likely because the percentage of carbohydrate in each of these supplements was equivalent and therefore their respective rates of carbohydrate gastric emptying were similar (13). At 0.5 h postexercise, the rise in insulin was associated with the rise in plasma glucose. The CHO and CHO/PRO supplements postexercise led to the greatest increase in insulin at 0.5 h after ingestion. The PRO treatment caused a lesser rise in insulin, which was above Control.

Immediately after exercise or muscle contraction, insulin-stimulated amino acid uptake is enhanced (1, 40). Chesley et al. (3) demonstrated that after resistance exercise muscle protein synthesis increased in the exercised muscle and that the increase was persistent for up to 24 h postexercise. However, after isolated muscle stimulation in rats, muscle net protein degradation transiently increases (1, 2). It has been shown that, in this state, the addition of insulin cannot prevent exercise-induced protein degradation but its ability to stimulate

protein synthesis is not compromised (1). Therefore insulin may counterbalance exercise-induced net protein degradation by increasing protein synthesis.

One important role of insulin after exercise is its effect on growth hormone during the recovery period. Insulin can stimulate the release of growth hormone by inducing hypoglycemia (30). Our results show that supplements that cause the greatest insulin spike after exercise lead to the greatest growth hormone levels 5–6 h postexercise. These results should be viewed with caution because the episodic secretion of growth hormone could not be adequately detected by our infrequent sampling of blood. However, it is unlikely that the spike in growth hormone observed 5–6 h postexercise was due to a normal growth hormone spike, because it occurred only during the CHO and CHO/PRO treatments and was common to all the subjects. Growth hormone stimulates an increase in amino acid transport and protein synthesis (18, 19). Crist et al. (6) found that heavy-resistance training and growth hormone injections caused significant increases in FFM and decreases in fat weight. Yarasheski et al. (38) found similar results in FFM, yet limb circumferences were not different from those of placebo controls. However, circumferences were not corrected for subcutaneous fat deposits. Furthermore, it is possible that the 12-wk duration was insufficient to separate the effects of growth hormone from placebo. Although not significant at the 0.01 α level, thigh girth measurements tended to be greater in the growth hormone-treated group.

Growth hormone and, to a lesser extent, insulin have been shown to stimulate the release of IGF-I from the liver (17); however, our results do not support such an effect. Our exercise and experimental protocol resulted in a large increase in insulin and growth hormone after supplements and exercise. These hormone responses, however, did not lead to significant increases in IGF-I. This is in agreement with results of other researchers (20). There is a 3- to 6-h lag between growth hormone administration and the release of IGF-I, and peak IGF-I does not occur for up to 16–28 h after growth hormone-stimulated release (4). It is possible that the changes in IGF-I were not manifested within the recovery time observed here.

The anabolic effects of testosterone, though controversial, are well documented (23). In agreement with other studies (7, 21, 22, 35), testosterone concentration

increased in response to weight-training exercise. Yet these results are also in contrast to other observations (5, 10, 20), although variations in exercise protocols and training status of the subjects are notable underlying mechanisms for these differences. After exercise in the present study, testosterone levels declined rapidly. Once testosterone attained its nadir, it remained below baseline until 6 h postexercise during the CHO, PRO, and CHO/PRO treatments. During the Control treatment testosterone levels returned to baseline shortly after exercise and stabilized throughout 6 h of recovery. After the standardized meal, increasing testosterone levels once again fell well below baseline in all treatments by the 8-h sampling. Decreases in testosterone after both the nutritive supplements and the meal were most likely caused by feeding. In support of this conclusion, the Control treatment had the least fall in testosterone levels after treatment administration and was significantly greater at its nadir than levels seen after the supplement treatments. This concept is further evidenced in *study 2* when no meal was given. Testosterone did not fall but remained stable from 6 to 8 h postexercise, as did insulin levels. An elevation in insulin concentration corresponded to a reduced testosterone level. It appeared that so long as the plasma insulin concentration remained above baseline, the testosterone concentration remained suppressed. There was no relationship, however, between insulin elevation and the degree of testosterone decrease. For example, testosterone levels were lowest during the PRO treatment, but the increase in insulin as a result of the PRO supplement was modest compared with the rise in plasma insulin during the CHO and CHO/PRO treatments.

The decrease in testosterone during the CHO/PRO, CHO, and PRO treatments could have been due to lower testicular secretion or an increase in testosterone clearance. To address this question, we measured the changes in plasma luteinizing hormone during the CHO trial. Luteinizing hormone is responsible for testosterone secretion from the testes. We reasoned that if the decline in plasma testosterone after the experimental supplements was due to a reduced rate of testosterone secretion, then this might require a reduction in plasma luteinizing hormone. However, luteinizing hormone was unaffected by the CHO supplement, thus providing indirect evidence that the decrease in plasma testosterone was due to an increased clearance rather than a reduced rate of secretion. Alternatively, the reduced plasma testosterone levels could have been a consequence of decreased testicular responsiveness to luteinizing hormone. If the decrease in testosterone was due to increased muscle uptake, it should lead to an increase in protein synthesis in the muscle via stimulation of mRNA synthesis. To our knowledge, only one other study has observed the acute effects of meals on plasma testosterone concentration (24). The authors determined that meals high in fat caused a significant reduction in testosterone and that an isocaloric diet of carbohydrate and protein mixture did not affect testosterone. This is in direct conflict with the results presented here, because the CHO/PRO supplement was associated with a large reduction in plasma testosterone. Yet these two studies cannot be directly

compared because there is the added stimulus of exercise in the present study. In a recent study by Kraemer et al. (20), subjects consumed a meal within 3 h after a bout of heavy-resistance exercise. Within 4 h after the meal, plasma testosterone concentration was below basal level, but the difference was not significant. The next blood sample was not measured until almost 3 h later, at which time the testosterone level was similar to the preexercise level. The relationship between supplements, exercise, and testosterone remains to be elucidated.

Of particular interest was the observation that the glucose and insulin responses were different than previously observed after aerobic exercise by trained cyclists (14, 15, 29, 39). In the study by Zawadzki and others (39), immediately and 2 h postexercise, subjects were given a supplement of carbohydrate or carbohydrate and protein mixture, both similar in carbohydrate content to that of the present study. Plasma glucose increased after the first supplement and remained elevated 3 h postexercise. After each supplement, insulin levels increased and remained above baseline values for 4 h. The lower insulin response and rapid glucose removal after the second CHO and CHO/PRO supplements in the present study are in direct contrast to this aerobic study. This observation seems to suggest that insulin action is greater after weight-training exercise than after sustained aerobic exercise. The effect of acute weight-training exercise on carbohydrate metabolism has not been studied in depth. Consequently, it is not known why there is a disparity between the two types of exercise in their responses to a carbohydrate challenge. Probable explanations are the differences between duration and intensity of exercise, the muscle tissue recruited for the exercise, and/or the hormonal environment during and/or after aerobic and weight-training exercise.

From these results it can be determined that nutritive supplementation can affect the anabolic hormonal milieu after exercise. Supplements that resulted in the greatest increases in insulin also caused the greatest rise in growth hormone 6 h postexercise. Supplements did not alter plasma IGF-I, but they did cause a decrease in testosterone concentration. Therefore we concluded that the CHO and CHO/PRO supplements stimulated an environment favorable for muscle growth throughout the body by increasing plasma concentrations of growth hormone and insulin during recovery.

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