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 ± 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-

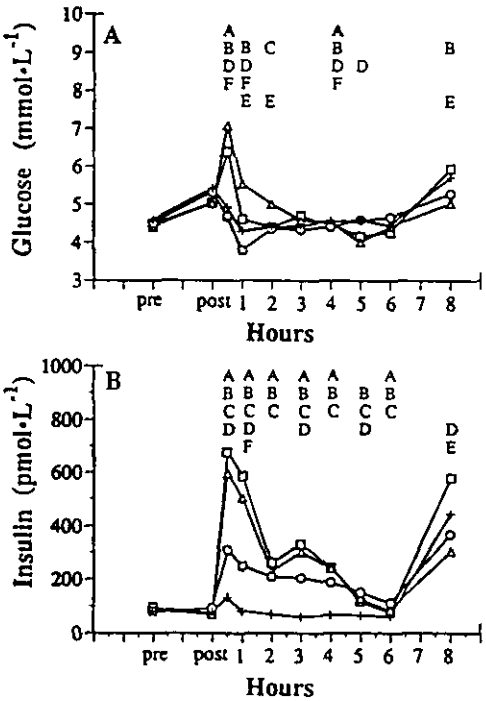

FIG. 1. *Study 1.* Plasma glucose (*A*) and insulin (*B*) concentrations for carbohydrate-protein (CHO/PRO; □), CHO (△), PRO (○), and Control (+) treatments. Values are means ± SE. Significant differences ($P \leq 0.05$): [A] CHO/PRO from Control; [B] CHO from Control; [C] PRO from Control; [D] CHO/PRO 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 × 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 concentration 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

TABLE 2. *Study 2. Hormone and metabolite concentrations*

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

Values are means ± 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

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