Growth hormone, IGF-I, and testosterone responses to resistive exercise

R. R. KRAEMER, J. L. KILGORE, G. R. KRAEMER, and V. DANIEL CASTRACANE

Exercise Physiology Laboratory, Department of Health, Physical Education, and Dance, Southeastern Louisiana University Hammond, LA 70402; Department of Kinesiology, Kansas State University, Manhattan, KS 66506; and Department of Obstetrics and Gynecology, Texas Tech University Health Sciences Center, Amarillo, TX 79106

ABSTRACT

KRAEMER, R. R., J. L. KILGORE, G. R. KRAEMER, and V. D. CASTRACANE. Growth hormone, IGF-I, and testosterone responses to resistive exercise. *Med. Sci. Sports Exerc.*, Vol. 24, No. 12, pp. 1346–1352, 1992. It has been suggested that growth hormone (GH), testosterone (T), and insulin-like growth factor I (IGF-I) play large roles in muscle tissue growth; however, in only two investigations IGF-I responses to resistive exercise have been examined. Eight young males who had not weight trained for a minimum of 5 months participated in the study. Three sets of bench press (BP), lat-pull (LP), leg extension (LE), and leg curl (LC) exercises were performed at a 10-RM load for 10 repetitions or until failure. Blood samples were collected from an IV catheter before exercise (−30 min and −10 min), after each individual exercise (BP, LP, LE, LC), and after the exercise session (+5, +15, +25, +35, +95 min; +5:35, +22:30, and +23:30 h). GH, IGF-I, and T determinations were corrected for plasma volume change. GH significantly increased ($P < 0.05$), but IGF-I did not change. Correction for plasma volume accounted for significant increases in T, but did not account for GH and IGF-I results. These data suggest that moderate resistive exercise may increase GH concentrations, whereas elevated T levels can be accounted for by exercise-induced alteration of plasma volume.

RESISTANCE EXERCISE, ANABOLIC, SOMATOTROPIN, SOMATOMEDIN-C, ANAEROBIC, HEMOCONCENTRATION

Resistive exercise is used extensively in the training and rehabilitation of athletes and is becoming increasingly recognized as an important exercise mode for the health-related exercise setting (2,22). Nevertheless, mechanisms that explain resistive exercise-induced skeletal muscle growth remain to be elucidated (14). Hypertrophy of skeletal muscle has been attributed, in part, to anabolic hormone/growth factor effects (8,14,19,23). Most of the investigations concerned with resistive exercise-induced endocrine changes have focused on testosterone (T), and fewer investigations have focused on growth hormone (GH) and insulin-like growth factor I (IGF-I). Testosterone is an anabolic and androgenic hormone that also is associated with bone growth, calcium retention, sodium reabsorption, and increased metabolic rate (11). Its role in protein formation and muscle development has long been recognized. GH has several major physiological roles including the stimulation of bone and cartilage growth, facilitation of protein deposition, and enhancement of fatty acid utilization with reduction in utilization of glucose and amino acids (11,19). Growth hormone promotes protein synthesis by facilitating the transport of amino acids across the cell membrane and affecting transcription in the nucleus, which results in increased amounts of RNA. IGF-I has been shown to have a strong anabolic effect upon muscle tissue (14) and is associated with regulatory feedback of GH (14,19). The effects of GH on skeletal muscle are thought to be mediated through the effects of IGF-I activity (13,14).

Previous T studies in males have shown increases from heavy loads or high volume resistive exercise (9,15) and no change following light loads and low volume (10). Although there are only a few studies in which GH responses to resistive exercise have been examined, a significant GH response has been observed from a protocol using a low volume (28,000 J) of resistive exercise and large muscle mass (21) and larger GH responses have occurred from a higher volume (49,000 and 59,000 J) of resistive exercise and large muscle mass (16). IGF-I responses to resistive exercise

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have been examined in only two previous studies by the same investigator (15,16). Among the few existing GH and IGF-I studies, differences in resistive exercise protocols have included equipment (free weights, fixed machine), muscle groups, exercise load, and duration of rest period between sets. Thus, the present study was designed to determine the effects of a low volume of resistive exercise using fixed machines and small muscle mass on GH, IGF-I, and T concentrations.

MATERIALS AND METHODS

Eight healthy males volunteered to participate in the study as experimental subjects. The subjects had the following descriptive characteristics: mean (±SE) age 26.9 ± 2.7 yr; height, 180.0 ± 2.9 cm; weight, 76.1 ± 3.6 kg; body fat, 14.9 ± 2.8%; fat-free mass, 64.4 ± 3.0 kg; $\dot{V}O_{2max}$, 53.9 ± 4.2 ml·kg^{-1}·min^{-1}; HR_{max}, 192.9 ± 2.6 b·min^{-1}. All of the subjects, with the exception of one, exercised on a regular basis and had experience with resistive exercise from a recreational standpoint; however, none of the subjects was a body builder or a competitive lifter, and no subject had performed any regular resistive exercise for at least 5 months. A medical history on each subject revealed that none of the subjects was a smoker or was taking any medication including anabolic steroids or other hormonal treatments. All subjects completed three testing sessions that were performed in accordance with policy statement of the American College of Sports Medicine (1).

Session 1. In the first session a one-repetition maximum (1-RM) for four exercises was determined (3). Bench press (BP) and seated lat-pulls (LP) were performed on a pulley-system weight training machine (Universal Gym, Inc., Cedar Rapids, IA); leg extension (LE) and leg curl (LC) were performed on a cam-system weight training machine (Nautilus Sports/Medical Industries; Deland, FL). These exercises were chosen because they represented two upper body and two lower body resistive exercises commonly used in weight training programs. Careful attention was paid to body position during each lift and joint angles were kept uniform for each lift among subjects.

The 1-RM assessment was followed by measurement of body composition determined using hydrostatic weighing with residual volume measured from an O_2 dilution technique (24). Treadmill maximal oxygen uptake ($\dot{V}O_{2max}$) was assessed with an automated $\dot{V}O_2$ measurement system during a Bruce Protocol treadmill test (20) to exhaustion.

Session 2. In session 2 a 10-RM was determined for the four exercises. The 10-RM resistance was used for each repetition completed in session 3. The 10-RM exercise load corresponded to approximately 75% of 1-RM for all four exercises (Table 1). Session 2 was separated from session 1 by at least 48 h. To keep work uniform for each repetition, exercise was performed to a metronome at a cadence of 15 repetitions·min^{-1}.

TABLE 1. Maximal strength, exercise load, and percentage of 1-repetition maximum.

Exercise	Strength, 1-RM (kg)	Exercise Load (kg)	% 1-RM
Bench press	70.73 ± 5.79	54.40 ± 4.89	76.48 ± 2.00
Lat pull	76.13 ± 4.08	56.82 ± 2.03	75.08 ± 1.91
Leg extension	104.35 ± 6.03	75.0 ± 6.48	71.13 ± 0.35
Leg curl	49.43 ± 3.26	38.64 ± 3.19	77.69 ± 2.91

Values are means ± SE; $N = 8$.

Session 3. Within 7 d of the second session the subjects reported to the lab after an 8-h fast for session 3. Subjects were instructed not to exercise 24 h prior to session 3. At 0800 h an IV catheter (Travenol, 20 g, 3.2 cm) was inserted into a forearm vein and kept patent with a heparin lock (1 ml sodium heparin, 10 U·ml^{-1}). Resting blood samples were taken in a sitting position at 0900 h, 30 min before the exercise session began (−30), and 0930 h, 10 min before the start of the exercise session (−10). The subjects then walked from the Exercise Physiology Lab to an air-conditioned weight room (50 yards), technicians readied themselves, and the exercise session began. Exercises were all performed at a 10-RM load in the same order: BP, LP, LE, LC. Each set was continued until muscular failure. Muscular failure was defined as the point at which the subject was not able to complete a repetition beyond the prescribed set. A 10.0% drop in the range of motion constituted failure to complete a repetition. A meter stick on a stand was adjusted to the appropriate height and placed behind the weight stack prior to each exercise. A technician recorded the distance the stack moved and stopped the set when there was a 10% drop in the range of motion. The number of repetitions completed by the subjects is shown in Table 2. Total work completed was calculated as the product of the weight and the vertical distance that the weight was moved. Mean (±SD) total work was 21,653.5 ± 3917.2 J.

Two min of rest were taken between sets. Five min of rest were taken between individual exercises (BP, LP, LE, LC) during which time blood sampling was completed. Resistive exercise was performed to a metronome at a cadence of 15 repetitions·min^{-1}. Each exercise and subsequent blood draw required approximately

TABLE 2. Repetitions completed at a 10-RM load for bench press, lat pull, leg extension, and leg curl resistive exercises.

	Set 1	Set 2	Set 3
Bench press	9.50 ± 1.3	7.25 ± 2.2	4.30 ± 1.4
Lat pull	9.75 ± 0.4	7.50 ± 1.1	5.62 ± 1.3
Leg extension	9.50 ± 1.0	8.9 ± 2.0	7.9 ± 1.9
Leg curl	9.4 ± 0.7	8.5 ± 1.6	6.6 ± 1.5

Values are means ± SE; $N = 8$.

9.0 min for completion, depending upon number of repetitions completed per set. For each time period, 3-ml discard volumes were drawn, proceeded by EDTA and serum tube draws; following centrifugation, plasma and serum aliquots were frozen at −70° and −20°C, respectively. After each sample was taken the catheter was flushed with physiological saline, and heparin was replaced in the catheter. Blood samples were collected in a sitting position after each exercise and after the entire exercise session at min +5, +15, +25, +35, +95, and h +5:35, +22:30, and +23:30. A sequence of events for the exercise session and each exercise is shown is Table 3 and 4, respectively. After the blood draw at +95 min, the catheter was taped more securely to the subject's arm. The subject was instructed to go home and eat a light, noncaffeinated lunch, then return to the lab for a blood draw at +5:35 post-exercise. Following the blood draw at +5:35, the catheter was removed and the subjects were instructed not to exercise and to return to the lab the following morning after an 8-h fast. A resting blood sample was drawn via venipuncture at +22:30 and +23:30 h.

Blood analyses. Blood samples were analyzed for hematocrit, hemoglobin, lactate, GH, T, and IGF-I. Hematocrit and hemoglobin were analyzed with a Coulter counter (Coulter Electronics, S880). Lactate samples were treated with a neutral phosphate buffer with added NaCl (YSI 2357) and were analyzed with a lactate analyzer (Yellow Springs Instruments, 2300 GL). Hematocrit and hemoglobin were used to calculate plasma volume change using the method of Dill and Costill (7). GH, T, and IGF-I were determined by radioimmunoassay (RIA). Serum concentrations of GH were determined in duplicate using an ^{125}I liquid phase double antibody procedure (Diagnostic Products, Los Angeles, CA). Serum samples for T were determined in duplicate with a solid phase ^{125}I RIA (Diagnostic Products). Plasma samples were analyzed for IGF-I with a liquid phase ^{125}I second antibody RIA (Nichols Institute, San Juan Capistrano, CA) following an acid ethanol extraction procedure. IGF-I binding proteins may interfere somewhat with quantification of plasma IGF-I. A standard acid ethanol precipitation and subsequent centrifugation was employed to separate IGF-I from its binding proteins. The method, which was used as described by the manufacturer, yielded accurate quantification of IGF-I and obviated the use of column chromatography.

All of the samples from an individual were run in the same assay to avoid any changes in interassay variability. Intraassay coefficients of variation for GH, IGF-I, and T were 2.46, 2.41, and 2.97%, respectively. Interassay coefficients of variation for GH, IGF-I, and T were 8.92, 14.6, and 8.24%, respectively.

A one-way ANOVA with repeated measures was used to determine whether GH, T, IGF-I, and lactate changed over time and a Student's *t*-test was applied where appropriate. All comparisons were considered significant at an alpha level of $P < 0.05$. Correlation coefficients were used to determine whether relationships existed between GH and IGF-I at different time points.

TABLE 3. Sequence of testing events for each exercise.

Event	Time (min)	Task
1	0	Set 1 of resistive exercise ca. 10-RM
2	0.5	Rest for 2 min
3	2.5	Set 2 of resistive exercise
4	3.0	Rest for 2 min
5	5.0	Set 3 of resistive exercise
6	6.5	First 3 ml of blood discarded
7	9.0	Blood sample collected/catheter flushed with physiological saline
8	10.0	Heparin injected into infusion plug

TABLE 4. Sequence of testing events for session 3.

Time	Event
0800 h	IV catheter insertion
0900 h	First resting blood sample
0930 h	Second resting blood sample
0940–0950 h	Bench press, post-exercise blood sample
0950–1000 h	Lat pulls, post-exercise blood sample
1000–1010 h	Leg extension, post-exercise blood sample
1010–1020 h	Leg curls, post-exercise blood sample
1025 h	Blood sample (+5)
1040 h	Blood sample (+15)
1105 h	Blood sample (+25)
1115 h	Blood sample (+35)
1155 h	Blood sample (+95)
1200 h	Subjects leaves lab with catheter secured and eats a noncaffeinated, nonalcoholic lunch
1555 h	Blood sample after subject returns to lab; IV catheter removed (+5:35)
0845 h	Blood sample via venipuncture
0945 h	Blood sample via venipuncture

RESULTS

Exercise lactate concentrations were significantly higher than resting levels (Fig. 1). The peak lactate of 8.54 ± 1.68 mmol·l^{-1} occurred after LE. GH, IGF-I, and T were corrected for plasma volume change using hematocrit and hemoglobin concentrations (7). Time 0 hematocrit and hemoglobin concentrations were in the normal range and were used as a reference for plasma volume change at subsequent times. Plasma volume was unaltered from −30 to 0 min (0.5 ± 2.1%) but was reduced during and after exercise. Plasma volume for BP, LP, LE, LC, +5, and +15 was −8.73 ± 1.18, −12.05 ± 1.25, −11.04 ± 1.67, −8.91 ± 1.41, −8.12 ± 0.75, and −1.52 ± 1.41%, respectively.

GH levels were significantly higher than resting concentrations after LE and stayed elevated until 35 min after exercise (Fig. 2). IGF-I and T concentrations did not change significantly (Figs. 3 and 4, respectively).

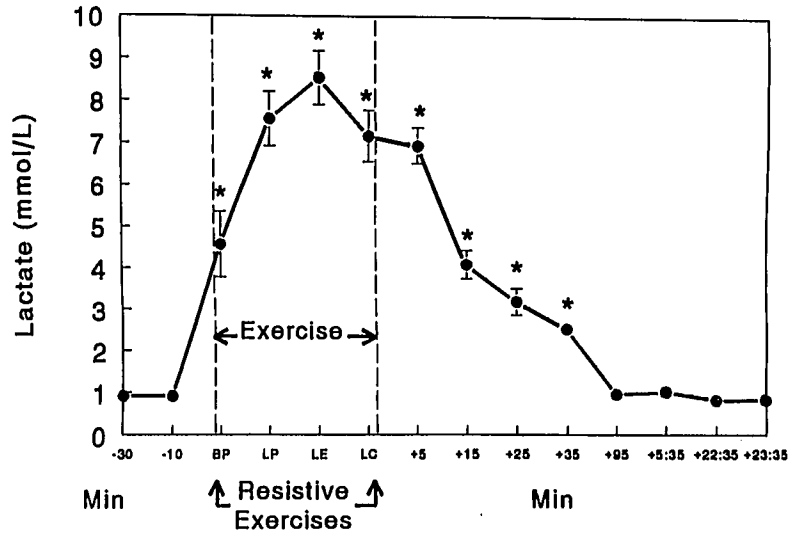

Figure 1—Alteration of lactate concentrations before, during and after resistive exercise. Data represent mean ± SEM; $N = 8$. * $P < 0.05$ compared with −10 value.

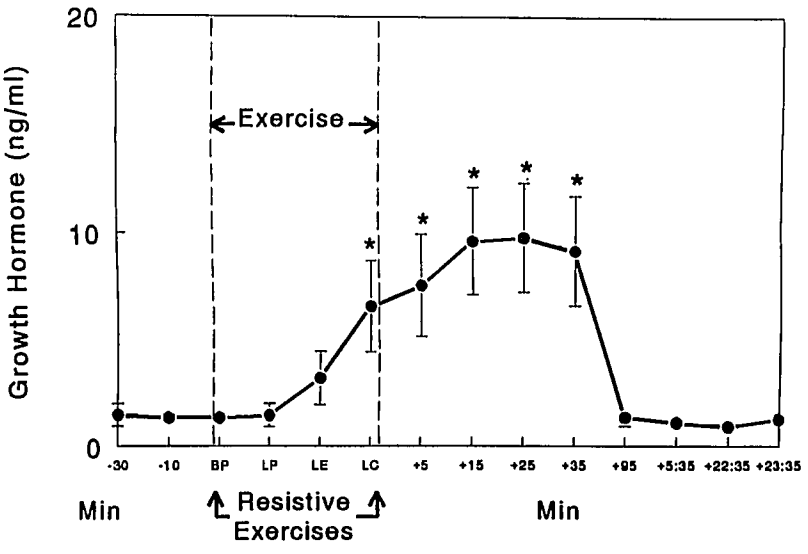

Figure 2—Alteration of growth hormone concentrations before, during and after resistive exercise that are corrected for plasma volume change. Data represent mean ± SEM; $N = 8$. * $P < 0.05$ compared with −10 value.

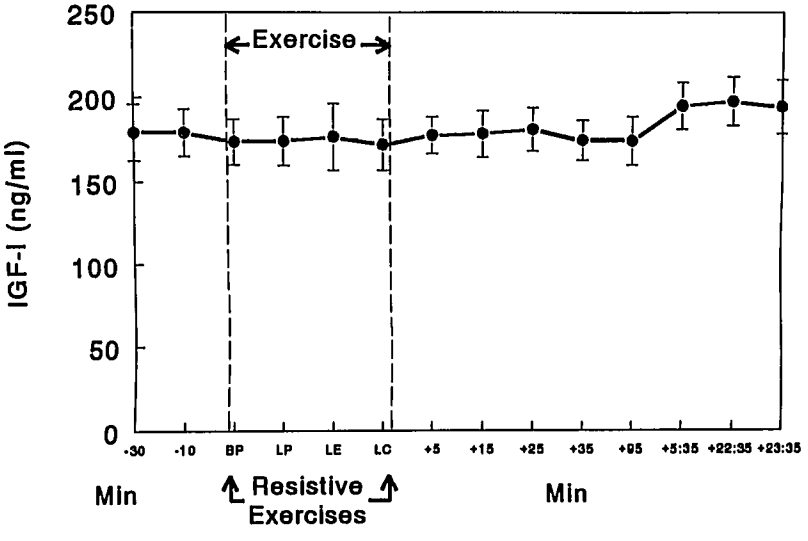

Figure 3—Alteration of IGF-1 concentrations before, during, and after resistive exercise that are corrected for plasma volume change. Data represent mean ± SEM; $N = 8$.

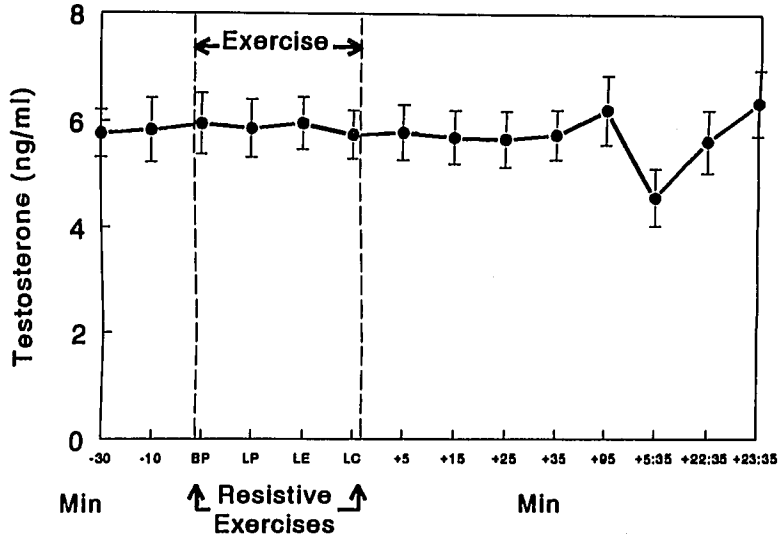

Figure 4—Alteration of testosterone concentrations before, during, and after resistive exercise that are corrected for plasma volume change. Data represent mean ± SEM; $N = 8$.

T levels that were *not* corrected for plasma volume change were significantly higher than resting levels at two time points. After LP, T concentrations uncorrected for plasma volume change were 23.15 ± 2.22 $nmol \cdot l^{-1}$ compared with 20.31 ± 1.87 $nmol \cdot l^{-1}$ for corrected values; uncorrected T concentrations after LE were 23.26 ± 2.15 $nmol \cdot l^{-1}$ compared with 20.66 ± 1.70 $nmol \cdot l^{-1}$ for corrected values. GH, IGF-I, and lactate concentrations were significantly different at the same time points regardless of whether they were corrected for plasma volume change. Additionally, correlation coefficients between GH at its peak (+25) and all subsequent IGF-I time points did not indicate a relationship existed between GH release and IGF-I concentration.

DISCUSSION

The objective of the study was to determine the effects of a low volume of resistive exercise using fixed machines and small muscle mass on GH, IGF-I, and T concentrations. These endocrine parameters were corrected for plasma volume change. Corrected GH levels rose significantly in response to the exercise; however, corrected IGF-I and T concentrations were not observed to be elevated over a 23-h period following the resistive exercise.

Both an increase (9,15,16,23) and no increase (10) in T levels in response to resistive exercise have been documented. In the present study hemoconcentration was responsible for significantly elevated T concentrations (uncorrected for plasma volume change) after LP and LE. Peak plasma volume change in the present study was very similar to that reported by Collins et al. (5) using three circuits of four resistive exercises at 70% of 1-RM. It is probable that the lack of a corrected T response was due to: 1) lower exercise load, 2) lower exercise volume, and 3) correction for plasma volume shifts (14). The degree of T rise for uncorrected values after LP was consistent with changes in uncorrected T in previous research following a resistive exercise protocol of similar loads, repetitions, and rest periods (23). The extent to which T played an anabolic role in response to resistive exercise of moderate intensity in the present study, may have been partly dependent upon the degree of hemoconcentration produced by the exercise; an increase in concentration of T would increase the exposure of muscle cell receptors to T and thus might affect muscle tissue growth (16).

Vanhelder and coworkers (21) found vertical leg lifts to elicit an increase in GH concentrations from resistive exercise at 85% of a 7-RM, whereas 28% of a 7-RM did not result in a significant rise. Kraemer et al. (16) recently documented different degrees of GH response to resistive exercise sessions controlled for load and rest between exercise. The highest GH responses were produced with a 10-RM exercise and 1 min of rest between sets. The pronounced GH response in the present study was produced with a 10-RM load and 2-min rests between sets.

There exist only two previous investigations by the same primary investigator in which effects of resistive exercise on IGF-I have been examined (15,16). Total work of those resistive exercise protocols was approximately 2 to 3 times the total work performed in the present study. In both previous investigations, only random increases in IGF-I were observed within 1 h following resistive exercise protocols, and the increases did not follow any pattern of GH release. Additionally, some of the IGF-I increase documented in the more recent study (15) could be accounted for by small plasma volume changes.

It is thought that increases in IGF-I levels are mediated through GH (13). However, this phenomenon

was not observed at any of the post-exercise time points throughout the 23 h following exercise. Furthermore, correlation coefficients between GH at its peak (+25) and all subsequent IGF-I time points did not substantiate a GH/IGF-I relationship in response to the exercise protocol. It has been suggested that IGF-I is not stored in a readily releasable form since the earliest detectable rise occurs 3–6 h following infusion of human GH with peak IGF-I concentrations resulting 16–28 h following *im* administration of human GH (6,12). The resistive exercise protocol in the present study used a low volume and small muscle mass compared with protocols used in the previous studies. Perhaps elevated IGF-I concentrations between 3 and 28 h post-GH rise would have been elicited in response to a greater exercise load of more muscle mass and subsequently higher GH concentrations.

It is possible that GH responses produced an unobserved effect on IGF-I concentrations. Minuto et al. (17) have studied the diurnal fluctuations of IGF-I during a 24-h interval. There is a nocturnal decline in IGF-I, which then increases in the morning hours. This would be consistent with a delayed response to the well-documented nocturnal increases in GH (17). In the present study we did not have the opportunity to obtain sleep-related samples and therefore were not able to determine whether resistive exercise was associated with prevention of this nocturnal decline, but this would seem to be consistent with the temporal pattern of growth hormone stimulation and subsequent morning increase in IGF-I.

If IGF-I increases were to occur, several proposed mechanisms could facilitate an increase. The mechanisms include stimulation by GH, stimulation by gonadal steroidal hormones, stimulation through synergistic effects of non-GH hormones, augmentation of GH effects on IGF-I production (19), or exercise-induced disruption of cells (e.g., adipose, skeletal muscle) containing endogenous IGF-I. In the present study the resistive exercise protocol involved small muscle mass at low workloads, which may not have disrupted cellular components enough to cause endogenous release.

There remains the possibility that training influences could have affected hormonal responses (4,18). All subjects, with one exception, participated on a regular basis in different forms of aerobic exercise. Training-affected GH responses to running have been demonstrated to some extent; however, the direction of the effect appears to depend upon the kind of exercise protocol (i.e., intensity and duration) employed (4). Additionally, it has been proposed that training-induced hormonal responses may be specific to the type of exercise (4). All of the subjects in the present study were untrained with respect to resistive exercise.

In conclusion, a resistive exercise of low volume using fixed machines and small muscle mass will produce elevated GH concentrations; however, IGF-I increases do not accompany elevated GH levels. In addition, serum concentrations of T are elevated in response to the resistive exercise and therefore may exert an effect on tissue metabolism. However, T levels can be accounted for by hemoconcentration, which suggests that there is not a significant increase in testicular production. Whether higher GH responses than those demonstrated in the present study are associated with elevated IGF-I levels over a 28-h period remains to be elucidated.

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Address for J. L. Kilgore: Kansas State University, Dept. of Anatomy and Physiology, College of Veterinary Medicine, Manhattan, KS 66506. Address for G. R. Kraemer: Southeastern LA University, Exercise Physiology Laboratory, Dept. of HPED, P.O. Box 845 SLU, Hammond, LA 70402. Address for V. Daniel Castracane: Texas Tech University Health Sciences Center, Dept. of Obstetrics and Gynecology, 1400 Wallace Blvd., Amarillo, TX 79106.

Address for correspondence: R. R. Kraemer, Southeastern LA University, Exercise Physiology Laboratory, Dept. of HPED, P.O. Box 845 SLU, Hammond, LA 70402.

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