LH secretion and testosterone concentrations are blunted after resistance exercise in men

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1Intercollege Graduate Program in Physiology, 2General Clinical Research Center at Noll Laboratory, and Departments of 3Kinesiology and 4Animal Science, The Pennsylvania State University, University Park, Pennsylvania 16801; 5Military Performance Division, US Army Research Institute of Environmental Medicine, Natick, Massachusetts 01760; and 6The Human Performance Laboratory, Department of Kinesiology, University of Connecticut, Storrs, Connecticut 06269-1110

Received 8 February 2001; accepted in final form 6 April 2001

Nindl, Bradley C., William J. Kraemer, Daniel R. Deaver, Jana L. Peters, James O. Marx, Jeffrey T. Heckman, and Gregory A. Loomis. LH secretion and testosterone concentrations are blunted after resistance exercise in men. J Appl Physiol 91: 1251–1258, 2001.—This study examined the hypothesis that exercise-induced changes in circulating testosterone would be centrally mediated via hypothalamic-pituitary release of luteinizing hormone (LH). We tested this hypothesis by examining overnight LH, total and free testosterone (TT and FT), and cortisol (C) concentrations in 10 young healthy men (21 ± 1 yr) during two experimental sessions: a control and an acute heavy-resistance exercise bout (50 total sets consisting of squats, bench press, leg press, and latissimus dorsi pull-down). Exercise was performed from 1500 to 1700, and blood sampling began at 1700 and continued until 0600 the next morning. Blood was sampled every 10 min for LH and every hour for TT, FT, and C. Hormonal concentrations were determined via RIA, and the secretion characteristics of LH were analyzed with deconvolution analysis. When overnight postexercise concentrations were compared with control concentrations, no statistically significant (P ≤ 0.05) differences were observed for LH half-life, LH pulse frequency, interpulse interval, pulse amplitude, or pulse mass. Significant differences were observed for LH production rate (13.6 ± 4 and 17.9 ± 5 IU·l distribution volume−1·day−1 for exercise and control, respectively, a 24% reduction). For the ANOVA marginal main effect means due to condition, C was significantly elevated (5.9 ± 0.7 vs. 4.0 ± 0.4 μg/dl), while TT (464 ± 23 vs. 529 ± 32 ng/dl) and FT (15.6 ± 0.7 vs. 18.3 ± 0.9 pg/ml) were significantly decreased for the exercise condition. These data demonstrate that the decline in overnight testosterone concentrations after acute heavy-resistance exercise is accompanied by a blunted LH production rate and elevated C concentrations.

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Luteinizing hormone (LH) is secreted from the anterior pituitary gland in an ongoing pulsatile fashion and into the systemic circulation (31, 32). The primary target tissue for LH is the Leydig cell within the testes, from which testosterone is released. Testosterone exhibits potent somatotrophic, anabolic, and metabolic effects (9, 15, 22). Testosterone has also been shown to be an important biomarker influencing the trainability of neuromuscular performance and the regional distribution of body fat (3). Acute exercise has been shown to increase (3, 10, 12, 14–16) or decrease (5, 9) testosterone concentrations when sampled after exercise, dependent on the mode, intensity, and duration of the exercise bout. No prior studies have employed long-term (i.e., >6 h) blood sampling to ascertain the extent to which the exercise-induced change in testosterone persists, and the question arises whether exercise induces transient or more sustained alterations in circulating testosterone concentrations beyond those typically observed due to normal circadian changes. Most studies examining testosterone have also ignored its signaling peptide, LH. Because of the episodic LH glandular release, multiple time-point measurements are essential to fully characterize the trophic effect this hormone may have (23, 25, 28–32). Inasmuch as the impact of strenuous exercise on LH pulsatility has been primarily focused on women (17, 18, 33), there is a glaring lack of LH data in response to exercise for men. Additionally, few studies have made concomitant serial measures for both testosterone and cortisol (C). Quantification of C concentrations is important because of its antagonistic and potential inhibitory role to that of testosterone. To provide further information on the hypothalamic-pituitary-testicular axis in men, the purpose of this study was to examine overnight LH, total (TT) and free testosterone (FT), and C concentrations after acute heavy-resistance exercise. We hypothesized that any exercise-induced effect on overnight testos-
terone concentrations would be centrally mediated by changes in LH secretion.

METHODS

Experimental approach. Long-term blood sampling (i.e., 13 h) was employed to evaluate the effects of acute heavy-resistance exercise performed in the late afternoon on overnight circulating concentrations of LH, TT, FT, and C in young healthy men. Blood was sampled every 10 min for LH and every hour for TT, FT, and C. Deconvolution analysis (28–31) was used to estimate the secretory parameters of pulsatile LH release.

Subjects. Ten healthy men gave informed consent to participate in this investigation, which was approved by The Pennsylvania State University Human Use Institutional Review Board and the University Park General Clinical Research Center (GCRC) Scientific Review Committees. The physical characteristics of the subjects were as follows: age, 21 ± 1 yr; height, 177 ± 2 cm; weight, 79 ± 3 kg; percent body fat, 11 ± 1%; maximal O2 uptake (V\textsubscript{O2 max}), 51 ± 3 ml·kg\textsuperscript{-1}·min\textsuperscript{-1}; and met the following criteria: no caffeine, aspartame, or other medical problems (e.g., eating or sleeping disorders) that could confound the data from this investigation. Percent body fat and V\textsubscript{O2 max} were measured by methods previously described (11). Subjects who had taken supplements (e.g., androstenedione) during the preceding 3 mo were excluded. To utilize a homogenous subject population that could be fairly characterized as having above-average degrees of aerobic and strength fitness and thus be able to tolerate the heavy demands of the exercise protocol, the following inclusion criteria were used: <25 yr of age, <20% body fat, >45 ml·kg\textsuperscript{-1}·min\textsuperscript{-1} V\textsubscript{O2 max}, and >1.5 times body mass 1-repetition maximum (RM) squat strength. The strength 1 RM (mean ± SD) were 135 ± 12 kg for squat, 110 ± 8 kg for bench press, 196 ± 11 kg for leg press, and 84 ± 3 kg for latissimus dorsi (lat) pull-down.

Strength assessment. Universal (Omaha, NE) and York Barbell equipment (York, PA) was used according to previously described procedures (11) to test 1 RM for squat, bench press, leg press, and lat pull-down. Briefly, warm-up consisted of performing 5–10 repetitions at 40–60% perceived maximum, a 3- to 5-min rest and stretching period, and the completion of three to five repetitions at 60–80% maximum. Three to five subsequent lifts were then made to determine the 1 RM, with 5-min rests between lifts. An attempt was considered successful when completed through a full range of motion without deviation from proper technique and form. Spotters supervised each lift, and no injuries were observed.

Dietary control. All subjects completed 3-day dietary intake diaries (before each overnight trial). Subjects were asked to replicate, as much as possible, their first 3-day dietary intake for the second 3-day period on their overnight visits. Dietary analyses (Nutritionist IV, First DataBank, San Bruno, CA) of these records verified that the caloric content and composition were similar for the 3 days before each overnight stay. On the day of their overnight trials, the entire day’s meals were provided for the subjects. These meals were prepared by registered dietitians at the GCRC and met the following criteria: no caffeine, aspartame, or snacks and 55% carbohydrate, 15% protein, and 30% fat; sodium was controlled at 3 g. Calories were based on the Harris-Benedict standard formula plus an appropriate activity factor for the subject’s age, gender, and physical activity. Meal times were breakfast at 0630, lunch at 1130, and dinner at 1900. Lunch and dinner times were scheduled around the 1500–1700 afternoon workout to ensure that all subjects exercised in the postabsorptive state and also to allow an acute postexercise sampling regimen that was not influenced by caloric consumption. Subjects were allowed to consume water ad libitum.

Acute heavy-resistance exercise protocol. The acute heavy-resistance exercise protocol was designed to be a high-volume workout (i.e., high total work) that recruited and activated a large amount of muscle tissue. This was accomplished by performing multijoint exercises that required the use of major muscle groups in the lower and upper body. The relative loads for each exercise alternated between 10- and 5-RM loads. The 10- and 5-RM loads were calculated as 70 and 85% of the exercise 1 RM, respectively. The relative loads for each exercise alternated between 10- and 5-RM loads. The 10- and 5-RM loads were calculated as 70 and 85% of the exercise 1 RM, respectively. Subjects terminated the exercise set on the completion of the number of repetitions or muscle failure. In the event that the desired number of repetitions (at 10- or 5-RM loads) was not achieved for a given set, the load was subsequently reduced before the next set of the exercise. A 90-s rest period was given after each exercise (Table 1). All subjects completed the entire workout demonstrating the ability to tolerate and to perform at a high level of strength fitness.

Overnight stays. Subjects underwent two randomized counterbalanced overnight trials at the GCRC, which was housed in the Noll Physiological Research Center. To familiarize the subjects with the facility and safeguard against a documented disrupted first-night sleeping effect, all subjects slept in the GCRC on the night before each overnight serial blood draw. Conditions for the night before were mimicked exactly to include the taping (but not puncture of the skin) of a catheter near the antecubital vein for the night. One of these overnight stays served as the control trial; when the subject reported to the GCRC at 1430, a resting blood sample was taken via venipuncture, and the subject rested quietly until 1700, whereupon a catheter was inserted into the antecubital vein and serial blood samples were drawn at a rate of one draw every 10 min from 1700 to 1800 and one draw every hour thereafter until 0600 on the following morning. Venous blood sampling followed the same procedures during the exercise condition. During the exercise the subject performed the acute heavy-resistance exercise protocol from 1500 to 1700. Blood was obtained as soon as possible after the completion of the last set [time after exercise for the 1st blood draw was 4.3 ± 0.7 (SE) min]. Typical of metabolic ward studies, during each overnight trial, subjects were allowed to move freely, watch television, receive telephone calls, and study quietly. Bedroom lights were turned off at 2200, and the television was turned off at 2300. Serial blood draws were performed throughout the night. Blood was collected, allowed to clot at room temperature, and centrifuged for 30 min at 800 g at 4°C. After centrifugation, serum was aliquoted into an Eppendorf tube, flash frozen in liquid nitrogen, and stored at −80°C until later analysis.

Hormonal analyses. TT, FT, and C concentrations were determined by radioimmunoassays (Diagnostic Product, Los Angeles, CA). The sensitivities and intra-assay variances for these assays were 4 ng/dl and <7.0%, respectively, for TT and 0.15 pg/ml and <5%, respectively, for FT. C concentrations were determined by a double-antibody radioimmunoas-
say (Diagnostic Systems Laboratories, Webster, TX). The sensitivity and intra-assay variance for this assay were 0.11 µg/dl and <4.0%, respectively. All tubes were run through a gamma counter for 60 s (EC & G Wallac Gamma Counter, Turku, Finland).

LH concentrations were determined by a competitive polyclonal radioimmunoassay, the reagents of which were obtained by written request to A. F. Parlow from the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK). The human LH (hLH) antigen (NIDDK-hLH-I-SIAFP-1) was iodinated on site. The primary polyclonal antibody was rabbit NIDDK-anti-hLH-3. The biological potency of 1 ng of the hLH reference preparation (LER 907) was 277 IU in terms of the second international reference preparation of human menopausal gonadotropin. The secondary antibody was sheep anti-rabbit and was used in conjunction with polyethylene glycol. After centrifugation, tubes were aspirated, and the remaining pellet was counted with a gamma counter for 120 s (EC & G Wallac Gamma Counter). The sensitivity for this assay, calculated using B0 ± 2SD, was 0.4 ng/ml. Intra-assay variances were <5.0%. For all assays, all samples were run in duplicate. To eliminate intra-assay variances, all samples for each assay for a single subject were analyzed within the same assay batch.

Deconvolution analysis. Multiparameter deconvolution analysis was used to estimate characteristics of pituitary LH secretion (28–31). For this method, duplicate measures of serum LH concentrations for each time point are resolved into its constituent secretory contributions, and the hormone half-life is simultaneously estimated. Thirteen-hour LH production rates were calculated by multiplying the LH secretory burst frequency by the LH secretory burst mass. The LH burst mass was derived as the analytic integral of the calculated secretory pulse. Deconvolution analysis was carried out at 95% joint statistical confidence intervals for all calculated LH secretory burst amplitudes in a blinded fashion for the exercise vs. control conditions.

Statistical analyses. Differences between the control and exercise conditions for TT, FT, and C concentrations were analyzed with a two-way ANOVA with repeated measures.
burst interval, LH secretory burst mass, or LH secretory burst amplitude. The difference in half-life of LH approached significance \( P = 0.06 \). The product of the mass secreted per burst and LH secretory burst frequency was calculated as the LH production rate. The LH production rate was 24% lower in the exercise than in the control condition (13.59 ± 3.83 vs. 17.91 ± 5.1 IU·l distribution volume\(^{-1}·day^{-1}\)). Representative profiles of LH concentrations for subjects 4 and 10 in the control and exercise conditions are illustrated in Fig. 4. The corresponding LH secretory profiles estimated by deconvolution analysis for these subjects are shown in Fig. 5. For both subjects, exercise dampened the number of LH pulses and the LH production rate observed during exercise.

**DISCUSSION**

This study has shown that high-volume, whole body heavy-resistance exercise in the late afternoon resulted in lowered concentrations of TT and FT throughout the night in young, healthy men. These lowered testosterone concentrations were accompanied by a lowered LH production rate and elevated C concentrations. Thus these data support an inhibited hypothalamic-pituitary regulation of LH secretion as a possible mechanism mediating lower androgen concentrations after strenuous resistance exercise. These findings also show that a single bout of resistance exercise can have an impact on endocrine function that is sustained for \( \geq 13 \) h after exercise. This study is unique, in that we have employed a rigorous blood sampling scheme (every 10 min overnight from 1700 to 0600 for LH and every hour for TT, FT, and C for 13 h after exercise) to more fully characterize the effect of resistance exercise on the pituitary-adrenal-testicular endocrine axis. Additionally, this report represents the only available study to use deconvolution analysis to assess pulsatile LH secretion after resistance exercise in men. This is an important distinction, inasmuch as the abundance of the previous exercise/LH literature has focused on
women (17, 18, 33) or endurance exercise in men (8, 11, 19, 20, 23).

**LH secretion.** A prominent and new finding from the present study is the 24% reduction in overnight LH production rate after resistance exercise. The decline in LH production rate appeared to be explained by small declines in LH pulse frequency and LH secretory burst mass, although it must be pointed out that, singularly, neither the LH pulse frequency nor the LH secretory burst mass was statistically significant in the exercise condition ($P = 0.21$ for both). However, we interpret the lowered LH production rate (i.e., the product of LH pulse frequency and LH secretory burst mass) to indicate a blunted hypothalamic/pituitary signal at the Leydig cell receptor. This, in turn, would have a negative-feedback effect on testosterone secretion. To our knowledge, this is the first report on men to suggest that a reduction in overnight testosterone observed after heavy-resistance exercise was mediated by an attenuation of pituitary LH secretory events.

The lowered LH production rate in the present study was observed with deconvolution analysis over a sampling period of 13 h. A longer and more sophisticated secretory detection program (i.e., deconvolution analysis) may have provided more power and resolution to detect an exercise impact on LH production than previous studies. Two previous studies examining the effects of acute aerobic exercise on LH pulsatility in men failed to find an effect. McColl et al. (20) reported that strenuous exercise (60 min of running at 5% below the anaerobic threshold) induced a small but significant lowering of the area under the LH curve, but no differences in pulsatile LH release were measured over 6 h. MacConnie et al. (19) also found that acute exercise (treadmill running at 72% $V_{\text{O}2\text{ max}}$ for 2 h) did not alter plasma LH concentrations when measured over 8 h.

Our finding of a reduced LH secretion after exercise further extends the results from previous investigations in women (18, 33). In women, a diminishment in LH secretion after exercise has been linked to the energy deficit induced by the metabolic cost of exercise. Williams et al. (33) reported that low energy availability in the presence of aerobic exercise stress created a slowing of the LH pulse frequency in women but did not change in mean LH concentrations or peak amplitudes. Loucks et al. (18) subsequently confirmed and extended these findings by showing that the reduction in LH pulsatility in malnourished women is slowly restored by refeeding. In our study, the subjects received the same amount of total calories during the control and exercise trials. By our not providing extra calories to cover the energy expenditure of the exercise session, it is also conceivable that the lowered LH production rate could be explained by inadequate caloric availability, inasmuch as fasting in men has also been shown to be a potent inhibitor of pulsatile LH secretion (1).

**Table 2. Deconvolution analysis of (13-h) pulsatile LH secretion and half-life in control and exercise conditions**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control</th>
<th>Exercise</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>LH half-life, min</td>
<td>62.8 ± 6.8</td>
<td>74.50 ± 13.1</td>
<td>0.06</td>
</tr>
<tr>
<td>LH secretory burst frequency, events/13 h</td>
<td>4.86 ± 1.3</td>
<td>4.29 ± 0.91</td>
<td>0.20</td>
</tr>
<tr>
<td>LH interburst interval, min</td>
<td>123.8 ± 28.6</td>
<td>141.3 ± 31</td>
<td>0.12</td>
</tr>
<tr>
<td>LH secretory burst mass, IU/l</td>
<td>3.96 ± 1.9</td>
<td>3.29 ± 0.92</td>
<td>0.21</td>
</tr>
<tr>
<td>LH secretory burst amplitude, IU·l⁻¹·min⁻¹</td>
<td>0.316 ± 0.15</td>
<td>0.262 ± 0.07</td>
<td>0.21</td>
</tr>
<tr>
<td>LH production rate/13 h, IU·l⁻¹·distribution volume⁻¹·day⁻¹</td>
<td>17.91 ± 5.1</td>
<td>13.59 ± 3.83</td>
<td>0.006</td>
</tr>
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Values are means ± SE for 10 healthy young men. LH, luteinizing hormone.
Testosterone responses. Overall, for the 13 overnight, 1-h serial measures, TT and FT concentrations were \( \approx 12 \) and \( \approx 15\% \) lower (although there was no significant difference at any individual time point), respectively, after resistance exercise. These data are in agreement with the study of Kern et al. (13), who reported \( \approx 17 \) and \( \approx 30\% \) declines in overnight TT concentrations after low- and moderate-intensity aerobic cycle exercise performed in the late afternoon. However, these data are in contrast to the only other available report for overnight TT concentrations after resistance exercise: McMurray et al. (21) reported elevations in the later morning hours (0500–0700) compared with the control condition. The study of McMurray et al. was comprised of lower total work (18 vs. 50 sets in this study). This is in contrast to the study of Kern et al., in which the two conditions lasted for 2.5 or 4 h. Other research has shown a decline in testosterone concentrations immediately after prolonged exercise ( \( > 2 \) h) (3). Our findings confirm these results and even suggest that such exercise-induced suppression can last for as long as 13 h. Such findings differ for exercise bouts of shorter duration ( \( \leq 90 \) min) (9, 15, 16), in which testosterone concentrations are reported to be elevated; however, this elevation is only transient, inasmuch as concentrations are normalized to resting concentrations within 2 h. Other studies have found that the reductions in testosterone concentrations after prolonged exercise are restored within 24–72 h (9). Hence, it seems that the time course for normalization of testosterone concentrations lies somewhere between 12 and 24 h after exercise.

Of interest is the mechanism whereby overnight testosterone concentrations appeared lower in the circulation after heavy-resistance exercise. In a recent study, De Leo et al. (4) injected saline or 5,000 IU of human chorionic gonadotropin (hCG, a stimulator of Leydig cell testosterone secretion) into a group of 18 men and showed that the reduction in testosterone concentrations after exercise did not occur when hCG was administered. Prior studies using hCG or gonadotropin-releasing hormone injec-
tion have also shown that the testes retain their ability to secrete testosterone in the face of prolonged physical or operational stress (27). The available data seem to point away from a gonadal defect to explain lowered testosterone concentrations and toward a centrally mediated mechanism that is hypothalamic-pituitary in origin.

We previously reported that acute resistance protocols lasting ≤1 h result in elevations in testosterone and that these elevations are influenced by the relative intensity and rest periods between sets (15, 16). We have hypothesized that these testosterone increases facilitate tissue repair and recovery processes after the microtrauma of repetitive near-maximal muscular contractions (i.e., a greater testosterone increase, a greater biological impact). At face value, this seems reasonable, inasmuch as testosterone is known to promote protein synthesis and glycogen metabolism. However, in light of this study’s observation that testosterone concentrations were lower after a resistance exercise bout configured with acute program variables known to increase muscle hypertrophy and strength when performed on a chronic basis (e.g., exercises, relative loads, sets, rest between sets), how should the lowered testosterone concentrations after the resistance exercise in this study be interpreted? Urhausen et al. (26) suggested that the physiological implications of lowered testosterone concentrations after physical exercise could result in an impaired resynthesis of protein and glycogen during the regeneration phase. This, in turn, would induce a shift in the energy metabolism toward an increased fat and decreased carbohydrate utilization. Hence, the lowered testosterone concentrations, in conjunction with elevated C concentrations, likely modulate enhanced lipolytic activity and protein catabolism. Immediately after the stress imposed by high-volume muscular contractions, the body is in a catabolic, rather than an anabolic, energy flux. Hence, a provision of fuels mitigated by lowered testosterone and elevated C assists to replenish energy stores and supply the precursors for later use in gluconeogenesis and protein synthesis.

**C responses.** Overall C concentrations were significantly elevated in the exercise condition. Close scrutiny of the C response patterns, however, indicated that the difference under the conditions was mainly attributed to the large C rise for the 2–3 h immediately after exercise. As mentioned above, the rise in C, along with other metabolic regulators (e.g., catecholamines and growth hormone), contributes to fuel provision to meet the metabolic demands of exercise. Our results differ from the only other study to evaluate overnight C responses after daytime resistance exercise and also from one report on endurance exercise. McMurray et al. (21) reported that resistance exercise conducted at 1800 and comprised of 18 sets did not alter overnight C concentrations compared with the control condition, whereas Hackney (8) reported decreased overnight C concentrations after 90 min of cycling at 70% V\textsubscript{O2\text{max}}. Consistent with our results, Kern et al. (13) reported that low and moderate daytime cycling endurance exercise induced higher C concentrations during the first half of sleep.

Differences in the intensity and duration of exercise, as well as the fitness level of the subject population, are possible factors explaining the varied results for C responses after exercise in the literature (2, 24). Few (7) hypothesized that a critical threshold may exist for exercise, below which C concentrations decline in the circulation as the hormone is taken up by peripheral tissues and above which massive secretion from the adrenal cortex results in elevated systemic concentrations. Pertaining to the resistance exercise protocol employed in the present paradigm, the elevated C concentrations for 3 h after exercise are possibly due to the intensity, volume, and duration of the exercise and the above-average strength and aerobic fitness levels of the subjects (as evidenced by their 1-RM values and V\textsubscript{O2\text{max}}). An important contrast between the C and testosterone results is that the influence of the resistance exercise perturbation did not appear to be long lasting. The fact that C concentrations were restored more rapidly than the testosterone concentrations might suggest that testosterone concentrations are a better “biomarker” of the lasting effects of the physiological strain created by acute exercise stress. In addition to serving as a metabolic regulator of energy needs, another physiological outcome of the elevated C seen in this study, albeit transient, could be in the suppression of testosterone secretion. Glucocorticoids have also been reported to suppress moderately LH secretion in men, inasmuch as Doerr and Pirke (6) showed that the mere production of C can suppress the secretion of testosterone.

In summary, acute heavy-resistance exercise in the late afternoon resulted in suppressed TT and FT concentrations in young healthy men. These changes were accompanied by elevated C concentrations and lowered LH flux concentrations. These data suggest that a centrally mediated mechanism via the hypothalamic-pituitary axis may be partially responsible for the decrease in testicular steroidogenesis. These changes in testosterone and C concentrations likely serve to mediate the need for energy provision during the recovery and regeneration processes incurred by the tissue damage of repeated muscular contractions.

This study could not have been successfully accomplished without the expertise and professionalism of the nursing staff at The Pennsylvania State University Park GCRC at Noll Laboratory (i.e., Nancy Lambert, Paula Kirwin, Laurie Aquilino, and Jan Dwelf). Judy True and Sara Diaz provided excellent dietary support. Adam Hittinger, Skip Hildrebrand, Dave Benson, and Kei Dohi demonstrated yeoman efforts in providing assistance for the overnight blood draws, data reduction, and logistical support. We thank Michael Johnson for providing instruction and software for deconvolution analysis, Chip Harris for providing exercise facilities, and Michele Ilgen for timely ordering of all equipment and supplies. Jeanne Indll and Shari Hallas provided invaluable assistance in the data analysis and preparation of the manuscript. We are also indebted to the highly motivated subjects who enthusiastically completed everything that was asked of them.

This study was supported, in part, by National Institutes of Health Grant M01-RR-10732 and grants from the American College
RESISTANCE EXERCISE AND THE PITUITARY-TESTICULAR AXIS

REFERENCES


