Effects of heavy-resistance training on hormonal response patterns in younger vs. older men

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1Human Performance Laboratory, Ball State University, Muncie, Indiana 47306; 2Center for Sports Medicine/ Noll Physiological Research Center, Pennsylvania State University, University Park, Pennsylvania 16802; 3Department of Biology of Physical Activity, University of Jyväskylä, Jyväskylä, Finland; 4School of Exercise Science and Sport Management, Southern Cross University, Lismore, New South Wales, Australia; 5Nutrition, Metabolism, and Exercise Laboratory, Donald W. Reynolds Department of Geriatrics, University of Arkansas for Medical Sciences, North Little Rock, Arkansas 72114-1706; and 6Department of Sport Science, Colorado College, Colorado Springs, Colorado 80913

Kraemer, William J., Keijo Hakkinen, Robert U. Newton, Bradley C. Nindl, Jeff S. Volek, Matthew McCormick, Lincoln A. Gotshall, Scott E. Gordon, Steven J. Fleck, Wayne W. Campbell, Margot Putukian, and William J. Evans. Effects of heavy-resistance training on hormonal response patterns in younger vs. older men. J. Appl. Physiol. 87(3): 982–992, 1999.—To examine the adaptations of the endocrine system to heavy-resistance training in younger vs. older men, two groups of men (30 and 62 yr old) participated in a 10-wk periodized strength-power training program. Blood was obtained before, immediately after, and 5, 15, and 30 min after exercise at rest before and after training and at rest at –3, 0, 6, and 10 wk for analysis of total testosterone, free testosterone, cortisol, growth hormone, lactate, and ACTH analysis. Resting values for insulin-like growth factor (IGF)-I and IGF-binding protein-3 were determined before and after training. A heavy-resistance exercise test was used to evaluate the exercise-induced responses (4 sets of 10-repetition maximum squats with 90 s of rest between sets). Squat strength and thigh muscle cross-sectional area increased for both groups. The younger group demonstrated higher total and free testosterone and IGF-I than the older men, training-induced increases in free testosterone at rest and with exercise, and increases in resting IGF-binding protein-3. With training the older group demonstrated a significant increase in total testosterone in response to exercise stress along with significant decreases in resting cortisol. These data indicate that older men do respond with an enhanced hormonal profile in the early phase of a resistance training program, but the response is different from that of younger men.

endocrine; aging; sarcopenia; strength; muscle; andropause; somatopause; growth factors

NORMAL AGING IS ASSOCIATED with declines in fat-free mass, particularly muscle mass (i.e., sarcopenia), and subsequent decrements in muscle strength and functional abilities (18–20, 44, 60, 62). With age, plasma concentrations of circulating anabolic hormones and growth factors [e.g., growth hormone (GH), testosterone, and insulin-like growth factor (IGF)-I] are also diminished (i.e., somatopause and andropause) (4, 11, 16, 22, 33, 42, 46, 56, 61, 62). Over the last 15 years, resistance training has been a primary intervention used to offset the effects of aging by minimizing age-related declines in strength, fat-free mass, and functional abilities (2, 14, 18, 20, 54). Campbell et al. (2) demonstrated that resistance training can positively enhance nitrogen retention and whole body muscle protein metabolism in older men. Enhanced protein turnover favoring the growth of muscle tissue is under the homeostatic interactive regulation of the endocrine system (5, 6, 17, 31, 32, 50, 70). The hormonal changes produced by weight-training exercise may be an important stimulus in older men, contributing to the prevention of sarcopenia, loss of strength, and loss of functional abilities. However, data on the effects of strength training on the acute response patterns of hormonal changes with resistance training in older men are limited (5, 18, 54).

Heavy-resistance exercise has been shown to be a potent stimulus for acute increases in circulating hormones in younger men (5, 23, 24, 30, 36–38, 40, 41, 59). In contrast, heavy-resistance exercise has not been shown to elicit the same magnitude of hormonal responses in older men (i.e., >60 yr of age) (5, 23, 54, 59). The importance of circulating hormones resides in the fact that lower amounts of circulating anabolic hormones are thought to influence the age-related decline in muscle mass and its associated strength capabilities observed in the sixth decade of life (4, 17, 61, 62). Because it has been observed in younger men that the alterations in hormonal concentrations may take place in the early phase of training (67), the question arises whether older men can engage similar hormonal mechanisms in the early phase of a resistance training program. Such changes, however, have not been typically observed (5, 18, 20, 54). This might be due to the type of resistance training program utilized, inasmuch as some protocols have not appeared to be as effective in stimulating hormonal responses because of lower intensity, small muscle mass involvement, and/or longer rest...
between sets and exercises (16, 24, 34, 36, 38). Furthermore, only a few studies have presented data on an entire ensemble of anabolic and catabolic hormones and acute and chronic responses of resistance exercise and training (23, 54). If the proper resistance training program (e.g., a periodized program) could enhance the resting and/or exercise-induced responses to resistance exercise, then it might be of therapeutic value for addressing endocrine changes in aging men. The primary purpose of this investigation was to examine the pattern of changes in resting and exercise-induced blood hormonal concentrations in younger and older men before and after a periodized resistance training program designed to enhance muscle size and strength.

METHODS

Subjects. Eight younger [30-yr-old (30Y)] and nine older [62-yr-old (62Y)] men volunteered to participate in this investigation, which was approved by The Pennsylvania State University Institutional Review Board for the Use of Human Subjects. The risks and benefits of the study was thoroughly explained to all subjects, and written informed consent was subsequently obtained. Before inclusion in the study, all subjects were medically screened and approved by a physician as being healthy (i.e., free from any orthopedic, endocrine, or medical problems). None of the subjects was on any medications during the study. All subjects were physically active but had not been involved in any previous structured resistance training programs. Activity questionnaires were utilized and revealed that all subjects were involved in recreational sports and jogging. Pretraining characteristics of the experimental subjects were as follows: for the 30Y group, age was 29.8 ± 5.3 yr, body mass was 90.0 ± 12.8 kg, height was 177.2 ± 5.3 cm, and percent body fat was 18.3 ± 4.6% for the 62Y group, age was 62 ± 3.2 yr, body mass was 84.3 ± 13.4 kg, height was 170.0 ± 7.3 cm, and percent body fat was 20.4 ± 4.6%. From the information collected, it was observed that physical characteristics and activity background of these subjects were essentially similar, with the only significant (P ≤ 0.05) difference between the two groups being age.

Body composition. Body composition was assessed using skinfolds. All anthropometric measurements were obtained by the same investigator on the right side of the subject's body. Skinfold thicknesses were obtained with a Harpenden skinfold caliper (Country Technology, Gays Mills, WI; 10 g/mm constant pressure) at the chest, midaxilla, abdomen, suprailiac, subscapula, triceps, and thigh following the procedures previously described (45). Repeated trials were performed until two measures within 1 mm were obtained, with the mean of these two measures being utilized. A seven-site skinfold equation was used to estimate body density (29), and percent body fat was subsequently calculated using the Siri equation (66). There was no significant difference in percent fat between the groups.

Muscle cross-sectional area. Thigh bone-free muscle cross-sectional area (MCSA) of the dominant leg was assessed before and after the resistance training program by means of a magnetic resonance imaging (MRI) 0.5-T superconduction magnet (Picker International, Highland Heights, OH) with MR6B software. Images were obtained by alteration of the spin-lattice or longitudinal relaxation time (T1). T1-weighted images were obtained using repetition time of 500 ms, echo time of 13 ms, radio frequency of 90°, and power absorption of 0.028 W/kg. MCSA was analyzed from the MRI scans by a gradient echo technique, which allows the greatest delineation and distinction between muscles. Once the subject was positioned within the magnet, the thigh of the dominant leg was supported under the knee to be parallel to the MRI table, and the feet were strapped together to prevent rotation. A sagittal image of the thigh was obtained. A 15-slice grid was placed over the sagittal image, and transaxial images were obtained. Fifteen 1-cm-thick transaxial images were obtained equidistantly between the base of the femoral head and the midknee joint of the thigh. All MRI images were then ported to a Macintosh computer for calculation of total and individual MCSAs with a modified National Institutes of Health (NIH) image software package. For the MCSA, slice 8 was used (slice 1 being the base of the femoral head). Tissue cross-sectional area was obtained by using the NIH 1.55.20A Image Analysis pixel-counting program. The same investigator made all area measurements, and the test-retest reliability intraclass correlation coefficient for this measurement was R = 0.99 (21).

One-repetition maximum test. Maximal strength was assessed using a concentric-only 1-repetition maximum (RM, i.e., the heaviest load that could be correctly performed once) squat test on the Plyometric Power System (Narcomed, Lismore, Australia) from a 90° knee angle (37, 73). Warm-up consisted of a set of 5–10 repetitions at 40–60% perceived maximum. Subjects then rested for ~1 min and performed some light stretching. Thereafter, three to five repetitions were performed with 60–80% of the perceived maximum. Three to four subsequent attempts were then made to determine the 1 RM, with 3–5 min of rest between lifts. Proper technique and a complete range of motion were required for each successful 1-RM trial. No injuries were observed during the 1-RM testing. The test-retest reliability coefficient for this test was R = 0.97.

Acute-heavy-resistance-exercise test. Each subject was familiarized with the experimental acute heavy-resistance exercise test (AHRET) and performed the AHRET before and at the conclusion of the 10-wk training program. The AHRET consisted of four sets of 10-RM squats with 90 s of rest between sets. If because of fatigue on any given set, the subject failed to perform 10 repetitions, the load was subsequently adjusted (e.g., lightened) to allow the completion of 10 repetitions on the following set. The squats were performed with the Plyometric Power System, as previously described (73).

Blood collection and analyses. For the AHRET, blood was obtained preexercise, immediately postexercise (IP), and 5, 15, and 30 min postexercise via an indwelling cannula kept patent with a saline lock 1 wk before training (pre) and at the conclusion of the 10-wk training program (post). In addition, resting blood samples were drawn with similar sterile techniques at ~3 wk (3 wk before the start of training) and at 0, 3, 6, and 10 wk during training. The blood samples drawn before training were obtained to serve as control comparisons, and for the ease of illustration, only the 0-wk values are depicted in RESULTS. Resting blood samples drawn at ~3 and 0 wk were indistinguishable (P > 0.05). Blood samples were obtained at different times of the day among subjects but at the same time of day for each subject during the experiment to limit the influence of any diurnal variations (1, 11, 28, 64). Blood was drawn after an overnight fast, and dietary intake was monitored and recorded across time points. Over the course of the resting baseline time line, blood was drawn from two 30Y subjects and one 62Y subject outside the 90-min window from week 0 (within 2.5 h). Blood was centrifuged at 1,500 g at ~4°C for 15 min. All serum and plasma samples were then
distributed to appropriate preservative tubes and stored at −84°C until analysis. Serum was obtained for total testosterone (TT), free testosterone (FT), cortisol, GH, and lactate analyses, whereas heparinized plasma was obtained for ACTH analysis. Resting values for serum IGF-I and IGF-binding protein-3 (IGFBP-3) were determined only pre- and postraining.

Hematocrit was determined in triplicate using a standard microcapillary technique. Hb was determined colorimetrically in duplicate using the cyanmethemoglobin method (Sigma Chemical, St. Louis, MO). All hormones were analyzed in duplicate using various RIA techniques. To eliminate interassay variance, all samples were analyzed within the same assay batch, and all intra-assay variances were <5%. Serum testosterone (TT) and FT and cortisol were analyzed by single-antibody (solid-phase) 125I-RIAs, whereas a double-antibody assay was used for plasma ACTH and serum GH (Diagnostic Products, Los Angeles, CA). Total IGF-I was analyzed with 125I liquid-phase double-antibody RIA with an octadecasyl silica preliminary column extraction to separate IGF-I from its binding proteins (INSTAR, Stillwater, MN). IGFBP-3 was measured using a two-site immunoradiometric 125I assay (Diagnostic Systems Laboratory, Webster, TX). Antibody sensitivities were 0.14 nmol/l for TT, 0.52 pmol/l for FT, 0.19 pmol/l for ACTH, 5.5 nmol/l for cortisol, <2 nmol/l for IGF-I, 0.5 ng/ml for IGFBP-3, and 0.9 µg/l for GH. An LKB model 1272 Clini gamma counter and on-line data reduction system (Pharmacia LKB Nuclear, Gaithersburg, MD) were used to determine immunoreactivity values. Whole blood lactate concentrations were measured in duplicate using a lactate analyzer (Sport Lactate Analyzer 1500, Yellow Springs Instruments, Yellow Springs, OH). Plasma volume changes were calculated using hematocrit and Hb values and the methods described by Dill and Costill (7). Hormonal concentrations were not corrected for plasma volume changes. The plasma volume changes in this study pre- to post-AHRET (t) were −11.4 ± 6.2 and −14.0 ± 5.6% pretraining and −10.4 ± 5.2 and −12.0 ± 4.6% postraining for the 30Y and 62Y groups, respectively. No significant differences in plasma volume changes were observed between groups or with training.

Periodized strength-power resistance training program. All subjects were carefully familiarized with all the exercise protocols used in the study. In addition, all exercise sessions were individually supervised. The periodized resistance training program consisted of a nonlinear, multiset, multiexercise periodized program performed three times per week for 10 wk (12, 13). The daily workouts were alternated by varying the intensity and the volume (sets × repetitions × load) over the week. On Monday, sets were performed at 3–5 RM with 2–3 min of rest between sets. On Wednesday, sets were performed at 8–10 RM with 1 min of rest between sets. Finally, on Friday, the load was 12–15 RM; however, only 6–8 repetitions were performed with the intention of performing these exercises with greater power output or in an explosive manner. Between sets, 1–2 min of rest were allowed. The program consisted of the following exercises: squats, knee extensions, lower back extensions, lat pull downs, leg curls, calf raises, bench presses, seated rows, military presses, and arm curls.

Statistical analysis. Data were analyzed using a two-way repeated-measures ANOVA, and a Fisher least significant difference post hoc test was used to find differences in the pairwise mean comparisons when appropriate. Statistical power calculations for this study ranged from 0.78 to 0.80. The significance level for this investigation was set at P ≤ 0.05.

Results

Strength and hypertrophy. The 30Y group had greater 1-RM squat strength and total mean thigh MCSA than the 62Y group before and after the training program. After 10 wk of the periodized resistance training program, 1-RM squat strength increased from 139 ± 22 to 163 ± 23 kg and from 102 ± 34 to 113 ± 37 kg for the 30Y and 62Y groups, respectively. Thigh MCSA increased from 186 ± 16 to 204 ± 18 cm² and from 159 ± 22 to 169 ± 26 cm² after 10 wk of the periodized resistance training program for the 30Y and 62Y groups, respectively. Significant age differences were observed for body mass or percent body fat after training for either group. Figure 1 shows the percent increases in 1-RM squat strength and MCSA. The 30Y and 62Y groups experienced similar percent increases for the 1-RM squat (~15%), but the 30Y group experienced a greater amount of MCSA hypertrophy than the 62Y group (10.1 ± 3.7 and 5.9 ± 2.9% for the 30Y and 62Y groups, respectively). Also, an increase of 8.4 ± 4.6% (P ≤ 0.05) in the MCSA calculated separately for the hamstring muscle group in the 30Y group was greater (P ≤ 0.05) than 3.6 ± 3.3% recorded for the 62Y group, whereas the increases of 12.2 ± 3.6 and 8.4 ± 4.6% for the quadriceps femoris did not differ significantly between the groups.

Serial resting hormonal concentrations. Figures 2–4 depict the changes in serial resting concentrations of TT, FT, ACTH, and cortisol at 0, 3, 6, and 10 wk and for IGF-I and IGFBP-3 at pre- and postraining. For TT (Fig. 2A), no significant differences were noted for age or for training. However, interaction effects indicated higher TT concentrations at 6 and 10 wk in the 30Y than in the 60Y men. Significant age and interaction effects were observed for FT responses (Fig. 2B). At 0 wk, there were no differences in FT between the 30Y and 62Y men, but significant differences appeared by 3 wk and persisted throughout the duration of the train-
ing program. In addition, for the 30Y men, resting serum concentrations for FT were elevated at 10 wk compared with 0 wk. For the 62Y men, FT remained unchanged throughout the training program. No age, training, or interaction effects were observed for plasma concentrations of ACTH (Fig. 3A). For cortisol (Fig. 3B), significant interaction effects were apparent, indicating lower concentration at 3 and 10 wk than at 0 wk for the 62Y group.

For GH (Fig. 4A), no age, training, or interaction effects were evident. Figure 4, B and C, shows pre- and posttraining assessment of serum IGF-I and IGFBP-3. Serum IGF-I (Fig. 4B) was higher for the 30Y than for the 62Y men pre- and posttraining. In addition, IGF-I did not change for the 30Y or 62Y men from pre- to posttraining. IGFBP-3 concentrations were higher in the 30Y than in the 60Y men before and after training (Fig. 4C). However, the 30Y men, but not the 62Y men, demonstrated an increase in serum IGFBP-3 after 10 wk of resistance training.

Hormonal responses to the AHRET. Figures 5–7 depict the hormonal and lactate recovery responses after the AHRET before and after 10 wk of training. For TT (Fig. 5A), significant main effects were observed for age. The means for the main effects due to age were 20.3 and 15.7 nmol/l for the 30Y and 62Y men, respectively. Before training, elevations above baseline were observed immediately after the AHRET and at 5 and 15 min of recovery for the 30Y men and immediately after the AHRET and at 15 min of recovery for the 62Y men. At 30 min of recovery, TT concentrations for both groups were similar to preexercise values. The 30Y men demonstrated elevations immediately and 5 min after AHRET. The 62Y men demonstrated elevations immediately after AHRET and at 5 min of recovery. Interaction effects for the 62Y men also indicated that the posttraining 5- and 30-min recovery time points were greater than the corresponding pretraining values.

For FT (Fig. 5B), significant main effects for age, recovery times, and training were observed. The means for the main effects of age were 80 and 60 pmol/l for the 30Y and 62Y men, respectively. The means for the main effects due to training were 66 and 74 pmol/l before and after training, respectively. For the main effect of recovery times, significant elevations above baseline were seen immediately after the AHRET and for 5 and
15 min, but not for 30 min into recovery. Interaction effects revealed differences between the 30Y and 62Y men at all time points, except for the pretraining preexercise value. Also, elevations above preexercise values were observed at IP and at 5 and 15 min post-AHRET for the 30Y men pre- and posttraining. In the 62Y men the FT values were higher than preexercise values at IP and 15 min post-AHRET pretraining and at IP and 5 min post-AHRET posttraining. For the 62Y men the pre-AHRET FT value posttraining was greater than the pretraining value.

Compared with pretraining, for ACTH (Fig. 6A) significant reductions after the AHRET were observed posttraining. In the 30Y men, ACTH was elevated pretraining at IP and at 5 and 15 min; however, posttraining elevations were evident only at IP. In the 62Y men, ACTH was elevated pretraining at IP and at 5 min, but only at IP for posttraining. In addition, in the 30Y men the posttraining values were lower than the corresponding pretraining values at IP and at 5 and 15 min. For the 62Y men the value was lower at IP posttraining than pretraining. Cortisol was elevated above pre-AHRET values at every time point for both age groups pretraining (Fig. 6B). Posttraining, cortisol was elevated above baseline at all time points in the 62Y men. In the 30Y men, cortisol was elevated over preexercise values at 15 and 30 min. Their IP and 5 and 15 min of recovery time points posttraining were lower than the corresponding pretraining values. Differences between the 30Y and 62Y men were observed posttraining at IP and 5 min of recovery.

Lactate was significantly elevated above baseline values after the AHRET for the 30Y and 62Y men before and after the training program (Fig. 7A). Lactate values were higher for the 30Y than for the 62Y group at every time measured, except at 30 min post-AHRET posttraining. Posttraining lactate values were lower than the corresponding pretraining values for the 30Y group at IP and at 5 and 15 min post-AHRET. The 62Y men demonstrated no elevations in GH (Fig. 7B) above pre-AHRET pre- or posttraining. The 30Y men demonstrated elevated values pretraining for 5, 15, and 30 min of recovery. Posttraining, in the 30Y men, values were higher at IP and at 5 and 15 min into recovery than corresponding preexercise baseline values. For the 30Y men the 30-min recovery value posttraining was lower than the corresponding pretraining value.
DISCUSSION

The hormonal responses in this study reflect adaptive changes in a very active and fit group of 60-yr-old men who were capable of undertaking a very aggressive whole body resistance training program. The efficacy and associated adaptations of such a resistance training program in less-fit 60-yr-old men or older populations require further study. However, total body resistance training programs have been used for older adults and may just require proper progression to ensure appropriate toleration of more advanced protocols (13).

Decreases in anabolic hormonal concentrations (e.g., testosterone, GH, and IGF-1) with age can influence the reduction in muscle size and strength observed with aging (32, 50, 63). Restoring an endocrine gland's function with exercise training remains an attractive hypothesis, which could help ameliorate the age-related declines in muscle tissue mass and strength. In this study we examined the early-phase adaptations of circulating hormones to a periodized heavy-resistance training program targeted at increasing muscle size and strength. Although the early phases of resistance training are characterized by considerable neural adaptations (e.g., increased activation of the agonist muscles), adaptations intrinsic to muscle also take place in the early phases (67). In a prior study of younger men, early-phase training adaptations included transformations in muscle fiber type and increases in strength that were associated with early-phase increases in serum testosterone and decreases in serum cortisol (67). Unique to the present study was the use of a periodized heavy-resistance training program, which was effective in eliciting increased muscle.

![Graph A](image1)

**Fig. 5.** Responses (means ± SD) of total testosterone (A) and free testosterone (B) after an acute heavy-resistance exercise test (AHRET) before and after 10 wk of periodized strength and power training for 30Y and 62Y men. *Significantly different (P ≤ 0.05) from corresponding preexercise value; $^\#$ statistically significant difference (P ≤ 0.05) between 30Y and 62Y men; $^\bullet$ statistically different (P ≤ 0.05) from corresponding pretreatment value. Pre, preexercise; IP, immediately after exercise; 5, 15, and 30: 5, 15, and 30 min after exercise.

![Graph B](image2)

**Fig. 6.** Responses (means ± SD) of ACTH (A) and cortisol (B) after AHRET before and after 10 wk of periodized strength and power training for 30Y and 62Y men. *Significantly different (P ≤ 0.05) from corresponding preexercise value; $^\#$ statistically significant difference (P ≤ 0.05) between 30Y and 62Y men; $^\bullet$ significantly different (P ≤ 0.05) from corresponding pretreatment value.
size and muscle strength in just 10 wk. A major finding of the present study was that the younger men demonstrated a significantly greater increase in the size of the whole thigh, yet the relative gains, at least in strength of the knee extensors, were not different between the older and younger groups. The ability of the younger men to elicit a greater relative hypertrophic response in 10 wk of training in this study appears to be associated with the differences in the resting and exercise-induced adaptational patterns of the hormones. The other primary finding of this study was that the endocrine system demonstrated a "plasticity" for adaptational changes in older and younger men in the early phase of a periodized heavy-resistance training program.

In men, testosterone is a potent anabolic hormone that mediates protein accretion and also enhances neural function (6, 51, 70). In general, exercise-induced concentrations of testosterone [e.g., exercise main effects for TT (20.3 vs. 15.7 nmol/l) and FT (80 vs. 60 pmol/l)] were significantly higher in the 30Y than in the 62Y group. In addition, area-under-the-curve analysis revealed a greater magnitude of increase in the exercise-induced responses of FT to a resistance training stimulus in younger men. These data support the maintenance of an "andropause," which is characterized by a decrease in testicular Leydig cell numbers, reductions in secretory capacity, and a decrease in resting episodic and stimulated gonadotropin secretion (69–71). Staron et al. (67) demonstrated that resting concentrations of TT in men averaging 23.5 yr of age increased over baseline values after just 4 wk of training. However, other studies have not shown a training-related increase in resting concentrations of testosterone. This indicates that, in young men, testosterone may exhibit a dynamic homeostatic hormonal response pattern to resistance training. However, in a study by Nicklas et al. (54), no alterations in resting concentrations of TT were observed over 16 wk of progressive training in men averaging 60 yr of age. Häkkinen and Pakarinen (25) observed a similar nonresponse in 70-yr-old men. Despite the lack of changes in hormonal concentrations, they reported significant relationships between testosterone concentrations and change in strength with training in the 70-yr-old men ($r = 0.61$). In the present study, although no changes in resting testosterone were observed with 10 wk of training in the 62Y men, the 62Y group did demonstrate an enhanced adaptational ability to stimulate TT after resistance exercise. This finding is unique and may be related to the periodized training program; however, the exact reasons and the physiological mechanism(s) mediating this adaptation remain speculative.

The mechanism(s) that mediate such an adaptation in exercise-induced serum testosterone concentrations in older men could be due to classic increases in luteinizing hormone (LH) pulsatility or production (34, 46, 51, 69, 71). However, Lu et al. (47) reported that increased testosterone concentrations in male rats during exercise were at least partially the result of a direct (LH-independent) stimulatory mechanism of exercise, with lactate influencing the secretion of testosterone by increasing testicular cAMP production. Recent evidence also implicates nitric oxide (via vasodilatory mechanisms) and blood flow patterns impacting testosterone release at the level of the testis (49). The impact of these exercise-induced changes in the older men remains unclear but could have contributed to the increases in muscle size and strength observed in this study. One might speculate that in older men such adaptational increases in testosterone may have contributed to a greater extent to the similar relative changes in strength by the potent influence of testosterone on neural mechanisms (e.g., increased neurotransmitter synthesis) (36, 51). Neural factors have been demonstrated to be very important, mediating strength increases with short-term resistance training in older men (19, 52, 53). In fact, the similar relative changes in strength of the knee extensors with lower magnitudes of increases in cross-sectional size of the whole thigh

![Graph A](image1.png)

![Graph B](image2.png)
(and also of the quadriceps femoris, although not statistically different from the 30Y group in older men) appear to support the theory that neural factors play a great role in explaining 1-RM force production (19, 52, 53, 58). More specifically, both age groups demonstrated smaller increases in the size of the hamstrings than that observed for the quadriceps femoris, and the 62Y group showed a smaller training-induced increase in the hamstring muscle than the respective increase recorded for the 30Y group. Consistent with the present data, smaller increases in the size of the hamstring muscle group than in the quadriceps femoris have been demonstrated in older individuals (65). Because strength of the knee flexors was not recorded, it was not possible to evaluate the contribution of neural factors to strength development of the hamstrings between the 30Y and 62Y men in this study.

Resting FT concentrations were higher in the younger men through most of the training period, and the younger men again showed the ability to elicit a higher exercise-induced response of FT than the older men. Häkkinen and Pakarinen (25) previously demonstrated the importance of biologically active FT for trainability in younger men. Testosterone circulates in a bound form in the blood primarily with sex hormone-binding globulin or albumin. Although the free hormone (FT is considered both the testosterone that is circulating unbound and the testosterone that circulates bound to albumin) has been thought to be regulated by TT, the bound testosterone complex, with a larger molecular weight, has been shown to be unable to traverse the capillary endothelium and penetrate the plasma membrane to interact with the regulatory elements of the nucleus (27, 36, 48, 57). Thus higher FT in the blood appears to indicate an improved "bioactivity status" for the younger men (51, 55, 56). Overall, a greater androgenic environment for the younger men at rest and after exercise sessions appears to support the higher overall magnitude of the observed increase in thigh muscle size. In addition, these data give potential insights into the age-related differences in the development of muscle tissue with short-term training programs, although this difference may not hold true to the same extent for all muscles or muscle groups. Thus there appears to be an age-related advantage in FT-mediated mechanism(s) to respond to a resistance training program in the early phases of training.

Another important finding of this study was that the amount of cortisol produced at resting levels was reduced and the response to the resistance exercise stress was lower in the older men. The decrease in resting concentrations of cortisol throughout the training program in the older men without significant changes in the ACTH concentrations indicates that the ACTH receptors in the adrenal gland may have been "downregulated" (27, 35). With training, reductions in the response of cortisol to resistance exercise stress were observed in only the younger men. In an earlier study, Staron et al. (67) showed a similar response in younger men. These changes in exercise-induced cortisol responses observed after exercise are apparently mediated by a reduction in ACTH responses to the resistance exercise stress. The reductions in cortisol have been thought to provide one possible mechanism by which protein accretion via reduced degradation in type I muscle fibers is enhanced (8, 36). Older men may also rely on hypertrophy of type I muscle fibers to elicit total muscle hypertrophy due to a loss of type II muscle fibers with the aging process (43). Thus the importance of these changes in cortisol cannot be minimized, especially in the older men, as a mechanism related to tissue hypertrophy and force production abilities. Collectively, these data indicate that the older men can elicit a reduction in the catabolic hormonal responses, resulting in a more favorable anabolic environment for reduced protein degradation or increased protein synthesis.

In this study no significant changes in GH were observed for resting concentrations throughout the training program for younger or older subjects. This result is supported by similar findings in the literature (5, 15, 25, 26, 54, 67). The lactate response to exercise was lower in the older than in the younger men with use of the same relative exercise stress. The younger men also demonstrated a slightly reduced lactate response to the same relative exercise intensity after training, and this may partially explain the trend for some of the postexercise GH values to be lower, inasmuch as acid-base mechanisms have been shown to be related to GH release (15). Although no changes have typically been observed for resistance training studies in GH adaptations of older men, GH responses following constant exercise responses after endurance-type training are reduced (72). Because of the pulsatile nature of GH, single resting measures must be interpreted cautiously, especially in younger men, who show a much more prominent pulsatile secretion (11, 71). Our data are consistent with the findings of other resistance training studies of GH in older and younger men (5, 25, 54, 67). In general, significant increases in the pattern of exercise-induced GH after resistance exercise were observed for only the 30Y group. This is in agreement with previous studies in which exercise did not induce a GH response in older men (5, 54) and in which the GH response was reduced after resistance training at some but not all times of recovery (63). These results may be in part due to differences in age of the subjects or differences in the training programs (i.e., total sets, repetitions, rest periods, and exercises). There is only a rudimentary understanding of the effects of exercise and aging on the physiological mechanisms underlying "somatopause" (i.e., the diminishment of the GH-IGF-I system) (4, 61). Because this endocrine axis has been thought to be of great importance in maintaining the integrity of the musculoskeletal system, it is conceptually pragmatic to suggest that exercise regimens should attempt to target it. GH also exhibits a great deal of molecular heterogeneity, and the standard RIA focuses on the 22-kDa variant (3, 10, 68). This is an especially important point for future studies, when it is considered that the higher molecular weight species of GH could possess greater biological...
activity and that the lack of changes or reductions in the immunoreactive GH may not present the complete picture of the adaptational responses of GH variants to resistance exercise training (3, 10, 15, 68).

Similar to other studies, IGF-I was lower in the 62Y than in the 30Y group (4, 54). Ten weeks of training did not affect this age-related difference, and therefore short-term training does not appear to be effective in altering IGF-I concentrations (54). However, unique to this study was the finding that resistance training significantly increased IGFBP-3 in the younger group. In addition, the concentration of IGFBP-3 was higher than in the older group pre- and postraining. IGF-I and IGFBP-3 are thought to be released from the liver, yet few studies have examined the effects of chronic resistance training on the components of the IGF system. In endurance training studies, IGFBP-3 has been shown to be independent of IGF-I responses and potentially have its own biological activity at the level of the cell (32). Recently, Elikiam et al. (9) showed that muscle IGF-I concentrations can increase with endurance training, despite lack of change in muscle IGF-I mRNA or serum IGF-I. Lack of change in serum IGF-I with training may suggest that IGF-I in the circulation may not be a meaningful marker of the implicit activity of the GH-IGF-I system. IGF-I may operate in more of an autocrine/paracrine fashion in muscle, or increased secretion from the liver may be quickly sequestered by the tissue in an effort to maintain a homeostatic balance of IGF-I in the systemic circulation. IGF-I exerts a negative inhibition on GH secretion at the level of the hypothalamus and the pituitary. Tissue sequestration of IGF-I could serve to augment its mitogenic actions and, at the same time, protect the system from a decline in hypothalamic and pituitary GH release.

In summary, this study has demonstrated differences in hormonal concentrations between men averaging 30 and 62 yr of age. However, periodized resistance training is very effective in producing initial gains in muscle size and strength. These adaptations are associated most likely with early neural adaptations of the trained muscles and also with changes in the endocrine system in older and younger men. The inability to engage similar hormonal mechanisms in response to heavy-resistance exercise training indicates that the plasticity of the endocrine system in older men has been altered or impaired. However, this study was the first to demonstrate that older men can make physiological adaptations in the endocrine system with resistance training. Whether longer periods of periodized resistance training in older men will continue to produce further hormonal adaptations that are associated with further changes in muscle size, strength, and functional abilities remains a provocative hypothesis for further study.

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