Compatibility of high-intensity strength and endurance training on hormonal and skeletal muscle adaptations

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Kraemer, William J., John F. Patton, Scott E. Gordon, Everett A. Harman, Michael R. Deschenes, Katy Reynolds, Robert U. Newton, N. Travis Triplett, and Joseph E. Dziados. Compatibility of high-intensity strength and endurance training on hormonal and skeletal muscle adaptations. *J. Appl. Physiol.* 78(3): 976–989, 1995.—Thirty-five healthy men were matched and randomly assigned to one of four training groups that performed high-intensity strength and endurance training (C; $n = 9$), upper body only high-intensity strength and endurance training (UC; $n = 9$), high-intensity endurance training (E; $n = 8$), or high-intensity strength training (ST; $n = 9$). The C and ST groups significantly increased one-repetition maximum strength for all exercises ($P < 0.05$). Only the C, UC, and E groups demonstrated significant increases in treadmill maximal oxygen consumption. The ST group showed significant increases in power output. Hormonal responses to treadmill exercise demonstrated a differential response to the different training programs, indicating that the underlying physiological milieu differed with the training program. Significant changes in muscle fiber areas were as follows: types I, IIa, and IIc increased in the ST group; types I and IIc decreased in the E group; type IIa increased in the C group; and there were no changes in the UC group. Significant shifts in percentage from type IIb to type IIa were observed in all training groups, with the greatest shift in the groups in which resistance trained the thigh musculature. This investigation indicates that the combination of strength and endurance training results in an attenuation of the performance improvements and physiological adaptations typical of single-mode training.

testosterone; cortisol; anaerobic power; muscle fibers

THE PHYSIOLOGICAL COMPATIBILITY of simultaneous strength and endurance training has been a subject of great interest over the past 10 years (6, 11). By the use of various experimental protocols, studies have shown that strength can be either compromised (10, 17, 18, 21, 34, 38) or increased (2, 20, 39) while no decreases in endurance capabilities are shown or that both strength and endurance capabilities can be attenuated, especially over longer periods of simultaneous training or in trained athletes (17, 34).

The physiological mechanisms that may mediate such adaptational responses to simultaneous training remain speculative but appear related to alterations in neural recruitment patterns and/or attenuation of muscle hypertrophy (6, 10, 11). Such physiological attenuation may, in fact, result in overtraining (i.e., a decrease in performance) (17, 34). It is also possible that if the simultaneous exercise-training programs are properly designed, they may just require a longer period of time for summation of the ultimate expression of the same magnitude of physiological adaptations.

Few cellular data are available to provide insight into changes at the muscle fiber level with concurrent strength and endurance training (34, 39). In addition, no data are available on endocrine responses to simultaneous strength and endurance training. Anabolic and catabolic hormones (e.g., testosterone and cortisol, respectively) may play a vital role in mediating any differential responses to simultaneous strength and endurance training. Kraemer et al. (25) had previously demonstrated that simultaneous sprint and endurance training produce differential cortisol responses compared with sprint or endurance training only. High-intensity strength training results in a potent stimulus for muscle cell hypertrophy that appears mediated via increases in protein synthesis and accretion of contractile proteins (12). Conversely, an oxidative endurance-training stress causes muscle to respond in an opposite fashion by ultimately degrading and sloughing myofibrillar protein to optimize oxygen uptake kinetics (22, 44, 45). Anabolic and catabolic hormones play a key role in such metabolic phenomena (16).

The majority of studies in the literature have utilized relatively untrained subjects to examine the physiological effects of simultaneous strength and endurance training (6, 11). Few data are available regarding the effects of simultaneous strength and endurance training that utilized previously active or fit individuals who are able to tolerate much higher intensity exercise-training programs (17). Athletes and specialized military units may need such high-intensity training programs to attain higher levels of performance. The primary purpose of this investigation was to examine the physiological adaptations to simultaneous high-intensity strength and endurance training in physically active men. In addition, we wanted to examine the effects of strength training with the upper body alone in combination with endurance training performed with lower body musculature. It was hypothesized that only the musculature that underwent simultaneous training would demonstrate an altered physiological response due to the duality of the exercise stimulus.

METHODS

Subjects

Before the study, the subjects had the investigation fully explained to them. Each was informed of all the potential risks of the investigation and then given an opportunity to

TABLE 1. *Subject characteristics*

Group	*n*	Age, yr	Height, cm	Body Mass, kg	Body Fat, %
Combined	9	23.3±3.6	174.1±6.4	74.2±6.7	13.1±6.1
Upper body combined	9	22.9±5.0	176.7±4.0	75.6±8.5	17.4±2.9
Strength	9	24.3+5.1	175.3±6.1	76.6+14.0	18.3+7.7
Endurance	8	21.4±4.1	177.6±7.8	75.3±6.7	18.5±7.7
Control	5	22.4±4.2	176.5±7.0	76.1±5.4	15.4±7.2

Data are means ± SD; *n*, no. of subjects.

sign an institutionally approved informed consent document. Investigators adhered to Army Regulation 70–25 and US Army Medical Research and Development Command Regulation 70–25 on Use of Volunteers in Research. All subjects were men and were cleared with a physical examination by a physician before the start of the study, and none had any medical or endocrine disorders that would confound or limit his ability to participate fully in the investigation. Each subject was a member of the US Army and classified as physically active, having been involved with standard military physical training programs at least 3 times/wk for at least 2 yr before the start of the study. All subjects were housed, fed, trained, and tested on base at the US Army Natick Research, Development, and Engineering Center, Natick, MA.

The subjects were matched by body size, age, and training status in sets of four, so that one individual of each matched set was randomly assigned to a different group. Training status was evaluated from an interview and an activity questionnaire that assessed the mode, frequency, duration, and intensity of training activities the subjects had been involved with over the year before the study. The soldier's most recent Army Physical Fitness Test (maximum number of sit-ups in 2 min, maximum number of push-ups in 2 min, and 2-mi run time) was also used to help establish the subject's training status. The randomization process was done by an independent investigator. One of the subjects in the endurance group had to be dropped from the study due to an acute hernia, not caused by the experiment, in the first week of training. The four training groups were high-intensity endurance training only (E; $n = 8$), high-intensity total body strength training only (ST; $n = 9$), combined high-intensity total body strength training and endurance training (C; $n = 9$), and combined high-intensity upper body strength training and lower body endurance training (UC; $n = 9$). Five subjects with similar profiles to the training groups served as control subjects for the muscle biopsy procedure. All of the other tests utilized in the investigation (utilizing various military subjects) had test-retest reliabilities over the 12 wk duration equal to or greater than $r = 0.94$. Body composition of the subjects was assessed with methods previously described (15, 46, 47). Subject characteristics are shown in Table 1. No significant differences were observed in any of the variables at the start of the investigation.

The training programs were 12 wk in duration. Subjects performed only the assigned training programs prescribed in this study and no other exercise training. Before the start of the 12-wk training program, 2–3 wk were used to fully familiarize every subject with each of the experimental tests and respective training protocols. Care was taken to have each subject practice the experimental tests to eliminate improvements due to simply learning how to perform the test (12). Each subject also practiced his respective training protocols.

Training Programs

Training took place 4 days/wk (Monday, Tuesday, Thursday, and Friday). All workouts were individually supervised and monitored for progress. Endurance run workouts were started at 0800, and strength-training workouts were started at 1300. The E and ST groups trained at the above times noted for their specific modes of exercise. The combined training groups (C and UC) waited 5–6 h after their run workout to do their lift workout. All subjects completed 100% of the workouts. As test subjects improved in strength and/or endurance, as indicated by weight-lifting repetitions performed, postrun heart rate, treadmill testing, or run times, workout intensities were progressively increased within the constraints of each exercise program type (weights increased for the lift programs while exercise-to-rest ratios decreased for run training as well as run speeds increased). No injuries were observed in this investigation.

The high-intensity strength training program, shown in Table 2, consisted of varied workouts within each week designed to enhance muscle size and strength (15). Thus, the subjects performed both moderate and heavy workouts, previously operationally defined as "hypertrophy" (H) and "strength" (S) workouts, respectively. Such workouts have been previously characterized as to their acute hormonal response patterns (24, 28). In addition, profiles of competitive body builders and power lifters showed that the midpoint repetition maximum (RM) utilized by these athletes were the 10-RM and 5-RM load schemes, respectively (29). Because body builders are primarily interested in the size of muscle and power lifters are most interested in maximal 1-RM force production, we utilized both of these qualities of training in this investigation to provide our needed program variation.

TABLE 2. *High-intensity strength-training workouts*

Monday/Thursday H Workout		Tuesday/Thursday S Workout	
Exercise	No. of sets × RM	Exercise	No. of sets × RM
	Upper body		
Bench press*	3 × 10 RM	Bench press	5 × 5 RM
Fly*	3 × 10 RM		
Military press*	2 × 10 RM	Military press	5 × 5 RM
Upright row*	2 × 10 RM		
Latissimus pull down*	3 × 10 RM	Arm curl	5 × 5 RM
Seated row*	3 × 10 RM		
Arm curl	3 × 10 RM	Latissimus pull down	5 × 5 RM
Sit-up	2 × 25 RM	Obliques (twists)	5 × 5 RM
		Sit-up	5 × 5 RM
	Lower body		
Single knee extension*	3 × 10 RM	Calf raise	3 × 10 RM
Single leg curl*	3 × 10 RM		
Calf raise	3 × 15 RM	Double knee extension	5 × 5 RM
Split squat	3 × 10 RM	Leg press	5 × 5 RM
		Dead lift	4 × 6 RM

Strength (S) workouts used 2- to 3-min rest periods between sets and exercises. Hypertrophy (H) workouts used 1-min rest periods between sets and exercises. RM, repetition maximum (maximum that could be lifted for indicated number of repetitions). * Superset of paired exercises that were performed in sequence and rest was taken after paired exercises were performed.

TABLE 3. *High-intensity endurance-running workouts*

Distance Workouts (Monday/Thursday)	Interval Workouts (Tuesday/Friday)
Warm-up	Warm-up
Maximum distance in 40 min	200- to 800-m intervals
80–85% $\dot{V}O_{2\,max}$	95–100+% $\dot{V}O_{2\,max}$
	Exercise-to-rest ratio went from 1:4 to 1:0.5

$\dot{V}O_{2\,max}$, maximal oxygen consumption.

The H workouts involved the selection of weights targeted for the performance of only 10 repetitions (10 RM) and were performed on Mondays and Thursdays. Similarly, the S workouts involved the selection of weights targeted for the performance of only 5 repetitions (5 RM) and were performed on Tuesdays and Fridays. A universal weight machine and free weights (York Barbell, York, PA) were used for all exercises. Strength testing utilized the same equipment. S workouts were split up during the week and paired with run workouts, so that on each training day only one of the exercise workouts [i.e., H or sprint-interval (SI) workouts] produced high levels of blood lactate for those subjects performing combined training (C and UC groups). To confirm the glycolytic nature of these workouts, we used finger-stick samples and measured the blood lactate levels 5 min after these workouts. The H and SI workouts demonstrated blood lactate levels of 10 mM or greater.

To provide variation, the endurance-training program consisted of both long-distance (LD) and SI protocols. The programs were designed to optimize oxidative aerobic stress (25). On Mondays and Thursdays, LD workouts were performed, and on Tuesdays and Fridays, SI workouts were performed. Exercise prescriptions were based on heart rates measured during treadmill testing. Heart rates were monitored for maintaining appropriate intensities based on each of the two protocols' exercise prescriptions. The LD training was performed on a 1-mi. course with varying terrain and each subject running as far as possible in 40 min. A 400-m track was used to perform all SI workouts. The SIs ranged from 200 to 800 m, and exercise-to-rest time ratios progressed from 1:4 to 1:0.5. A 1,500-m warm-up and cool-down run was performed during each SI training session. Excluding warm-up and cool-down distances, LD running encompassed ~70% of the total distance run in training. The total distance increased over the course of the training as the subjects increased their exercise tolerance. Nevertheless, the ratio of LD to SI distance remained relatively constant. Based on treadmill heart rate and maximal oxygen consumption ($\dot{V}O_{2\,max}$) relationships, the percentage of $\dot{V}O_{2\,max}$ for the workouts was estimated. The run workouts are shown in Table 3.

Testing Schedule

Subject testing took place before the start of the study, at 4 and 8 wk of training, and after 12 wk of training. Biopsy samples were obtained first, followed by a 24-h recovery before other testing. Except for treadmill tests performed between 0800–1000 due to known diurnal hormonal variations, all other tests were balanced and randomized for the time of day. Care was taken to allow at least 1 h of rest between strength and anaerobic tests, and only one treadmill test took place on a given day. Although testing took place throughout the day to reduce variance from any unknown diurnal variations, all tests for a given subject were administered at the same time of day as the first test (e.g., if a subject performed a bench press test at 1300 in the first testing, he always performed it at 1300 for the subsequent tests). Training was integrated into the test week schedules. A 48-h rest was allowed after the last training session of *week 12* of training, a biopsy sample was again obtained, and the same sequence of testing followed.

Strength Testing

1-RM strength was determined for the bench press, leg press, military press, and double leg extension exercises (Universal Weight Machine, Universal Gym, Cedar Rapids, IA) to gain measures of maximal dynamic force production in the upper and lower body musculature. The 1 RMs were the maximal weights that could be lifted through a full range of motion and utilized methods previously described (26, 27, 29). No injuries were observed in any of the strength testing.

$\dot{V}O_{2\,max}$ Determination

Because of the measurement of the relative hormonal changes to exercise stress, we had the opportunity to gain repeat $\dot{V}O_{2\,max}$ test data on two occasions. We hoped that this would allow even more assurances that no anomalies existed with single test results, and none was observed. A continuous treadmill exercise test protocol to exhaustion was used to determine $\dot{V}O_{2\,max}$. The treadmill speed was based on the fitness level of the subject (2-mi. run time) and ranged from 6 to 7 mi./h starting at 0% grade for 4 min and was raised by 2% grade every 2 min thereafter. $\dot{V}O_{2\,max}$ was measured again during a discontinuous progressive exercise treadmill test used for blood collections. Criteria for determination of $\dot{V}O_{2\,max}$ have been previously described (32, 43). $\dot{V}O_{2\,max}$ data from the two tests were within 3%. An on-line metabolic system and electrocardiogram (lead II configuration) were utilized for cardiorespiratory data acquisition (7). For the discontinuous test, 7-min stages at exercise intensities of 25, 50, and 75% of $\dot{V}O_{2\,max}$ were used, and a 2- to 3-min stage at 100% $\dot{V}O_{2\,max}$ was used with a 1-min rest period between stages to obtain blood samples to evaluate serum testosterone and cortisol responses, which represent the primary anabolic and catabolic hormones in men (16, 23).

Anaerobic Power Determinations

To examine the effects of simultaneous strength and endurance training on power production capabilities, upper and lower body anaerobic power measurements were determined using the Wingate anaerobic test (WAT). A computer-interfaced Monark ergometer was used for both upper and lower body tests. The equipment and testing protocols have been previously described (33, 35, 36).

Muscle Biopsy Samples

To determine the potential differential training effects in the muscle fibers, percutaneous needle biopsy samples were obtained from muscle ~10 days before the start of training and ~48 h after the last training session. Samples were obtained from the superficial portion of the vastus lateralis muscle of the dominant thigh by utilizing the percutaneous needle biopsy technique of Bergström (3) as modified by Evans et al. (13). Due to possible variation in fiber type distribution from superficial to deep and proximal to distal sites, special care was taken to extract tissue from approximately the same location each time by using the prebiopsy scar (~0.5 cm from scar going from medial to lateral) and marked needle depth (usually 2 cm) (4, 31). We utilized a procedure similar to one previously published (42). Data from repeat biopsies

(randomly performed) demonstrated insignificant intrabiopsy variations in fiber type distributions.

Muscle tissue samples were oriented in embedding medium (i.e., tragancanth gum), frozen in isopentane cooled to −159°C with liquid N_2, and stored at −120°C until analyzed. Serial cross sections (12 μm thick) were cut on a cryostat (American Optical, Buffalo, NY) at −20°C for histochemical analyses. Pre- and posttraining samples were histochemically analyzed in the same assay to avoid interassay variances.

Histochemical analyses used for fiber typing consisted of assaying for myofibrillar adenosinetriphosphatase (ATPase) activity at pH 4.3, 4.6, and 10.3. Muscle fiber types were divided into four groups (types I, IIa, IIb, and IIc) based on the stability of their ATPase activity in the preincubation medium (5, 40, 41). Type IIab fibers were classified with the type IIb muscle fibers for quantification (41).

Fiber type percentages were calculated from the total number of fibers in the muscle tissue sections that contained an average of 1,850 ± 320 fibers (range 947–2,830 fibers). Fiber type percentages were computed by a Zeiss Interactive Digital Analysis System (ZIDAS; Zeiss, Thornwood, NY) from projections at a constant magnification with a Zeiss microscope (standard 16 drawing tube) onto a digitizing tablet with a self-contained computer running appropriate morphometric programs (30). This was interfaced with a mainframe computer (VAX 11/780, Digital Equipment, Maynard, MA) system for data storage and analysis. In addition to myosin ATPase quantification, muscle fiber areas were determined using nicotinamide-adenine dinucleotide tetrazolium reductase-stained fibers to avoid any possible shrinkage due to the alcohol used in the ATPase histochemical assay. The perimeters of all intact fibers of each muscle fiber type were measured. Cross sections were projected at a constant magnification with a Zeiss microscope onto the digitizing tablet. Fiber areas were determined by tracing the perimeter of each fiber on the digitizing tablet and calculating the area with the ZIDAS computer system.

Blood Collections

Thirty minutes before the discontinuous treadmill test, an indwelling 20-gauge Teflon cannula was placed into a superficial arm vein and kept patent with a continuous flow of isotonic saline (30 ml/h). Samples were collected via a syringe-and-stopcock arrangement on the cannula. A resting blood sample was collected in the standing position after 20 min of positional equilibration. Subsequent samples were obtained after each exercise stage and at 5 and 15 min into recovery. Blood samples were processed and centrifuged, and the serum was stored at −120°C until analyzed.

Biochemical Blood Analyses

Hemoglobin was analyzed in triplicate using the cyanmethemoglobin method (Sigma Chemical, St. Louis, MO), and hematocrit was analyzed in triplicate using standard microcapillary technique. The percent changes in plasma volume were calculated according to equations by Dill and Costill (9). Hormones were not corrected for plasma volume changes, which were all less than −15%. Analyses of corrected values demonstrated the same statistical response patterns. Serum testosterone and cortisol were determined in duplicate via solid-phase ^{125}I radioimmunoassays (Diagnostics Products, Los Angeles, CA). Intra- and interassay variances for testosterone were 4.7 and 6.4%, respectively, with a sensitivity of 0.14 nM. Intra- and interassay variances for cortisol were 5.3 and 6.2%, respectively, with a sensitivity of 5.5 nM. All samples were thawed only once for analysis, with each subject's samples run in the same assay to reduce variation.

Statistical Analyses

Appropriate statistical assumptions for each analysis were tested before evaluation of the data. Area under the curve (AUC) was also calculated for the hormonal data using a standard trapezoidal method. The statistical evaluation of the data started with a multicovariate analysis of variance with the pretraining value acting as the covariate. It was determined that the pretraining value in none of the data sets had a significant influence on the pattern of response. Appropriate (two-way or three-way) multivariate analysis of variance (power range = 0.459–0.665) was then used for the primary data analyses using repeated measures and subsequent Tukey's post hoc tests for appropriate pairwise comparisons. Selected Δ percentages of change pretraining to 12 wk of training were analyzed via a one-way analysis of variance. Statistical significance was chosen as $P \leq 0.05$.

RESULTS

1-RM Strength

Figure 1 shows the results of the strength testing. No significant differences were observed among groups in pretraining strength levels for each 1 RM. Significant increases in 1 RM for double leg extension strength were observed for the C and ST groups at 4, 8, and 12 wk. In the leg press, significant increases in 1 RM were demonstrated at *weeks 8* and *12* for the C group and at *weeks 4, 8,* and *12* for the ST group. Significant increases in 1 RM for the bench press were observed for the C, UC, and ST groups at 4, 8, and 12 wk. In the military press, 1 RM significantly increased at *weeks 8* and *12* for the C, UC, and ST groups. Percent improvements for the leg press were 19.50 ± 9.50 (SD), 9.60 ± 6.83, 30.00 ± 7.67, and 1.70 ± 1.20% for the C, UC, ST, and E groups, respectively, and those for the double leg extension were 34.40 ± 8.61, 10.90 ± 6.5, 34.40 ± 11.4, and 3.10 ± 1.7% for the C, UC, ST, and E groups, respectively. A significant difference was found in the percentages for leg press improvements (ST > C > UC > E) and for double leg extension (C and ST > UC > E).

$\dot{V}O_{2\,max}$

Table 4 presents the changes in $\dot{V}O_{2\,max}$ for each group over the 12-wk training program. *Groups C, UC,* and *E* demonstrated significant increases in treadmill $\dot{V}O_{2\,max}$ by *week 12* of training. Percent improvement pre- to posttraining for each of the groups was 7.69 ± 4.5, 9.62 ± 3.2, −0.99 ± 1.3, and 11.82 ± 3.9% for the C, UC, ST,

TABLE 4. *Changes in* $\dot{V}O_{2\,max}$

Group	Pretraining	4 wk	8 wk	12 wk
Combined	58.88±5.95	59.65±7.38	56.96±8.32	63.41±8.02*
Upper body combined	51.43±6.92	51.80±3.87	51.10±4.44	56.38±4.69*
Strength	53.47±4.95	51.60±5.39	47.04±5.71	53.02±4.34
Endurance	52.45±5.59	54.03±7.69	54.46±3.48	58.65±6.87*

Values are means ± SD in $ml \cdot kg^{-1} \cdot min^{-1}$. * $P < 0.05$ vs. corresponding pretraining value.

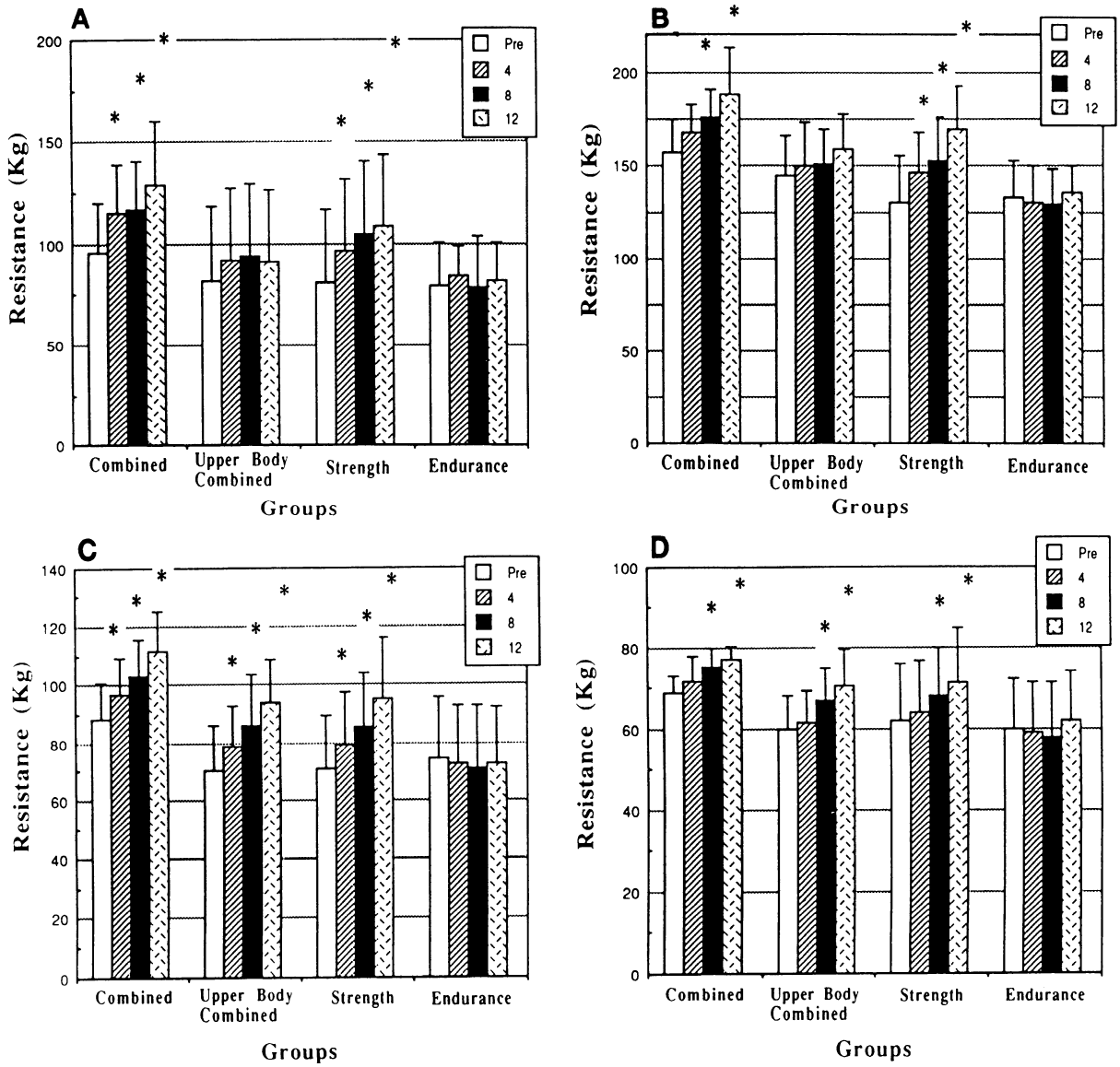

FIG. 1. 1-Repetition maximum (1-RM) strength changes over training program [double leg extension (*A*), leg press (*B*), bench press (*C*), and military press (*D*)]. Values are means ± SE. * $P < 0.05$ vs. corresponding pretraining value.

and E groups, respectively. No significant difference was observed between the C, UC, and E groups, which were all significantly greater than the ST group.

Anaerobic Power

Table 5 shows the results of the WAT for each training group. The C group demonstrated a significant increase in the mean power output of the arms at *week 12*. The ST group demonstrated significant increases in peak and mean power output for the legs and the arms by *week 12* of the training program. No changes were observed for any of the other training groups.

Muscle Fiber Data

The changes in muscle fiber morphology are presented in Table 6. *Group C* demonstrated a significant decrease in the percentage of type IIb muscle fibers and a significant increase in the percentage of type IIa muscle fibers pre- to posttraining. In addition, the C group demonstrated a significant increase in only type IIa muscle fiber area. The ST group demonstrated a significant decrease in the percentage of type IIb muscle fibers and an increase in percentage of type IIa muscle fibers. The ST group also demonstrated significant increases in muscle fiber areas for types I, IIc, and IIa pre- to posttraining. For the E group, a significant increase in the percentage of types IIc and IIa muscle fibers was observed along with a significant decrease in the percentage of type IIb muscle fibers. The E group demonstrated a significant decrease in the muscle fiber areas in the type I and type IIc fibers. The UC group demonstrated a significant increase in the percentage of the types IIc and IIa muscle fibers and a decrease in percentage of type IIb muscle fibers pre- to posttraining. The UC group demonstrated no changes in the muscle fiber areas. No significant changes were ob-

TABLE 5. *Changes in Wingate anaerobic test measures*

	Pretraining	4 wk	8 wk	12 wk
		Combined group		
Peak power, legs	742±87	710±70	756±119	784±101
Mean power, legs	502±48	487±47	488±87	525±68
Peak power, arms	651±43	634±74	665±59	684±68
Mean power, arms	476±33	473±59	494±52	516±44*
		Upper body combined group		
Peak power, legs	650±145	702±109	658±137	697±112
Mean power, legs	443±97	469±70	438±118	458±79
Peak power, arms	635±110	614±70	665±78	676±63
Mean power, arms	443±80	433±51	462±64	478±43
		Endurance group		
Peak power, legs	645±87	624±78	648±123	637±65
Mean power, legs	441±58	430±86	421±94	427±62
Peak power, arms	576±74	588±47	599±62	573±44
Mean power, arms	396±89	406±46	434±61	414±49
		Strength group		
Peak power, legs	627±89	659±82	690±149	735±123*
Mean power, legs	399±62	430±71	442±95	480±82*
Peak power, arms	595±90	610±119	636±129	656±125*
Mean power, arms	425±80	433±51	462±64	478±43*

Values are means ± SD in W. * $P \leq 0.05$ vs. corresponding pretraining value.

served in the control values pre- to posttraining. The ST group had a significantly higher percent increase in muscle fiber areas for the type I, type IIc, and type IIa fibers compared with the E, UC, and control groups. The percent increase in fiber areas for the types I and IIc fibers for the ST group was significantly greater than that for the C group. The percent decrease in muscle fiber areas for the E group for all of the fiber subtypes was significantly different from the C, ST, UC, and control groups.

Hormonal Data

Serum testosterone concentrations. Figure 2 presents the changes in serum testosterone during the graded treadmill test and the acute recovery (R) for each training group. The AUC analyses are shown in Fig. 3.

C GROUP. For the C group, increases in serum testosterone concentrations were significantly higher than the preexercise values at 75 and 100% $\dot{V}O_{2\,max}$ and 5 min of R for each training time point. At 12 wk, there was an increase above rest at 15 min after exercise. The testosterone concentrations at every time point in the *week 12* test were significantly higher than pretraining and 4- and 8-wk tests. At 12 wk, the AUC was significantly higher compared with any of the other training time points.

UC GROUP. For the UC group, increases in serum testosterone concentrations were significantly higher than the preexercise values at 100% $\dot{V}O_{2\,max}$ and 5 and 15 min of R for the pretraining and 4-wk training time points, at 75 and 100% $\dot{V}O_{2\,max}$ and 5 min of R for the 8-wk training time point, and at 75% $\dot{V}O_{2\,max}$ for the 12-wk training time point. No differences were seen in the AUCs at any training time point.

ST GROUP. For the ST group, increases in serum testosterone concentrations were significantly higher than the preexercise values at 100% $\dot{V}O_{2\,max}$ and 5 min of R for the pretraining time point, at 75 and 100% $\dot{V}O_{2\,max}$ and 5 min of R for the 4-wk training time point, at 75 and 100% $\dot{V}O_{2\,max}$ and 5 and 15 min of R for the 8-wk training time point, and at 100% $\dot{V}O_{2\,max}$ and 5 min of R for the 12-wk training time point. Again, no differences were seen in the AUCs.

E GROUP. For the E group, increases in serum testosterone concentrations were significantly higher than the preexercise values at 75 and 100% $\dot{V}O_{2\,max}$ and 5 and 15 min of R for all the training time points.

Serum cortisol concentrations. Figure 4 presents the changes in serum cortisol concentration during the graded treadmill test and the acute R for each training group. The AUC analyses are shown in Fig. 5.

C GROUP. For the C group, serum cortisol concentrations were significantly higher than preexercise values at 15 min of R for the 8-wk training time point and at 100% $\dot{V}O_{2\,max}$ and 5 and 15 min of R for the 12-wk test. Cortisol values at 100% $\dot{V}O_{2\,max}$ and 5 and 15 min of R at 8 wk were significantly higher than the corresponding pretraining time points. Cortisol values at 50, 75, and 100% $\dot{V}O_{2\,max}$ and 5 and 15 min of R for the 12-wk test were significantly higher than the corresponding

TABLE 6. *Muscle fiber characteristics pre- and posttraining*

	Combined Group		Strength Group		Endurance Group		Upper Body Combined Group		Control Group	
Fiber	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
					Percentage					
Type I	55.6±11.1	57.7±11.1	55.21±11.7	55.44±11.5	54.1±5.9	54.6±5.3	50.6±8.0	51.1±7.9	52.0±11.5	52.8±10.8
Type IIc	1.9±2.2	1.8±2.7	2.4±1.6	2.0±1.3	0.9±0.6	2.5±2.0*	1.3±1.0	3.0±2.2*	1.6±0.9	1.3±1.3
Type IIa	28.4±15.4	39.4±11.1*	23.3±11.5	40.5±10.6*	25.75±4.8	34.1±3.9*	25.5±4.2	34.2±6.9*	25.6±1.6	26.6±4.6
Type IIb	14.11±7.2	1.6±0.8*	19.1±7.9	1.9±0.8*	19.2±3.6	8.8±4.4*	22.6±4.9	11.6±5.3*	20.8±7.6	19.2±6.4
					Area, μm^2					
Type I	5,008±874	4,756±692	4,883±1,286	5,460±1,214*	5,437±970	4,853±966*	5,680±535	5,376±702	4,946±1,309	5,177±1,344
Type IIc	4,157±983	4,658±771	3,981.2±1,535	5,301±1,956*	2,741±482	2,402±352*	3,050±930	2,918±1,086	3,733±1,285	4,062±1,094
Type IIa	5,862±997	7,039±1,151*	6,084±1,339	7,527±1,981*	6,782±1,267	6,287±385	6,393±1,109	6,357±1,140	6,310±593	6,407±423
Type IIb	5,190±712	4,886±1,171	5,795±1,495	6,078±2,604	6,325±1,860	4,953±1,405	6,052±1,890	5,855±867	5,917±896	6,120±1,089

Values are means ± SD; $n = 9$ subjects for combined, strength, and upper body combined groups; 8 subjects for endurance group; and 5 subjects for control group. * $P \leq 0.05$ vs. corresponding pretraining value.

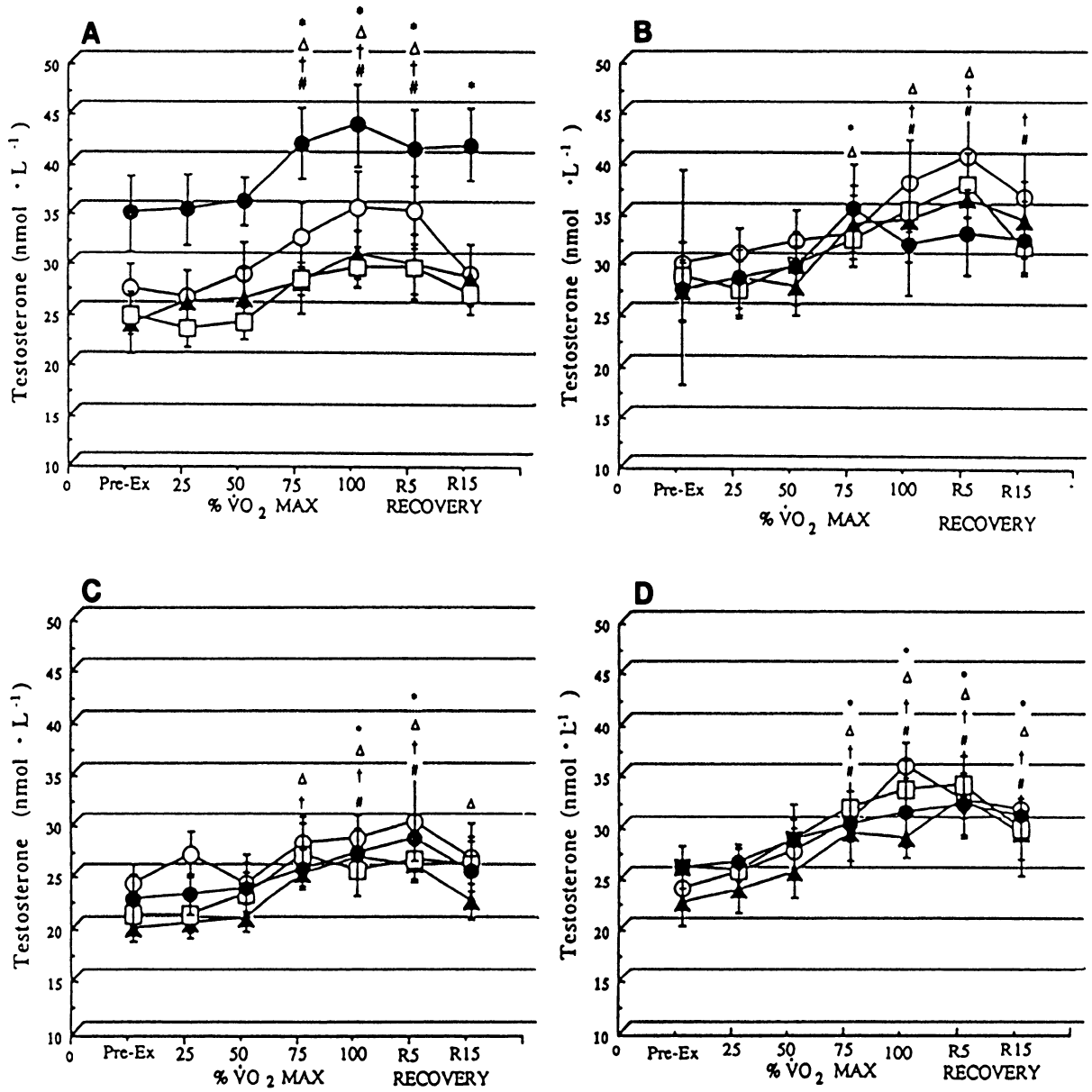

FIG. 2. Serum testosterone concentrations to graded treadmill exercise tests over training period for combined (*A*), upper body combined (*B*), strength-training (*C*), and endurance-training groups (*D*). Pre-Ex, preexercise; $\dot{V}O_{2\,max}$, maximal O_2 consumption; R5 and R15, 5 and 15 min of recovery, respectively. ○, Pretraining; ▲, 4 wk of training; □, 8 wk of training; ●, 12 wk of training. Values are means ± SE. $P \leq 0.05$ vs. corresponding preexercise value: # pretraining; † 4 wk; △ 8 wk; * 12 wk.

pretraining and 4-wk values at those time points. The 8- and 12-wk AUCs were significantly higher than the pretraining and 4-wk tests. The 12-wk AUC was also significantly higher than the 8-wk test.

UC GROUP. For the UC group, serum cortisol concentrations were significantly higher than the preexercise values at 15 min of R for all tests. Resting concentrations of cortisol were significantly higher at the 4- and 8-wk training time points compared with pretraining and 12 wk. In addition, serum cortisol concentrations at the 4-wk training time point were significantly higher than pretraining and 12-wk values at 25 and 50% $\dot{V}O_{2\,max}$. Eight-week values were greater than the pretraining and 12-wk values at 25% $\dot{V}O_{2\,max}$. All of the training time points at 75% $\dot{V}O_{2\,max}$ were significantly greater than the corresponding pretraining value. The 4- and 8-wk AUCs were significantly greater than the pretraining and 12-wk training time points.

ST GROUP. For the ST group, serum cortisol concentrations were significantly higher than the preexercise values at 5 and 15 min of R for the pretraining and 8- and 12-wk training time points. Increases were observed at 15 min of R for all of the training time points. Cortisol concentrations at 4 wk were significantly higher than those at the 8-wk training time point at rest, pretraining, and 8- and 12-wk at 25% $\dot{V}O_{2\,max}$; at 8 and 12 wk at 50% $\dot{V}O_{2\,max}$; and at 8 wk at 75 and 100% $\dot{V}O_{2\,max}$. Pretraining values were also greater than the 8-wk values at 100% $\dot{V}O_{2\,max}$. AUC values for the 8- and 12-wk training time points were significantly lower than the cortisol values at pretraining and 4 wk. The

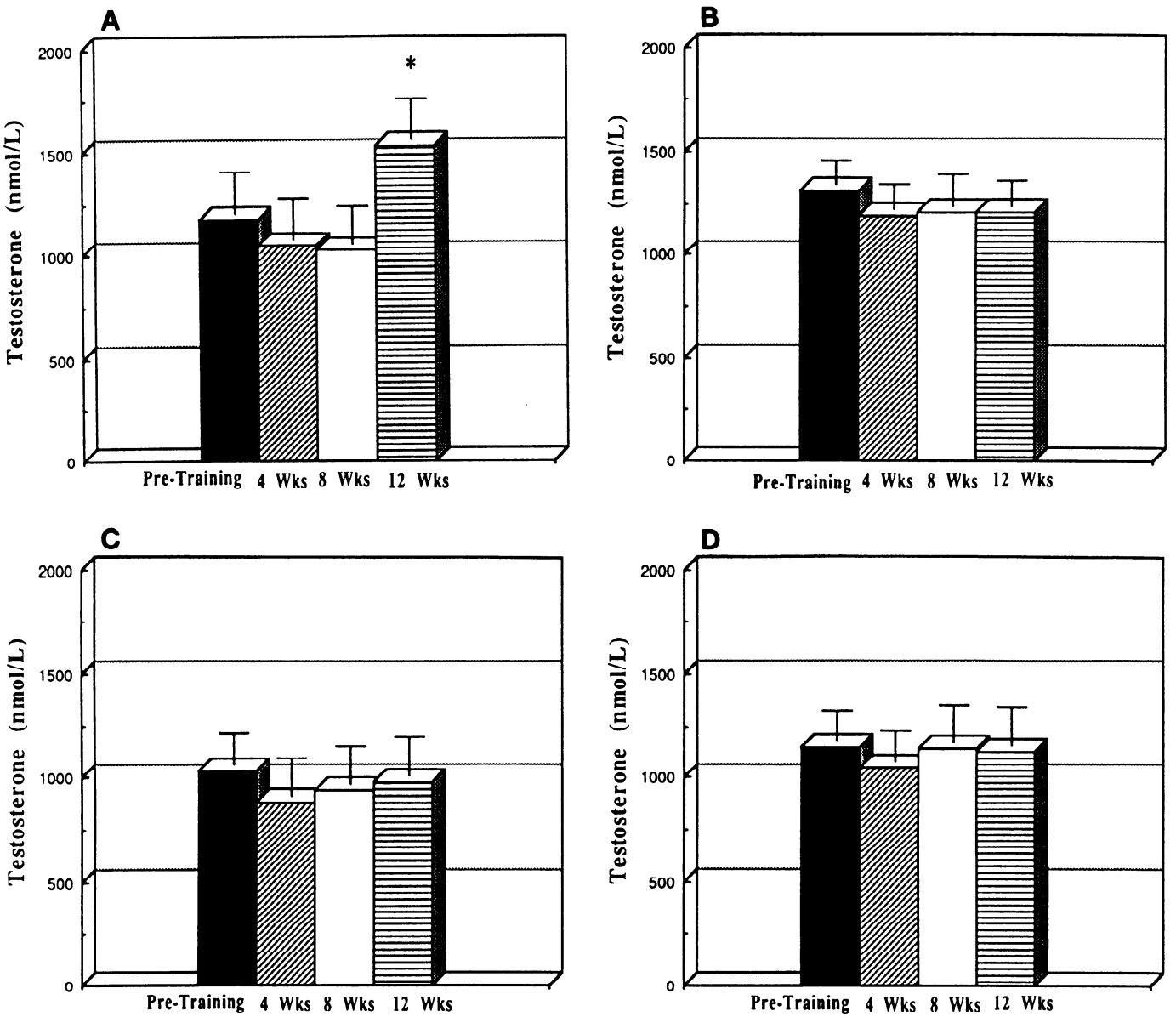

FIG. 3. Area under curve for serum testosterone concentrations to treadmill exercise and recovery (Pre-Ex to R15) over training period for combined (*A*), upper body combined (*B*), strength-training (*C*), and endurance-training groups (*D*). Values are means ± SE. * $P \leq 0.05$ vs. corresponding pretraining value.

AUC cortisol value at 8 wk was also significantly lower than that at 12 wk.

E GROUP. For the E group, serum cortisol concentrations were significantly higher than the preexercise values at 5 and 15 min of R for pretraining and 8- and 12-wk training time points. Cortisol values were significantly higher at rest and at 4 wk at 15 min of R. The preexercise cortisol concentration at 4 wk was significantly higher than the other training time points. Cortisol concentrations at 50 and 75% $\dot{V}O_{2\,max}$ were significantly lower than preexercise values at 4 wk. The 4- and 12-wk AUCs were significantly higher than the pretraining and 8-wk training time points.

DISCUSSION

The primary findings of this investigation were that the underlying hormonal and muscle fiber adaptations demonstrated a differential response to the training programs. It is proposed that these differential adaptations at the cellular level may help explain the subtle performance differences that were starting to emerge after only 12 wk of training. In this investigation, the subjects performed comprehensive high-intensity strength- and/or endurance-training programs that allowed us to examine the compatibility of programs used by many athletes and specialized military units (15). In addition, one group (UC) performed only upper body high-intensity strength training along with endurance training. Muscle strength and $\dot{V}O_{2\,max}$ increased in groups performing the independent training, but a possible attenuation of muscular power and some strength responses resulted when both forms of training were performed using the same musculature. The influence of simultaneous training on endurance performance remains unclear, as only a weak nonsignificant trend was

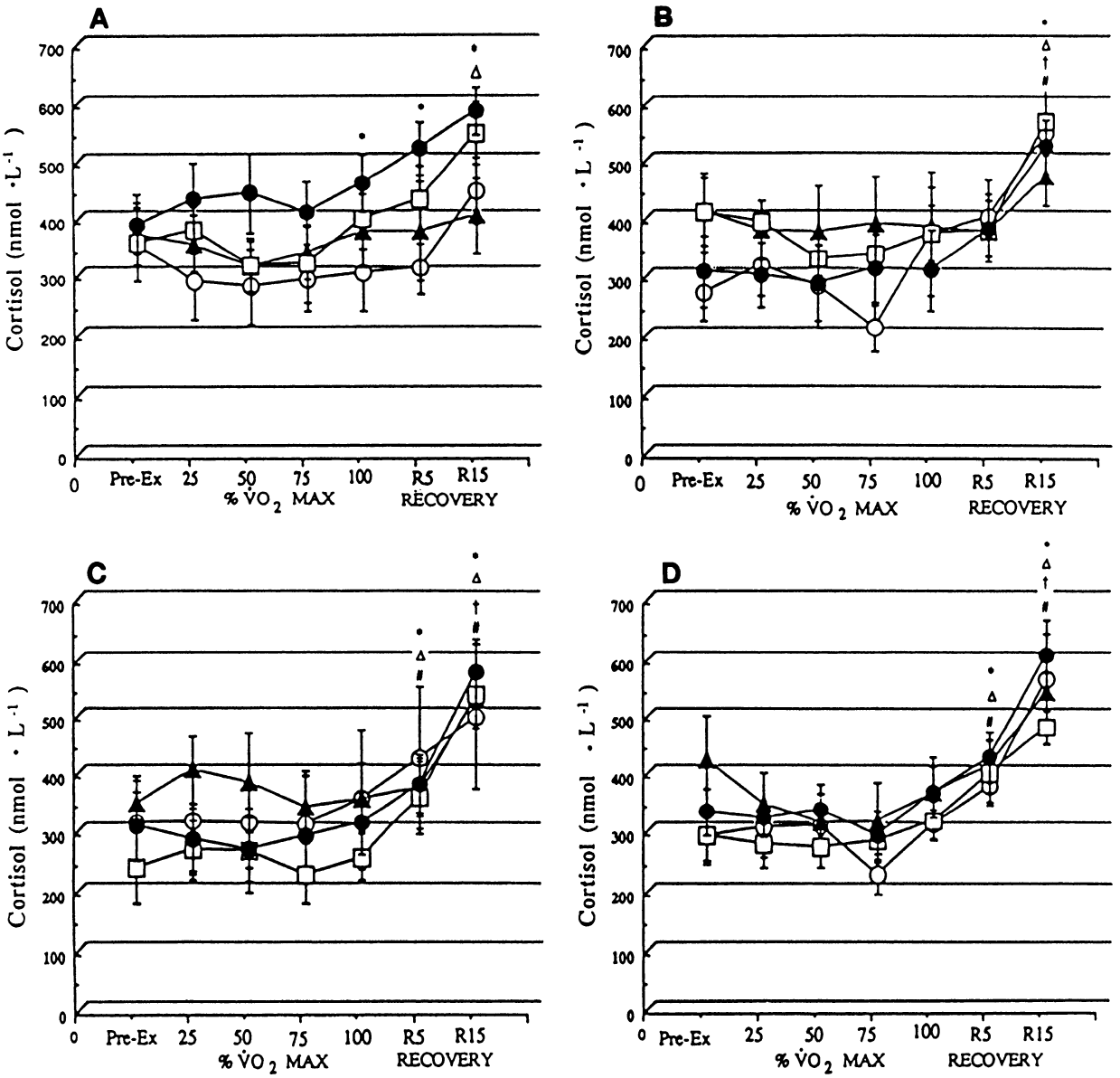

FIG. 4. Serum cortisol concentrations to graded treadmill exercise tests over training period for combined (*A*), upper body combined (*B*), strength-training (*C*), and endurance-training groups (*D*). ○, Pretraining; ▲, 4 wk of training; □, 8 wk of training; ●, 12 wk of training. Values are means ± SE. $P \leq 0.05$ vs. corresponding preexercise value: # pretraining; † 4 wk; △ 8 wk; * 12 wk.

observed for a lower percent increase in $\dot{V}O_{2\,max}$ for the C group compared with the E group. In addition, the effects of upper body strength training performed with endurance training (UC group) seem to be generally compartmentalized to the upper body musculature, as it did not significantly affect the force production or endurance capabilities of the lower body musculature. However, subtle differences were observed in muscle fiber and hormonal changes compared with those of endurance training alone.

Whether the combined training of the UC group might have compromised strength or power capabilities of the upper body is also of interest. Increases in 1-RM strength did occur in the UC group, and the improvement was not different from the ST or C groups. Simultaneous training appears to compromise strength improvement only when both modes of training engage the same muscle group. To our surprise, no changes were noted in the WAT for the arms in the UC group. Even though we have no explanation for these results, it does give an indication that it may again be physiological mechanisms related to power production that are most affected by high-intensity endurance training even in musculature that is not directly involved in the training. The mechanism for such a compromise remains unknown.

Power indexes, as measured by the WAT, demonstrated that combined training compromised power development. This may be due to a wide variety of factors differentially related to neuromuscular function (6, 11, 37). Our data extend the findings of Dudley and Djamil (10), who demonstrated compromises in isokinetic strength at higher velocities of movement with combined training. Thus, it may be that power develop-

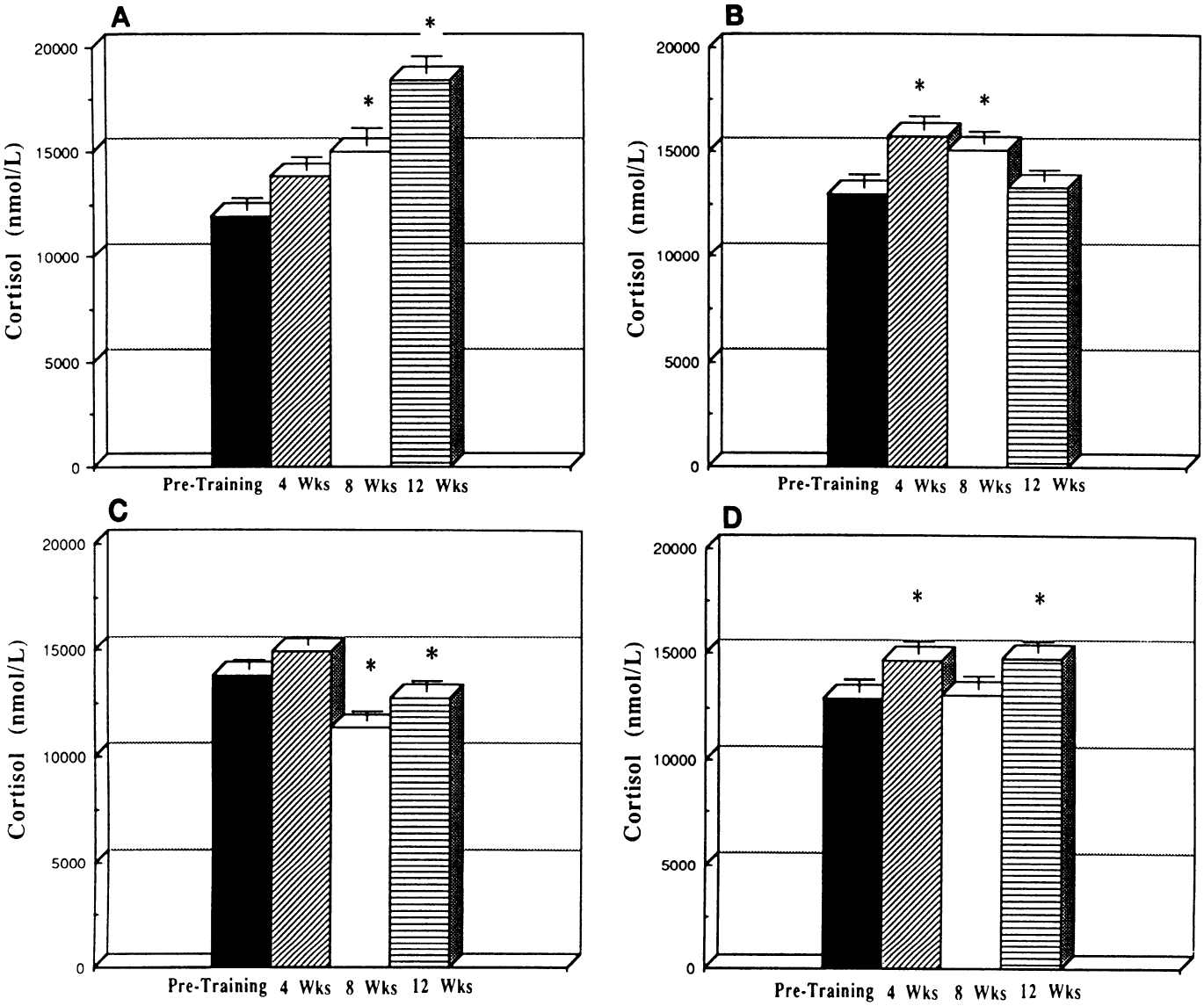

FIG. 5. Area under curve for serum cortisol concentrations to treadmill exercise and recovery (Pre-Ex to R15) over training period for combined (*A*), upper body combined (*B*), strength-training (*C*), and endurance-training groups (*D*). Values are means ± SE. * $P \leq 0.05$ vs. corresponding pretraining value.

ment is much more susceptible to the antagonistic effects of combined strength- and endurance-training programs than slow-velocity strength (6, 11, 17).

Changes in muscle fiber areas due to high-intensity strength training or high-intensity endurance training were attenuated when the training programs were performed simultaneously. These findings of size antagonism on the cellular level are unique. It appears that the type I and type II muscle fibers were differentially responsible for the endurance- and strength-training adaptations in the C group. Type I muscle fibers in the C group did not hypertrophy in response to the strength-training program nor did they decrease in response to the endurance-training program, as was observed in the ST and E groups, respectively. Such an intermediate response of the type I muscle fibers and the inability of the type II muscle fibers to apparently compensate for the needed magnitude of hypertrophy required for some 1-RM strength and power performances indicate support for the hypothesis that strength, power, and endurance performance decrements may be influenced to some extent over 12 wk of training due to differential muscle fiber adaptations. We also observed a decrease in the size of the type IIc muscle fiber areas in the E group and an increase in these fiber areas in the ST group that were not observed in the C group, again suggesting a compromising effect at the cellular level for both endurance and high force and power production capabilities. Whereas limited data are available on muscle fiber responses to simultaneous strength and endurance training, the two previous studies examining adaptations of muscle fiber areas are equivocal and did not demonstrate this differential training adaptation. Simultaneous training has been shown to either result in no changes or increases in type I and type II muscle fiber areas (34, 39). Our data support the concept that muscle fiber type area adaptations to simultaneous training differs from the single-training mode adaptations.

The mechanisms that mediate such differential ad-

aptations in muscle fiber areas remain speculative. In a recent study, Deschenes et al. (8) demonstrated soleus muscle fiber atrophy in endurance-trained rats. In addition, they observed differential alterations in the morphology of the neuromuscular junction (e.g., in the high-intensity group, more dispersed synapses and greater total length of branching) with different intensities of endurance training. Previous studies have also shown decreases in muscle fiber size in humans with endurance training (22, 44). Decreases in muscle fiber size and increased nerve cell branching and morphology may contribute to more optimal kinetics for oxygen utilization and innervation patterns promoting endurance capabilities (45). Conversely, such changes would be hypothesized to compromise muscle size and strength adaptations (6). The lack of change in type I muscle fiber areas and the sole increase in type IIa muscle fiber areas in *group C* appear to represent a cellular adaptation representative of the antagonism of simultaneous strength and endurance stimuli because strength training alone produced increases in both type I and type II muscle fiber areas. The use of only an upper body strength-training program also kept the size of the type I muscle fibers from decreasing due to endurance training. It is hypothesized that this might have been due to the isometric muscle actions of the lower body musculature utilized for stabilization during the upper body strengthening exercise movements. The subtle influence of such force development underscores the sensitivity of muscle fibers to resistance stimuli. In addition, no hypertrophy was observed in the other muscle fiber types even with high-intensity endurance run training. These data suggest that the subtle influence of isometric muscle actions used for stabilization when performing upper body resistance exercises in the UC group was not enough to create a hypertrophy stimulus. In addition, the already physically active status of our subjects may have eliminated the potential for any possible increases in muscle fiber areas, with only high-intensity endurance training (including SIs) as an overload stimulus.

Alterations in muscle fiber type percentages were observed with strength training even when performed in conjunction with endurance training, resulting in significantly greater reductions in type IIb muscle fibers. Thus, simultaneous training does not appear to affect the myosin heavy chain transformation of protein in strength-trained musculature. Due to the low number of fibers in the type IIb and IIc populations, one must look cautiously at these data, but a lack of change in area measurements in the type IIb fibers in each group and the alterations of type IIc fibers in selected groups may indicate a differential response to the exercise recruitment process. High-intensity endurance training did significantly reduce the percentage of type IIb muscle fibers but not to the extent that occurred when a strength-training stimulus was added to the program (i.e., posttraining type IIb fibers were 1.6 ± 0.8 and $1.9 \pm 0.8\%$ for the C and ST groups, respectively, compared with 8.8 ± 4.4 and $11.6 \pm 5.3\%$ for the E and UC groups, respectively). It might be hypothesized that the loads used in the strength-training program resulted in greater motor unit recruitment and therefore more muscle tissue was activated to perform the exercise. Recent studies have demonstrated a change from type IIb to type IIa muscle fibers similar to what was found in this study with high-intensity strength training (1, 42). It now appears that the type II muscle fiber subtype transition is initiated in the early phases of training (42), with the complete transition to type IIa fibers almost complete by 12 wk (i.e., 48 training sessions) of high-intensity strength training. Strength training appears to affect both the quality and quantity of contractile proteins, but only the quantity of contractile proteins appears to be affected by simultaneous training over 12 wk. The higher percentage of type IIb fibers in endurance-trained vs. strength-trained muscle, along with a lack of hypertrophy in the remaining type IIb fibers, supports the concept of a "reserve population" of type II fibers that, once recruited, start to make changes toward a type IIa fiber type (42). Data from this study suggest that even high-intensity endurance training does not recruit type IIb muscle fibers to the same extent as does heavy-resistance training.

We did not observe any alteration in the percentage of type I muscle fibers, but our type I muscle fiber percentage, which ranged from ~36 to 50%, was a bit higher than reported in typical untrained males (1, 10, 34, 39, 42). Patton et al. (35) showed that physically active soldiers may have a typical type I muscle fiber percentage of ~50%, or the upper limit of the untrained population. Sale et al. (39) observed an increase (~12–30%) in the percentage of type I muscle fibers after both endurance training and combined strength and endurance training. Nelson et al. (34) observed increases in the percentage of type I muscle fibers only in a combined strength- and endurance-training group. The pattern of results observed in this study may be due to the high aerobic fitness of the individuals at the start of the study due to their prior physical training. It is also possible that the lighter (i.e., 15–20 RM and isokinetic training) strength-training loads in the other studies contributed to greater fiber changes in the type I population (39). We did observe increases in the type IIc fiber type percentage when endurance training (E and UC groups) was performed. This change did not occur when combined training (C group) was performed, again pointing to a possible incompatibility for optimizing endurance mechanisms when both forms of training are performed together over a 12-wk training program.

The pattern and time course of changes in the hormones provide some insights into hormonal influences on cellular adaptations of muscle that ultimately influence performance changes. Whereas these data only examine the circulating peripheral alterations, a stronger case may now be made for further study at the molecular level to better understand the hormonal mechanisms of protein metabolism. In the ST group, testosterone increased in response to exercise stress, but no changes were observed in the resting or exercise-induced concentrations over the course of the training program. Of greater importance in understanding the

enhanced anabolic environment was the concomitant decrease in the exercise-induced response of cortisol by the 8th wk of training, thus producing an enhanced anabolic environment due to the enhanced testosterone-to-cortisol ratio for total exposure (i.e., AUC). Testosterone and cortisol are representative of anabolic and catabolic hormones in the body and have been used to reflect training adaptations of the endocrine system (16, 23). The training programs produced a different hormonal environment for muscle and nerve cells over their course. Such differences in the hormonal environment can influence the cellular changes related to protein synthesis, neurotransmitter synthesis, and subsequent muscle fiber adaptations as well as substrate utilization and endurance capabilities (16, 23, 25). Alterations in resting testosterone and cortisol concentrations in untrained men in the early phase of a resistance-training program have been observed (42). The present study demonstrated that varied strength-training programs using higher volumes of exercise may be needed to alter resting concentrations because previous studies have not demonstrated alterations (19, 25). However, too much exercise may result in an undesirable increase in cortisol, as observed in the C group, which might compromise muscle strength, power, and size gains.

The E group demonstrated no significant changes in resting or exercise-induced testosterone concentrations with training but did show an increased total cortisol exposure (i.e., AUC) response at 4 and 12 wk, suggesting that the progressive high-intensity endurance-training program was at least creating a greater adrenal cortical response to exercise stress at certain times of the training program (e.g., acute stress response and a later chronic response) than strength training alone. In general, a decrease in cortisol has been observed with high-intensity strength training, whereas an increase has been attributed to high-intensity sprint training (25, 28, 42). Because cortisol has been associated with protein degradation mechanisms, the increased amounts of cortisol in the face of no changes in testosterone could influence the reductions in cell size noted in the types I and IIc muscle fibers (16, 23).

The combination of both forms of training resulted in dramatic and stepwise increases in the exercise-induced and total cortisol exposure (i.e., AUC) responses. This preceded a large increase in the exercise-induced and total testosterone exposure (i.e., AUC) at the end of the 12-wk training program. The dramatically different response of both cortisol and testosterone to the simultaneous training suggests that the increased total work may have resulted in a type of "overtraining" response, at least at the level of the hypothalamic-pituitary-adrenal axis, by 8 wk. The increased cortisol along with associated increases in catecholamine production (unpublished data) help explain the dramatic increases observed in testosterone after 12 wk of training (16). However, due to the fact that the total cortisol response in *week 12* was even higher than that in *week 8*, how successful the concomitant and large testosterone response (i.e., increase in resting, exercise-induced, and AUC total exposures) would be in offsetting continued catabolic effects remains speculative. Nevertheless, muscle fiber size, power, and strength adaptations were all somewhat compromised by 12 wk of training. Again, due to the measurement of variables every 4 wk, it is not possible to study the day-to-day time course of these events that culminated after 12 wk of training. Thus, the exposure time of these hormones at target tissues from *weeks 8* to *12* remains unknown. The influence of such a dramatic increase in endogenous testosterone on physiological and performance variables (e.g., supercompensation) with further training remains unknown. However, it appears that a reduction in training volume would be needed to create an environment where an anabolic rebound in muscle size, strength, and/or power could continue to increase and overtraining would be avoided (17, 18).

Thus, incompatibility of training may be attributed to a large extent to the extreme stress of adrenal activation due to the total amount of high-intensity exercise. Whether successful adapations can occur remains dependent on the ability of various anabolic compensatory mechanisms (e.g., testosterone, insulin-like growth factors, growth hormone) to eventually override a catabolic environment (15, 23). This ability to overcome the catabolic environment was in part demonstrated by the UC group that performed the upper body strength-training program along with the endurance-training program. By *week 12*, the UC group demonstrated a total cortisol exposure response (i.e., AUC) that was no different from the pretraining level. Not performing the lower body strength-training program resulted in a reduction in the total work that was associated with the program. Similar to the ST and E groups, no changes occurred in the concomitant testosterone response over the 12 wk of training. Even though no decrease or increase in the testosterone-to-cortisol ratio was observed, the training did not enhance the catabolic environment and may again have influenced the lack of changes in types I and IIc muscle fiber areas. Unfortunately, data on the impact of a controlled reduction in the volume of total work and its effects on muscle undergoing the simultaneous training are not directly available from this study. Still, such data and previous studies have indicated that total work stress may be a potentially significant factor in the development of incompatibility of exercise training (6, 11). This concept is now supported from an endocrine perspective.

Our data indicate that single-mode training tends to be the most effective for strength or endurance performance and its concomitant muscle fiber changes. Similar to other studies in the literature, the exercise programs utilized caused the C group to increase both 1-RM strength and $\dot{V}O_{2\,max}$ performance capabilities (2, 20, 21, 39). However, the C group increased strength by a smaller percentage than did the ST group in the leg press and also increased $\dot{V}O_{2\,max}$ by a smaller (but not significant) percentage than did the E group. As demonstrated in all of the previous studies, the impact of simultaneous training appears to be more detrimental to potential strength and power gains and not to $\dot{V}O_{2\,max}$.

It is interesting to note that the percent improvement observed in this study for the leg press was greater in the ST group compared with that in the C group, but no differences were observed for the percent improvement in the double leg extension exercise. These data indicate that incompatibility may also be a function of the type of movement being tested (single vs. multiple joint).

Simultaneous increases in both 1-RM strength and $\dot{V}O_{2\,max}$ could be attributed to a number of design features including three complete rest days within each training week and periodized training programs within the week (15). Nevertheless, because we only evaluated 1-RM strength and $\dot{V}O_{2\,max}$ every 4 wk, it is possible that we missed transient decreases between *weeks 8* and *12*. We also had the ability to better control other stress-related factors (e.g., schedule overloads, class pressures, job-related stress as this was their job, etc.) that may otherwise have contributed to overtraining manifesting itself as incompatibility of training programs (15). Programs that utilize higher training frequencies, longer training periods, reduced rest, and/or potential stressors from other sources may show greater incompatibility for enhancing both strength and endurance performance (17, 18, 34).

In summary, our data indicate that, when performed singly, endurance and strength training elicit adaptations in muscle fiber morphology and serum hormones that are different from those induced by concurrent strength and endurance training. Combining strength and endurance training attenuates the muscle fiber hypertrophy produced by resistance training alone and produces increases in cortisol that enhance the catabolic environment. Conversely, strength training alone promotes reductions in cortisol that enhance the testosterone-to-cortisol ratio. The simultaneous strength and endurance training produced smaller muscle strength and power increases than strength training alone. Whereas endurance improvements were lower when performing both modes of training with the same musculature, our data did not support a significant reduction in endurance performance or percent improvement when strength training was added to the endurance-training program. Finally, the observed incompatibility of strength and endurance training may be due, at least in part, to some type of overtraining, a possibility that warrants further investigation.

The authors thank the following people from both the US Army Research Institute of Environmental Medicine and the Center for Sports Medicine at Pennsylvania State University for help in data collection, laboratory biochemistry, test subject supervision, technical support, data analyses, research support, manuscript preparation, and a wide range of logistical support activities for a very busy project: Louis Marchitelli, Robert Mello, Peter N. Frykman, Dan Jones, Jeff Crawshaw, Chuck Cruthirds, John Hallmark, Pat Fitzgerald, and Duncan Muffles. We also thank a very dedicated group of soldiers who acted as test subjects in this investigation and without whose hard work and dedication this project would not have been possible. We also thank Dr. James A. Vogel for support of this line of research in soldier preparation and combat readiness. We also thank Dr. Keijo Häkkinen for insightful and helpful comments on the manuscript while a visiting professor in W. J. Kraemer's laboratory in the Center for Sports Medicine.

The preparation of this manuscript was supported in part by a grant from the Robert F. and Sandra M. Leitzinger Research Fund in Sports Medicine at The Pennsylvania State University.

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Received 1 June 1994; accepted in final form 27 October 1994.

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