Neuromuscular adaptations to concurrent strength and endurance training

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ABSTRACT

McCARTHY, J. P., M. A. POZNIAK, and J. C. AGRE. Neuromuscular adaptations to concurrent strength and endurance training. *Med. Sci. Sports Exerc.*, Vol. 34, No. 3, pp. 511–519, 2002. **Purpose:** The purpose of this study was to examine muscle morphological and neural activation adaptations resulting from the interaction between concurrent strength and endurance training. **Methods:** Thirty sedentary healthy male subjects were randomly assigned to one of three training groups that performed 10 wk of 3-d·wk^{-1} high-intensity strength training (S), cycle endurance training (E), or concurrent strength and endurance training (CC). Strength, quadriceps-muscle biopsies, computed tomography scans at mid-thigh, and surface electromyogram (EMG) assessments were made before and after training. **Results:** S and CC groups demonstrated similar increases ($P < 0.0001$) in both thigh extensor (12 and 14%) and flexor/adductor (7 and 6%) muscle areas. Type II myofiber areas similarly increased ($P < 0.002$) in both S (24%) and CC (28%) groups, whereas the increase ($P < 0.004$) in Type I area with S training (19%) was also similar to the nonsignificant ($P = 0.041$) increase with CC training (13%). Significant increases ($P < 0.005$) in maximal isometric knee-extension torque were accompanied by nonsignificant ($P \leq 0.07$) increases in root mean squared EMG amplitude of the quadriceps musculature for both S and C groups. No changes ($P > 0.38$) in the EMG/torque relation across 20 to 100% maximal voluntary contractions occurred in any group. A small 3% increase ($P < 0.01$) in thigh extensor area was the only change in any of the above variables with E training. **Conclusions:** Findings indicate 3-d·wk^{-1} concurrent performance of both strength and endurance training does not impair adaptations in strength, muscle hypertrophy, and neural activation induced by strength training alone. Results provide a physiological basis to support several performance studies that consistently indicate 3-d·wk^{-1} concurrent training does not impair strength development over the short term.

Key Words: HYPERTROPHY, MUSCLE FIBERS, CT SCAN, EMG, RESISTANCE EXERCISE

Strength and power development has been impaired when endurance training is added to strength training (4,11,21–23,25). Other investigators, however, report no interference in strength development with concurrent strength and endurance training over a short term (1,14,27,31,34,38). Studies investigating the interaction of these two diverse types of training, however, provide strong evidence that concurrent training does not impair endurance development as measured by maximal aerobic power (21,22,25,27,34). It thus appears, that with concurrent strength and endurance training, the major consideration is that of endurance training possibly interfering with the neuromuscular system's ability to generate maximal force.

Although a considerable number of studies have addressed performance adaptations with concurrent training, only a few have attempted to address underlying physiological mechanisms responsible for impairment in strength development. Muscle hypertrophy and changes in motor unit recruitment are two of the most salient factors associated with strength development (16). Kraemer et al. (25) indicate an impairment in hypertrophy of Type I fibers when endurance training is added to strength training. Bell et al. (4) reported no increase in Type I fiber area with concurrent strength and endurance training, but there were no differences in fiber type area adaptations (both Type I and II) compared with the same type of strength training performed alone. With concurrent training, Nelson et al. (31) reported hypertrophy in Type I, IIa, and IIb fibers, whereas the strength-only training employed in this study resulted in hypertrophy of only the Type IIb fibers. None of these studies considered muscle adaptations at the macroscopic or whole muscle level as may be assessed with imaging technology. Sale et al. (34) reported similar muscle area increases with both strength-only and concurrent training as measured at both the microscopic (Type I and II fibers) and macroscopic (quadriceps area as assessed using computed tomography) levels. It is evident that the pattern of specific fiber type hypertrophy with concurrent training is inconsistent in the few investigations addressing this question and little is known about adaptations at the whole muscle level (26).

With strength training there are several different lines of evidence that indicate adaptations within the nervous system occur that are related to strength development (33). One of the major areas of investigation with strength training focuses on adaptations in motor unit activation in prime movers as measured by electromyography (EMG) (30,33). The integrated EMG signal attained during a maximal isometric contraction has been shown to increase in several longitudinal strength-training studies (16,17,29), although other investigations report no change in this measure (13,30,37). It has been suggested that an impairment in

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force development with concurrent training, as compared with strength-only training, may be related to altered neural activation associated with maximal voluntary contractions (6,25,26). The relatively small number of maximal or near maximal contractions involved in strength training demand different patterns of motor unit activation than the fairly continuous low level contractions involved with endurance training. No studies to date have investigated the effects of concurrent training on neural activation of prime movers.

To investigate major mechanisms associated with changes in strength performance, the purpose of this study was to evaluate the influence of 3-d·wk^{-1} concurrent strength and endurance training on muscle morphology and neural activation in previously sedentary individuals. We tested hypotheses that the diversity of demands of traditional endurance and strength training on skeletal muscle would limit muscle hypertrophy at the macroscopic (whole muscle) and microscopic (myofiber) levels. In addition, we tested the hypothesis that concurrent-training adaptations are different than any changes that may occur with strength-only training in neural activation of muscle.

METHODS

Subjects and experimental design. The data were collected as part of a larger study that also examined the effect of concurrent training on body composition, $\dot{V}O_{2peak}$, and strength performance (27). Thirty sedentary healthy men, who had not exercised regularly for at least 3 months before the start of the study, served as subjects. After approval from the Health Sciences Human Subject Committee at University of Wisconsin-Madison, all subjects were informed of the procedures, risks, and benefits, and provided written consent before participation. Each subject was screened via a medical history questionnaire and a physical evaluation given by a physician. Measures of vastus lateralis fiber area and type distribution (from muscle biopsies), thigh cross-sectional muscle area (from computerized tomography [CT] scans), knee-extension isometric torque and associated quadriceps RMS-EMG amplitude across 20–100% maximal voluntary contractions (MVC) were taken before and after 10 wk of training. After all pretraining measurements, subjects were randomly assigned to either a strength-only (S), an endurance-only (E), or a concurrent (CC) strength- and endurance-training program. Characteristics of subjects in the three groups are presented in Table 1. Sample size was estimated as described by Cohen (7) for paired *t*-tests and analysis of variance (ANOVA) (see Statistical Analysis section) by using the variable of maximal isometric torque and data from a pilot study (27). To detect an effect size difference of 15 N·m for paired *t*-tests with $\alpha = 0.0167$, the power $(1\text{-}\beta) = 81$; and for ANOVA with $\alpha = 0.05$, the power = 76. All subjects proceeded through all testing procedures with one exception. One subject in the CC group did not have muscle-biopsy samples taken.

Training. Conventional high-intensity strength- and continuous-endurance-training regimens were employed in this study and have been previously described in detail (27). All subjects completed 10 wk of 3-d·wk^{-1} exercise training on alternate days. The S group performed eight weight-training exercises for one warm-up set and three maximal-effort sets. The goal for number of repetitions throughout the training was six (range 5–7). After the warm-up set in each exercise, subjects performed as many repetitions as possible until muscular failure (could not perform another full repetition). If a set was performed outside of the range of 5–7 reps, the load of subsequent sets was adjusted accordingly. The goal was always to perform a 6-repetition maximum (6RM); thus, progression was always incorporated into this program. Warm-up sets were performed with two-thirds of the load used to perform a 6RM. During the first week of training, starting weights were determined by trial and error. Each exercise utilized low contraction velocities with subjects performing concentric and eccentric phases of repetitions in approximately 1–2 s each, with the last repetition of a set generally the slowest with subjects close to muscular failure. Rest between maximal sets was approximately 75 s (60–90 s). All sets of each exercise were performed in succession before moving to another exercise. Exercises performed using barbells included parallel squats, bench presses, and standing curls. Exercises performed using plate loaded machines (Badger Fitness Equipment, South Milwaukee, WI) included knee extensions, leg curls, wide-grip lat pull-downs, overhead presses, and heel raises.

TABLE 1. Subject characteristics by group.

Group	*N*	Age (yr)	Height (cm)	Weight (kg)
Strength	10	27.9 ± 1.2	180.4 ± 1.3	82.0 ± 4.4
Endurance	10	26.5 ± 1.6	179.3 ± 2.7	84.5 ± 5.5
Concurrent	10	27.3 ± 1.7	179.2 ± 1.5	82.1 ± 4.3

Values are means ± SE; *N*, no. of subjects.

The E group performed 50 min of continuous cycle ergometry at an intensity of 70% heart rate reserve. The first 5 min of exercise served as a warm-up and was performed at two-thirds of the normal training workload of the following 45 min. S and E programs were designed to elicit substantial improvements in either strength or aerobic capacity, and both were designed to require substantial involvement of the knee extensor muscle group. The CC group completed both S and E programs in the same training session. Order of S and E training was rotated each training day with a 10- to 20-min rest period between training modes. S training was performed in its entirety before proceeding to endurance training and *vice versa*. All training was closely supervised and monitored by investigators. Compliance to training was high with subjects in each training group completing an average of 30 of 31 training sessions. No more than two training sessions were missed by any of the subjects.

Computerized tomography. Cross-sectional area (CSA) of the dominant knee extensor and flexor/adductor muscles were measured using a computed tomographic (CT) technique. Scans were obtained on a DR3 CT scanner (Siemens, Erlangen, Germany). Scans were taken while subjects were in the supine position with the thighs relaxed. Radiation exposure was minimized by low-exposure factors (125 kVp, 3 s, 180 mAs). A single-axial transverse image

was obtained through the thigh at a level 18 cm above the superior border of the patella.

All CT scans were blinded and analyzed by the same individual. Cross-sectional areas were digitized using an image processing and analysis system. A COHU video camera (no. 6415-2000) with a Javelin 18- to 108-mm macro zoom lens was interfaced with a Sony 1343 monitor and a microcomputer equipped with a PC Vision Plus frame grabber board (Jandel Scientific, Corte Madera, CA). JAVA (Jandel Video Analysis Software; Jandel Scientific) computer-enhanced tracing and digitizing software was used for the analysis. From CT images, thigh muscles were divided into extensor and flexor/adductor compartments as follows. On the lateral side, the dividing line was between the vasti anteriorly, and the hamstrings and adductor magnus posteriorly, extending to the posterior border of the femur. From the posterior border of the femur, the line was extended on the medial side between the vastus medialis anteriorly, and adductor longus and sartorius posteriorly. The femur bone was included in the initial extensor area but was measured separately and subtracted out to yield only the muscle area within the extensor compartment. Muscles included in the extensor area were limited to the four quadriceps. The flexor/adductor area contained the hamstrings, sartorius, gracilis, and adductor magnus and longus muscles. Coefficients of variation were calculated on 16 random samples redigitized for extensor, flexor/adductor, and bone areas. The coefficient of variation was less than 1% for each of these areas.

Muscle biopsy. Biopsy specimens were removed from the vastus lateralis muscle of the dominant leg by percutaneous needle biopsy as previously described (10). Muscle samples were oriented cross-sectionally using a dissecting microscope, mounted on cork by using Gum Tragacanth, and then quickly frozen in isopentane cooled with liquid nitrogen. Tissue was stored at −70°C until analyzed.

Frozen muscle tissue was serial sectioned in 10-μm sections on a Reichert Histostat cryostat microtome at −20°C. Myofibers were classified as slow twitch (ST, or Type I) or fast twitch (FT, or Type II) by the myofibrillar adenosine triphosphatase reaction at pH 9.4, after preincubation at pH 10.4 (15). Pre- and post-experimental muscle samples on the same subject were processed simultaneously to avoid differences attributable to the procedure.

For planimetric measurements of the cross-sectional myofiber area, a Houston Instruments computerized digitizing tablet was attached to an IBM microcomputer. Muscle sections were projected at 200 times original size on a Nikon Optiphot microscope, equipped with a zoom drawing tube. A Lovins Micro-slide Field Finder (Teledyne Gurley, Troy, NY) was used to verify projection accuracy. Myofiber perimeters were manually traced and Bioquant software (Biometrics, Nashville, TN) was used to calculate fiber areas. Myofibers selected for area measurement were without longitudinal tendencies, had distinct cell borders, were free of artifacts, and were centrally located in the sample. Samples were blinded to protect the investigator from knowledge of treatment group and initial or final biopsy status.

Myofiber distribution (% fiber types) was determined from samples containing an average (±SD) of 718 ± 153 fibers (range 402–1045). From the samples, 152 ± 2 fibers (range 146–158) were traced to measure fiber area. There were 64 ± 13 (range 51–100) ST and 87 ± 12 (range 51–100) FT fibers used to compute mean areas. In addition to the areas for Type I and Type II fibers, mean fiber area (MFA) was calculated to account for fiber distribution as follows: MFA = [(area of Type I fibers × percent of Type I fibers) + (area of Type II fibers × percent of Type II fibers)]/100. FT/ST area ratio was calculated by dividing FT fiber area by ST fiber area.

Isometric torque and neural activation. After submaximal warm-ups, maximal voluntary isometric knee-extension torque in the dominant leg was determined at a 0.52 rad angle below horizontal using a LIDO Active isokinetic loading dynamometer (Loredan Biomedical, Inc., Davis, CA) as previously described (27). Three maximal 5-s isometric contractions were performed with 3-min rest intervals between each contraction to prevent fatigue. The contraction with the highest torque value was used in data analysis. We have previously shown this method to be highly reliable with an intraclass correlation coefficient of 0.99 between different days (27). For determining the EMG/torque relation, submaximal isometric torque levels were then registered in random order at 20% intervals between 20 and 80% of MVC. For posttesting, the same relative (i.e., same percentages of post MVC) submaximal torques were used in assessments. The same order of performing the submaximal contractions was maintained pre- to posttesting for each subject. Each submaximal contraction was held at the desired torque level for 5 s with 2-min rest intervals between each contraction. To maintain torque at the required level, each subject matched his generated torque to the target level imposed on a computer screen linked with the dynamometer.

During isometric knee-extension contractions electromyographic (EMG) activity was recorded from the vastus medialis muscle by using bipolar skin surface electrodes by methods we have previously described (32). Consistency in electrode placement was maintained pre- to post-test for each subject (32). Skin impedance was kept below 5000 Ω for all tests by standard skin preparation. Raw EMG was full-wave rectified and converted to root mean squared (RMS) by a microprocessor linked to the electromyograph (Tracor Northern, Middleton, WI) and then to a strip chart recorder (Gould TA 550 Strip Chart Recorder, Eastlake, OH) to record the RMS-EMG signal. We have previously shown measures of maximal RMS-EMG amplitude to be reliable with this method with an intraclass correlation coefficient of 0.82 as assessed with repeated tests over a 12-month interval (32). In recording only from the vastus medialis, we are assuming that the behavior of RMS-EMG amplitude in this muscle is representative of all knee extensors. The EMG/torque relation was investigated by measuring the RMS-EMG amplitude during isometric contractions at 20, 40, 60, 80, and 100% of MVC.

Statistical analysis. Group scores are reported as means ± SE. Magnitude of changes produced by training in the three groups was compared using a one-way analysis of variance (ANOVA) on the difference (post minus pre) scores. The level of statistical significance was set at 0.05. *Post hoc* Fisher least significant difference tests were employed to locate specific significant differences between groups. Effects of training within each group were assessed using Dunn's multiple comparison procedure incorporating the Bonferroni correction to maintain the family-wise Type I error rate at 0.05 (24). By using the Bonferroni correction, the 0.05 significance level was divided by three (three *t*-tests), yielding a Type I error rate of 0.0167 for each *t*-test.

Statistical analysis for the EMG/torque relation for the five levels of isometric torque (20, 40, 60, 80, and 100% MVC) was investigated via linear trend analysis across EMG/torque data points (20). Relevant planned comparisons were tested among the six trends (pre- and post-training for each of the three group).

RESULTS

Thigh extensor area increased ($P < 0.0001$) after training in both S (12%) and CC (14%) groups (Fig. 1). A smaller increase ($P < 0.011$) occurred with E (3%) training, which was significantly smaller ($P < 0.0001$) than changes in the S and CC groups. Thigh flexor/adductor area increased ($P < 0.01$) in both S (7%) and CC (6%) groups but did not change in E (0%, $P = 0.924$). Femur bone cross-sectional areas did not change ($P > 0.307$) pre- to post-training in any group (S: 8.8 ± 0.3 to 8.9 ± 0.3 cm^2; E: 9.0 ± 0.4 to 9.0 ± 0.3 cm^2; and CC: 9.2 ± 0.4 to 9.3 ± 0.5 cm^2).

Myofiber areas and distribution results are presented in Table 2. For muscle fiber distribution, there were no changes ($P > 0.273$) pre- to post-training. The only significant finding in Type I fiber area was a pre to post increase in the S group (18.5%, $P = 0.004$). The pre to post increase in the CC group (12.5%) approached significance ($P = 0.041$). The reader is reminded that due to the application of the Bonferroni procedure (to maintain Type I error rate at 0.05), the criterion for finding a difference within a group was more stringent than the criterion for finding a difference between groups. The same pattern of findings occurred in both Type II fiber area and in mean fiber area. Both S and CC groups had similar increases ($P < 0.002$) in Type II fiber area (24 and 28%) and in mean fiber area (21 and 23%). These increases were significantly greater than the nonsignificant increases seen in the E group (Type II fiber area = 4.5%, $P = 0.186$; and mean fiber area = 3.9%, $P = 0.167$). Increases in FT/ST area ratio approached significance in S (5%, $P = 0.066$) and CC (15%, $P = 0.044$) groups. FT/ST area ratio showed no change in the E group (0%). There were no significant differences in changes in FT/ST area ratio between groups ($P = 0.081$).

Knee-extension isometric torque results across 20–100% MVC are paired with associated quadriceps RMS-EMG amplitudes in the EMG/torque relation curves presented in Figure 2. There were no significant differences among linear trends pre to post training ($P > 0.384$) or between groups ($P = 0.776$). This indicates that for a given level of torque output there was no change in level of neural activation. Training-induced increases in maximal RMS-EMG amplitude approached significance in the S (13.5%, $P = 0.044$) and CC (10.6%, $P = 0.073$) groups (Fig. 2). The smaller nonsignificant ($P = 0.639$) increase in the E group (5.0%) was not significantly different than changes in the other groups. As we reported previously, maximal knee-extension isometric torque increased similarly in both S (12%, $P = 0.004$) and CC (7%, $P = 0.003$) groups but did not change with E (−2%, $P = 0.223$) training (27). Increases in S and CC groups were significantly greater ($P = 0.0006$) than changes in the E group.

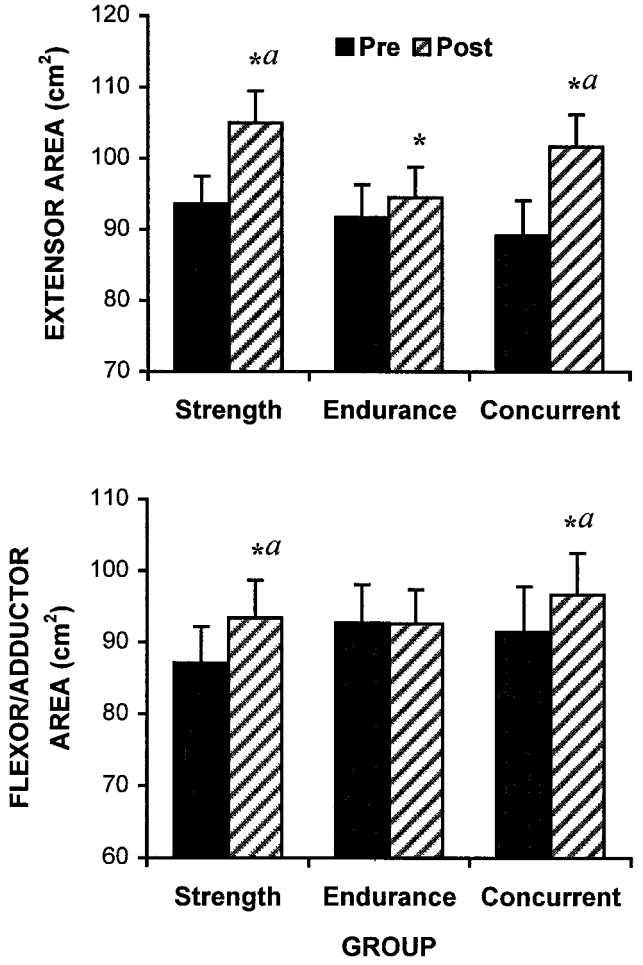

FIGURE 1—Thigh extensor (top) and flexor/adductor (bottom) muscle cross-sectional area. Values are means ± SE *Within group, post significantly different from pre; $P < 0.0167$. [a]Change significantly different from endurance group; $P < 0.05$.

DISCUSSION

Our focus on investigating neuromuscular mechanisms related to strength development with 3-d·wk^{-1} concurrent strength and endurance training revealed similar findings in all variables in both S and CC groups. Maximal isometric torque increased in both groups (S = 22 and CC = 14 N·m)

TABLE 2. Myofiber areas and distribution.

Variable/Group	Pretest	Posttest
Percent Type I fibers		
Strength	32.9 ± 3.5	31.8 ± 2.7
Endurance	34.5 ± 3.2	37.9 ± 4.6
Concurrent	37.5 ± 5.6	39.5 ± 6.7
Percent Type II fibers		
Strength	67.1 ± 3.5	68.2 ± 2.7
Endurance	65.5 ± 3.2	62.1 ± 4.6
Concurrent	62.5 ± 5.6	60.5 ± 6.7
Type I area (μm^2)		
Strength	4429 ± 222	5249 ± 259*
Endurance	4661 ± 395	4863 ± 324
Concurrent	4279 ± 452	4816 ± 506
Type II area (μm^2)		
Strength	4975 ± 399	6181 ± 286*[a]
Endurance	5523 ± 443	5772 ± 418
Concurrent	4422 ± 469	5651 ± 528*[a]
Mean fiber area (μm^2)		
Strength	4843 ± 338	5874 ± 233*[a]
Endurance	5203 ± 389	5404 ± 346
Concurrent	4331 ± 448	5307 ± 485*[a]
FT/ST area ratio		
Strength	1.13 ± 0.08	1.19 ± 0.07
Endurance	1.20 ± 0.05	1.20 ± 0.07
Concurrent	1.04 ± 0.05	1.20 ± 0.05

Values are means ± SE.
* Post significantly different from pre ($P < 0.0167$).
[a] Change significantly different from endurance ($P < 0.05$).

without an accompanying significant increase in maximal RMS-EMG amplitude. No changes in the EMG/torque relation across 20–100% MVC occurred indicating the level of neural activation remained the same for a given level of torque output in all groups. At the gross muscle level, quadriceps area (11.4 and 12.6 cm^2) and flexor/adductor area (6.2 and 5.2 cm^2) increased similarly in both S and CC groups. At the myofiber level, there was no change in fiber type distribution or in the FT/ST area ratio in any group. Substantial and similar levels of hypertrophy occurred in Type II fibers (1205 and 1228 μm^2) in both S and CC groups. Although significant Type I fiber hypertrophy occurred only in the S group (820 μm^2), this change was not different than the nonsignificant increase seen in the CC group (537 μm^2). With the exception of a small increase (3%) in quadriceps cross-sectional area, there were no changes in any of the above variables in the E group. We have previously shown that the E-training regimen, as employed in the current study, substantially increases (18%) $\dot{V}O_{2peak}$ (27). Our findings indicate that 3-d·wk^{-1} concurrent training for both strength and endurance, in sedentary subjects, does not impair the magnitude of muscle hypertrophy induced by S training alone. Results of the concurrent strength and endurance training were similar to the strength-only training in all neuromuscular measures. These findings provide a physiological basis to support a number of studies addressing performance adaptations that indicate concurrent training does not impair strength development over the short term with 3-d·wk^{-1} training (1,9,14,27,34,38).

Why strength development is attenuated in only some concurrent-training studies has generally been attributed to differences in training-design variables (26,34). These include training volume, duration, frequency, intensity, mode or type of S and E training, and initial training status of subjects. However, few if any guidelines for designing concurrent-training programs for preventing or minimizing strength development impairment have been proposed (12,26). A striking contrast in concurrent-training regimens that induce impairment in strength development with regimens that do not induce impairment involves the frequency of training. When both strength and endurance training is performed on the same day and for only 3 d·wk^{-1} on alternate days, as in the current study, strength development is not compromised as compared with performing the strength training only (1,9,14,27,34,38). The similar increases in isometric strength performance in our S and CC groups agree with the similar strength increases in S and CC groups of all other concurrent-training studies we know of employing 3-d·wk^{-1} CC training (1,9,14,27,34,38). Four of these studies involved untrained or sedentary subjects (9,27,34,38), and two of these studies involved physically fit subjects who regularly exercised (details limited) (1,14). In

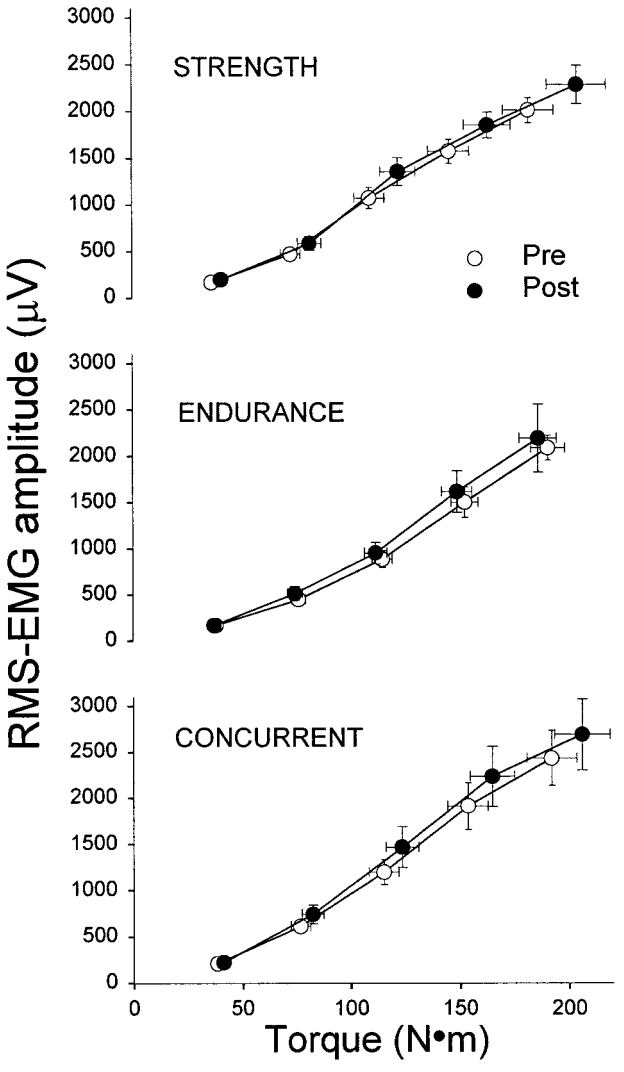

FIGURE 2—The EMG/torque relation in the quadriceps of the three groups of subjects, pre and post training. Values are means ± SE. No significant changes in this relation were seen pre to post training ($P < 0.0167$) or between groups ($P < 0.05$). Maximal RMS-EMG amplitude did not change significantly pre to post training ($P < 0.0167$) or between groups ($P < 0.05$).

our previous study (27), we also reported almost identical increases in one repetition maximum (1RM) barbell squats (S = 24 kg [23%], CC = 23 kg [22%]) and maximum vertical jump height (S = 3 cm [6%], CC = 4 cm [9%]) employing the same training regimens as in the current study. Due to specificity of training, we expected smaller increases in isometric strength and maximum vertical jump height than in 1RM squat strength, because barbell squats were used in the training (27,30). Although Craig et al. (9) reported a significant 5.8% (7.9-kg) increase in leg strength with S training, the CC group in this study did not significantly increase strength. These authors concluded that leg strength does not increase to the same degree when endurance training is added to strength training. We disagree with this conclusion in that there was no statistical analysis performed comparing between group changes and little actual difference between the two groups in strength improvement existed (the CC group showed a similar level change of 4.6% [6.5 kg], although this was not statistically significant from pre to post training within the group). Thus, we are assuming similar strength adaptations in both S and CC groups for the Craig et al. (9) investigation.

The similar strength gains seen in both S and CC groups with 3-d·wk^{-1} training are in sharp contrast to concurrent training that takes place 5 or 6 d·wk^{-1}. Concurrent-training investigations that employ a CC group, with some type of training (S, E, or both) being performed 5 (21) or 6 d·wk^{-1} (2,4,11,22,23), report impairment in some type of strength performance measure with CC training compared with S training. The initial training status of subject groups in these studies varied considerably from untrained (11,22,23), to athletes out of training for at least 4 wk (21), to experienced rowers or experienced weight trainers (2), to physically active subjects who were not formally training to develop strength or endurance at the time of entry into the study (4). In another study, Bell et al. (3) report similar strength increases in S and CC groups with the CC group training 6 d·wk^{-1}. But the group labeled S in this study was also allowed to perform one E-training session per wk. Thus, as indicated by Leveritt et al.(26), in this investigation, two CC-training protocols were really being compared. Equivocal results have been reported in whether an impairment in strength development occurs when endurance training is added to strength training in studies that employ 4-d·wk^{-1} CC training. Whereas Kraemer et al. (25) conclude impairment in strength development occurs, Nelson et al. (31) report similar increases in strength in both S and CC groups over 20 wk of training. It thus appears that at least over the short term (the longest duration of training in a study employing 3-d·wk^{-1} CC training was 22 wk (34)), designing CC-training regimens that will not impair strength develop may need to limit training to no more than 3 d·wk^{-1}. In contrast, if some type of S or E training is performed for the same muscle group 5 or 6 d·wk^{-1}, some type of strength impairment is likely to occur with CC training. Whereas frequency of training may be an important factor in influencing outcomes with concurrent training, it appears no clear pattern of results are obtained when other training-design variables (including initial training status of subjects) are compared across studies (1,14,26). Our current findings provide a physiological basis to support the consistency across studies that indicate the same level of strength improvements can be achieved using 3-d·wk^{-1} concurrent training as that which occurs with 3-d·wk^{-1} strength-only training.

The current investigation indicates substantial similar muscle hypertrophy occurred in both S and CC groups as assessed at the macroscopic and microscopic levels. Our whole muscle and fiber area S training results are similar in magnitude with results reported in the literature employing similar resistance-training regimens in young adults (4,18,19,28). Hakkinen et al.(19), employing a very similar strength-training regimen for the quadriceps (10 wk of 3-d·wk^{-1} barbell squat and machine leg-extension training) as in the current study, reported increases in vastus lateralis Type I, IIa, and IIb fiber areas of 23%, 26%, and 14%, which are similar in magnitude to increases in our S group of 19% and 24% in Type I and II fiber areas. Identical increases in quadriceps CSA of 12.2% were found in our S group subjects and in similar age subjects in the Hakkinen et al. (19) investigation. With a similar 12-wk strength-training regimen, we previously reported a 14.4% increase in quadriceps volume (28).

Muscle morphology adaptations have only been considered in a few concurrent-training investigations. At the whole muscle level, our findings are in agreement with Sale et al. (34), who report similar increases in quadriceps CSA with both S (13%) and CC (11%) 3-d·wk^{-1} training. Similar strength improvements from both S and CC training are also accompanied with similar increases in both Type I and II fiber areas for both these training conditions. Although our results are consistent with these findings, there are substantial differences in the design of the Sale et al. (34) study that appear to limit appropriate comparisons with other concurrent-training investigations. The endurance-only training (3-min bouts at 90–100% VO_{2max}) employed in this study produced substantial increases in strength (20%) and substantial increases in quadriceps cross-sectional area (14%). Also, their results may be affected in employing a unilateral-leg–training model where potential cross-over adaptations may occur in the comparative (control) contralateral limb (40).

In adding endurance training to strength training, a limitation in muscle hypertrophy has been suggested as a mechanism for an interference in strength gains with concurrent training (4,26). Bell et al. (4) and Kraemer et al. (25) both report some type of impairment in strength development with 12 wk of CC training compared with S training, and both implicate an underlying factor may be a limitation in hypertrophy of Type I fibers. Bell et al. (4) reported very similar fiber type adaptations as in our current findings. In their study, both Type I (27%) and II (28%) fiber areas increased with S training, whereas in their CC group, only a significant within group increase occurred in Type II (14%) fiber area (Type I fiber area increased 11%, which was not statistically significant). Although the within-group

increases resulting from the S training appear to be about twice as large as changes within the CC group, there were no significant differences in the increases between the groups in both Type I and II fiber areas. The critical criterion in determining whether an impairment occurs with concurrent training is whether there is a difference in adaptations between groups. Failure in the Bell et al. (4) investigation not to find a significant difference between groups could be due to low statistical power for this test, which may relate to the fair amount of variability with fiber type area measures (35).

Kraemer et al. (25), employing 4-d·wk^{-1} training programs in physically active soldiers, found similar adaptations in Type IIa fiber areas in both S (24%) and CC (20%) trained subjects. In contrast, the S training induced a 12% increase in Type I fiber area, which was greater than the change seen with CC training (a nonsignificant within group decreased of 5%). We are not considering changes in Type IIb and IIc fibers from the Kraemer et al. investigation (25), because after training their percentage distributions were 2% or less in both S and CC groups. Also of interest in the Kraemer et al. (25) study was that the running type endurance-only training employed in the study induced an 11% decrease in Type I fiber area. This finding was different from our current results and all other E-training regimens used in concurrent-training investigations that assessed fiber areas (4,31,34). Combining an E-training program that induces a reduction in muscle fiber size with a strength-training program would account for the attenuation in fiber hypertrophy produced by the strength training alone. Other concurrent-training investigations that have combined running E training with strength training have found no impairment in strength development compared with the strength training alone (9,38).

Nelson et al. (31) also reported that concurrent S and E training produces different fiber hypertrophy patterns than S training. However, they report significant hypertrophy occurred in response to both E and CC training in all fiber type areas (I, IIa, IIb). Their knee-extension isokinetic S training, performed at 0.52 rad·s^{-1}, only induced hypertrophy in Type IIb fibers. With both S and E training producing increases in strength, there was no impairment in strength development when the E training was added to the S training. The apparent training effects of the individual S and E protocols employed by Nelson et al. (31) were substantially different that ours and other individual protocols employed by other investigations considering fiber type adaptations. The eclectic array of training-design variables, and somewhat limited descriptions of protocols employed in the different concurrent-training investigations, makes it difficult to compare studies directly.

It has been proposed that an impairment in strength development with concurrent training may be due to increases in Type I fiber composition, with concomitant decreases in Type II fiber percentage (6,12). In response to training in the present study, fiber distribution remained the same across all three groups. Studies reporting similar strength improvements with S and CC training (31,34), and a study reporting an impairment in strength development with concurrent training (25), have indicated little difference in fiber type change between S and CC training. Bell et al. (4) did not report fiber-type distribution despite reporting fiber-area results. In the few studies to date, none have indicated that CC training alters the fiber distribution pattern seen with strength training alone.

Results of the current investigation do not give any credence to the hypothesis that an interference in strength development with concurrent training may be related to neural activation. In response to S training in the current study, maximal RMS-EMG amplitude was not significantly different ($P = 0.044$) from that before training, possibly due to rather large methodological error of this measure (30). Again, the reader is reminded that due to the application of the Bonferroni procedure (to maintain family-wise Type I error at 0.05), the criterion for finding a difference within a group (level of statistical significance = 0.0167, for each *t*-test for the three groups) was more stringent than the criterion for finding a difference between groups (level of significance = 0.05). Although a number of studies report an enhancement in maximal EMG amplitude with strength training (16,17,29), our results are in agreement with several other investigations that report no change in maximal EMG amplitude with strength training (5,13,30,37,39). The 13.5% nonsignificant increase in our S group is similar in magnitude to significant 11.8 and 12.5% increases reported in other strength-training regimens by Moritani and deVries (29) and Hakkinen and Komi (17). Very similar nonsignificant increases within our S (272 μV) and CC (258 μV) groups indicate no interference in maximal neural activation is likely when E training is added to S training in 3-d·wk^{-1} concurrent training. Further, the closeness of EMG/torque relation changes (across 20–100% MVC), in both S and CC groups, indicate RMS-EMG amplitude as an unlikely mechanism related to strength impairment with 3-d·wk^{-1} training (Fig. 2). We are unaware of any research investigating neural activation changes due to endurance training. As indicated earlier, the E training in the present study did not affect strength, but did substantially increase $\dot{V}O_{2peak}$. No change in maximal RMS-EMG amplitude with E training, and the closeness of the pre- and post-EMG/torque relation curves, indicates little effect of the continuous cycling E training on neural activation. We cannot rule out, however, that other possibly more intense, or other modes of E training, may influence neural activation and could potentially interfere with strength development when added to S training.

In the current study, although we have investigated some major mechanisms that may account for the possibility of strength impairment occurring when endurance training is added to strength training, other mechanisms may also be involved. Concurrent-training investigations compare CC training with other individual training regimens that are performed less frequently or with much less volume. This argues that overtraining and/or chronic muscle glycogen depletion, which may occur with consecutive days of training (8,36), may be other mechanisms that negatively

influence strength adaptations with concurrent training (12,25,26). Although inconsistent in the literature, there is evidence that a catabolic state related to increased cortisol levels, combined with little change in concentrations of anabolic hormones (i.e., testosterone, growth hormone), may indicate an overtrained state with concurrent training (2,4,25). It appears future investigations should consider examining indices of overtraining and compare concurrent-training protocols that are more similar in volumes to individual S and E regimens. It should be noted, however, that no strength development impairment has been reported with CC training performed 6 $d \cdot wk^{-1}$ (2), whereas fairly low volume training (11) has produced significant strength development impairments in CC versus S training. In the Bell et al. (2) study where CC training was performed 6 $d \cdot wk^{-1}$, there was nonrandom assignment of college students to two groups of S and CC training. A group of experienced rowers performed CC training 6 $d \cdot wk^{-1}$ (3-$d \cdot wk^{-1}$ endurance and 3-$d \cdot wk^{-1}$ strength training), and a group of experienced strength trainers (nonrowers) performed the same S training 3 $d \cdot wk^{-1}$. In this study, only female subjects showed an impairment in strength development with CC training as compared with S training, whereas male subjects did not show any impairment. For the present study, we chose a training model to help control for potential overtraining effects by stressing the same muscle group (quadriceps) only 3 $d \cdot wk^{-1}$ with concurrent training.

In summary, our findings indicate that, in sedentary subjects, 3-$d \cdot wk^{-1}$ concurrent training for both strength and endurance does not impair the magnitude of muscle hypertrophy induced by strength training alone. The S training employed induced increases in maximal knee extensor torque that was accompanied by substantial muscle hypertrophy of the quadriceps as measured at both the macroscopic and microscopic levels, whereas maximal neural activation of the quadriceps was not significantly increased. With S training, no changes were seen in fiber-type distribution or in the EMG/torque relation across 20–100% MVC. With the exception of a small 3% increase in quadriceps cross-sectional area, there were no changes in any of the above variables with E training. Results in our study of the concurrent strength and endurance training were similar to the strength-only training in all neuromuscular measures. Our findings provide a physiological basis to support a number of studies addressing performance adaptations that consistently indicate concurrent training does not impair strength development over the short term with 3-$d \cdot wk^{-1}$ training (1,9,14,27,34,38).

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