Effect of resistance training on muscle fatigue and recovery in intact rats

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ABSTRACT

WILLEMS, M. E. T., and W. T. STAUBER. Effect of resistance training on muscle fatigue and recovery in intact rats. *Med. Sci. Sports Exerc.*, Vol. 32, No. 11, pp. 1887–1893, 2000. **Purpose:** To examine the effect of resistance training on muscle fatigue from intermittent contractions and subsequent recovery in intact rats. **Methods:** By using electrical stimulation, plantar flexor muscles were trained with eccentric and concentric contractions (5 × 10 repetitions, 5 d·wk^{-1} for 6 wk) during ankle rotations. By using nerve stimulation, concentric contractions (40) imposed on isometric contractions (stimulation time, 1.9 s; rest period, 13.6 s; intermittent contractions) induced fatigue. During recovery, equivalent contractions were used every 5 min for 30 min. **Results:** Training increased isometric forces (19% and 23% at ankle positions of 1.57 and 0.70 rad), but muscle weights were not changed. After training, smaller declines in isometric (control, 68.9 ± 1.4%; trained, 58.8 ± 2.9%) and average concentric force (control, 71.6 ± 0.7%; trained, 65.5 ± 2.8%) occurred from fatigue. Recovery for 5 min returned isometric and average concentric force to 61.7 ± 2.2% and 65.1 ± 2.5% of initial values for controls and 76.9 ± 2.2% and 77.1 ± 2.2% after training. After recovery for 30 min, these forces were 87.6 ± 0.7% and 89.2 ± 1.1% of initial values for controls and recovered almost completely (94.2 ± 1.3% and 94.6 ± 1.6%) in trained muscles. During fatigue, the decline in force during successive concentric contractions was larger after training (from 19.7 ± 1.1% to 50.1 ± 2.0%; controls, from 19.9 ± 2.0% to 41.7 ± 1.4%). Recovery of this decline in force was training-independent and complete within 5 min. **Conclusions:** Rat plantar flexor muscles adapt to 6 wk of 5 d·wk^{-1} resistance training with: 1) increased isometric force, 2) smaller losses in isometric and average concentric force during fatigue, 3) larger force decline during concentric contractions during fatigue, and 4) improved recovery following fatigue. Different mechanisms might account for the recovery of the average concentric force and the decline in force during concentric contractions. **Key Words:** SKELETAL MUSCLE, ISOMETRIC CONTRACTION, CONCENTRIC CONTRACTION, MUSCLE FORCE, STRENGTH TRAINING

Skeletal muscles exposed to high loads during resistance training adapt by changes in structure and performance (15). Usually, the performance is improved by enhanced force output due to hypertrophy (16) resulting from load-induced muscle growth (1). After voluntary resistance training, a reduced fatigability of skeletal muscles has been observed in healthy humans (5) and animals (6,24) and in patients after anterior cruciate ligament surgery (20). Patients with chronic heart failure improved isometric strength and also fatigue resistance after resistance training with neuromuscular electrical stimulation (23). In general, muscle fatigue from isometric contractions has been quantified by the time to maintain a specific force level during long-duration isometric contractions, a decline in isometric force during intermittent isometric contractions, or a decline in force during sustained isometric contractions. However, muscle fatigue induced by intermittent isometric contractions is different than for concentric contractions (i.e., shortening) (12). Thus, resistance-training programs would be expected to produce different responses to intermittent isometric and concentric contractions.

In humans, resistance training often consists of one to five bouts of 4–10 repetitions performed not more than three times a week (e.g., 25). Significant changes in muscle performance can be seen by 6 wk of training (28). Various animal models have been used to study adaptations to human strength-training protocols (for a review see ref. 27). In rats, an increase in weight and force of the plantar flexor muscles was reported after 16 wk of resistance training when 3 d of rest occurred between training sessions (30). No increase in muscle weight was observed with similar training sessions performed every other day. Whether such an intense resistance-training program can lead to changes in muscle performance by intermittent contractions and subsequent recovery is not known. In the present study, we exposed plantar flexor muscles of intact rats to a intense resistance training protocol, five bouts of 10 repetitions, 5 d·wk^{-1} for a period of 6 wk. The effect of resistance training on muscle fatigue by intermittent contractions and subsequent recovery was examined using high-intensity nerve stimulated isometric and concentric contractions. In the present study, isometric fatigue and recovery were quantified by the relative decline in isometric force. Concentric fatigue and recovery were quantified in two ways. First by the relative decline in average concentric force and second

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by the decline in force during a concentric contraction. The hypothesis that a heavy resistance-training program, 5 $d \cdot wk^{-1}$ for 6 wk, could improve the isometric and concentric muscle performance without changes in muscle weight was tested.

METHODS

Female Sprague-Dawley rats (4–5 months) were used. Animals used for this study complied with Animal Welfare Act P.L 91–579 and DHHS Guidelines governing the care and use of laboratory animals. All procedures in this study were approved by and followed the guidelines of the West Virginia University Animal Care and Use Committee (WVU-ACUC #9809–02) and were conducted in accordance with the principles of the American College of Sports Medicine. Rats were provided water and laboratory chow *ad libitum* and were housed (2–3 per cage) in animal facilities maintained at 21°C with a 12:12 h light:dark cycle. Experiments were performed to determine the effect of resistance training (5 $d \cdot wk^{-1}$ for 6 wk, with eccentric and concentric contractions) on isometric force, muscle fatigue, and recovery during high-intensity intermittent contractions by using nerve stimulated concentric contractions imposed on isometric contractions.

Resistance training. Training sessions were performed on rats given atropine (0.08 mg kg^{-1} s.c.) and anesthetized with ether. Animals were placed under a fume hood during the entire training session. Electrodes for electrical stimulation were subcutaneously inserted and positioned bilaterally along the surface of the plantar flexor muscles of the lower left hindlimb (31). Electrical stimulation of muscles with 0.2-ms pulses at 70 Hz and 20 V was selected to produce submaximal muscle activation. We have observed, using dynamometry, that force with 20 V stimulation produces 50–90% of the force with 40-V stimulation producing maximal force (data not shown). Eccentric and concentric contractions of plantar flexor muscles were induced following onset of stimulation by the experimenter (W.S.) by manual rotation of the ankle by pushing at the sole of the foot (i.e., eccentric contractions by dorsiflexion of the ankle) and returning the foot to the starting position (i.e., concentric contractions by plantarflexion of the ankle). Rotation of the ankle was approximately between 2.44 rad (140°) and 0.70 rad (40°) at an estimated average angular velocity of about 0.87 $rad \cdot s^{-1}$. The basic training regime consisted of five bouts of 10 repetitions each (time needed to turn on and off stimulation and perform 10 repetitions was about 5 s) with 30-s rest intervals between bouts performed 5 $d \cdot wk^{-1}$ for 6 wk. The resistance-training program was used to create resistance-trained plantar flexor muscles without changes in muscle weight. Rats exposed to the resistance-training program will be referred to as resistance-trained rats.

Fatigue and recovery of skeletal muscles. Experiments for fatigue and recovery were performed on plantar flexor muscles in resistance trained ($N = 7$) and control rats ($N = 7$) by using direct nerve stimulation. Control rats consisted of sedentary rats of the same age. Rats were anesthetized with sodium pentobarbital (75 mg kg^{-1} i.p.) and supplementary doses administered as required. Details on the dissection procedure for nerve cuff placement, animal positioning, dynamometer, and force recording are described elsewhere (3,29). Briefly, the foot of the rat was positioned on an aluminum plate that was connected to a dynamometer. The dynamometer consists of a DC permanent magnet servomotor (Model 1410C) and an Unidex 1 single axis motion controller (Aerotech Inc, Pittsburgh, PA). Below the aluminum plate is a Z-11/5 kg load cell (HBM Inc, Marlboro, MA). Force of the plantar flexor muscles by stimulation via the tibial nerve was recorded under the sole of the foot (29).

The stimulus parameters for high-intensity activation of the plantar flexor muscles by electrical stimulation of the tibial nerve were determined using five to seven isometric contractions with a duration of 600 ms at a force plate position of 2.09 rad. These stimulus parameters (200 μs pulse duration; 80 Hz; 5.4 $\pm$ 0.4 V, mean $\pm$ SE) produced isometric force levels above 90% of the maximal isometric force possible at an ankle position of 2.09 rad without substantial fatigue (i.e., force decline) during isometric contractions of 600 ms (data not shown) and were used for all nerve stimulated contractions.

Isometric force. Before fatigue testing, three isometric contractions with a duration of 600 ms at an ankle position of 1.57 rad were produced. Two-minute rest periods between isometric contractions of 600 ms avoids fatigue (i.e., range of force decline is 0–4%).

Fatigue and recovery. Fatigue and recovery were tested using concentric contractions between ankle positions of 0.70 rad and 1.57 rad imposed on isometrically active muscles at an ankle position of 0.70 rad. These contractions were equivalent to those used by James et al. (12) to study muscle fatigue in humans. The rat's ankle was rotated from 1.57 rad to 0.70 rad with an angular velocity of 0.87 $rad \cdot s^{-1}$ with relaxed muscles. After a 100-ms pause, the plantar flexors were activated (i.e., isometric preload) and 600 ms later the ankle rotated to 1.57 rad at an angular velocity of 0.87 $rad \cdot s^{-1}$ (i.e., concentric contraction). Figure 1 shows a typical force-time pattern of a contraction, illustrating the isometric contraction phase and concentric contraction phase (i.e., shortening). Total stimulation time for such a contraction was 1.9 s. Forty contractions were performed to produce fatigue with rest periods between contractions of 13.6 s. Consequently, duty cycle (i.e., contraction time/rest period) was 0.14. After fatigue, additional contractions were made, using contractions as described above, every 5 min for 30 min to monitor recovery. All contractions (fatigue and recovery) were preceded by one movement of the same magnitude but with no activation of the muscles. Animals were sacrificed with an intracardial injection of sodium pentobarbital immediately following the final testing.

Data collection and analysis. Concentric forces (i.e., forces during muscle shortening) were calculated as the difference between the total force during movements with concentric contractions and the passive force recorded

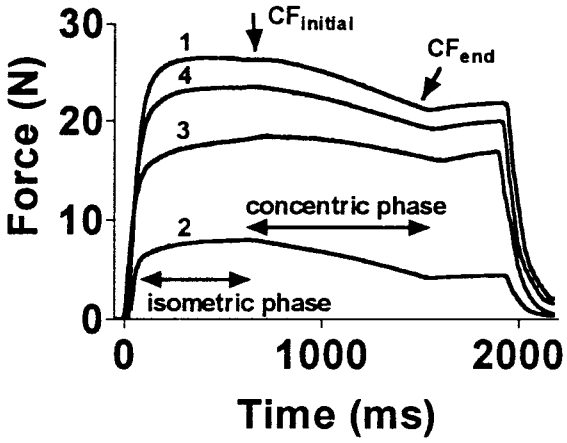

Figure 1—Typical examples of force-time traces during individual contractions with the isometric and concentric phase indicated. These phases were used to measure muscle fatigue and subsequent recovery. *Solid lines* indicate the 1st (no. 1) and 40th (no. 2) contraction during fatigue testing and contractions after 5 min (no. 3) and 30 min (no. 4) of recovery. *Arrows* indicate the concentric force at the beginning (i.e., $CF_{initial}$) and end (i.e., CF_{end}) of the concentric contraction.

during movements with no muscle activation. For each contraction, the isometric force was calculated by subtracting the average force 100 ms before stimulation from the average total force between 500 and 600 ms after stimulation (i.e., on the tetanic plateau during the isometric phase). The average concentric force was calculated during the concentric contraction phase.

Isometric fatigue and isometric recovery. Isometric contractions were analyzed by the relative decline in isometric force during the fatigue protocol and recovery period. The relative decline in isometric force was calculated as the isometric force decline index (IFDI) by $(F_1\text{-}F_x)/F_1 \cdot 100$, where F_1 is the isometric force (i.e., isometric preload) of the first contraction during fatigue testing and F_x the isometric (preload) force for each contraction during fatigue and recovery testing.

Fatigue and recovery of concentric contractions. Fatigue and recovery of concentric contractions were quantified in two ways: 1) by the relative decline in average concentric force and 2) by the relative decline in force during the concentric phase of the contractions. The relative decline in average concentric force was calculated as the concentric force decline index (CFDI) by $(CFDI_1 - CFDI_x)/CFDI_1 \cdot 100$, where $CFDI_1$ is the average concentric force for the first contraction during fatigue testing and $CFDI_x$ is the average concentric force for a contraction during fatigue and recovery testing. The relative decline in force during the concentric contraction was calculated by $(CF_{initial} - CF_{end})/CF_{initial} \cdot 100$, where $CF_{initial}$ and CF_{end} are the muscle forces at the beginning and end of the concentric contraction phase in each contraction during fatigue and recovery testing.

Plantar flexor muscles (i.e., soleus muscle, plantaris muscle and gastrocnemius muscle) of right and left hindlimb of resistance trained rats and right hindlimb of control rats were dissected, removed of external fat, and weighed. Adrenal glands in resistance-trained rats and control rats were dissected, removed of external fat, and weighed. In each group, weights of the left and right adrenal glands were averaged.

Statistics. Student *t*-tests were used to test for differences between resistance-trained rats and control rats for 1) body weight, 2) mass of the plantar flexor muscles of the right hindlimb, 3) weight of the adrenal glands, 4) isometric force at 1.57 rad, 5) isometric force at 0.70 rad, 6) IFDI at the end of fatigue and recovery testing, and 7) CFDI at the end of fatigue and recovery testing. Two-way analysis of variance (ANOVA) with Bonferroni *post hoc* testing (GraphPad Prism v3.0 for Windows, GraphPad Software, San Diego, CA) was used to test for differences between resistance-trained rats and control rats on 1) the decline in IFDI, 2) the decline in CFDI, and 3) the relative decline in force during a concentric contraction. Significance was accepted at $P < 0.05$.

RESULTS

Body weights were 7% lower in resistance-trained rats, but the weights of the plantar flexor muscles were not different than control rats (Table 1). In contrast, the isometric forces of the plantar flexor muscles of resistance-trained rats were 19% and 23% greater at ankle positions of 1.57 rad and 0.70 rad, respectively, than control rats (Table 1). Weights of the adrenal glands were 17% larger in resistance-trained rats than in control rats (Table 1).

Contractions used to produce fatigue and monitor recovery. Typical examples of force-time traces of the plantar flexor muscles during fatigue (1st and 40th contraction) and recovery (5 and 30 min after fatigue testing) showing the concentric contraction phase (i.e., shortening) after an isometric contraction phase are presented in Figure 1. Repeated contractions to produce fatigue resulted in a substantial decrement (>50% decrease) in total force

TABLE 1. Body weight, adrenal weight, muscle weight, and isometric forces of control rats and resistance-trained rats.

Group	Control		Training	
Body wt. (g)	281 ± 3		261 ± 6*	
Adrenal wt. (mg)	30.3 ± 0.5		35.6 ± 2.0*	
	Right hindlimb	Left hindlimb	Right hindlimb	Left hindlimb
Plantar-flexors wt. (mg)	2195 ± 45	ND	2228 ± 50	2197 ± 49
Isometric force at 1.57 rad, (N)	ND	22.3 ± 0.9	ND	27.7 ± 0.5*
Isometric force at 0.70 rad, (N)	ND	23.9 ± 1.1	ND	29.5 ± 0.6*

Values are presented as mean ± SE; $N = 7$ per group.
* Significant difference between control and resistance-trained rats (Student's *t*-test, $P < 0.05$).
ND, not determined; isometric forces were measured at two ankle positions.

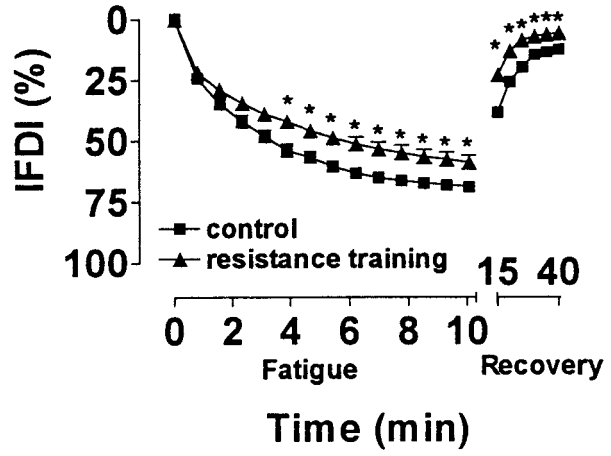

Figure 2—The isometric force decrement index (IFDI) of the rat plantar flexor muscles as a function of time during fatigue testing and subsequent recovery for control rats (■) and resistance-trained rats (▲). Data are presented as mean ± SE for seven animals. For illustrative purposes, only force data of every third contraction during fatigue testing are plotted. * Significant difference between control and resistance trained rats, $P < 0.05$.

output. Total force output increased during the recovery after fatigue (Fig. 1).

Decline in isometric force and average concentric force: fatigue and recovery The decrements in isometric force (IFDI) and average concentric force (CFDI) of plantar flexor muscles were used as indexes of fatigue and recovery for control and resistance-trained rats (Figs. 2 and 3). The isometric force and average concentric force of the plantar flexor muscles decreased exponentially during repeated contractions (i.e., 40 contractions in 10.3 min) and increased exponentially during recovery testing (i.e., 6 contractions in 30 min) in control and resistance-trained rats (Figs. 2 and 3). In both groups, half of the total loss in isometric force (Fig. 2) and average concentric force (Fig. 3) occurred within 2 min (i.e., after 6–7 contractions). Both

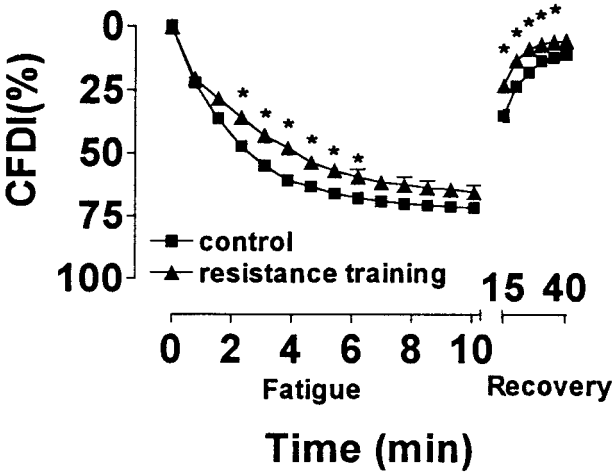

Figure 3—The average concentric force decrement index (CFDI) of the rat plantar flexor muscles as a function of time during fatigue testing and subsequent recovery for control rats (■) and resistance-trained rats (▲). Data are presented as mean ± SE for seven animals. For illustrative purposes, only force data of every third contraction during fatigue testing are plotted. * Significant difference between control and resistance-trained rats, $P < 0.05$.

TABLE 2. Indexes after 40 repeated contractions (duty cycle 0.14) (i.e., fatigue) and subsequent recovery for 30 min for control rats and resistance-trained rats.

	Fatigue		Recovery for 30 min	
	Control	Training	Control	Training
IFDI	68.9 ± 1.4%	58.8 ± 2.9%*	12.4 ± 0.7%	5.8 ± 1.3%*
CFDI	71.6 ± 0.8%	65.5 ± 2.8%*	10.8 ± 1.1%	5.4 ± 1.6%*

Values are presented as mean ± SE; $N = 7$ per group.
* Significant difference between control and resistance-trained rats (Student's t-test, $P < 0.05$).
IFDI, isometric force decrement index; CFDI, average concentric force decrement index.

indexes (IFDI and CFDI) became significantly smaller for resistance-trained rats during fatigue testing (i.e., reduced fatigability, Figs. 2 and 3) (IFDI; $F[1,480] = 387.6$; CFDI: $F[1,480] = 408.2$; two-way ANOVA). At the end of the 40 contractions used to induce fatigue, indexes were less for resistance-trained rats and recovered more completely for resistance-trained rats compared with control rats (Table 2).

For trained and control rats, both the average values for the relative decline in isometric force and relative decline in average concentric force during fatigue and recovery were subtracted and presented in Figure 4. A trend for a difference between the groups in fatigue and recovery by isometric contractions (IFDI) or concentric (CFDI) contractions can be seen, but statistical analysis would be inappropriate. For isometric contractions during fatigue, plantar flexor muscles of trained rats were less fatigable than control rats. This difference was maximal at 4 min (i.e., 15–17 contractions) and remained relatively the same over time. For concentric contractions during fatigue, plantar flexor muscles of trained rats were less fatigable than control rats, reaching a maximum also at 4 min (i.e., 15–17 contractions). After 4 min, there was a tendency for this difference to become smaller with time. Plantar flexor muscles of trained rats seem to recover faster from isometric and concentric fatigue than control rats for the first 20 min of recovery. After 20 min, there is no change in the difference between either IFDI or CFDI of resistance-trained and control rats. The force decrements by isometric or concentric contractions between trained rats and control rats seem to follow different time courses during fatigue but not during recovery.

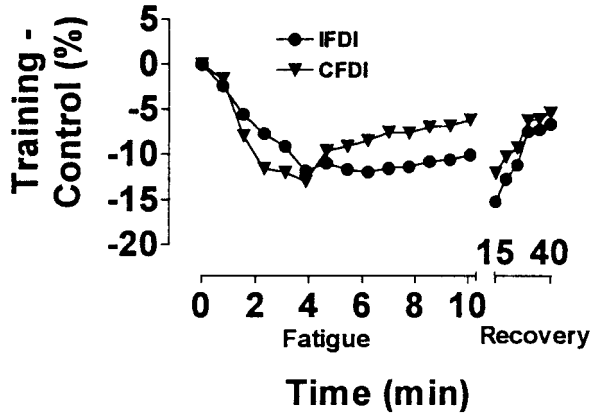

Figure 4—The difference for either the isometric force decrement index (●) or average concentric force decrement index (▼) between resistance-trained and control rats (seven animals in each group). For illustrative purposes, only differences of indexes of every 3rd contraction during fatigue testing are plotted.

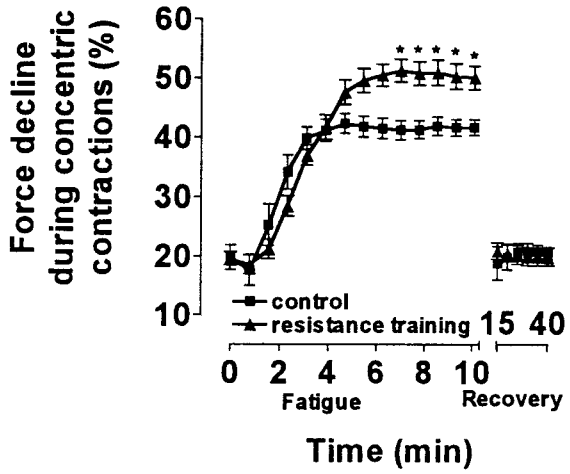

Figure 5—The relative decline in force during the concentric contractions as a function of time for rat plantar flexor muscles during fatigue and subsequent recovery for control rats (■) and resistance-trained rats (▲). Data are presented as mean ± SE for seven animals. For illustrative purposes, only relative declines of every third contraction during fatigue testing are plotted. * Significant difference between control and resistance trained rats, $P < 0.05$.

Decline in force during concentric contractions: fatigue and recovery In control and resistance trained rats, the relative decline in force during a concentric contraction increased during the fatigue protocol (Fig. 5) (F[1,480] = 87.0; two-way ANOVA). Resistance-trained rats and control rats reached a limit for the decline in force during concentric contractions. During fatigue testing, the decline in force during concentric contractions became significantly larger for resistance-trained rats at 6.5 min compared with control rats (Fig. 5) (two-way ANOVA). During recovery, the relative decline in force during a concentric contraction was complete within 5 min in both control and resistance trained rats (Fig. 5).

DISCUSSION

Animals in resistance-training programs, mimicking those in humans, have been used to study muscle performance (strength) and hypertrophy (for a review see ref. 27). In this study, the hypothesis was tested that a heavy resistance-training program with electrically stimulated skeletal muscles, 5 $d \cdot wk^{-1}$ for 6 wk, could improve the isometric and concentric muscle performance (without changes in muscle weight). Subcutaneous electrical stimulation (stimulation parameters: 1-ms pulse duration, 100 Hz, 15 V) was also used by Wong and Booth (30) for strengthening plantar flexor muscles in ether anesthetized rats. Wong and Booth (30) trained rats for 16 wk with a progressive increase in the load lifted with four sets of six repetitions every 4th day. Weights of the gastrocnemius, plantaris, and soleus muscles were increased by 18%, 18%, and 13%, respectively. Remarkably, no enlargement of plantar flexor muscles was reported for one rat after 16 wk of training with just one recovery day between the training sessions (30). This observation led to the suggestion that, in their model, an increased frequency of training (days) would hinder muscle enlargement (i.e., no increase in muscle weights). The absence of muscle enlargement in our study might be due to the fact that the training period was too short [6 wk compared with 16 wk by Wong and Booth (30)]. However, Jaweed et al. (13) trained rats 5 $d \cdot wk^{-1}$ for 6 wk and did find an increase in weight of the gastrocnemius and soleus muscles. On the other hand, a decrease in weight of gastrocnemius and soleus muscles was reported after mild weight-lifting for 4 $d \cdot wk^{-1}$ for 6 wk (9). Because Gardiner et al. (9) used electric shocks to stimulate weight-lifting by the rats, the stress of repeated shocks could have led to some muscle catabolism if cortisol levels were increased. Evidence of chronic stress was reported after daily swim training in female rats (5 $d \cdot wk^{-1}$ for 10 wk): adrenal weights were increased by 12% (26). In our study, weights of the adrenal glands were also increased (17%). Increased stress levels by the training protocol could result in weight loss of skeletal muscles and counteract weight gains by the resistance training.

The absence of muscle enlargement in the present study did not diminish an increase in isometric force of the plantar flexor muscles. The isometric force increased 19% and 23% at ankle positions of 1.57 rad and 0.70 rad, respectively, by the resistance training (i.e., 0.63–0.80% per day). Rat plantar flexor muscles produce nearly constant isometric force at ankle positions between 0.61 rad and 2.18 rad (29). Therefore, small length changes of the muscles induced by the resistance training cannot lead to a 20% increase in isometric force at the ankle positions tested. In animals with electrically evoked muscle contractions, discrepancies between increases in muscle force by resistance training (16, this study) or jump training (4) and muscle weight can only result from structural and/or mechanical changes in the skeletal muscle. The increase in isometric force could be due to an increased density of myofibrillar protein (10)—an increased number of cross-bridges (i.e., greater forces) per cross-sectional area. An increased isometric force could also be related to a training-induced increase in collagen content and collagen cross-links in individual plantar flexor muscles which altered force transmission (14), producing more effective force transfer to the Achilles tendon of the foot. It is unlikely that the increase in force was due to an upward and rightward shift of the force-position relationship of the plantar flexor muscles due to the addition of sarcomeres in series (21) by resistance training because a similar increase in force was observed at different ankle positions (i.e., different muscle lengths). In addition, a shift in fiber type from low force-generating slow-twitch muscle fibers to high force-generating fast-twitch muscle fibers seems unlikely as such a shift is not consistent with the observed decreased fatigability in resistance-trained rats unless all the fibers became fast oxidative. It is concluded that resistance training, 5 $d \cdot wk^{-1}$ for 6 wk, with eccentric and concentric contractions of rat plantar flexor muscles using electrical stimulation can increase isometric force substantially (~20%) without muscle enlargement.

An enhanced force output by resistance training could improve the performance of high-intensity contractions. However, high-intensity contractions result in substantial muscle fatigue and is associated with large decrements in

force output of skeletal muscles (e.g., 12). Physiological mechanisms of muscle fatigue are dependent on the intensity, type, and duration of the exercise (for a review of muscle fatigue see ref. 8). It is well known that endurance training improves muscle fatigue. The information on effects of resistance training involving eccentric and concentric contractions on muscle fatigue by isometric and concentric contractions in animals is absent. Diminished fatigue for isometric contractions was reported for EDL and SOL after a 26-wk weight-lift training program (i.e., vertical rack climbing with weights attached to the tail) (6) and for adductor longus muscle after 8 wk of squats (24). These resistance-training studies involved a large number of contractions and might have produced some endurance-training stimulus. In our study, muscles were activated for 5 bouts of 5-s stimulation (5 $d \cdot wk^{-1}$ for 6 wk) producing fatigue. Short-duration fatiguing contractions such as we used are not known to produce changes in muscle endurance.

To assess fatigue from intermittent contractions, the decline in isometric and average concentric force and the decline in force during individual concentric contractions were measured. For high-intensity intermittent contractions, fatigue might be due to disturbances in excitation-contraction coupling, increases in intracellular H^+, increases in intracellular P_i (2,19), and decreased ion transport. In addition, a strong correlation between muscle intracellular H^+ and force during tetanic stimulation and recovery has been found (18). Some or all of these mechanisms could have been influenced by training and reduced the effect of metabolic fatigue. For example, chronic electrical stimulation and endurance training in humans resulted in reduced pH and P_i changes at the same exercise intensities (22). Our experimental design does not allow an evaluation of specific metabolic adaptations as a result of resistance training.

The force decline during concentric contractions has been attributed to 1) shortening-induced temporary decrease in the affinity of calcium bound to troponin when filament sliding occurs during muscle shortening (7), 2) an increase of low-force producing cross-bridges as high-force cross-bridges are reduced during muscle shortening (11), and 3) metabolic changes that are larger during concentric than isometric contractions (17). In this study, the decline in average concentric force was less following training (i.e., decreased fatigability), but we found an increase in the decline in force during the concentric contractions (i.e., increased fatigability). Because metabolic fatigue should alter the force decline during the concentric contractions to the same degree as the decline in average concentric force, training probably altered contractile properties by decreasing the affinity of binding calcium to troponin and/or increasing the population of low-force producing cross-bridges which appears while fatiguing skeletal muscles. As a result, a larger decline in force was observed in resistance trained muscles during concentric contractions during fatigue. Further support that altered contractile properties account for the force decline during concentric contractions is provided by the complete recovery within 5 min to initial values of the force decline. In contrast, the recovery of average concentric force (i.e., recovery of the metabolic and/or other factors) is not complete after 5 min (Fig. 3). Thus, the mechanism(s) responsible for the force decline during individual concentric contractions in a series of concentric contractions is altered during fatigue in resistance-trained muscles but not after 5 min of recovery.

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