Performance decrements with high-intensity resistance exercise overtraining

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ABSTRACT

FRY, A. C., W. J. KRAEMER, F. VAN BORSELEN, J. M. LYNCH, J. L. MARSIT, E. P. ROY, N. T. TRIPLETT, and H. G. KNUTTGEN. Performance decrements with high-intensity resistance exercise overtraining. *Med. Sci. Sports Exerc.*, Vol. 26, No. 9, pp. 1165–1173, 1994. The purpose of this investigation was to study a high-intensity resistance exercise overtraining protocol resulting in muscular strength decrements. Seventeen weight-trained males were divided into an overtraining group (OT; $N = 11$; $\bar{X} \pm$ SE, age = 22.0 ± 0.9 yr,) that exercised on a squat machine daily for 2 wk with 100% of 1 repetition maximum (RM) relative intensity, and a control group (CON; $N = 6$; age = 23.7 ± 2.4 yr) that exercised 1 $d \cdot wk^{-1}$ with low intensity (50% 1 RM). Test batteries were conducted at the beginning (test 1), after 1 wk (test 2), and after 2 wk (test 3) of the study. One RM performance significantly decreased from test 1 to test 3 ($P < 0.05$) for the OT group ($\bar{X} = -12.2 \pm 3.8$ kg), but not the CON group ($\bar{X} = -1.1 \pm 0.8$ kg). Isokinetic and stimulated isometric muscle force significantly decreased for the OT group compared with the CON group by test 3. The primary site of maladaptation appeared to be in the periphery as indicated by changes in stimulated force, circulating CK activity, and exercise-induced lactate responses. This protocol produced a significant decrease in 1 RM performance, thus providing a model for the study of short-term, high-intensity resistance exercise overtraining.

ISOMETRIC STRENGTH, ISOKINETIC TORQUE, ELECTRICAL MUSCLE STIMULATION, ONE REPETITION MAXIMUM

Optimal athletic performance is dependent in large part on a properly designed training program. High levels of performance, however, may be compromised by an inappropriate training regimen known as overtraining, and may result in an overtraining syndrome, characterized by performance plateaus or decrements. The physiology of overtraining is not entirely understood since it is difficult to monitor such a process until it has already occurred. Thus, the need exists for the development of overtraining exercise protocols to allow the study of overtraining phenomena in controlled laboratory conditions.

The stimulus of overtraining includes inappropriate rest and recovery from training (5,18,30) and results in increased levels of fatigue (5,6,30,34). Overtraining should be differentiated from muscular overstrain, which has been used to describe short-term fatigue that occurs immediately postexercise, and is recovered from in 1–2 d (30). For the purposes of this investigation, overtraining will be operationally defined as the training stimulus involving an increase in training volume and/or intensity that results in decreases in physical performance that are not due to muscular overstrain (5,30,40). The present investigation will focus on the role of exercise intensity. Numerous other terms have been suggested for the resulting overtraining syndrome, including burnout, staleness, overfatigue, and chronic overwork, among others (18,30,40). Overtraining is also different from planned phases of increased training volume and/or intensity called overreaching, from which recovery can occur within a short period, resulting in enhanced performance (5,18,30,40).

Overtraining may affect many physiological systems (5,18,30,40). Symptoms of the syndrome always include impaired physical performance, and may or may not include associated alterations of body mass, resting heart rate, sleep patterns, and mood states, to mention a few (5,34,40). An overtraining stimulus may attenuate only some types of physical performance, with these decrements occurring before many commonly listed symptoms (9). Therefore, it is unlikely that all the above listed symptoms are observed in the presence of performance decrements due to overtraining. It appears that different

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physiological profiles accompany overtraining in aerobic and anaerobic activities (6,30,42). Since most of the previously listed symptoms of an overtraining syndrome have been associated with aerobic exercise, it is possible that they may not be applicable to overtraining with anaerobic exercise. It has also been suggested that training for highly anaerobic activities can result in a sympathetic overtraining syndrome with increased sympathetic activity at rest (18,40), whereas aerobic overtraining may produce at parasympathetic overtraining syndrome with decreased sympathetic activity at rest (6,30), further evidence of different etiologies for aerobic and anaerobic overtraining syndromes.

Although increased volume aerobic overtraining with significant performance decrements has been studied (31), a protocol for the study of overtraining with high-intensity resistance exercise has not been successfully developed. Only one resistance exercise investigation has attempted to isolate the effects of increased training intensity from those of increased training volume (16), but was unable to develop a definitive state of overtraining. The development of a resistance exercise overtraining protocol would allow the examination of physiological events associated with specific sites of action during overtraining. From previous investigations and pilot work, it was found that consecutive days of exercise using greater numbers of repetitions with maximal relative intensity (100% 1 repetition maximum) were required to elicit an overtraining response (16). Therefore, the purposes of the present investigation were to determine the efficacy of a high-intensity resistance exercise protocol for producing performance decrements, and to determine the time course and sites of physiological adaptation accompanying high-intensity resistance exercise overtraining.

METHODS

Seventeen weight-trained males were randomly divided into either an overtraining (OT; $N = 11$) or a control group (CON; $N = 6$). All subjects had consistently performed weight training for the legs for a mean of 4.5 yr preceding the study, were capable of at least a 1.5 × body weight 1 repetition maximum (RM) for the parallel back squat (36), and reported no history of anabolic steroid use over the previous year. Prior to participation in the study, all subjects signed an informed consent statement as approved by the Pennsylvania State University Institutional Review Board, and were medically screened for orthopedic abnormalities of the knee by a physician (32). Subject characteristics for each group are presented in Table 1.

TABLE 1. Subject characteristics ($\bar{X}$±SE) for the overtraining (OT) and control (CON) groups. No significant changes occurred for any of these variables from test 1 to test 3 ($P > 0.05$).

Variable	OT		CON	
	Test 1	Test 3	Test 1	Test 3
N	11	—	6	—
Age (yr)	22.0 ± 0.9	—	23.7 ± 2.4	—
Height (cm)	176.6 ± 2.1	—	175.7 ± 3.1	—
Body weight (kg)	83.4 ± 4.9	83.4 ± 4.8	81.2 ± 5.6	81.2 ± 5.6
Relative fat (%)	12.2 ± 1.9	12.2 ± 2.0	10.0 ± 2.1	9.7 ± 2.1
Lean body mass (kg)	72.2 ± 2.5	74.5 ± 2.5	72.7 ± 4.3	73.1 ± 4.5
1 Repetition maximum (kg) (2nd familiarization session)	116.7 ± 5.6	—	122.5 ± 10.1	—
Leg weight training experience (yr)	4.0 ± 1.0	—	5.7 ± 2.5	—

Lower body training was performed on a squat resistance exercise machine (Tru-Squat, Southern Xercise, Inc., Cleveland, TN), using a shoulder width stance with the feet angled laterally 15–30° (36). Foot positions were held constant throughout the study with the aid of numbered scales secured to the foot platform. The torso was secured by a belt and a padded shoulder yoke to a rigid vertical padded board. Proper exercise range of motion was signalled by a magnetically activated electronic buzzer that sounded when a parallel position was attained (i.e., the greater trochanter level with the knee joint center). Exercise on this particular squat machine is not identical to barbell squats, but does primarily involve the hip and knee extensor muscle groups (15). This device was appropriate for this study since it uses a large muscle group, multiple joint activity, within a regulated joint range of motion. Due to the low exercise technique requirement, a rapid familiarization process was possible. Subject safety was ensured since a single person could easily assist the subject during a failed repetition. This machine has also been used in a previous resistance exercise overtraining study (16).

Lower body resistance exercise was controlled in the present study, whereas current upper body resistance exercise programs were held constant. If upper body resistance exercise was terminated, it would have resulted in a considerable decrease in the total training stress, thus possibly confounding the results of the study. Two familiarization sessions (F1 and F2) on the squat machine were used for all subjects during the first week. Both F1 and F2 also included a 1 RM assessment using a previously recommended progression (28). One RM test reliability determined from F1 and F2 was r = 0.96, with mean differences between F1 and F2 = 4.4 kg.

During weeks 2 and 3 of the study, the OT group performed resistance exercise at a maximal relative intensity (100% 1 RM) and a low volume of exercise (10 sets × 1 repetition = 10 total repetitions) in order to induce an overtraining syndrome. After a controlled warm-up (1 × 5–32 kg, 1 × 5–40% 1 RM, 1 × 3–60% 1 RM, 1 × 1–80% 1 RM), OT subjects were tested for their current 1 RM each training day, typically requiring 2–4 additional repetitions after the warm-up. They then performed a total of 10 single repetitions at 100% of their 1 RM on the squat machine with 2 min of rest between each attempted lift. If any attempted repetition was unsuccessful, the resistance for subsequent repetitions was decreased by 4.5 kg, allowing each successful lift to be as close to each subject's maximal capability as possible. This protocol was based on previous research attempting to produce resistance exercise overtraining (16). Although this protocol was used as a tool to elicit an overtraining response with concomitant performance decrements, similar exercise protocols (i.e., multiple 1 RM lifts) have been used for physiological investigations (20,22,23), as well as for daily sports training (1,19, personal observations). Variables recorded for each OT group exercise session included total joules (J; load × distance) of work successfully performed, and percent fatigue from the heaviest lift to the last lift. The CON group performed a low volume, low relative intensity exercise session one day each week using the following protocol; 1 × 5–32 kg, 1 × 5–40% 1 RM, 3 × 5–50% 1 RM. A second day each week was devoted to 1 RM testing. All exercise sessions were monitored by at least one member of the research team.

Test batteries were administered before, during, and after the 2nd and 3rd weeks of the study. Each battery consisted of voluntary isometric and isokinetic, and stimulated isometric quadriceps torque on Friday evenings, followed by muscular endurance tests at 70% of 1 RM and blood sampling on Saturdays. Body composition was estimated anthropometrically (26,38), with all skinfold measurements taken by the same investigator who demonstrated reliabilities of $r \geq 0.94$ for each site.

One RM capabilities on the squat machine were determined for all subjects using the same warm-up progression as was used for the OT exercise sessions (28). Subjects were also tested for concentric leg extension peak torque (N·m) of their dominant leg at angular limb velocities of 0.00, 0.53, and 5.24 $\text{rad}\cdot\text{s}^{-1}$ on a Cybex II isokinetic dynamometer (Lumex, Inc., Ronkonkoma, NY). The dynamometer was calibrated before each test battery according to the manufacturer's specifications (10). Isometric leg extension testing was performed at 90° of knee flexion (0° = leg fully extended) for a 5 s maximal effort (28). This knee angle was selected to allow comparison with the accompanying nerve stimulation test. Dynamometer damping for this test was set at 2. Although these tests are not specific to the exercise modality, they were included to determine velocity specific force characteristics that may result from the overtraining exercise protocol.

Stimulated quadricep force production was also assessed at 90° of knee flexion after each subject completed the voluntary isometric assessment. Surface stimulation was applied to the femoral nerve with a Teca Electromyograph/Synchronized Stimulator with built-in oscilloscope (Model B-2) and a Teca 9523 hand-held stimulator at the inguinal fold, immediately distal to the inguinal ligament (12). Maximal muscle activation of the vastus medialis was determined via electromyographic (EMG) activity, using two silver/silver-chloride electrodes with a built-in preamplifier, and a constant interelectrode distance of 2 cm. Maximal activation of the quadriceps muscle group was verified by the amplitude of the raw EMG signal displayed on an oscilloscope. Beginning with single rectangular wave stimulations of 0.05-ms duration, the nerve stimulation was increased in steps of approximately 60 V, followed by duration increases to a maximum of 2.0 ms until the EMG signal did not increase in amplitude. Supramaximal stimulation of 30–60 V greater than the stimulation of maximal activation was used to ensure complete activation. Isometric peak torque due to the supramaximal stimulation was determined in triplicate on the Cybex dynamometer, with all trials producing identical results. Dynamometer damping was set at 0 due to the extremely short duration of the stimulated muscle activity. All muscle force assessments were performed at the same time of day for each test battery, and were at least 3 h postexercise session to permit acute neural recovery (25). After test 1, training protocols prior to the stimulated force test were held constant since short-term training status can affect efficiency of electrical activity of skeletal muscle (14).

The maximum number of repetitions at 70% of 1 RM were performed on the squat machine on Saturday for each test battery, preceded by a 6-h fast with water *ad libitum*. The time of day for each test was constant for each subject to minimize diurnal variations. After a warm-up of three repetitions at 32 kg, sets of 10 continuous repetitions at 70% of the most recent 1 RM load, separated by 1 min of rest, were performed until failure. Total repetitions, as well as total work (J), were recorded for each test. Upon reporting for the 70% 1 RM test session, a 20-gauge Teflon catheter with four-way stopcock was inserted in a superficial antecubital vein while the subject was kept in a calm seated position similar to the terminal position on the squat machine for a minimum of 30 min preexercise. The cannula was kept patent with a continuous saline drip (0.9% sodium chloride) at a rate of approximately 45 $\text{ml}\cdot\text{h}^{-1}$. Blood samples were taken 15 min preexercise (Pre 1) for determination of serum total creatine kinase (CK), and immediately preexercise (Pre 2) to determine resting whole blood lactate (Lac^-). After the 70% 1 RM test for repetitions, the subjects were seated and a 5 min postexercise (5 post) blood draw was obtained to determine exercise-induced blood Lac^- responses.

Pre 1 blood samples were allowed to clot at room temperature, and were centrifuged at 3000 g for 15 min. A 1-ml aliquot of the resulting serum was frozen at –90°C for later analysis. Samples were thawed only once for analysis, and decoded after all analyses were completed (blinded analysis). Serum CK activity was determined in duplicate using a kinetic assay with spectrophotometric absorbance at 340 nm at an adjusted temperature of 30°C (Sigma Diagnostics, St. Louis, MO). Absorbance was determined with a Pharmacia LKB Biotechnology Novaspec II visible spectrophotometer (Uppsala, Sweden). Intraassay variance for serum creatine kinase was 6.0%, with interassay variance eliminated by analyzing all samples in the same assay. Pre 2 and five postblood samples were mixed with sodium heparin in a chilled vacutainer. A 0.5-ml aliquot was immediately used for duplicate enzymatic amperometric analysis for Lac^-, using a YSI 1500 Sport L-lactate analyzer (Yellow Springs, OH), which was calibrated prior to use.

Prior to each exercise session, including the 70% 1 RM test session, subjects completed an abridged profile of mood states (POMS) (11). In addition, a questionnaire including several additional questions regarding self-perception of physical status were given to the subjects. This questionnaire has been pretested on similar subjects in a previous study (16) and was found to be suitable for the present investigation (4). After instruction on monitoring their heart rates and assessment for accuracy by a member of the research team, each subject kept a record of morning heart rate upon awakening, as well as sleep patterns (i.e., hours of sleep and sleep quality; 5 = excellent, 4 = good, 3 = fair, 2 = poor, 1 = restless). Dietary records were kept for the day prior to each 70% 1 RM test. All subjects were instructed on proper nutritional recording, including estimating portion sizes. Total kcal and % protein, fat, and carbohydrates were determined using the Macdiet Professional dietary computer program (version 2.0, Drexel University, Philadelphia) and were analyzed by the same investigator throughout the study. Figure 1 describes the exercise and test schedule for both groups.

Subject characteristics ($\bar{X} \pm SE$) for each group were compared with independent *t*-tests. Responses of all performance variables for both groups were compared with mixed model analyses of variance with Fisher's least significant difference *post-hoc* tests. When the Kolmogorov-Smirnov test for normality of distribution showed this assumption was violated, nonparametric chi-square analysis was used. Relationships between changes in performance variables were determined with Pearson product-moment correlation coefficients. The level of significance for all statistical procedures was $P = 0.05$.

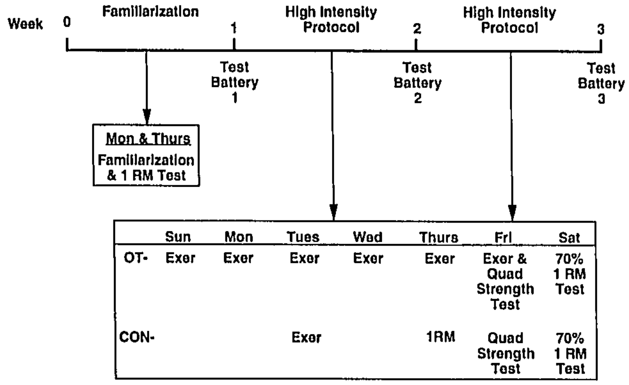

Figure 1—Exercise and test session time line for the overtraining (OT) and control (CON) groups. Each test battery consisted of the following; quadriceps strength test (Friday evening)—voluntary isokinetic and isometric, and stimulated isometric quadriceps torque; 70% 1 RM test (Saturday)—maximal repetitions on the squat machine, blood sampling, anthropometric measures, dietary reports, and profile of mood states and questionnaires. Each exercise session for the OT group included a 1 RM test, a profile of mood states, and a questionnaire. Each exercise and 1 RM session for the CON group included a profile of mood states and a questionnaire.

TABLE 2. A 2 × 2 chi-square contingency table categorizing decreases in 1 repetition maximum (RM) strength over the course of the 2-wk study for both the overtraining (OT) and the control (CON) groups.

	Decrease in 1 RM	
Group	≥4.5 kg	<4.5 kg
OT (*N* = 11)	8 (73%)	3 (27%)
CON (*N* = 6)	0 (0%)	6 (100%)

Categories for decreases (i.e., ≥4.5 kg and <4.5 kg) are based on mean absolute differences in 1 RM values between the first and second familiarization sessions ($\bar{X}$ = 4.4 kg). Changes for the OT group were significantly different from those for the CON group ($\chi^2 = 8.24$, $P < 0.005$).

RESULTS

Using nonparametric chi-square analysis, changes in 1 RM were significantly different for the OT group compared with the CON group, with more OT subjects experiencing decreased 1 RM capabilities compared with the CON group ($\chi^2 = 8.24$, $P < 0.005$; see Table 2 and Fig. 2). Grouping for changes in 1 RM (≥4.5 kg or <4.5 kg) was based on the mean absolute changes of the combined groups for 1 RM when comparing the two familiarization sessions ($\bar{X}$ absolute change = 4.4 kg). Changes of ≥4.5 kg represented differences larger than the expected test-retest variability. Subjects were then classified by their individual changes in 1 RM (max 1 RM to last 1 RM) and their exercise group assignment. The Yate's correction for continuity was not applied to this procedure due to the resulting unnecessary loss of power (7). In addition, independent *t*-tests indicate that the decreased 1 RM for the

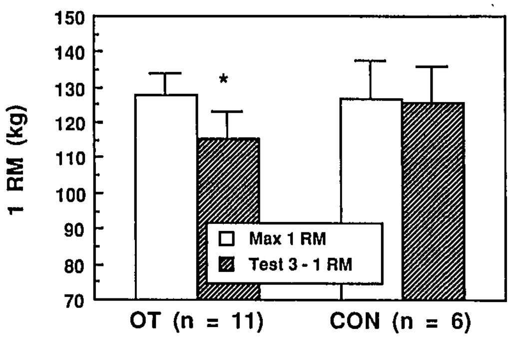

Figure 2—One repetition maximum (RM) performances on the squat machine for the OT and CON groups ($\bar{X} \pm SE$). Maximum 1 RM values are reported rather than test 1 values since some subjects demonstrated maximal 1 RM performances after several exercise sessions. * Indicates patterns of strength decrements different from CON group ($\chi^2 = 8.24$, $P < 0.005$).

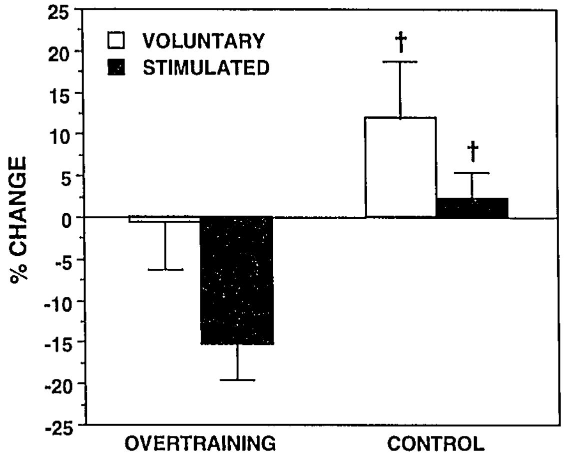

Figure 4—Percent changes ($\bar{X}$ ± SE) from test 1 to test 3 of isometric (0 rad·s^{-1}) leg extension torque during maximal voluntary actions and maximal nerve stimulated actions. † Different from overtraining (OT) group ($P < 0.05$).

OT group ($\bar{X}$ = −12.2 kg) was significantly greater than for the CON group ($\bar{X}$ = −1.1 kg).

Body weight, relative fat (or sum of skinfolds), and lean body mass for both groups of subjects were similar and did not change during the course of this investigation (see Table 1). Two of the subjects in the present study were medically diagnosed with overuse syndrome of the knees by the end of the overtraining protocol, but were included in all analyses since they completed all training and test sessions. Low (0.53 rad·s^{-1}) and high (5.24 rad·s^{-1}) velocity isokinetic leg extension torque, as well as stimulated torque (0 rad·s^{-1}), all demonstrated significant decreases for the OT group by tests 2 and 3, and were also significantly less than the CON group performances by tests 2 and 3. Voluntary isometric (0 rad·s^{-1}) leg extension torque did not significantly decrease for the OT group, but like the other quadricep strength measures, was significantly less than the CON group performances by tests 2 and 3 (Fig. 3). Comparisons of %Δ for both voluntary and stimulated isometric leg extension torque (see Fig. 4) demonstrate a significant decrease in stimulated torque for the OT group ($\bar{X}$ = −15.3%), and a significant increase in voluntary isometric torque for the CON group ($\bar{X}$ = +11.9%).

Variables measured during the 70% 1 RM test are listed in Table 3. The OT group demonstrated significant decreases during the course of the study for total work (J) and postexercise Lac$^-$, whereas total CK significantly increased, as well as total and carbohydrate caloric intakes. Absolute changes in 1 RM during the entire study (test 1 − test 3) were negatively related to relative changes (%Δ) in repetitions at 70% 1 RM (r = −0.91) for the OT group, whereas this relationship for the CON group was significantly positive (r = 0.65).

Exercise session variables during the first exercise session for the OT group were % fatigue = 15.2 ± 2.4%, and total work = 5,521.9 ± 353.8 J. Other characteristics reported at the first exercise session were hours of sleep (OT = 7.7 ± 0.3 h; CON = 7.7 ± 0.4 h), sleep quality (OT = 4.3 ± 0.2; CON = 3.8 ± 0.4; see methods for

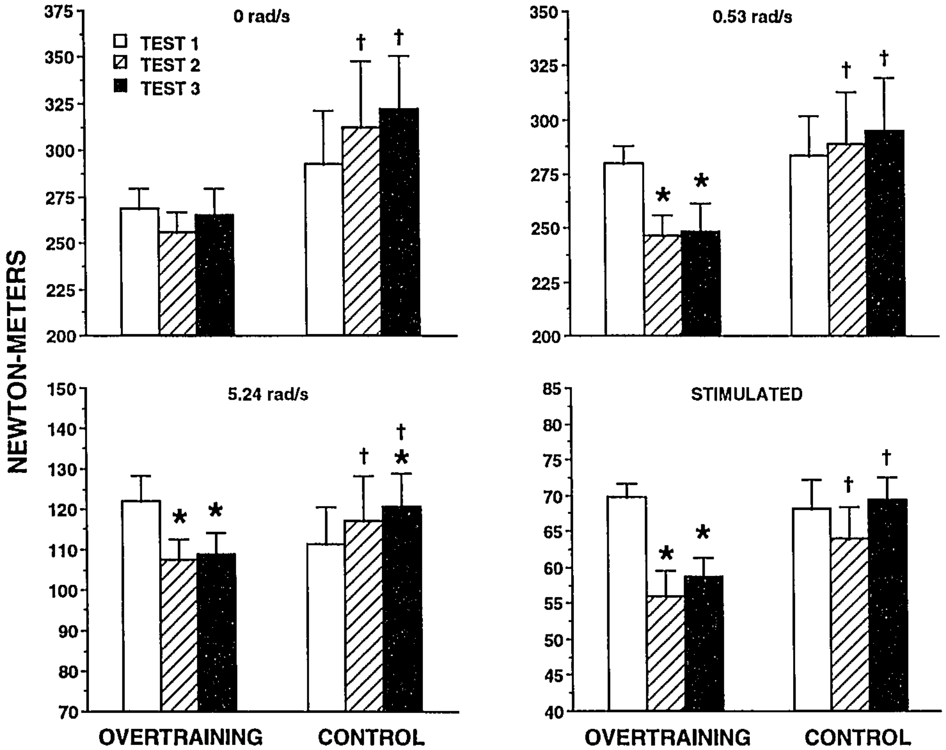

Figure 3—Torque production (N·m) during voluntary isometric (0 rad·s^{-1}) and isokinetic (0.53 and 5.24 rad·s^{-1}), and involuntary stimulated (0 rad·s^{-1}) leg extension tests ($\bar{X}$ ± SE). * Different from test 1; † different from overtraining (OT) group ($P < 0.05$).

TABLE 3. 70% 1 RM test variables ($\bar{X}$±SE) for the overtraining (OT) and control (CON) groups.

Variable	Group	Test 1	Test 2	Test 3
Repetitions				
	OT	27.2 ± 2.3	27.3 ± 3.0	30.2 ± 4.4
	CON	30.2 ± 4.6	30.5 ± 3.7	33.2 ± 3.7
Work (J)				
	OT	11,627.1 ± 1,199.0	10,802.8 ± 682.7	11,859.0 ± 921.7
	CON	13,267.7 ± 1,730.2	13,758.2 ± 1,623.6†	15,420.9 ± 2,028.3†
Lactate (mmol·l^{-1})				
	OT			
	Pre	1.04 ± 0.08	0.80 ± 0.07	0.68 ± 0.04
	Post	10.93 ± 0.56	8.63 ± 0.52*	9.64 ± 0.49*
	CON			
	Pre	0.93 ± 0.14	0.73 ± 0.08	0.68 ± 0.04
	Post	9.45 ± 0.83†	9.91 ± 0.67†	9.92 ± 0.87
Creatine Kinase (IU·l^{-1})				
	OT	337.7 ± 45.8	661.4 ± 96.1*	613.1 ± 129.1*
	CON	299.4 ± 79.8	342.1 ± 96.3†	299.4 ± 92.5†
24 h dietary recall total kcal				
	OT	2,853.6 ± 363.5	2,953.2 ± 260.4	3,696.9 ± 395.0*
	CON	2,402.7 ± 294.3	2,786.0 ± 474.8	2,775.2 ± 621.2
Protein kcal				
	OT	476.1 ± 102.4	523.9 ± 56.6	520.1 ± 88.4
	CON	347.2 ± 141.7	415.6 ± 102.8	348.3 ± 101.1
Fat kcal				
	OT	842.6 ± 165.2	789.5 ± 96.9	1,066.9 ± 191.4
	CON	913.1 ± 171.8	955.4 ± 253.8	855.4 ± 317.5
Carbohydrate kcal				
	OT	1,550.1 ± 176.3	1,667.9 ± 198.0	2,122.1 ± 166.8*
	CON	1,167.2 ± 168.2	1,466.4 ± 259.3	1,595.7 ± 290.6†

* Different from test 1 ($P < 0.05$).
† Different from OT group ($P < 0.05$).

scale), and morning HR (OT = 58.8 ± 2.7 bpm; CON = 67.3 ± 5.5 bpm). None of these variables demonstrated significant changes during the course of this investigation. The OT group reported a slightly decreased perception of anxiety on the abridged POMS by the end of the study (test 1 = 1.5 ± 0.2, test 3 = 0.7 ± 0.2). The OT group also significantly decreased perceptions of strength (test 1 = 2.2 ± 0.3, test 3 = 1.5 ± 0.2) and recovery from their previous exercise session (test 1 = 2.5 ± 0.2, test 3 = 1.4 ± 0.2). Perceptions of increased leg muscle soreness for the OT group were not reported, although perceptions of soreness of the knees and lower back increased by the last several days. All questions were answered on a Likert scale of 0 = not at all, 1 = a little, 2 = moderately, 3 = quite a bit, 4 = extremely.

DISCUSSION

The most critical finding of the present investigation was that, for the first time, high-intensity resistance exercise overtraining was induced in a controlled scientific setting, as indicated by the attenuated 1 RM performance ($\bar{X} = -12.2$ kg) for the OT group. Previous attempts to induce resistance exercise overtraining with increased training volume (17) have resulted in no performance decrements, although increased relative intensity resistance exercise (16) and increased training volumes for other anaerobic activities (e.g., judo) have been detrimental to isokinetic strength and sprint times (6). The controlled overtraining protocol utilized permitted study of the development of an overtrained state that could be attributed in large part one particular acute training variable, the relative intensity (% 1 RM) of the exercise. Furthermore, gross motor patterns were controlled by the squat machine throughout the investigation, thus eliminating a possible mechanism of preserving 1 RM performance (37). A follow-up survey determined that 2–8 wk elapsed before members of the OT group could utilize previous levels of resistance for their normal leg resistance exercise training programs, further evidence that overtraining actually occurred rather than short-term muscular overstrain.

Besides decreased 1 RM performance, strength decrements were also evident for both isokinetic and stimulated isometric measures. Even when voluntary isometric strength did not significantly decrease for the OT group, it had become significantly less than for the CON group by the end of the study, although this appeared to be mostly due to nonsignificant increases for the CON group. These strength decrements were accompanied by a decreased perception of strength reported in the daily questionnaire for the OT subjects. The fact that voluntary leg extension torque was only attenuated when dynamic activity was tested suggests that proprioceptive afferent input may have been affected, thus decreasing dynamic torque production (21). These attenuated muscular strength performances for the OT group provide evidence of the efficacy of the high-intensity resistance exercise protocol for eliciting an overtrained state. Although the CON group increased quadriceps torque at 5.24 rad·s^{-1}, small nonsignificant torque increases were observed for all voluntary efforts. It is likely that this trend was the result of a learning effect for the CON group that was simply overwhelmed by the overtraining protocol for the OT group. It should be noted that the CON group demonstrated no changes for 1 RM performances, indicating that the abbreviated training protocol for this group was effective in maintaining 1 RM strength.

Comparisons of voluntary versus stimulated isometric torque production at identical anatomical positions provide insight as to the site of action (central vs peripheral) for force production decrements (3). Maximal voluntary isometric torque is regulated by both central (proximal to the point of stimulation) and peripheral factors (distal to the point of stimulation). These stimulated torques were of short enough duration ($\leq$2 ms) to avoid sensations of pain for all subjects and to eliminate contributions of spinal reflexes to the resulting quadriceps torque production. Although activation characteristics of only the vastus medialis were monitored, according to standard clinical procedures (12), other heads of the quadriceps muscle group possess comparable fiber type profiles (27), suggesting that activation characteristics of the vastus medialis would be representative of the entire muscle group. Central input via descending pathways were avoided by cutaneously stimulating the peripheral motor nerve. Therefore, the decreased stimulated torque for the OT group indicates that peripheral factors were responsible for this decrement. Voluntary isometric force, however, did not change for the OT group, suggesting that central factors may be counteracting compromised peripheral strength regulation. On the other hand, central input was responsible for increased voluntary force production for the CON group, since force production increased in the absence of peripheral changes. Although not monitored in the present study, these central factors could have included recruitment patterns, sympathetic activity, or psychological factors. Stimulated torques ranged from 20 to 26% of maximal voluntary isometric torque since such short stimulus durations would permit only the fastest contracting muscle fibers to contribute to the measured force production. Therefore, the stimulated force test may only be indicative of changes in the fastest contracting fibers of the quadriceps. Although changes in muscle fiber contractile characteristics can occur with chronic resistance exercise, perhaps affecting stimulated force production, greater than 2 wk is required for these adaptations in males (39). Therefore, peripheral factors other than fiber type characteristics are most likely responsible for the strength decrements observed.

These findings provide important direction for future investigations of high-intensity resistance exercise overtraining. It appears that the site of action of short-term, high-intensity resistance exercise overtraining is primarily in the periphery, although central input increased in an attempt to maintain voluntary force production. Several studies have focused on central factors (e.g., neural) that may account for attenuated strength performances (14,24,25), which may be the case for normal training. However, decreased performance with high-intensity training of such short duration may be dependent on alterations at a peripheral site such as the skeletal muscle cell, or neuromuscular junction level (2).

Further evidence of peripheral alterations is provided by the decreased exercise-induced blood Lac^- responses for the OT group. Similar decreases in exercise-induced Lac^- responses have been observed with increased volumes of resistance exercise (17). It has been speculated that decreased Lac^- production may be caused by inadequate muscle glycogen stores, due in part to a low dietary caloric intake (8). Increased caloric and carbohydrate intakes for the OT group suggest that dietary causes for the decreased Lac^- response are unlikely, although actual muscle glycogen content must be directly measured in order to adequately address this question. It is unlikely that the decreased exercise-induced Lac^- response was part of a positive adaptation to the exercise program. The low volume, high-intensity overtraining protocol was not designed to emphasize glycolytic metabolism. Furthermore, if the 70% 1 RM test for repetitions was sufficient to elicit a training response, it should have been evident for both the OT and CON groups, since all subjects performed these tests.

Large levels of muscle-tissue damage for the OT group are not evident, since muscle damage studies report dramatically greater CK activity levels of up to 34,000 $IU \cdot L^{-1}$ after exercising smaller muscle groups (i.e., elbow flexors) (13). The CK activity levels observed in the present study are similar to values observed after a typical resistance exercise training session with trained subjects (29). They may also be indicative of muscle-tissue disruption resulting in decreased muscle glycogen stores, thus contributing to the decreased lactate responses observed (35). Since evidence exists of glycogen depletion of only type II fibers with high-intensity resistance exercise (41), disruption may have been specific to these fibers, resulting in the attenuated maximal force production for the OT group. Lower intensity exercise, such as the 70% 1 RM test for repetitions, would be unaffected since the highest threshold motor units would not be activated throughout the activity. The relatively low levels of muscle disruption were also apparent from the daily questionnaires, with a self-reported lack of delayed onset muscle soreness for the OT subjects.

An important finding is that the OT group exhibited a strong negative relationship ($r = -0.91$) between absolute changes (kg) in 1 RM capabilities and changes in local muscular endurance for the entire study (test 1 − test 3). The CON group exhibited a significant positive relationship ($r = 0.65$) as was expected. Thus, the OT subjects with the greatest 1 RM decrements did not have similar decrements for local muscular endurance (i.e., 70% 1 RM for repetitions). It should be noted that tests of local muscular endurance, such as maximal repetitions at 70% 1 RM, are not adequate indicators of 1 RM decrements with the overtraining protocol used in the present study.

Although numerous symptoms have been suggested as characteristics of overtraining due to aerobic exercise or increased volumes of resistance exercise (5,18,30,40,42), no changes were apparent for body weight, relative fat, lean body mass, sleep patterns (i.e., hours and quality), or morning heart rate during the 2 wk of high-intensity resistance exercise overtraining. This concurs with previous weight-training studies that have not observed altered physical characteristics with increased training stresses (16,17), although other anaerobic activities (i.e., judo) have resulted in decreased relative body fat as determined anthropometrically (6). Even in the presence of aerobic exercise induced overtraining, commonly accepted symptoms such as altered cardiac function do not necessarily occur (31). Exercise session % fatigue and total work also did not change during the 2 wk, despite decreased muscle force production capabilities. The abridged POMS indicated that slight decreases in anxiety had developed by the end of the study, which is contrary to previous reports on overtraining (5,18,40). Overall, an "inverted iceberg profile" did not develop as has been suggested (5), and POMS variables were not associated with performance decrements for the high-intensity, low volume resistance exercise protocol used in the present study, as they are for high-volume aerobic exercise (33,34). Training session questionnaires filled out each day indicated the OT subjects did not look forward to the daily exercise sessions, and reported decreased perceptions of strength and recovery. In general, however, a number of the often cited symptoms of overtraining associated with aerobic exercise or increased volumes of resistance exercise did not occur with this high-intensity overtraining protocol. This serves to reinforce the theory that overtraining for different types of physical activity is manifested through different physiological systems (42).

The major finding of this investigation was that it was possible to induce 1 RM performance decrements using a high-intensity, low-volume resistance exercise overtraining protocol for the lower body with previously weight-trained males. Along with decreased 1 RM values, stimulated isometric and voluntary isokinetic muscle force capabilities were also decreased. The primary site of the short-term strength maladaptation with this overtraining model appears to be in the periphery as indicated by nerve stimulation tests, circulating CK activity, and decreased exercise-induced Lac$^-$ responses. Many typically cited symptoms of overtraining were not manifested, emphasizing the unique characteristics of high-intensity overtraining. The onset of performance decrements resulting from overtraining using the high-intensity resistance exercise protocol was quite rapid, with most physiological maladaptations developing within 1 wk of the initiation of the high-intensity protocol. By localizing the site of initial physiological maladaptations, future studies can begin to focus on a more detailed understanding of the specific mechanisms that mediate these changes. In addition, coaches and athletes must be aware that indices of overtraining with high-intensity resistance exercise may not be identical to those associated with other variations of exercise.

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