
ELIMINATION OF DELAYED-ONSET MUSCLE SORENESS BY PRE-RESISTANCE CARDIOACCELERATION BEFORE EACH SET

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ABSTRACT

We compared delayed-onset muscle soreness (DOMS) induced by anaerobic resistance exercises with and without aerobic cardioacceleration before each set, under the rationale that elevated heart rate (HR) may increase blood perfusion in muscles to limit eccentric contraction damage and/or speed muscle recovery. In two identical experiments (20 men, 28 women), well-conditioned athletes paired by similar physical condition were assigned randomly to experimental or control groups. HR (independent variable) was recorded with HR monitors. DOMS (dependent variable) was self-reported using Borg's Rating of Perceived Pain scale. After identical pre-training strength testing, mean DOMS in the experimental and control groups was indistinguishable ($P \geq 0.19$) for musculature employed in eight resistance exercises in both genders, validating the dependent variable. Subjects then trained three times per week for 9 (men) to 11 (women) weeks in a progressive, whole-body, concurrent training protocol. Before each set of resistance exercises, experimental subjects cardioaccelerated briefly (mean HR during resistance training, 63.7% HR reserve), whereas control subjects rested briefly (mean HR, 33.5% HR reserve). Mean DOMS among all muscle groups and workouts was discernibly less in experimental than control groups in men ($P = 0.000019$) and women ($P = 0.0007$); less for each muscle group used in nine resistance exercises in both genders, discernible ($P < 0.025$) in 15 of 18 comparisons; and less in every workout, discernible ($P < 0.05$) in 32% (men) and 55% (women) of workouts. Most effect sizes were moderate. In both genders, mean DOMS per workout disappeared by the fourth week of training in experimental but not control groups. Aerobic cardioacceleration immediately before each set of resistance

exercises therefore rapidly eliminates DOMS during vigorous progressive resistance training in athletes.

KEY WORDS muscle recovery, concurrent training, resistance training, weight training, progression, aerobic exercise, integrated

INTRODUCTION

Delayed-onset muscle soreness (DOMS) is a familiar and widespread adverse consequence of unaccustomed or unusually intense physical activity or exercise. Symptoms can appear within hours, persist for as long as several days, and include pain (41), compromised muscle physiology and recovery (10,18,36,43), reduced performance (18,19), increased risk of further injury (55), and reduced adherence to exercise programs (34,45). Substantial research has therefore been devoted to strategies aimed at reducing DOMS, including behavioral approaches (8,23,60,61), diet (12), supplements (37,54), systemic analgesics (3,38), and technological approaches (5).

Despite this research, DOMS has proved resistant to amelioration. The most effective strategy for reducing DOMS remains preceding exercise, termed the "repeated bout effect" (44,47). The most successful behavioral strategies include yoga (8) and compression therapy (33), whereas the most effective dietary strategies include ingestion of the phospholipid phosphatidylserine (30) or a carbohydrate-protein-antioxidant beverage (53). Supplements and analgesics have proven generally ineffective, although some technological approaches to DOMS reduction, such as transcutaneous nerve stimulation and its combination with cold (16), have reduced DOMS moderately. Most strategies reported to reduce DOMS are ameliorative rather than preventive, none has been shown directly to influence muscle recovery, and none has eliminated DOMS entirely.

Finding a simple, effective, and inexpensive antidote and/or prevention for DOMS could help to eliminate a major barrier to physical conditioning and exercise adherence at all levels. Moreover, any process or procedure that reduces or prevents DOMS may imply more rapid muscle recovery after an exercise stimulus, which could benefit exercisers at all

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levels—competitive athletics, recreational, and rehabilitative. In a research context, the discovery of an effective remedy for DOMS could lead to a better understanding of its causes. We therefore explored and report herein a new approach to the management and elimination of DOMS induced by resistance training; namely, aerobic elevation of heart rate (HR), or cardioacceleration, immediately before each set of anaerobic resistance exercises.

The rationale for this approach to DOMS reduction is based on its probable causes. DOMS is induced by eccentric or lengthening muscle contraction. In both human and animal models, eccentric contraction immediately damages the muscle cytoskeleton and reduces contractile tension (20,39). Eccentric contraction also immediately compromises the muscle membrane system, interfering with excitation-contraction coupling (58), and alters the phospholipid membrane structure (22) and function (2,49). Eccentric exercise disrupts the sarcomere and myofibrils (e.g., 9 and 41, but see 46). The collective cellular injury generates free radicals (6,36), edema (10), and inflammation products (17,42) that act as cellular mediators (24) to alter muscle sensory systems (42), increase afferent discharge in muscle sensory receptors (56) and register centrally as the perception of DOMS.

Elevated HR could intervene at any stage in this presumptive causal cascade to reduce DOMS and speed muscle recovery by increasing cardiac output and systolic blood pressure (1), enhancing muscle perfusion, and re-supplying nutrients and other substances and clearing waste and injury products faster. Resistance exercises that induce DOMS generally do not increase HR significantly (1), however, because they are largely anaerobic, and the increase in blood pressure caused by resistance exercises is brief and transient (40,48). In contrast, brief bouts of aerobic exercise performed immediately before each set of resistance exercises can accelerate HR significantly, increasing cardiac output and elevating systolic blood pressure chronically (1). Elevated HR could combine with exercise hyperemia (35,57) to increase muscle perfusion, accelerate sarcolemmal materials flux, reduce or prevent eccentric contraction injury, promote faster muscle recovery, and reduce or prevent DOMS.

METHODS

Experimental Approach to the Problem

Under this experimental rationale, we prospectively tested the hypothesis that aerobic cardioacceleration immediately before every set of resistance exercises reduces DOMS. Subjects matched as closely as possible were divided randomly into experimental and control groups for several weeks of concurrent exercise training. Experimental subjects performed brief, vigorous aerobic exercise immediately before each set of resistance exercises to elevate their HR during subsequent resistance training to 60–84% of HR reserve (HRR), typical of vigorous aerobic exercise and higher than can be achieved generally by conventional anaerobic

resistance exercises (1). Control subjects performed the same resistance exercises at the same intensity, ensuring identical eccentric muscle loading, but they rested briefly before each set of resistance exercises to lower their HR during the subsequent resistance training to 20–39% of HRR, typical of anaerobic resistance exercises. Therefore, the primary difference between experimental and control groups was HR.

In both experiments, subjects reported DOMS for muscle groups used in every resistance exercise in every workout during several weeks of concurrent exercise training. The dependent variable (self-reported DOMS) was validated by comparing DOMS induced by strength testing before and after resistance training, which was not discernibly different between the experimental and control groups. The independent variable (HR) was validated by recording HR during resistance training and calculating mean values following the training program, which fell within the prescribed ranges for experimental and control groups. The null hypothesis (elevated HR does not affect DOMS) was rejected in favor of the alternative hypothesis (elevated HR reduces DOMS) if the mean DOMS reported by experimental groups during resistance training was discernibly less than the mean DOMS reported by control groups at the 5% level ($P < 0.05$) and effect sizes (ES) were at least moderate.

Subjects

Identical experiments were conducted in successive years with 20 men and 28 women, undergraduate student-athletes aged 18–22 years. The institutional review board evaluated and approved all aspects of the research program before implementation. After the recruitment of prospective subjects from university sport teams, the experiment was explained in a group setting without divulging its purpose. Each prospective subject then signed a witnessed informed consent statement describing the risks, benefits, and responsibilities of participation, and the option to withdraw at any time without prejudice. Prospective subjects then underwent laboratory blood tests (lipid panel, electrolytes, fasting blood glucose) and cardiovascular risk stratification (1). Athletes accepted as subjects were healthy, generally asymptomatic, and exhibited no more than one risk factor for coronary artery disease. After subjects were matched and assigned to experimental and control groups as described below, mean demographics were compared for the experimental and control groups (two-tailed Wilcoxon matched-pairs sign-ranked tests). These comparisons revealed no discernible differences in age, body weight, height, or maximal aerobic capacity in men (Table 1) ($P = 0.10$ – 0.95) or women (Table 2) ($P = 0.11$ – 0.65). Subject demographics were therefore similar in the experimental and control groups.

Procedures

The overall design of this experiment included three phases (Figure 1a): pre-training assessments and instruction, concurrent exercise training and collection of data on the

TABLE 1. Demographic variables for male subjects.

Variable	Experimental	Control
Age, y	20.36 ± 0.34	20.44 ± 0.41
Body mass, kg	70.97 ± 1.81	76.53 ± 1.98
Height, cm	176.65 ± 2.07	178.93 ± 2.21
$\dot{V}O_{2max}$, mL·kg ⁻¹ ·min ⁻¹	52.83 ± 2.76	50.37 ± 2.45

Values are mean ± SEM.

independent variable (HR) and the dependent variable (DOMS), and post-training assessments and debriefing.

Before strength and endurance testing, subjects were instructed uniformly in resistance training form (Figure 1b) for each resistance exercise performed in this study. Subjects were told to give equal attention to the concentric and the eccentric phases of each resistance exercise using a 4-second duty cycle (2 seconds each for the concentric and eccentric phases). Certified or trained supervisors confirmed that subjects followed this protocol during training. All subjects restricted resistance training during the experiment to that prescribed by the program. Fewer than 10% of subjects had recently participated in resistance training, and these individuals were distributed in both the experimental and control groups. Pre-exposure to resistance training therefore did not provide a significant protective effect against DOMS in either group.

During pre-training instruction in each experiment, subjects were instructed as a group in the properties of DOMS, including the suspected causes, typical effects and sensations, time course, and the muscle groups affected for each resistance exercise performed during testing and training. All subjects indicated that they had experienced DOMS and were familiar with its characteristics. Both experiments were conducted in the off-season for participating athletes, but several of the men reported at the beginning of the concurrent training phase that they were experiencing moderate DOMS

from sport-related activities. All of the subjects were well-conditioned athletes, but in view of the specificity principle (1), exposure to unaccustomed resistance exercise would be expected to induce DOMS, as confirmed by the results.

Subjects were also instructed uniformly in the use of the Category-Ratio Rating of Perceived Exertion (RPE) and Rating of Perceived Pain (RPP) 11-point scales (7) during pre-training instruction. Directions were read aloud in a group setting, quoting the recommended instructional language (1, 7), and followed by a question-and-answer session. Subjects were then provided with the written instructional language and scales and asked to study them. Subjects were subsequently trained in the practical application of the RPE and RPP scales during the pre-training assessments, with feedback from trained and certified supervisors.

Pre-training assessments (Figure 1b) included measurements of: (a) muscle strength (1 repetition maximum weight or 1RM) for eight machine or free-weight resistance exercises (seated inclined bilateral leg press, seated leg [knee] extension, seated leg [knee] flexion or leg curls, seated front lat pull-down, flat bench press, overhead or military press, biceps or arm curl, and triceps kickback); (b) muscle endurance (number of repetitions to failure at a fixed percentage of 1RM weight) for the same eight exercises; and (c) maximal aerobic capacity ($\dot{V}O_{2max}$), estimated using a graded treadmill exercise test (1) and automated using a Technogym treadmill (The Technogym Wellness Company, Gambettola, Italy). All measurements were conducted following formats, procedures, and protocols recommended by the American College of Sports Medicine (1). During the 48-hour period immediately after the 1RM trials, subjects recorded any DOMS in muscle groups used for the eight exercises performed during 1RM tests every 12 hours, using purpose-designed forms.

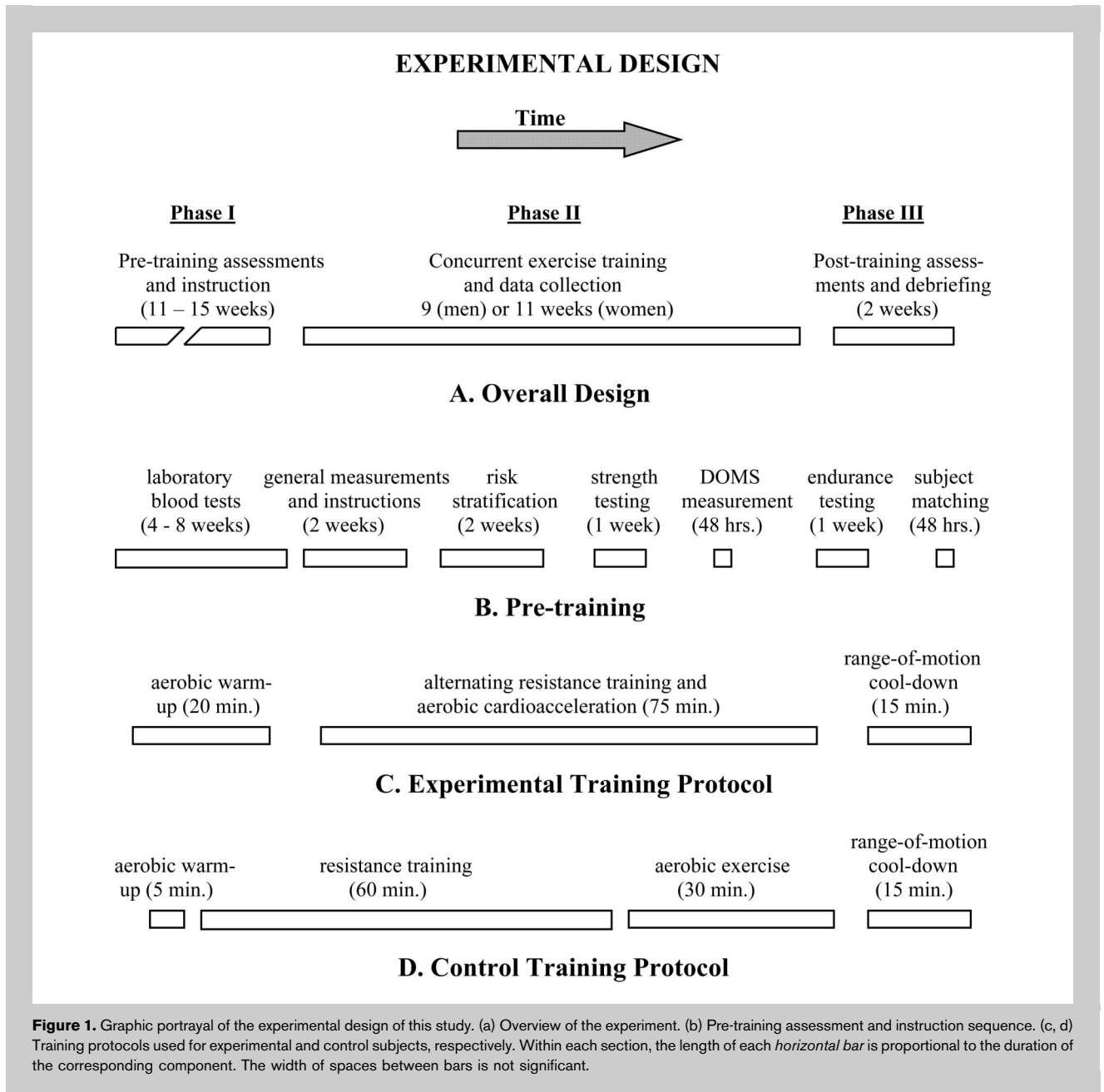
At the end of pre-training assessments, a matched-pairs design was implemented (Figure 1b). Subjects were first ranked according to three measured variables related to initial physical condition: muscle strength adjusted for body weight squared or cubed, muscle endurance, and estimated $\dot{V}O_{2max}$. A mean ranked list was prepared from these three separate rank listings, and adjacent subjects on the composite list were defined as matched pairs. Members of each matched pair were then assigned to either the experimental or control group by a random process (coin flip). Matching subjects as closely as possible on the basis of initial physical condition ensured similar starting points for the experimental and control groups, eliminating differences between groups arising from nonlinear training adaptations and enabling more powerful matched-pairs statistical tests to compare means.

Experimental and control groups then began a 9-week (men) or 11-week (women) concurrent training program, during which data were collected on the independent variable (HR during resistance training) and dependent variable (self-reported RPP for DOMS). Each subject wore a Polar A-5 HR

TABLE 2. Demographic variables for female subjects.

Variable	Experimental	Control
Age, y	19.71 ± 0.30	19.40 ± 0.21
Body mass, kg	65.78 ± 2.50	60.85 ± 2.59
Height, cm	163.69 ± 2.92	163.79 ± 1.10
$\dot{V}O_{2max}$, mL·kg ⁻¹ ·min ⁻¹	46.57 ± 2.09	47.03 ± 2.37

Values are mean ± SEM.



transmitter and wrist receiver during workouts to observe instantaneous HR, adjust aerobic work rate as required by the experimental design, and store mean HR data. DOMS was recorded for muscle groups used for each of the nine resistance exercises performed by each group, i.e., the eight previously listed and weighted abdominal curl-ups on an inclined bench (Figure 1a). In every workout, subjects recorded the RPP for residual DOMS in the muscle group used for each resistance exercise immediately after the third (final) set using a purpose-designed workout log (15). The rationale for this procedure was that DOMS can be evaluated most accurately during an exercise that utilizes the affected

musculature, and recalled and reported most accurately immediately upon completing the evaluation.

Under a similar rationale, subjects recorded the RPE for each resistance exercise and the RPP for any non-DOMS pain experienced during that resistance exercise immediately upon concluding the final set. At the end of each workout, subjects recorded mean HR during the resistance exercise stage, stored in HR monitors; water consumption during the exercise session, measured to the nearest 0.1 l; data pertinent to a number of control variables, including aggregate RPE and RPP for the entire exercise session; and other data. All data were recorded using a purpose-designed workout log (15).

Experimental and control subjects performed three vigorous workouts per week consisting of concurrent aerobic, resistance, and range-of-motion (ROM) exercise (Figure 1c, d). Experimental subjects performed integrated concurrent training divided into three stages (Figure 1c): aerobic, resistance, and a ROM cool-down. Experimental subjects began each exercise session with a 20-minute aerobic exercise warm-up (generally treadmill running) in which they rapidly attained and sustained a HR corresponding to exercise of vigorous intensity, 60–84% of HRR (1), calculated using the Karvonen method. The rationale for prolonging the warm-up was to induce sufficient cardiovascular fatigue to support more rapid subsequent cardioacceleration and to limit subsequent HR recovery in these well-conditioned athletes, enabling experimental subjects to achieve and sustain a higher HR during the resistance training that immediately followed the warm-up.

After the aerobic warm-up, experimental subjects began integrated concurrent resistance training consisting of three sets each of the nine resistance exercises previously identified in the sequence listed. Immediately before every set of resistance exercises, experimental subjects elevated their HR to the upper portion of the vigorous range (60–84% of HRR) (1) by performing a short (0.5–1 minute) bout of vigorous aerobic exercise (cardioacceleration), enabling them to sustain an elevated HR through the subsequent set of resistance exercises. Experimental subjects used the cardiovascular machine or exercise of their choice for cardioacceleration, usually treadmill running. Experimental subjects concluded each exercise session with a cool-down consisting of 12 ROM exercises during which they reduced their HR to the low end of the range corresponding to light exercise (20–39% of HRR) (1).

Control subjects performed serial concurrent training divided into four stages (Figure 1d): warm-up, resistance, aerobic, and a ROM cool-down. Control subjects began each exercise session with a brief (5-minute) aerobic exercise warm-up in which they raised their HR to 60–84% of HRR using the cardiovascular exercise of their choice, usually treadmill running. The rationale for abbreviating the warm-up was to minimize cardiovascular fatigue to support slower HR acceleration and faster HR recovery during subsequent resistance training, enabling control subjects to achieve and sustain a lower HR during the resistance training that immediately followed the warm-up.

After the aerobic warm-up, control subjects began resistance training, consisting of three sets each of the same nine resistance exercises performed by experimental subjects and in the same sequence. Immediately before every set of resistance exercises, control subjects lowered their HR to the low end of the range corresponding to light-intensity exercise (20–39% of HRR) by resting briefly (0.5–1 minute) in a seated position, enabling them to sustain a lowered HR during the subsequent set of resistance exercises. Resistance training was followed by 30 minutes of vigorous aerobic exercise

(treadmill running), 10 minutes longer than the aerobic session of experimental subjects to help to compensate for pre-resistance cardioacceleration time in experimental subjects and equalize the volume and intensity of aerobic exercise among the experimental and control groups. Control subjects concluded each exercise session with the same ROM cool-down as experimental subjects.

During concurrent training, experimental and control subjects performed the same nine resistance exercises in the same sequence: lower body (seated inclined bilateral leg press, seated leg [knee] extension, seated leg [knee] flexion or leg curl) followed by upper body (seated front lat pull-down, flat bench press, overhead or military press, biceps or arm curl, triceps kickback), and concluded with weighted abdominal curl-ups (crunches) on an inclined bench. The initial starting weights for resistance exercises were 65% of 1RMs for men and 50% of 1RMs for women. The rationale for beginning with these relatively light weights was to minimize the risk of injury. The resistance weight was increased rapidly during the first few exercise sessions to accommodate subjects' capacities (see below). The use of the same resistance exercises performed at the same intensity ensured equivalent eccentric muscle loading between experimental and control groups. The use of different starting levels for resistance exercises between men and women did not influence the interpretation of results because all statistical comparisons were performed within genders.

To minimize extraneous variance and ensure equivalent eccentric muscle loading among groups, experimental and control subjects used the same method of progression during resistance training. For the first few exercise sessions, subjects in both groups were instructed to increase the weight used for each resistance exercise as rapidly as necessary to achieve three sets of eight repetitions for each resistance exercise with an RPE no greater than 5 ("strong" exertion) and an RPP no greater than 2 ("weak" pain). This enabled subjects to increase workloads rapidly but safely from the relatively light initial weight of 50% (women) to 65% (men) of 1RM weight to a heavier weight more appropriate to their capacities.

After subjects reached this level, which occurred in the first two or three exercise sessions, advances in weight or repetitions were prescribed for each resistance exercise when the following five criteria were met: (a) the RPE for the exercise assessed after the third (final) set was "strong" or less (numerical value of 5 or less); (b) the aggregate RPE of the corresponding workout, recorded at its conclusion, was 5 or less; (c) the RPP for DOMS during each resistance exercise assessed after the third set was "weak" or less (numerical value of 2 or less); (d) the RPP for non-DOMS pain during each resistance exercise assessed after the final set was 2 or less; and (e) aggregate RPP for the corresponding exercise session was 2 or less.

When these five criteria were met, either the number of repetitions per set or the weight per exercise was increased and entered immediately into workout logs as the

prescription for the corresponding resistance exercise in the next workout. Repetitions per set began at eight, increased on progression by one or two to a maximum of 12, and reverted to eight when weight was increased. Weight was increased upon progression by 5 lb for small muscle groups (generally upper body) and 10 lb for large muscle groups (seated lat pull-down and lower body). This method of progression was easy in practice for athletes to learn, and they implemented it without difficulty within one or two workouts.

Several additional control and monitoring procedures were implemented to minimize extraneous variance and ensure identical eccentric muscle loading among groups. Experimental and control training protocols were equalized for exercise modes, types, volume, intensity, and duration (approximately 1.8 hours). Experimental and control groups exercised on different floors of the same training facility during the same morning hours (6:00 to 10:00 AM) of the same days (Tuesday, Thursday, and Saturday). Supervising trainers alternated between the two groups several times in each exercise session to preclude differential training effects or motivational influences. Prescribed HR ranges for each subject were registered in their HR monitor as lower and upper limits to assist compliance with HR prescriptions and were confirmed after training as reported in the results.

Post-training assessments and debriefing (Figure 1a) followed the same format, procedures, and sequence as pre-training assessments, including measurements of muscle strength, muscle endurance, and $\dot{V}O_{2\max}$. To conclude each experiment, every subject completed a written evaluation of the training program in the form of a questionnaire that contained an embedded question asking the purpose of the experiment. No subject answered the question correctly, indicating that the experimental protocol was blind. Because subjects who were unaware of the purpose of the experiment also self-reported the dependent variable (DOMS), the experimental protocol was double-blind.

Statistical Analyses

All data were entered into electronic spreadsheets, and entries were confirmed by trained personnel before statistical analysis and graphical display. The Wilcoxon test (29) was used for most comparisons of means. Student's *t*-test was used to compare DOMS means in different exercise sessions (Figure 4) because the underlying assumptions of normality and independence were justified. All hypotheses were tested at the 5% level ($P \leq 0.05$). Exact probabilities associated with most tests are reported to permit critical evaluation of differences. ES was calculated using the Cohen *d* method (51), and differences in means were characterized using the ranges and descriptive terminology for highly trained individuals (ES < 0.25, "trivial;" ES = 0.25–0.50, "small;" ES = 0.50–1.0, "moderate;" and ES > 1.0, "large."). Unless otherwise noted, statistical tests were one-tailed. Sample sizes varied because of missing data from some subjects for some tests and are therefore reported separately for each statistical test.

Quantitative results are reported as the mean \pm standard error of the mean.

RESULTS

To evaluate and validate the dependent variable (self-reported RPP for DOMS), the DOMS induced by identical strength testing was recorded by all subjects every 12 hours for 48 hours after both pre- and post-training 1RM trials. DOMS in the four measurements was recorded separately for muscle groups used in each of eight resistance exercises listed in the methods (abdominal curl-ups excluded). Before training in men, mean DOMS per measurement for all eight muscle groups after strength testing was not discernibly different between experimental and control groups (2.5 ± 1.4 and 2.6 ± 1.4 , respectively; two-tailed Wilcoxon test, $n = 9$, $P = 0.19$). Similarly, before training in women, mean DOMS per measurement for all eight muscle groups after strength testing was not discernibly different between experimental and control groups (5.0 ± 2.7 and 4.8 ± 3.8 , respectively; two-tailed Wilcoxon test, $n = 13$, $P = 0.81$). After training, mean DOMS per measurement for all eight muscle groups after strength testing in women was not discernibly different (3.5 ± 3.1 and 4.5 ± 3.5 for experimental and control groups, respectively; two-tailed Wilcoxon test, $n = 8$, $P = 0.78$). Mean DOMS after training was not evaluated in men because of an inadequate sample size resulting from incomplete data and withdrawal of some subjects.

DOMS after pre-training strength testing was then analyzed for each of the above eight muscle groups separately. Mean DOMS for lower body exercises exceeded that for upper body exercises almost 2:1, discernibly different in both genders (men: two-tailed *t*-test, $n = 9$, $P = 0.001$; women: two-tailed *t*-test, $n = 13$, $P = 0.001$). For each of the muscle groups corresponding to the eight resistance exercises performed, the mean DOMS per measurement (Figure 2) after identical strength testing was in no case discernibly different between experimental and control groups in either gender (two-tailed Wilcoxon tests; men: $n = 9$, $P = 0.44$ – 0.86 ; women: $n = 13$, $P = 0.23$ – 1.0). Experimental and control groups in both genders therefore self-reported DOMS induced by the same relative exercise exertion (maximal) similarly for every muscle group using the RPP scale. This outcome was prerequisite to the use of self-reported DOMS as the dependent variable and comprises a necessary and sufficient validation of the dependent variable (RPP for DOMS) for use in the current experimental application.

To test the main hypothesis (elevated HR reduces DOMS), DOMS induced by nine resistance exercises during training (the previously listed eight plus abdominal curl-ups) was first averaged among all muscle groups and all workouts separately for experimental and control groups. In men, the experimental group mean DOMS (1.4 ± 0.5) was less than one-fourth the control group mean (6.3 ± 1.4), discernibly smaller (Wilcoxon test, $n = 28$ workouts, $P = 0.0000019$; ES = 0.66, moderate). In women, the experimental group

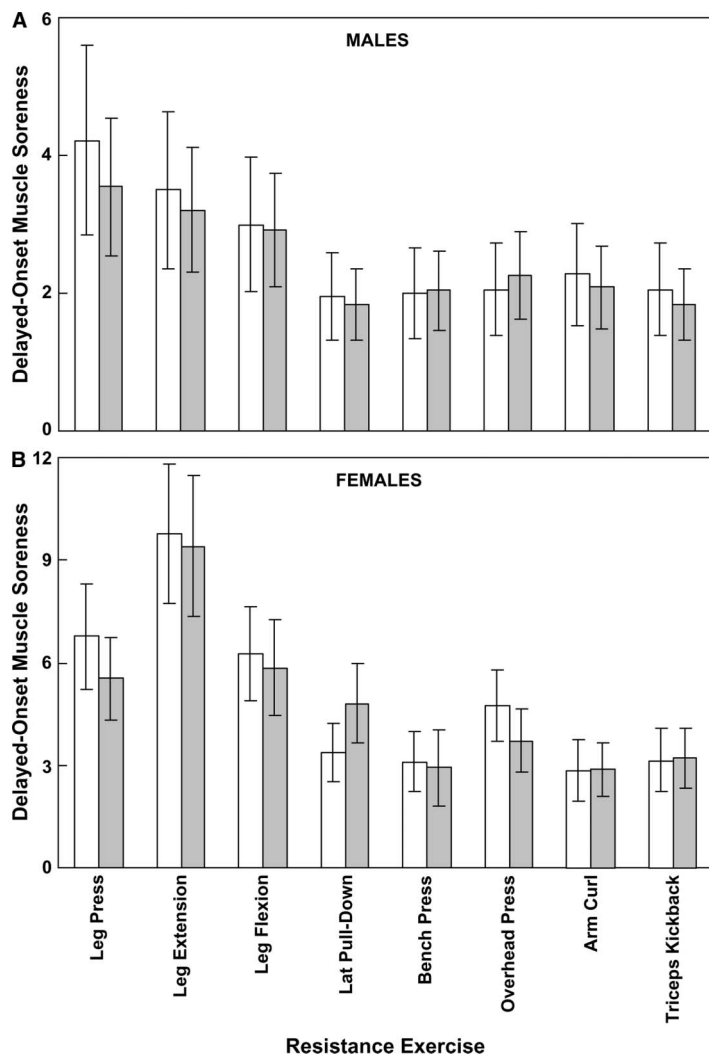


Figure 2. Mean delayed-onset muscle soreness (DOMS) per measurement in muscle groups used in eight resistance exercises over four measurements during the 48 hours immediately after pre-training strength (1RM) trials in male (a) and female (b) athletes. *Open bars* = experimental means; *shaded bars* = control means. Mean DOMS for each exercise was not discernibly different between experimental and control groups for any muscle group in either gender (see Results). Error bars represent 2 SEMs.

mean DOMS (0.3 ± 0.3) was less than one-tenth the control group mean (3.6 ± 1.1) also discernibly smaller (Wilcoxon test, $n = 33$ workouts, $P = 0.0007$; ES = 0.52, moderate).

Mean DOMS during the 9- to 11-week concurrent training programs was then calculated separately for muscle groups used in each of the nine resistance exercises (Figure 3). In men, lower body muscle groups showed a tendency toward smaller mean DOMS in experimental groups, but the differences between experimental and control means during training were not discernible at the 5% level (Wilcoxon tests, $n = 10$, $P = 0.12-0.17$). Mean DOMS in men during training for each of the six upper body resistance exercises was discernibly smaller in experimental groups (Wilcoxon tests,

$n = 10$, $P = 0.005-0.046$). ES for the nine comparisons ranged from 0.31 (small) to 0.70 (moderate). In women (Figure 3b), mean DOMS during training for muscle groups used in all nine resistance exercises was discernibly smaller in the experimental group than the control group (Wilcoxon tests, $n = 13$, $P = 0.0002-0.023$). ES for the nine comparisons ranged from 0.32 (small) to >0.50 (moderate).

Examination of DOMS data from individual subjects during training revealed large intersubject variation. Control subjects fell into two categories: those who repeatedly experienced weak to moderate DOMS throughout the training period (“DOMS-susceptible”; 35.7% of women, 50.0% of men) and those who experienced little or no DOMS after the first 2 or 3 weeks of training (“DOMS-insusceptible”). The greater mean DOMS in control groups during training represents mainly the contribution of the DOMS-susceptible subjects. The smaller mean DOMS in experimental groups during training resulted from the reduction of DOMS in the DOMS-susceptible subjects. The absence in men of a discernible difference in DOMS between experimental and control groups for the three lower-body resistance exercises (Fig-

ure 3) may therefore be attributable to high intersubject variation, combined with a relatively small sample size.

Mean DOMS averaged among all nine resistance exercises for each workout and graphed as a time series showed that, in both genders, experimental groups experienced a smaller initial rise in mean DOMS during training than control groups, and a faster decline that tended toward 0 by the end of the fourth week of training (lower curves in Figure 4a, b). Mean DOMS in experimental groups was less than mean DOMS in corresponding control groups in every exercise session in both genders. In men (Figure 4a), the difference was discernible in 32% of all exercise sessions (t -tests, $n = 6-10$; $P < 0.05$; *Figure 4a). ES ranged from 0.27 (small) to

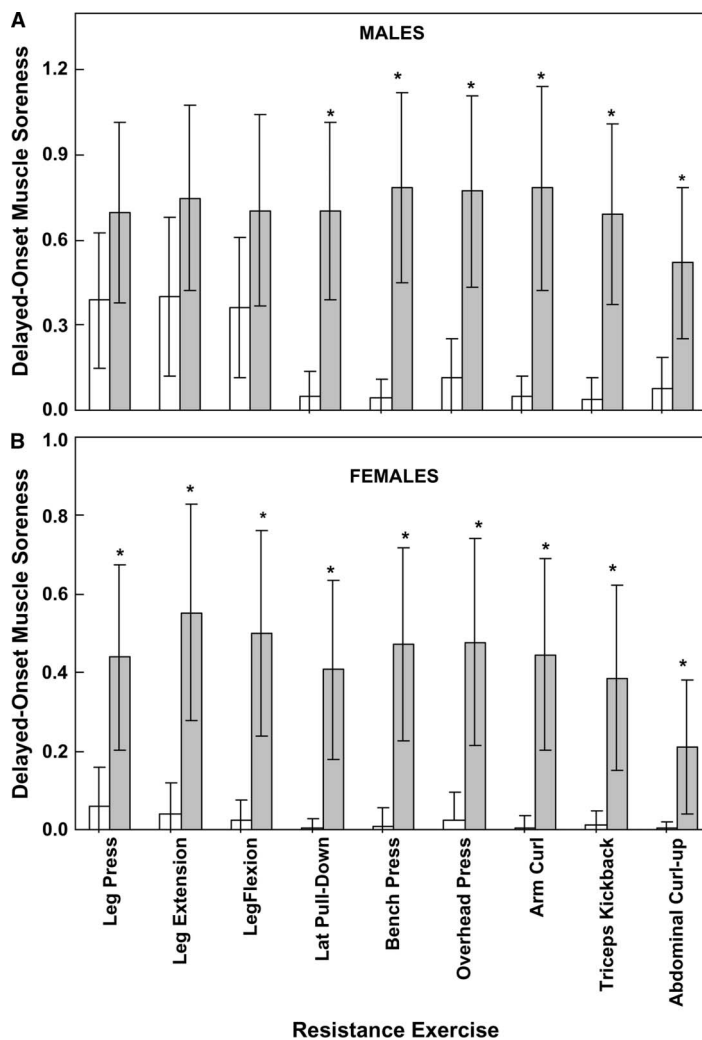


Figure 3. Mean delayed-onset muscle soreness (DOMS) in muscle groups used in nine resistance exercises over all exercise sessions during 9 weeks (men; a) to 11 weeks (women; b) of concurrent exercise training. Open bars = experimental means; shaded bars = control means. Experimental groups (cardioacceleration before each resistance exercise) reported less DOMS than control subjects (rest before each resistance exercise) for all muscle groups in both genders. *Control means discernibly larger than the corresponding experimental means (see Results). Error bars represent 2 SEMs.

1.31 (large) and, in most cases (60.7%), was moderate. In women (Figure 4b), the difference was discernible in 55% of all exercise sessions (t -tests, $n = 8-14$; $P < 0.05$; *Figure 4b). ES ranged from 0.25 (small) to 1.0 (large) and, in most cases (55%), was moderate.

To confirm expected variation of the independent variable (HR), mean HR during the resistance training portion of all workouts (Table 3) was calculated at the end of the concurrent training program from values recorded by athletes during each workout. In both genders, the mean HR during the 70-minute period of resistance training fell within the prescribed HR ranges for experimental (60–84% of HRR) and control (20–39% of HRR) groups. The mean

HR during resistance training of experimental and control groups was discernibly different for men (Wilcoxon test, $n = 10$, $P = 0.003$) and women (Wilcoxon test, $n = 13$, $P = 0.0007$). This outcome, which was anticipated based on instructions given to subjects, validates the independent variable. The HR of experimental subjects during resistance exercises was higher than the mean calculated during resistance training (Table 3) by 10–15% because after pre-resistance cardioacceleration, HR declined during and after resistance sets while HR recording continued. The mean HR of control subjects during resistance exercises was similar to the mean calculated (Table 3) because the resistance exercises alone generally had little impact on HR.

To further validate the dependent variable (RPP for DOMS), we analyzed two related control variables: non-DOMS pain and RPE during individual resistance exercises. Mean non-DOMS pain averaged among all nine resistance exercises and all exercise sessions (Table 4) was not discernibly different between experimental and control groups in either gender (two-tailed Wilcoxon tests; men: $n = 10$, $P = 0.72$; women: $n = 13$, $P = 0.70$). This result reinforces

the conclusion that subjects evaluated pain from a common exercise experience similarly using the RPP scale and provides additional, indirect validation of the dependent variable.

Mean RPE averaged among all nine resistance exercises and all exercise sessions (Table 5) was not discernibly different between experimental and control groups in either gender (two-tailed Wilcoxon tests; men: $n = 10$, $P = 0.44$; women: $n = 13$, $P = 0.86$), as anticipated from the balanced experimental design. This result confirms that differences in mean DOMS between experimental and control groups did not arise from differences in actual or perceived exertion.

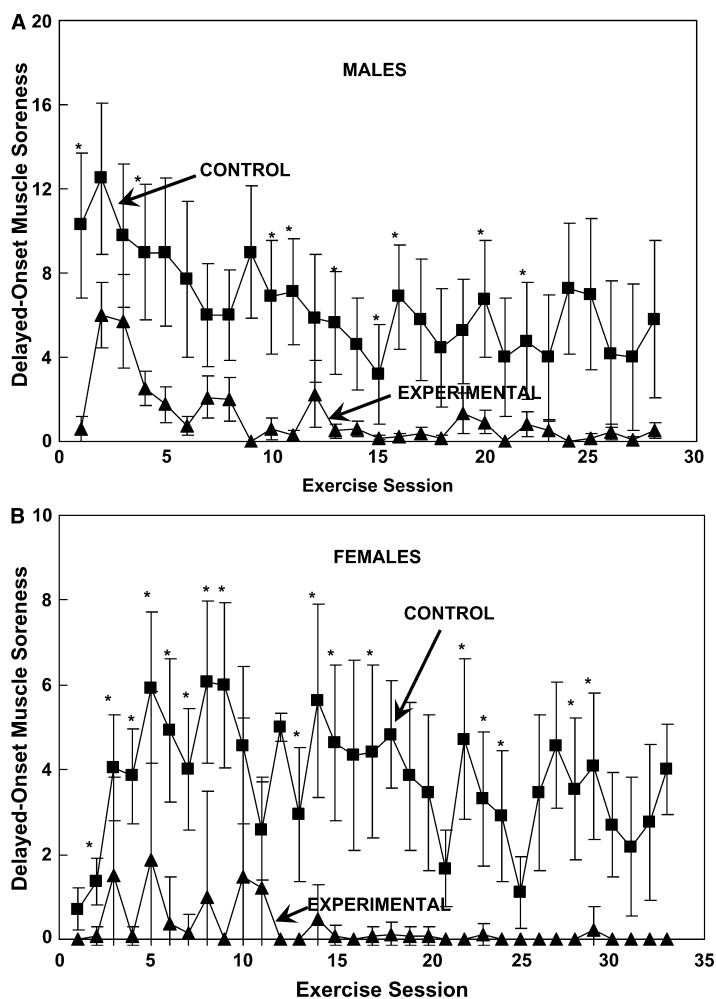


Figure 4. Time series showing mean delayed-onset muscle soreness (DOMS) per exercise session summed over all muscle groups used in nine resistance exercises during 9 weeks (men; a) to 11 weeks (women; b) of concurrent exercise training. Experimental groups (cardioacceleration before each resistance exercise) reported less DOMS than control groups (rest before each resistance exercise) for every exercise session in both genders. *Control means discernibly larger than the corresponding experimental means (see Results). Error bars represent 2 SEMs.

We analyzed two additional potentially confounding variables that could, in principle, lead to different DOMS in the experimental and control groups; namely, water consumption during workouts and attendance at prescribed exercise sessions. Dehydration during exercise can induce or exacerbate muscle injury and potentially increase DOMS in hyperthermic men (11); therefore, differential water intake during exercise sessions could yield differences in DOMS. Mean water consumption (Table 6) was not discernibly different, however, between experimental and control groups in either gender (two-tailed Wilcoxon tests; men: $n = 9$; $P = 0.77$; women: $n = 14$, $P = 0.11$).

A second potentially confounding variable, differential compliance with prescribed exercise sessions between experimental and control groups, could lead to different

overall workloads and, in particular, different eccentric loading of muscles between experimental and control groups, which could also contribute to different DOMS between the groups. Mean exercise compliance ($[\text{exercise sessions attended}/\text{total possible exercise sessions}] \times 100$) (Table 7) was not discernibly different between experimental control groups in either gender (two-tailed Wilcoxon tests; men: $n = 9$, $P = 0.31$; women: $n = 14$, $P = 0.38$).

DISCUSSION

The main finding of this study is that physiologic DOMS caused by conventional anaerobic resistance exercises is reduced immediately and eliminated within 4 weeks by aerobically elevating HR before each set in comparison with control subjects who did not elevate HR. Several potentially confounding variables, including differential eccentric muscle loading, the method of using the RPP scale to report DOMS, actual and perceived exercise exertion, water consumption during exercise sessions, and compliance with training sessions, were eliminated. None differed discernibly between the experimental and control groups. We conclude that the reduction of

DOMS in experimental groups compared with control groups was caused largely or exclusively by variation of the independent variable, i.e., by elevated HR. This finding implies that elevated HR during resistance training speeds muscle recovery.

The discernible reduction of DOMS in experimental groups below that of control means in the first (men) or second (women) workout suggests that cardioacceleration before each set of resistance exercises rapidly reduces any residual DOMS induced in the corresponding musculature by earlier exercise. Male subjects in particular began the training program under the influence of residual DOMS induced by earlier sport-related activities (see Methods). The smaller mean DOMS in experimental groups in every exercise session after the first could reflect either the reduction of residual

TABLE 3. Heart rate during the resistance training phase of the exercise program.

Gender	Variable	Experimental	Control
Male	HR, b·min ⁻¹	142.5 ± 0.7	108.1 ± 1.1
	HR, HRR	62.5% ± 0.5%	35.1% ± 0.8%
Female	HR, b·min ⁻¹	151.1 ± 0.4	107.9 ± 0.5
	HR, HRR	64.8% ± 0.3%	31.9% ± 0.4%

Values are mean ± SEM.

HR = heart rate; HRR = heart rate reserve.

DOMS induced by resistance exercises performed in the preceding exercise session(s), the blockage of new DOMS formation in preceding exercise session(s), or both. The experimental design of this study did not distinguish among these alternatives.

The design of this study also did not distinguish between the effects on DOMS of elevated HR before versus during resistance exercise. Experimental subjects warmed up longer to enable them to sustain an elevated HR during subsequent resistance training (see Methods), which could have reduced DOMS in comparison with control groups. This interpretation seems unlikely, however, because DOMS is not diminished by a warm-up (23,52), although other negative effects of eccentric exercise may be partially alleviated (52). Aerobic exercise alone was not responsible for the reduction of DOMS in experimental subjects because the intensity and volume of aerobic exercise performed in each workout was the same for experimental and control groups. We conclude that elevated HR immediately before and during resistance training in experimental subjects, and not the longer warm-up, caused the accelerated reduction and elimination of DOMS in experimental but not control groups.

The experimental design of this study required subjects to assess cumulative DOMS in muscle groups as they were employed in each resistance exercise and to record DOMS immediately after the final (third) set, when their HR was still elevated from pre-resistance cardioacceleration (see Methods). It is therefore possible that exercise-induced analgesia (25,31), mediated perhaps by elevated blood pressure (32), contributed to the reduction of self-reported DOMS in experimental subjects. This interpretation seems unlikely, however, because experimental subjects evaluated and self-reported DOMS while their HR was only marginally above the threshold for exercise-induced analgesia (50% of $\dot{V}O_{2max}$) (25). Moreover, unlike other forms of pain, DOMS seems to be undiminished by exercise-induced analgesia (14).

The design of this study conforms to the model generally used in previous controlled studies of DOMS. Groups of subjects, i.e., men in the first experiment and women in the

second experiment, were each divided into two matched groups, experimental and control. Each group received the same DOMS model exercise exposure, i.e., resistance training using identical program variables. The “treatment,” i.e., elevated HR in experimental groups, reduced and eliminated DOMS compared with the absence of treatment, i.e., lower HR in control groups. The discernible difference between mean DOMS in experimental and control groups confirmed

the hypothesized effect of the treatment, i.e., reduction and elimination of DOMS.

This study differs from previous investigations of DOMS, however, in at least five respects. First, DOMS has been evaluated typically in untrained subjects, because unconditioned muscles are more susceptible to DOMS. In contrast, we examined DOMS in trained athletes. Although the athletes were well conditioned for their respective sports, they generally had not participated in resistance training programs in the recent past (see Methods); therefore, they had not acquired specific protection against DOMS from the repeated bout effect. The specificity principle (1) implies that these athletes would be expected to experience DOMS in muscles unaccustomed to resistance exercise, which was confirmed in averaged data.

Second, the majority of studies on DOMS have employed maximal or supramaximal (>1RM weight) eccentric contraction to induce maximal muscle injury. In contrast, DOMS was induced in the present study in both experimental and control groups by the same submaximal resistance exercises, which contained concentric and eccentric components equally. The eccentric loading was therefore physiologic and equivalent for the experimental and control groups. Accordingly, although the DOMS in the present study was physiologically more realistic, it was also substantially smaller.

TABLE 4. Self-reported non-delayed-onset muscle soreness pain per exercise session during resistance training averaged among all nine resistance exercises and all exercise sessions.

Gender	Experimental	Control
Male	0.09 ± 0.19	0.06 ± 0.17
Female	0.57 ± 0.15	0.59 ± 0.20

Values are mean ± SEM.

TABLE 5. Self-reported Rating of Perceived Exertion summed among all individual resistance exercises and all exercise sessions.

Gender	Experimental	Control
Male	5.43 ± 0.10	5.57 ± 0.12
Female	5.09 ± 0.09	5.11 ± 0.11

Values are mean ± SEM.

Based on previous studies, it seems likely that DOMS in the present study was induced primarily by the eccentric component of the resistance exercises. The design of the present experiments did not, however, enable distinction between eccentric and potential concentric contributions to DOMS. The impact of elevated HR on the much greater muscle injury and DOMS induced by less physiologic maximal eccentric contraction was not addressed in this study.

Third, most studies on DOMS have examined one or at most a few muscles, typically elbow flexors (biceps brachii) or knee extensors (vastus lateralis). In contrast, we examined all major muscle groups used in nine conventional lower and upper body resistance exercises. The results show that DOMS affects both upper and lower body musculature and that pre-resistance cardioacceleration reduces DOMS in both. Retroactive (post hoc) comparisons showed that mean DOMS in lower body musculature exceeded mean DOMS in upper body musculature discernibly after 1RM testing (Figure 2). During training, mean DOMS in experimental groups also seemed greater in lower body musculature (Figure 3a, b), but mean DOMS in control subjects seemed similar in lower and upper body musculature (Figure 3). Evidence obtained herein for differential lower versus upper body DOMS is therefore inconclusive.

Fourth, most studies on DOMS have evaluated muscle soreness during the 72-hour period immediately after eccentric contraction, when DOMS presents with greatest intensity. In contrast, we measured cumulative DOMS over several weeks during a vigorous concurrent training program.

TABLE 6. Water consumption during exercise sessions.

Gender	Experimental	Control
Male	1.29 ± 0.09	1.21 ± 0.12
Female	1.19 ± 0.12	0.96 ± 0.12

Values are mean ± SEM and are expressed as liters.

TABLE 7. Compliance with exercise sessions.

Gender	Experimental	Control
Male	76.62 ± 3.38	83.24 ± 4.35
Female	81.92 ± 2.97	84.58 ± 2.63

Values are mean ± SEM and are expressed as percentages.

This much longer time series (Figure 4) revealed that mean cumulative DOMS rose initially and then declined steadily in control groups over a few weeks to approximately half the maximum, which then persisted for the remainder of the training program. The initial decline of cumulative DOMS in control groups may be attributed to the repeated bout effect, which presumably also contributed to the decline of DOMS in experimental groups. Evaluation of data recorded during training from individual subjects showed that many control subjects ceased experiencing DOMS within 2–3 weeks after training began, presumably reflecting the protection against DOMS that is afforded by the repeated bout effect. The persistence of mean DOMS in control groups resulted from sustained DOMS in a sizeable minority of DOMS-susceptible individuals. The disappearance of DOMS in experimental groups resulted from the elimination of DOMS in DOMS-susceptible individuals. The design of the present study provided no insight, however, into the reasons for the differences in susceptibility of individual subjects to DOMS, which remains to be investigated.

Fifth, most studies of DOMS have used either men or women as subjects, preventing evaluation of gender effects. In contrast, we studied both male and female subjects in separate but identical experiments and observed significant similarities in results (Figures 2–4a, b). In particular, the time course of DOMS reduction in control groups, and the magnitude and time course of DOMS reduction and elimination in experimental groups, were similar in both genders (Figure 4a, b). Remaining comparisons between genders in the present study may be of limited validity because women used less relative weight initially during resistance training and trained for 2 weeks longer. Women nonetheless reported twice the DOMS per muscle group of men after pre-training 1RM trials (Figure 2) and half the DOMS of men per workout during the several-week training program (Figures 3 and 4). These findings are consistent with the conclusion of other investigators (13) that the outcome of gender comparisons varies with the method of inducing and measuring DOMS.

The present study demonstrates rapid reduction and elimination of DOMS caused by aerobically elevating HR immediately before each set of resistance exercises but does not address directly the proximate biological causes of DOMS suppression. The time course of DOMS' decline

and disappearance in experimental subjects during training nonetheless provides empirical constraints on models of the underlying physiological mechanisms. The rapid initial reduction of DOMS in experimental groups compared with control groups in both genders (Figure 4) is congruent with the rapid dynamics of increased membrane flux that is presumably induced in skeletal muscle by the higher cardiac output and greater systolic blood pressure caused by pre-resistance aerobic cardioacceleration (1). The slower disappearance of DOMS by the end of the fourth week in experimental subjects compared with control subjects in both genders (Figure 4), however, suggests a different mechanism with a longer time constant, e.g., exercise-induced vascularization of skeletal muscle.

Increased muscle capillarization after exercise training is induced by both resistance and aerobic exercise (4,21,26,35). High-intensity intermittent endurance training increases the ratio of capillaries to muscle fibers in 4 weeks (26), similar to the time to disappearance of DOMS in experimental subjects in the present study. Concurrent strength and endurance training (but not strength or endurance training separately) significantly increased vascularization in the vastus lateralis muscle after 12 weeks of training (4), suggesting that concurrent training accelerates muscle vascularization disproportionately. The increased capillarization of leg muscle (tibialis anterior) in response to exercise is similar in men and women (50), consistent with the similar time course of DOMS reduction and elimination observed in our male and female experimental groups (Figure 4). The known properties and time course of muscle angiogenesis in response to a strong exercise stimulus are therefore congruent with the dynamics of long-term DOMS reduction and disappearance observed in experimental groups of both genders in the present study.

We propose the following hypothetical two-stage model to account for the reduction and elimination of DOMS caused by elevated HR during resistance exercises in experimental subjects. In the first (early) stage, aerobic elevation of HR immediately before each set of resistance exercises increases the flow of blood to skeletal muscle, enhancing muscle perfusion during resistance exercises (35,57), which accelerates lactate/H⁺ release from stressed muscle (27,28) and re-supplies nutrients such as glucose/glycogen and other substances more rapidly. In this first stage, therefore, the increased muscle perfusion induced by pre-resistance cardioacceleration retards cellular destruction induced by eccentric contraction and/or accelerates tissue repair, limiting muscle inflammation and therefore reducing DOMS in the first few workouts.

In the second (late) stage of this hypothetical model, continued aerobic elevation of HR immediately before each set of resistance exercises during integrated concurrent training stimulates longer-term angiogenesis, accelerating the capillarization of skeletal muscle. The increased HR, cardiac output, and systolic blood pressure induced by continuing cardioacceleration before each set of resistance

exercises now act on an expanding peripheral vascular bed in skeletal muscles to further enhance muscle perfusion (59), deliver nutrients and other substances and clear waste and injury products faster, repair the cytoskeleton and sarcolemma more rapidly, restore the metabolic and contractile machinery, and reduce inflammation, eliminating DOMS within a period of 4 weeks.

This hypothetical two-stage model is based on the probable causes of DOMS (see Introduction) combined with the documented responses of muscle to exercise. The model accounts for all features of the reduction and elimination of DOMS by aerobic elevation of HR before each set of resistance exercises observed in experimental groups in the present study. The time course of the reduction in DOMS from the repeated bout effect in control subjects was similar to the attenuation of DOMS in experimental subjects (Figure 4), suggesting that this two-stage model could operate in parallel with or through the mechanism(s) of the repeated bout effect to enhance protection against eccentric contraction injury. The proposed model can be readily tested using cardioacceleration to regulate DOMS as the independent variable and physiological, biochemical, and ultrastructural measures as dependent variables.

PRACTICAL APPLICATIONS

This study has two practical applications. First, it utilizes a new method of progression during resistance training, which optimizes exertion (RPE) and minimizes pain (RPP) during sets of resistance exercises. This method promotes the fastest possible advances in training adaptations consistent with balancing the two competing goals of exercise programs: maximizing effects and minimizing the risk of injury or overtraining. The training protocols used in this study were designed to increase general strength and endurance by combining medium weight, repetitions, and velocity with strong exertion. The method of progression used herein can be adapted, however, to specialized training purposes such as athletic strength and power conditioning or patient rehabilitation by raising or lowering, respectively, the RPE threshold for progression.

Second, this study documents a new training protocol, integrated concurrent exercise (15), which rapidly eliminates DOMS. The rapid attenuation of DOMS during integrated concurrent training implies faster recovery of skeletal muscle after a strong exercise stimulus, which should support more frequent training and faster progression during all high-intensity training modes, including endurance, strength, power, and agility. Even when DOMS is not an issue in a training program, integrated concurrent training would be expected to support faster development. The present experiments entailed vigorous training with well-conditioned athletes, but integrated concurrent exercise may also reduce DOMS in medium- and low-intensity training applications with recreational exercisers, the elderly, and rehabilitating patients. In this case, the prescription of integrated

concurrent exercise could help to eliminate a demonstrated barrier to exercise initiation, adherence, and progression at all levels and ages and could have potential clinical applications.

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Note: W. Jackson Davis is now with The Miracle Workout, LLC, P.O. Box 2221, Boulder, Colorado 80306.

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