Muscle damage and resting metabolic rate after acute resistance exercise with an eccentric overload

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ABSTRACT

DOLEZAL, B. A., J. A. POTTEIGER, D. J. JACOBSEN, and S. H. BENEDICT. Muscle damage and resting metabolic rate after acute resistance exercise with an eccentric overload. *Med. Sci. Sports Exerc.,* Vol. 32, No. 7, pp. 1202–1207, 2000. **Purpose:** The purpose of this investigation was to determine whether muscle damage caused from acute resistance exercise with an eccentric overload would influence resting metabolic rate (RMR) up to 72 h postexercise in resistance-trained (RT) and untrained (UT) subjects. **Methods:** Nine RT and 9 UT male subjects (mean ± SD; age = 20.7 ± 2.1 yr; body mass = 79.0 ± 1.4 kg; height = 178.4 ± 3.1 cm; and body fat = 10.2 ± 1.6%) were measured for RMR, creatine kinase concentration ([CK]), and rating of perceived muscle soreness (RPMS) on five consecutive mornings. To induce muscle damage, after the measurements on day 2, each subject performed leg presses that emphasized the eccentric movement for 8 sets at his six-repetition maximum (6-RM). **Results:** Compared with baseline, the RMR ($kJ \cdot d^{-1}$ and $kJ \cdot kg\ FFM^{-1} \cdot h^{-1}$) was significantly elevated for RT and UT at 24 h and 48 h postexercise. From 24 h to 48 h to 72 h postexercise, RMR significantly decreased within both groups. The UT group had a significantly higher RMR at 24 h (9705.4 ± 204.5 $kJ \cdot d^{-1}$) and 48 h postexercise (8930.9 ± 104.4 $kJ \cdot d^{-1}$) when compared with the RT group (9209.3 ± 535.3 and 8601.7 ± 353.7 $kJ \cdot d^{-1}$). Both [CK] and RPMS showed a similar time course. **Conclusion:** There was a significantly higher [CK] for the UT group at 24 h postexercise (320.4 ± 20.1 $U \cdot L^{-1}$) and for both [CK] and RPMS at 48 h (1140.3 ± 37.1 $U \cdot L^{-1}$ and 4.4 ± 0.5, respectively) and 72 h postexercise (675.9 ± 41.7 $U \cdot L^{-1}$ and 1.67 ± 0.5, respectively) when compared with the RT group (24 h, 201.9 ± 13.4 $U \cdot L^{-1}$; 48 h, 845.4 ± 30.7 $U \cdot L^{-1}$ and 3.7 ± 0.5; and 72 h postexercise, 420.2 ± 70.2 $U \cdot L^{-1}$ and 0.89 ± 0.3). These data indicate that eccentrically induced muscle damage causes perturbations in RMR up to 48 h postexercise. **Key Words:** POSTEXERCISE RECOVERY, CREATINE KINASE, RATINGS OF PERCEIVED MUSCLE SORENESS, METABOLISM

In addition to factors such as exercise intensity and duration, the mode of exercise may play a critical role in postexercise metabolism. There is a greater occurrence of muscle damage after eccentric-type exercise compared with concentric-type exercise (1). Evidence of damage after eccentric-type exercise includes performance decrements, morphological changes, delayed-onset muscle soreness, and elevations of myocellular enzymes in the circulation, especially creatine kinase concentration ([CK]) (10,27). Examples of exercises that have a heavy eccentric component and therefore cause muscle damage are high-intensity sprinting, high-intensity resistance exercise, and downhill running (10).

Exercise involving eccentric muscle actions has been found to induce damage in skeletal muscle fibrils, membranes, and ion transport mechanisms (12), and may subsequently cause perturbations in postexercise metabolic rate (13). Considerable speculation exists that eccentric forms of exercise may cause greater trauma to muscle, especially relative to protein breakdown and synthesis (20,24). Because the resynthesis of protein is energetically expensive (possibly accounting for up to 20% of resting metabolic rate (RMR) (30)), others have proposed that there may be greater energy utilization from muscle damage and that this increased energy utilization may be reflected in elevations of RMR for extended periods of time afterward (19,29).

Recent studies by Thomas et al. (29) and Kolkhorst et al. (19) failed to support the hypothesis that muscular degradation induced by eccentric muscular actions from running and/or jogging would increase recovery energy expenditure beyond 24 h. However, their data suggest that exercise with a significantly stronger eccentric component (i.e., resistance exercise) may be necessary to evoke large amounts of muscle damage so that the energy required for muscle repair and protein synthesis may be reflected in prolonged elevated postexercise RMR. These studies also failed to assess muscle damage and repair, which would have limited them from drawing any conclusions between muscle degradation and/or protein synthesis with postexercise RMR. In addition, very few studies have examined the influence of physical conditioning on these types of interventions. Research has shown attenuated muscle soreness and blunted increases in [CK] activity in trained versus untrained individuals,

0195-9131/00/3207-1202/0
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Submitted for publication January 1999.
Accepted for publication September 1999.

TABLE 1. Subject demographic data.

Characteristic	Group	
	UT	RT
Age (yr)	20.6 ± 1.8	20.9 ± 2.5
Height (cm)	178.6 ± 1.1	178.2 ± 3.1
Body mass (kg)	78.8 ± 1.2	79.2 ± 1.6
Fat-free mass (kg)	70.8 ± 0.6	71.2 ± 0.5
Body fat (%)	10.2 ± 1.5	10.1 ± 1.7
Daily macronutrients		
(% CHO)	54.3 ± 6.5	51.9 ± 4.1
(% FAT)	20.7 ± 2.9	22.6 ± 3.2
(% PRO)	25.0 ± 3.4	25.5 ± 4.8
Daily energy intake (kJ)	12,590 ± 923	12,881 ± 1176
Predicted 6-RM (kg)	149.7 ± 8.7	202.6 ± 8.4*

Values are means ± SD; $N = 9$ in each group.
* Significantly different from UT, $P < 0.05$.

suggesting that there may also be a lesser degree of muscle damage in the trained state (6,10,16,23). Whether this difference may be reflected in postexercise metabolism remains to be determined.

Therefore, the purpose of the present investigation was to determine whether muscle damage caused by acute resistance exercise with an eccentric overload would influence RMR up to 72 h postexercise. Additionally, we compared RMR, ratings of perceived muscle soreness (RPMS), and creatine kinase concentration ([CK]) between resistance-trained (RT) and untrained (UT) subjects.

METHODS

Subjects. Eighteen college-aged male subjects participated in this experiment. All subjects voluntarily read and signed an informed consent form and completed a health history questionnaire in accordance with guidelines set forth by the Advisory Committee for Human experimentation at the University of Kansas. Depending on prior resistance training experience, subjects were categorized into one of two groups: 9 were resistance trained (RT) (subjects included lower-body resistance exercises with their resistance training program for at least 2 $d \cdot wk^{-1}$ for a minimum of 2 yr) and 9 were untrained (UT) (no prior resistance training experience). Group demographics are provided in Table 1.

Research design. Preliminary testing included a body composition measurement, determination of total thigh muscle cross-sectional area (thigh-CSA) to predict 6-repetition maximum (6-RM) on the leg press machine, and familiarization of the subject with the test protocol. Subsequently, the subjects were tested on five consecutive mornings for an RMR measurement followed by a 5-mL venous blood collection to determine [CK]. To induce muscle damage, after the RMR on day 2, each subject performed leg presses that emphasized the eccentric movement for 8 sets at his 6-RM. For the next three mornings, the subjects were asked to rate the soreness of their leg muscles.

Determination of thigh-CSA to predict leg press 6-RM load. It was necessary to ensure that the UT subjects of this investigation remained truly untrained and that the RT subjects encountered no unplanned physical stress. Therefore, before the start of the study, two regression analyses of thigh-CSA to predict 6-RM were developed on a separate cohort sample of resistance-trained ($N = 10$) and untrained ($N = 10$) male volunteers. In this way, we eliminated the trial and error involved with 6-RM determination and subsequent muscle damage and possible carryover into RMR associated with the leg press exercise for the subjects participating in the study.

Using the anthropometric equation for thigh-CSA (17), measurements were made for a midthigh circumference and an anterior thigh skin-fold. Anthropometric dimensions were taken on the right thigh with the subject standing with knees slightly bent, body weight evenly distributed, and feet approximately shoulder-width apart.

The 6-RM was recorded as the maximal weight that could be lifted through a full range of motion six times. Weights were added or removed between sets in 10-pound increments (up to 3-min rest between each set) until the subject's 6-RM was determined. The results of a linear regression analysis yielded equations for RT ($y = 2.021x + 99.808$, where x = thigh-CSA and y = weight; $R = 0.87$, SEE = 13.6) and UT ($y = 1.231x + 173.664$; $R = 0.96$, SEE = 2.3) subjects.

Body composition measurement. Hydrostatic weighing was performed to determine body density on college-aged Caucasian male subjects. The body mass of the subject, while wearing shorts and a shirt, was measured and recorded in kilograms using an electronic scale. Afterward, six trials of underwater weight were performed with the average of the last four trials used as the mean value (25). Residual lung volume was measured via the helium dilution method (25). The equation of Siri (28) was used to estimate percent body fat from body density (14), with fat-free mass and fat mass calculated accordingly.

Dietary journal. We attempted to foster dietary compliance or to account for unexpected fluctuations due to unplanned, temporary changes in dietary intake. Thus, each subject kept a dietary journal of the foods that were consumed 24 h before the first RMR measurement and for the remainder of the study. Subjects were provided with examples of food samples, written guidelines, and a record book for keeping track of daily food intake. The subjects were asked to consume foods they normally eat and to maintain those same foods in like amounts at the same time of day until the last RMR measurement. Each dietary record was analyzed for daily kJ intake and the percentage of macronutrients using a commercially available nutritional software program (Nutritionist III; N-Squared Computing, Inc., Salem, OR).

Resting metabolic rate. Subjects were tested for RMR on five consecutive mornings. Subjects had a restful night's sleep (~8 h) and did not eat or consume any food or liquids except water for 12 h before each RMR assessment. Subjects refrained from all exercise for 48 h before the first day of testing and for the remainder of the 5-d experiment with exception of the exercise performed in conjunction with the study. The verification of these conditions occurred via verbal questions. Each subject was transported by a motor vehicle to the testing site to ensure minimal activity before RMR determination. After entering the laboratory,

subjects rested in a supine position for at least 30 min in a dark, quiet, thermoneutral (22°−24°C) environment. Each subject was instructed to remain as quiet as possible and minimize any movement before and during the entire RMR measurement period. During this rest period, a Hans-Rudolph (Kansas City, MO) flow-by face mask was fitted on the subject and interfaced with a SensorMedics 2900 Metabolic Measurement Cart (Yorba Linda, CA). The 30-min habituation period was immediately followed by a 30-min measurement period of RMR. The metabolic system was calibrated before each RMR test by using calibration gases of a known concentration and a 3-L syringe. Reproducibility of test values for RMR on each subject was determined by acquiring a 10-min steady state value, recalibrating the metabolic cart, and establishing another 10-min steady state value. The interclass correlation for this was $r = 0.98$. Additionally, the mean of each subject's two pretreatment RMR trials (i.e., the first and second day) was used as the baseline RMR unless the difference between the two trials exceeded a 4% variance (3,21), in which case the subject was not used in the study. All subjects RMR trials fell within this limit and thus were used in the study.

Eccentric resistance exercise. After the RMR measurement on day 2, and immediately after a 5-min warm-up at 1.0 kp on a cycle ergometer, subjects performed leg presses that emphasized the eccentric movement for 8 sets at their 6-RM as determined previously using a separate cohort as described above. The leg press exercise was selected because it has previously been shown to elicit large eccentric muscle actions in the quadriceps muscles (9).

The subject assumed a seated position on the leg press machine (Pro Star Sports, Blue Springs, MO) and placed his feet shoulder-width apart so that they were flat on the resistance platform. The position of the legs remained parallel to each other and in a semiflexed position (~45° between the femur and tibia) while the subject's back remained flat against the back pad during the entire exercise. The subject then proceeded to extend his legs and push the weight stack forward until his knees were only slightly bent. While maintaining a correct position on the back pad, the subject moved the resistance platform slowly backward and under control to the beginning position; this was considered one repetition. The backward movement phase (i.e., the eccentric muscle action) was timed by the technician so that it entailed a slow, deliberate movement that was 4 s in duration. A 3-min rest period was taken between each set. A spotter was positioned on each side of the leg press machine and assisted in the concentric phase when the subject began to experience local muscle fatigue. At all times, the subject was encouraged to exhale through the forward movement phase and inhale during the backward movement phase.

Blood collection and analysis. After each RMR measurement, venous blood (~4 mL) was collected into a syringe from an antecubital vein. The blood was then transferred into a nontreated Vacutainer, allowed to clot at room temperature for 10 min, and then centrifuged for 15 min at −10°C. The separated serum was extracted and kept frozen at −70°C until it was analyzed for [CK]. Each blood sample was analyzed spectrophotometrically, in duplicate, according to specific assay procedures (Sigma kit no. 315–47, St. Louis, MO). The mean of each subject's two pretreatment [CK] measurements (i.e., the first and second day) was used as the baseline [CK]. The interclass correlation for this was $r = 0.97$.

Ratings of perceived muscle soreness (RPMS). Subjects rated soreness for their leg muscles after each RMR measurement. Ratings of perceived muscle soreness were scored on a scale ranging from 0 to 6 (27). Each number corresponded with a verbal description of soreness (0); no soreness (1), dull feeling of soreness (2), light continuous soreness (3), more than light soreness (4), annoying soreness (5), severe soreness, and (6) intolerable soreness.

Statistical analyses. A 2 × 4 (group by time) repeated-measures ANOVA was used to determine significance for RMR ($kJ \cdot d^{-1}$ and $kJ \cdot kg\ FFM^{-1} \cdot h^{-1}$), [CK] ($U \cdot L^{-1}$), and RPMS using SPSS (*v*7.5). Where significance was found, the least significant difference (LSD) *post hoc* test was used. Demographic data (age, height, body mass, and body fat %), predicted 6-RM, and daily energy intake and percentage of macronutrients were analyzed using independent-samples *t* tests. Significance was set at $P < 0.05$. All values reported are means ± SD.

RESULTS

The RT and UT subjects did not differ as to age, height, body mass, fat-free mass, body fat %, daily percentage of macronutrients, and daily energy intake (Table 1). The mean predicted 6-RM, as expected, was significantly higher for the RT group (by 26%) compared with the UT group. There were no significant changes in each group's normal dietary patterns (i.e., daily kJ intake and percentage of macronutrients) over the five day investigation (data reported as the average of the 5 d).

Baseline and 72 h postexercise values for RMR in $kJ \cdot d^{-1}$ and $kJ \cdot kg\ FFM^{-1} \cdot h^{-1}$ were not significantly different between or within the RT and UT groups. However, 24 h and 48 h postexercise values for RMR were significantly different both between and within the two groups (Fig. 1). RMR ($kJ \cdot d^{-1}$ and $kJ \cdot kg\ FFM^{-1} \cdot h^{-1}$) was significantly elevated for both RT and UT groups from baseline at 24 h and 48 h postexercise. From 24 h to 48 h to 72 h postexercise, RMR significantly decreased within both groups. The UT group showed a significantly higher RMR at 24 h (9705.4 ± 204.5) and 48 h postexercise (8930.9 ± 104.4) when compared with the RT group (9209.3 ± 535.3 and 8601.7 ± 353.7).

Significant differences were observed for [CK] and RPMS over time both between and within groups. Both [CK] and RPMS showed a similar time course; that is, they significantly increased for both groups from baseline to 24 h and peaked at 48 h postexercise. At 72 h postexercise, although still significantly elevated from baseline, [CK] and RPMS had significantly decreased from 48 h postexercise. There was a significantly higher [CK] for the UT group at 24 h postexercise (320.4 ± 20.1 $U \cdot L^{-1}$) and for both [CK]

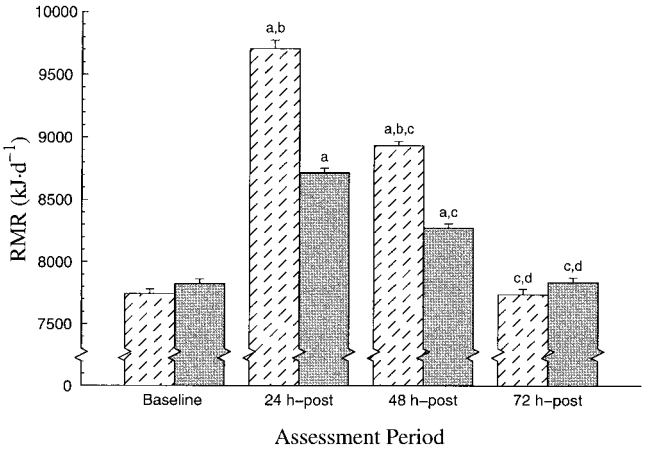

Figure 1—Mean RMR (kJ·d^{-1}) for UT (*N* = 9; *hatched bars*) and RT (*N* = 9; *solid bars*) before and after eccentrically induced muscle damage (*P* < 0.05); a, significantly different from baseline; b, significantly different from RT group; c, significantly different from 24 h postexercise; d, significantly different from 48 h postexercise.

and RPMS at 48 h (1140.3 ± 37.1 U·L^{-1} and 4.4 ± 0.5, respectively) and 72 h postexercise (675.9 ± 41.7 U·L^{-1} and 1.67 ± 0.5, respectively) when compared with the RT group. Figure 2 illustrates a comparison of the time course for RMR (kJ·kg FFM^{-1}·h^{-1}), [CK], and RPMS for both UT and RT groups.

DISCUSSION

The main finding of this investigation indicates that RMR remains elevated above baseline levels during the postexercise period for up to 48 h after a bout of acute resistance exercise with an eccentric muscle action overload. Additionally, although RT subjects had a similar time course in RMR elevation when compared with the UT subjects, we believe that their attenuated [CK] and RPMS response to the eccentric exercise suggested that there was a lesser degree of muscle damage thereby equating to a diminished elevation in postexercise RMR. Although our findings are not directly comparable to those of others (13,19,20,29) due to protocol differences (i.e., exercise mode, intensity, and duration), some comparisons are in order.

Gillette et al. (13) and Melby et al. (20) reported a prolonged recovery RMR from resistance exercise; however, unlike the results in the present investigation, RMR remained elevated only up to 15 h. Although it was not their intention to identify the influence of eccentric based exercise on postexercise RMR, they suspected that exercise with eccentric muscle actions may induce muscle damage and subsequently lead to perturbations in RMR.

Our results were in contrast with other investigators who were not able to show that muscle damage induced by eccentric muscular actions from downhill running (29) and level treadmill jogging (19) increased postexercise RMR beyond 24 h. Elevation of [CK] in blood after exercise is considered an indicator of muscle damage (6,10,23,27). Consequently, the [CK] and RPMS results (Fig. 2) in the present study suggest that muscle damage and soreness

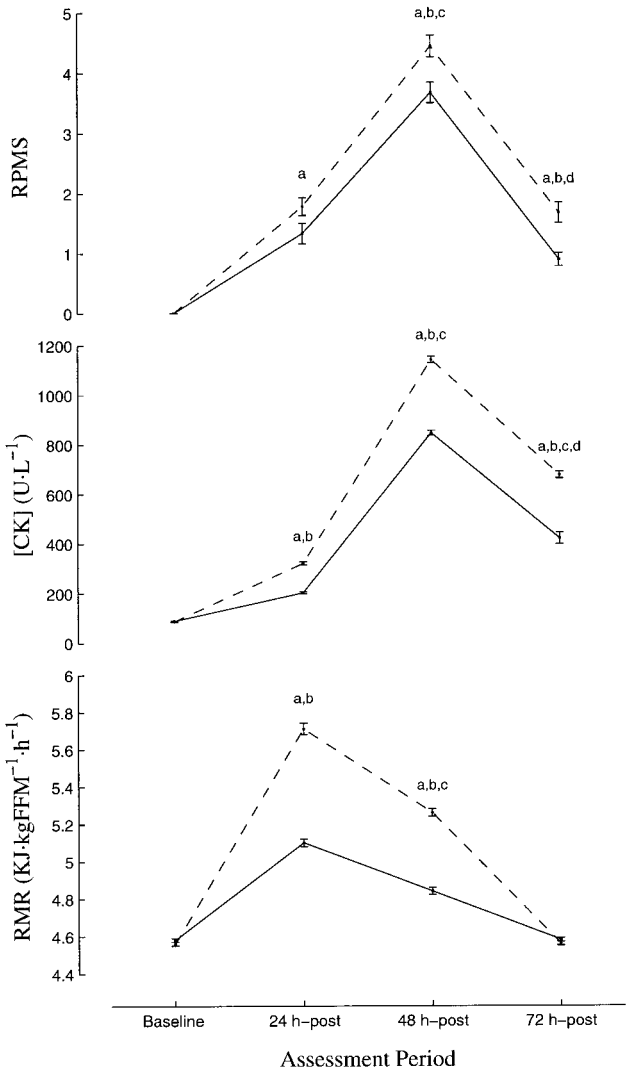

Figure 2—A comparison of the time course for mean RMR (kJ·kg FFM^{-1}·h^{-1}), [CK] (U·L^{-1}), and RPMS for both UT (*N* = 9; *dotted lines*) and RT (*N* = 9; *solid lines*) before and after muscle damage (*P* < 0.05); a, significantly different from baseline; b, significantly different from RT group; c, significantly different from 24 h postexercise; d, significantly different from 48 h postexercise.

occurred from the leg press protocol. Creatine kinase levels reached peak values (>1100 U·L^{-1}) at 48 h postexercise and then began to subside (remaining elevated above baseline at 72 h postexercise), an observation consistent with the reported time course of [CK] response after many forms of high-intensity eccentric exercise (6). The subjects' soreness ratings were also in agreement with research that characterize an increase in the intensity of soreness after exercise, peaking at 48 h (light to annoying soreness), and followed by a subsequent decrease in soreness (22).

Some factors that contribute to an elevated postexercise metabolism after exercise with an equal concentric-eccentric component include resynthesis of glycogen from lactate, elevated body temperatures, resaturation of venous blood and myoglobin, phosphagen resynthesis, residual effects of hormones, and redistribution of ions within tissue compartments (2,4). In our findings, we believe that these factors contributed minimally to the 18% increase in RMR from

baseline at 24 h (for both groups averaged together) because these processes have been shown to have immediate and temporary effects (21); therefore, other factors must have contributed to an elevated RMR at 24 h and 48 h postexercise (11% increase from baseline).

Currently, discrepancies still exist as to the sequence of events that lead to muscle damage and ultimately repair. Mechanical factors (muscle tension) and active strain have been known to initiate damage during eccentric exercise in animal studies (6). The responses to damaging exercise often do not manifest themselves until the postexercise period, at which time a cascade of host-specific defense reactions is activated. This cascade of events, characterized as the "acute phase response," includes complement activation, neutrophil mobilization, cytokine production, and rise in circulating CK levels (10). In addition, these events trigger increases in skeletal muscle protein synthesis and degradation. Although it would be impossible to speculate as to the energy cost during this period, it is reasonable to suggest that some of these processes may contribute to a postexercise elevation in metabolism. In fact, it is believed that there is a decreased protein metabolism during exercise that is compensated by an augmented rate of protein turnover during the postexercise period (16).

Recently it has been postulated that eccentric based exercise induces dramatic perturbations in homeostasis that would require energy to repair muscle trauma, especially relative to protein breakdown and synthesis (20,24). Although we did not measure protein turnover directly, we suspected that the leg press protocol in our study influenced myofibrillar protein metabolism for a period of time postexercise. This has been confirmed in number of studies (5,11,16) where maximum rates of muscle protein turnover lasted for 2 d in response to an acute bout of heavy eccentric resistance exercise. Because the energy cost of protein turnover may account for as much as 20% of resting metabolism (30), the energy utilization during the 48-h recovery period for the present study may have been reflected in elevations of RMR. In fact, in clinical studies involving traumatized (i.e., burned) adults and children, protein synthesis during recovery correlated moderately well with elevations in oxygen consumption and RMR (7,18). More recently, it was suggested that tissue damage and the stimulus for repair and tissue hypertrophy resulting from resistance exercise may be sufficient to contribute to the increased total energy during recovery (4).

We believe that the differences resulting from the subjects' training status in this investigation lends further support that the events associated with muscle damage from exercise may contribute to the elevations found in postexercise RMR. It is well established that physical conditioning results in reductions in muscle damage, RPMS, and [CK] activity after acute bouts of eccentric exercise (10). Figure 2 shows that the RT subjects had diminished responses to the eccentric exercise in [CK] and RPMS at 24 h and 48 h postexercise when compared with the UT subjects. It appears that the RT subjects' prior performance of leg resistance exercise encountered in their daily training regimen may have resulted in an adaptation in the muscle such that it became more resistant to the effects of a subsequent bout of intense leg presses that emphasized the eccentric movement. More efficient recruitment of motor units and structural adaptations in the muscle in which membrane, extracellular matrix, cytoskeleton, and myofibrils are more resistant to damage have been offered to explain this "repeated bout effect" (23).

Resistance-trained subjects also exhibited attenuated responses to the eccentric exercise in RMR at 24 h and 48 h postexercise when compared with the UT subjects. As seen in Table 1, we had very homogeneous groups that were evenly matched in all characteristics except resistance training experience and consequently in the predicted 6-RM. To equalize the overload between the two groups, 26% more weight (as determined by the cohort sample) was assigned to RT subjects 6-RM leg press exercise versus the UT subjects. When compared with the RT subjects, the UT subjects had a 12% and 8.5% greater elevation in RMR at 24 h and 48 h postexercise, respectively. It has been reported that the training state of the subjects is considered among one of numerous variables that may affect the duration of increases in protein muscle synthesis after muscle damage from heavy resistance exercise (5). Because fat-free mass (particularly skeletal muscle) represents an important reserve of amino acids that can be mobilized during muscle trauma (11), we believe the [CK] and RPMS data suggested that the RT group experienced a lesser degree of muscle damage, which may have equated to smaller amounts of protein turnover. Consequently, this may have been reflected in the smaller elevation found in postexercise RMR.

In summary, as evidenced by the present data and other researchers who have speculated from the results of their own studies (13,19,20,29), we believe that the events associated with muscle damage from leg presses that emphasized the eccentric movement partially contributed to the elevations found in postexercise RMR. Although the reasons for this observation are not completely understood and need considerably more invasive measurements, the events which led to differences in postexercise RMR for the UT and RT subjects seem to lend additional support to this theory.

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