Psychological Stress Impairs Short-Term Muscular Recovery from Resistance Exercise

MATTHEW A. STULTS-KOLEHMAINEN1,2 and JOHN B. BARTHOLOMEW3
1Yale Stress Center, Yale University School of Medicine, New Haven, CT; 2Department of Kinesiology and Physical Education, Northern Illinois University, DeKalb, IL; and 3Exercise and Sport Psychology Laboratory, University of Texas at Austin, Austin, TX

ABSTRACT
STULTS-KOLEHMAINEN, M. A., and J. B. BARTHOLOMEW. Psychological Stress Impairs Short-Term Muscular Recovery from Resistance Exercise. Med. Sci. Sports Exerc., Vol. 44, No. 11, pp. 2220–2227, 2012. Purpose: The primary aim of this study was to determine whether chronic mental stress moderates recovery of muscular function, perceived energy, fatigue, and soreness in the first hour after a bout of strenuous resistance exercise. Methods: Thirty-one undergraduate resistance training students (age = 20.26 ± 1.34 yr) completed the Perceived Stress Scale and Undergraduate Stress Questionnaire (USQ; a measure of life event stress) and completed fitness testing. After 5 to 14 d of recovery, they performed an acute heavy-resistance exercise protocol (10-repetition maximum (RM) leg press test plus six sets: 80%–100% of 10 RM). Maximal isometric force (MIF) was assessed before exercise, after exercise, and at 20, 40, and 60 min postexercise. Participants also reported their levels of perceived energy, fatigue, and soreness. Recovery data were analyzed with hierarchical linear modeling growth curve analysis. Results: Life event stress significantly moderated linear (P = 0.013) and squared (P = 0.05) recovery of MIF. This relationship held even when the model was adjusted for fitness, workload, and training experience. Likewise, perceived stress moderated linear recovery of MIF (P = 0.023). Neither USQ nor Perceived Stress Scale significantly moderated changes in energy, fatigue, or soreness. Conclusion: Life event stress and perceived stress both moderated the recovery of muscular function, but not psychological responses, in the first hour after strenuous resistance exercise. Key Words: ECCENTRIC EXERCISE, LIFE EVENT STRESS, PERCEIVED STRESS, MAXIMAL ISOMETRIC FORCE, ENERGY, FATIGUE

To the uninitiated, exercise seems counterintuitive in that people must first break down their bodies to enhance strength, endurance, and power (22). In fact, the response to the acute stress of strenuous high-volume–high-intensity training follows a bimodal (two-wave) trajectory. There is an immediate reduction in strength postexercise (up to 50%–65% loss) followed by a short-lived rebound. After this time, strength again decreases for 24–48 h (16,30) along with concomitant increases in pain and swelling (3,30). The first hour of recovery after exercise appears to be critical. Decrements associated with strenuous exercise have a rapid rebound within the first 60 min, including rapid changes in metabolic (5), neuromuscular (25), immune (1), and cardiovascular systems (7). This general pattern of disruption and recovery of the homeostatic state is called the stress response (37). With physical stress often follows the experience of mental strain and emotional distress. More broadly, stress may be conceptualized as a perceived inability to exert control or the number of major life events that a person has experienced over the last several months (21,28). Regardless of the assessment, psychological stress is associated with a host of training outcomes. For example, those higher in life stress experience lesser strength adaptations from resistance training (4) and are more likely to experience an illness (43) or sport-related injuries (21). Similar patterns of effects have been shown for recovery from injury.

Studies indicate that those who are stressed take much longer to recover from surgery and illness than do those low in stress (8). In addition, an elegant series of wound healing studies, in which a wound is developed by the experimenter, provides strong evidence that stress prolongs the time it takes to recover (10,17). In one study of note, Marucha et al. (32) found that healing time was extended for 100% of dental students during academic finals as opposed to during summer break. Remarkably, this finding has replicated across the types of wounds induced (e.g., mucosal, epidermal), the types of stressors evaluated (e.g., caregiving and marital conflict), the magnitude of the stressor (e.g., examinations and restraint), and whether stress was measured objectively or by self-report (10,17).

There is reason to expect this relationship to apply to recovery from acute, exercise-induced muscle impairments. Wound healing and muscular repair are both highly structured and elaborate processes involving the activation of cytokines, macrophages, growth factors, and stem cells (9,10). Many of...
the specifics regarding these mechanisms’ roles in exercise recovery are not fully understood (16). However, to the extent that it is reasonable to view acute, exercise-induced muscle impairments as an induced wound, it follows that mental strain may impair muscular adaptation through the recovery process. Little data exist to address this gap (12). Perna and McDowell (35) found that elite endurance athletes with similar training histories differentially recovered from an exhaustive, acute aerobic trial on the basis of their experience of chronic mental strain. There are, however, some limitations to this study. For instance, recoveries of muscular function and psychological state were not monitored.

Given the limited findings, the purpose of this study is to determine whether a relationship exists between psychological stress and recovery of muscular force after a bout of very strenuous resistance training. Obviously, recovery is a multistage process. Despite this, an initial assessment of these relationships may best begin with the first 60 min of recovery. If evidence supports this effect, further investigations may focus on later stages of recovery. It was hypothesized that self-reported psychological stress would moderate recovery of maximal isometric force (MIF), a measure of functional muscle performance, for a 1-h period after strenuous leg press exercise. Because psychological stress may affect force production through sensations of pain or fatigue, we also hypothesized that fatigue, soreness, and the recovery of psychological energy would be moderated by chronic self-reported stress.

**METHODS**

All subjects were recruited from a pool of approximately 1200 undergraduate students enrolled in resistance training classes. Of these, 210 students completed an online screening for perceived stress (Perceived Stress Scale (PSS)) (13). These responses were used to recruit a group of high-stress (1/2 SD above group mean) and low-stress (1/2 SD below group mean) individuals for further testing. Ineligibility criteria included recent illness or injury, musculoskeletal limitations or pain of the spine and lower body, steroid use, medications that could affect testing (e.g., muscle relaxants), consumption of ≥200 mg of caffeine per day, and depression levels greater than 27 on the Center for Epidemiological Studies Depression Scale (19). All participants signed an informed consent before beginning any laboratory procedures. This study was approved by the Institutional Review Board of The University of Texas at Austin.

Participants were eliminated for the following reasons: minor injury, volitional dropout, inability to complete the acute heavy-resistance exercise protocol (AHREP) or fitness testing with proper form, lingering soreness from previous workouts, and inability to complete the study within the needed time frame. The final sample included 31 participants who were 18–23 yr old (mean = 20.26 yr, SD = 1.34 yr), including 9 women and 22 men. Most subjects were recreationally trained and had not previously received formal instruction in strength training. No subject was an intercollegiate athlete. All students had a minimum of 5 wk of class instruction (approximately 10 class sessions) and experience with leg press exercise before the beginning of the AHREP protocol to minimize risk of macroadamage to the musculature. The physical characteristics of subjects are presented in Table 1.

**Procedures.** To control for various factors affecting recovery, eligible participants were instructed to abstain from the following: anti-inflammatories, sports supplements, supplemental antioxidants, and flax seed and fish oils for the experimental period (including 48 h before laboratory testing). Up to 3 h before measurements, caffeinated drinks were not allowed; meals were not allowed 2 h beforehand, and proper hydration was strongly encouraged. Participants were instructed to perform only light recreational exercise during the 48 h before laboratory testing. Fitness testing and protocol familiarization was scheduled within 1–3 wk after screening. Complete fitness testing took place in the following order: resting heart rate, body composition, MIF, vertical jump, maximal cycling power, muscular strength, and aerobic capacity. Five to 14 days after fitness testing, individuals returned for additional testing and the AHREP. On this day, participants completed the stress questionnaires and warmed up on a cycle ergometer (50 W, 60 rpm, 5 min). MIF, squat jump, and cycling power were retested. After lower body static stretches (15- to 20-s hold), they completed

<table>
<thead>
<tr>
<th>Variable</th>
<th>Women Mean</th>
<th>Women SD</th>
<th>Men Mean</th>
<th>Men SD</th>
<th>All Mean</th>
<th>All SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mass (kg)</td>
<td>62.003</td>
<td>14.198</td>
<td>75.013</td>
<td>12.842</td>
<td>71.236</td>
<td>14.326</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>21.127</td>
<td>13.979</td>
<td>27.124</td>
<td>18.952</td>
<td>25.124</td>
<td>15.983</td>
</tr>
<tr>
<td>Heart rate reserve (bpm)</td>
<td>120.111</td>
<td>11.634</td>
<td>125.273</td>
<td>13.548</td>
<td>123.774</td>
<td>13.048</td>
</tr>
<tr>
<td>V̇O₂max (L·min⁻¹)</td>
<td>1.979</td>
<td>0.290</td>
<td>2.888</td>
<td>0.616</td>
<td>2.624</td>
<td>0.681</td>
</tr>
<tr>
<td>V̇O₂max (mL·kg⁻¹·min⁻¹)</td>
<td>32.474</td>
<td>6.254</td>
<td>39.165</td>
<td>8.824</td>
<td>37.223</td>
<td>8.629</td>
</tr>
<tr>
<td>Leg press 1 RM (kg)</td>
<td>160.054</td>
<td>42.743</td>
<td>277.252</td>
<td>58.749</td>
<td>243.227</td>
<td>76.338</td>
</tr>
<tr>
<td>Leg press 1 RM (kg per mass)</td>
<td>2.581</td>
<td>0.640</td>
<td>3.696</td>
<td>0.708</td>
<td>3.414</td>
<td>0.879</td>
</tr>
<tr>
<td>Bench press 1 RM (kg)</td>
<td>39.217</td>
<td>17.376</td>
<td>72.43</td>
<td>16.310</td>
<td>62.787</td>
<td>22.396</td>
</tr>
<tr>
<td>Bench press 1 RM (kg per mass)</td>
<td>0.633</td>
<td>0.213</td>
<td>0.966</td>
<td>0.191</td>
<td>0.881</td>
<td>0.246</td>
</tr>
<tr>
<td>Jump power (W)</td>
<td>2680.904</td>
<td>1258.799</td>
<td>4089.336</td>
<td>680.211</td>
<td>3680.437</td>
<td>1081.095</td>
</tr>
<tr>
<td>Maximum jump height (cm)</td>
<td>35.719</td>
<td>13.836</td>
<td>46.124</td>
<td>9.611</td>
<td>43.349</td>
<td>11.619</td>
</tr>
<tr>
<td>Maximum cycling power (W)</td>
<td>1047.111</td>
<td>250.594</td>
<td>1445.810</td>
<td>307.099</td>
<td>1326.200</td>
<td>341.984</td>
</tr>
<tr>
<td>Maximum isometric force (N)</td>
<td>2125.788</td>
<td>726.006</td>
<td>3179.865</td>
<td>999.586</td>
<td>2873.849</td>
<td>977.880</td>
</tr>
</tbody>
</table>
the AHREP protocol. MIF, squat jump, cycling power, energy, fatigue, and soreness were measured again immediately. These same measures minus squat jump and cycle power were also measured every 20 min for 1-h postexercise. Water was provided ad libitum, but no food or other drinks were allowed during the testing procedures.

**Psychological measures.** Perceived chronic mental stress was measured with the PSS: 10-item version. The PSS measures the degree to which situations in one’s life are appraised as stressful (13). It is correlated with both quantity of life event stressors \( r = 0.32 \) and the negative effect of these events \( r = -0.27 \) (13,14). A large national sample of young adults, age 18–29 yr \( N = 645 \), had a mean PSS of 14.2 \( \text{SD} = 6.2 \). The pilot data for this study collected from 357 undergraduate students showed that these students had a mean PSS score of 14.4 \( \text{SD} = 5.5 \) at the beginning of the semester and 17.8 \( \text{SD} = 6.1 \) in the finals period. This was supplemented by the Undergraduate Stress Inventory (USQ), which was used to measure school and non-school-related life events that occurred in the month before evaluation. The objective form is a checklist that has 83 items representing common stressors for undergraduates (15). The USQ correlates well with other stress inventories (e.g., \( r = 0.79 \) with Subjective Distress Scale and \( r = 0.97 \) with the Objective Stressor Scale) (15). Finally, fatigue and energy were measured using the Visual Analog Scales (VAS) (23). Respondents placed a mark on 12 standard 10-cm lines that asked about the physical aspects of energy and fatigue. Examples of anchors include “I have no energy” to “strongest feelings of energy ever felt.” Soreness was assessed as an additional VAS item attached to the energy and fatigue scales. Anchors for the soreness VAS were “I have no feelings of soreness” to “strongest feelings of soreness ever felt.” The correlation between visit 1 and visit 2 pre-AHREP soreness was 0.58. The test–retest reliability \( (\alpha) \) over these two visits was 0.728.

**Fitness measures.** To quantify the fitness of our sample, aerobic capacity, body composition, and strength measures were collected. Aerobic capacity was determined with an incremental protocol test on an Excalibur Sport electronically braked cycle ergometer (Lode BV, Groningen, The Netherlands). This protocol was selected to minimize the risk of soreness before strength testing. This test starts at 25 W of resistance and increases in wattage progressively until volitional failure. \( \text{VO}_{2\text{peak}} \) is estimated from the peak wattage achieved at exhaustion (42). Resting and maximal heart rates were recorded with a Polar-OY (Kempele, Finland) telemetric heart rate monitor. Body composition (percentage body fat and fat-free mass (FFM)) was determined from dual-energy x-ray absorptiometry (Lunar DXA; GE Healthcare, Madison, WI). Female subjects indicated they were not pregnant before the scan. Lower and upper body strength was assessed via the bench and leg presses. Participants completed four to six sets of both these strength measures with a standard bench and a plate-loaded 45° Cybex machine (Cybex International Inc., Medway, MA). Weight progressively increased with each set. Strength was determined from muscular failure at three to five repetitions (reps) in the last set. 1-RM measures were determined from the coefficients reported by Brzycki (6).

MIF was determined on a modified leg press machine (45° plate-loaded Cybex). The machine was adjusted so that each individual was at a 110° knee joint angle, and the sled was fixed in place with adjustable attachments. Feet placement was approximately shoulder width, and measures were taken to reproduce foot placement with each measurement. An Omega LC101-3.0 k load cell (Omega Engineering, Inc., Stamford, CT) was used with an Omega DMD 460 (115 V) amplifier/signal conditioner and a Measurement Computing USB-1208FS Analog to Digital (A/D) Board. Data acquisition was accomplished with a Measurement Computing DAS-Wizard 3.0 for MS Excel at 1000 Hz for 3.0 to 4.0 s of maximal performance. Participants were given three trials to press maximally against the sled platform for 4 s, with force data collected by the load cell. Rest periods between trials were 30 s. Maximal force output (N) for each of these three trials was averaged. The correlation between visit 1 MIF and visit 2 preexercise MIF was 0.95, and the test–retest reliability \( (\alpha) \) was 0.972. The interitem correlation mean was 0.86, and the intraclass correlation was 0.85 for single measures and 0.98 for average measures (consistency index type; two-way mixed effects model where people effects are random and item effects are fixed). MIF correlates moderately with leg press 1 RM \( (r = 0.79) \), vertical jump power \( (r = 0.60) \), and maximal cycling power \( (r = 0.74; n = 55 \) for all correlations,

![FIGURE 1—Graphical representation of AHREP.](http://www.acsm-msse.org)
The correlation between visit 1 vertical jump height and visit 2 pre-AHREP jump height was 0.97, and the test–retest reliability (α) was 0.986. The correlation between visit 1 cycling power and visit 2 pre-AHREP cycling power was 0.95, and the test–retest reliability (α) was 0.974.

Vertical jump power was determined from a squat jump measured with a Vertec apparatus (Sports Imports, Inc., Columbus, OH). Power was calculated from the equation by Sayers et al. (39). Participants started in a crouched position (90° at the knee and hip) and were instructed not to use a countermovement. Participants were given three trials per collection period to achieve peak maximal jump height. Maximal cycling power was determined from a modified Monark cycle ergometer fitted with an optical sensor to determine velocity of the flywheel (31). The bike seat was adjusted so that knee flexion was between 10° and 20°. Participants started with the right crank arm of the bike parallel to the crossbar and cycled maximally against the resistance of the flywheel for 33 revolutions. Sixteen data points were collected per revolution. Participants were given three trials for each measurement period with 1 min of rest between performances.

**AHREP.** An AHREP was developed to reliably produce quantifiable amounts of muscular microtrauma and decrements in muscular function over a selected period (Fig. 1).

![Graph](image-url)

**FIGURE 2—**Predicted MIF over the first hour of recovery as moderated by USQ and PSS (measured at the first laboratory visit). Curves are adjusted for imputed values of the stress measure (mean and approximately ± 1 SD).

The AHREP session consisted of two stages. The first was a “ramping phase,” in which a 10-RM load was determined. This consisted of a variable number of sets of 10 reps, each performed with an increasing load until a full set could no longer be completed. The cadence of the movement was kept steady with a metronome, as 3-s eccentric action/2-s concentric action with a 1-s isometric hold at full extension (without locking the knees). Two minutes were provided for rest between each set. After the last set, 3 min of rest were provided before the beginning of the “burnout phase.” In this phase, six sets of leg presses were performed, each to volitional exhaustion (10 ± 2 reps). The load for the first set was the 10-RM capacity of the subject determined in the ramping phase. The second set was 90% of this value. If the subject was able to perform 10+ reps during the second set, sets 3–6 were maintained at this weight. Otherwise, the load was reduced to 80%. Participants were given strong verbal encouragement to complete the protocol.

**Statistical analysis.** Descriptive statistics (mean and SD) were calculated for PSS scores at the first visit (PSS-V1) and USQ. Kolmogorov–Smirnov (K-S) tests were conducted to determine normality of stress measures, and a Pearson’s product correlation was calculated between these variables. Chronic stress may have a relationship with measures of physical fitness (20); therefore, correlations were calculated between stress measures, workload (e.g., total weight lifted), body composition, and fitness-related constructs. Stepwise regression was used to determine whether a relationship existed between stress measures and changes in muscular function and feelings of energy and fatigue pre- to post-AHREP. Post-AHREP outcomes were regressed onto stress measures holding constant pre-AHREP values.

A two-level (observations, level 1 nested within persons, level 2) hierarchical linear modeling growth curve analysis (36) was used to detect differences in recovery trajectories by stress for MIF, energy, fatigue, and soreness for a 1-h postexercise period. First, simple intercepts-and-slopes-as-outcomes analyses were conducted to determine the functional form of time (linear, quadratic, and exponential) for each variable’s recovery curve analysis. Functions of time significant below a P value of 0.05 were retained for further moderation analyses. When modeling with two or more functions of time, it was necessary to fix the variance for at least one functional form of time. In such cases, these parameters had a higher number of degrees of freedom (df) associated with them. It was determined a priori that the PSS scale would be modeled as a linear/continuous variable if its distribution met normality requirements as determined by K-S tests. Next, stress measures were added as covariates at the level 2 to determine whether these variables moderated the recovery curves. Finally, it was planned that if USQ was significantly related to the recovery trajectories further models, it would be adjusted for covariates that had a significant relationship with muscular function (hours awake, muscular fitness, amount of FFM (kg), finals period (dichotomous), workload (total kilograms), magnitude of disruption from the...
AHREP protocol (percent), and training experience on the basis of completed semesters in the resistance training class (continuous). A similar model would be created for PSS-V1.

RESULTS

Of the 31 participants who completed the study, 18 scored into the low-stress and 13 scored into the high-stress groups from the online PSS screening (PSS-O). PSS scores from the first visit (PSS-V1; range, 3−27; mean = 14.9; SD = 6.8) indicated sufficient variability to test hypotheses. This variable was distributed normally, which indicated regression to the mean from the online survey (K-S statistic = 0.117, df = 30, P = 0.200). USQ scores also had sufficient variability (range 7−50, mean = 25.5, SD = 10.9) and had a normal distribution (K-S statistic = 0.109, df = 30, P = 0.200). Hence, hypothesis testing was conducted with stress measures as continuous linear variables. PSS-O had a positive linear relationship with PSS-V1 (r = 0.75, P < 0.001), and the latter had a positive linear relationship with USQ (r = 0.59, P = 0.001). Therefore, it was determined that separate multilevel (hierarchical linear modeling) analyses would be conducted for each measure of stress. USQ was related to bench press 1 RM (r = −0.37, P = 0.040), FFM (r = −0.50, P = 0.013), and MIF (r = −0.40, P = 0.028). PSS-V1 was only related significantly to percentage body fat (r = 0.36, P = 0.047). Both USQ and PSS-V1 were related to workload relative to body mass (i.e., total mass lifted per body mass; USQ, r = 0.40, P = 0.028; PSS, r = 0.40, P = 0.025), workload relative to FFM (USQ, r = 0.43, P = 0.017; PSS, r = 0.46, P = 0.009), and total reps (USQ, r = 0.47, P = 0.009; PSS, r = 0.49, P = 0.005). Stress measures were not related to absolute workload (total mass lifted), peak heart rate, or average heart rate in the burnout phase of the AHREP.

Changes in outcome variables pre- to postprotocol. The AHREP resulted in decreases in MIF (mean = 44.4%), squat jump (18.2%), cycle power (16.6%), and energy (35.1%) and increases in fatigue (126.5%) and soreness (69.7%). The main effect of time (pre- to post-AHREP) was significant for all variables (energy, P = 0.005; all others, P < 0.001), which indicated that all parameters of muscular and psychological function changed pre- to postexercise. After holding the preexercise values constant, baseline stress measures (USQ, PSS-V1) did not predict changes postexercise in MIF, jump height, perceived energy, fatigue, or soreness (all P > 0.05). USQ did not predict cycling power; however, PSS-V1 did predict this variable (β = −0.316, t = −2.350, P = 0.028).

Changes in outcome variables in recovery. After 60 min of recovery, MIF (mean = −17.1%), fatigue (+76.0%), and soreness (+161.1%) remained noticeably altered, although perceived energy had recovered (−0.5%). For MIF, linear (β = 1481.755, SE = 285.226, r ratio = 5.195, df = 30, P < 0.001) and squared (quadratic) curves (β = −796.535, SE = 203.440, r ratio = −3.915, df = 30, P = 0.001) provided the best functional form of time. Linear time provided the best functional form of time for energy (β = 158.270, SE = 36.902, r ratio = 4.289, df = 30, P < 0.001) and for fatigue (β = −194.750, SE = 41.825, r ratio = −4.656, df = 30, P < 0.001). Cubed (exponential) time provided the best fit of the data for soreness (β = −90.486, SE = 32.087, r ratio = −2.820, df = 115, P = 0.006). MIF, energy, and fatigue models were able to converge with the variance components estimated freely. For each, examination of the variances associated with linear time revealed that there was variability left to continue modeling additional variables (P < 0.001). Furthermore, there was significant variability in the squared function of time (P = 0.001). However, to facilitate model convergence for soreness, the cubed time parameter was fixed and no variance components were estimated. There was very little missing data for the first hour of recovery, which ranged from 2.5% (MIF) to 5.6% (soreness).

Moderation analyses for stress. Life event stress (USQ) significantly moderated the time–MIF relationship for both linear time (P = 0.013) and squared time (P = 0.050). In fact, the stress–time interaction remained significant for linear time (β = −62.527, SE = 17.828, r = −3.508, P = 0.002) and for squared time (β = 37.716, SE = 15.573, t = 2.422, P = 0.017) even after controlling for covariates, including recent examinations, fitness, FFM, number of semesters of the resistance training class, workload, and reduction in force. Comparing the variance from the unconditional model to the USQ conditional model determined that 9.2% of the variance was explained by the addition of this variable (Fig. 2).

Unlike life event stress, PSS-V1 did not significantly moderate the time–MIF relationship for linear time (P = 0.143) or squared time (P = 0.372). When the intercept was changed to 60 min and the analysis rerun, there was no difference at the intercept (PSS-V1, P = 0.363), which indicates that no differences existed at the end of the first hour of recovery. However, after controlling for significant covariates, including final exams, fitness, FFM, number of semesters of the resistance training class, workload, and reduction in force, the stress–time interaction was significant for linear time (β = −69.931, SE = 29.018, t = −2.410, df = 27, P = 0.023). PSS was not significant for squared time (P = 0.118). A median split of subjects by PSS at the first laboratory visit is

<table>
<thead>
<tr>
<th>Table 2. Recovery of MIF (R) by perceived stress.</th>
</tr>
</thead>
<tbody>
<tr>
<td>First Visit</td>
</tr>
<tr>
<td>Low stress (n = 16)</td>
</tr>
<tr>
<td>High stress (n = 15)</td>
</tr>
<tr>
<td>All subjects (n = 31)</td>
</tr>
</tbody>
</table>

Participants grouped by median split for PSS at the first laboratory visit (PSS > 13). Values are means (SD) and recovery percentage change from value immediately postexercise protocol.
DISCUSSION

This investigation explored the possibility that chronic psychological stress could have a measurable relationship with recovery of muscular and affective responses to a single bout of strenuous exercise. The current results from AHREP, therefore, are not immediately clear where stress may have its greatest effect on these processes. However, a burgeoning literature provides evidence that psychological stress is adversely associated with numerous physiological systems. For instance, wound healing studies demonstrate that stress impairs inflammatory responses, including those of interleukin-6 (10, 12), a cytokine that is highly responsive in the first few hours after strenuous eccentric movement (16). It is also well known that chronic stress results in dysregulated glucocorticoid responses and hypercortisolism (17, 38). Other mechanisms include depletion of catecholamines, irregular muscular activation (26), breathing patterns, blood pH (40), sympathetic neural burst firing patterns (27), glucose metabolism (34), atrophic gene expression and myostatin-dependent muscle atrophy (2), operation of the PI3-kinase/Akt signaling pathway, and increased apoptosis (18). In short, multiple central and peripheral mechanisms are implicated and may lead to musculoskeletal dysfunction over the long term, which should be an avenue for further investigation. Considering the multifactorial effects of stress throughout the human organism, is it of any surprise that stress is related to recovery over a single hour post-strenuous exercise?

There may also be a complex biobehavioral explanation for the effect of stress that goes beyond the immune and glucocorticoid hypothesis. Stressed individuals frequently experience poor sleep and nutrition and have less time for leisure and rest (33). Such conditions place the individual in a crisis of energy. Because the recovery process is energetically expensive (5), it would be protective for strained individuals to avoid or abstain from any behavior that could induce damage necessitating energy-expensive repair unless absolutely necessary (e.g., being chased by a lion, hunting for food to avoid starvation, and earning income) or imperative for the psyche (e.g., athletic competition and proving one’s competence) (38). It is possible that stress could be related to recovery simply because lesser stressed individuals approach the challenge with greater intensity and expend more effort, resulting in greater declines in function and therefore greater magnitudes of recovery (i.e., steeper slopes) for 1 h (29). However, these data do not confirm such a proposal. For instance, stress measures were not related to absolute workload, peak heart rate, or average heart rate throughout the AHREP exercise. Accordingly, stress was unrelated to force production, energy, and fatigue immediately postexercise when controlling for baseline values. Consequently, it does not appear that the observed results are due to differences in effort exerted during the AHREP protocol. Of course, this may not be the case when a person is in an unstructured/naturalistic environment or without resources to ensure completion of exercise. In such situations, individuals may resist expending much energy, regardless of the intensity (29). It was beyond the scope of the current investigation, however, to explore behavioral pathways connecting stress to recovery.

Limitations in the current study are evident but are outweighed by substantial strengths. Our timing of recovery was limited to 1 h. This was selected because pilot modeling of...
MIF after exhaustive exercise demonstrated that a single hour was sufficient to demonstrate a significant recovery. Furthermore, it is well established that numerous physiological changes occur within the first hour after strenuous exercise (1, 5, 7, 25). Indeed, interventions to enhance recovery, most prominently, nutritional interventions, have focused on the first hour as a critical window (24). As for the strenuous exercise protocol, the magnitude of sets being completed for a single exercise (in this case leg press) does not represent a typical workout. This, however, was required to ensure sufficient muscular disruption. Future studies should be conducted in naturalistic settings of high-intensity training, including any type of beginning exercise experience in which muscle damage is common. Another limitation was that chronic stress was measured retrospectively via self-report of stress events, which increases the opportunity for measurement error. It is, of course, not possible or ethical to manipulate chronic stress in humans to definitively determine causation. However, measures were taken over several time points to avoid regression to the mean, and these values of life stress correlated with present values of perceived stress. Thus, although a limitation, it does represent the most reasonable assessment. Future studies may wish to use naturally occurring stressors (e.g., final examinations) as an alternative. Likewise, a potential extension of this study would be a stress intervention to help individuals to monitor and manage their sources of stress and potentially enhance recovery. Either approach would serve to reinforce the present finding.

In summary, the present research demonstrates the relationship of psychological stress on physical recovery from strenuous exercise. Our data suggest that although stress does not affect the relative decline in muscular function and strenuous exercise. Our data suggest that although stress has an adverse effect even when recovery trajectories were adjusted by other factors related to stress and recovery, such as muscle mass, fitness, and workload completed during exercise. Thus, this relationship has a robust effect. Indeed, life event stress as measured by the USQ accounted for 9.2% of the variance explained in recovery. Our results are concordant with observations from Selye (41) who said, “When general stress is excessive the whole organism needs a rest; it cannot afford a struggle anywhere.” Interestingly, mental stress does not modulate recovery of perceived physical energy, fatigue, and soreness over a single hour. Regardless, delayed recovery comes at a high cost to those desiring enhanced fitness. Therefore, in light of our major findings, athletes and others undergoing bouts of strenuous exercise should undertake strategies to a) obviate the negative effects of chronic mental stress or b) monitor for stress and engage carefully in strenuous exercise when chronic stress is unavoidable. Because this is both a novel and important finding, further investigation into processes of stress and recovery represents an important area for continued inquiry.

The authors received no external funding for this research from the National Institutes of Health, Wellcome Trust, Howard Hughes Medical Institute, or any other funding agency.

The authors thank Charles “Chuck” Abolt and David Lassiter for their assistance on many facets of this manuscript.

In addition, there are no other conflicts of interest.

The results of the present study do not constitute endorsement by the American College of Sports Medicine.

REFERENCES


