Chronic Psychological Stress Impairs Recovery of Muscular Function and Somatic Sensations Over a 96-Hour Period

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Abstract

Stults-Kolehmainen, MA, Bartholomew, JB, and Sinha, R. Chronic psychological stress impairs recovery of muscular function and somatic sensations over a 96-hour period. *J Strength Cond Res* 28(7): 2007–2017, 2014—The primary aim of this study was to determine whether chronic mental stress moderates recovery of muscular function and somatic sensations: perceived energy, fatigue, and soreness, in a 4-day period after a bout of strenuous resistance exercise. Undergraduate resistance training students ($n = 31$; age, 20.26 ± 1.34 years) completed the Perceived Stress Scale and the Undergraduate Stress Questionnaire, a measure of life event stress. At a later visit, they performed an acute heavy-resistance exercise protocol (10 repetition maximum [RM] leg press test plus 6 sets: 80–100% of 10RM). Maximal isometric force (MIF), perceived energy, fatigue, and soreness were assessed in approximately 24-hour intervals after exercise. Recovery data were analyzed with hierarchical linear modeling growth curve analysis. Life event stress significantly moderated linear ($p = 0.027$) and squared ($p = 0.031$) recovery of MIF. This relationship held even when the model was adjusted for fitness, workload, and training experience. Perceived energy ($p = 0.038$), fatigue ($p = 0.040$), and soreness ($p = 0.027$) all were moderated by life stress. Mean perceived stress modulated linear and squared recovery of MIF ($p < 0.001$) and energy ($p = 0.004$) but not fatigue or soreness. In all analyses, higher stress was associated with worse recovery. Stress, whether assessed as life event stress or perceived stress, moderated the recovery trajectories of muscular function and somatic sensations in a 96-hour period after strenuous resistance exercise. Therefore, under conditions of inordinate stress, individuals may need to be more mindful about observing an appropriate length of recovery.

Key Words resistance training, mental stress, growth curve analysis

Introduction

Exercise, including resistance training, is generally touted for its profound health enhancing effects, including reduced insulin resistance, improved blood pressure, and less incidence of illness (43). The attainment of these benefits, however, does come at a physiological and psychological cost, particularly when exercise is unaccustomed, or of very high volume or intensity or any combination of these factors. For instance, high intensity resistance training results in profound fatigue, soreness, and reductions in muscular function immediately after a workout (40). Although these perturbations recover quickly over a several hour period, reductions in muscular force along with concomitant somatic symptoms reemerge over the course of multiple days of recovery. This pattern of immediate and longer-term change is known as the bimodal recovery response (14). Although this general pattern is clear, there is great variability in the magnitude of disruption and in the duration of recovery from intense training, which is not well explained by factors such as age, gender, and genetic factors (8). The identification of factors that influence trajectories of pain, function, and performance postexercise is important because (a) this is a period of physical vulnerability when further damage must be mitigated, (b) adverse psychological responses may dampen adherence to future exercise, and (c) such factors may be intervened on to accelerate the recovery response.

An ever-growing body of literature from diverse fields implicates psychological stress as a factor that modulates physiological recovery (42). Stress is a disruption from the homeostatic state that is frequently experienced as feeling out of control and taxed beyond one's capacity, generally resulting in feelings of emotional distress (10,22,36). Other theories posit that perceptions of distress are not necessary. Instead, the mere accumulation of life events can be

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sufficient to alter one's equilibrium even when one is not aware of their emotional impact (10,26). In a series of elegantly designed studies, it has been demonstrated that psychological stress measured using a variety of methods predicts speed of healing with those under higher levels of stress recuperating more poorly than their lesser stressed counterparts (7,42). For instance, 100% of dental students subjected to a mucosal wound during the summer academic recess and at final examinations recovered more slowly during this latter and more stressful period (25). Perna and McDowell(32) found that recovery of cortisol 1 day after an exhaustive aerobic performance trial was influenced by the experience of life stress. Indeed, stress has been related to a multitude of exercise and training-related outcomes, including impaired development of strength (3), smaller increases in aerobic capacity (35), altered immune function in the recovery period post-marathon (34), incidence of illness (44), and sport-related injuries (18).

A recent study implicates the effects of both life event stress and perceived stress on 60 minutes of muscular recovery from a bout of strenuous resistance (40). Specifically, 9.2% of the variance in maximal isometric force (MIF) was explained by life stress during this initial phase of the bimodal recovery response. Therefore, the effect appears to fall in the range reported for other physical outcomes, such as wound healing (42). Although this was the first investigation to report the effects of psychological stress on muscular recovery, there remains a need to follow recovery through its multistage process (8). There is reason to suspect that psychological stress may influence this longer-term recovery. For example, repair from exhaustive locomotion and wound healing are both highly elaborate processes involving the mobilization of cytokines, neutrophils, macrophages, growth factors, and stem cells over a multiday period (6,7). Because similar factors are likely to impede progression of recovery in the phase immediately after exercise, they are similarly likely to have an influence on longer-term processes. Consequently, there is reason to believe that mental strain may impair muscular adaptation over a multiday period of recovery.

At the present time, no data exist to substantiate an influence of psychological stress on multiday recovery from very strenuous resistance training. Therefore, the aim of this study is to analyze the 96-hour muscular recovery of 31 subjects previously reported (40). Specifically, it was hypothesized that self-reported psychological stress would moderate recovery of MIF, squat jump, and maximal cycling power, measures of functional muscle performance, over a 96-hour period following an acute heavy-resistance exercise protocol (AHREP). Because psychological stress may impact force production through sensations of pain or fatigue, it was additionally hypothesized that the recovery of perceived energy, fatigue, and soreness would be moderated by self-reported chronic stress.

Methods

Experimental Approach to the Problem

This study is concerned with the effects of chronic stress on 4 days of recovery, as opposed to the effects of an acute stress manipulation. Because it is both practically and ethically impossible to manipulate chronic stress, it was decided to use a quasi-experimental approach where a large sample of individuals (>1,200) would be screened for chronic stress (40). Those in the extremes (both high and low) were selected for further study participation (more details below). If chronic mental stress has a relationship with muscular recovery, it should be observable with a single cycle of strenuous exercise (32). To ensure that recovery processes were observable in a trained population, a strenuous bout of resistance training was implemented to produce muscular microtrauma and soreness.

Subjects

Subject recruitment, inclusion criteria, and characteristics have been published elsewhere (40). In short, 210 students in resistance training classes completed an online screening for perceived stress (Perceived Stress Scale [PSS]) (12). These responses were used to recruit a group of high stress (scores $\geq$19) and low stress (scores $\leq$13) individuals for further testing. Individuals scoring higher than 27 on the Center for Epidemiological Studies Depression Scale were not included (15). All participants signed an informed consent before beginning any laboratory procedures. The final sample included 31 participants who were 18–23 years of age (mean = 20.3, SD = 1.3) including 9 women and 22 men. This study was approved by the Institutional Review Board of The University of Texas at Austin. Physical characteristics of subjects are presented in Table 1.

Procedures

Overview. Participants made 6 visits to the laboratory for testing, the first of which occurred approximately 1–2 weeks after screening. At each visit, participants were assessed for somatic sensations (e.g., fatigue), and then they completed a brief warm-up on a cycle ergometer along with light lower-body stretches. Testing for muscular function (i.e., MIF, squat jump, and cycling power) was then administered. Visit 1 also included testing for fitness (described below) and familiarization with protocols. After 5–14 days, subjects returned for a strenuous exercise protocol (AHREP; explained below). Immediately after this protocol, MIF, squat jump, cycling power, energy, fatigue, and soreness (in this order) were remeasured. Participants stayed for 1-hour of post-exercise measurements and returned for their third to sixth visits for follow-up measures, which occurred in approximately 24-hour intervals. Participants were readministered the PSS at visits 1, 2, and 6. To control for various factors affecting recovery, participants were instructed to abstain from various substances 48 hours before and during the experimental period (e.g., anti-inflammatories), and caffeine

Table 1. Physical and performance descriptives for the current sample from the first laboratory visit ($n = 31$).*

Variable	Women		Men		All	
	Mean	*SD*	Mean	*SD*	Mean	*SD*
Mass (kg)	62.0	14.2	75.0	12.8	71.2	14.3
Fat mass (kg)	19.1	9.7	16.0	9.6	16.9	9.6
FFM (kg)	40.6	7.8	56.5	7.9	51.9	10.7
DXA body fat (%)	31.0	8.9	21.1	10.0	24.0	10.6
Max cycling work (W)†	154.4	28.8	183.4	59.4	175.7	54.1
Heart rate peak	189.1	12.9	188.5	9.5	188.7	10.3
Heart rate reserve ($b \cdot min^{-1}$)	120.1	11.6	125.3	13.6	123.8	13.1
$\dot{V}O_2$peak ($L \cdot min^{-1}$)‡	2.0	0.3	2.9	0.6	2.6	0.7
$\dot{V}O_2$peak ($ml \cdot kg^{-1} \cdot min^{-1}$)‡	32.5	6.3	39.2	8.8	37.2	8.6
Leg press 1RM (kg)	160.1	42.7	277.3	58.5	243.2	76.3
Leg press 1RM (kg per mass)	2.6	0.6	3.7	0.7	3.4	0.9
Bench press 1RM (kg)	39.2	17.4	72.4	16.3	62.8	22.4
Bench press 1RM (kg per mass)	0.6	0.2	1.0	0.2	0.9	0.3
Jump power (W)	2,680.9	1,258.8	4,089.3	680.2	3,680.4	1,081.1
Max jump height (cm)	35.7	13.8	46.1	9.6	43.4	11.6
Max cycling power (W)§	1,047.1	251.0	1,445.8	307.1	1,326.2	342.0
Max isometric force (N)	2,125.8	726.0	3,179.9	909.6	2,873.9	977.9

*RM = repetition maximum; FFM = fat free mass.
†From Storer aerobic capacity test on a excalibur sport ergometer (see text).
‡Aerobic capacity tests were completed at the end of the fitness assessment session.
§From cycling power test on a modified Monark ergometer fitted with an optical sensor (see text).

and food intake were limited before the AHREP protocol. Participants were instructed to perform only light recreational exercise during the 48 hours before laboratory testing. Additional procedural detail is supplied by Stults-Kolehmainen and Bartholomew (40).

Acute Heavy-Resistance Exercise Protocol. A leg press AHREP was developed to reliably produce quantifiable amounts of muscular microtrauma and decrements in muscular function over a selected time period (see Stults-Kolehmainen and Bartholomew (40) for a graphical representation). The AHREP session consisted of 2 stages. The first was a "ramping phase," in which a 10 repetition maximal (RM) load was determined. This consisted of a variable number of sets of 10 repetitions, 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 seconds eccentric action/2 seconds concentric action with a 1-second isometric hold at full extension (without locking the knees). Two minutes were provided for rest between each set. After the last set, 3 minutes of rest were provided before the beginning of the "burnout phase." In this phase, 6 sets of leg presses were performed, each to volitional exhaustion (10 ± 2 repetitions). The load for the first set was the 10RM 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+ repetitions 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.

Measures

Psychological Assessments. Perceived chronic mental stress was measured with the Perceived Stress Scale: 10-Item Version. The PSS measures the degree to which situations in one's life are appraised as stressful, with scores ranging from 0 to 40 (12). It is correlated with both quantity of life event stressors ($r = 0.32$) and the negative impact of these events ($r = -0.27$) (12). A large national sample of young adults (age, 18–29 years; $N = 645$), had a mean PSS of 14.2 ($SD = 6.2$). Pilot data for this study were collected from 357 undergraduate students in weight-training classes. These students had a mean PSS score of 14.4 ($SD = 5.5$) at the beginning of a semester and 17.8 ($SD = 6.1$) in their final examination period. The internal consistency for this earlier sample (Chronbach's alpha) was 0.76. This was supplemented by the Undergraduate Stress Questionnaire (USQ), which was used to measure school- and nonschool-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 (13). Finally, somatic

sensations were measured using visual analogue scales (VAS) (20). 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 (α) over these 2 visits was 0.73.

Fitness Assessments. To quantify the fitness of our sample, body composition, strength, and aerobic capacity were measured (in that sequence). Percent body fat and lean mass were determined from Dual emission x-ray absorptiometry (Lunar DXA, G.E., Madison, WI, USA). Female subjects indicated that they were not pregnant before the scan. To assess lower- and upper-body strength, participants completed 4–6 sets of leg and bench press movements with a standard bench and a plate-loaded 45° Cybex machine. Strength was determined from muscular failure at 3–5 repetitions in the last set, and 1RM measures were determined from coefficients reported by Brzycki (4). Aerobic capacity was determined with the Storer incremental protocol (39) on an Excalibur Sport electronically braked cycle ergometer (Lode BV, Groningen, The Netherlands). The $\dot{V}O_2$peak was estimated from the peak wattage achieved at volitional exhaustion. The test ended when subjects were not able to maintain 60 RPM (39). Heart rate was recorded with a Polar-OY telemetric heart rate monitor (Polar-OY, Kempele, Finland). Additional details are provided by Stults-Kolehmainen and Bartholomew (40).

Maximal isometric force 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 (21), and the sled was fixed in place with adjustable attachments. Participants were given 3 trials to press maximally against the sled platform for 4 seconds, with force data collected by an Omega LC101–3.0 k load cell (Omega Engineering, Inc., Stamford, CT, USA). Rest periods between trials were 30 seconds. Maximal force output (N) for each of these 3 trials was averaged.

Vertical jump power was determined from a squat jump measured with a Vertec apparatus (Sports Imports, Inc., Columbus, OH, USA). Power was calculated from the equation by Sayers et al. (37). Participants started in a crouched position (90° at the knee and hip) and were instructed not to use a countermovement. Maximal cycling power was determined from a modified Monark cycle ergometer fitted with an optical sensor to determine velocity of the flywheel, following the guidelines of Martin et al. (24). 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. For both assessments, participants were given 3 trials for each measurement period to achieve peak power.

Statistical Analyses

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 (16); 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 if 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 hierarchical linear modeling (HLM) growth curve analysis (33) was used to detect differences in recovery trajectories by stress for MIF, cycle and jump power, energy, fatigue, and soreness over a 96-hour postexercise period. First, simple intercepts-and-slopes-as-outcomes analyses were conducted to determine the functional form of time for each variable's recovery curve analysis (see Stults-Kolehmainen and Bartholomew (40) for more details). 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 to determine if these variables moderated the recovery curves. The p values ≤ 0.05 were deemed significant. Finally, it was planned that if USQ was significantly related to recovery trajectories, additional models would be adjusted for covariates that had a significant relationship with muscular function. These included hours awake, muscular fitness, fat-free mass (FFM, kg), finals period (dichotomous), workload (total kg), magnitude of disruption from the AHREP protocol (%), and training experience based on completed semesters in the resistance training class (continuous). A similar model would be created for PSS-V1 and mean PSS measured 4 times across the study period.

The correlation between visit 1 MIF and visit 2 pre-exercise MIF was 0.95 and test-retest reliability (α) 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; 2-way mixed effects model where people effects are random and item effects are fixed). Maximal isometric force correlates moderately with leg press 1RM ($r = 0.79$), vertical jump power ($r = 0.60$), and maximal cycling power ($r = 0.74$; $n = 55$ for all correlations, $p \leq 0.0001$ for all). The correlation between visit 1 vertical jump height and visit 2 pre-AHREP jump height was 0.97 and test-retest reliability (α) was 0.99. The

Table 2. Recovery of maximal isometric force (N) by perceived stress for low ($n = 16$) and high ($n = 15$) stress participants.*†

	First visit	Pre-AHREP	Post-AHREP	60-minute postexercise
Maximal isometric force				
Low stress	2,930.2 (1,048.3)	2,939.1 (1,064.0)	1,514.1 (635.5)	2,427.1 (1,089.5); 60.3%
High stress	2,813.5 (929.7)	2,801.8 (1,039.5)	1,685.8 (554.1)	2,330.1 (719.8); 38.2%
All subjects	2,873.9 (977.9)	2,872.4 (1,036.6)	1,596.5 (607.0)	2,380.1 (914.0); 49.1%
Jump height				
Low stress	44.4 (10.5)	45.6 (11.9)	38.1 (11.7)	N/A
High stress	42.2 (13.1)	41.7 (12.1)	36.4 (8.8)	N/A
All subjects	43.4 (11.6)	43.8 (11.9)	37.3 (10.3)	N/A
Cycle power				
Low stress	1,359.0 (327.1)	1,319.7 (367.5)	1,067.8 (275.6)	N/A
High stress	1,288.7 (366.9)	1,344.3 (413.7)	1,152.9 (371.2)	N/A
All subjects	1,326.2 (342)	1,332.0 (384.7)	1,109.0 (322.7)	N/A
	24 h	48 h	72 h	96 h
Maximal isometric force				
Low stress	2,745.9 (1,093.1); 81.4%	2,958.8 (1,024.5); 95.4%	2,827.4 (1,005.2); 86.7%	2,928.6 (1,076.9); 93.4%
High stress	2,497.9 (999.2); 48.1%	2,489.5 (844.5); 47.7%	2,598.5 (938.5); 54.1%	2,775.0 (1,079.8); 64.6%
All subjects	2,625.6‡ (1,038.8); 64.5%	2,715.5‡ (947.8); 70.1%	2,704.2§ (957.1); 69.4%	2,854.9 (1,058.5); 78.8%
Jump height				
Low stress	45.0 (11.5); 18.1%	49.6 (9.1); 30.2%	45.8 (12.1); 20.2%	47.5 (9.8); 24.7%
High stress	40.4 (12.7); 11.0%	38.6 (10.2); 6.0%	43.9 (12.4); 20.6%	41.1 (11.2); 12.9%
All subjects	43.0 (12.0); 15.3%	44.6§ (11.0); 19.6%	44.8 (12.0); 20.1%	44.9 (10.7); 20.3%
Cycle power				
Low stress	1,337.3 (297.7); 25.2%	1,385.2 (207.4); 29.7%	1,322.9 (281.5); 23.9%	1,455.8 (356.6); 36.3%
High stress	1,293.9 (413.1); 12.2%	1,301.2 (365.3); 12.9%	1,358.2 (380.4); 17.8%	1,357.8 (401.8); 17.8%
All subjects	1,317.1 (350.5); 18.8%	1,343.2‖ (294.2); 21.1%	1,341.2 (330.5); 20.9%	1,408.7 (374.3); 27.0%

*AHREP = acute heavy-resistance exercise protocol; PSS = Perceived Stress Scale; N/A = no measures collected at this time point.
†Values are given as means (*SD*) and percent change from post AHREP value. Participants grouped by median split for PSS at the first laboratory visit. High stress (>13), mean = 9.4 (3.0). Low stress (≤13), mean = 20.7 (4.1).
‡$p \leq 0.01$.
§$p \leq 0.05$.
‖$p = 0.067$.

correlation between visit 1 cycling power and visit 2 pre-AHREP cycling power was 0.95 and test-retest reliability (α) was 0.97.

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 = 0.12, $df = 30$, $p = 0.200$). Undergraduate Stress Questionnaire scores also had sufficient variability (range 7–50, mean = 25.5, $SD = 10.9$) and had a normal distribution (K-S statistic = 0.11, $df = 30$, $p = 0.200$). Hence, hypothesis testing was conducted with stress measures as continuous linear variables. The 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 (HLM) analyses would be conducted for each measure of stress. The Undergraduate Stress Questionnaire was related to bench press 1RM ($r = -0.37$, $p = 0.040$), FFM ($r = -0.50$, $p = 0.013$), and MIF ($r = -0.40$, $p = 0.028$). PSS-V1 was only related significantly to percent 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 repetitions (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.

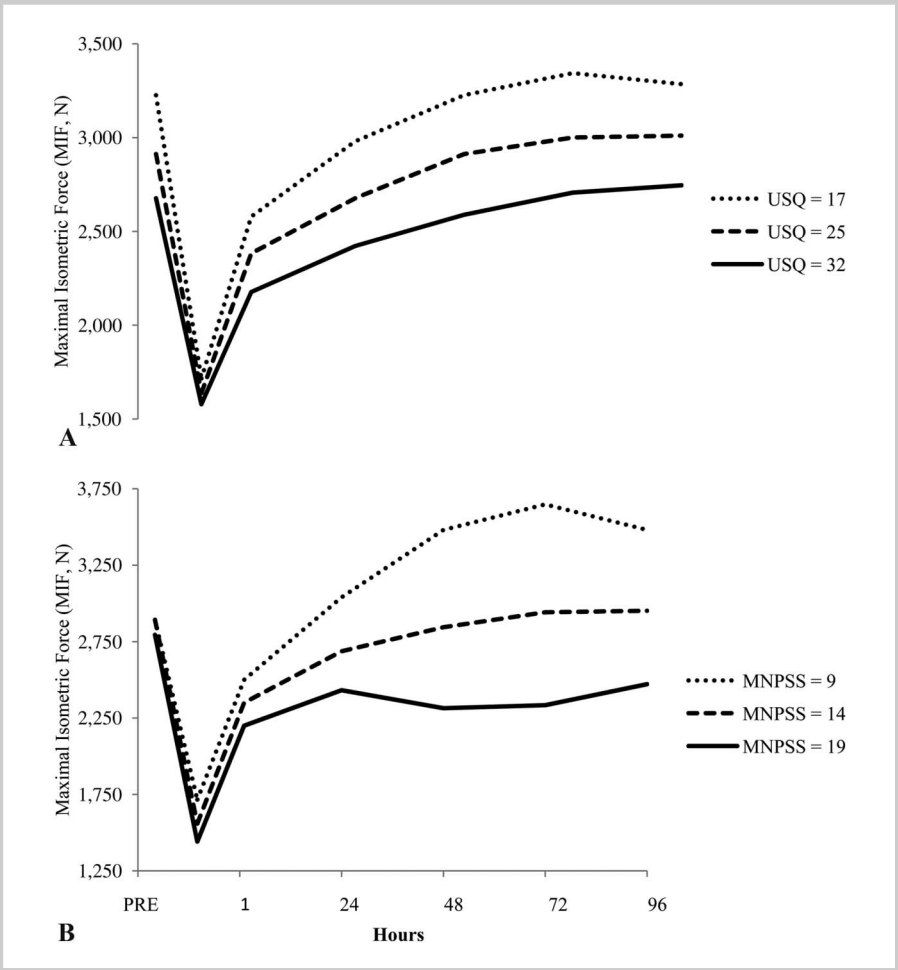

Figure 1. Predicted maximal isometric force (MIF) over 4 days of recovery as moderated by (A) life event stress (Undergraduate Stress Questionnaire [USQ]) and (B) mean perceived stress (MNPSS). Linear and quadratic time beyond the first hour is modeled. Curves are adjusted for imputed values of the stress measure (mean and approximately ±1 *SD*).

Changes in Outcome Variables

Pre- to Post-protocol. 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 $p \leq 0.001$), which indicated that all parameters of muscular function and somatic sensations changed pre- to post-exercise. After holding the pre-exercise values constant, baseline stress measures (USQ, PSS-V1) did not predict changes immediately postexercise in MIF, jump height, perceived energy, fatigue, or soreness (all p's > 0.05). The USQ did not predict cycling power; however, PSS-V1 did predict this variable ($\beta = -0.32$, $t = -2.35$, $p = 0.028$).

During Recovery. Visual examination of the trajectories confirmed that MIF, jump height, and cycling power increased with time. Table 2 shows percent change in these parameters over 96 hours of recovery. Following 2 days of recovery (a

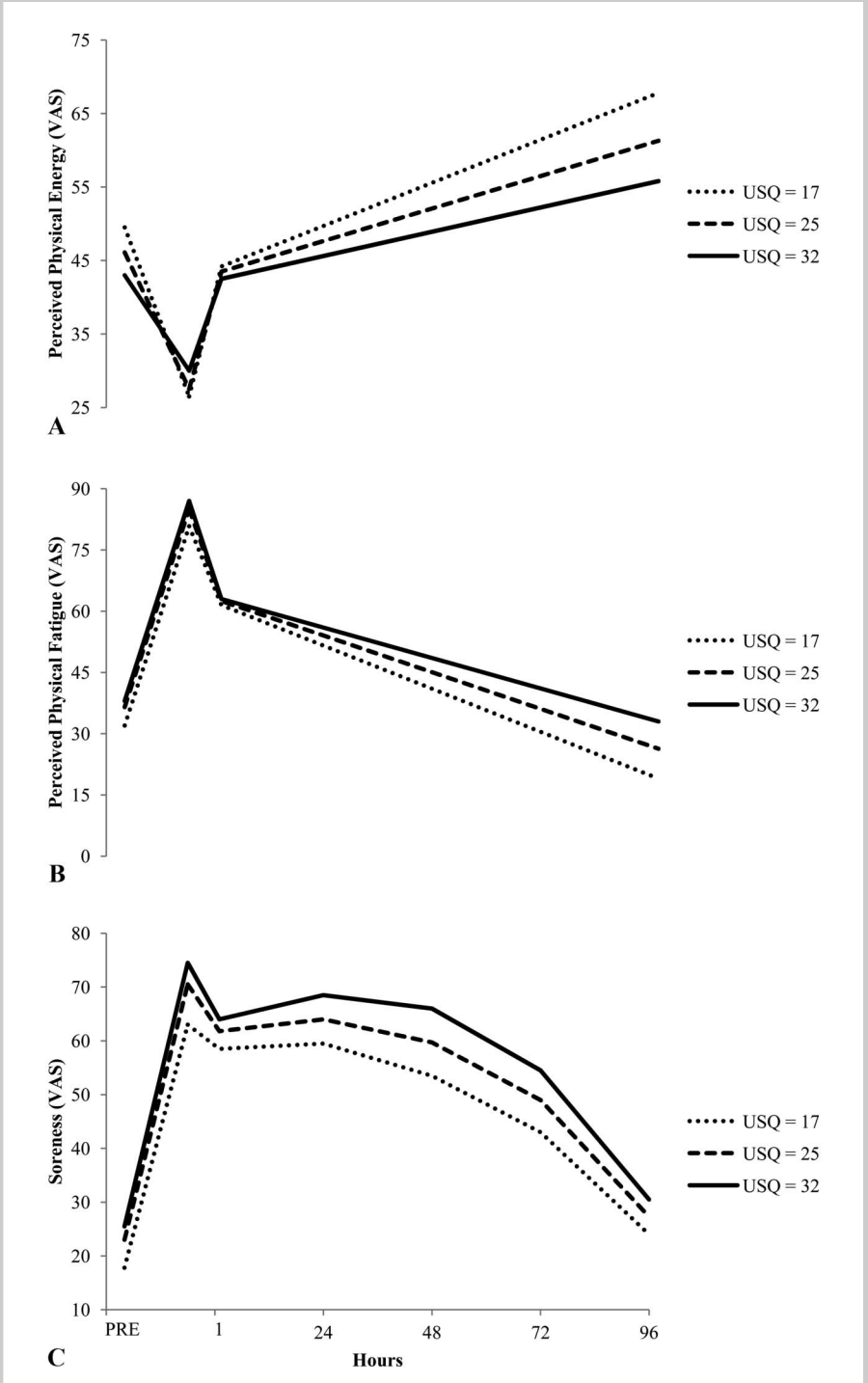

Figure 2. Predicted (A) perceived physical energy, (B) fatigue and (C) soreness over 4 days of recovery as moderated by life event stress (Undergraduate Stress Questionnaire [USQ]). Time beyond the first hour of recovery is modeled linearly for energy and fatigue. Linear, quadratic and exponential time is modeled for soreness. Curves are adjusted for imputed values of USQ (mean and approximately ±1 *SD*).

key time point for recovery), MIF (mean = −5.5%) had recovered across all subjects when compared with the initial baseline. This was also true for cycling power (0.0%) and jump height (+2.5%) and perceived energy (+2.0%). However, fatigue remained noticeably altered (+47.7% at 24 hours and +28.6% at 48 hours) as well as soreness (+224.3% at 24 hours and +191.6% at 48 hours). The functional form of time was the same for MIF and jump height, but not for maximal cycling power. For MIF, linear ($\beta = 1.51$, $SE = 0.28$, t-ratio = 5.38, $df = 30$, $p < 0.001$) and squared (quadratic) time ($\beta = -0.01$, $SE = 0.003$, t-ratio = −3.34, $df = 135$, $p = 0.001$) provided the best fit. For jump height (cm), again linear ($\beta = 0.10$, $SE = 0.03$, t-ratio = 3.17, $df = 29$, $p = 0.004$) and squared time ($\beta = -0.001$, $SE = 0.000$, t-ratio = −2.31, $df = 96$, $p = 0.023$) were optimal. For maximal cycling power, squared time by itself provided the best fit ($\beta = 0.02$, $SE = 0.003$, t-ratio = 6.25, $df = 106$, $p < 0.001$). As with cycling power, squared time provided the best functional form of time for physical energy ($\beta = 0.002$, $SE = 0.000$, t-ratio = 5.21, $df = 134$, $p < 0.001$). Linear time provided the best fit for physical fatigue ($\beta = -0.36$, $SE = 0.05$, t-ratio = −7.58, $df = 30$, $p < 0.001$). All forms of time were significant predictors of soreness, including linear time ($\beta = 1.06$, $SE = 0.27$, t-ratio = 3.95, $df = 30$, $p = 0.001$), squared time ($\beta = 0.000$, $SE = 0.000$, t-ratio = 3.39, $df = 132$, $p = 0.001$), and cubed (exponential) time ($\beta = -0.03$, $SE = 0.01$, t-ratio = −4.36, $df = 132$, $p < 0.001$).

Moderation Analyses

Life Event Stress. The USQ moderated the MIF and time relationship for both linear time ($p = 0.027$) and squared time ($p = 0.031$). Higher stress values were related to deeper or lower recovery curves for this muscular parameter. Furthermore, USQ moderated the MIF and time relationship for linear time ($p = 0.032$) and squared time ($p = 0.021$) after adjusting for covariates related to muscular functioning and recovery (recent exams, fitness, fat free mass, training experience, workload, and reduction in force). The relationship after controlling for covariates was in the same direction as previously. In contrast, USQ did not moderate the jump height and time relationship for linear time ($p = 0.497$) or for squared time ($p = 0.391$). The USQ did not moderate the linear time-cycling power relationship ($p = 0.856$). Life event stress also did not moderate this relationship for squared time ($p = 0.919$). However, the effect of life event stress approached significance for the intercept, indicating that higher stress individuals had lower power values at 24 hours after the AHREP workout ($p = 0.059$) (Figure 1).

There was a linear time by life event stress (USQ) interaction for the recovery trajectories of perceived physical energy ($p = 0.038$) and perceived physical fatigue ($p = 0.040$), over a 4-day period. For fatigue, those with higher life stress had higher recovery trajectories over 96 hours, which indicates that their fatigue declined at a slower rate. Life events stress was significantly related to the linear effect of time for soreness ($p = 0.027$) and approached significance for the quadratic effect of time ($p = 0.052$). Those with higher life event stress had a higher soreness trajectory over the 4 days (Figure 2).

Perceived Stress. Stress moderated the MIF and time relationship for both linear time (PSS at the first visit, $p = 0.003$; mean PSS, $p = 0.001$) and squared time (PSS at the first visit, $p = 0.002$; mean PSS, $p < 0.001$). Higher stress values for perceived stress were related to deeper or lower recovery curves for this muscular parameter. After adjusting for covariates, PSS moderated the MIF and time relationship for linear time (PSS at the first visit, $p = 0.004$; mean PSS, $p = 0.001$) and quadratic time (PSS at the first visit, $p = 0.001$; mean PSS, $p < 0.001$) related to muscular functioning and recovery. The relationship after controlling for covariates was in the same direction as previously (Figure 1 and Table 1).

Perceived Stress Scale at the first visit did not moderate the jump height and time relationship for linear time ($p = 0.094$), but approached significance for squared time ($p = 0.061$). Mean perceived stress did moderate the jump height and time relationship for both linear time ($p = 0.035$) and squared time ($p = 0.019$). Those reporting higher stress values were lower or deeper in their recovery curves. After adjusting for covariates, perceived stress moderated the jump height and time relationship for linear time (PSS at the first visit, $p = 0.035$; mean PSS, $p = 0.024$) and squared time (PSS at the first visit, $p = 0.028$; mean PSS, $p = 0.015$). The relationship after controlling for covariates was in the same direction as previously. Perceived stress did not moderate the linear time-cycling power relationship (PSS at the first visit, $p = 0.093$; mean PSS, $p = 0.289$). Stress also did not moderate this relationship for squared time (PSS at the first visit, $p = 0.083$; mean PSS, $p = 0.292$). However, there was a significant effect of stress on the intercept, indicating that higher stress individuals had lower power values at 24 hours after the AHREP workout (PSS at the first visit, $\beta = -23.62$, $SE = 11.29$, t-ratio $= -2.09$, $df = 29$, $p = 0.045$; mean PSS, $\beta = -26.02$, $SE = 13.15$, t-ratio $= -1.98$, $df = 29$, $p = 0.057$). Gender did not moderate recovery for any outcome variable.

Perceived stress moderated changes in perceived energy over time. There was a linear time by stress interaction for the recovery trajectories of energy over a 4-day period (PSS at the first visit, $p = 0.009$; mean PSS, $p = 0.004$). However, there was no moderation of 4-day recovery curves evident for perceived fatigue (PSS at the first visit, $p = 0.327$; mean PSS, $p = 0.210$). Perceived stress at the first visit and mean perceived stress did not moderate the linear time-soreness relationship (PSS at the first visit, $p = 0.898$; mean PSS, $p = 0.806$). No relationships were evident for the squared time-soreness relationship (PSS at the first visit, $p = 0.713$; mean PSS, $p = 0.683$).

Discussion

This investigation demonstrates that chronic mental stress has a measurable impact on the rate of functional muscle recovery from strenuous resistance training over a 4-day period. Specifically, higher levels of stress resulted in lower recovery curves and, conversely, lower levels of stress were associated with superior levels of recovery. Consequently, these data extend the results from Stults-Kolehmainen and Bartholomew (40), who found that stress impairs recovery in the short-term (1 hour) period after exercise. Remarkably, the current data demonstrate that those low in perceived stress returned to baseline several times faster than those reporting high levels of stress. Unlike the initial report, however, the current data provide evidence that stress also moderates the rebound of affective responses (i.e., soreness, energy, fatigue) associated with training. Because these relationships may be explained by factors such as fitness, workload, and training experience, statistical models of recovery were adjusted for these variables with a similar pattern of results emerging. In each case, the pattern of effects held for the impact of stress on the recovery of affective responses. Thus, these data explain some of the large variability associated with postexercise muscle function and soreness. It also serves to identify a source of vulnerability for those engaging in very strenuous exercise.

Functional recovery was also assessed through tests of physical performance: jump height and maximal cycling power. The stress-recovery relationship was less consistent for these measures. In regard to jump height, only mean perceived stress, and not life events, was related significantly

to recovery. For maximal cycling power, neither perceived stress nor life event stress was related to recovery curves over 96 hours. These differences were likely because of response specificity (30). The machine used to induce muscle damage was identical to the machine used to assess MIF (both 45° angle leg presses). Likewise, vertical jumps use musculature in a manner more similar to leg press than cycling. As such, it is not surprising that MIF was the most sensitive measure of recovery, with jump height producing similar but less pronounced effects. In contrast, cycling underemphasizes the hip and gluteus muscles. Therefore, muscles used to produce cycling power were likely to be less damaged than muscles used to leg press and jump. Consequently, there is less variability in the recovery curve potentially explained by psychosocial factors. While this interpretation fits these data, it is, of course, speculative. It would require the induction of muscle damage through various means, (e.g., leg press and cycling) to test definitively, which may provide fodder for future research.

Stress was also significantly related to recovery trajectories of energy, fatigue, and soreness over a 4-day period. For energy, this relationship held for perceived stress and for life stress. As with muscular measures, higher stress was related to deeper/lower recovery. However, for fatigue and soreness, this relationship held only for life event stress. Higher life event stress was associated with higher recovery slopes, which indicates slower recovery. These data stand in contrast with the results of Stults-Kolehmainen and Bartholomew (40), who found that stress was not related to recovery of somatic sensations over the first hour of recovery. It is not clear why these various differences were observed for these different time frames. One possibility is that affective measures demonstrated both a ceiling (soreness, fatigue) and basement effect (energy) in the first hour of recovery. This is the first study to test the recovery of perceived physical energy and fatigue from a damaging bout of resistance training, and more work is required to determine a reliable pattern of effects.

Although this study was not designed to assess specific mechanisms for the effect, there are numerous areas where future research might be aimed. Factors that overlap with both life events stress and subjective (perceived) stress may be a prime target for further inquiry because both indices of mental strain resulted in similar outcomes in this and former studies (40). This is interesting because perceived stress is a marker of one's cognitive evaluation (i.e., appraisal) of stress, while simple checklists of life event stress are not weighted by the subjective rating of stressor severity and magnitude (9). From a theoretical standpoint, however, these measures both are sensitive to the notion of physiological overload, which is a combination of high physical and mental demands and low resources resulting in overreaching beyond one's adaptive capacity (9,10). Under such conditions, one generally experiences increasing hedonic displeasure and negative affectivity, factors highly correlated with both types of stress (11). Personality traits, such as neuroticism, physical symptoms, stress reactivity, heart rate variability/parasympathetic activity, and sympathetic activation are but a few of a large number of factors covarying with multiple stress measures.

Activation of the HPA axis and the immune system are examples of numerous physiological processes involved in both repair from exercise-induced damage and psychological adjustments to physical stress (8,14). Such interplay between these systems is typically a highly synchronized process that ensures quick recuperation from challenging experiences (1,6). However, as reported in the wound-healing literature, key psychoneuroimmunological factors responsible for regeneration may be undermined by psychological stress (7,42). Inordinate mental strain is associated with a dysfunctional response of cytokines, protein signals that both promote and dampen inflammation. For instance, IL-6, IL-1β, and TNF-α, examples of cytokines involved in multiple processes of regeneration, are dysregulated by the experience of stress (7,8). Additionally, cytokines, such as IL-1β, play a central role in the perception of pain and fatigue after muscle damaging exercise (5). Alterations to the normal activation and quiescence of other immune-related factors have been linked to glucocorticoids, which are released in excess during times of unremitting stress (28).

Muscular recovery and specific responses from the immune system and HPA axis are responsive to a variety of lifestyle and behavioral factors. Sleep hygiene, proper nutrition, social support, and recreation are just a few variables associated with both stress and recovery (27). As a specific example, alcohol intake is both accentuated under periods of stress and related to poor muscular recovery (2,19). These factors may account for both the vulnerability of some individuals under the face of stress and the resilience of others (44). With increasing stress-related pressure, individuals may dampen their exercise effort, both in terms of intensity and volume of exercise, which would impact both the decline and recovery of muscular function (41). Nevertheless, our previous analysis discounts such a possibility because both perceived and life event stress measures were unrelated to indicators of workload, cardiovascular strain during the AHREP exercise protocol and baseline-adjusted force production, energy, and fatigue immediately after exercise (40). The current protocol is limited because it was highly structured, and participants were carefully acclimated, guided, and motivated throughout the experimental procedure. In a naturalistic setting, stressed individuals–particularly those unaccustomed to exercise–may be intolerant of the unpleasant sensations associated with locomotion, which is itself both a mental and physical stressor. Given this distinction, individuals will engage in a multitude of behaviors in attempts to cope with a disrupted state. This includes eating high fatty foods, use of drugs, alcohol, and nicotine, but in some cases over-employing healthy behaviors, such as physical activity itself (38). Unfortunately, it was beyond the

scope of the current analysis to explore behavioral pathways connecting stress to recovery.

Despite the substantial strengths of this study, there are some additional limitations. First, the participants in this study were college students in weight-training classes. Therefore, it is not clear that these data would replicate in more athletic populations. Moreover, the current sample was queried on retrospective measures of stress, which prohibits drawing firm conclusions about causation. It is possible that poor recovery becomes a source of stress, resulting in the experience of more daily hassles and distress. Future work employing longitudinal designs would permit firmer conclusions, particularly if active populations were examined over a series of strenuous training sessions–with resulting accumulation of fatigue. Given the prevalence of stress in society, however, there is a greater need for tailored interventions, such as mindfulness-based stress reduction (MBSR), cognitive behavior therapy, and restricted environmental stimulation technique (29,31). If such investigations provide evidence of reduced distress and enhanced recovery, this would further support the accumulating literature that supports a relationship between stress and physiological recuperation. Finally, the current investigation modeled perceived energy, fatigue, and soreness as separate outcomes, each of which had unique recovery trajectories. Future research should explore the possibility that affective responses are mechanisms mediating the relationship between stress and physical recovery.

In conclusion, the present research demonstrates a continuing association of psychological stress and recovery from strenuous exercise over a 96-hour period. These data extend previous work that found recovery trajectories for MIF were moderated by chronic mental stress. A unique finding captured in these data was that other indicators of muscular performance and psychological adjustments made over a 4-day period were also adversely impacted by stress. These findings are strengthened by the fact that these relationships were similar for multiple indices of stress. This provides evidence that stress undermines recuperation of physical function and somatic sensations regardless of how stress is measured. Similar results were obtained even when statistical models were adjusted for factors that might explain recovery trajectories, such as FFM, fitness, and workload. Taken together, these data provide a remarkably consistent picture of poorer recovery for individuals reporting inordinate stress. Considering the multitude of information concerning the salubrious effects of mild to moderate exercise on stress-related phenomena, further inquiry needs to carefully delineate the conditions under which exercise may optimize physical and psychological functioning.

Practical Applications

Psychological stress is related to many adverse outcomes, but it is not currently known how stress gets "under the skin" (17). These data provide evidence that stress has an impact on recovery from heavy bouts of training. Those reporting high levels of stress take several more days to recover than those reporting less stress. This robust effect likely has practical significance for those facing the dual challenges of chronic mental strain and strenuous exercise, which suggests that those engaging in strenuous strength training should "exercise caution when stressed" (23). Consequently, it may be prudent for such individuals to monitor recovery and prescribe more time for recuperation during periods of inordinate mental stress. Furthermore, coaches and trainers may elect to use stress instruments, such as the PSS, a measure that has generated a great deal of normative data. Administering these brief instruments at baseline and over several time points across the season may provide a wealth of information about an individual's risk for poor recovery. Providing opportunities for rest and relaxation and encouraging the use of stress management techniques, such as MBSR, are suggested methods to help athletes cope or mitigate the effects of mental strain. In cases of unremitting stress, however, coaches may need to consult with and refer athletes to licensed mental health professionals.

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References

1. Armstrong, RB. Initial events in exercise-induced muscular injury. *Med Sci Sports Exerc* 22: 429–435, 1990.
2. Barnes, MJ, Mündela, T, and Stannarda, SR. Acute alcohol consumption aggravates the decline in muscle performance following strenuous eccentric exercise. *J Sci Med Sport* 13: 189–193, 2010.
3. Bartholomew, JB, Stults-Kolehmainen, MA, Elrod, CC, and Todd, JS. Strength gains after resistance training: The effect of stressful, negative life events. *J Strength Cond Res* 22: 1215–1221, 2008.
4. Brzycki, M. Strength testing: Predicting a one rep max from a reps-to-fatigue. *J Phys Educ Recreation Dance* 64: 88–90, 1993.
5. Carmichael, MD, Davis, JM, Murphy, EA, Brown, AS, Carson, JA, Mayer, EP, and Ghaffar, A. Role of brain IL-1 beta on fatigue after exercise-induced muscle damage. *Am J Physiol Regul Integr Comp Physiol* 291: R1344–R1348, 2006.
6. Charge, SBP and Rudnicki, MA. Cellular and molecular regulation of muscle regeneration. *Physiol Rev* 84: 209–238, 2004.
7. Christian, LM, Graham, JE, Padgett, DA, Glaser, R, and Kiecolt-Glaser, JK. Stress and wound healing. *Neuroimmunomodulation* 13: 337–346, 2007.
8. Clarkson, PM and Hubal, MJ. Exercise-induced muscle damage in humans. *Am J Phys Med Rehabil* 81: S52–S69, 2002.
9. Cohen, S. Contrasting the Hassles scale and the perceived stress scale–who's really measuring appraised stress? *Am Psychol* 41: 716–718, 1986.

10. Cohen, S, Kessler, RC, and Gordon, LU. Strategies for measuring stress in studies of psychiatric and physical disorders. In: *Measuring Stress: A Guide for Health and Social Scientists*. New York, NY: Oxford University Press, 1995. pp. 3–24.
11. Cohen, S, Tyrrell, DAJ, and Smith, AP. Negative life events, perceived stress, negative affect, and susceptibility to the common cold. *J Pers Soc Psychol* 64: 131–140, 1993.
12. Cohen, S and Williamson, G. Perceived stress in a probability sample of the United States, In: *The Social Psychology of Health: Claremont Symposium on Applied Social Psychology*. S. Spacapam and S. Oskamp, eds. Newbury Park, CA: Sage, 1988. pp. 31–67.
13. Crandall, CS, Preisler, JJ, and Aussprung, J. Measuring life event stress in the lives of college students: The undergraduate stress Questionnaire (USQ). *J Behav Med* 15: 627–662, 1992.
14. Dousset, E, Avela, J, Ishikawa, M, Kallio, J, Kuitunen, S, Kyrolainen, H, Linnamo, V, and Komi, PV. Bimodal recovery pattern in human skeletal muscle induced by exhaustive stretch-shortening cycle exercise. *Med Sci Sports Exerc* 39: 453–460, 2007.
15. Ensel, WM. Measuring depression: The CES-D scale. In: *Social Support, Life Events, and Depression*. N. Lin, A Dean, and W.M. Ensel, eds. New York, NY: Academic Press, 1986. pp. 51–70.
16. Ensel, WM and Lin, N. Physical fitness and the stress process. *J Community Psychol* 32: 81–101, 2004.
17. Epel, ES, Blackburn, EH, Lin, J, Dhabhar, FS, Adler, NE, Morrow, JD, and Cawthon, RM. Accelerated telomere shortening in response to life stress. *Proc Natl Acad Sci U S A* 101: 17312–17315, 2004.
18. Ford, IW, Eklund, RC, and Gordon, S. An examination of psychosocial variables moderating the relationship between life stress and injury time-loss among athletes of a high standard. *J Sports Sci* 18: 301–312, 2000.
19. Fox, HC, Bergquist, KL, Hong, KI, and Sinha, R. Stress-induced and alcohol cue-induced craving in recently abstinent alcohol-dependent individuals. *Alcohol Clin Exp Res* 31: 395–403, 2007.
20. Herring, MP and O'Connor, PJ. The effect of acute resistance exercise on feelings of energy and fatigue. *J Sports Sci* 27: 701–709, 2009.
21. Hulmi, JJ, Ahtiainen, JP, Selänne, H, Volek, JS, Häkkinen, K, Kovanen, V, and Mero, AA. Androgen receptors and testosterone in men-Effects of protein ingestion, resistance exercise and fiber type. *J Steroid Biochem Mol Biol* 110: 130–137, 2008.
22. Lazarus, RS and Folkman, S. Cognitive theories of stress and the issue of circularity. In: *Dynamics of Stress Physiological, Psychologcal, and Social Perspectives*. M. Appley and R. Trumbull, eds. New York, NY: Plenum, 1986. pp. 63–80.
23. Lutz, RS, Stults-Kolehmainen, MA, and Bartholomew, JB. Exercise caution when stressed: Stages of change and the stress-exercise participation relationship. *Psychol Sport Exerc* 11: 560–567, 2010.
24. Martin, JC, Wagner, BM, and Coyle, EF. Inertial-load method determines maximal cycling power in a single exercise bout. *Med Sci Sports Exerc* 29: 1505–1512, 1997.
25. Marucha, P, Kiecolt-Glaser, J, and Favagehi, M. Mucosal wound healing is impaired by examination stress. *Psychosom Med* 60: 362–365, 1998.
26. McEwen, BS. Physiology and neurobiology of stress and adaptation: Central role of the brain. *Physiol Rev* 87: 873–904, 2007.
27. Miller, GE, Cohen, S, Pressman, S, Barkin, A, Rabin, BS, and Treanor, JJ. Psychological stress and antibody response to influenza vaccination: When is the critical period for stress, and how does it get inside the body? *Psychosom Med* 66: 215–223, 2004.
28. Miller, GE, Cohen, S, and Ritchey, AK. Chronic psychological stress and the regulation of pro-inflammatory cytokines: A glucocorticoid-resistance model. *Health Psychol* 21: 531–541, 2002.
29. Morgan, PM, Salacinski, AJ, and Stults-Kolehmainen, MA. The acute effects of Flotation restricted environmental stimulation technique on recovery from maximal eccentric exercise. *J Strength Cond Res*. In press.
30. Morrissey, MC, Harman, EA, and Johnson, MJ. Resistance training modes–specificity and effectiveness. *Med Sci Sports Exerc* 27: 648–660, 1995.
31. Perna, FM, Antoni, MH, Baum, A, Gordon, P, and Schneiderman, N. Cognitive behavioral stress management effects on injury and illness among competitive athletes: A randomized clinical trial. *Ann Behav Med* 25: 66–73, 2003.
32. Perna, FM and McDowell, SL. Role of psychological stress in cortisol recovery from exhaustive exercise among elite athletes. *Int J Behav Med* 2: 13–26, 1995.
33. Raudenbush, SW and Bryk, AS. *Hierarchical Linear Models: Applications and Data Analysis Methods*. Thousand Oaks, CA: Sage, 2002. pp. 3–15.
34. Rehm, KE, Elci, OU, Hahn, K, and Marshall, GD. The impact of self-reported psychological stress levels on changes to peripheral blood immune biomarkers in recreational marathon runners during training and recovery. *Neuroimmunomodulation* 20: 164–176, 2013.
35. Ruuska, PS, Hautala, AJ, Kiviniemi, AM, Makikallio, TH, and Tulppo, MP. Self-rated mental stress and exercise training response in healthy subjects. *Front Physiol* 3: 51, 2012.
36. Sapolsky, RM. *Why Zebras Dont Get Ulcers: An Updated Guide to Stress, Stress-related Diseases, and Coping*. New York, NY: Owl Books, 2004, p. 6.
37. Sayers, SP, Harackiewicz, DV, Harman, EA, Frykman, PN, and Rosenstein, MT. Cross-validation of three jump power equations. *Med Sci Sports Exerc* 31: 572–577, 1999.
38. Sinha, R. Stress and addiction: A dynamic interplay of genes, environment, and drug intake. *Biol Psychiatry* 66: 100–101, 2009.
39. Storer, TW, Davis, JA, and Caiozzo, VJ. Accurate prediction of VO_{2max} in cycle ergometry. *Med Sci Sports Exerc* 22: 704–712, 1990.
40. Stults-Kolehmainen, MA and Bartholomew, JB. Psychological stress impairs short-term muscular recovery from resistance exercise. *Med Sci Sports Exerc* 44: 2220–2227, 2012.
41. Stults-Kolehmainen, MA and Sinha, R. The effects of stress on physical activity and exercise: A systematic review. *Sports Med*. In press.
42. Walburn, J, Vedhara, K, Hankins, M, Rixon, L, and Weinman, J. Psychological stress and wound healing in humans: A systematic review and meta-analysis. *J Psychosom Res* 67: 253–271, 2009.
43. Westcott, WL. Resistance training is medicine: Effects of strength training on health. *Curr Sports Med Rep* 11: 209–216, 2012.
44. Yi, JP, Smith, RE, and Vitaliano, PP. Stress-resilience, illness, and coping: A person-focused investigation of young women athletes. *J Behav Med* 28: 257–265, 2005.