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Physiological and Psychological Recovery from Muscle Disruption following Resistance Exercise: The Impact of Chronic Stress and Strain

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Physiological and Psychological Recovery from Muscle Disruption following Resistance Exercise: The Impact of Chronic Stress and Strain

by

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Dedication

I dedicate this dissertation to those who seek respite in times of stress and uncertainty.

"Tempest-tossed souls, wherever ye may be, under whosoever conditions ye may live,
know this – in the ocean of life the isles of Blessedness are smiling,
and the sunny shore of your ideal awaits your coming."

James Allen (1864-1912)

Epigraph

"When general stress is excessive the whole organism needs a rest;

it cannot afford a struggle anywhere."

-Hans Selye (1976, p. 435)

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Preface

No topic was more improbable to pique my interest than stress. Indeed, no material baffled me more throughout my undergraduate and masters' studies. What is stress? To ask others this question is to subject oneself to a litany of responses, most of which will center on the effects of stress and not on stress itself! To espy the branches of a tree rustling one may be tempted to describe this motion as the wind; it is, rather, a reaction. Perhaps the wind causes this response, or perhaps it is a child at play.

Many academics expound on the effects of stress without attending fully to its definition. Hence, the conversation begins with each person at a different starting point. One student imagines stress as a strong emotional reaction; another recalls the concept from a physics perspective; a third thinks about a recent trip to her advisor's office. Such an approach transforms concrete concepts into abstractions. My own personal experience was marked by a general discomfort born of this ambiguity. It is no wonder that I lost interest in these discussions. The only comments memorable were invariably in reference to Selye's GAS (General Adaptation Syndrome).

Little did I know, however, that stress had permeated the studies in which I cultivated great interest. My master's thesis was on the topic of exercise training periodization (how one directs training over long periods of time), a subject built on the tenets of General Adaptation Syndrome. I utilized this knowledge to craft my own training programs and coach others. In essence, I had become educated on how to purposely use stress to physically grow stronger. An incomplete understanding of stress, however, left me susceptible to injuries. This vulnerability created topical relevance and ignited an interest in pursuing a better understanding of stress, strain, and recovery.

Arriving at the University of Texas at Austin, I was immersed in conversation about the effects of stress on fitness adaptations. I discovered the struggles of others who have pursued this line of research and succeeded to make contribution to the topic. I also learned about the process of stress, which ends in recovery and adaptation. Armed with new knowledge and confidence I rigorously embarked on a series of pilot studies on chronic stress and muscle development (described within). That led me to develop the model (Appendix A) from which I crafted this dissertation. It has been a challenging journey through which the concept of recovery entered the forefront and is the centerpiece of my dissertation. As a result, I have become welcoming of the multifarious nature of the topic, and intrigued at how an increased understanding of recovery will certainly refocus the discussion; in this lay the future.

Physiological and Psychological Recovery from Muscle Disruption following Resistance Exercise: The Impact of Chronic Stress and Strain

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A large body of evidence supports the notion that chronic stress and strain may impact healing from physical trauma. However, no evidence exists to substantiate whether chronic stress impacts recovery from exercise-induced muscle damage. In this study, a group of 31 undergraduate weight-training students completed the Perceived Stress Scale (PSS), Undergraduate Stress Survey (USQ, a measure of life event stress) a series of fitness tests and then returned 5 to 10 days later for an exhaustive resistance exercise stimulus (E-RES) workout. This workout was performed on a leg press to the cadence of a metronome to ensure a strong eccentric component of exercise. Participants were monitored for 1 hour after this workout and every day for 4 days afterwards. Hierarchical Linear Modeling (HLM) multi-level growth curve analyses demonstrated that stress measures were related to recovery from maximal resistance exercise for both functional muscular (maximal isometric force, jump height, and cycling power) and psychological (perceived energy, perceived fatigue, and soreness) outcomes. Stress was

not related to outcomes immediately post-workout (except maximal cycling power) after controlling for pre-workout values. Thus, the effect of stress on recovery is not likely due to magnitude of disruption from maximal exercise. After controlling for significant covariates, including fitness and percent disruption from baseline, individuals scoring a 10 on the PSS at their first visit reached baseline 288% (2.88 times) faster than individuals who scored a 19 at this same time point. There were significant moderating effects of stress on affective responses during exercise. Feeling (pleasure/displeasure), activation (arousal), muscular pain and RPE (exertion) trajectories were moderated by stress. Exploratory analyses found that stress moderated physical recovery, but not psychological recovery in the first hour after the E-RES workout. Also, stress was related to the increase in IL-1β, a pro-inflammatory cytokine, in the 48 hour period after exercise for a sub-set of participants. These findings likely have important theoretical and clinical implications for those undergoing vigorous physical activity. Those experiencing chronic loads of stress and mental strain should include more rest time to ensure proper recovery.

Table of Contents

List of Tables xviii

List of Figures xxii

List of Abbreviations xxvi

CHAPTER ONE − INTRODUCTION 1

Disruption and Recovery within Exercise Training 1

Protein Breakdown and Recovery 3

Similarity of Muscle Recovery to Other Recovery Processes 4

Potential Impact of Mental Strain on Recovery from Exercise Stress 6

The Current Study: Aims 7

Brief Methods 7

Hypotheses 9

Hypothesis 1a 9

Hypothesis 1b 9

Hypothesis 2a 10

Hypothesis 2b 10

Hypothesis 2c 10

Hypothesis 2d 10

Hypothesis 2e 10

Brief Statistical Analysis 11

Definitions of Terms 11

Delimitations 13

Experimental design 13

Sample 13

Generalizability of workout and damage 14

Fitness measurement 14

Timing of measures 15

Limitations 15

Generalization 15

Experimenter bias 16
Strain caused by the study 16
Quantification of physical work 16

CHAPTER TWO − LITERATURE REVIEW 17
Muscular Disruption and Recovery in the Context of Exercise 17
Affective Responses to Resistance Exercise: The Good, Bad, and Ugly 19
Acute Responses 19
Pain, Soreness and Long-Term Responses 20
Pain during exercise 20
Pain after exercise 21
Repeated bout effect 22
Methodological Problems with Exercise-Affect Research: Using Resistance Training Paradigms 22
The Nature of Physiological Breakdown and Recovery from Exercise 25
Stress and Strain Processes 25
Mechanical Breakdown 31
Inflammation 32
Chronic Breakdown with Lack of Recovery 34
Similarity of Muscle Recovery to other Recovery Processes 35
Processes of Regeneration 35
Wound Healing Studies of Recovery. A Decade of Research (1995-2007) 37
Specific Mechanisms of the Stress and Healing Relationship 40
Inflammatory Cytokines 40
Glucocorticoids 41
General Cytokine and Cortisol Interactions 42
Exercise-specific cytokine and cortisol interactions: Providing Answers to Paradoxical Relationships 43
Stress, Strain, and Recovery Processes: Is Responsiveness The Most Important Factor? 44
Key Concepts 44
Reactivity and Responsiveness 47

Impact of Mental Stress/Strain on Athletic Injuries, Health and Fitness Adaptations 53
Stress, Pain, Fatigue, and Recovery 56
Methodological Problems with Previous Research 57
Issues related to Quantification of Stress, Strain, and Recovery 57
Issues related to subject characteristics and ecological validity 57
CHAPTER THREE − METHODS 58
Experimental Design 58
Description of Subjects 59
Demographics 59
Health History 59
Menstrual and Pregnancy Status 60
Exercise Experience 60
Behavioral Restrictions 60
Supplements and Over-the-Counter Medications 60
Caffeine 61
Exercise and Therapy 61
Procedures 61
Recruiting 61
Screening 62
Fitness Testing, Blood Draw, and Familiarization 62
Assessments: General Screening, Psychological Screening, and Personality 65
Assessments: Measures of Fitness and Blood Draw 67
E-RES Workout 68
Exhaustive Resistance Exercise Stimulus (E-RES) Protocol 69
Post-E-RES Workout (Visits 3-6) 70
Dependent (Outcome) Variables 71
Statistical Analysis 79
CHAPTER FOUR − RESULTS 84
Introduction and Recap of Methods 84
Summary of Experimental Design and Statistical Analysis 85

Descriptive Statistics and Preliminary Analyses 88
Demographics and Finals Status 88
Diagnostics to Determine Scale of Measurement for Stress 89
Collinearity of Stress/Strain Measures 90
Physical Fitness, Body Composition, and Anthropometric Characteristics 91
Missing Data 91
Workload Completed E-RES Workout 92
The Effect of the E-RES Workout (Time) on Functioning and the Sphericity Assumption 93
Examining the Functional Form of Time: Do Outcomes Change? 93
Relationship of Hours Awake to Muscular Function 95
Summary 96
Hypothesis Testing 96
Research Questions 96
Hypotheses 97
Life Event Stress (USQ) 98
Perceived Stress 106
Hypothesis 2a: 110
Finals 115
Exploratory Analyses 118
Exploratory Research Questions 118
First Hour of Recovery 118
Missing Data 119
Muscular Functioning (MIF Only) 120
Psychological Function 122
Cytokines 123
Summary 124
CHAPTER FIVE − DISCUSSION 125
Introduction 125
Interpretation of Major Findings 126
Muscular Recovery 126

Psychological Recovery 129
Affective Disruption during Exercise 130
Mechanisms of the Stress-Recovery Relationship 133
Interpretation of Physical Sensations 133
Cytokines 133
Motor Control 136
Strengths of the Current Study 137
Limitations 137
Sample Size and Composition 138
Measurement Error 139
Manipulation of Stress 139
Summary 140
Practical and Theoretical Implications 140
Future Directions 143
Experience with Intense Exercise 143
Health Behaviors and Other Factors 144
Interventions 145
Bi-Directional Effects Research 146
Conclusion 147
Appendix A – Proposed Model 183
Appendix B – PSS 186
Appendix C – Energy and Fatigue: How You Feel Right Now 188
Appendix D – Demographic Questionnaire 190
Appendix E – Health Questionnaire 191
Appendix F – PRETIE-Q−TOL 198
Appendix G – PAIN 199
Appendix H – RPE (Category-Ratio-10, Borg, 1998) 201
Appendix I – Feeling Scale (F.S.), Felt Arousal Scale 202

Appendix J – CES-Depression Inventory.....203

Appendix K – PANAS: Positive and Negative Affect Schedule (Watson, Clark, & Tellegen, 1988).....205

Appendix L – USQ: Undergraduate Stress Questionnaire (Crandall, Preisler, & Aussprung, 1992) 206

References.....209

Vita 232

List of Tables

Table 2.1 Leg press strength and muscular endurance in trained and untrained men and Women. Adapted from Hoeger et al. (1987, 1990). 25

Table 2.2 Comparison of wound-healing and muscle regeneration. These processes share common immunological mechanisms and the employment of adult stem cells for repair (see Chargé & Rudnicki, 2004; Christian et al., 2007; Labarge & Blau, 2002 & Werner & Grose, 2003). Besides obvious differences between muscle tissue and the epidermis, there are several other notable differences. 36

Table 3.1 Overview of study visits and study time allotment..... 64

Table 3.2 Ramping phase of E-RES workout 70

Table 3.3 Duty cycles and testing schedule for leg press exercise stimulus protocol. Work was for 60 seconds. Rest was for 120 seconds..... 78

Table 3.4 Dissertation timeline. 83

Table 4.1 Baseline, disruption and recovery values for muscular and psychological function 4-day post-workout (means and % change)..... 86

Table 4.2 Disruption and recovery for muscular and psychological function 4-day post-workout (means and % change). 87

Table 4.3 Disruption and recovery for MIF 1 hour post-workout (means and % change) 120

Table 4.4 Pearson Product Correlations 149

Table 4.5 Descriptives for stress/strain measures *.....150

Table 4.6 Physical and performance descriptives for the current sample (n = 31) and for a group of physical activity class students (n = 380) 150

Table 4.7 Correlations between physical characteristics, USQ, and PSS at the first visit 151
Table 4.8 HLM level 1 descriptives for four days of recovery 152
Table 4.9 HLM level 1 descriptives for during the E-RES workout 153
Table 4.10 Relation of life event stress to four days of recovery for maximal isometric force (MIF; kg) * 154
Table 4.11 Relation of life event stress to 4 days of recovery for maximal isometric force (MIF; kg) adjusting for significant covariates * 155
Table 4.12 Relation of stress/strain measures with physical energy in the four days after workout for USQ 156
Table 4.13 Relation of USQ with physical fatigue in the four days after workout157
Table 4.14 Relation of USQ with soreness in the four days after workout 158
Table 4.15 Relation of perceived stress (visit 1) to four days of recovery for maximal isometric force (MIF; kg) 159
Table 4.16 Relation of perceived stress (mean) to four days of recovery for maximal isometric force (MIF; kg) 160
Table 4.17 Relation of perceived stress (mean) to four days of recovery for maximal isometric force (MIF; kg) 161
Table 4.18 Relation of perceived stress (average over all visits) to 4 days of recovery for maximal isometric force (MIF; kg) adjusting for significant covariates 162
Table 4.19 Relation of perceived stress (first visit) to 4 days of recovery for maximal isometric force (MIF; kg) adjusting for significant covariates 163

Table 4.20 Relation of perceived stress (average over all visits) to 4 days of recovery for vertical jump height (cm) 164
Table 4.21 Relation of perceived stress (average over all visits) to 4 days of recovery for vertical jump height (cm) 165
Table 4.22 Relation of perceived stress at the first visit to feeling (pleasure) during the E-RES workout * 166
Table 4.23 Relation of perceived stress at the first visit to activation (arousal) during the E-RES workout * 167
Table 4.24 Relation of perceived stress at the first visit to muscular pain during the E-RES workout * 168
Table 4.25 Relation of perceived stress at the first visit to RPE during the E-RES workout *.....169
Table 4.26 Relation of stress/strain measures with physical energy in the four days after workout for PSS at the first 170
Table 4.27 Relation of perceived stress with physical fatigue in the four days after workout.....171
Table 4.28 Relation of finals (average over four visits) to four days of recovery for maximal isometric force (MIF; kg).....172
Table 4.29 Relation of being in the finals period to 4 days of recovery for maximal cycling power (watts).....173
Table 4.30 Relation of finals with physical energy in the four days after workout174
Table 4.31 Relation of finals with physical fatigue in the four days after workout. 175
Table 4.32 Relation of finals with soreness in the four days after workout 176
Table 4.33 HLM level 1 descriptives for the first hour of recovery 177

Table 4.34 Relation of USQ (life event stress) to the first hour of recovery for maximal isometric force (MIF; kg).....178
Table 4.35 Relation of USQ (life event stress) to first hour of recovery in maximal isometric force (MIF; kg) after adjusting for covariates.....179
Table 4.36 Relation of perceived stress (first visit) to first hour of recovery in maximal isometric force (MIF; kg) after adjusting for covariates *180
Table 4.37 IL-1β, IL-6 and TNFα for low and high stress groups (means and standard deviations). Units are pg/ml.....181
Table 4.38 Relation of stress to IL-1β for linear time.....182

List of Figures

Figure 2.1 Parameters describing disruption of physiological homeostasis and functioning with various recovery curves. A. Thriving, B. Return to baseline functioning, C. Adaptation to a level just below baseline functioning, D. Significant decrease or impairment in functioning following disruption. E. Extreme maladapation or death. Adapted from Carver, 1998. 27

Figure 2.2 Biphasic or bimodal recovery curve as seen in MacIntyre et al. (1996). Eccentric torque values are displayed as means + SD. Pre, pretest. 29

Figure 2.3 Functional muscle capability is affected by energy availability and structural capacity. 30

Figure 2.4 Model of chronic, unremitting physical stress and excessive cortisol release. Diagram simplified from Smith's (2000) Cytokine Hypothesis of Overtraining..... 35

Figure 2.5 Healing time for 11 individuals at summer vacation (low stress, black bars) and at examination time (high stress, gray bars). Note that all 11 individuals had longer or delayed recovery during the high stress period. Chart from Marucha et al., 1998..... 38

Figure 2.6 Kellman's Scissors Model of Stress States and Recovery Demands (1991, 1997, 2000). When demands are chronically high and recovery is poor, adaptation will not be optimal. The model demonstrates the balance that must be achieved in order to reach a high level of performance. 48

Figure 2.7 Cortisol responses in well-trained endurance athletes of high- and low-stress after maximal exercise (Perna & McDowell, 1995). The pre-test means for the high- and low-stress groups is 1.267 (SD = 0.3481) and 1.559 (SD = 0.381), respectively. 49

Figure 2.8 The impact of psychological stress on physical strain reactions. Stress reactivity, comprised of disruption and recovery, has many identifiable parameters, including: i. mobilization (time to reach asymptote; brackets A & B), ii. point of recovery (rings on curves), iii. return time to baseline (i.e., recovery; brackets C & D), iv. disruption magnitude (difference from baseline; brackets E & F) and v. disruption magnitude (AUC – area under the curve; shaded area). Disruption and recovery can also be quantified as slope (rise over run). Conceptual basis adapted from Dienstbier (1989, 1992) and visual representation created by the author..... 51

Figure 3.1 Flowchart of participants through study..... 63

Figure 3.2 Individual demonstrating set-up for Maximal Isometric Force (MIF) measurement. 72

Figure 3.3 Detail of MIF apparatus. Notice turnbuckle, black strap, and orange ratchet tie-down. Lines around platform of sled and along frame provide reference for positioning of feet on platform and angle of knee. 72

Figure 4.1 Distribution of PSS stress scores from online screening. Participants scoring between 14 and 18 were not eligible for the study. 89

Figure 4.2 Distribution of PSS stress scores from the first visit. 90

Figure 4.3 Relation of USQ to MIF recovery curves over 4 days. Time is modeled linearly and quadratically. Logarithmic time was not significant. Solid line indicates data analyzed with HLM. Dashed lines indicate level baseline values and level of disruption. 99

Figure 4.4 Relation of USQ to jump height recovery curves over 4 days. Time is modeled linearly and quadratically. Logarithmic time was not significant. Solid line indicates data analyzed with HLM. Dashed lines indicate level baseline values and level of disruption. 100

Figure 4.5 Relation of USQ to jump height recovery curves over 4 days. Time is modeled linearly and quadratically. Logarithmic time was not significant. Solid line indicates data analyzed with HLM. Dashed lines indicate level baseline values and level of disruption. 101

Figure 4.6 Rating of perceived exertion over the course of the E-RES workout.103

Figure 4.7 Perceived physical energy at baseline, disruption pre- to post- workout and rise over a 4-day recovery period. 104

Figure 4.8 Perceived physical fatigue at baseline, disruption pre- to post- workout and rise over a 4-day recovery period. 105

Figure 4.9 Soreness (pain) at baseline, disruption pre- to post- workout and rise over a 4-day recovery period. 106

Figure 4.10 Relation of mean PSS to MIF recovery curves over 4 days. Time is modeled linearly and quadratically. Logarithmic time was not significant. 108

Figure 4.11 Relation of mean PSS to jump height baseline values, disruption pre- to post-workout, and recovery curves over 4 days. 109

Figure 4.12 Relation of PSS at the first visit to changes in feeling during the E-RES workout.....111
Figure 4.13 Relation of PSS at the first visit to changes in activation (arousal) during the E-RES workout. 112
Figure 4.14 Relation of PSS at the first visit to changes in muscular pain during the E-RES workout. 113
Figure 4.15 Relation of PSS at the first visit to changes in RPE during the E-RES workout. Note that in this diagram, time is modeled linearly, quadratically, and exponentially. 114
Figure 4.16 Relationship of finals period to cycling power recovery curves over 4 days. Time is modeled linearly and quadratically. 116
Figure 4.17 Maximal isometric force over the first hour of recovery by USQ..121
Figure 4.18 Maximal isometric force over the first hour of recovery by first visit PSS. 122
Figure 4.19 IL-1β (pg/ml) over 48 hours. 124
Figure 5.1 Potential paths from chronic stress to muscular function during the recovery period after strenuous exercise. 135

List of Abbreviations

APES – Adolescent Perceived Events Scale

CHO – Carbohydrate

CRP – C – Reactive Protein

DEXA – Dual Energy X-Ray Absorpiometry

DOMP – Delayed-onset muscle pain

E-RES – Exhaustive Resistance Exercise Stimulus protocol (the “workout” completed at the second visit)

FAS – Felt Arousal Scale

FS – Feeling Scale

GAS – General Adaptation Syndrome

GXT – Graded exercise test

HIIT – High intensity interval training

HLM – Hierarchical Linear Model

HPA – Hypothalamic-Pituitary-Adrenal

IL-1β – Interleukin-1β

IL-6 – Interleukin-6

MIF – Maximal isometric force

MVPA- Moderate to vigorous physical activity

NK – Natural Killer cell

OTS – Overtaining Syndrome

PAART – Psychosocial Aspects of Adaptation to Resistance Training

PANAS – Positive and Negative Affect Scale

PSS – Perceived Stress Scale

RM – Repetition maximum

RPE – Rating of perceived exertion

SAM – Sympathetic Adrenal Medullary system

SRSS – Social Readjustment Rating Scale

TNF-α – Tumor necrosis factor-α

USQ – Undergraduate Stress Questionnaire

VAS – Visual analogue scale

CHAPTER ONE

INTRODUCTION

DISRUPTION AND RECOVERY WITHIN EXERCISE TRAINING

To the novice exerciser, physical training is counter-intuitive. The ostensible goal for this purposeful training is to increase muscular strength, endurance, power, and hypertrophy. Yet, to achieve this, one begins by draining energy systems and damaging muscle (Fry, 1999, p. 149). In fact, the physical stress or overload of intense exercise actually results in a short-term reduction in strength. This dissertation is concerned with individual variation in the nature of this strength reduction and a person's ability to recover muscular and psychological equanimity following intense exercise-induced muscle damage. It is specifically focused on the impact of psychological stress and mental strain on these relationships.

The period following intense exercise actually produces a bimodal (two-humped) strength response. There is an immediate reduction in strength post-exercise (up to 50-65% loss) followed by a short-lived rebound (Nosaka et al., 1991). Strength again decreases 24-48 hours later (MacIntyre, Reid, Lyster, Szasz, & McKenzie, 1996; Dousset et al., 2007). This bimodal return to homeostatic balance is characterized by fatigue during the initial period (several hours) followed by inflammation, swelling and inflexibility along with a concomitant experience of extreme pain and stiffness in the later period (several days). The experience of these sensations is referred to as delayed onset muscle pain/soreness or DOMP. Interestingly, DOMP does not correlate well with classic physiological measures of muscle damage, such as creatine kinase, a muscle enzyme that seeps from the myofiber cytosol after tissue disruption (Clarkson & Hubal,

2002). Whereas DOMP occurs within 24-48 hours post intense exercise, creatine kinase (CK) usually does not peak for several days after DOMP has subsided (Clarkson & Hubal). Accordingly, although pain may subside, strength may not return to baseline for up to a week or longer after a bout of unaccustomed, fatiguing exercise.

Certain types of exercise training, such as weight lifting and high intensity interval training (HIIT), are particularly associated with pain and prolonged recovery from muscular damage.[1] This is especially true when training is unaccustomed. These training methods are increasingly popular with both athletes and non-athletes. Indeed, current exercise recommendations include resistance training several times per week (American College of Sports Medicine & American Heart Association, 2007) and many publications are touting the utility of HIIT, even for relatively untrained individuals (Coyle, 2005; Gibala & McGee, 2008; Laursen & Jenkins, 2002; Trapp, Chisholm, Freund, & Boutcher, 2008). These protocols commonly call for several training sessions per week. Unfortunately, those who are initiating an exercise regimen often fail to consider the need for additional recovery time. Without this, subsequent training when in a damaged condition may lead to further physiological disturbance, extended healing time, exacerbated sensations of pain and fatigue, and further decrements in performance (Mair et al., 1995; O'Connor, Morgan, & Raglin, 1991). Heightened feelings of pain have long been deemed a barrier to further participation in physical training (Pasley & O'Connor, 2008; Roth, 1974, Vogt, et al., 2002). Likewise, negative mood responses to exercise have been associated with drop out (Tompkins, Kwan, Bryan, Marcus, & Ciccolo, 2007). Buckworth and Dishman (2007, p. 511) and Hall, Ekkekakis, and Petruzzello (2002) indicate that negative mood is the most detrimental factor to exercise

[1] HIIT programs, a generic designation for all training programs with a highly intense, short-period nature, are not to be confused with HIT training, which was first described by Arthur Jones (the inventor of Nautilus resistance training machines) in 1970 and is currently touted by Ellington Darden, Ph.D. in a series of popular books.

behavior. Thus, it is likely that a failure to properly recover from intense exercise may eventually lead to a diminished sense of enjoyment and motivation for exercise and eventual dropout, commonly reported amongst new exercisers (Fleck & Kraemer, 2004, p. 44; Ryan, Frederick, Lepes, Rubio, & Sheldon, 1997). Consequently, it is not surprising that high-intensity interval exercise programs have lower adherence than moderate- and low-intensity programs (Perri et al., 2002; Lee et al., 1996). While repeated exposure to intense exercise is protective against damage and pain (Nosaka, Sakamoto, Newton, & Sacco, 2001), athletes are not immune from the experience of soreness and poor recovery. Athletes often experience excessive training coupled with poor recovery, which has been strongly implicated in burnout or over-training syndrome (OTS; Kellman, 2000). OTS is characterized by intolerance to further training, sensitivity to pain and excessive feelings of fatigue even when training is greatly reduced. Thus, O.T.S. may represent a complete depletion of physiological and psychological compensatory mechanisms. In sum, exercise-induced muscle damage is an area of concern for a wide swath of exercisers, from beginners to advanced individuals and even highly competitive athletes.

PROTEIN BREAKDOWN AND RECOVERY

Loss of muscle function, including strength, is likely related to acute energy disruption, protein breakdown (proteolysis), loss of muscle architecture (sarcomere disruption), biochemical changes (excitation-contraction coupling dysfunction), inflammation, oxidative stress (directly or indirectly) and other factors. Muscle degeneration occurs at the level of the surface membrane (sarcolema), t-tubules, sarcoplasmic reticulum and the individual muscle sarcomere (Allen, 2001). Specific mechanisms responsible for this damage are not clearly understood, but initial disruption is due to the mechanical perturbation of the fiber itself (Clarkson & Hubal, 2002). This

results in sarcomere inhomogeneity, whereas these structures become weakened or overstretched (Allen) along with concomitant accumulation of intracellular calcium (Gissel & Clausen, 2001). Later disruption is related to: inflammatory processes, further degeneration of muscle fibers making them inexcitable, failure of individual filaments within the muscle to reinterdigitate after disruption (Allen,), and changes in excitation-contraction coupling within the muscle leading to reduction of calcium release (Clarkson & Hubal, 2002). These disruptions are reflected in the impairment of functional measures such as isometric muscle strength.

Recovery from muscular disruption is a highly synchronized process (Chargé & Rudnicki, 2004). As mentioned previously, recovery of muscular function follows a bimodal pattern with the immediate recovery phase taking up to five hours and the long recovery phase taking up to 7-10 days (MacIntyre et al., 1996; Dousset et al., 2007). This pattern suggests that many different physiological mechanisms are at play. Following tissue breakdown and inflammation response comes proliferation and differentiation of myogenic and satellite cells. Myogenic cells fuse to damaged fibers to complete repair or fuse to one another to form new fibers (Chargé & Rudnicki, 2004). Signaling factors such as IGF-1 and regulation from myostatin are important players in this process (Chargé & Rudnicki, 2004). Despite this understanding, outside influences on muscular recovery from individual bouts of training are not well understood. Even when controlling for gender, age, fitness, and training history, variance explaining muscular recovery time is still largely unexplained (Clarkson & Hubal, 2002; Ebbeling & Clarkson, 1989).

SIMILARITY OF MUSCLE RECOVERY TO OTHER RECOVERY PROCESSES

The recovery process from damage is remarkably similar regardless of the method of induction or the tissue involved. Wound healing, for instance, requires several more actions than muscle regeneration to ensure proper repair (e.g., neutrophil accumulation to

prevent infection and re-epithelialization to cover the wound). Nevertheless, the mechanisms involved are quite similar (e.g., growth factors and cytokines; see Table 2.2 for a comparison of wound healing and muscle repair; Chargé & Rudnicki, 2004; Christian, Graham, Padgett, Glaser, & Kiecolt-Glaser, 2007; Labarge & Blau, 2002; Werner & Grose, 2003).

These similarities are important because the wound-healing literature has identified individual factors that have explained significant speed of recovery. For instance, there is a growing body of evidence concerning the potential effects of psychological stress on wound healing. The tissue healing process, though highly structured, is impacted by the experience of chronic mental stress and strain. Specifically, mental strain may delay the recovery (or regeneration) phase, thus impairing or weakening physical adaptations. A series of wound healing studies demonstrates that psychological stress impacts the ability to recover from a variety of naturalistic and induced physical injuries, such as superficial wounds to the epidermis. This effect of mental stress and strain on healing has been corroborated in numerous investigations studying a variety of populations (Broadbent, Petrie, Alley, & Booth, 2003; Kiecolt-Glaser & Glaser, 1995; Kiecolt-Glaser, Glaser, Gravenstein, Malarkey, & Sheridan, 1996; Kiecolt-Glaser et al., 2005; Marucha, Kiecolt-Glaser, & Favagehi, 1998). Likewise, the effects have been replicated across a range of types of tissue damage, with an amazing degree of consistency in the magnitude of statistical effect size. Mental strain impairs wound healing in a variety of different tissues (Ebrecht et al., 2004; Marucha et al., 1998; Wikesjo, Nilveus, & Selvig, 1992) and the effect is the same regardless of the method used to inflict damage (Altemus, Rao, Dhabhar, Ding, & Granstein, 2001; Glaser et al., 1999; Kiecolt-Glaser & Glaser, 1995; Roy, et al., 2005). Across these studies, stress-related time delays in healing range from 24-40% and the effect sizes (cited by the

authors as squared correlation as a proportion of variance explained by stress) are between $r^2 = 0.30$ and $r^2 = 0.74$. Glaser et al. (1999) notes that widespread evidence from stress interventions targeting the ill and infirm also supports the stress-recovery hypothesis for systemic recovery.

Potential Impact of Mental Strain on Recovery from Exercise Stress

There is reason to believe that mental strain may affect recovery from exhaustive exercise. Recovery from exhaustive exercise follows a structured process involving physiological mechanisms similar to those implicated in the wound healing studies (P. M. Clarkson, personal communication, November 17, 2007; Daruna, 2004, p. 217). Consequently, to the extent that it is reasonable to view exercise-induced muscle damage as an induced wound, it follows that mental strain may impair muscular adaptation through the recovery process. Perna & McDowell (1995) found that elite endurance athletes with similar training histories differentially recovered from an exhaustive, acute aerobic trial based on their experience of chronic mental strain. Likewise, Bartholomew, Stults-Kolehmainen, Elrod, and Todd (2008) found that those who reported a disproportionate level of negative, stressful events experienced reduced strength gains following 13 weeks of resistance training.

Given these relationships, it is reasonable to posit that psychological stress and mental strain may slow recovery from exercise-induced muscle damage. To date, no study has tested this possibility. A review by Clow & Hucklebridge (2001, p. 10) concludes, "The question as to whether chronic psychological stress alters responses to physical stress does not appear to have been investigated." This is surprising, as resistance exercise provides an ideal paradigm to test these hypotheses (Chargé & Rudnicki, 2004). This modality of exercise incorporates significant eccentric movement, resulting in structural damage in addition to depletion of energy reserves. One may

readily measure muscular recovery and adaptation. The current dissertation is designed to fill this void.

THE CURRENT STUDY: AIMS

This investigation is designed to achieve the following general aims:

i. to determine whether chronic mental strain, as measured by the Undergraduate Stress Questionnaire (USQ) and Perceived Stress Scale (PSS) is associated with physiological recovery from muscle damage induced by an exhaustive resistance exercise stimulus (E-RES).

ii. to determine whether chronic mental strain, as measured by the Undergraduate Stress Questionnaire (USQ) and Perceived Stress Scale (PSS) is related to psychological recovery from an exhaustive resistance exercise stimulus (E-RES).

BRIEF METHODS

Individuals 18-30 years of age (n = 210), both women and men, were screened online for perceptions of chronic mental strain using the Perceived Stress Scale (PSS; Cohen, Kamack, & Mermelstein, 1983). Our laboratory has existing pilot data on 357 students drawn from U.T.-Austin physical activity courses.[2] The mean PSS score for U.T. students in the first month of classes is 14.4 (SD = 5.5) whereas the mean for finals period is 17.8 (SD = 6.1). Those scoring equal to or above 19 (approximately the semester-long mean for PSS plus 1/2 standard deviation) were placed into a high stress group and those equal to or below 13 (approximately the semester long mean for PSS minus 1/2 standard deviation) were in a low stress group. These individuals completed physical fitness testing (body composition via DEXA, leg and bench press strength, squat jump power, maximal isometric force, maximal cycling power, and aerobic capacity).

[2] These pilot studies, along with the current dissertation study, were collectively known as PAART (Psycho-social Aspects of Adaptation to Resistance Training).

Seven to ten full days after fitness testing, individuals returned to my laboratory for an exhaustive resistance exercise stimulus protocol (E-RES, also known as the "workout"). E-RES is performed on a plate-loaded, 45 degree Cybex leg press with two phases of work; a ramping phase akin to a 10-RM test and a burnout phase in which multiple sets to muscular exhaustion are completed (described in greater detail later). All repetitions were performed in a six second duty-cycle with 3 seconds of eccentric contractions, 2 seconds of concentric contractions, and one second of hold (in that order). There is approximately 120-130 seconds of rest between sets with 180 seconds of rest between phases. The E-RES protocol is based on procedures from Arent, Landers, Matt, and Etnier (2005), MacIntyre et al. (1996), MacIntyre, Reid, Lyster, & McKenzie (2000), MacIntyre, Sorichter, Mair, Berg, & McKenzie (2001), Hortobagyi & Katch (1990), as well as suggestions from Baechle, Earle, & Wathen (2000). In addition, pilot testing of 28 male and female students (conducted Spring 2007-Spring 2008) found this to reliably induce delayed-onset muscle pain (DOMP). Mechanical and metabolic workload were quantified with total weight lifted (kg), total repetitions, and peak heart rate (HR_{pk}). To control for the large effect of CHO intake on recovery, post-exercise nutrition was standardized. Specifically, each individual received 0.715g of CHO per kg of body weight 60 minutes after the E-RES workout. Blood draws were completed before each visit for 36 participants with an additional blood draw 50 minutes post workout at the second visit. Thirty-one people finished all measures and were included in the present analysis.

The primary dependent variables are parameters of functional muscular and psychological recovery over 96+ hours of recovery post-workout. Exploratory analyses presented later explored these outcomes over the first hour of recovery and markers of systemic inflammatory cytokines. Functional muscular recovery was assessed through:

(1) maximal isometric force (MIF), (2) squat jump height, and (3) maximal cycling power from the Texas Power Bike. These measures were collected just before and after E-RES workout, plus 1-hr post, and 24, 48, 72, and 96 hours post. MIF was also collected at 20, 40, and 60 minutes post E-RES as part of first hour exploratory analyses. Efforts were made to collect all data at the same time of day (+/- 2 hours of the workout start time). Subjects were instructed to refrain from eating 3 hours before all testing or to fast if completing testing within the first few hours of waking. Psychological disruption during the workout was gauged by feeling (Feeling Scale), activation (Felt Arousal Scale), rating of perceived exertion (Omni RPE scale), and muscular pain (Cook Scale). Psychological recovery was assessed through self-reports of perceived physical energy, perceived physical fatigue, and soreness. The first two psychological recovery constructs were measured with Visual Analogue Scales (VAS) recently developed and validated by O'Connor (2006). Soreness was measured as a single visual analogue item inserted into the O'Connor Energy/Fatigue VAS scales.

HYPOTHESES[3]

Hypothesis 1a

In regards to functional muscular recovery, mental stress/strain (e.g. PSS score at the first visit, USQ) will **not** predict changes in maximal isometric force, squat jump height, and maximal cycling power pre- to post-workout.

Hypothesis 1b

In regards to functional muscular recovery, higher mental stain (e.g., PSS score at the first visit, mean PSS, and USQ) will be related to deeper (lower) recovery slopes and,

3 Several exploratory analyses were completed, but specific hypotheses were not formulated. See Chapter 4.

therefore, a **prolonged** return to baseline over 96 hours post E-RES workout in terms of maximal isometric force, squat jump height, and maximal cycling power.

Hypothesis 2a

In regards to psychological functioning, higher mental strain (e.g., higher PSS at time 1, higher mean PSS, and higher USQ) will be related to greater mental disruption *during* the E-RES protocol (FS, FAS, RPE, pain).

Hypothesis 2b

In regards to psychological functioning, mental strain (e.g. PSS score at the first visit, USQ) will predict decrements in perceived physical energy and perceived physical fatigue pre- to post-workout with higher strain related to greater disruptions.

Hypothesis 2c

In regards to psychological functioning, mental strain (e.g. PSS score at the first visit, USQ) will predict increases in soreness (VAS scale) immediately post E-RES workout (VAS scale).

Hypothesis 2d

In regards to psychological functioning, mental strain (e.g., PSS score at the first visit, mean PSS, and USQ) will predict recovery in fatigue and energy 1-hour post E-RES workout with higher stress scores being related to lesser recovery.

Hypothesis 2e

In regards to psychological functioning, mental strain (e.g., higher PSS at time 1, higher mean PSS, and higher USQ) will predict greater soreness (DOMP) in the 96 hour period post E-RES protocol as self-reported on the VAS inventory item.

BRIEF STATISTICAL ANALYSIS

Diagnostics were performed to determine the best scale of measurement for stress measures, whether dichotomous or continuous. Stress measures were correlated with each other, other measures of affect and fatigue, fitness, and workload during the leg press protocol. Hierarchical Linear Modeling growth curve modeling (HLM, Raudenbush & Bryk, 2002, pp. 185-186) analysis was used to examine differences in linear and/or curvilinear trajectories of recovery between low and high stress individuals. HLM has several advantages, including: a) reduced chance of committing a Type I error compared to repeated measures ANOVA, b) the ability to use all data points and not just group means, c) missing data points are less burdensome, d) exact time points where groups differ may be pinpointed by recoding for the intercept. Inserting baseline times as "Y" (the outcome) into the regression equations and solving for hours "X" (the predictor) determined return time to baseline for selected perceived stress scores. HLM tests recovery trajectories as slopes (rise over run). This is advantageous as exact recovery or healing time cannot often be precisely quantified.[4] Hypothesis 1 was tested through stepwise regression (1a) and HLM (1b). Hypothesis 2 was tested through HLM (2a and 2e by testing slopes) and stepwise regression (2b-2d).

DEFINITIONS OF TERMS

Disruption

A deviation from homeostasis resulting in impaired functioning. The first period of stress reactivity.

Homeostasis

A steady state whereas stress and strain is balanced.

[4] If healing or recovery from disruption was a discrete event, one could use Survival Analysis instead of HLM. Furthermore, if healing or recovery time were finite processes (e.g., 4.5 days long) then traditional ANOVA would work well. As definite criteria identifying a precise point of recovery are often not available, the current statistical analysis is preferred.

Reactivity

A measurable deviation from homeostasis. A period after stress initiation characterized by two phases: disruption and recovery.

Recovery

Both the process of restoration and the state in which a return to homeostasis after disruption (or the disruption phase) has been achieved.

Operational definition: Time to recovery is measured as the difference in time from the end of the E-RES workout to the "point of recovery". "Point of recovery", in turn, is defined as a final return to baseline values (without further decrement) after initial disruption in the parameter measured.

Stress (derivative: stressor)

a) In general terms, a disruption from physiological or psychological homeostasis or equilibrium and includes the impinging object, the force it exerts, and reactivity from the human organism.
b) A stimulus, such as physical exercise, or a stressor from an impinging object, or a force imposed on an individual.

   Operational definition for chronic psychological stress: Chronic life event stress is measured with the Undergraduate Stress Questionnaire (USQ).

   Operational definition for physical stress: Total workload in kilograms and repetitions (see Fry, 1999).

Strain

In general, the response to stress or the physical and mental reaction from exposure to a stressor. Effort that has to be expended to resist stress forces (Green, 2007) or the total collection of resistance forces.

Operational definition for acute physiological strain: Increase in heart rate in response to the E-RES workout.

Operational definition for chronic psychological strain: Score on the Perceived Stress Scale (PSS).

Operational definition for acute psychological strain: Rating for single-item scales (FS, FAS, REP, muscular pain) during the E-RES workout protocol.

Supercompensation

A more than expected increase in physical ability after a short-term transient period of physical incompetence (adapted from Lehman, Foster, Gastmann, Keizer & Steinacker, 1999, p. 2)

Operational definition for supercompensation: An increase in muscle function above baseline in the recovery curve.

DELIMITATIONS

Experimental design

It is not possible or ethical to induce chronic stress. Hence, this study is not a true experiment and causation is not inferred absolutely. Rather, stress was measured retrospectively over several time points and these data were related to recovery trajectories.

Sample

The population studied was a convenience sample of university physical activity students. This may not represent a limitation, however, because activity courses are required for a large proportion of students at the university and thus this population is representative of the larger student body. Additionally, a large percentage consisted of minority students.

Generalizability of workout and damage

The magnitude of sets being completed does not represent a typical workout that would be performed by an average recreational weightlifter; however, the E-RES workout generalizes to a greater degree than isokinetic dynamometry protocols utilized in muscle damage studies (e.g., MacIntyre et al., 1996, 2000, 2001; Ebbeling & Clarkson, 1989; Nosaka & Clarkson, 1996b). For instance, the apparatus utilized is a Cybex 45 degree angle leg press, which is available in many American gymnasiums.

Fitness measurement

In the effort to prevent soreness and excessive damage from fitness testing, I abstained from conducting graded treadmill tests (GXT) and tests assessing for muscular endurance (i.e., leg press 10-RM). A small pilot study conducted in our laboratory found that a leg press 1-RM test, in conjunction with 10-RM testing and a GXT, elicits high levels of soreness for many individuals. Furthermore, this reported soreness and stiffness often lasted a lengthy period of time (e.g., > 7 days). Our goal was to avoid inducing muscular damage and/or soreness from fitness testing. This would be especially problematic if muscular damage or soreness carried over into future testing, thus preventing individuals from completing the E-RES workout protocol. Even after taking this caution, one individual was unable to complete the E-RES workout due to soreness lingering from previous fitness testing. Perhaps of equal concern could be what has been termed a "repeated bout effect" (Ebbeling & Clarkson, 1989). In short, the repeated bout effect explains how a single experience of damage with or without soreness protects individuals against future damage/soreness, even within a short one to two week timeframe. In this scenario, damage and soreness would be greatly *minimized* during and after E-RES in which case recovery curves would be un-observable (i.e., there would be flat lines or 0 slope), thus diminishing the testability of our hypotheses.

Timing of measures

The primary emphasis of this dissertation was on the second recovery curve (96 hours of recovery) after a strenuous workout. Consequently, this study did not examine the full initial recovery curve (i.e., up to 8 hours). As part of exploratory analyses, post-E-RES workout observation occurred in 20-minute intervals for 1 hour at which point individuals were allowed to leave. It is possible that the initial recovery curve (i.e., about the first six hours) is most impacted by the experience of chronic stress and strain. For instance, IL-6, a cytokine implicated in the stress-adaptation relationship, is fully recovered within the first five-hour period (Dousset et al., 2007; Pedersen, Steensberg, Fischer et al., 2001). Furthermore, the 4 days of recovery tracked may represent only the intermediary phase of recovery. Indeed, some individuals do not fully recover for weeks or even months after very hard physical labor (Sayers, Clarkson, Rouzier, & Kamen, 1999).

LIMITATIONS

Generalization

Our sample did not represent the university at large in terms of ethnicity and may not represent populations outside of the university. To elaborate, the current sample was only 32.2% Caucasian (compared to 54.5% in the general student population) and there were no African-Americans in the current sample (versus 4.4% in the general student population). Asians accounted for 25.8% of the study sample (versus 15.5%), Hispanics accounted for 22.6% (versus 15.9%) and other/mixed accounted for 19.4% (versus 10.1%). Therefore, our study includes many more minorities than the overall student body with the exception of African-Americans.

Experimenter bias

The experimenter was not blind to the stress level of the individual. It is possible that knowledge of an individual's stress status may have biased the administration of the physical stressor in subtle ways (Semmer, Grebner, & Elfering, 2004).

Strain caused by the study

The study may have caused some individuals considerable stress. Grebner and colleagues (2005) and Semmer et al., (2004, p. 231) report that a number of participants found these previous studies to be very demanding and 2.4% of the stressful events they cited were related to their participation. Halson et al. (2002) report that several bouts of intensified training result in increased symptoms of mental strain. While I assessed emotional/mental strain caused by the E-RES workout, I did not assess perceived psychological strain from the study as a whole. Some participants in the current study did report duress related to the workout-induced soreness.

Quantification of physical work

Our calculations of workload are not in kilocalories or joules, as recommended by Fry (1999, p. 152, see also Dhabhar & McEwen, 2001). To calculate such as measure, one would have to know how far the sled of the leg press was pushed. I recorded the prescribed depth of the sled for each individual, which was 90 degrees at the knee joint. However, I did not measure the distance the sled traveled or the lower limb length of our participants.

CHAPTER TWO

LITERATURE REVIEW

MUSCULAR DISRUPTION AND RECOVERY IN THE CONTEXT OF EXERCISE

As mentioned previously, one must self-induce muscular damage and suffer from reductions in muscular strength in order to attain desired fitness and health benefits (Fry, 1999). Indeed, exercise is a purposeful, self-imposed stressor (Daruna, 2004, p. 217). To the novice exerciser, this is both counter-intuitive and unexpected. Even more perplexing is that some individuals experience much greater damage than others and prolonged periods of recovery (Sayers & Clarkson, 2003). Factors such as gender, age, fitness level or other physical characteristics cannot explain these variations (Ebbeling & Clarkson, 1989, Nosaka & Clarkson, 1995; Nosaka & Clarkson, 1996b) although at least one study reports that female gender is a protective factor attenuating the impact of exercise stress on muscle damage, particularly as measured by creatine kinase (Stupka et al., 2000).[5] Whereas recovery usually takes 7 to 10 days, as many as 21% of individuals exposed to an intense eccentric exercise protocol bout (50 maximal eccentric contractions) may not completely recover for over 3 weeks (Sayers & Clarkson, 2001). These researchers report that some individuals require up to almost 90 days for complete recovery. Chronic life stress and mental strain, however, is a possible individual factor that may explain additional variation in the muscle damage phenomenon.

High-intensity interval training (HIIT) is receiving more attention in the scientific literature (in terms of total publications).[6] This is reflected in the popularity of this

[5] The reasons for these possible gender differences are not clear; however, women are less fatigable than men and perhaps incur less damage during a bout of physical work (Hunter, 2009).

[6] There is some confusion concerning the definition of HIIT. A recent review (Wisløff, Ellingsen, & Kemi, 2009) on the effects of HIIT describes it purely in the realm of aerobic activities, such as running. They

exercise even among relatively untrained individuals (e.g., recreational boot camps, spinning, et cetera). HIIT is effective towards a variety of health and performance parameters (Fry, Kraemer, Ramsey, 1998; Fry, Kraemer, Van Borselen et al., 1994). It is superior to lower intensities in improving insulin resistance and improving body composition (Trapp et al., 2008). Subjects in this investigation performed 8 seconds of "all-out" exercise with 12 seconds of slow movement (Trapp et al.). This was continued for up to 60 cycles. Results showed that compared to a control and steady-state exercise group, high intensity interval exercise was the only condition of the three resulting in a decrease in total body mass, fat mass, trunk subcutaneous fat, and resting plasma insulin levels. Effectiveness has even been demonstrated with coronary heart disease patients (Warburton et al., 2005). Specifically, these individuals had a greater time to exhaustion on a treadmill test and a higher anaerobic threshold compared to traditional rehabilitation training without any greater risk. High intensity interval training is also extremely effective at increasing aerobic capacity and muscle enzyme activity. One study found an increase in citrase synthase activity of over 38% with only 24-36 minutes of total exercise time, which indicates substantial improvements to aerobic capacity (Burgomaster, Hughes, Heigenhauser, Bradwell, & Gibala, 2005). The efficacy of high intensity interval exercise has lead the ACSM and AHA (2007) to recommend as little as 3 sessions of 20 minutes of high intensity exercise (activity > 6.0 METS), which causes heavy breathing and a much higher heart rate. This is indicated as a viable alternative to slower endurance training for the general population.

The long-term implications of using high intensity interval training are complex and largely unexplored. Gibala and McGee (2008) suggest that high-intensity exercise may be more conducive of long-term exercise adherence. This may be due to the fact that

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say, "high intensity aerobic interval training refers to walking or running intensity at bouts of 85%-90% of peak oxygen uptake or 90%-95% of peak heart rate separated by 2-3 minutes of active recovery…"

high-intensity exercise is extremely time efficient (Coyle, 2005). For instance, Burgomaster et al. (2005) reported substantial benefits with only 4 to 6 minutes of extremely intense cycling exercise. This is important as "lack of time" is the most common barrier for exercise for most individuals (Godin, Desharnais, Valois, Lepage, Jobin, & Bradet, 1994). A cautionary note, however, must be exerted because the psychological consequences of high-intensity exercise over the intermediate-term (e.g., soreness and fatigue over days and weeks post initiation of exercise) are unknown. Tolerance of this type of exercise is likely highly variable between individuals. In essence, there appears to be a trade-off between efficiency of time and training-related dysphoria for this training paradigm, at least at training initiation. High-intensity exercise may serve to attenuate perceptions of barriers from time constraints, but it may also deter exercise due to intolerance of painful sensations (Hall, Ekkekakis, & Petruzzello, 2002).[7]

AFFECTIVE RESPONSES TO RESISTANCE EXERCISE: THE GOOD, BAD, AND UGLY

Acute Responses

The affective response to resistance training is influenced by several factors. These include: exercise intensity, workload (total work, as quantified by total repetitions), rest time between sets, type of contraction (e.g., eccentric, concentric, isometric) and speed of contraction. To date, the effects of temporary increases in workload (overreaching) are the most well known (e.g., Coutts, Wallace, & Slattery, 2007; O'Connor et al., 1991; Rietjens et al., 2005) with a small number of studies devoted to the effects of exercise intensity. In short, high intensity resistance training is associated with decrements in mood, anxiety and affect/emotion (Arent et al., 2005; Bartholomew &

[7] A further concern stems from inappropriate implementation of high-intensity programs in the community. Whereas experimenters have been careful to execute these programs in a progressive manner (Trapp et al., 2008 started individuals at just five minutes of exercise and increased gradually to 20 minutes over 15 weeks), this conscientiousness may not translate in application and individuals may suffer from excessive soreness or injury.

Linder, 1998; Raglin, Turner, & Eksten, 1993) while moderate intensity resistance exercise has a salutary effect on mood. Arent et al. (2005) reports a curvilinear relationship of exercise intensity with affective responses. High intensity exercise resulted in increased negative affect (via PANAS), anxiety, and tense arousal.

Pain, Soreness and Long-Term Responses

According to the International Association for the Study of Pain (1979, p. 249), pain is "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms with such damage." It is described by Cook (2006, p. 203) as a subjective experience arising from stimulation of the nocioceptors and transmission to the brain via afferent nerves. Chronic musculo-skeletal pain is a large barrier to both initiation of and adherence to exercise behavior in a variety of populations, including older adults, the infirm and injured, and injured athletes (Cohen-Mansfield, Marx, & Guralnik, 2003; Ditor et al., 2003; Mori et al., 2006; Patil, Johnson, & Lichtenberg, 2008; Pen & Fisher, 1994). One study (Ditor et al., 2003) found that 83% of the variability in exercise adherence at a 3-month, post-intervention follow-up was due to intensity of pain. Furthermore, while not explicitly speaking of pain, it has been suggested that individuals may want to participate in low-intensity exercise to maximize enjoyment and decrease aversion to physical activity (Pate et al., 1995).

Pain during exercise

Repeated and fatiguing muscular contractions often are associated with musculoskeletal pain. Muscular pain appears to increase quadratically with increased intensity of exercise (Cook, 2006, p. 209). During exercise, muscle pain is described as diffuse, sharp, intense, exhausting, and burning. Leg pain is not associated with RPE in some studies (Cook, O'Connor, Oliver, & Lee, 1997), but contradictory evidence has been reported (Hollander et al., 2003). Pain also does not appear to be the main reason

individuals discontinue an acute round of exercise (contradicting the notion that pain is the primary inhibitor of exercise, hence the often-used aphorism “no pain, no gain”). Rather, fatigue has a more prominent effect (Cook et al., 1997).

Pain after exercise

Unfortunately, post-workout affective responses to resistance training, particularly delayed-onset muscle pain (DOMP, formerly known as DOMS), are not well understood and have not been systematically investigated. DOMP has not received much attention in the extant exercise pyschobiology literature (Acevedo & Ekkekakis, 2006; for an exception see O’Connor et al., 1991) and was conspicuously excluded from a recent review on pain and physical activity (Cook, 2006). The extant literature on muscle damage, however, does provide a glimpse into the fundamentals of the DOMP phenomenon.

The sensation of DOMP, usually classified as a type of pain, is characterized by minor tenderness or stiffness to a dull, aching pain to severe and debilitating pain. It is likely evolutionarily conserved because soreness promotes less strenuous physical activity; thus, the healing process is strengthened (Sapolsky, 2004, p. 191). Recovery from muscular pain appears to be of a bimodal pattern. There is a dramatic decrease in pain immediately after exercise (Cook et al., 1997), which is thought to result from a lack of input from the local musculature. Shortly thereafter, a noticeable increase in pain (or DOMP, often described as soreness or stiffness) occurs. This is followed by a subsequent decrease in pain along with a gradual clearance of noxious chemicals (i.e., Bradykinin). Clarkson and Hubal (2002) report that exercises such as downhill running and isokinetic leg extension produce smaller ratings of DOMP (in the 4-5 range, 1 being no soreness and 10 being very sore) than eccentric exercise of the elbow flexors (7-8 range). During inflammation of an injured area, chemicals are released that make pain receptors more

sensitive. One key biochemical associated with pain sensation is Substance P. This chemical, interestingly, also shows a delayed recovery curve (Dousset et al., 2007).

DOMP does not correlate well with other indices of muscular damage, such as creatine kinase (Clarkson & Hubal, 2002); however, there appears to be a lagged relationship between DOMP and inflammatory cytokines. In one study DOMP at 24 and 96 hours post-workout significantly correlated with interleukin-6 (IL-6; Miles et al., 2008). This relationship may indicate that inflammation-related chemicals or cells may cause increased pressure on the afferent nerves (via swelling or edema) in the effected or damaged area. These observations agree with evidence that soreness reaches its maximum well before swelling of the extremity (Rodenburg et al., 1994 as cited in Clarkson & Hubal, 2002). A further concern is the fact that sensations of pain subside well before muscular function is completely recovered.

Repeated bout effect

Acute physiological and sensory reactions to muscular damage are altered (up to 12 months) by rapid adaptations to a single experience of physical stress, called the repeated bout effect. Physical labor associated with an experience of DOMP may provide protection or resiliency against subsequent damaging exercise (Ebbeling & Clarkson, 1989). Pain, as with muscle markers of creatine kinase (CK), shows a repeated bout effect whereas a single session of eccentric training will protect against further pain in subsequent sessions (Dannecker, O'Connor, Atchison, & Robinson, 2005).

Methodological Problems with Exercise-Affect Research: Using Resistance Training Paradigms

Much of this research has been plagued with methodological problems, particularly, quantification of training intensity and volume. Typically, training intensity has been identified as a percentage of 1-repetition maximum (1-RM; Kraemer &

Ratamess, 2004) but methods of quantifying intensity have varied widely (Bartholomew & Linder, 1998; Focht & Koltyn, 1999; Focht, 2002; Koltyn, Raglin, O'Connor, & Morgan, 1995 as cited in Arent et al., 2005). Researchers fail to report if individuals reach subjective/volitional and objective failure (i.e., unable to complete another repetition without assistance), which may be important as it represents extreme lack of motivation, spikes in distress, and/or total physiological failure (Tuson, Sinyor, & Pelletier, 1995). Researchers also fail to report muscle contraction speed (e.g., 2 seconds pushing a load, 3 seconds relaxing it) which represent labor from concentric versus eccentric muscle actions. Protocols with greater eccentric motion are pervasive in the muscle damage and DOMP literature. Eccentric muscle actions produce greater force but require less energy per unit of muscle force. Consequently, this type of contraction results in lower RPE than concentric actions (Hamlin & Quigley, 2001; Hollander et al., 2003; Komi, Kaneko, & Aura, 1987). On the other hand, eccentric contractions result in greater levels of muscle damage and also greater perceptions of soreness in the period following exercise. Indeed, eccentric contractions propel gains in strength and hypertrophy.

Rest periods constitute an important part of the physical stress and mental strain relationship. Kraemer (1997) reports that with 3 minutes of rest, a relatively long rest-period, individuals are able to perform 100% of their 10-RM for 10 repetitions. While longer rest periods are often utilized in trials to maximize muscular strength, rest periods this long are rarely utilized in muscle damage research (see Vaile, Gill, & Blazevich, 2007, for an exception). Bodybuilders and those seeking enhanced muscular hypertrophy often utilize short rest periods. Exercise protocols with short rest periods (< 1 minute to 90 seconds) are associated with greater anxiety, discomfort, and mental strain and thus demonstrate the greatest increases in negative affect and diminished mood (Arent et al., 2005; Tharion, Rausch, Harman, & Kraemer, 1991). With only 1 minute of rest,

individuals are also less able to sustain a high level of absolute work. Kraemer found that, on average, those using 1-minute rest periods are only able to perform 10, 8, and 7 reps at their 10-RM over 3 sets. This may be due to greater physiological demand and resulting muscle fatigue. Short rest periods are associated with greatly elevated blood lactate concentrations and increased muscle damage (Fleck & Kraemer, 2004, p. 165). Some researchers have utilized super-short rest periods (~20-30 seconds) with very high volume of work to induce muscular damage (MacIntyre, 1996, 2000, 2001).

Workload (e.g., repetitions completed, joules of work) is also an important construct that is often not reported or simply overlooked (Fry, 1999). Frequently, researchers have commonly failed to standardize workload or fail to report the amount of work accomplished. The number of sets needed to elicit adaptations is highly variable and is secondary to the consideration of total repetitions (Fleck and Kraemer, 2004). Reportedly, nine sets are needed for any muscle group to maximize metabolic and hormonal adaptations. This is based on findings that a minimum of 3 sets and 3 different exercises are needed to maximize hypertrophy (Baechle et al., 2000). Researchers have typically used a 10-RM load (the amount of weight one is able to lift exactly 10 times). 10-RM protocols are longer overall, include more repetitions and longer time-under-tension (Fleck & Kraemer, 2004, p. 165). Therefore, a 10-RM protocol to muscular failure (exhaustion) or near-failure allows for high intensity (75-85% of 1-RM) and high lactate responses. Increasing the contraction time (in other words, decreasing the contraction speed) of the eccentric phase of movement results in an additional muscular strain and may result in faster adaptation (Fleck & Kraemer, 2004, pp. 170-171).

The effect of training experience or fitness, the muscle group strained, and gender are other important considerations, particularly since the current study included individuals that varied in each of these characteristics. In a series of classic studies,

Hoeger, Barette, Hale, and Hopkins (1987) and Hoeger, Hopkins, Barette, and Hale (1990) determined the relationship between intensity and repetitions-to-failure on a Universal leg press machine (starting position, 100 degree angle) depended on muscle group strained, gender, and training status. See Table 2.1. When working out the lower body, one needs a higher intensity to stay within an RM zone, apparently because of the greater muscle mass utilized (Fleck & Kraemer, 2004, p. 168). 10-RM for the leg press, therefore, necessitates much higher intensities than other exercises. Fleck, Kraemer et al. (2004) found that power lifters could lift 80% of their 1-RM for 22 repetitions. Untrained individuals in this study could perform only 12 repetitions at 80% of their 1-RM. These considerations have great consequence for research in the area of exercise-related affect.

Table 2.1 Leg press strength and muscular endurance in trained and untrained men and Women. Adapted from Hoeger et al. (1987, 1990).

	N	1-RM (kg)	Reps to Failure: 60% of 1-RM	Reps to Failure: 80% of 1-RM
Untrained Males	38	137.9 +/- 27.2	33.9 +/- 14.2	15.2 +/- 6.5
Trained Males	25	167.2 +/- 43.2	45.5 +/- 23.5	19.4 +/- 9.0
Untrained Females	40	85.3 +/- 16.6	38.0 +/- 19.2	11.9 +/- 7.0
Trained Females	26	107.5 +/- 16	57.3 +/- 27.9	22.4 +/- 10.7

THE NATURE OF PHYSIOLOGICAL BREAKDOWN AND RECOVERY FROM EXERCISE

Stress and Strain Processes

Muscular breakdown in otherwise healthy populations is typically the consequence of physical overload (stress). Usually, breakdown is followed by recovery and a higher level of functioning. Hence, stress or overload provides the mechanism by which an individual becomes more adapted to one's environment. How does one break down, however, without becoming "broken"? This is where an understanding of strain

and what Selye (1956) calls “resistance” is most important (see below for an exposition of his General Adaptation Syndrome, or GAS, theory). Strain is the total set of resistive forces that counteracts stressful impinging forces and thus prevents collapse of the stressed tissue. At first, a stressor of great enough magnitude will cause disruption to a system, primarily because resistive forces have not been activated or modified to meet the new demand. When resistance forces meet demand, a steady state occurs (a new homeostasis).[8] Recovery is when resistive forces have been sufficiently activated to slow disruption and finally cause a return to homeostasis. Both disruption and recovery combined describe the larger phenomenon of “stress reactivity”. While the topic of interest is recovery, this process is only observed as part of a larger process of reactivity. One sees that the stress concept is a dynamic and complex set of processes.

Understanding these processes is facilitated by examining the response of the human organism to a single stressor, such as a cardiovascular stress test. Disruption begins once stressful impinging forces of severe enough strength begin to act on an organism and therefore perturb homeostasis. However, this perturbation is counterbalanced almost immediately by resistive forces designed to regain homeostasis, such as increased heart rate. This is the process of resistance.

The final outcome from stress depends on the ability to enact resistive forces. This capability is sometimes called fitness. In other words, a given stress and strain response may result in differential degrees of adaptation or maladaptation depending on the fitness or capacity of the individual and the associated vulnerability to disruption. Figure 2.1 demonstrates recovery trajectories for individuals who all experience the same relative

[8] The VO2max protocol I used, the Storer protocol, prescribes increases in wattage every minute. Therefore, heart rate, or physical strain, is never allowed to enter a steady state. This is a general criticism of this protocol.

magnitude of disruption.[9] In cases where stress is suddenly released, one is able to adequately rest, and resistance forces continue to exert an effect (such as when an athlete tapers for competition), one may observe super-compensation for a limited period of time or until homeostasis is again reached.

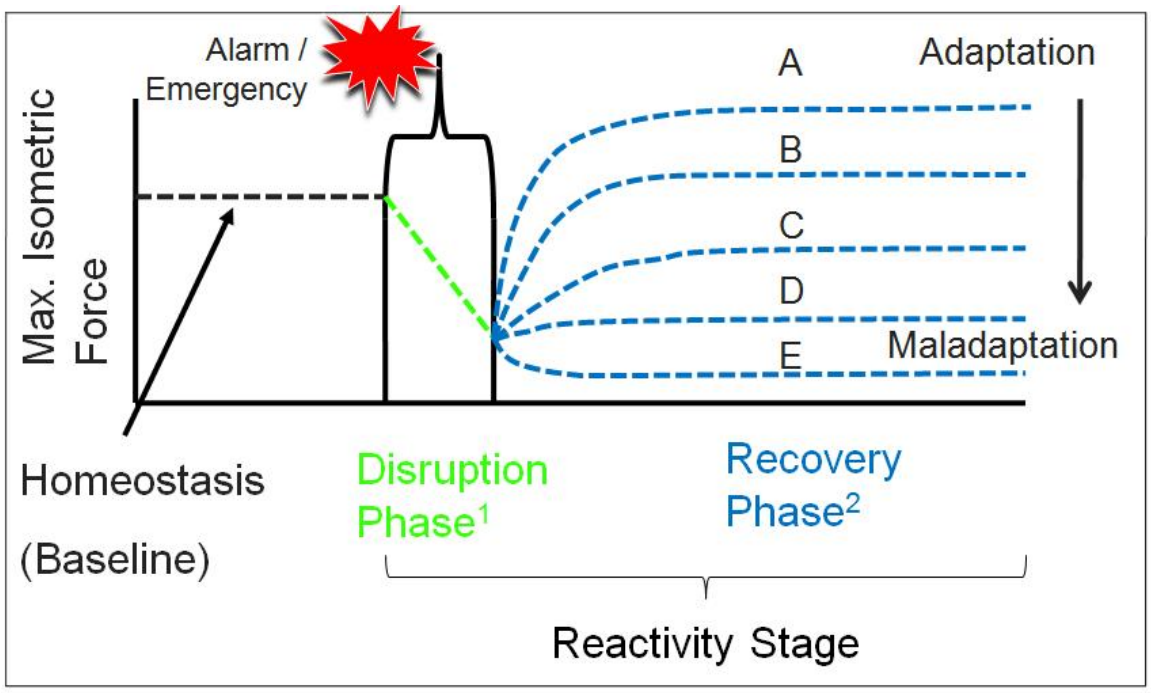


Figure 2.1 Parameters describing disruption of physiological homeostasis and functioning with various recovery curves. A. Thriving, B. Return to baseline functioning, C. Adaptation to a level just below baseline functioning, D. Significant decrease or impairment in functioning following disruption. E. Extreme maladapation or death. Adapted from Carver, 1998.

1. Disruption varies in magnitude (amount of decrease in functioning) and duration [in this diagram, all individuals experience the exact same disruption in function].

[9] For any stressor of absolute magnitude individuals will experience a large variation in the amount of initial disruption to this acute event, thus creating an infinite number of possible recovery curves. This variation, however, is often proportional to the strain experienced, which in turn is determined by the fitness or capacity of the organism. Individuals able to enact a large resistive response may experience no disruption whatsoever.

2. Recovery varies in: a.) point of initiation, b.) length of recovery period, and c.) final recovery status (adaptation).

Force regeneration (again, a functional measure) after maximal exercise shows a bi-modal (or biphasic) recovery curve (Dousset et al., 2007; Malm, Lenkei, & Sjodin, 1999; MacIntyre et al.,1996; see Figure 2.2). MacIntyre et al. (1996) was one of the first to demonstrate this pattern. After 300 eccentric contractions, a group of ten young women had bi-modal reductions in eccentric torque (MacIntyre et al., 2001). Dousset et al. (2007) has replicated this result with young men performing hack squat-type, short-cycling contraction movements.

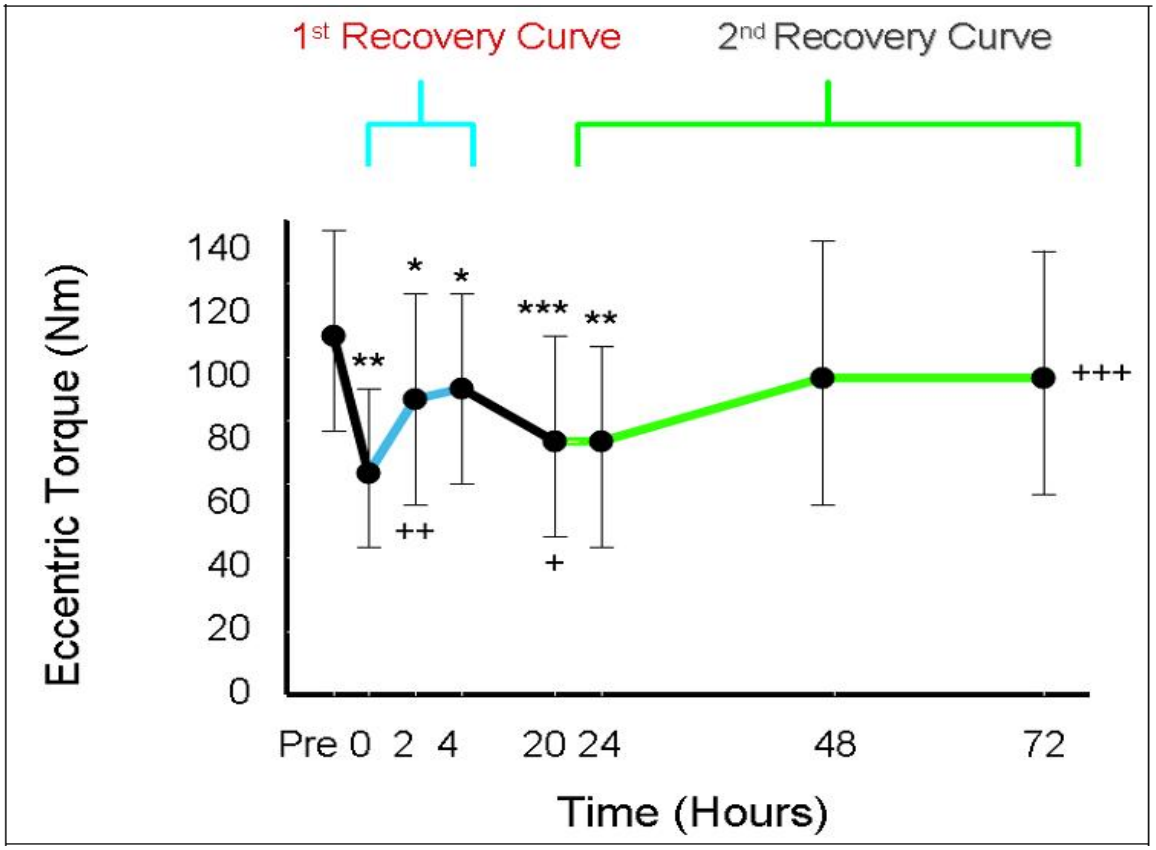


Figure 2.2 Biphasic or bimodal recovery curve as seen in MacIntyre et al. (1996). Eccentric torque values are displayed as means + SD. Pre, pretest.

*Significantly different from Pre, $P < 0.05$. + Significantly different from pretest, $P < 0.01$. **Significantly different from pretest, $P < 0.001$. ++ Significantly different from preceding test, $P < 0.05$. ***Significantly different from preceding test, $P < 0.01$. +++ Significant differences over time, $P < 0.001$. See also the bimodal recovery curve in Dousset et al., 2007.

Bimodal decrements in function, such as decreases in power and strength, are likely related to energy resource depletion partnered with structural breakdown (Figure 2.3). The systems involved are loosely coupled and roughly equivalent systems with the greatest disruption to these systems following different time courses.[10]

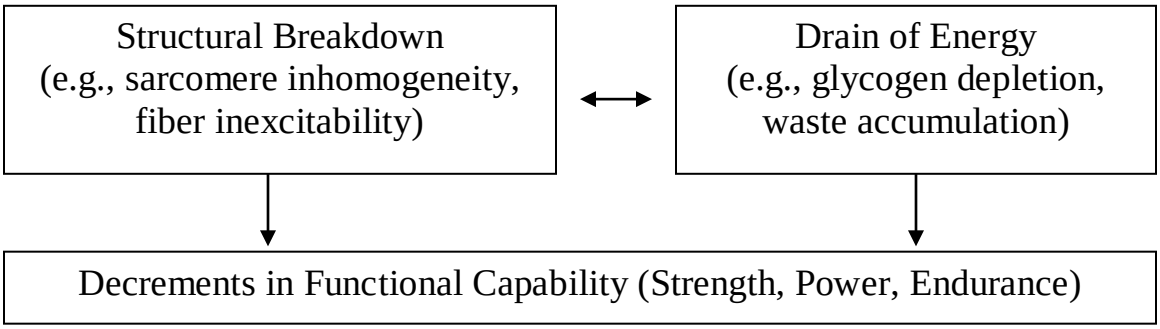


Figure 2.3 Functional muscle capability is affected by energy availability and structural capacity.

Armstrong (1990) describes how these factors are related in his 4-stage healing process (Armstrong). The stages are 1) initial, 2) autogenetic, 3) phagocytic, and 4) regenerative. The initial stage includes the maximal exercise or injury that triggers further physiological responses.[11] Immediately after eccentric exercise there is a large concomitant decrease in functional and performance measures (Garcia-Lopez et al., 2006; Mair et al., 1995; Skurvydas, Dudoniene, Kalvenas, & Zuoza, 2002). Garcia-Lopez et al. (2006) utilizing a protocol using 120 eccentric actions of the knee extensors found large decreases in vertical jump height in the magnitude of 28%, 29%, and 34% immediately afterwards, 6 hours later and 24 hours later, respectively.

[10] Semmer, McGrath & Beehr (2005, pp. 11-12) note important distinctions between energetic versus structural stress and resources.

[11] Whereas Armstrong (1990) does not imply that restoration of energy systems is reflective of initial strength regeneration, it is the belief of several researchers that these gains within the first few hours are due to energy system recovery.

The autogenetic stage, lasting up until 6-8 hours post-exercise, is characterized by initial increases in energy system waste products (e.g., lactate). IL-6 peaks somewhere between 2 hours (Dousset et al., 2007) and 8 hours (Miles et al., 2008) after the termination of the physical stressor. This stage typically is also when cellular degradation of muscle tissues begins.

The phagocytic stage starts 4-6 hours after injury and is when phacocytic cells invade the injured area to assist in clearing protein debris. This stage marks the start of major inflammatory processes along with symptoms of concomitant damage (e.g., burning, swelling, redness). The regenerative stage is in its fullness about 4-6 days after the injury when evidence of the injury starts to greatly diminish. It may last up to 10-14 days. Dousset et al., (2007) reports that these later phases of recovery are characterized by high levels of Substance P (the protein associated with pain sensation) and C-Reactive Protein (CRP; a marker of systemic inflammation associated with heart disease). It is important to note that success in the early stages of recovery predicts success in later stages of recovery.

Mechanical Breakdown

Armstrong (1990) suggests that irreversible damage may primarily occur to muscle fibers that are "stress susceptible".[12] These fibers may be fragile and in a state of decline already and thus are cleaved with maximal eccentric exercise. Evidence from Foley, Jayaraman, and Prior (1999) corroborates this hypothesis. These authors assert that stress-susceptibility develops over time and with disuse. In other words, disused fibers become vulnerable to mechanical strain. Clarkson and Hubal (2002, p. S64) revised this theory by claiming that only parts of these fibers are vulnerable and not the entire fiber. Otherwise, massive amounts of muscle would be lost over a long period of time.

[12] Several reviews of muscle damage and repair have been published within the last year. See Jarvinen, Kaariainen, Aarimaa, Jarvinen, and Kalimo (2008) and Tiidus (2008).

Disruption to the surface membrane (sarcolema), sarcoplasmic reticulum, t-tubules and remainder of the cytoskeleton, as mentioned in the introduction, has also been observed (Clarkson & Sayers, 1999). High muscle forces degrade the architecture of the individual sacromere as the muscle undergoes proteolysis (Allen, 2001). Sarcomere inhomogeneity and fiber over-stretching is clearly observable under the microscope as a total loss of z-line synchronization. The concomitant influx of intracellular calcium (Gissel & Clausen, 2001) and reduction in calcium release also results in changes in excitation-contraction coupling and thus a myofiber that is unexcitable and unable to generate force (e.g., isometric muscle force). Destruction of proteins and failure of filaments to reinterdigitate in itself results in function loss (Allen). Inflammation in the hours afterwards (see below) contributes to loss but Clarkson and Hubal (2002) note that specific mechanisms are still not clearly understood.

Inflammation

Chargé & Rudnicki (2004) assert that the inflammation response demarks the beginning of the adaptation process after a strenuous physical challenge. Indeed, healing cannot occur without inflammation (Hart, 2002). Current evidence suggests that this process evolves rapidly, often within minutes (MacIntyre, Reid, & McKenzie, 1995). The primary positive outcome of inflammation is the repair of injured tissue (Tidball, 1995). It is characterized by rapid increase in local blood flow and vascular permeability (MacIntyre et al., 2001, p. 181). Muscle injury results in an activation of mononucleated cells (inflammatory cells within the cells and in the bloodstream and myogenic cells). Neutrophils (a type of leukocyte) are the first to appear (approximately 1-6 hours afterwards). Neutrophils migrate towards chemoattractants, such as cytokines, which accumulate at the site of injury for 6-12 hours (Walker & Fantone, 1993). Neutrophils and monocytes may induce more muscle damage as they enter the muscle fiber (likewise,

with repeated training, there is less entrance of these cells into the fiber and perhaps less damage, see Pizza et al., 1996 and Clarkson & Hubal, 2002, p. S64). IL-1β and tumor necrosis factor alpha (TNF-α) are released by resident macrophages (activated monocytes) at the site of injury (Tidball, 1995; Smith et al., 2000) and initiate the inflammatory cytokine response. They also stimulate the release of IL-6, usually via the local endothelium. IL-6 stimulates satellite cell proliferation, helps to recruit inflammatory proteins and enzymes, and also inhibits production of IL-1β and TNF-α (Miles et al., 2008). Macrophages appear about 48 hours post-exercise to breakdown (phagocytose) cellular debris and activate the myogenic cells. Both neutrophils and macrophages are phagocytes, which release oxygen radicals and proteases and potentially contribute to further damage to the injured muscle tissue (Roitt, 1991; MacIntrye et al., 2001). Myogenic cells later provide the new myonuclei for muscle regeneration. The inflammation process is nearly the same for wound healing and for muscular damage due to maximal exercise (Clarkson, 2007; Daruna, 2004, p. 217).[13]

Cytokines are regulatory proteins secreted by white blood cells and a variety of other cells and are intercellular signals of inflammation (Cannon & St. Pierre, 1998; MacIntyre et al., 1996; Pedersen, Rohde, & Ostrowski, 1998, p. 327). They are messenger molecules that aid in directing inflammatory-related events (Dinarello, 1997; Smith et al., 2000) and may be both anti-inflammatory and pro-inflammatory (such as IL-6; Miles, 2008). They have low molecular weight and evoke activities after binding to a receptor on a responsive target cell (Pedersen et al., 1998, p. 327). When endothelial cells are stimulated by the acute inflammatory response, one of the responses is the release of cytokines. Cytokines are largely divided into four groups based on their functions: a) pro-inflammatory (e.g., IL-1β, TNF-α) which are released from macrophage/monocyte

[13] See Table 2.2 for a comparison of wound healing and muscle damage healing.

lineage cells (non-specific, innate immunity), b) T-Helper-1 (Th1) cytokines (IL-2, IFN-gamma), c) Anti-Inflammatory T-Helper-2 (Th-2) cytokines (e.g., IL-4, IL-10), and d) Transforming Growth Factor family (TGF)- β which are largely immuno-suppressive. Cytokines are also divided into several general families: a) interleukins, b) tumor necrosis factors, c) interferons, d) growth factors, e) colony stimulating factors (CSF) and f) cell adhesion molecules (CAM). IL-6 is the cytokine most consistently related to stress and strain processes, including muscular trauma (Biffl, Moore, Moore, & Peterson, 1996; Curfs, Meis, & Hoogkamp-Korstanje, 1997; Smith et al., 2000) and intense and/or high volume exercise (Chargé & Rudnicki, 2004; DeRijk et al., 1997; Dousset et al., 2007; Ostrowski et al., 1999; Pedersen et al., 1998; Smith et al., 2000).

Chronic Breakdown with Lack of Recovery

Dysregulation of pro-inflammatory cytokines and other mechanisms of chronic inflammation are strongly implicated in hyper-catabolism and physical deterioration (Smith, 2000, see Figure 2.4). Stress and strain-related increases in cortisol may be the by-product of inflammation working on the pituitary gland (Smith, 2000, p 327). IL-6 receptors on the adrenal cortex have been identified and IL-6 is thought to cause release of stress hormones and androgens (Path, Borstein, Ehrhart-Borstein, & Scherbaum, 1997). Increases in TNF-α associated with muscle damage lead to inhibition of PKB (also known as AKT) signaling, which is detrimental to reactions along the AKT-mTor pathway, which contributes strongly to muscle growth. Normally, chronic exercise inhibits TNF-α in muscle thus promoting a more anabolic milieu (Spiering, 2008). The enduring consequences of hyper-catabolism include a failure of musculature to properly adapt to training even when the training stimulus is strong.

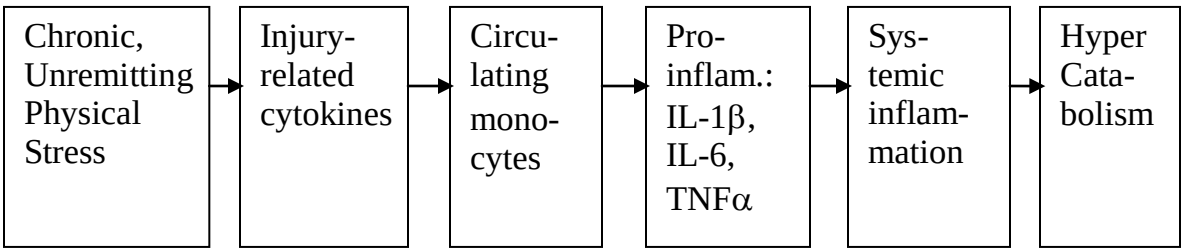


Figure 2.4 Model of chronic, unremitting physical stress and excessive cortisol release. Diagram simplified from Smith's (2000) Cytokine Hypothesis of Overtraining

SIMILARITY OF MUSCLE RECOVERY TO OTHER RECOVERY PROCESSES

Processes of Regeneration

The recovery process is remarkably similar regardless of the type of damage or the tissue involved. Wound healing of superficial tissue may be more complicated as these processes require several more actions than muscle regeneration to ensure proper repair (e.g., neutrophil accumulation to prevent infection and re-epithelialization to cover the wound). Nevertheless, the mechanisms involved, such as cytokines and growth factors, are quite similar (Chargé & Rudnicki, 2004; Christian et al., 2007; P. M. Clarkson, personal communication, November 17, 2007; Labarge & Blau, 2002; Werner & Grose, 2003). See Table 2.2 below for a comparison of wound healing and muscle repair.

Table 2.2 Comparison of wound-healing and muscle regeneration. These processes share common immunological mechanisms and the employment of adult stem cells for repair (see Chargé & Rudnicki, 2004; Christian et al., 2007; Labarge & Blau, 2002 & Werner & Grose, 2003). Besides obvious differences between muscle tissue and the epidermis, there are several other notable differences.

Similarities	Dissimilarities
Contribution of growth factors: Fibroblast growth factor (FGF) and transforming growth factor (TGF-ß), see Werner & Grose, 2003	Magnitude of response: Muscle disruption due to exercise has a larger systemic response (as opposed to local)
Contribution of cytokines: IL-6, IL-1α and cytokines of other families involved.	Time frame: Epidermis repair may take months or years (e.g., scars)
Macrophage activation: Limits excessive tissue disruption, phagocytose cellular debris, and continue repair (e.g., activate myogenic cells in muscle)	Contamination: Disruptions to the skin tissue must also initiate neutrophil accumulation to prevent contamination by bacteria and infection.
Utilization of adult stem cells: In muscle regeneration, to replenish myogenic and satellite cells.*	Scabbing: Muscle repair is also simpler than epidermis repair in that re-epithelialization to cover the open wound does not need to occur.
	Characteristics of tissue: Muscle is more stable as a tissue, myonuclei in rodents turn over only 1-2% at most per week, see Chargé & Rudnicki, 2004.

*After injury, bone marrow-derived and muscle-derived stem cells contribute to new myofibers and replenishment of the satellite cell pool (LaBarge & Blau, 2002).

Wound Healing Studies of Recovery. A Decade of Research (1995-2007)

In contrast to exercise-induced muscle damage, there is a growing body of literature on the potential effects of psychological stress on wound healing. The tissue healing process, though highly structured, is impacted by the experience of chronic mental strain. Specifically, mental strain may delay the recovery (or regeneration) phase, thus impairing or attenuating physical adaptations. A series of wound healing studies demonstrate that stress impacts the ability to recover from a variety of naturalistic and induced physical injury, such as superficial wounds to the epidermis. This effect has been replicated across a range of psychological stressors including: care given to Alzheimer's patients (Keicolt-Glaser et al., 1995; Kiecolt-Glaser et al., 1996), patients undergoing surgery (Broadbent et al., 2003), couples experiencing marital hostility (Kiecolt-Glaser et al., 2005), and students experiencing examination stress (Marucha et al., 1998). In each of these cases, healing was slower in individuals with higher experience of and/or perceptions of stress and mental strain. Burns (2006, p. 233) interpreted the last study cited as evidence that "even mild transient stressors may have clinically relevant implications for the rate healing of wounds."

Likewise, these deleterious effects have been replicated across a range of tissue damage, with an amazing degree of consistency in magnitude of statistical effect. Mental strain impairs mucosal (Marucha et al., 1998), oral (Ebrecht et al., 2004; Wikesjo et al., 1992), and dermal wound healing. The effect is similar for standardized punch biopsies (Kiecolt-Glaser & Glaser, 1995; Marucha et al., 1998), suction blister wounds (Glaser et al., 1999; Roy et al., 2005), and skin barrier tape-stripping (Altemus et al., 2001). Across these studies, stress-related time delays in healing range from 24-40% and the effect sizes (cited by the authors as squared correlation as a proportion of variance explained by stress) are between $r^2 = 0.30$ and $r^2 = 0.74$. See Figure 2.5.

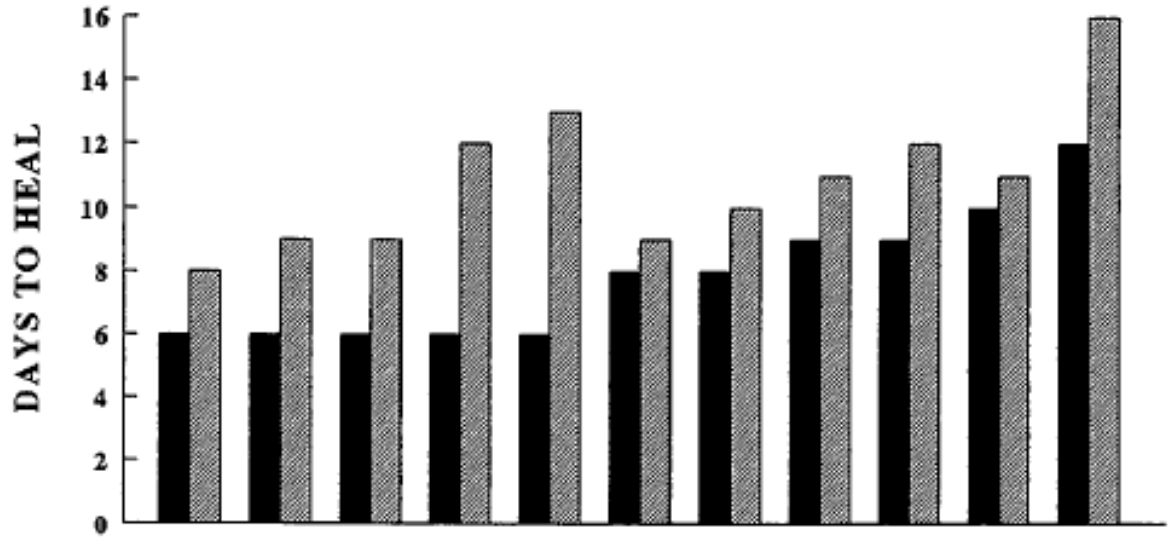


Figure 2.5 Healing time for 11 individuals at summer vacation (low stress, black bars) and at examination time (high stress, gray bars). Note that all 11 individuals had longer or delayed recovery during the high stress period. Chart from Marucha et al., 1998.

Evidence from stress interventions also supports the stress-recovery hypothesis for systemic recovery. Glaser et al. (1999) notes, "Over 200 studies spanning three decades have demonstrated that programs aiming to buffer psychological stress result in reductions of hospital stays, fewer post-operative complications, better treatment compliance, less pain, less use of analgesics, and improvements in numerous physiological variables" (see also Kiecolt-Glaser, Page, Marucha, MacCallum, & Glaser, 1998). Glaser et al. (1999) suggests that the stress-related disruption has significant consequences in both the statistical and clinical sense. It should be noted, however, that at

least one study has not found a relationship between stress and speed of recovery (Ievleva & Orlick, 1991).

At this time point, there is a dearth of direct evidence to fully authenticate the notion that chronic mental stress and strain impacts adaptation from training (see Perna & McDowell, 1995 for an exception, described below). Nevertheless, in light of the wound healing studies, it is reasonable to posit that psychological stress and mental strain may slow recovery from exercise-induced muscle damage, thus weakening adaptation. This is true to the extent that it is reasonable to view exercise-induced muscle damage as an induced wound. Recovery from exhaustive exercise follows a structured process involving physiological mechanisms (e.g., cytokines) similar to those implicated in the wound healing studies (Clarkson, 2007; Daruna, 2004, p. 217). Inspecting the nature of these mechanisms, along with other key stress agents such as glucocorticoids, may provide clues and help to identify the most important factors in regards to stress and strain processes. Clow and Hucklebridge (2001), in their wide-reaching review, attempt to consolidate these diverse lines of research to answer the question at hand. They conclude (along with Burns, 2006) that there is no available published evidence to support that neither chronic stress nor strain modify responses to physical (e.g., training) stimulation. Nevertheless, these researchers purport that the literature in immunology, physiology, and sport and exercise psychology provide important linkages substantiating the proposed relationship between mental stress and impaired recovery. What follows is an inspection of two of the most important physiological mechanisms, cytokines and glucocorticoids.

SPECIFIC MECHANISMS OF THE STRESS AND HEALING RELATIONSHIP

Inflammatory Cytokines

The mechanisms that modulate the perceived stress and healing relationship are unclear at this time, but the crucial link may be through inflammation and processes at the sub-cellular level (Clow & Hucklebridge, 2001; Christian et al., 2007). Essentially, chronic mental strain may impair inflammatory processes important for adaptation after a physical challenge. Chronic stress and strain appears to result in a shift from Th1 (IL-2) to Th2 (anti-inflammatory IL-4, IL-10) responses (Kim & Maes, 2003). The response of pro-inflammatory cytokines to mental strain, however, is equivocal. Kim and Maes (2003) report that chronic stress results in an up-regulation of pro-inflammatory cytokines. Likewise, their review suggests IL-6 (again, a cytokine with inhibitory and pro-inflammatory properties) is consistently affected by acute stress in humans. However, several wound-healing studies demonstrate a down-regulation of pro-inflammatory cytokines. Ideally, pro-inflammatory cytokines would be quickly released when necessary, followed by a rapid quiescence. Release of these substances is adversely delayed early after an induced wound in strained individuals (Glaser et al., 1999). Roy et al. (2005) found that chronic perceived stress suppresses neutrophil transcriptome at the level of DNA, also attenuating beneficial adaptations.

Cytokines, the main messengers in the inflammation process, are known to have an influence in mood and depression (Maes, 1995 as cited in Smith, 2000; Maes et al., 1993; Maier & Watkins, 1998, as cited in Smith, 2000). In accordance, injection of exogenous cytokines results in disruptions in mood (Dinarello, 1997 as ctied in Smith, 2000). Cytokines act on the brain to produce sickness symptoms and behavior, apparently to force individuals to rest and conserve energy after infection and other physical challenge (Besedovsky, Del Rey, & Sorkin, 1983; Clow & Hucklebridge, 2001, p. 10).

In a review of the literature on the effects of acute stress on cytokines, Steptoe, Hamer, & Chida (2007) found that psychological stress had a substantial effect on IL-1β (a pro-inflammatory cytokine) and IL-6 as well as a marginal effect on C-Reactive Protein (CRP, an indicator of systemic inflammation). This review, however, was limited to acute laboratory stressors such as the Stroop Color-Word Test, cold pressor test, and the Trier Social Anxiety Test and may not be consistent with more naturalistic stressors. Another limitation is that most studies included in their review studied recovery from stress in a very limited time period (up to several hours post-manipulation) even though biomarkers like CRP may not peak for over 24 hours. Despite these limitations, it is reasonable to posit an impact of mental strain on recovery through its effect on cytokines that are tied to the recovery process.

Glucocorticoids

Cortisol is a primary agent in the stress-adaptation process, being activated via the Hypothalamic-Pituitary Axis (HPA). It is released in response to both physical and mental stressors (Webb et al., 2007) and is related to negative affect on a moment-by-moment basis (Smyth et al., 1998) and to ongoing, negative, daily life events (Van Eck, Berkhof, Nicholson, & Sulo, 1996). In an extensive meta-analysis (208 studies) of the human and animal literature, Dickerson and Kemeny (2004) found that cortisol responses were greatest and recovery was longest when induced stressors were uncontrollable and caused substantial social evaluation. In relation to physical activity, cortisol is released only after long duration of exercise (Kjaer, 1989; Pedersen et al., 1998). This contrasts the extant literature purporting that cortisol starts to release 10-30 minutes after the onset of an acute stressor. Webb et al. (2007) found that exercise combined with acute mental stress in a strained population (firefighters) produced cortisol responses greater than with

a matched group who underwent exercise alone. Unfortunately, these groups were not compared to a non-chronically strained population.

More importantly, cortisol has been identified as a central element in the wound-healing process. Ebrecht et al. (2004), found that wound healing was inversely related to cortisol-upon-awakening, which is a good indicator of chronic physiological stress (Wust, Federenko, Hellhammer, & Kirschbaum, 2000).[14] As mentioned earlier, perceived stress, as measured via the PSS (Cohen et al., 1983) is also related to wound healing. Interestingly, though morning cortisol and perceived stress are both related to healing, they have not consistently been related to each other, contrary to conventional expectations (Ebrecht et al., 2004). Is it possible that both cortisol and perceived stress are related to a third variable, life event stress? Conversely, there is some evidence that cortisol does not have influence in the wound-healing process. Specifically, Padgett, Marucha, and Sheridan (1998) demonstrated in a well-designed study that restrained (thus, stressed) mice with blocked glucocorticoid receptors had similar healing rates from wound-inducement as un-restrained mice.

General Cytokine and Cortisol Interactions

The relationship between stress hormones (e.g. cortisol) and inflammation is bi-directional (see Dhabhar & McEwen, 2001) and is influenced by training status, general health, type of stimulation, intensity of the stimulus, temporal effects, and other factors. Kunz-Ebrecht, Mohamed-Ali, Feldman, Kirschbaum, and Steptoe (2003) and DeRijk et al. (1997), describing the acute effect of cortisol on inflammation, conclude that cortisol is immunosuppressive in the face of threat, preventing release of key immune system actors until after the threat has subsided. Indeed, immune responses are expensive, and

[14] It must be noted that this study utilized a median split of healing with the outcome being perceived stress and optimism.

thus cortisol helps to both release (gluconeogenesis, lypolysis) and conserve energy when it is needed to fuel demanding levels of work. Evidence supports that Type I immunity, such as Natural Killer (NK) cell activity, is affected to the greatest degree (Clow & Hucklebridge, 2001). Cortisol is also immuno-enhancing when down-regulated and thus may play a prominent role in the stress and healing relationship.

Studies focused on the other direction of the cortisol-cytokine relationship (effects of cytokines on glucocorticoids) report that there is a negative feedback loop between cytokines released peripherally and the HPA axis (DeRijk et al., 1997). Cytokine stimulation of the HPA axis protects against immune overshoot. Less favorable is the pathology created from over-release of inflammatory cytokines (Malarkey & Mills, 2006), which may result in OTS and a host of other maladies (see Smith's Cytokine Hypothesis of Overtraining, 2000). As mentioned previously, dysregulation of inflammation is related to hypercatabolism (Path et al., 1997) and may explain why glucocorticoids are released in excess after unremitting stress. Clow & Hucklebridge (2001) and Dhabhar & McEwen (2001) note that there are paradoxical relationships between cortisol and immunity that are left to be explained.

Exercise-specific cytokine and cortisol interactions: Providing Answers to Paradoxical Relationships

Studies of exercise stimulus stressors provide an ideal paradigm to study interactions between stress mechanisms. Pedersen et al. (1998), after reviewing the extant literature on bouts of endurance training and inflammation, asserts that cortisol release is not related to cytokines released via exercise stimulus (see Lancaster, 2006 for a review). McCarthy and Dale (1988) provide evidence that immediate leukocytosis during exercise is due to increased release of catecholamines and cortisol results in delayed release of neutrophils. DeRijk et al. (1997) propose that IL-6 is resistant to the effects of cortisol, TNF-α (another pro-inflammatory cytokine) is greatly sensitive to cortisol production,

and IL-1β has intermediate sensitivity to cortisol. These authors report that exercise at 100% of VO_2 max results in increases of glucocorticoids but suppressed IL-6 and TNF-α production. Therefore, the picture is clouded by the intensity and volume of exercise workload.

Dhabhar & McEwen (2001, p. 307) propose that paradoxical relationships between cortisol and immune cells are probably explained by the magnitude of exercise stress. These authors utilize a military metaphor to explain this paradox. With low-intensity exercise, as with other mild stressors, catecholamine hormones and neurotransmitters act to increase leukocytes in the blood stream (which they term, the "boulevards"). Exercise that results in large activation of the HPA axis (such as with high intensity exercise) results in decreased leukocytes from the spleen, lung, and bone marrow (the "barracks"). However, these leukocytes do not return to their place of origin. On the contrary, they appear to take place at "battle stations" in preparation for large-scale challenge. These battle stations include the mucosal lining of the gastro-intestinal tract and urinary-genital tracts, lungs, liver, and lymph nodes. Their evidence provides broad implications for the current study in that the magnitude of the stimulation, relative to the capacity of the individual, greatly impacts the responses expected to be observed.

STRESS, STRAIN, AND RECOVERY PROCESSES: IS RESPONSIVENESS THE MOST IMPORTANT FACTOR?

Key Concepts

Stress, sometimes called strain, is a disruption of physiological and/or psychological homeostasis and/or allostasis.[15] It is the reaction of the human organism to any demand (Selye, 1993, p. 7). Homeostasis is a stable state or tendency towards equilibrium (in Greek, literally "same stable") and allostasis is stability within a larger

[15] Selye (1976) reflected later that he did not mean to call stress by that name but rather strain. Stress was merely a useful monosyllabic word that needed no translation in other languages.

range of operating values (literally, "variable stable"; McEwen, 1998).[16] Selye (1956) posits that stress is a non-specific, diffuse, general response to environmental demands/threats. He argues that humans respond the same physiologically to all types of stressors, regardless of whether they are socially, psychologically, or physiologically generated. Weiner (1992*)* counters by suggesting stressors elicit more specific responses (see Sapolsky, 2003) unless an individual is facing the most dire of circumstances, such as impending death.[17] In this conceptualization, exercise (as both a physical stressor and a mental stressor) can result in various stress responses. It results in numerous physiological disruptions but also highly predictable psychological responses, particularly when well above anaerobic threshold (Ekkekakis, 2003).

Stress theories have historically partitioned into two major lines of thought: response versus stimulus. Stimulus theories, as encapsulated by the original life stress theorists (e.g., Holmes & Rahe, 1967) suggest that the human organism experiences damage constant to the specific type of event (e.g., death of spouse, holiday) experienced. When measuring stress along these lines the magnitude of the event is determined through expert ratings and is not dependent on individual perception of event impact. The perception of stressor impact may be irrelevant. A stressor such as inordinate chronic exposure to radiation or a latent virus (e.g., HIV) impacts the body in a deleterious manner but is not likely to be perceived as an environmental stressor by the afflicted individual. In the Social Readjustment Rating Scale (SRRS), death of a spouse, therefore, is regarded as the worst possible stimulus (a rating of 100) while a major holiday (a rating of 12) would be considered a lesser stimulus with smaller associated damage. Some

[16] McEwen (1998) provides an extensive review of homeostasis and allostasis and its relationship to physiological adaptation.

[17] Exercise may also be construed as a psychological stressor for those who are not acclimated to difficult or unaccustomed forms of physical activity. Physiological responses to exercise may lead to positive adaptations.

might not consider a holiday a source of stress as the associated time off may provide an enjoyable and relaxing reprieve from other stressors. Nevertheless, it places upon the human organism other demands which may attenuate possible adaptations.[18] In the same vein, stressors may be classified as distress (bad stress) or eustress (good stress), depending on the appraisal of the individual.

Life event stress is further regarded as the summation of major change experiences (such as a marriage, divorce or loss of employment) that occur over a period of time (usually 3 to 12 months). Traditional life event stress instruments, such as the SRRS, count the number of "hits" an individual accumulates. These perturbations may include divorce, exams, breakups, incarceration, or other stressful events. Stressful life events are often associated with a lasting stress response, sometimes with effects lingering for months or years.

Stimulus and response theories of stress have innumerable detractors, however. John Dewey, the preeminent American philosopher and psychologist, denounced stimulus and response theories as inconsequential to the greater notion of an "organic circuit" (Dewey, 1896; Menand, 2001, p 328-329). He espoused the idea that stimulus and response were two sides of the same coin, or merely different aspects of a single, indivisible process. In his interpretation, I can only know the stimulus because of the response it elicits.

Modern scientists, such as Lazarus & Folkman (1984), also dismiss the notion of stimulus and response. They conclude that the individual perception and appraisal are more important considerations. In their Transactional Model of Stress and Coping, they define stress as "a relationship with the environment that the person appraises as

[18] As noted by Semmer et al. (2004), this rating of stressful experiences is very general. If one's spouse died on Christmas, then this particular holiday may always be associated with her/his death thus eliciting more negative stressful experiences on future Christmas holidays.

significant for his or her well-being and in which the demands tax or exceed available coping resources" (Lazarus & Folkman, 1986, p. 63). Stress must be perceived, receive attention, and be rated as threatening in order for it to have an impact. The secondary process in their Transactional Model states that stress and strain is maximized only when the individual perceives that they do not have the resources available to them to cope with the ordeal. Theory from these researchers spearheaded the development of testing instruments such as the Perceived Stress Scale (PSS; Cohen et al., 1983). Furthermore, their theory has resulted in refined life stress checklists, many of which now include stress impact scores (such as the APES, which was utilized by Bartholomew et al., 2008, see below). Considering the multifarious nature of the stress concept, Lazarus suggests that, "stress can be treated as an organizing concept for understanding a wide range of phenomena of great importance in human and animal adaptation. Stress, then, is not a variable, but rather a rubric consisting of many variables and processes" (Lazarus & Folkman, 1984, pp. 11-12).

Reactivity and Responsiveness

As previously noted, the terms stress and strain are often used interchangeably but each refers to a different component of a dynamic process (Ekkekakis, 2003; Kirschbaum, Kudielka, Gaab, Schommer, & Hellhammer, 1999; Rietjens et al., 2005; Selye, 1993). Although generally placed in a negative light, stress can be facilitative when paired with adequate resistive forces and strong recovery (Weiss, Glazer, Pohorecky, Brick, & Miller, 1975 as cited in Dienstbier, 1989). Indeed, Kellman (1991, 1997, 2000) reports that stressful demands must be balanced with periods of recovery (see Figure 2.6). Selye (1956) theorized under the General Adaptation Syndrome that under conditions promoting recovery, the stress response provides the means for

adaptation by developing greater stress resistance and improving capacity.[19] Indeed, the specific goal of periodized exercise training is to balance stress and recovery as a means to improve strength and fitness (Bompa, 1999).

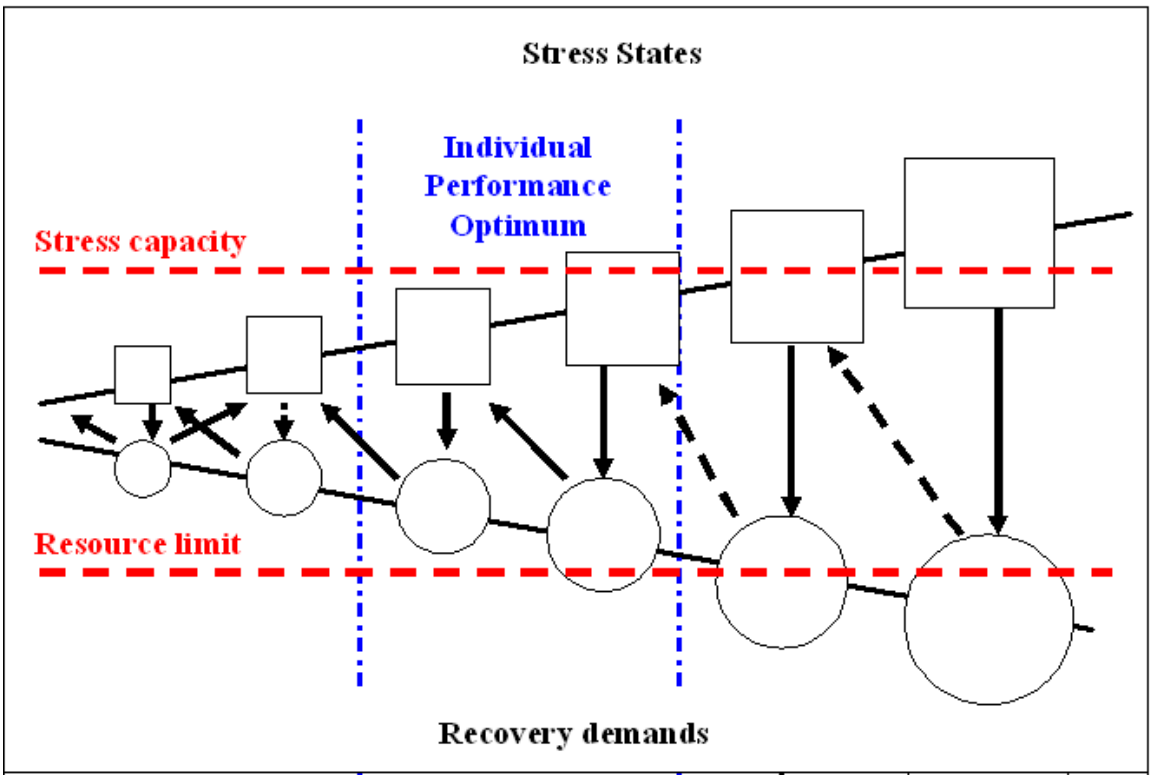


Figure 2.6 Kellman's Scissors Model of Stress States and Recovery Demands (1991, 1997, 2000). When demands are chronically high and recovery is poor, adaptation will not be optimal. The model demonstrates the balance that must be achieved in order to reach a high level of performance.

Chronic stress and strain that lacks the opportunity for full recovery takes its toll on the human organism. Unremitting stress results in lack of regeneration whereas superior recovery results in maximal adaptation and associated performance (see Figure 2.7). Stressful events may result in a cascade of physiological sequelae, beginning with

[19] Selye's General Adaptation Syndrome (GAS) posits that stress reactions are divided into 3 phases: a) shock, b) resistance and c) exhaustion.

an increased activation of the Sympathetic/Adrenal-Medullary (SAM) system and Hypothalamic-Pituitary-Adrenal (HPA) axis (Stratakis, Gold, & Chrousos, 1995; Chrousos & Gold, 1992).

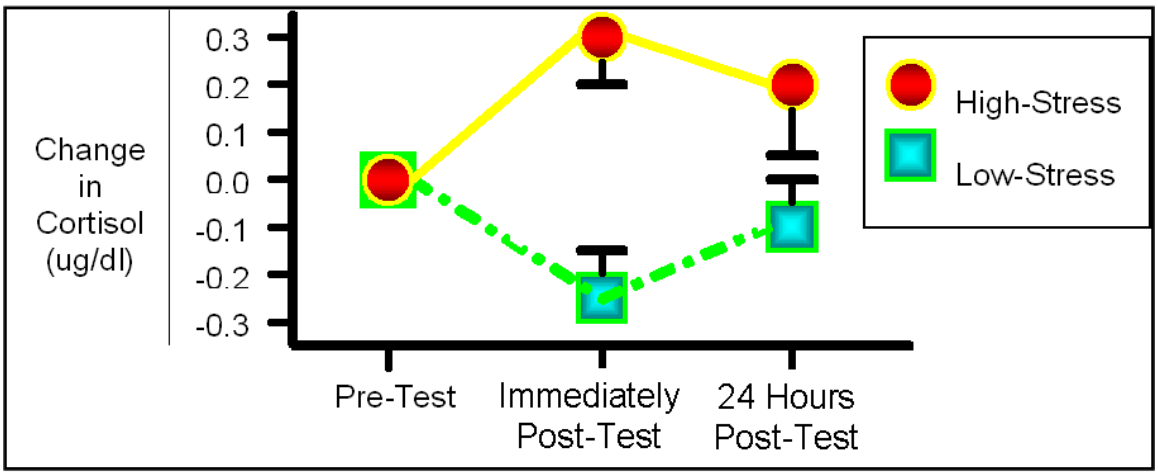


Figure 2.7 Cortisol responses in well-trained endurance athletes of high- and low-stress after maximal exercise (Perna & McDowell, 1995). The pre-test means for the high- and low-stress groups is 1.267 (SD = 0.3481) and 1.559 (SD = 0.381), respectively.

Individuals high in chronic life stress are more reactive to laboratory stressors (Pike et al., 1997; Semmer et al., 2004) and several studies find that chronically stressed individuals have greater cortisol reactivity to awakening (Ebrecht et al., 2004; Wust et al., 2000). Recent evidence demonstrates that both actual and perceived chronic stress results in increased oxidative stress and shorter telomeres in the DNA (Epel et al., 2004). Furthermore, it has been well documented that a disproportionate exposure to life stress has negative implications for people's health (Damush, Hays, & DiMatteo, 1997), including the number of illnesses they experience as well as rates of morbidity (House, Strecher, Metzner, & Robbins, 1986). Minor stressors, known as daily hassles, are also associated with deleterious effects on one's health (Kanner, Coyne, Schaefer, & Lazarus, 1981).

It is not sufficient to classify the stress response in a wholly negative light. In fact, the ability to respond to stress is a key component of general health. According to Sapolsky (1999), Semmer et al. (2004), and Semmer, McGrath, & Beehr (2005, pp. 12-13), health is defined to a greater degree as a *state of responsiveness* as opposed to a range of physiological values. A very fast reaction to stress (swift responsiveness) may be a sign of a strongly functioning system, particularly when followed by a very quick return to baseline (Dienstbier, 1989, 1991, 1992). Those exhibiting strong physiological reaction followed by a strong recovery demonstrate what Dienstbier (1989, 1991, 1992) refers to as physiological "toughness", a positive stress adaptation (see Figure 2.8). Dominant baboons, for instance, demonstrate strong cortisol responses and rapid recoveries compared to more passive, and thus more strained, baboons. The latter have more delayed physiological recovery (Sapolsky, 1995). Roy, Kirschbaum, & Steptoe (2001) found that after exposure to strenuous mental tasks in the laboratory, those who had experienced greater psychological "activation" and more daily hassles in the previous week exhibited the most delayed cortisol recovery. What is unclear from this study, however, was whether their groups differed in initial and basal levels of cortisol and whether their cortisol during recovery receded to sub-baseline levels. Thus, the line between stress reactivity and stress recovery remains unclear.

This general premise applies to chronic stress as well. Those who are able to flexibly manage and adapt to chronic stress likely recuperate more quickly from the transient stressors couched within the greater stressful environment (Eden, 2001; Repetti, 1992, as cited in Semmer et al., 2005, p. 12). Lack of responsiveness to stress is often mislabeled as adaptive and may actually be maladaptive and indicative of physiological exhaustion. Whereas cortisol is a primary stress hormone, its mobilization is absolutely essential for metabolism and immune function. Consequently, physiological disruption is

adaptive in the acute or short-term whereas chronic disruption leads to physiological burnout (Selye, 1993; Semmer et al., 2004).[20]

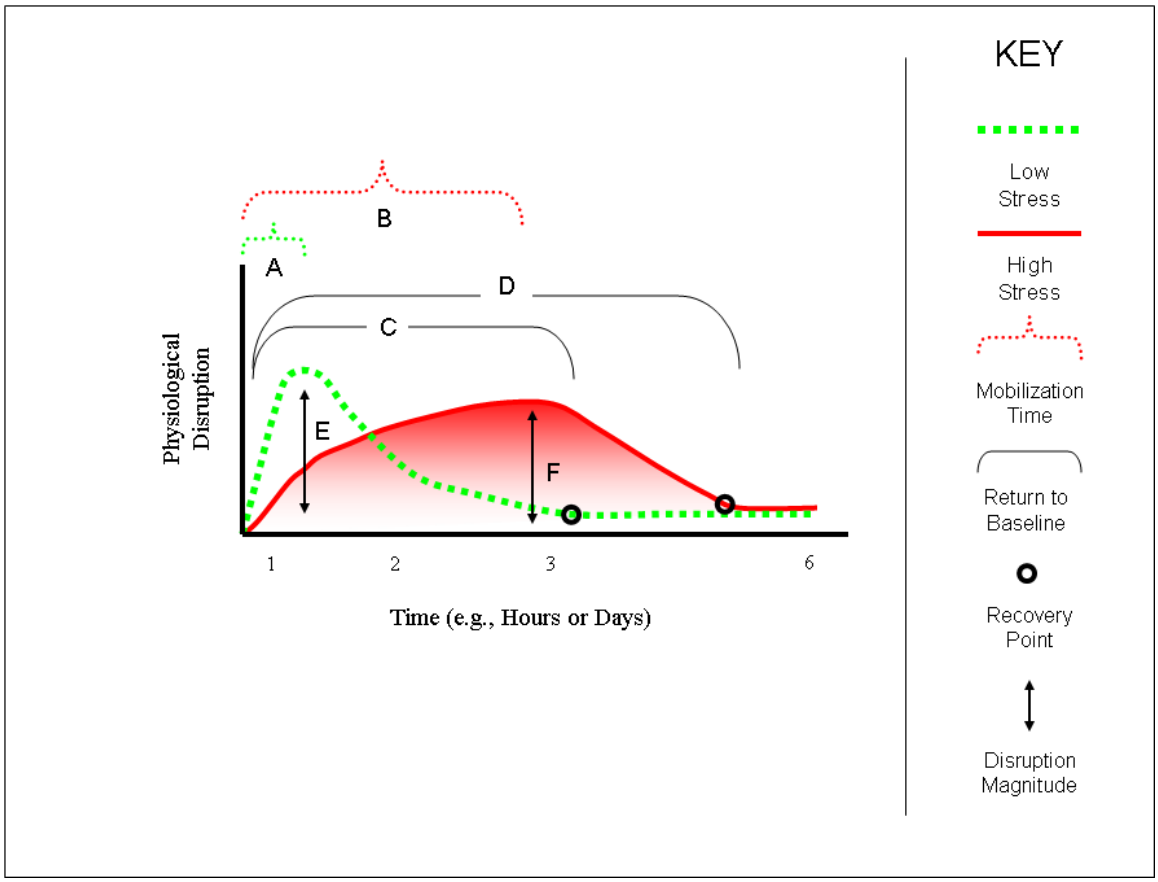


Figure 2.8 The impact of psychological stress on physical strain reactions. Stress reactivity, comprised of disruption and recovery, has many identifiable parameters, including: i. mobilization (time to reach asymptote; brackets A & B), ii. point of recovery (rings on curves), iii. return time to baseline (i.e., recovery; brackets C & D), iv. disruption magnitude (difference from baseline; brackets E & F) and v. disruption magnitude (AUC – area under the curve; shaded area). Disruption and recovery can also be quantified as slope (rise over run). Conceptual basis adapted from Dienstbier (1989, 1992) and visual representation created by the author.

[20] See Semmer et al. (2004, p. 227-229) for a discussion of recovery parameters and how they are related to chronic stress.

Recovery patterns vary greatly with dysfunctional adaptations. For instance, according to McEwen (1998),McEwen and Lasley (2002), and Temoshok (2000) physiological response may be characterized by:

a) delayed mobilization (weak activation, see parameters a/b in hypothesis 1 diagram, also see Siegrist, 1998, p. 199),[21]
b) exaggeration (high reactivity or hyper-responsivity, see parameters c/d in hypothesis 1 diagram),
c) non-remittance (cannot "turn off" the response, e.g., Cushing's Disease), or
d) no response at all (exhausted, e.g., Addison's Disease).

Impaired recovery may be indicative of diminished ability of protective mechanisms, exhaustion of resistive forces, and activation of last-resort compensatory mechanisms (Linden, Earle, Gerin, & Christenfeld, 1997; Roy et al., 2001). The phenomenon of "slow unwinding" is a well-known consequence of unremitting stress (Semmer et al., 2004, p. 227). Both instances may be facilitative, even chronically, in that they may ultimately allow for conservation of energy. McEwen (1998) and McEwen and Lasley (2002) refers to this breakdown as an "allostatic load" in which the human organism degrades systemically over years and even decades. Tenets of McEwen's theory explain the progression of disease and dysfunction from pre-disease states (e.g., pre-hypertension, pre-diabetes) to clinical diagnosis (McEwen, 1998; McEwen & Seeman, 1999), functional and muscular decline over time (Karlamangla, Singer, McEwen, Rowe, & Seeman, 2002) and even the aging process itself (Seeman, McEwen, Rowe, & Singer, 2001).

[21] See the reduced responsivity (hypo-sensitivity) of cortisol found in Heim, Ehlert & Hellhammer (2000, as cited in Semmer et al., 2004, p. 228). Temoshok (2000) provides an graphical representation of dysfunctional stress responses.

Perna & McDowell (1995), in a rare study investigating the effects of chronic stress on exercise stimulus responses, found that those with high-stress have exaggerated cortisol responses from maximal exercise. High-performing endurance athletes (cycling and rowing) who were high in life stress had an increase in cortisol after a maximal treadmill test. The effect size (Cohen's d) associated with this group difference was 0.72 immediately post-exercise and 0.20 a full day later. Interestingly, those low in life event stress had a decrease in cortisol immediately after exhaustive exercise (See Figure 2.7 above). The investigators note that the high-stress group had lower cortisol values at the pre-test, which possibly relates to their inability to properly activate a stress response in anticipation to an acute stressor. This corroborates well with McEwen's theory of stress responsivity. Both groups were also able to achieve maximal exhaustion and had similar levels of fitness, agreeing with work from the laboratory of Noakes (St. Clair Gibson et al., 2006 that strained and depressed individuals are similar to non-strained individuals in their ability to reach maximal fitness values. In this study, these groups had similar resting, maximal isometric force (MIF), VO_{2max}, maximal heart rate, and lactate responses.

Impact of Mental Stress/Strain on Athletic Injuries, Health and Fitness Adaptations

The relationship between life event stress, illness, and susceptibility to sports injuries is well established (Andersen & Williams, 1999; Fawkner, McMurray, & Summers, 1999; Ford, Ekland, & Gordon, 2000; 1999; Junge, 2000). There is a strong positive correlation between life event stress and injury. Life event stress is also associated with illness over the course of an entire sports season (Yi, Smith, & Vitaliano, 2005). Clow & Hucklebridge (2001) posit that chronic stress is also associated with prolonged rehabilitation from sports injury, though they credit mainly personality and behavioral constructs in this relationship.

Exercise training is a stressor (Coyle, 2000; Pedersen & Hoffman-Goetz, 2000) and is an exemplar of dynamic stress and strain processes. Some researchers partition stress stimuli into two types: training and non-training (Lehman et al., 1993; Kenttä & Hassmén, 1998). Together they make up "total stress" or "cumulative stress". Training stress is further decomposed into individual bout units of exercise, called training impulses or TRIMPs (Kenttä & Hassmén, 1998; Morton, 1997). As alluded above, exercise stimuli is described in terms of the type or mode (e.g., aerobic, resistance), total load or training volume, the intensity, and whether failure was achieved (also known as an "overload stimulus").[22] Kenttä & Hassmén (1998, p 14) suggests that each individual exhibits a dynamic adaptation threshold, which is responsive to the magnitude of exercise training. In this sense, muscular strength is an example of functional stress capacity, which is genetically endowed, influenced by environmental factors and accentuated through physical training. Through training, an individual may increase her or his exercise stress tolerance, thus habituating to heavier and heavier loads. Fitness, or stress capacity, is essentially the uppermost ability to resist stress. Several stress theorists have utilized this model to demonstrate principles of stress and strain (Carver, 1998; Semmer et al., 2005). In short, fitness adaptations are responses to physical stress. Is it tenable that non-training stressors (e.g., mental stress and strain) impact these physical responses?[23]

This hypothesis was tested by Bartholomew et al. (2008). This investigation demonstrated that life event stress negatively impacts the development of fitness. In a

[22] Long-term manipulation of training stress to maximize adaptations is known as periodization (Bompa, 1999). There is substantial evidence to demonstrate the training stress cannot increase linearly and indefinitely without deleterious effects on one's health. Even within the scope of a single semester, stress of training results in maximal adaptations when the exercise volume undulates. This line of research also has demonstrated that adequate rest is absolutely essential for recovery and adaptation.

[23] In the opposite direction, the effects of fitness and exercise on anxiety and stress are well known (see Holmes, 1993).

prospective study, these investigators found that those above the median for negative life event stress, as reported via the Adolescent Perceived Events Scale (APES), developed less upper and lower body strength over the course of a 14-week resistance-training program. Because the effect of mental strain on muscular hypertrophy and strength was not invariant these data can be used to infer conditions under which this relationship does not hold and also potential mechanisms through which stress may impact fitness.

Bartholomew et al. (2008) posited that the stress-adaptation relationship may be mediated or moderated by several mechanisms: biological responses such as increased basal cortisol (Perna & McDowell, 1995; Sapolsky, Romero & Munck, 2000), changes in nutrition (Baum & Posluszny, 1999), illness and related absence from training (Yi et al., 2005), or health-related and training behaviors (Lutz, Stults-Kolehmainen & Bartholomew, 2009). Specifically, those who are stressed may experience central fatigue and may exert a decreased level of training effort or may fail to exercise, particularly if not a habitual exerciser (Davis & Bailey, 1997; Lutz et al., 2009). Another possibility is that these individuals may experience an exaggerated or premature fatigue to the same stimulus, although this has not been supported (Perna & McDowell, 1995; St Clair Gibson et al., 2006). A recent paper by Marcora et al., (2008) found that the experience of acute mental stress was related to the amount of work accomplished. Individuals just completing a grueling mental task also had greater sensations of exertion with exercise. It is not clear, however, if this effect extends to chronic stress. It is tenable that chronically stressed individuals do not make gains to the level of lower-stressed counterparts because they are not willing to complete, or simply cannot complete as much physical work. Lastly, it is possible that those higher in chronic stress may have compromised ability to recover from training stress.

Stress, Pain, Fatigue, and Recovery

Pain is both conceptualized as an outcome of stress and as a negative contributor to the process of recovery (Keicolt-Glaser et al., 1998, p. 1215). Stress is related to pain (Graham et al., 2006; Melzack & Katz, 2004; Christian et al., 2007) and illness symptoms (Stone, Reed, & Neale, 1987). Elfering, Grebner, Semmer, and Gerber (2002), studying a cohort of nurses, found that a occupational stress (derived from a lack of control over time use during work) was significantly related to musculo-skeletal pain and low back aches. Affleck, Tennen, Urrows, and Higgins (1994), investigating a group of individuals with active inflammatory disease, found that the experience of pain was related to stress on the same day and the previous day. Physical stress, such as illness, is related to increased fatigue, sickness behavior, and decreases in positive affect (but not changes in negative affect; Janicki-Cohen, Cohen, Doyle, Turner, & Treanor, 2007).

As alluded to above, pain is related to slower healing (McGuire et al., 2006 as cited in Christian et al., 2007). DOMP is related to performance decrements over several days. In one study, vertical jump height decreased for 3 to 4 days (Mair et al., 1995). Pain is related to use of anti-inflammatory and pain medications and may initiate the negative coping behavior process (increased use of cigarettes, alcohol consumption, and drugs). It is also likely related to decreased movement, exercise, as well as quality and quantity of sleep.[24]

[24] See Maier & Watkins (1998) for discussion of stress-induced analgesia and pain-induced recuperative behavior.

METHODOLOGICAL PROBLEMS WITH PREVIOUS RESEARCH

Issues related to Quantification of Stress, Strain, and Recovery

Studies on recovery from physical and psychological stress suffer from major methodological problems. The largest problems stem from quantification of stress, strain, and recovery processes.

i. Workload has not been quantified.
ii. Only one measure of psychological strain is utilized.
iii. Recovery has been over-simplified (Dickerson & Kemeny, 2004; Linden et al., 1997; Matthews, 1986 as cited in Dienstbier, 1989). Researchers typically choose a single recovery parameter for study (e.g., days to full recovery or area-under-the-curve).
iv. Follow-up and timing of the recovery period has been limited.
v. Stress is often assessed in the health literature only *after* people become ill or injured. Also, there is a general lack of well-designed prospective studies (See Sapolsky, 2004).

Issues related to subject characteristics and ecological validity

i. Rarely is fitness measured. Individuals are simply selected by training status.
ii. The physical stressor is rarely applicable to the real world. Most studies are completed on isokinetic dynamometers or only use eccentric actions.

It is with the findings and limitations of previous studies that this dissertation was developed to examine the effect of chronic mental stress and strain from exhaustive resistance exercise.

CHAPTER THREE

METHODS

EXPERIMENTAL DESIGN

The purpose of this study is to examine variations in recovery from disruptive exercise and test the possibility that mental strain may account for differences in force production. This investigation was prospective; individuals completed an exhaustive resistance exercise protocol (E-RES) and were tracked for 96 hours post-exercise. This workout was strong enough to ensure that a *micro*-level of muscular damage was induced, which was necessary to demonstrate reductions in muscular function (e.g., reduced power output) and thus recovery. I selected a multi-level approach to investigating recovery. In other words, individual observations were nested hierarchically into groups of observations classified by the study participant. This varies from nomothetic analyses focusing on group differences (i.e., low stress versus high stress groups) and from idiographic analyses, which study how single individuals experience recovery over time (Hanin, 2002).

The design was quasi-experimental as chronic stress and mental strain was not manipulated. One problem associated with these studies is that variability is often constrained for the stress measure. This limits the ability to detect an effect, particularly because the effect of stress is likely most pronounced for values in the tails of the stress distribution. Therefore, it makes sense to oversample at the extremes in order to detect an effect with a manageable number of participants. A screening tool was selected to ensure variability for stress measures (see below). A dilemma that emerges when utilizing this procedure, however, is that participants may regress to the mean. In particular, this may

be a problem when there is a significant amount of time (2-4 weeks) between screening and laboratory visits. Nevertheless, considering the necessity of ensuring enough variance, the practicality of this approach must be balanced against the chance of regression to the mean. To prevent order effects, subjects were recruited over two semesters (Fall 2008 and Spring 2009) with both low and high stress individuals in both semesters.

DESCRIPTION OF SUBJECTS

Demographics

Participants were 18-23 years of age, including nine women and 22 men. All subjects were undergraduates enrolled in morning and afternoon weight-training classes that met twice weekly for 1.5 hours each session. There were approximately 40 sections of this course in the Fall of 2008 and Spring of 2009 with about 1,200 total students. All sections were actively recruited to participate in the study. Ten individuals self-reported ethnicity as "Caucasian," seven reported "Hispanic/Latino/a," eight reported "Asian/Pacific Islander," and six reported "other." Of those reporting "other," four considered themselves of Middle Eastern origin.

Health History

All students were in good health and with no history of musculoskeletal disorders or recent injury. All participants completed a health-status questionnaire that placed them in low, moderate, or high risk for medical problems during exercise (ACSM & AHA, 2000). The *American College of Sports Medicine's Guidelines for Exercise Testing and Prescription, Seventh Edition* (2006) were used to determine risk level. Only low-risk participants were allowed to participate in the study. Students stratified as moderate or high risk were notified of their status and disqualified from the study. The health-status questionnaire also assessed individuals for physical limitations, musculoskeletal

problems, and/or other factors (e.g., medications, steroids) that would affect physical training/adaptation or strength testing. Students with lower body musculoskeletal problems within the last two years, history of lower back injury, history of or current lower back pain, past or current use of muscle relaxants, etc. were disqualified from the study.

Menstrual and Pregnancy Status

Eligible women were in the luteal phase of their menstrual cycle (17-25 days after the end of their menstrual cycle) or taking contraception medications (Kirschbaum et al., 1999). Women indicated their pregnancy status in written and oral form. Those who answered 'yes' or 'unknown' were ineligible for the study.

Exercise Experience

These individuals had a wide variety of training experiences, from none to highly formal high school training backgrounds. Most of these students, however, were recreationally trained and had not previously received formal instruction in strength training. A small minority (16%) of the students were in the class for a second, third, or fourth semester. All students had a minimum of 5 weeks of instruction and experience with leg press exercise before the beginning of the protocol to minimize risk of *macro*-damage to the musculature (e.g., muscle strain).

BEHAVIORAL RESTRICTIONS

Supplements and Over-the-Counter Medications

Participants were instructed to abstain from a list of anti-inflammatories and supplements, including ibuprofen, creatine monohydrate, supplemental anti-oxidants, flax seed oil, and fish oil for the entire experimental period (48 hours before laboratory testing and up to 96 hours afterwards).

Caffeine

Supplements and drinks with caffeine were not allowed for 3 hours before any physiological/functional measurements. Before this period, caffeine was permitted up to about 200 mg per day, or the equivalent to: (a) 2 cups of coffee, (b) one “tall” Starbucks coffee, (c) 4-12 oz. cans of Mountain Dew, (d) 6-12 oz. cans of coke, (e) 2-8 oz. cans of Red Bull, (f) 4 cups of tea, or (g) two caffeine tablets. Participants were told to be well hydrated before all laboratory visits. Chronic high caffeine users (e.g., three or more cups of coffee per day) were disqualified from the study.

Exercise and Therapy

Participants were instructed to perform only light recreational exercise during the experimental period (2 days before laboratory testing and up to 96 hours post-testing). This included exercise in their weight-training class (they were excused from lifting). Very light lifting with the upper body was allowed. Daily commuting (e.g., bicycling, walking) and other physical activities (excluding resistance training) were only permitted if they could be classified as ‘easy’ in intensity (not causing moderate to excessive perspiration, heavy breathing, and/or a fast heart rate). They were not permitted to have special therapy sessions for the lower body, such as massage, electrical stimulation (e-stim), ultrasound, cold-water emersion, or other forms of hydrotherapy.

PROCEDURES

Recruiting

The weight-training classes were visited and students received a brochure explaining the details of the study along with the benefits, incentives (which included two absence make-ups), and risks. Students were then invited to go online to complete a screening instrument. Recruitment in classes was staggered throughout the semester.

All participants signed an informed consent following a lengthy explanation of the study after which they were allowed to ask questions. Subjects were reassured that (1) the study was completely voluntary, (2) they could volitionally discontinue completion of the protocol at any time, (3) they could leave the study at any time without penalty or loss of benefits for which they were already entitled, and (4) their participation or lack of participation would not affect their relationship with the investigators, course instructors, or the university.

Screening

Students went online to complete a short survey to determine eligibility for the study. Individuals were screened for perceptions of chronic mental strain and depression using the Perceived Stress Scale (PSS; Cohen et al., 1983) and the CES-D inventory (Radloff, 1991). Those who scored at and above 19 (approximately ½ a standard deviation above the semester-long mean) or at and below 13 (approximately ½ a standard deviation below the semester-long mean) were eligible. Individuals who scored at or above 28 on the CES-D were not be eligible for the study. See Figure 3.1.

Fitness Testing, Blood Draw, and Familiarization

After online screening for mental stress/strain, eligible individuals were scheduled over e-mail for their first visit. They completed the informed consent process and then completed the PSS (paper/pencil version) an additional time to corroborate initial stress/strain results. Complete fitness testing took place, including body composition, muscular strength and power, and aerobic capacity. Settings for the isometric muscle force apparatus were set-up during this initial visit and maximal isometric force (MIF) was tested. Careful familiarization of the isometric force apparatus and the MIF force protocol took place. At the beginning of each visit, participants were given a bottle of

water to drink to ensure good hydration. This entire visit took about 2 hours. See Table 3.1 for an outline of the procedures.

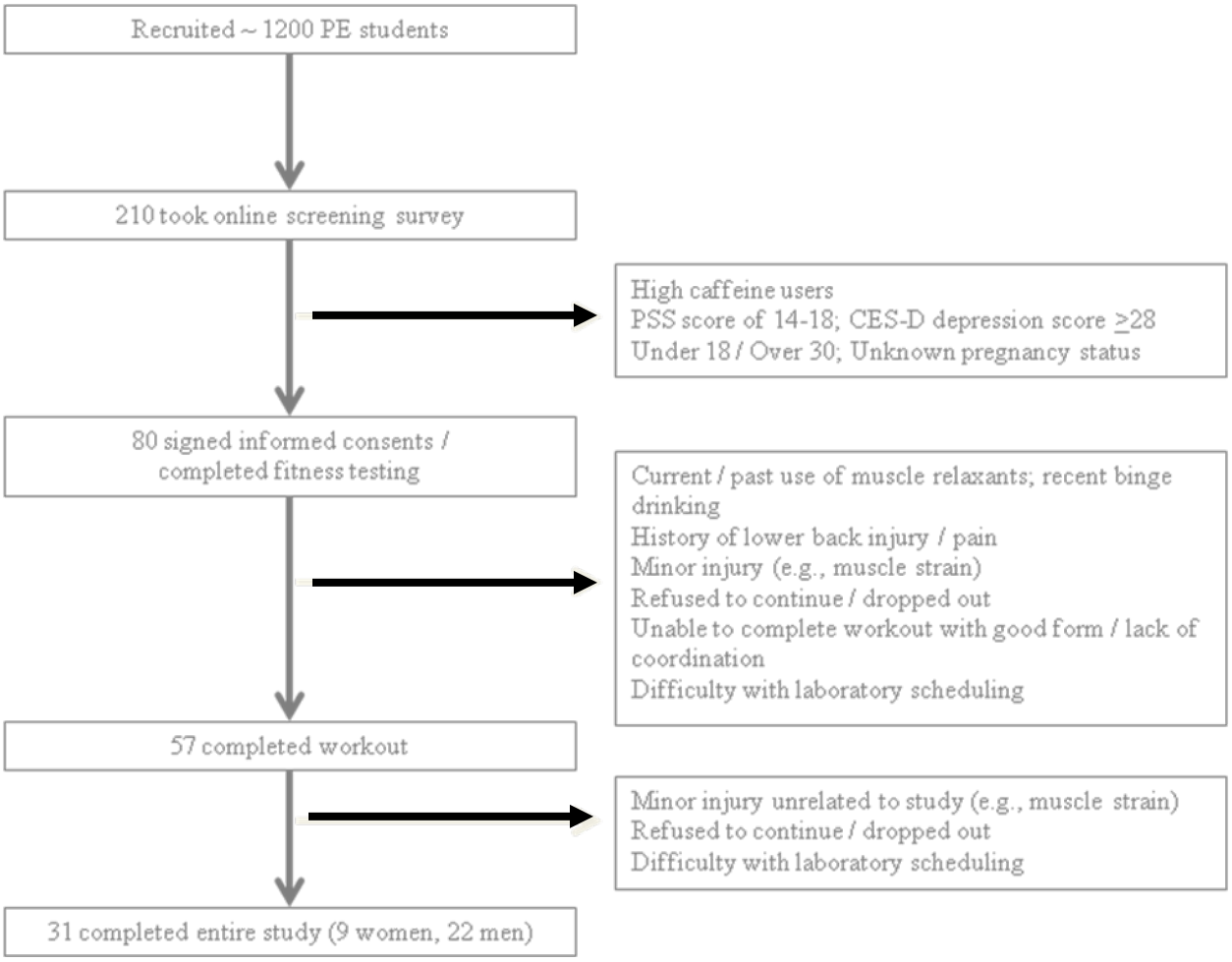


Figure 3.1 Flowchart of participants through study

Table 3.1 Overview of study visits and study time allotment.

VISIT	#	Action Item	Time
Screen	A	Eligibility Questions (e.g., age 18-30)	
	B	PSS-10 (score $\leq$ 13 or $\geq$ 19)	< 5 minutes
	C	CES-D (score $\leq$ 27)	
Visit 1:	A	Study Introduction	
	B	Informed Consent	
	C	Demographics Questionnaire	
	D	Health/Fitness History Questionnaire	
	E	PSS-10 (second time)	
	F	PANAS	
	G	Energy, Fatigue	
	H	UG Stress Questionnaire	2.0 hours
	I	PRETIE-Q	
	J	Blood Draw (fully rested)	
	K	Body Composition (DEXA)	
	L	MIF Apparatus Set-Up	
	M	Criterion measures: MIF*, Squat Jump, Power	
	N	Strength measures (leg press, bench press)	
	O	Bike Cycle Test	
Visit 2:	A	FS, FAS, PANAS, Energy, Fatigue, Soreness, PSS	
	B	Blood Draw	
	C	Criterion Measures: MIF*, Squat Jump, Power Bike	2.0 hours
	D	Leg Press Exercise Stimulus Protocol	
	-	(see Tables 3 and 5 for complete details)	
	E	FS, FAS, Psychological Recovery	
Visit 3:	A	FS, FAS, PANAS, Energy, Fatigue, Soreness	
	B	Blood Draw	0.5 hours
	C	Criterion Measures: MIF*, Squat Jump, Power Bike	
Visit 4:	A-C	Same items as in Visit 3	0.5 hours
Visit 5:	A-C	Same items as in Visit 3	0.5 hours
Visit 6:	A-C	Same items as in Visit 3	0.5 hours

* Muscular pain assessed for each MIF push

Assessments: General Screening, Psychological Screening, and Personality

Online Screening Questionnaire. These items screened for (a) age (must be 18-30 years of age), (b) participation in a current PED 106c class, (c) student status (e.g., undergraduate, graduate), (d) excessive caffeine use, (e) chronic mental stress and strain, and (f) depression.

Demographics Questionnaire. Participants indicated gender, age, ethnicity, work hours, loans, and number of semesters taking the weight-training class.

Perceived Stress Scale: 10-Item (PSS). The PSS measures the degree to which situations in one's life are appraised as stressful (Cohen et al., 1983). The current study utilized the 10-item version, which has high reliability of 0.78 (Cronbach's α), which is similar to the original 14-item PSS. The PSS-10 was determined to have two factors via principal components analysis, with the factors divided by negatively and positively worded items (48.9% of the variance was explained by the two factors) (Cohen & Williamson, 1988). In a national probability sample of 2,270 people, the PSS-10 was correlated with the number of life events (r = .32, p < .0001) and the negative impact of life events (r = -.27, p < .0001) (Cohen et al., 1983; Cohen & Williamson, 1988). A large national sample of young adults in college, the military, and the general work force, ages 18-29 years (N = 645), had a mean PSS of 14.2 (SD = 6.2).

Existing data on 357 undergraduate students drawn from The University of Texas at Austin's physical activity courses showed that these students had a mean PSS score of 14.4 (SD = 5.5) at the beginning of the semester and 17.8 (SD = 6.1) in the finals period. In this sample, the PSS was correlated with a measure of life events (APES, r = .55). The PSS was completed online outside of class and in the laboratory as a paper/pencil survey.

Undergraduate Stress Inventory (UGS). The UGS was used to measure school and non-school related life events that occurred in the last month. The objective form is a

checklist that has 83 items. Respondents did not weigh the items, thus the contamination from negative affectivity is reportedly low (Crandall, Preisler, & Aussprung, 1992). The UGS has adequate psychometric properties: internal consistency was 0.80, split-half reliability was 0.71, and test-retest reliability over the course of a 6-week summer semester was 0.59. The correlation of the USG with state mood (BMIS) was -.26, with negative affect (NEM) of .09, and with physical symptoms (PILL) of .53. The average inter-item correlation was .050-.083. The UGS correlates well with other stress inventories (r = .79 with Subjective Distress Scale, r = .97 with the Objective Stressor Scale) and has been related to immune function (Burns et al., 2003). It takes 3-5 minutes to complete (Crandall, Preisler, & Aussprung, 1992).

Center for Epidemiological Studies Depression Scale (CES-D). The CES-D has 20 items, which were selected from five previously used depression scales (Ensel, 1986). These items relate to all dimensions of depressive symptoms, including depressed mood, feelings of guilt and worthlessness, feelings of helplessness and hopelessness, loss of appetite, sleep disturbance, and psychomotor retardation. Each item is rated on 4-point scales indicating the degree of their occurrence during the last week. The anchors range from "rarely or none of the time" to "most all of the time." In a sample of 214 college-going emerging adults, Radloff (1991) found a mean score of 15.46 (SD = 9.67). If a traditional cut-off for depression is utilized, 40.65% of the sample would be considered clinically depressed. Therefore, a more stringent cut-off of 28 was recommended, which represents 13.55% of this group. Those who scored above 28 were not eligible for the study because these individuals were at risk for psychomotor retardation, likely to be less motivated to complete the protocol, may not achieve maximal performance values, or were at an elevated risk for drop out (St. Clair Gibson et al., 2006). The CES-D was completed online.

PRETIE-Q (Tolerance Sub-scale). The PRETIE-Q (Ekkekakis, Hall, & Petruzzello, 2005; Ekkekakis, Lind, Hall, & Petruzzello, 2007; Ekkekakis, P., Lind, E., & Joens-Matre, R. R., 2006). was used to measure tolerance for exercise intensity. This scale has eight items with four that assess high exercise tolerance (e.g., ‘‘I always push through muscle soreness and fatigue when working out.’’) and four that tap low exercise tolerance (e.g., ‘‘during exercise, if my muscles begin to burn excessively or I find myself breathing very hard, it is time for me to ease off.’’). Every item has a 5-item response with anchors ranging from “I totally disagree” at 1, to “neither agree nor disagree” at 3, to “I strongly agree” at 5. Low tolerance items (1, 3, 9, and 13) are reversed-scored. Internal consistency ranges from $\alpha = 0.82$ to $\alpha = 0.87$. Test–retest reliability (after 3 and 4 months) ranges from $\alpha = 0.85$ to $\alpha = 0.72$, respectively.

Assessments: Measures of Fitness and Blood Draw

Aerobic Capacity Test. Aerobic capacity was determined with a Storer incremental protocol test with an Excalibur Sport electronically braked testing bicycle (Lode BV, Groningen, The Netherlands). This protocol was selected to minimize soreness before the leg press test, which is more common than treadmill testing. This test starts at 25 watts of resistance and increases in wattage progressively until volitional failure. VO_{2peak} is estimated from the peak wattage achieved at exhaustion. Resting and maximal heart rates were recorded with a Polar-OY (Kempele, Finland) telemetric heart rate monitor.

Body Composition (DEXA). After measurement of height and weight, participants’ percent body fat was determined. This study used dual-energy x-ray absorpiometry (DEXA) with a Lunar apparatus and Prodigy software (G. E., Madison, WI). Female subjects administered a DEXA scan indicated that to the best of their knowledge, they were not pregnant. The DEXA technician verbally verified this

information. In the DEXA procedure, subjects laid down on a padded table while a small probe that emitted energy to measure tissue density was passed over the body.

Upper Body and Leg Strength. Strength was assessed via the bench (an upper-body movement) and leg presses. Participants completed 4-6 sets of leg press with a plate-loaded, 45 degree Cybex. Weight progressively increased with each set. Strength was determined from muscular failure at 3 to 5 repetitions in the last set. 1-RM was determined from coefficients reported by Brzycki (1993).

Blood Draw. Inflammatory cytokines were collected for about half of the participants by taking one blood sample (about 8 teaspoons) obtained by venipuncture from an anticubital vein. About 250 ml of blood over seven total draws was collected. The blood samples were analyzed for IL-6, TNF-α, and IL-1ß. To ensure the safety of the participants and study personnel, procedures were followed in accordance with the Environmental Health and Safety Exposure Control Policies and the University Handbook of Operating Procedures, Section 7.26.

E-RES Workout

Five to 10 full days after fitness testing, individuals returned for the exhaustive resistance exercise stimulus (E-RES) workout. Participant scheduling for this workout was determined by their availability and subjects' arrival times varied greatly, between 8 am and 5 pm. An effort was made for individuals to arrive within +/- 1 hour of their first visit time. After their arrival, they received a bottle of water to drink, completed questionnaires, were briefed again on the protocol, had a blood draw (for about 30% of subjects), and warmed-up on an electronically braked bicycle (50 watts at 60 rpm). Criterion recovery measures (MIF, squat jump, Texas power bike) were conducted. Then they were allowed to stretch their lower bodies while they observed a demonstration of the workout technique. After putting on a heart rate monitor and answering single-item

affect measures, they completed the E-RES workout. Criterion recovery measures (MIF, squat jump, Texas power bike) were recollected just after the E-RES, plus every 20 minutes for 1-hour post.

To control for the large effect of CHO intake on recovery and inflammation (Nieman et al., 1997 as cited in Pedersen & Toft, 2000), individuals were not allowed to eat for 3 hours beforehand. Each participant was provided a carbohydrate drink with electrolytes standardized to be equal to 0.715 grams of CHO per kg of body weight (approximately 195 kcal for a 70 kg person). This was given after the 1-hour post-measures were completed. Water was provided ad libitum.

Exhaustive Resistance Exercise Stimulus (E-RES) Protocol

Protocol Type. Ramping, then fatiguing resistance work

Muscle Group. Lower body (hips and legs)

Apparatus. Plate-loaded, 45 degree Cybex leg press (feet placement on platform standardized to same as MIF assessment and vertical jump)

Muscle Contraction Speed. 3 seconds eccentric/2 seconds concentric contraction speed with a 1-second isometric hold (60 seconds total for 10 rep sets). Cadence was paced with a pocket metronome.[25]

Load, Intensity, and Volume: Ramping Phase. Workload increased incrementally from 20.45 kg (warm-up) to load at which 10 repetitions cannot be volitionally completed (muscular failure, e.g., a 10-RM test). See Table 3.2.

Load, Intensity, and Volume: Burnout Phase. Approximately 200-300 repetitions altogether

Set 1: 10-RM capacity load to volitional exhaustion

[25] A pilot study conducted with 28 individuals was conducted to determine contraction speed. The specific speed selected minimizes the use of momentum, minimizes stress on the lower back, maximizes safety, and reliably produces muscular soreness and damage post-exercise.

Set 2: 90% of 10-RM to volitional exhaustion

Sets 3-6: If 10+ repetitions can be completed in last set, then remain at the same load, but

if < 10 repetitions, then 80% of 10-RM.

The last four sets were also to volitional exhaustion.

Rest Period (Duty Cycle). 120-second rest periods (1:2 duty cycle).

Warm-up. A treadmill warm-up was performed at 2.2 mph for 5 minutes or cycling at 50 watts for 60 RPM for five minutes. Participants performed 6 sets of stretches (2 for quads, 2 for hamstrings, 2 for lower back) for 30 seconds each (Drury, Stuempfle, Mason, & Girman, 2006). Resistance warm-up sets were included in the leg press protocol (see above).

Motivation. Participants were given verbal encouragement to complete the protocol from the test administrator(s).

Table 3.2 Ramping phase of E-RES workout

Set	1	2	3	4	5	6	7	8	9	- ->
Load	20.45 kg	30% 1RM	50% 1RM	60% 1RM	65% 1RM	70% 1RM	75% 1RM	80% 1RM	85% 1RM	- -> 1RM

Post-E-RES Workout (Visits 3-6)

Participants returned for four visits (24, 48, 72, and 96 hours post). Post-assessments for most participants were collected at a time of day +/- 3 hours as the E-RES workout. Upon arrival, they completed surveys for pleasure/displeasure (FS), activation/arousal (FAS), affect (PANAS), energy, fatigue, and soreness. Participants had a blood draw, completed assessments for maximal isometric force (MIF), squat vertical jump height, and maximal cycling power. Individuals rated muscular pain immediately after each MIF.

Dependent (Outcome) Variables

The primary dependent variables are related to functional muscular and psychological recovery. Functional muscular recovery was assessed through: (i) maximal isometric force (MIF), (ii) vertical jump height, and (iii) maximal cycling power. Psychological recovery was assessed through self-reports of perceived energy, perceived fatigue, and soreness.

Criterion Measures of Functional Recovery.

i. Maximal Isometric Force (MIF). MIF was determined on a modified leg press machine (45 degree, plate-loaded Cybex). Note that this leg press machine was not the one on which maximal leg press strength was measured. The machine was adjusted so that each individual was at a 110-degree knee joint angle and the sled fixed in place with adjustable attachments. An Omega LC101-3.0 k load cell was used with an Omega DMD 460 (115 v) amplifier/signal conditioner and a Measurement Computing USB-1208FS Analogue to Digital (A/D) Board. Data acquisition was accomplished with a Measurement Computing, DAS-Wizard 3.0 for MS Excel at 1,000 Hz for 3.0 to 4.0 seconds of maximal performance. Participants were given three attempts to push their feet maximally against the sled platform. The maximal data point was utilized for further analysis. Maximal force output (kg) for each trial was averaged. The correlation between visit 1 MIF and visit 2 pre-workout MIF was .946 and test-retest reliability (α) was 0.972. The reliability (α) over all visits was 0.983. The inter-item correlation mean was .859 and the intra-class correlation was .849 for single measures and .983 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 = .790), vertical jump power (r = .599), and maximal cycling

power (r = .742; n = 55 for all correlations, p = .000 for all). See Figures 3.2 and 3.3.

Figure 3.2 Individual demonstrating set-up for Maximal Isometric Force (MIF) measurement.

Figure 3.3 Detail of MIF apparatus. Notice turnbuckle, black strap, and orange ratchet tie-down. Lines around platform of sled and along frame provide reference for positioning of feet on platform and angle of knee.

ii. Vertical Jump Height and Power. Vertical jump power was determined from a squat jump measured with a Vertec apparatus (Sports Imports, Inc., Columbus, OH). This method calculates the difference between standing reach height and peak jump height. Power was calculated from the equation: Peak Power (Watts) = (60.7) x (jump height [cm]) + 45.3 x (body mass [kg]) – 2,055 (Sayers, Harackiewicz, Harman, Frykman, & Rosenstein, 1999). Positioning of the individual relative to the Vertec was standardized. Feet placement was exactly in the same dimensions as foot placement on the platform of the isometric force apparatus. Participants started in a crouched position (90 degrees at the knee and hip) and were instructed not to use a counter-movement. Participants were given three trials per collection period to achieve peak maximal jump height. Squat jump is a highly reliable measure of recovery (Garcia-Lopez et al., 2006; Mair et al., 1995). The reported test reliability is r = .985, with an objectivity of .981. The correlation between visit 1 vertical jump height and visit 2 pre-workout jump height was .972 and test-retest reliability (α) was 0.986.

iii. Maximal Cycling Power. Cycling power was determined from the Texas power bike. The bike seat was adjusted so that the knee bends were between 10 and 20 degrees. Participants started with the right crank arm of the bike parallel to the crossbar and cycled maximally for 33 revolutions of the flywheel. The power bike uses an optical sensor to determine velocity of the flywheel. Sixteen data points were collected per revolution. Participants were given three trials for each collection period to achieve maximal power, which takes about 4 seconds per attempt. The correlation between visit 1 cycling power and visit 2 pre-workout cycling power was .950 and test-retest reliability (α) was 0.974.

Criterion Measures of Psychological Recovery (all paper/pencil).

i. Fatigue and Energy. The fatigue and energy measures were the Visual Analogue Scales (VAS) developed by O'Connor (2006). Respondents placed a mark on 12 standard 10-cm lines that asked about both the physical and mental aspects of energy and fatigue. This measure has been validated with resistance exercise (Herring & O'Connor, 2008; O'Connor, 2008, personal correspondence). Chronbach α reliability for the state scales range from 0.89 to 0.91. Examples of anchors include "I have no energy" to "strongest feelings of energy ever felt."

ii. Soreness. 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-workout soreness was .576. The test-retest reliability (α) over these two visits was 0.728. The test-retest reliability (α) overall measurements (both the first hour of recovery and 4 days of recovery) was 0.806 and the average inter-item correlation was .270. The intra-class correlation was .269 for single measures and .786 for average measures (consistency index type; two-way mixed effects model where people effects are random and item effects are fixed).

iii. Positive and Negative Affectivity (PANAS). Negative affectivity (NA), an alternative measure of mood, was measured with the Positive and Negative Affect Schedule (Watson, Clark, & Tellegen, 1988). Unlike the Feeling Scale (FS, see above), which asks respondents to rate their affectivity as either negative or positive, the PANAS is useful for detecting the degree to which respondents are high or low on each. The PANAS asks respondents to rate themselves on 10 different negative and positive emotions with instructions to make ratings "right now, that is, at the present moment." Examples of NA items include guilty,

nervous, or jittery. Each emotion is rated on a 5-point Likert scale with the anchor at 1 being "very slightly or not at all" and the anchor at 5 being "extremely." A score was created for negative affect only. Affectivity shows high reliability, internal consistency, and discriminate validity with perceived stress. NA is correlated with the PSS scale (r = .44; Watson, 1988). Two studies have determined that there is no relationship between self-reported exercise behavior and NA (there is a relationship for PA, however; Watson, 1988; Watson & Pennebaker, 1989).

Measures of Psychological Response During the Workout

i. Muscular Pain during Movement. The pain measure was a category/ratio scale developed and validated by Cook and colleagues (Cook et al., 1997; Cook, O'Connor, Oliver, & Lee, 1998). The scale has 12 responses with 0 being "no pain at all," 5 being "somewhat strong pain," and 10 being "extremely intense pain (almost unbearable)." The scale has high reliability (ICC = 0.88-0.98) and is highly reproducible with different samples performing the same test (Cook et al., 1997, 1998). Pain ratings are highly related to objective measures of power output. VAS measures of pain on a standard 10-cm line ('no pain' to 'worst possible pain') are correlated with this measure of pain (.74-.94). Detailed written instructions for the participants were adapted from Cook et al. (1997).

ii. Pleasure/Displeasure. Affective valence, as measured from the Circumplex Model, is an effective measure of pleasure/displeasure during exercise (Ekkekakis & Petruzzello, 1999; Hall, Ekkekakis, & Petruzzello, 2002). The Feeling Scale (FS; Hardy & Rejeski, 1989) was used as a single-item measure of affective valence. This is an 11-point bi-polar measure ranging from -5 to +5. The anchors include "very bad" at -5 to "neutral" at 0 to "very good" at +5. The FS exhibits

correlations ranging from .51 to .88 with the valence scale of the Self Assessment Manikin (SAM; Lang, 1980) and from .41 to .59 with the valence scale of the Affect Grid (AG; Russell, Weiss, & Mendelsohn, 1989).

iii. Activation/Arousal. Perceived activation was measured with the Felt Arousal Scale (FAS) of the Telic State Measure (Svebak & Murgatroyd, 1985). This is a single-item self-report measure used extensively in exercise research (Hall et al., 2002; Kerr & Van den Wollenberg, 1997; Kerr & Vlaswinkel, 1993). This 6-point scale ranges from 1 to 6 with anchors including "low arousal" at 1 to "high arousal" at 6. Correlations of the FAS with the SAM arousal scale range from .45 to .70. Correlations with the arousal scale of the AG range from .47 to .65.

iv. Rating of Perceived Exertion (Omni).

Rating of perceived exertion (RPE) was measured with the Omni Scale for resistance exercise (Lagally & Robertson, 2006). Instructions were followed in line with advice from Borg (1998).

Cytokines. Blood specimens were collected with vacutainers with sodium citrate (BD Corporation, Franklin Lakes, NJ). Whole blood was centrifuged at 3,000 rpm at 4 degrees Celsius on a Sorvall RC6 (HS4 swing-out rotor) for 10 minutes. Plasma was extracted being careful not to disturb the buffy coat. Plasma was immediately frozen in microcentrifuge tubes at -80 degrees Celsius. Later, plasma for 39 samples (3 time points for 13 participants) was thawed for two hours. This plasma was filtered with Millipore sterile, 0.22 micron, low-protein binding, ultra-free centrifugal filter units (Millipore Corp, Bedford, MA). Plasma was re-centrifuged in a Sorvall 1500 with a fixed-angle rotor at 9,000 rpm for 4 minutes. IL-1β, TNF-α, and IL-6 were analyzed with a Bio-Plex cytokine multiplex assay kit (Bio-Rad Laboratories, Hercules, CA). Analysis for

cytokines was completed on a Bio-Plex Suspension Array System and values were generated using Bio-Plex Manager software.

Table 3.3 Duty cycles and testing schedule for leg press exercise stimulus protocol. Work was for 60 seconds. Rest was for 120 seconds.

	Pre	W	Rest	W	Rest	W	Rest	W	Rest	W	Rest	W	Rest	W	Rest	W	Rest	W	Rest	W	Post 1	Post 5	Post 20	Post 40	Post 60
Panas	•																				•				•
RPE			•				•				•				•				•		•	•	•	•	•
Pleas./ Displ.	•		•				•				•				•				•		•	•	•	•	•
Activ- ation	•		•				•				•				•				•		•	•	•	•	•
Energy	•		•				•				•				•				•		•	•	•	•	•
Fatigue	•		•				•				•				•				•		•	•	•	•	•
Soreness	•		•				•				•				•				•		•	•	•	•	•
Pain	•		•				•				•				•				•		•	•	•	•	•
Heart Rate	•		•				•				•				•				•		•	•			
MIF		•																				•			•
Jump Height		•																				•			•
Cycling Power		•																				•			•
Blood Draw	•																				•				

Statistical Analysis

As mentioned above, the ideal method for this analysis would be to run stress measures as continuous instead of dichotomous variables. The effects of stress are most pronounced at the tails of the distribution, and typically, when recruiting a small to medium number of participants, it is possible that the variance in a given stress measure may be limited. I oversampled at the extremes in order to detect an effect with a manageable number of participants. Participants may regress to the mean, so I surveyed for perceived stress on three more occasions and conducted K-S tests to determine normality of second visit PSS scores.

It was decided a priori that if regression to the mean was not problematic, participants would be coded into two groups, high and low stress. However, if regression was detected, then I would run analyses with stress run continuously.

Descriptive statistics (means and standard deviations) were calculated for each measure for the high- and low-strain groups based on the median split of PSS scores at the first visit. A Pearson's correlation was calculated between the PSS-10 and USQ according to the recommendation by Semmer et al. (2004). Designated groups (high vs. low perceived stress as the fixed factor) were compared in regards to depression, negative affectivity, perceived stress at time 1, mean perceived stress over the course of the experiment, and life event stress to ensure that groups differed on mental stress and strain using a one-way MANOVA. Because chronic stress may have a relationship with measures of physical fitness (Ensel & Lin, 2004), these groups were also compared in regards to fitness (resting heart rate (RHR), VO_{2peak}, bench press and leg press 1-repetition maxes (1-RMs), vertical jump peak power, maximal cycling power, maximal isometric force (MIF), indices of body composition and anthropometrics, exercise

behavior (kcals of physical activity), and indices of workload completed (e.g., repetitions, total weight lifted) using a one-way MANOVA with stress as the fixed factor.

Repeated measures ANOVA were utilized to compare groups in disruption from homeostasis (see hypothesis 1a, 2b-2d). A significant 2 x 2 (stress x time) factorial repeated measures ANOVA were used to compare groups on reductions in MIF, vertical jump height, and Texas power bike power. A 2 x 2 (stress x time) analysis was used to test the hypotheses (2b and 2d) that higher stress individuals have greater fatigue and reductions in energy immediately post-exercise.

In addition to nomothetic analyses, multi-level (hierarchical) analyses were used as recommended by Morton (2005) and Van Eck et al. (1996) to test hypotheses 1b and 2a. HLM is similar to regression where a single outcome is regressed onto predictors. This analysis allows 2 or 3 random factors (people and places) to be modeled with an unlimited number of predictors. An individual's observations (level 1 data) were nested within the individual (level 2 data). Each random factor has its own variance component. A two-level (observations within persons) hierarchical linear modeling growth curve (HLM) analysis was used to detect differences in individual recovery trajectories for maximal isometric force, peak jump height, and cycling maximal power (Raudenbush & Bryk, 2002, pp. 185-186). Simple intercepts-and-slopes-as-outcomes analyses were conducted to determine the functional form of time in HLM for each variable's recovery curve.

Linear, quadratic, and exponential (cubic) functions were modeled to find best functional form of time for further analyses. Functions of time significant below a *p*-value of .05 were retained for further moderation analyses. When modeling two of more functions of time, it was necessary to fix the variance for at least one functional form of

time in accordance with HLM procedures for growth curve modeling. Consequently, these parameters had a higher number of degrees of freedom (*df*) associated with them.

To detect significant effects, > 30 individuals were included in the analysis (approximately ½ low stress and ½ high stress). This analysis, though complex, was desirable for several reasons:

a) to decrease the chance of committing a Type I error by modeling error at the level of the observations (level-1) and the individual (level-2),
b) to use all data points and not just group means,
c) to allow the data to have any type of functional form (linear, quadratic, or cubic),
d) to pinpoint exact time points (e.g., 48 hours post) where groups may differ by recoding for the intercept,
e) to allow for missing data in case an individual misses one or two measurements, and
f) to easily test and diagram potential moderators.

For these reasons, the HLM analysis was considered the most appropriate method of analyzing these data (Morton, 2005; Raudenbush & Bryk, 2002).

Stress and strain measures were run as linear/continuous variables if they meet normality requirements as determined by Kolmogorov-Smirnov tests. Because it was not possible to schedule participants at the exact same time every day of the recovery period, hours awake and hours-out-of-bed were modeled as person-centered level-1 variables. It was determined a priori that if these variables were significant at a p-value < .05, they would be retained in the final multi-level models. Finally, stress and strain measures that were significantly related to the recovery trajectories were adjusted for covariates that had a significant relationship with muscular function (hours awake, muscular fitness, amount of fat-free mass (FFM, kg), finals period (dichotomous), workload (total kg),

magnitude of disruption from the E-RES workout (decimal), and semesters taking the weight-training class (continuous).

Power analyses based on effect sizes (Cohen's D and squared multiple correlation) from Perna and McDowell (1995) and Glaser et al. (1999) were conducted to determine sample size. With an effect size of .50, p set to .05, and a desired power set to .80 or greater, fewer than 30 subjects were needed (Lenth, 2001, 2006-2009). Due to volitional study incompletion, physical inability to complete the leg press protocol, voluntary withdrawal, and loss of follow-ups, it was the goal of the investigator to have 40 subjects participate in the study (approximately 20 men and 20 women). This is considered acceptable, as the goal is to balance quality of the manipulation with practicality of the protocol.

Table 3.4 Dissertation timeline.

	S/O '07	N '07	D '07	Ja '08	F '08	M '08	Ap '08	M '08	Jn '08	Jl '08	A '08	S '08	O '08	N '08	D '08	Jn '09	F '09	M '09	A '09	M '09	Jn '09	Jl '09	A '09
Tabulate / explore existing stress data	X	X	X																				
Proposal Writing Class	X	X	X																				
E-RES workout pilot study			X	X	X	X	X																
Proposal Second/Third Drafts				X	X	X																	
Proposal Presentation							X																
Create MIF machine								X	X	X	X	X											
IRB write-up (Prim/Secondary)								X	X	X	X	X											
Recruitment of subjects (pilot & non-pilot)			X	X	X	X							X	X			X	X					
Data Collection (Main study)													X	X	X		X	X	X				
Data Analysis	X				X	X	X									X	X			X	X	X	
Write Abstract for NASPSPA															X	X							
Present Preliminary Results																					X		
Write Discussion																					X	X	
Complete Dis. & Oral Defense																						X	
Revisions &Submission Deadline																							X

CHAPTER FOUR

RESULTS

INTRODUCTION AND RECAP OF METHODS

This study explored the relationship of various indices of stress and strain on the recovery of muscular and psychological function after a very vigorous and novel bout of lower body exercise (exhaustive resistance exercise stimulus, or E-RES). Weight training students were recruited to complete a short psychological screening (the PSS and CES-D) online. Those who met all qualifications and completed the study were 31 undergraduate students enrolled full time in studies and participating in a well-designed physical education, weight-training class (Beckwith, 2008). After refraining from moderate to vigorous activity (MVPA) for 2 days, participants were assessed for physical fitness, anthropometric, body composition, and psychological measures. Blood draws were completed for over half the participants (analyses were performed for 13). The lag time from screening, to scheduling, to the first visit was in the range of 1 to 3 weeks. Participants returned 7 to 10 days after the first visit, typically on a Sunday or Monday, for the E-RES workout, again after refraining from MVPA. To determine the potential effects of stress on recovery, trajectories of change were created for maximal isometric force (MIF), vertical jump height, maximal cycling power, perceived physical energy, perceived physical fatigue, and soreness. The time frame for these trajectories started at the first hour after the E-RES workout and included values for every 24 hours over the next 4 days.

Summary of Experimental Design and Statistical Analysis

The ideal method for this analysis was running stress measures as continuous (instead of dichotomous) variables. The challenge with this approach was that the effects of stress are most pronounced at the tails of the distribution, and typically, when recruiting a small to medium number of participants, it is possible that the variance in a given stress measure may be limited. Therefore, it makes sense to oversample at the extremes in order to detect an effect with a manageable number of participants. A dilemma that emerges when utilizing this procedure, however, is that participants may regress to the mean. In particular, this may be a problem when there is a significant amount of time (2 to 4 weeks) between screening and laboratory visits. Nevertheless, considering the necessity of ensuring enough variance, the practicality of this approach must be balanced against the chance of regression to the mean. Thus, I selected a tool to screen potential participants in advance of study participation. It was decided a priori that if regression to the mean was not problematic, participants would be coded into two groups, high and low stress. However, if both regression and normality was detected for the stress measures (as determined by K-S tests), then I would run analyses with stress run continuously.

Stress and strain measures were modeled as moderators of the outcome–time trajectory. Because recovery curves are likely curvilinear, I conducted analyses to determine the functional form of time (linear, quadratic, cubic, and/or logarithmic). These are important because they (a) confirm that the recovery slopes were significant across 4 days and (b) provide a starting point from which further modeling of the stress and recovery interactions may take place. For hypothesis testing, I decided a priori to run analyses regardless of whether the variance was significant after incorporating the effect of time on the outcomes.

Results are presented first for life event stress (USQ) and then for perceived stress (both PSS at the first visit and mean PSS, when appropriate). USQ does not measure the individual experience or impact of stressors, but PSS does measure this aspect of stress. Thus, both measures were utilized. Because USQ and PSS both increase linearly over the course of an academic semester and tend to peak at finals, I also coded for this period and analyzed finals as a dichotomous variable. Table 4.1 presents a summary of the means and percent reductions for the three measures of muscular and psychological function over 4 days (baseline, disruption and recovery values). Table 4.2 includes additional data for a median split of PSS at the first visit.

In this chapter, the results are presented in the following sections: (1) descriptive statistics and preliminary analysis, (2) hypothesis testing, and (3) exploratory analysis.

Table 4.1 Baseline, disruption and recovery values for muscular and psychological function 4-day post-workout (means and % change).

	BASE	Post 0 Min	1 hour	24 hours	48 hours	72 hours	96 hours
MIF (kg)	293.0	162.8 (-44.4%)	242.7 (-17.2%)	267.8 (-8.6%)	286.6 (-2.2%)	275.7 (-5.9%)	291.1 (-0.6%)
Jump height (cm)	43.5	35.6 (-18.2%)	N/A	43.0 (-0.9%)	44.6 (+2.5%)	44.8 (+3.1%)	44.9 (+3.2%)
Cycle power (watts)	1342.7	1119.7 (-16.6%)	N/A	1317.1 (-1.9%)	1343.2 (0.0%)	1341.2 (+0.1%)	1408.7 (+4.9%)
Perceived physical energy	46.1	29.0 (-35.1%)	44.5 (-3.5%)	47.6 (+3.3)	47.0 (+2.0%)	52.6 (+14.1%)	63.6 (+38.0%)
Perceived physical fatigue	35.0	82.9 (+126.5%)	64.4 (+84.0%)	51.7 (+47.7%)	45.0 (+28.6%)	37.1 (+6.0%)	24.9 (-28.9%)
Soreness	21.4	69.7 (+208.4%)	69.7 (+225.7%)	69.4 (+224.3%)	62.4 (+191.6)	41.8 (+95.4%)	29.1 (+36.0%)

Table 4.2 Disruption and recovery for muscular and psychological function 4-day post-workout (means and % change).

	BASE	1 hour	24 hours	48 hours	72 hours	96 hours
MIF						
Low Stress	299.2	247.5 (-17.3%)	280.0 (-6.4%)	321.8 (7.6%)	288.1 (-3.7%)	298.6 (-0.2%)
High Stress	287.0	237.6 (-17.2%)	254.7 (-11.3%)	253.9 (-11.5%)	265.0 (-7.7%)	283.0 (-1.4%)
All	293.0	242.7 (-17.2%)	267.8 (-8.6%)	286.6 (-2.2%)	275.7 (-5.9%)	291.1 (-0.6%)
Jump height						
Low Stress	44.9	N/A	45.0 (+2.7%)	49.6 (+10.6%)	45.8 (+2.1%)	47.5 (+5.8%)
High Stress	41.8	N/A	40.4 (-3.3%)	38.6 (-7.8%)	43.9 (+4.9%)	41.1 (-1.8%)
All	43.5	N/A	43.0 (-0.9%)	44.6 (+2.5%)	44.8 (+3.1%)	44.9 (+3.2%)
Cycle power						
Low Stress	1340.7	N/A	1337.3 (-0.3%)	1385.2 (+3.3%)	1322.9 (-1.3%)	1455.8 (+8.6%)
High Stress	1344.9	N/A	1293.9 (-3.8%)	1301.2 (-3.3%)	1358.2 (+1.0%)	1357.8 (-1.0%)
All	1342.7	N/A	1317.1 (-1.9%)	1343.2 (0.0%)	1341.2 (+0.1%)	1408.7 (+4.9%)
Perceived physical energy						
Low Stress	51.6	41.7 (-19.2%)	46.8 (-9.3%)	53.8 (+4.3%)	52.8 (+2.3%)	68.3 (+32.4%)
High Stress	38.9	47.7 (+22.6%)	48.6 (+24.9%)	39.6 (+1.8%)	52.5 (+35.0%)	58.5 (+50.4%)
All	46.1	44.5 (-3.5%)	47.6 (+3.3)	47.0 (+2.0%)	52.6 (+14.1%)	63.6 (+38.0%)
Perceived physical fatigue						
Low Stress	29.2	63.9 (+118.8%)	48.9 (+67.5%)	40.1 (+37.3%)	28.7 (-1.7%)	17.6 (-39.7%)
High Stress	42.6	64.9 (+52.3%)	54.7 (+28.4%)	50.4 (+18.3%)	45.5 (+6.8%)	32.9 (-22.8%)
All	35.0	64.4 (+84.0%)	51.7 (+47.7%)	45.0 (+28.6%)	37.1 (+6.0%)	24.9 (-28.9%)
Soreness						
Low Stress	17.2	61.3 (+256.4%)	66.9 (+289.0%)	60.4 (+251.2%)	39.4 (+129.1%)	22.4 (+30.2%)
High Stress	26.6	78.6 (+195.5%)	72.0 (+170.7%)	64.5 (+142.5%)	44.2 (+66.2%)	33.8 (+27.1%)
All	21.4	69.7 (+225.7%)	69.4 (+224.3%)	62.4 (+191.6)	41.8 (+95.4%)	29.1 (+36.0%)

DESCRIPTIVE STATISTICS AND PRELIMINARY ANALYSES

Demographics and Finals Status

Gender. Nine females and 22 males completed the study. PSS at the first visit correlated with gender, indicating that females reported higher perceived stress (r = .376, p = .037). The USQ and gender correlation was not significant, though the relationship trended in the same direction with women reporting more life events (r = .314, p = .091). A MANOVA with gender as the fixed factor revealed that women were significantly higher in PSS (F = 5.606, df = 1, p = .025, η = 0.167), but not USQ (p = .098).

In regards to workload completion during the workout, women were able to complete more repetitions (F = 10.665, df = 1, p = .003; η = 0.269), but did not complete more absolute work (F = 2.402, df = 1, p = .132) or work relative to body mass (F = 0.035, df = 1, p = .853). A repeated measures ANOVA was conducted to determine if reduction in MIF was related to gender. This analysis revealed a time by gender interaction. Women had lesser reductions in MIF (F = 13.040, df (1, 29), p = .001, η = 0.310).

Ethnicity. PSS at the first visit correlated with being non-White, indicating that this group reported higher perceived stress (r = .454, p = .010). Being non-White also correlated with USQ (r = .588, p = .001). A MANOVA with non-White/White as the fixed factor revealed that non-Whites reported higher PSS (F = 7.722, df = 1, p = .010, η = 0.216) and higher USQ (F = 14.776, df = 1, p = .001, η = 0.345).

Age. Subjects ranged in age from 18 to 23 years (M = 20.26, SD = 1.34). Age was not linearly related to PSS (r = .169, p = .362) or to USQ (r = .149, p = .431).

Finals. Eleven individuals completed the study during finals (Fall 2008), while 20 completed the study in the time before finals (Fall 2008 and early Spring 2009). The

linear relationship between being in the academic finals period and various stress/strain measures is detailed in Table 4.4.

Diagnostics to Determine Scale of Measurement for Stress

Of the 31 participants who completed the study, 18 originally scored into the low-stress grouping and 13 scored into the high-stress grouping (see Figure 4.1). Examination of the distribution of scores demonstrated that there was a clear bi-modal pattern of stress scores with no overlap of groups. See Table 4.5.

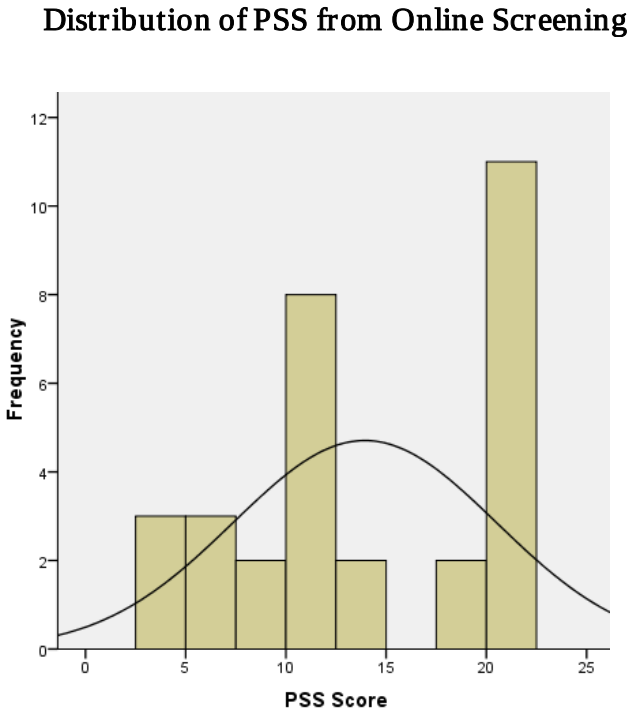


Figure 4.1 Distribution of PSS stress scores from online screening. Participants scoring between 14 and 18 were not eligible for the study.

However, the distribution of PSS scores measured at the first laboratory visit exhibited a different form. See Figure 4.2.

Distribution of PSS Scores at the First Visit

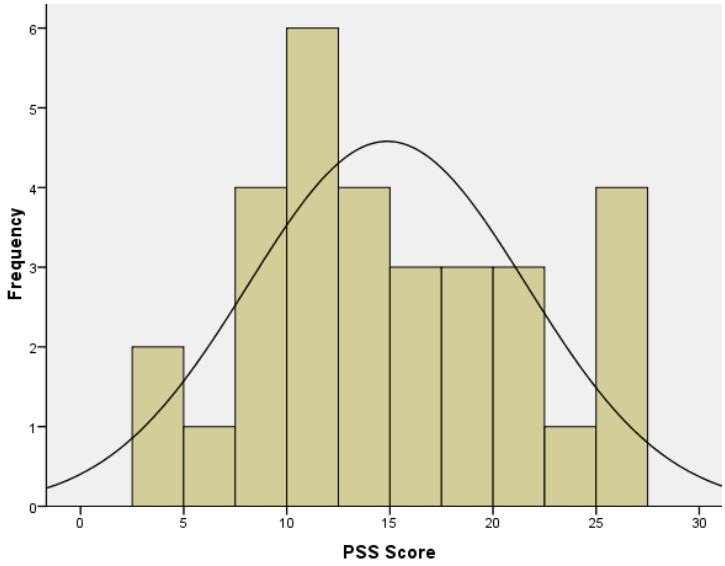


Figure 4.2 Distribution of PSS stress scores from the first visit.

To help determine the appropriate scale of measurement for stress, Kolmogorov-Smirnov (K-S) tests were conducted to determine normality of PSS scores from online and from the first visit. PSS from online did not meet normality criteria, as expected (K-S statistic = 0.199, $df = 30$, $p = .003$). However, the PSS score from time 1 was normal (K-S statistic = 0.117, $df = 30$, $p = .200$). K-S tests were also conducted for the mean PSS score (K-S statistic = 0.138, $df = 30$, $p = .151$) and for USQ (K-S statistic = 0.109, $df = 30$, $p = .200$). Hence, the strategy of using a categorical scale of measurement for stress was abandoned. Hypothesis testing was conducted with stress and strain measures as continuous, linear variables instead of stress groupings.

Collinearity of Stress/Strain Measures

PSS from online had a positive linear relationship with PSS at the first visit ($r = .747$, $p < .001$). PSS at the first visit also has a positive linear relationship with USQ,

which was also administered at the first visit (r = .594, p = .001). Academic finals was not related to USQ (r = .156) or PSS (r = .339). Therefore, it was determined that separate multi-level (HLM) analyses would be conducted for each measure of stress/strain. The relationship between various indices of stress/strain, finals, and measures of negative affect, energy, fatigue, and soreness is presented in Table 4.4.

Physical Fitness, Body Composition, and Anthropometric Characteristics

The current sample was similar in fitness to a cohort of 380 physical activity class students measured in our laboratory from 2004 to 2007 (Stults-Kolehmainen et al., 2007). My subjects were compared to this set of data for vertical jump power (watts), leg press 1-RM, and aerobic capacity (VO_{2max}). Both women and men in the current sample were similar for vertical jump power, leg press 1-RM, and aerobic capacity. See Table 4.6.

Four correlations analyzing relationships between stress/strain variables, body composition, and absolute physical fitness were significant. USQ was related to bench press 1-RM (r = -.376, p = .040), fat free mass (r = -.449, p = .013), and MIF (r = -.400, p = .028). Perceived stress was only related significantly to percent body fat (r = .360, p = .047). Overall, most of the correlations were negative (fat mass, percent body fat, and relative VO2max were exceptions). All of these measures were gauged at the first laboratory visit. See Tables 4.6 and 4.7.

Missing Data

There were missing data, which was not problematic for analyses.

Level-1 (Nested) Data. Data were missing for several variables on the level of the observation. For MIF and psychological function variables, 155 measures were possible. Missing data ranged from 9% to 12.3% for these variables. For jump height and maximal cycling power, 124 values were possible. Jump height had 20.1% missing data and cycling power had 12.9% missing information. Missing data for jump height were due to

temporary loss of the Vertec apparatus. One individual lacked vertical jump height data over the entire 4-day recovery period. Three individuals had missing data pre/post E-RES workout for both jump height and cycling power for the same reason.

There was a greater amount of missing data from the variables collected during the workout. Missing data ranged from 12.4% (for heart rate) to 29.5% for FS and FAS. This was considered acceptable because asking about pleasure, exertion, etc. at the end of each set may bias responses and be burdensome for the respondents. Furthermore, missing data are not considered a great limitation as HLM analyses are not unduly affected by missing data at level-1. See Tables 4.8 and 4.9 for level-1 descriptives for 4 days of recovery and variables during the workout.

Level-2 (Time Invariant) Data. Stress/strain data (USQ) were missing only for one subject out of the 31 that completed the study. PSS data were missing from the second visit for eight individuals (25%) and from the last visit for five individuals (16%). No participant was missing both PSS at the second visit and the last visit. As HLM analyses are not able to correct for missing data at level-2, these variables were not analyzed as moderators of the DV-time relationships.

Workload Completed E-RES Workout

Both USQ and PSS at the first visit were related to workload relative to body mass (for USQ, $r = .401$, $p = .028$; for PSS, $r = .401$, $p = .025$) and relative to FFM (for USQ, $r = .433$, $p = .017$; for PSS, $r = .461$, $p = .009$), and total repetitions (for USQ, $r = .469$, $p = .009$; for PSS, $r = .492$, $p = .005$). Stress was not related to absolute workload, peak heart rate, or heart rate over the last phase of the workout. See Table 4.7.

The Effect of the E-RES Workout (Time) on Functioning and the Sphericity Assumption

The main effect of time (pre/post E-RES workout) was significant for all variables ($p < .001$ for all except physical energy; $p = .005$ for physical energy), which indicated that all parameters of muscular and psychological function (MIF, jump height, maximal cycling power, perceived physical energy, perceived physical fatigue, and soreness) decreased pre- to post-workout. The assumption of sphericity was not violated for any analysis.

Examining the Functional Form of Time: Do Outcomes Change?

Visual examination of the trajectory confirmed that MIF, jump height, and cycling power increased with time. Tables 4.1 and 4.2 show percent change in these parameters over 96 hours of recovery. Energy increased with time, however, fatigue and soreness appeared to decrease with time. Feeling (pleasure) during the workout appeared to decrease, while pain and exertion appeared to increase. Activation visually appeared to have an inverted-u form.

Muscular Function. The functional form of time was the same for MIF and jump height, but not for maximal cycling power. For MIF, linear (ß =1.506, *SE* = 0.280, *t*-ratio = 5.382, *df* = 30, $p < .001$) and squared (quadratic) time (ß = -0.009, *SE* = 0.003, *t*-ratio = -3.337, *df* = 135, $p = .001$) provided the best fit. For jump height (cm), again linear (ß = 0.103, *SE* = 0.033, *t*-ratio = 3.165, *df* = 29, $p = .004$ and squared (quadratic) time (ß = -0.001, *SE* = 0.000, *t*-ratio = -2.309, *df* = 96, $p = .023$) provided the best fit. For maximal cycling power, squared (quadratic) time by itself provided the best fit (ß = 0.017, *SE* = 0.003, *t*-ratio = 6.253, *df* = 106, $p < .001$).

Psychological Function. As with cycling power, squared time provided the best functional form of time for physical energy (ß = 0.002, *SE* = 0.000, *t*-ratio = 5.212, *df* =

134, $p < .001$). Linear time provided the best fit for physical fatigue (ß = -0.355, *SE* = 0.047, *t*-ratio = -7.577, *df* = 30, $p < .001$). All forms of time were significant predictors of soreness, including linear time (ß = 1.057, *SE* = 0.267, *t*-ratio = 3.954, *df* = 30, $p = .001$), squared (quadratic) time (ß = 0.000, *SE* = 0.000, *t*-ratio = 3.386, *df* = 132, $p = .001$), and cubed (exponential) time (ß = -0.026, *SE* = 0.006, *t*-ratio = -4.357, *df* = 132, $p < .001$).

Variance Remaining. It was determined a priori to test models of stress-time interactions despite significance of the variance components from models of the effects of time. The variance components were examined to determine if variability was left over to model. Examination of the variance components associated with linear time revealed that remaining variance was not significant for MIF ($p > .500$), indicating a strong effect of time on this physical parameter. The MIF initial status variance (T_{00}) was linearly related to the variance (T_{10}) for the recovery slope ($r = .912$).[26] For jump height, variability was left over in the linear component for further modeling ($p < .001$). The variance associated with maximal cycling power was fixed to allow this model to converge. The variance associated with energy was fixed to allow the model to converge. The variance associated with fatigue was significant ($p = .038$), indicating that variance was left over to model other variables. To allow model convergence for soreness, squared, and cubed time parameters were fixed. Examination of the variance components associated with linear time revealed that variance approached significance ($p = .058$).

Affective Responses during the E-RES Workout. There was a main effect of time on feeling, with feeling decreasing quadratically over the course of the workout (ß = -0.023, *SE* = 0.005, *t*-ratio = -4.471, *df* = 26, $p < .001$). A positive exponential effect was also significant (ß = 0.001, *SE* = 0.000, *t*-ratio = 2.200, *df* = 282, $p < .029$). The linear term was not significant. All forms of time were significant predictors of exertion,

[26] Initial status is the first hour after E-RES workout for MIF.

including linear time (ß = 3.336, *SE* = 0.129, *t*-ratio = 25.927, *df* = 26, *p* < .001), squared (quadratic) time (ß = -0.346, *SE* = 0.017, *t*-ratio = -20.512, *df* = 26, *p* < .001), and cubed (exponential) time (ß = 0.011, *SE* = 0.001, *t*-ratio = 16.449, *df* = 296, *p* < .001). All forms of time were significant predictors of pain, including linear time (ß = 1.321, *SE* = 0.179, *t*-ratio = 7.383, *df* = 30, *p* < .001), squared (quadratic) time (ß = -0.088, *SE* = 0.020, *t*-ratio = -4.413, *df* = 30, *p* < .001), and cubed (exponential) time (ß = 0.002, *SE* = 0.001, *t*-ratio = 2.620, *df* = 340, *p* = .010). Significant predictors of activation (arousal) included linear time (ß = 0.253, *SE* = 0.093, *t*-ratio = 2.725, *df* = 26, *p* = .012) and squared (quadratic) time (ß = -0.029, *SE* = 0.010, *t*-ratio = -2.897, *df* = 26, *p* = .008). Cubed (exponential) time approached significance (ß = 0.001, *SE* = 0.000, *t*-ratio = -1.912, *df* = 281, *p* < .056).

Variance for the squared effect of time for feeling was significant (*p* < .001). The variance for linear time for exertion was significant (*p* = .001), but not for squared time (*p* = .118). The variances for linear and squared time for pain were significant (*p* < .001). Examination of the variance components for soreness associated with linear and squared time for activation revealed these variances were significant (*p* < .001 for both).

Relationship of Hours Awake to Muscular Function

Because participants performed laboratory testing in the 96-hour period following the workout at irregular times, a conditional model was run to determine the relationship between muscular and psychological function and hours awake. There was no relationship between these variables (*p* = .816 for MIF, *p* = .470 for jump height, *p* = .543 for cycling power, *p* =.494 for physical energy, *p* = .516 for physical fatigue, *p* = .790 for soreness). A similar analysis was run for function and hours out-of-bed (*p* = .565 for MIF, *p* = .739 for jump height, *p* = .586 for cycling power, *p* = .468 for physical energy, *p* = .407 for physical fatigue, *p* = .631 for soreness). Consequently, it did not appear that

these factors were directly related to muscular or psychological functioning and therefore were not further modeled.

Summary

Our sample was young and diverse and included an uneven number of men and women. Based on our diagnostics of regression to the mean, I decided to run stress as a continuous variable. The E-RES workout resulted in changes for all outcome variables during and after the exercise. I did not control for time of laboratory visit, however, since awakening and hours out of bed were not related to muscular or psychological functioning.

HYPOTHESIS TESTING

Research Questions

This investigation was primarily focused on the potential moderating effects of stress/strain on muscular and psychological recovery after a disruptive bout of resistance exercise. However, before this could be addressed, the relationship between stress and disruption from a difficult bout of resistance training had to be tested. Relationships between stress and disruption may account for any potential stress and recovery relationship. Likewise, if a relationship between stress and recovery exists, it may be due to other explanatory factors and not stress per se. Therefore, it was important to determine if stress and strain effects on recovery trajectories remained after adjusting for significant covariates, including hours awake, muscular fitness, amount of fat-free mass (FFM, kg), finals period (dichotomization), workload (total kg), magnitude of disruption from the E-RES workout (percent decrease in decimal form), and number of semesters taking the weight-training class.

Recaps of the hypotheses and then analyses are presented, first for life event stress followed by the analyses for perceived stress. Additionally, results are presented for

academic finals period, a naturalistic stressor. See Tables 4.1 and 4.2 for changes in muscular outcomes as a percent.

Hypotheses

Hypothesis 1a. In regards to functional muscular disruption, mental strain (e.g., USQ) will **not** predict changes in maximal isometric force, squat jump height, and maximal cycling power pre- to post-workout.

Hypothesis1b. In regards to functional muscular recovery, higher mental stain (e.g., USQ) will be related to deeper (lower) recovery slopes, and therefore, a **prolonged** return to baseline over 96 hours post E-RES workout in terms of maximal isometric force (MIF), squat jump height, and maximal cycling power.

Hypothesis 2a. In regards to psychological functioning, higher mental strain (e.g., higher USQ) will be related to greater mental disruption during the E-RES protocol (FS, FAS, RPE, pain).

Hypothesis2b. In regards to psychological functioning, mental strain (e.g., USQ) will predict decrements in perceived physical energy and perceived physical fatigue pre- to post-workout with higher strain related to greater disruption.

*Hypothesis2c. I*n regards to psychological functioning, mental strain (e.g., USQ) will predict increases in soreness (VAS scale) **immediately post**-E-RES workout (VAS scale).

Hypothesis 2d. In regards to psychological recovery, those higher in mental strain (e.g., USQ) will self-report lower recovery in fatigue and energy, as assessed by a return to baseline, 1-hour post-E-RES.

Hypothesis2e. In regards to psychological recovery, those higher in mental strain will self-report greater soreness (DOMP) in the 96-hour period post-E-RES protocol.

Life Event Stress (USQ)

Hypothesis1a. After holding the pre-workout value constant using a stepwise regression, life event stress (USQ) did not predict post-workout MIF (β = 0.027, t = 0.247, p = .807), jump height (β = -0.066, t = -0.548, p = .589) or maximal cycling power (β = -0.020, t = -0.132, p = .895).

Hypothesis1b. MIF: USQ moderated the MIF and time relationship for both linear time (p = .027) and squared (quadratic) time (p = .031). Higher stress values were related to deeper or lower recovery curves for this muscular parameter. There was no difference at the intercept (p = .128), which indicates that no differences existed at the end of the first hour of recovery after the E-RES workout. See Figure 4.3. Also, see Table 4.10. USQ moderated the MIF and time relationship for linear time (p = .032) and squared (quadratic) time (p = .021) after adjusting for covariates related to muscular functioning and recovery. The relationship after controlling for covariates was in the same direction as previously. See Table 4.11.

Jump height: USQ did not moderate the jump height and time relationship for linear time (p = .497) or for squared (quadratic) time (p = .391). There was no difference at the intercept (p = .312), which indicates that no differences existed at the end of the first hour of recovery after the E-RES workout. See Figure 4.4.

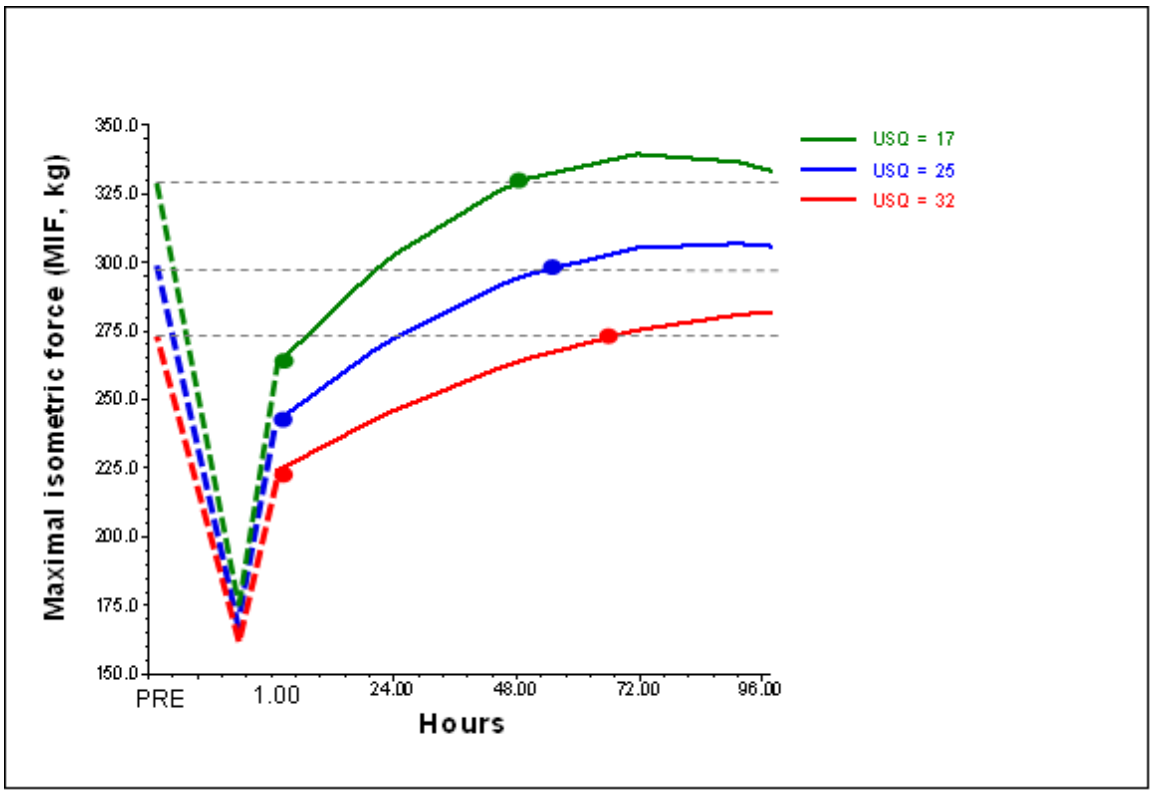


Figure 4.3 Relation of USQ to MIF recovery curves over 4 days. Time is modeled linearly and quadratically. Logarithmic time was not significant. Solid line indicates data analyzed with HLM. Dashed lines indicate level baseline values and level of disruption.

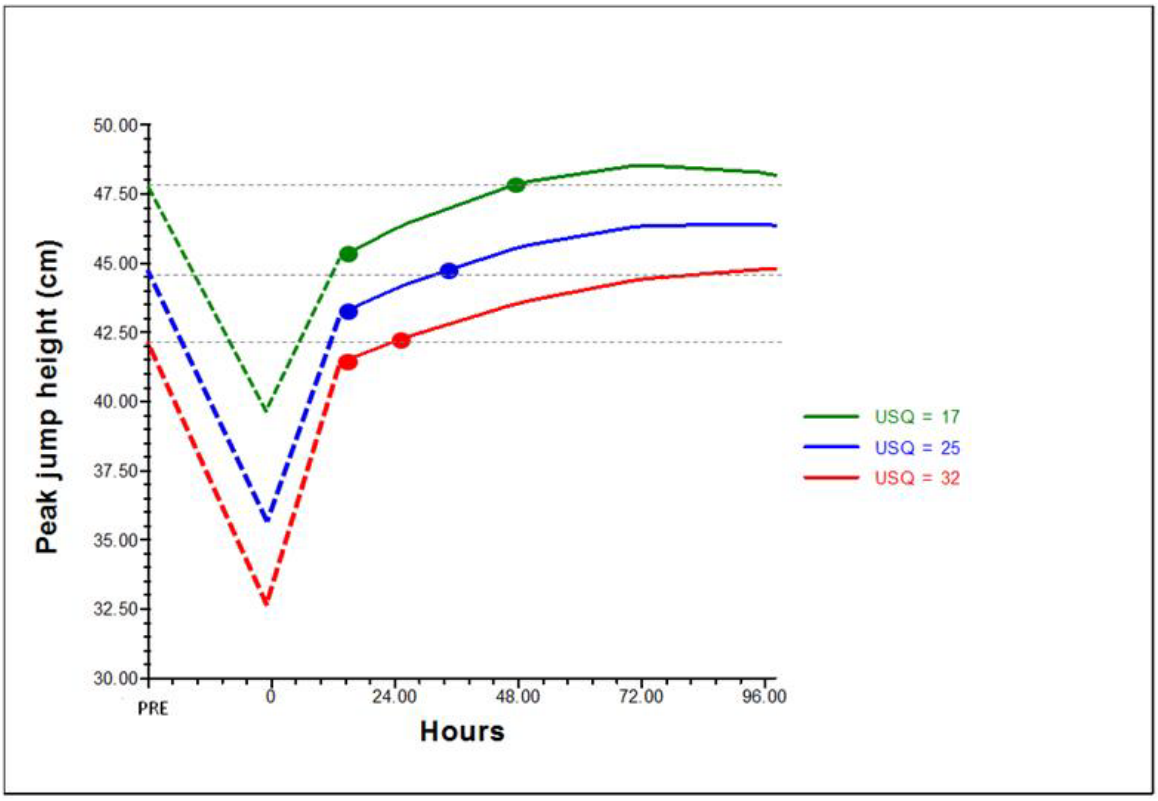


Figure 4.4 Relation of USQ to jump height recovery curves over 4 days. Time is modeled linearly and quadratically. Logarithmic time was not significant. Solid line indicates data analyzed with HLM. Dashed lines indicate level baseline values and level of disruption.

Cycling power: USQ did not moderate the linear time–cycling power relationship (p = .856). Stress/strain also did not moderate this relationship for squared time (p = .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 E-RES workout (p = .089). See Figure 4.5.

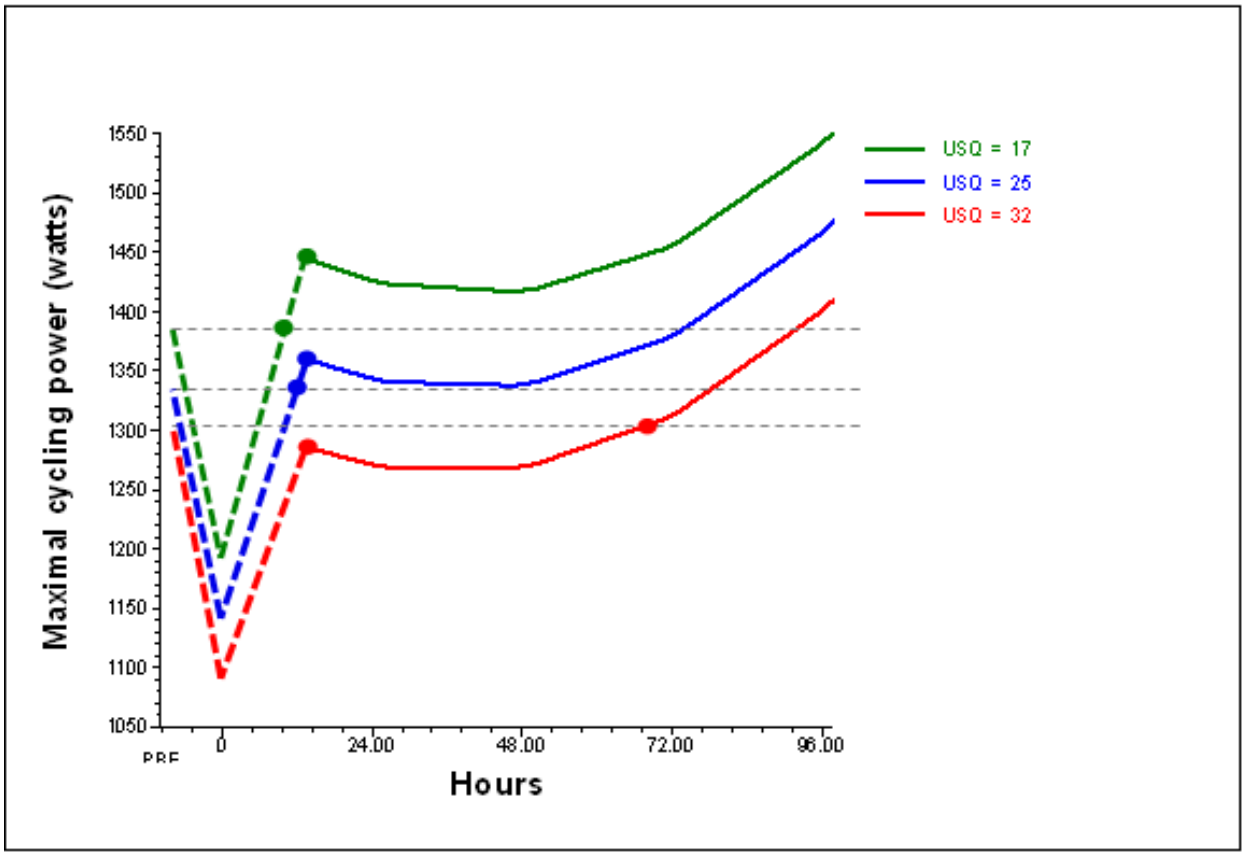


Figure 4.5 Relation of USQ to jump height recovery curves over 4 days. Time is modeled linearly and quadratically. Logarithmic time was not significant. Solid line indicates data analyzed with HLM. Dashed lines indicate level baseline values and level of disruption.

Hypothesis2a. Feeling (Pleasure): There was not a significant stress/strain by time interaction. Stress was not related to feeling for squared time (p = .255) or cubed (exponential) time (p = .406). The intercept approached significance, indicating that those higher in stress may be lower in feeling at the beginning of the workout (p = .065).

Activation (Arousal): There was no stress/strain moderation of the time–activation relationship for squared time (p = .326) or for cubed time (p = .932). The intercept was not significant (p = .693).

Muscular Pain: There was a significant stress/strain by time interaction. Those reporting higher stress had *lower* increases in pain. This held for squared time (β = -0.001, *SE* = 0.001, *t*-ratio = -2.132, *df* = 28, *p* = .042) and cubed time (β = 0.000, *SE* = 0.000, *t*-ratio = 2.395, *df* = 325, *p* = .017). The intercept, however, was not significant, meaning that there was no stress time relationship at the beginning of the workout (*p* = .696).

RPE: There was a significant perceived stress at the first visit by time interaction. Similar to pain, those reporting higher perceived stress at the first visit had lower increases in exertion over the workout. This effect held for squared time (β = -0.002, *SE* = 0.001, *t*-ratio = -2.341, *df* = 24, *p* = .028) and for cubed time (β = 0.000, *SE* = 0.000, *t*-ratio = 2.722, *df* = 281, *p* = .007). There was no relationship at the intercept (β = 0.044, *SE* = 0.027, *t*-ratio = 1.875, *df* = 24, *p* = .073). See Figure 4.6.

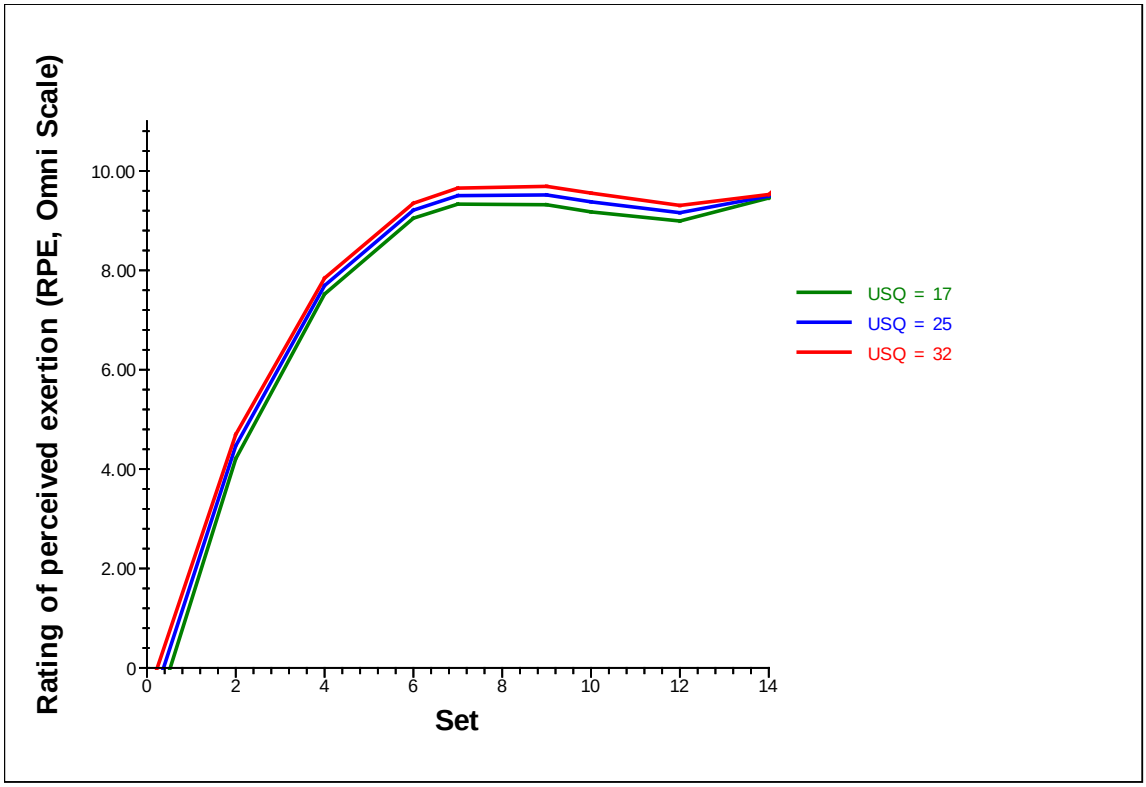


Figure 4.6 Rating of perceived exertion over the course of the E-RES workout.

Hypothesis 2b: In regards to psychological functioning, mental strain (e.g., USQ) will predict decrements in perceived physical energy and perceived physical fatigue pre- to post-workout with higher strain related to greater disruption.

After holding the pre-workout value constant using a stepwise regression, life event stress (USQ) did not predict perceived physical energy ($\beta = 0.180$, $t = 0.454$, $p = .654$) or perceived physical fatigue ($\beta = 0.210$, $t = 1.163$, $p = .255$).

Hypothesis2c. After holding the pre-workout value constant using a stepwise regression, life event stress (USQ) did not predict soreness ($\beta = 0.318$, $t = 1.805$, $p = .082$).

Hypothesis2d.

Perceived Physical Energy. There was not a relationship between stress and energy at 1 hour ($p = .764$). However, there was a linear time by stress interaction for the

recovery trajectories of energy over a 4-day period (p = .038). (This relationship did not have a formal hypothesis.) See Table 4.12 and Figure 4.7.

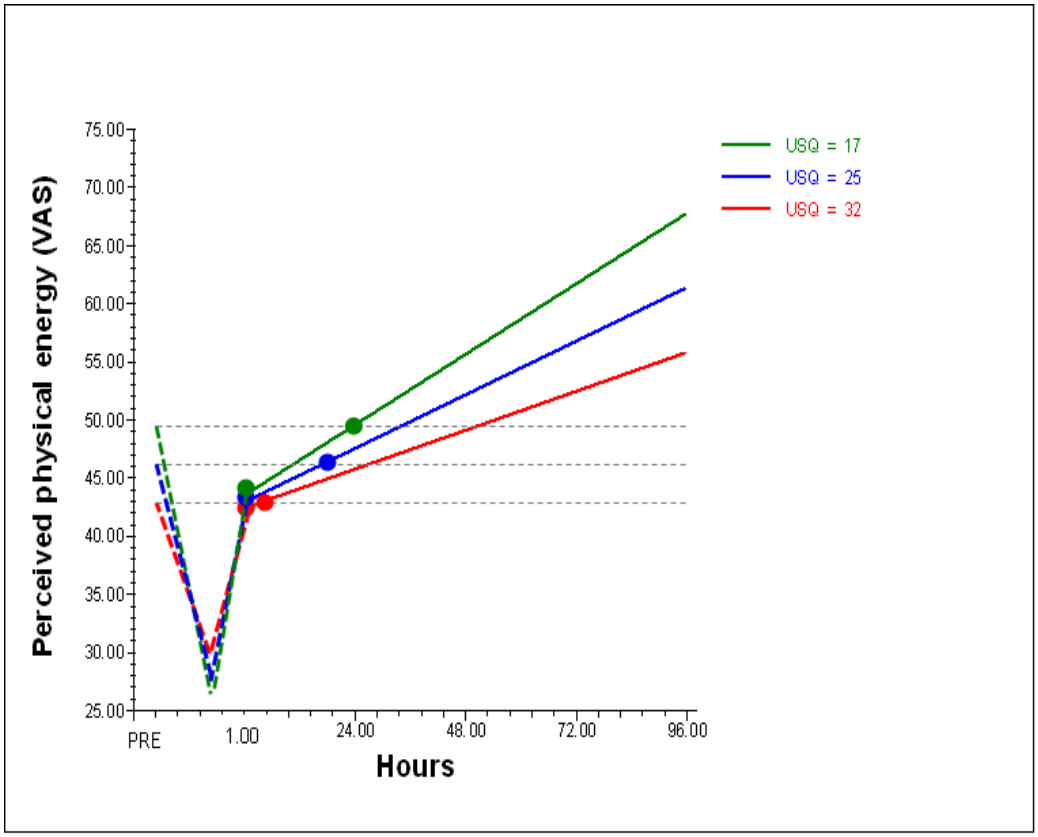


Figure 4.7 Perceived physical energy at baseline, disruption pre- to post- workout and rise over a 4-day recovery period.

Perceived Physical Fatigue. There was not a relationship between stress and fatigue at 1 hour (p = .866). However, there was a linear time by stress interaction for the recovery trajectories of fatigue over a 4-day period for life event stress (p = .040). (This relationship did not have a formal hypothesis.) Those with higher life stress and those who completed the workout during finals had higher recovery curves over 96 hours, which indicates that they decreased from their fatigue at a slower pace. See Table 4.13 and Figure 4.8.

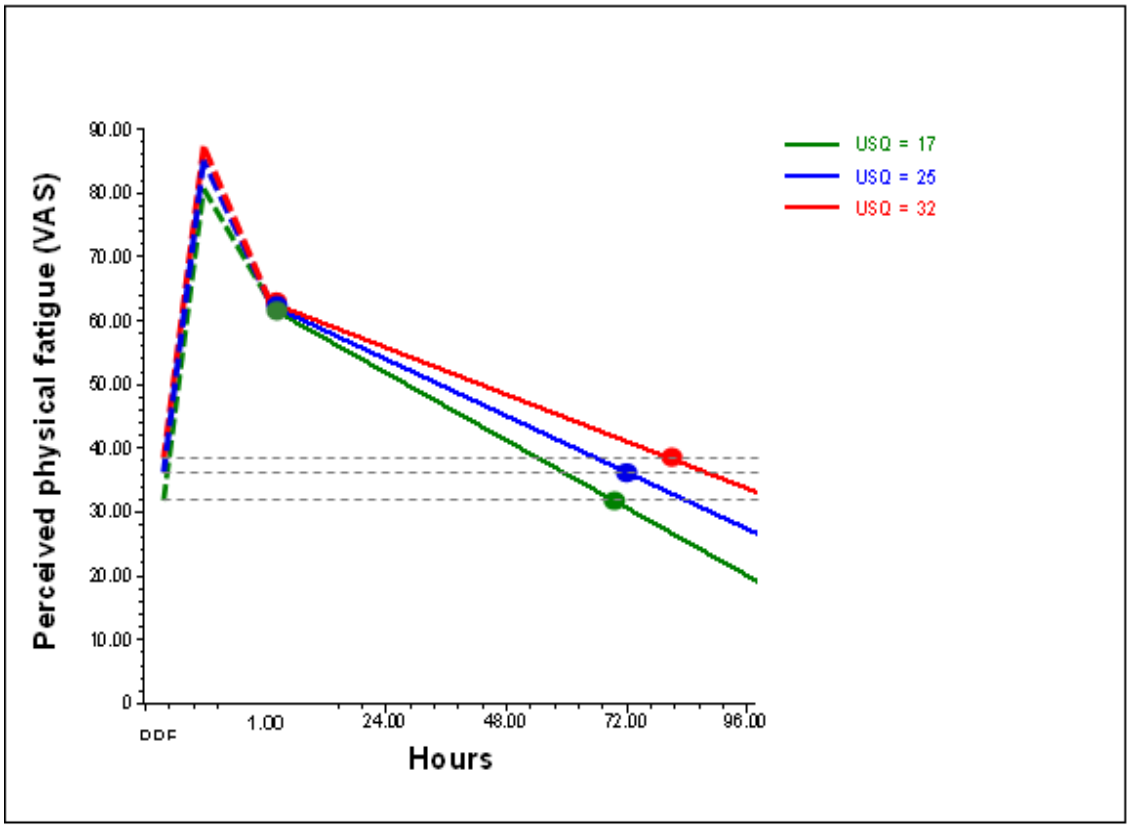


Figure 4.8 Perceived physical fatigue at baseline, disruption pre- to post- workout and rise over a 4-day recovery period.

Hypothesis 2e. Life events stress (USQ) was significantly related to the linear effect of time for soreness (p = .027) and approached significance for the squared (quadratic) effect of time (p = .052). Those with higher life event stress had a higher soreness trajectory over the 4 days. See Table 4.14 and Figure 4.9.

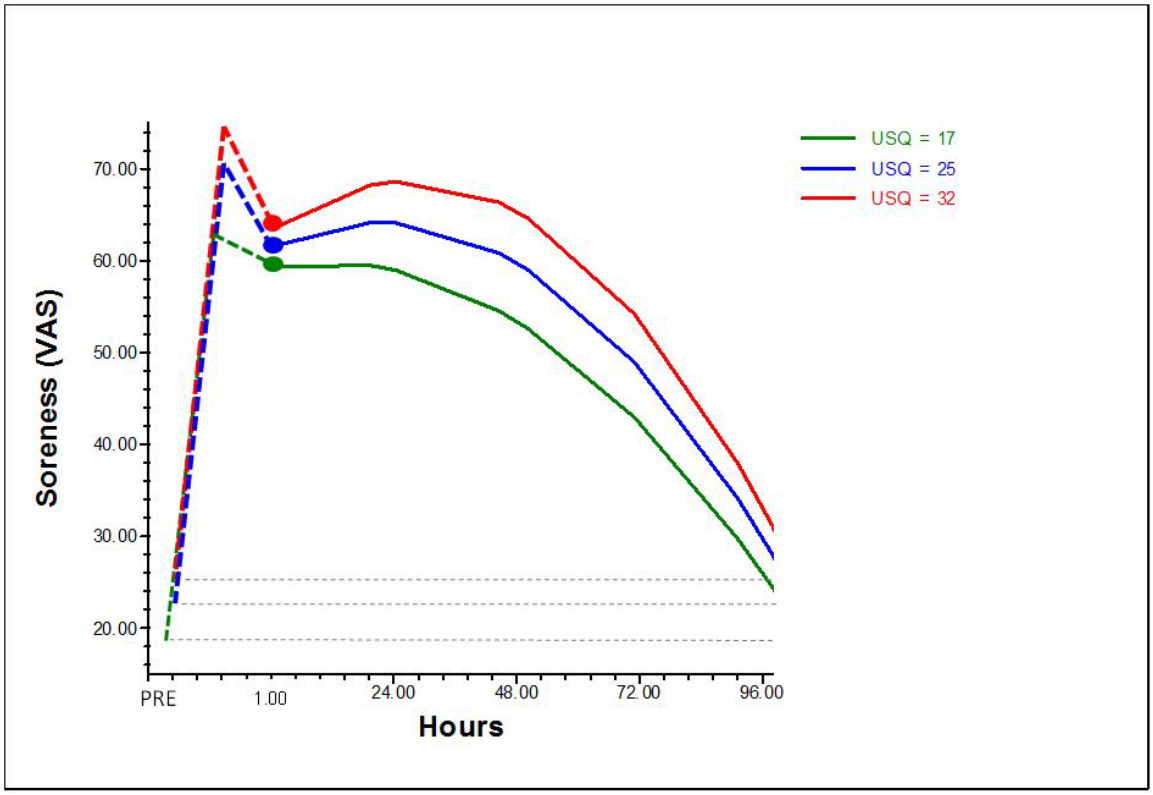


Figure 4.9 Soreness (pain) at baseline, disruption pre- to post- workout and rise over a 4-day recovery period.

Perceived Stress

For hypotheses 1a, 1b, 2d, and 2e, both perceived stress at the first visit and mean PSS (over four visits) were modeled as modulators of recovery trajectories. For all other hypotheses only perceived stress at the first visit was modeled.

Hypothesis 1a. Using the same statistical protocol, perceived stress (PSS) at the first visit did not predict post-workout MIF (β = 0.074, t = 0.506, p = .617) or jump height (β = -0.118, t = -1.113, p = .278). However, it did predict maximal cycling power (β = -0.316, t = -2.350, p = .028).

Hypothesis 1b. MIF: Stress/strain moderated the MIF and time relationship for both linear time (PSS at the first visit, p = .003; mean PSS, p = .001) and squared

(quadratic) time (PSS at the first visit, p = .002; mean PSS, p < .001). Based on examination of the raw data (Tables 4.1 and 4.2), the natural log of time was modeled for PSS at the first visit (p = .046) and for mean PSS (p = .018). Higher stress values for perceived stress values were related to deeper or lower recovery curves for this muscular parameter. There was no difference at the intercept (PSS at the first visit, p = .363; mean PSS, p = .379), which indicates that no differences existed at the end of the first hour of recovery after the E-RES workout. See Figure 4.10. Also, see Tables 4.15 through 4.17.

PSS moderated the MIF and time relationship for linear time (PSS at the first visit, p = .004; mean PSS, p = .001) and squared (quadratic) time (PSS at the first visit, p = .001; mean PSS, p < .001) after adjusting for covariates related to muscular functioning and recovery. The relationship after controlling for covariates was in the same direction as previously. See Tables 4.18 and 4.19.

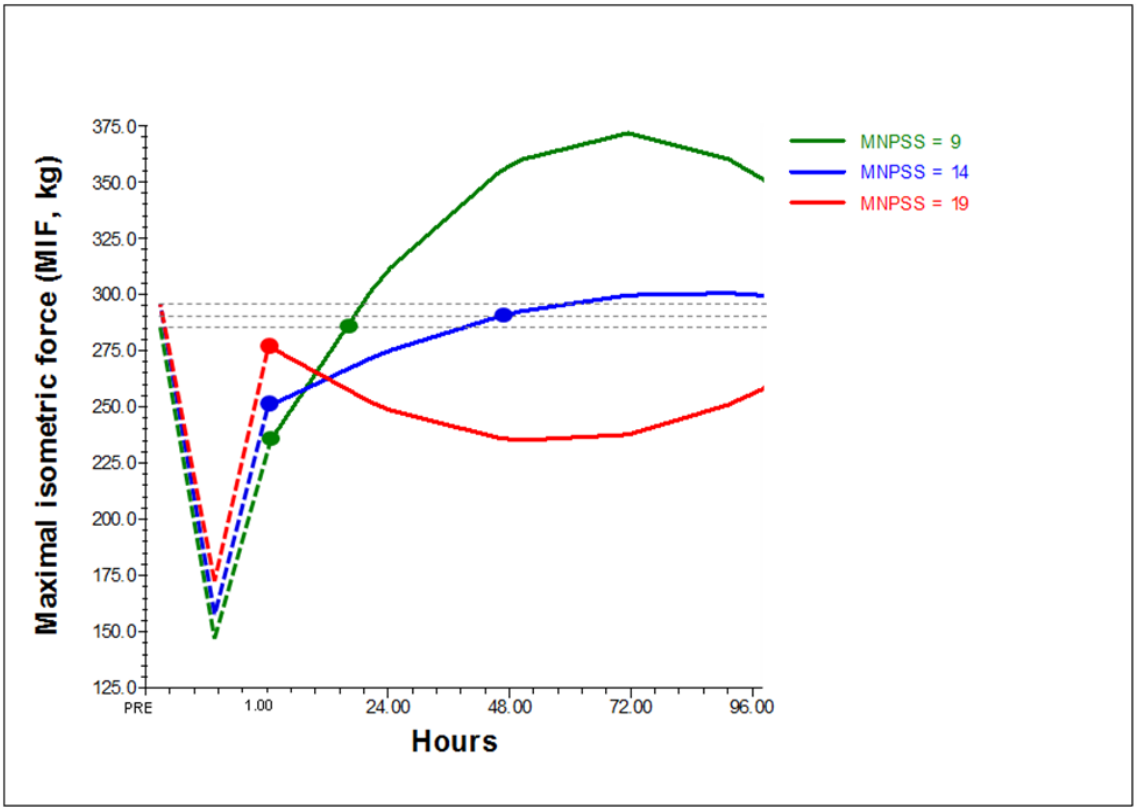


Figure 4.10 Relation of mean PSS to MIF recovery curves over 4 days. Time is modeled linearly and quadratically. Logarithmic time was not significant.

Jump height: PSS at the first visit did not moderate the jump height and time relationship for linear time (p = .094), but approached significance for squared (quadratic) time (p = .061). Mean perceived stress did moderate the jump height and time relationship for both linear time (p = .035) and squared (quadratic) time (p = .019). There was no difference at the intercept (PSS at the first visit, p = .391; mean PSS, p = .274), which indicates that no differences existed at the end of the first hour of recovery after the E-RES workout. Those reporting higher stress/strain values were lower or deeper in their recovery curves. See Table 4.20 and Figure 4.11.

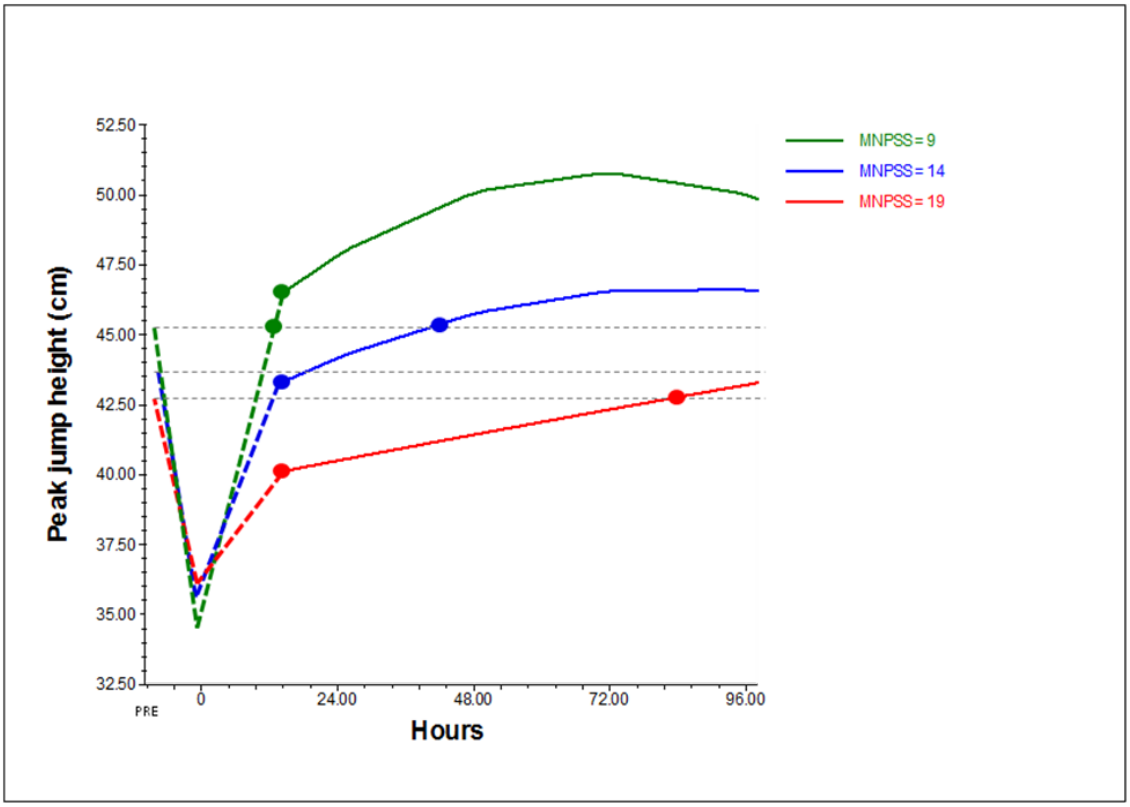


Figure 4.11 Relation of mean PSS to jump height baseline values, disruption pre- to post-workout, and recovery curves over 4 days.

Perceived stress moderated the jump height and time relationship for linear time (PSS at the first visit, $p = .035$; mean PSS, $p = .024$) and squared (quadratic) time (PSS at the first visit, $p = .028$; mean PSS, $p < .015$) after adjusting for covariates. The relationship after controlling for covariates was in the same direction as previously. See Table 4.21.

Cycling power: Stress did not moderate the linear time–cycling power relationship (PSS at the first visit, $p = .093$; mean PSS, $p = .289$). See Tables 4.1 and 4.2. Stress/strain also did not moderate this relationship for squared time (PSS at the first visit, $p = .083$; mean PSS, $p = .292$). 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 E-RES workout (PSS at the first visit, β = -23.622, *SE* = 11.291, *t*-ratio = -2.092, *df* = 29, *p* = .045;mean PSS, β = -26.022, *SE* = 13.146, *t*-ratio = -1.979, *df* = 29, *p* = .057).

Hypothesis 2a:

Feeling. There was a significant stress/strain by time interaction. Those reporting higher stress had greater decreases in feeling for squared time (*p* = .008) and for cubed (exponential) time (*p* = .014). The intercept was also significant, indicating that those higher in stress also were lower in feeling at the beginning of the workout (*p* = .011). See Table 4.22 and Figure 4.12.

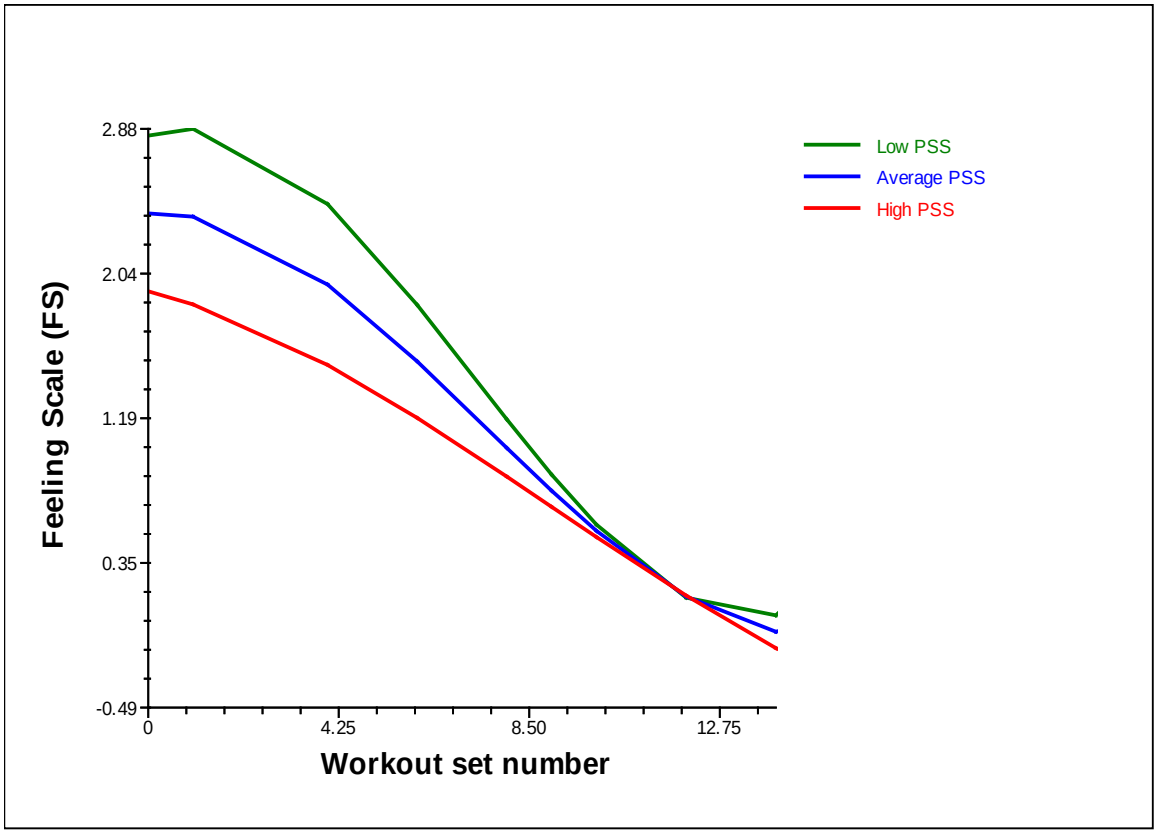


Figure 4.12 Relation of PSS at the first visit to changes in feeling during the E-RES workout.

Activation (Arousal). There was no stress/strain moderation of the time–activation relationship for squared time (p = .238) or for cubed time (p = .912). The intercept was not significant (p = .580). However, when the intercept was recoded to the end of the workout, perceived stress at the first visit moderated the squared (quadratic) effect of time for activation (p = .015) and the cubed effect (p = .034). Those higher in stress had higher a lesser decline in activation in the last third of the workout period. See Table 4.23 and Figure 4.13.

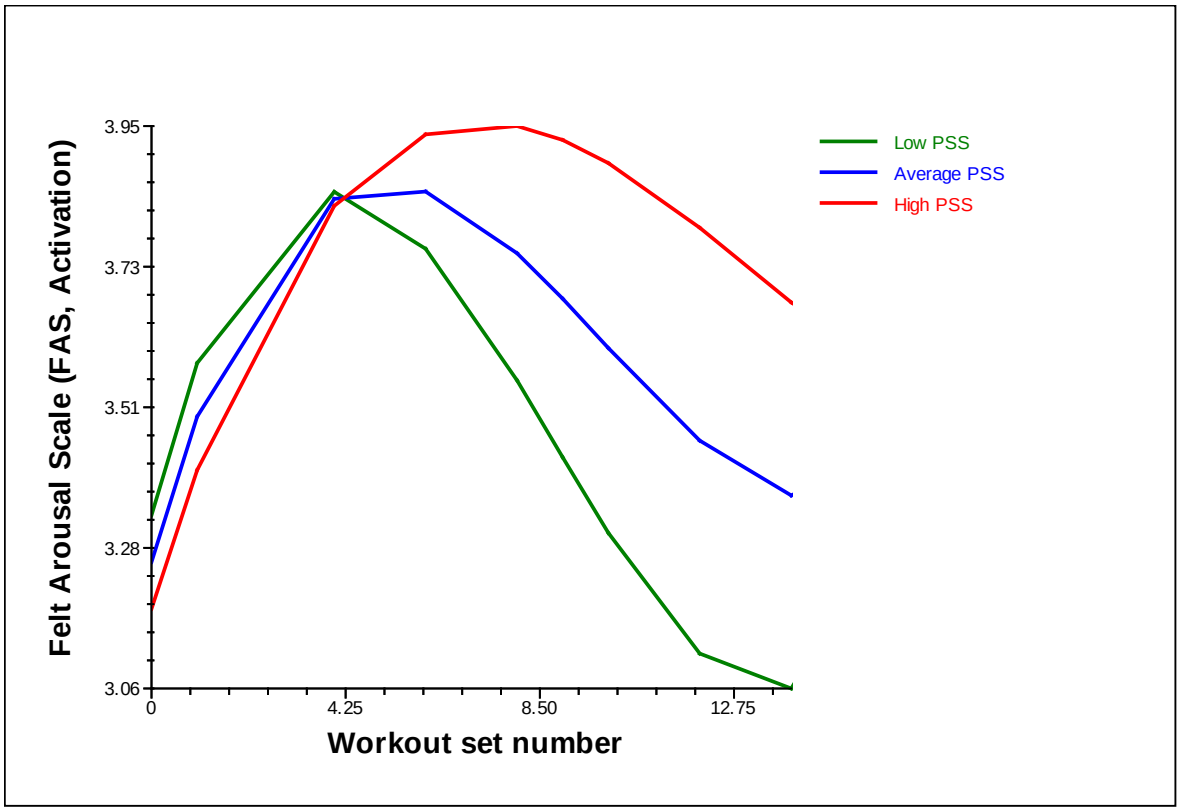


Figure 4.13 Relation of PSS at the first visit to changes in activation (arousal) during the E-RES workout.

Muscular Pain. There was a significant stress/strain by time interaction. Those reporting higher stress had lower increases in pain. This held for squared time ($p < .001$) and cubed time ($p < .001$). The intercept, however, was not significant, meaning that there was no stress time relationship at the beginning of the workout ($p = .746$). See Table 4.24 and Figure 4.14.

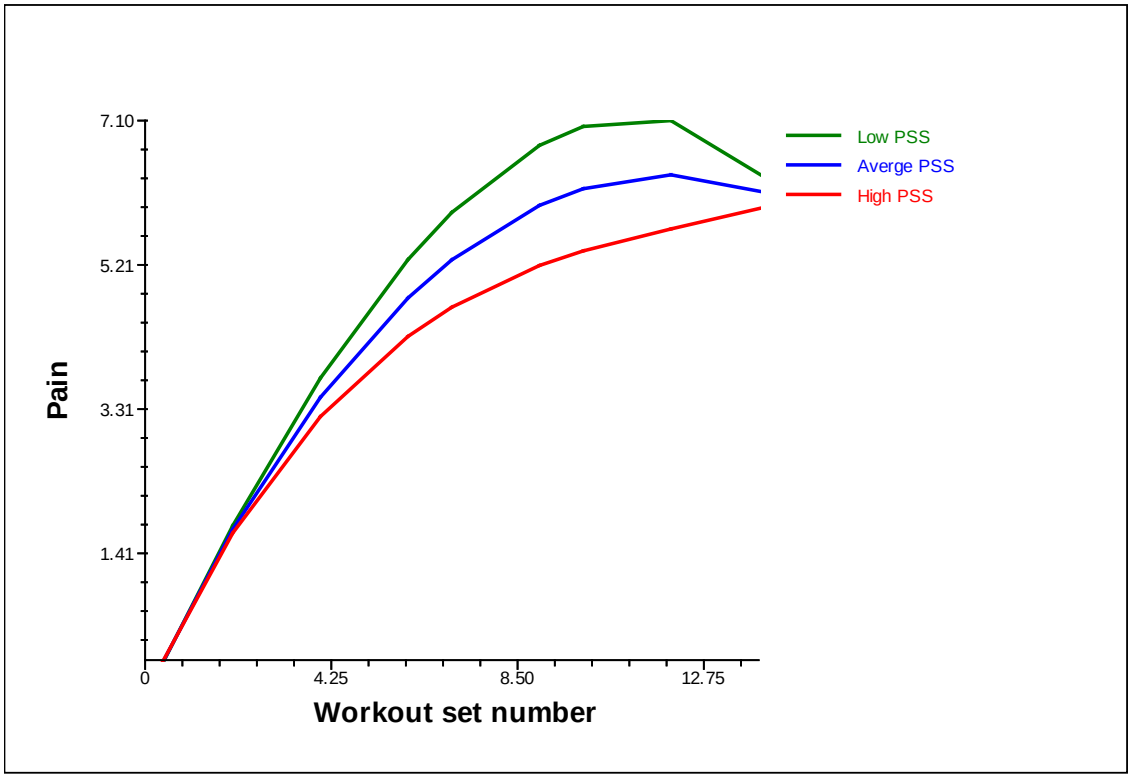


Figure 4.14 Relation of PSS at the first visit to changes in muscular pain during the E-RES workout.

RPE. There was a significant perceived stress at the first visit by time interaction. Similar to pain, those reporting higher perceived stress at the first visit had lower increases in exertion over the workout. This effect held for squared time (p = .022) and for cubed time (p = .007). There was no relationship at the intercept (p = .363). See Table 4.25 and Figure 4.15.

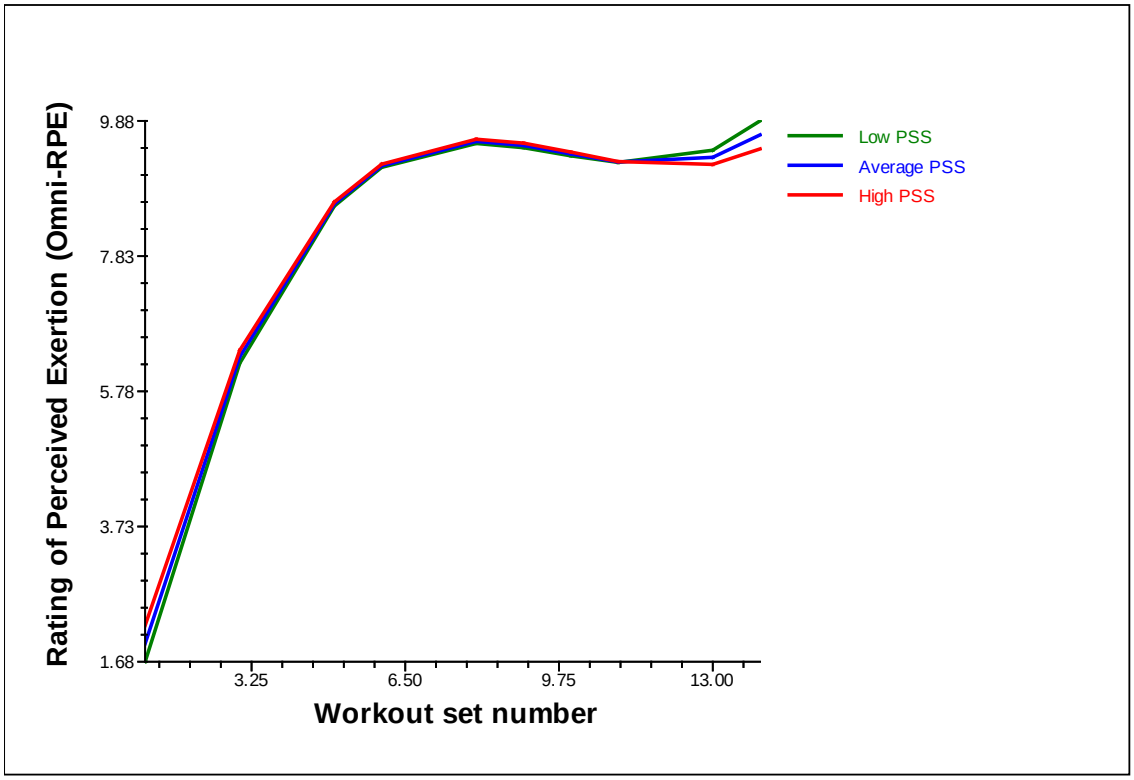


Figure 4.15 Relation of PSS at the first visit to changes in RPE during the E-RES workout. Note that in this diagram, time is modeled linearly, quadratically, and exponentially.

Hypothesis 2b & 2c:

Perceived stress (PSS) at the first visit did not predict post-workout perceived physical energy (β = -0.022, t = -0.111, p = .913) or perceived physical fatigue (β =0.275, t = 1.617, p = .118). Perceived stress (PSS) at the first visit did not predict post-workout soreness (β = 0.000, t = -0.002, p = .998).

Hypothesis 2d:

Perceived Physical Energy. There was not a relationship between stress and energy at 1 hour (PSS at time 1, p = .229; mean PSS, p = .279). However, there was a linear time by stress interaction for the recovery trajectories of energy over a 4-day period (PSS at time 1, p = .009; mean PSS, p = .004). See Table 4.26.

Perceived Physical Fatigue. There was not a relationship between stress and fatigue at 1 hour (PSS at time 1, p = .714; mean PSS, p = .869). There was also no moderation of 4-day recovery curves evident for perceived stress (PSS at the first visit, p = .327; mean PSS, p = .210). See Table 4.27.

Hypothesis 2e:

Perceived stress at the first visit and mean perceived stress did not moderate the linear time–soreness relationship (PSS at the first visit, p = .898; mean PSS, p = .806). No relationships were evident for the squared time–soreness relationship (PSS at the first visit, p = .713; mean PSS, p = .683).

Finals

Hypothesis 1b:

MIF: Finals period, a naturalistic stressor, did not moderate the MIF and time relationship for both linear time (p = .101) and squared (quadratic) time (p = .232). When eliminating the dichotomous finals variable for the intercept and squared time, however, the moderating effect on linear time approached significance (p = .066). There was no difference at the intercept (p = .702), which indicates that no differences existed at the end of the first hour of recovery after the E-RES workout. See Table 4.28.

Jump height: Finals did not moderate the jump height and time relationship for linear time (p = .384) and or for squared (quadratic) time (p = .442). There was no difference at the intercept (p = .556), which indicates that no differences existed at the end of the first hour of recovery after the E-RES workout.

Cycling power: Finals did not moderate the linear time–cycling power relationship (p = .156). Finals also did not moderate this relationship for squared time (p = .087). There was also no significant effect of finals on the intercept, indicating that these individuals had similar power values at 24 hours after the E-RES workout (p =

.257). After eliminating the effect of finals from the intercept and the effect of linear time, finals moderated the squared time-finals relationship. Those in finals had a lower or deeper recovery curve (p = .040). See Table 4.29 and Figure 4.16.

Hypothesis 2a.

Feeling. There was not a significant stress/strain by time interaction. Those reporting higher stress had greater decreases in feeling for squared time (p = .267) and for cubed (exponential) time (p = .108). The intercept was also significant, indicating that those higher in stress also were lower in feeling at the beginning of the workout (p = .355).

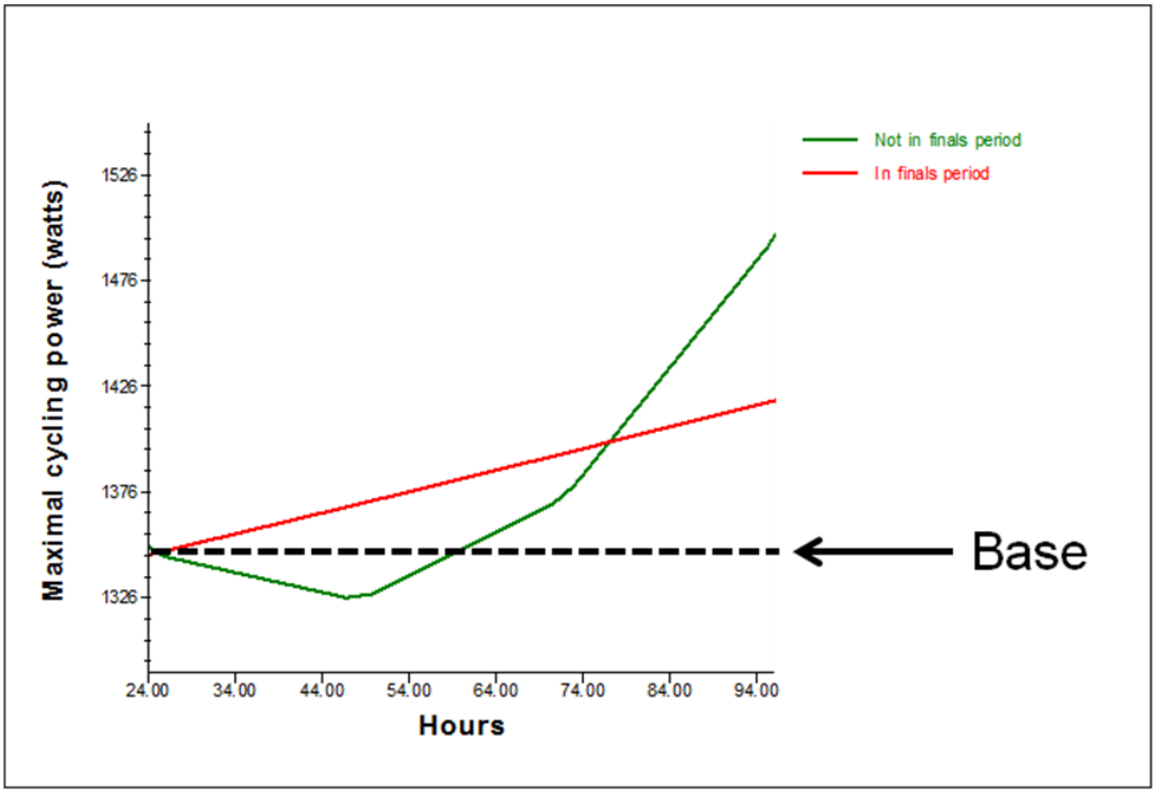


Figure 4.16 Relationship of finals period to cycling power recovery curves over 4 days. Time is modeled linearly and quadratically.

Activation (Arousal). There was no stress/strain moderation of the time–activation relationship for squared time (p = .277) or for cubed time (p = .676). The intercept was not significant (p = .780).

Muscular Pain. There was a significant stress/strain by time interaction. Those reporting higher stress had *lower* increases in pain. This held for squared time (β = -0.056, *SE* = 0.016, *t*-ratio = -3.496, *df* = 29, p = .002) and cubed time (β = 0.004, *SE* = 0.001, *t*-ratio = 3.993, *df* = 338, p < .001). The intercept, however, was not significant, meaning that there was no stress time relationship at the beginning of the workout (p = .621).

RPE. There was a significant perceived stress at the first visit by time interaction. Similar to pain, those reporting higher perceived stress at the first visit had lower increases in exertion over the workout. This effect held for squared time (p = .463) and for cubed time (finals, p = .392). There was no relationship at the intercept (p = .563).

Hypotheses 2d & 2e:

Perceived Physical Energy. There was not a relationship between finals and perceived physical energy at 1 hour (p = .084). There was no linear time by stress interaction for the recovery trajectories of energy over a 4-day period (p = .227). See Table 4.30.

Perceived Physical Fatigue. There was not a relationship between stress and fatigue at 1 hour (finals, p = .095). However, there was a linear time by stress interaction for the recovery trajectories of fatigue over a 4-day finals period (p = .020). Those who completed the workout during finals had higher recovery curves over 96 hours, which indicates that they decreased from their fatigue at a slower pace. See Table 4.31.

Soreness. Finals was significantly related to the linear effect of time (p = .047). Those in finals had a higher level of soreness over the 4-day recovery period. See Table 4.32.

EXPLORATORY ANALYSES

Exploratory Research Questions

Is it possible that stress/strain measures are related to recovery in the first hour after an exhaustive resistance-training workout? I collected data for MIF, perceived physical energy, perceived physical fatigue, and soreness in 20-minute intervals for 1 hour immediately after the E-RES workout. No data were collected for vertical jump height or for maximal cycling power.

Exploratory analyses were also conducted to test the possibility that stress and strain measures were related to immune parameters in the 48-hour period following exhaustive resistance exercise. A subset of 13 participants with blood draws collected pre-workout, 24 hours, and 48-hour post-workout were selected for this analysis. Stress was run as a dichotomous variable with six individuals in a high stress group and seven individuals in a low stress group. Stress groupings were based on PSS score at the first visit.

First Hour of Recovery

Functional Form of Time. To assist in modeling time over the first hour of recovery for each dependent variable, tests were performed to determine functional form of time, whether linear or curvilinear. For MIF, linear (ß =151.097, *SE* = 29.085, *t*-ratio = 5.195, *df* = 30, $p < .001$) and squared (quadratic) time (ß = -81.224, *SE* = 20.745, *t*-ratio = -3.915, *df* = 30, $p = .001$) provided the best fit. Unlike with the full 96 hours of recovery, linear time provided the best functional form of time for physical energy (ß = 16.139, *SE* = 3.763, *t*-ratio = 4.289, *df* = 30, $p < .001$). Linear time provided the best fit for physical

fatigue (ß = -19.859, *SE* = 4.265, *t*-ratio = -4.656, *df* = 30, $p < .001$). Cubed (exponential) time provided the best fit of the data for soreness (ß = -9.227, *SE* = 3.272, *t*-ratio = -2.820, *df* = 115, $p = .006$).

For MIF, the model was able to converge with the variance components estimated freely. Examination of the variance associated with linear time revealed that there was variability left to continue modeling additional variables ($p < .001$). Furthermore, there was significant variability in the squared function of time ($p = .001$). For physical energy and fatigue, the model was able to converge with the variance component estimated freely. Examination of the variance associated with linear time (both energy and fatigue) revealed that there was variability left to continue modeling ($p < .001$). To facilitate model convergence for soreness, the cubed time parameter was fixed and no variance components were estimated.

MISSING DATA

There was very little missing data for the first hour of recovery. Missing data ranged from 2.5% to 5.6%. See Tables 4.3 and 4.33.

Table 4.3 Disruption and recovery for MIF 1 hour post-workout (means and % change)

	Pre	0 min	20 min	40 min	60 min
MIF					
Low stress	299.7	154.4 (-48.5%)	213.1 (-28.9%)	225.6 (-24.7%)	247.5 (-17.4%)
High stress	285.7	171.9 (-39.9%)	209.2 (-26.8%)	226.2 (-20.8%)	237.6 (-16.8%)
All	292.9	162.8 (-44.4%)	211.2 (-27.8%)	225.9 (-22.9%)	242.7 (-17.1%)
Energy					
Low stress	47.7	25.1 (-47.4%)	28.4 (-40.5%)	38.1 (-20.1%)	41.7 (-12.6%)
High stress	41.5	33.2 (-20.0%)	33.7 (-18.8%)	39.9 (-3.9%)	47.7 (+14.9%)
All	44.7	29.0 (-35.1%)	31.0 (-30.6%)	39.0 (-12.8%)	44.5 (-0.5%)
Fatigue					
Low stress	31.3	80.2 (+156.2%)	73.5 (+135.8%)	65.2 (+108.3%)	63.9 (+104.2%)
High stress	42.3	85.7 (+102.6%)	74.0 (+74.9%)	69.3 (+63.8%)	64.9 (+53.4%)
All	36.6	82.9 (+126.5%)	73.7 (+101.4%)	67.2 (+83.6%)	64.4 (+76.0%)
Soreness					
Low stress	20.3	61.3 (+202.0%)	58.1 (+186.2%)	62.2 (+206.4%)	55.1 (+171.4%)
High stress	25.1	78.6 (+213.1%)	70.6 (+281.3%)	72.3 (+288.0%)	63.5 (153.0%)
All	22.6	69.7 (+208.4%)	64.1 (+183.6%)	67.1 (+196.9%)	59.0 (+161.1%)

Muscular Functioning (MIF Only)

Life event stress (USQ) significantly moderated the time-MIF relationship for both linear time (p = .013) and squared time (p = .050). Even after controlling for significant covariates, including finals, fitness, FFM, number of semesters of the weight-training class, workload, and reduction in force, the stress–time interaction was still significant for linear time (p = .002) and for squared time (p = .017). Consequently, a similar pattern was observed for both analyses. 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. See Figure 4.17, Table 4.34, and Table 4.35.

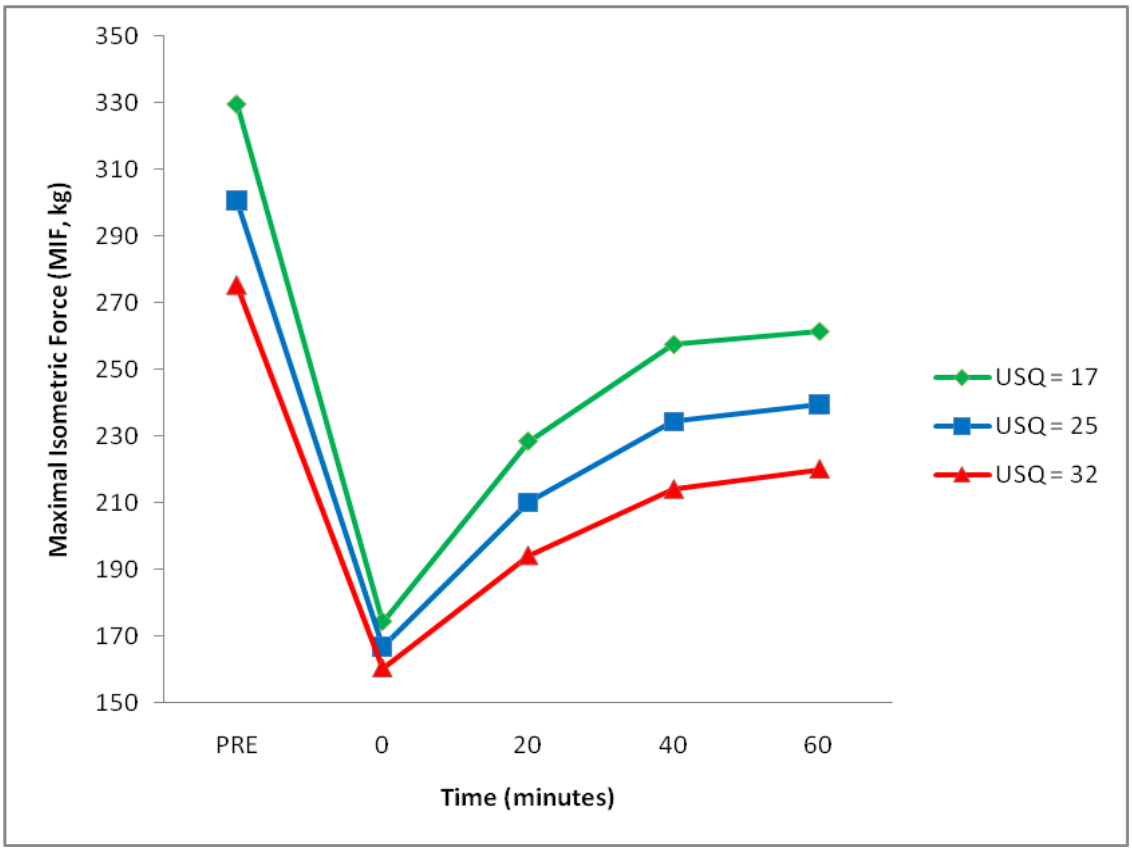


Figure 4.17 Maximal isometric force over the first hour of recovery by USQ.

Unlike life event stress, perceived stress at the first visit did not significantly moderate the time-MIF relationship for linear time (p = .143) or squared time (p = .372). However, after controlling for significant covariates, including finals, fitness, FFM, number of semesters of the weight-training class, workload, and reduction in force, the stress–time interaction was significant for linear time (p = .023). PSS was not significant for squared time (p = .118). See Figure 4.18 and Table 4.36.

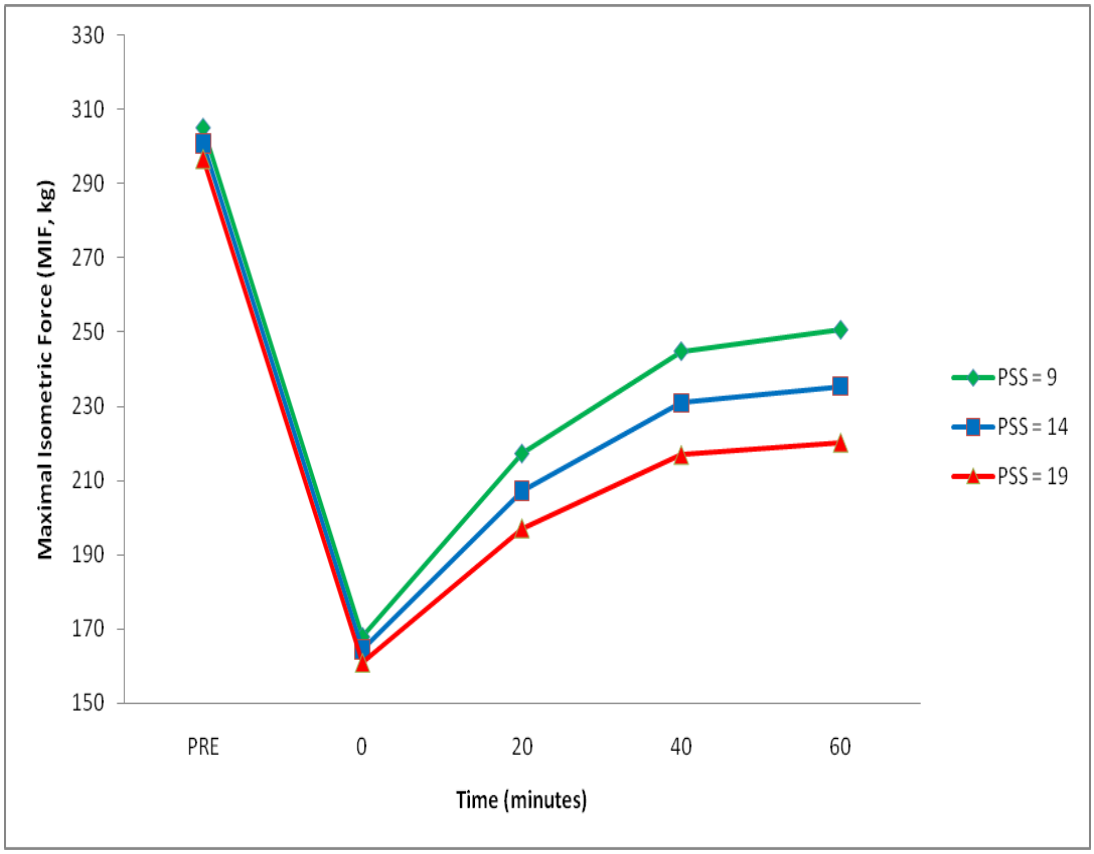


Figure 4.18 Maximal isometric force over the first hour of recovery by first visit PSS.

Psychological Function

There was no moderating effect of stress/strain measures on recovery of perceived physical energy, perceived physical fatigue, or soreness over the course of the first hour after the E-RES workout. There was no statistical significance for all outcomes with various functions of time modeled (all *p*-values > .100).

Cytokines

It is reasonable to hypothesize that there may be a relationship between stress/strain and changes in inflammatory makers in the period after the E-RES workout. See Table 4.37.

IL-1β. No functional form of time clearly emerged for IL-1β. There was a significant difference for IL-1β for linear time (p = .004) or when modeled separately for squared time (p = .035). When IL-1β was modeled with both squared and cubed time, however, both of these parameters were also significant (p = .019 and p = .023, respectively). See Figure 4.19 and Table 4.38.

TNF-α. There was no significant stress/strain effect for TNF-α (squared time, p = .324; cubed time, p = .346) [the functional form was both squared(.032) and cubed (.020)].

IL-6. There was no significant stress/strain effect for IL-6 (squared time, p = .195; cubed time, p = .166). [the functional form was both squared(.070) and cubed (.061)].

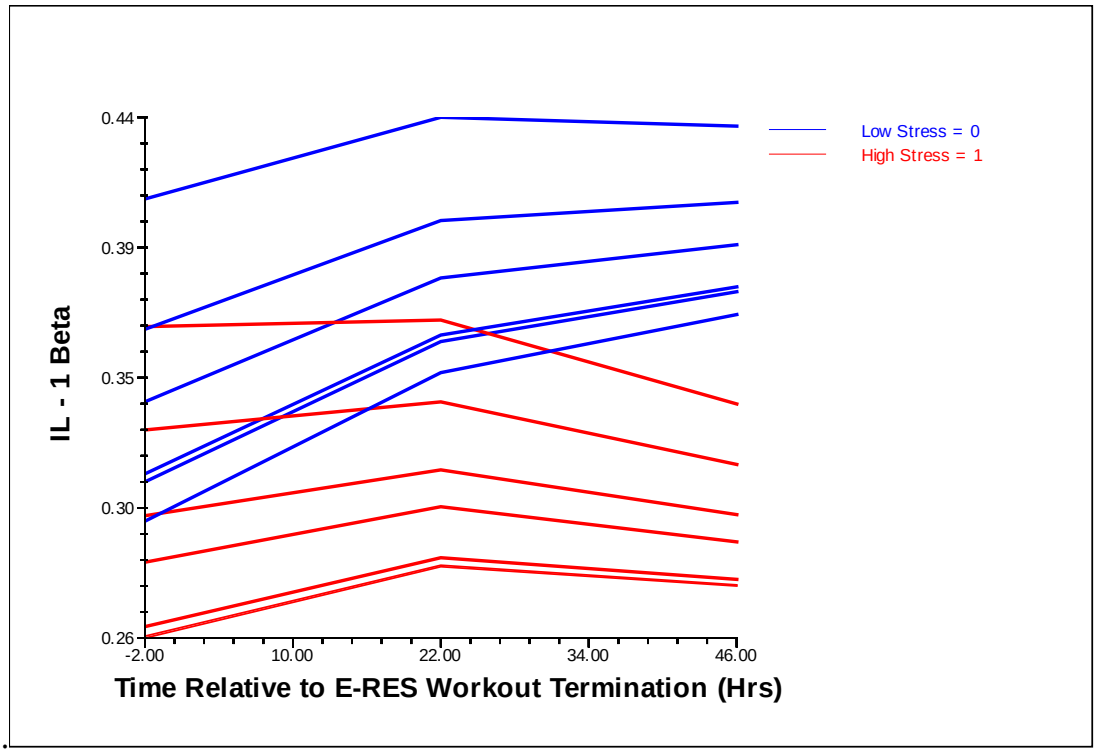


Figure 4.19 IL-1β (pg/ml) over 48 hours.

Summary

The exploratory analyses demonstrated that stress moderates the recovery curves for the first hour of recovery. Furthermore, the effects of stress hold even after adjusting for covariates. The pattern of the effects was the same. Life event stress was related to recovery for both models, while perceived stress was only related to recovery in the model adjusting for covariates. Results were differential for cytokines. IL-1β was related to stress; however, IL-6 and TNF-α were not related to stress.

CHAPTER FIVE

DISCUSSION

INTRODUCTION

This dissertation provides evidence that chronic stress is related to muscular and psychological recovery from a difficult bout of resistance training. I selected a quasi-experimental design for this investigation because it is neither ethical nor practical to manipulate chronic stress. Specifically, I screened a group of weight-training students (n = 210) for chronic stress. Those eligible were approximately half a standard deviation above or below the mean for perceived stress. Participants were asked to complete an exhaustive resistance exercise stimulus (E-RES) workout that induced muscular micro-trauma. Functional recovery over 4 days was followed for lower body maximal isometric force (MIF), vertical jump height, maximal cycling power, perceived physical energy and fatigue, and soreness. The slopes of the recovery curves for 31 participants were tested for relationship to stress/strain measures, including life event stress (Undergraduate Stress Questionnaire, USQ) and perceived stress (Perceived Stress Survey, PSS). In addition, exploratory analyses focused on recovery in the first hour following this bout of exercise and on cytokine responses in the 48-hour period post-workout.

I predicted that stress measures would affect recovery curves for these outcomes, and for each physical variable, higher stress would be related with deeper (lower) recovery curves. My hypotheses were largely confirmed. Stress and strain measures were related to recovery from maximal resistance exercise for both functional muscular (MIF, jump height, and cycling power) and psychological (perceived energy, perceived fatigue, and soreness) outcomes. Higher stress was related to deeper recovery curves (higher for

fatigue and soreness), and hence, slower recovery for all six outcomes. Lower stress, on the other hand, was associated with superior recovery.

Higher stress is possibly related to recovery simply through differential experiences of disruption from a workout. To state this possibility another way, stress may be related to the total decrement in any given outcome pre- to post-workout. Differential amounts of disruption from baseline, in turn, may be highly related to aspects of recovery. Stress measures, however, were largely not related to post-workout outcomes after adjusting for baseline values as determined by two-step regression analyses.

There were significant moderating effects of stress on affective responses during exercise. Feeling (pleasure/displeasure), activation (arousal), and muscular pain trajectories were moderated by stress, whereas RPE (exertion) was not. High stress was associated with greater decrements in feeling, less increase in pain and exertion, and higher activation, (due to *less* steep slopes, or rather, a slower decline) during the latter half of the workout. In addition, exploratory analyses showed that stress moderated physical recovery, but *not* psychological recovery in the first hour after the E-RES workout. Finally, stress was related to the increase in IL-1β, a pro-inflammatory cytokine, in the 48-hour period after exercise for a subset of participants. Lower stress individuals, therefore, exhibited a greater IL-1β response.

INTERPRETATION OF MAJOR FINDINGS

Muscular Recovery

As hypothesized, stress/strain was related to recovery from an exhaustive bout of resistance exercise over a 4-day period post-workout. Higher stress scores were related to lower (or deeper) recovery curves, meaning that individuals reporting greater stress recovered slower and returned to baseline functioning at a slower pace. The 48-hour

period appears to be the point of greatest difference between low and high stress/strain individuals. Those reporting high-perceived stress at the first visit had an 11.5% reduction in force even at 48 hours, while those reporting low stress had a gain in force production of 7.6% at this same time. Differences between these groups were greatest at 48 hours for peak jump height (7.8% reduction for high stress; 10.6% gain for low stress) and maximal cycling power (3.3% reduction for high stress; 3.3% gain for low stress).

Functional recovery was also assessed through jump height and maximal cycling power. The stress-recovery relationship was less consistent for these measures of recovery. In regards 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 were related to recovery curves over 96 hours. These differences were likely due to the machine used to induce muscle damage, which was identical to the machine used to assess maximal isometric force (both 45 degree angle leg presses). As such, it is not surprising that maximal isometric force was the most sensitive measure. Likewise, vertical jumps utilize musculature in a manner more similar to leg press than cycling. For instance, cycling underemphasizes the hip and gluteus muscles unlike both leg press and vertical jump. Therefore, muscles utilized to produce cycling power are less damaged than muscles used to leg press and jump. This interpretation is, of course, speculative. It would require the induction of muscle damage through various means, (e.g., leg press and cycling) to test definitively, but it does fit the observed pattern of results.

It has been argued that life event stress is less than ideal as a moderating variable because it fails to test the perceived impact of each event (Compas, Davis, Forsythe, & Wagner, 1987). As a result, stress was modeled as both life events and perceived severity.

The observed patterns of effects were largely identical for both. This suggests that stress, regardless of personal impact, exerts a strong effect on recovery.

These effects do not appear to be limited to a single, shared stressor. In other words, it is plausible that a single event, such as final exams, an event that all students experience during the course of a semester, could be related to and predict recovery better than scores of self-reported stress. If this were true, then students who completed the E-RES workout during the last few weeks of the semester would largely have worse recovery than students who completed the workout during earlier weeks. As students were recruited across the semester there was large variation in the timing of study entry and completion. Consequently, I coded finals status as a binary variable (0 or 1) and analyzed whether being in finals (a score of 1) was related to recovery trajectories. For isometric muscle force, my analyses determined that finals status was not significantly related to recovery of maximal isometric force ($p = .101$).

In addition, the stress/strain and recovery relationship held even when adjusting for indicators of potential compromising (hours of sleep, magnitude of disruption from the E-RES workout) or beneficial physical covariates (muscular fitness, fat-free mass, workload (total kg), and semesters taking the weight-training class). Even with each of these factors entered in the equation; mean perceived stress was the strongest predictor of recovery across the physical outcome measures.

My exploratory analysis focused on the hour immediately following the exercise stimulus. These data revealed that both perceived stress and life event stress were also related to functional muscular recovery in the first hour after the resistance workout. Higher stress was related to lower recovery for maximal isometric force when measured over 20-minute intervals. Just as with the 4-day recovery analyses, these relationships held even after adjusting for covariates related to the production of muscular force.

Overall, the observed results mirror the wound healing studies (e.g., Marucha et al., 1998). In this body of research, higher chronic stress was consistently related to slower healing from a wide variety of induced wounds. For instance, Marucha et al., in a within subject design, found that healing time was extended for 100% of dental students during academic finals period as opposed to during summer break. Burns (2006) found this study noteworthy because the deleterious effects of stress were observed in a young and healthy group of individuals experiencing a relatively mild, transient constellation of stressors. Consequently, this dissertation replicates and extends this body of knowledge into the area of exercise.

To summarize the results for physical outcomes over four days, both life event stress and perceived stress moderated recovery for MIF values. Additionally, this pattern of results replicated for vertical jump height (at least for perceived stress). Finally, exploratory analysis of physiological recovery over one hour revealed that stress and strain was significant over this time frame as well. USQ explained 9.8% of the variance of recovery for MIF over the first hour.

Psychological Recovery

Even though stress was not related to energy, fatigue, or soreness at precisely 1-hour post-workout, it was significantly related to recovery trajectories over 4 days. For energy, this relationship held for perceived stress and for life stress. Higher stress was related to deeper/lower recovery. However, for fatigue and soreness, this relationship held only for life event stress. Higher stress was associated with higher/flatter recovery slopes, which indicates slower recovery. Psychological recovery in the first hour, however, was not related to any form of stress/strain. Both USQ and PSS at the first visit were not related to soreness ratings taken at the first visit or before the E-RES workout.

It is not clear why these various differences were observed for psychological outcomes and for 4-day versus 1-hour time frames. This is the first study to test the psychological recovery of perceived physical energy and fatigue from a damaging bout of resistance training. There is considerable criticism regarding measurement in exercise-related fatigue and soreness, which may undermine the results–especially when compared to measures with less error, such as maximal isometric force. The crux of these criticisms is that one cannot be certain about what is actually being measured by single-item state measures of affect, exertion, and pain. The experience of painful sensations, for instance, varies by numerous inter- and intra-individual factors, such as gender, experience, sensitivity, and tolerance. On the other hand, there is some evidence that these items are not overly problematic and that they tap into more objective constructs than dispositional measures (Tenenbaum, Kamata, & Hayashi, 2007, p. 768). Moreover, the current data support results seen for soreness by other researchers (e.g., MacIntyre et al., 1996).

Affective Disruption during Exercise

Stress/strain was related to affective responses during exercise, but in a more complex manner than expected. Stress was related to feeling (pleasure/displeasure), activation (arousal), and muscular pain even after adjusting for these variables at the beginning of the workout (or in the case of activation, at the *end* of the workout). Those who were higher in stress reported more negative feeling states at the beginning of the workout and had stronger decreases in feeling states throughout the workout. However, the pattern of these responses differed by the type of stress measure. Specifically, the decrease in affect was affected by perceived stress, but not life event stress. It is possible that those who perceive stress are more attuned to *changes* in affective state during times of general acute stress. On the other hand, the correlation between PSS and USQ with

negative affectivity (from the first visit) was very similar (r = .487 versus r = .463, respectively).

It is uncertain why different results manifested for life event stress and perceived stress. However, other researchers, particularly in the area of psychoneuroimmunology, have found similar differences between life events and perceived stress on physiological adaptation (Burns, Carroll, Drayson et al., 2003; Cohen, Tyrrell, & Smith (1993, as cited in Burns, 2006). For example, Cohen, Tyrrell, & Smith (1993) found that there were differential effects of these types of stress, whereas life event stress was related to the manifestation of the common cold through symptoms and perceived stress was not related to symptomology. These differences may be due to the nature of perceived stress, which is cognitively mediated. Several of the items ask respondents about their perception of control in their life over the last month. Perhaps individuals high in perceived stress feel threatened by the stressors they encounter. Life event stress, on the other hand, may not reflect perceptions of threat and distress. While each type of stress would require greater recruitment of energy (via greater release of epinephrine and glucocorticoids), only perceived stress may be related to physical responses resulting from a lack of perceived control. For instance, Blascovich and Tomaka (1996) argued that stress responses devoid of distress result in differential physical responses. Stresses resulting in challenge appraisals and perceived maintenance of control are associated with increased Sympathetic-Adrenal Medullary (SAM) responses. Hence, there are increased levels of catecholamines, increased heart rate, cardiac output, and decreased peripheral resistance. However, stresses with threat appraisals and perceptions of diminished control result in hypothalamic pituitary adrenocortical (HPA) activation, which results in lower cardiac responses and possibly slight increases in peripheral resistance. Perhaps it follows that differences in affective responses for life event stress

and perceived stress were due to varying perceptions of control or effect of reported stress.

Stress was associated with reported pain and exertion during the E-RES workout. Participants reporting lower stress reported higher muscular pain sensations during the E-RES workout. This was true regardless of the type of stress measured (perceived stress or life event stress), and thus, it may represent the most robust effect demonstrated. These findings may be due to the difficulty in interpreting painful sensations. For instance, individuals reporting lower stress may exhibit a cognitive bias, such as a contrast effect. In other words, these individuals may experience lower levels of pain in daily life, and thus, when facing a painful situation may overrate painful sensations (Gilovich, Griffin, & Kahneman, 2002). Perhaps a greater possibility is that lower stress individuals accurately rate pain, but individuals under higher stress suppress pain sensations or dampen ratings of pain to protect the self. Recognizing these bodily sensations as painful would provide additional threat (Krohne, 1989, as cited in Stemmler, 2003).[27] Lower stress was also related to greater perceived exertion, although the trend for activation (arousal) was dissimilar. In this case, lower stress was associated with decreased activation at the end of the E-RES workout. This is perplexing because stress was not related to physical strain parameters indicative of workload. Peak and average heart rates, indicators of acute physical strain, had no relationship to mental stress. On the other hand, higher stress was correlated with higher completion of total work. This was likely a result of a greater number of warm up sets completed in the ramping phase of the workout by individuals reporting higher levels of stress and strain.

[27] Consider the fact that it is impossible to sense the intensity of painful sensations accurately when they are elicited from multiple regions of the body. If you are receiving signals of pain from your lungs (from breathing hard), your heart (from it pumping vigorously), your hands (from clenching the leg press handles tightly), and your leg musculature (which is dynamically laboring), then any survey of pain targeting any single region will be lower. Bear in mind that ratings of pain were targeted to the lower body working muscles. If ratings were based on overall painful sensations from the entire body it is possible that results may have been different. However, this is merely speculation.

MECHANISMS OF THE STRESS-RECOVERY RELATIONSHIP

The purpose of the exploratory analyses was to assist in assessments of potential mechanisms that might be responsible for the stress-recovery phenomenon, and thus, serve as the basis for future research. These mechanisms may be physical, such as cytokines, or psychological, such as the interpretation of physical sensations.

Interpretation of Physical Sensations

As noted above, higher stress was related to greater activation (arousal), particularly in the final six sets of the E-RES workout. During this period of the workout, participants likely produced a substantial amount of metabolic waste products associated with anaerobic metabolism. Average peak heart rates for these sets were over 160 beats per minute. This is significant because metabolic strain is the primary determinant of affective responses to exercise (Ekkekakis & Petruzzello, 1999). I did not collect lactate samples, which would certify whether participants were depleting their anaerobic energy system; however, an analysis of the relationship between relative heart rate (percent of maximum) and arousal determined that a negative relationship existed between these variables, but only for high stress individuals (r_s = -.412 to -.569 for high stress and r_s = .059 to .168 for low stress).

Cytokines

There were no stress-related differences for IL-6 and TNF-α. It is possible that I analyzed too few data points. Consequently, the functional form of cytokine change trajectories may be masked because there was not enough data to plot a curve properly.[28] Capturing precise trajectories also necessitates larger sample sizes than the one utilized in this dissertation. Another possibility is that stress only has an effect on muscular damage, per se, and not recovery of energy. Lancaster (2006) provides evidence, for instance, that

[28] Three points can only produce one bend in a growth or recovery curve.

of the three cytokines that I measured, only IL-1β is actually reflective of muscular damage. However, in the current study, *lower* stress was related to a greater IL-1β response in the 48 hours after exercise. This result is perplexing because one might anticipate that individuals reporting higher chronic stress would have greater cytokine values during recovery. Nevertheless, it is also possible that a stronger immune response is actually more adaptive as it propels the recovery process from damage. This is coupled with the fact that flexibility of physiological responses in the face of stress is key for successful health (Sapolsky, 2004). It is desirable to have a strong response followed by a quick return to quiescence. It may also be adaptive to have a preparatory increase in one or more cytokines before an acute stressor just as it is normal to have an elevated heart rate before exercise (Dhabhar, 2002). Although these differences may be expected as evidence of a healthy immune system, this pattern does not necessarily explain the observed differences in recovery.

When interpreting the current results, one must be mindful of the differential effects of acute versus chronic stress and strain. Acute stress is often adaptive, while chronic stress may impair the mobilization of the immune system (Dhabhar, 2002; Dhabhar, 2009; Dhabhar & McEwen, 1997). It is likely that the effects of acute and chronic stress also interact. Kiecolt-Glaser et al. (1996), one of the first studies on the effects of an acute physical stressor (influenza virus vaccination) in a chronically stressed population, found that long-term caregivers of ill or disabled individuals had lower levels of virus-specific-induced IL-2 and IL-1β. Interestingly, IL-6 was not different between these groups, as in the current study. The authors noted that this response pattern may have predisposed the chronically stressed to a higher level of infection vulnerability associated with inoculation. This group of individuals, however, was elderly, and there may be profound effects associated with aging. Nevertheless, these findings were

replicated by other researchers, such as Costanzo et al. (2004), who found that those with better mental dispositions had a greater cytokine response after acute stress.

Acute stress exerts its effects depending on the intensity of the stimulus. It is likely that the present exercise protocol was a much stronger stimulus than they would have otherwise experienced. One hundred percent of the subjects rated the workout as "the heaviest exercise session or one of the three heaviest workout sessions in my entire life." Consequently, the subjects likely experienced an extremely strong glucocorticoid response, which is immunosuppressive (see Sapolsky, 2004, pp. 162-163).

Clearly, IL-1β plays a central role in the perception of pain, however, whether IL-1β is related to the intensification or alleviation of pain is contentious. IL-1β, along with corticotropin releasing factor (CRF), inhibits pain (Shafer, Carter, & Stein, 1994). The release of IL-1β in peripheral tissue has been associated with increased sensitivity to pain (hyperalgesia; Morgan, Clayton, & Heinricher, 2004). Alternatively, a recent review by Ren and Torres (2009) implicates IL-1β in exacerbated pain after injury. Evidence also associates brain IL-1β with intensified fatigue after muscle-damaging exercise (Carmichael et al., 2006). Considering the current and past literature supporting the effects of chronic stress on IL-1β, it seems plausible that the stress phenomenon may follow a path similar to one presented below (Figure 5.1).

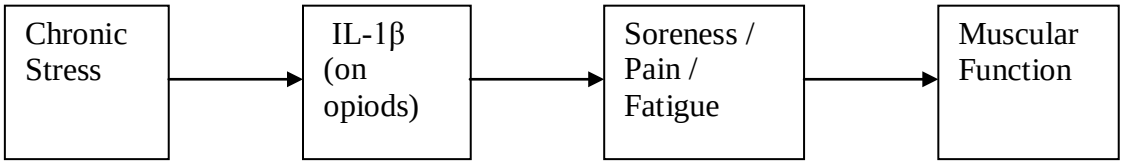


Figure 5.1 Potential paths from chronic stress to muscular function during the recovery period after strenuous exercise.

Motor Control

It is possible that stress had an effect on neural mechanisms and/or motor control. The muscular function recovery curves I observed gauge not only the effect of recovery, but also development of muscular control[29]. I infer this because of the great increase in function over baseline for all of the measures of muscular function. These three outcome measures necessitate differential amounts of learning. Maximal isometric force requires little learning, as individuals are simply pressing as hard as they are able. Maximal cycling requires a greater degree of learning and coordination of the muscular agonists and antagonists. Vertical jump requires the greatest amount of coordination and muscular control. We observed that poor performers could increase values rapidly over a set of trials. In light of this trend, some individuals were given a fourth or fifth attempt to reach a peak jump height. It is also possible that learning effects were of modest to negligible magnitude and that all gains in function were due to supercompensatory mechanisms. Perhaps a sufficient way to discern the effect of stress on recovery versus motor control would be by including a condition in which participants did not complete a workout–only the functional assessments.

On the other hand, there is evidence that negates the claim of learning. It is reasonable to infer that learning effects may be detectable after a single session of practice (between session improvements). Using paired-sample t-tests, I found that there was no difference between muscular function maximum values at visit 1 and before the workout at visit 2 (MIF, $p = .978$; vertical jump, $p = .727$; cycling power, $p = .753$).

[29] Just as initial status is influenced by chronic training, fitness, gender, health and other factors, the slope/trajectory is likely influenced by several underlying factors as well.

STRENGTHS OF THE CURRENT STUDY

The current study has several strong characteristics. First, I screened several hundred students to ensure enough variability in the stress measures. Second, I measured perceived stress at four time points, although there were missing data at both the second and last time points. I also measured stress from several theoretical standpoints: life event stress and perceived stress, which is cognitively mediated.

The assessment of muscular function was novel and combined both controlled isometric and naturalistic (vertical jump, cycling) exercise. Thus, it is reasonable to anticipate that the observed effects are likely applicable to a wide range of functional/purposeful movements. This adds relevance for the trainer, coach, and/or layperson unfamiliar with models of isokinetic dynamometry, which are typically utilized to induce micro-trauma in muscle damage research. Additionally, this is the first study to test psychological responses (i.e., perceived physical energy and fatigue) associated with recovery from a heavy bout of resistance training. Finally, this dissertation fills a gap noted in several recent reviews (Burns, 2006; Clow & Hucklebridge, 2001). These authors both noted that no study exists to extend the wound healing paradigm into the area of exercise research.

LIMITATIONS

Timing. Our follow up was limited to 96 hours of recovery post-exercise. Consequently, it is possible that I missed the end of the recovery curve for those under high stress. In other words, the recovery trajectory for high perceived stress may not have reached its plateau within the time-frame of my observations (Figure 4.10). Therefore, it is unknown whether higher stress is related to a lack of supercompensation or merely delayed supercompensation from strenuous exercise.

I did not measure jump height and cycling power at 1-hour post-workout. This may explain some of the differences in significance for these outcomes over the four days of recovery. Furthermore, the timing of blood collections may have masked any effect of stress on IL-6. This measure of inflammation peaks at 2 to 8 hours after strenuous exercise (Dousset et al., 2007; Miles et al., 2008).

The timing of the stress measurement is very important. I only measured life stress once, 5 to 10 days before the E-RES workout. Ebrecht et al. (2004) found that perceived stress at the time of biopsy was more predictive of healing time than stress 14 days before the biopsy or 14 days afterwards. In their review, Cohen, Miller, and Rabin (2001, as cited in Burns, 2006) found that a key marker of the immune system, s-IgA, is only related to psychological stress when measured very close in time. However, given the similarities in the pattern of results for perceived stress and life event stress, it is likely that this was not a major issue.

Sample Size and Composition

A relatively small number of individuals completed this study; however, this is less of an issue in studies incorporating screening for stress (Stemmler, 2003, p. 283).[30] While screening alleviates problems related to constrained variance, it generates issues in regards to generalizability, which may be limited due to non-random selection of participants. This threat is present whenever screening for a small set of variables and it has been a persistent problem in studies focusing on stress and emotion (see Stemmler, 2003, p. 238 for a discussion of threats to validity in psychophysiological studies of emotion). My sample included a large percentage of minorities (68%). Thus, it is not clear if my findings will generalize to other student populations. Furthermore, all of the

[30] Nevertheless, I did not have enough power to detect some effects. For instance, being in finals was not related to recovery trajectories for MIF. However, it is possible that there were not enough individuals in the finals period to detect this effect.

participants were non-athletes in a weight-training program and were not likely to have universal experience of or consistent muscle damage. Athletes who routinely exercise at a level of intensity similar to that elicited in the E-RES workout may have a far different response. Thus, generalizability of these data is limited to non-athletes.

Measurement Error

I utilized several single-item scales that have received criticism (Tenenbaum et al., 2007). As noted above, it is not fully certain what is actually being measured by these single items (e.g., personality, avoidance versus approach behavior, coping with stress). Generally, surveys with more items have better reliability. Nevertheless, the number of items does not erase problems with face or construct validity. Tenenbaum (2009) argued that while single-item measures such as exertion (RPE) suffer from poor reliability, as they certainly must because of their transient, state-like nature, they frequently are valuable tools because they tap into "objective" psychological constructs (p. 783). Finally, it must be noted that my item for soreness was not validated in previous studies, but was added as a single item to another validated scale (VAS scales; O'Connor, 2006).

Manipulation of Stress

The academic setting certainly causes strain for some individuals; however, others are rather immune to stress within this arena. Nevertheless, I feel that my screening technique was effective in finding individuals in this environment who perceived high or low levels of stress. I am not familiar with a better experimental protocol because it is not possible to manipulate chronic stress effectively or ethically. However, naturalistic stressors may provide clearer insights (e.g., caregiving, military boot camp) as a majority of individuals in these settings are under verifiable chronic stress.

In her review of the stress and healing relationship, Burns (2006, p. 222) criticized the stress and healing literature, in general, by asserting that most of this work actually

measures state anxiety and mood rather than stress. I did not triangulate self-ratings of chronic stress and strain with an interview of the participants or with people who knew the participants well, as recommended by Semmer et al. (2004). However, the use of life events serves to counter this criticism as it is less dependent on subjective experience.

Summary

Despite its numerous limitations, the findings of this novel experiment are in line with predictions. Specifically, I found that stress/strain measures moderated the time-muscle function relationship. In other word, stress was deleterious to recovery. This is the first study to explore this issue quantitatively within the realm of exercise-induced disruption. As such, and given the strengths, these data add to our understanding of relevant intrapersonal factors that contribute to adaptation from high intensity interval training.

Practical and Theoretical Implications

These findings likely have important clinical implications for those undergoing vigorous physical activity, both athletes and general fitness enthusiasts alike. I demonstrated that in the latter group, perceived stress and life event stress at the first laboratory visit was predictive of recovery from a vigorous resistance training workout. Individuals scoring a 10 on the PSS at their first visit reached baseline 288% (or 2.88 times) faster than individuals who scored a 19 on the PSS-10 at this same time point. Lower stress was also associated with a continued increase in function beyond baseline (supercompensation) before tapering off after approximately 3 days (see Figure 4.10). This held even after holding constant the effects of fitness, percent disruption, finals status and other factors. It is likely that individuals lower in stress were fully recovered and had experienced the maximum benefit from training and temporarily detraining.

Therefore, the 72-hour mark would be a good time for low stress individuals to begin training again.

High stress levels, however, were not associated with the experience of supercompensation post workout, even with reduced training. Higher stress was related to a prolonged return to baseline. However, as alluded to above, it is unknown whether higher stress is related to a lack of supercompensation or merely delayed supercompensation from strenuous exercise because of the abbreviated follow-up time of my dissertation (4 days post-workout). Regardless, these data suggest serious implications for those reporting higher stress. If high stress is related to an obviation of supercompensation, then these individuals may want to avoid very strenuous exercise altogether. On the other hand, if higher stress is merely related to slower recovery, then those with higher stress may simply want to include greater rest time (less frequent exercise). This information is likely most relevant for general fitness enthusiasts, as our sample was comprised of physical activity students and not athletes. Working out very vigorously during a period of great strain may require prolonged periods of recovery. Starting an unaccustomed workout regimen in a period of higher strain may not be advisable. On the other hand, there is considerable evidence that physical training with a lower level of intensity and/or volume may help to alleviate perceptions of stress (Spalding, Lyon, Steel, & Hatfield, 2004).

Findings from this study contribute to the ever-growing body of literature on damage, OTS, and under-performance. Perhaps the experience of chronic stress might explain some of the variability in muscle damage from exercise, which is unexplained by demographics, training status, fitness, and physical activity (Beaton, Tarnopolsky, & Phillips, 2002; Clarkson & Sayers, 2008). This study also compliments the literature that addresses OTS/burnout syndrome, a syndrome with depression-like symptoms related to

excessive training stress (Smith, 2000). These data may also inform research focused on performance incompetence in general (Lehman, Foster, Gastmann et al., 1999). More importantly, this research demonstrates that non-training stress, when experience is in excess, may result in impaired adaptation. This is in line with the hypotheses of Kenttä & Hassmén (1998) and Kellman (2000), both of whom posit that psychological stress is a powerful inhibitor of healing following training.

Finally, the results of this study may also help to explain findings from Bartholomew et al. (2008), who found that life event stress was related to lesser strength adaptations over the course of a semester-long weight-training class. A new wave of research is focusing on personal growth, both physical and psychological, that comes from acute challenges (Kumpfer, 1999, p. 189). I observed greatly enhanced perceptions of physical energy and greatly reduced perceptions of physical fatigue for most subjects in the four days after the E-RES workout. Perhaps this constitutes psychological growth and not just recovery.

No discussion of stress would be complete without recognizing the visionary writings of Hans Selye. He theorized in the revised edition of his book *The Stress of Life* (1976, pp. 434-435) that general stress responses (the General Adaptation Syndrome or GAS) would interact with disturbances localized to isolated areas, which he calls the Local Adaptation Syndrome (LAS). He states,

> When too much is going on in any one place within the body, that part is temporarily put out of action, by tissue breakdown, acute inflammation, or mere fatigue–which comprise essentially the alarm phase of the LAS. This forces other parts to take over, and thereby gives them a chance to develop as far as they can.
>
> But even without there being excessive activity in any one part, too much may be going on in the body as a whole. Then, the central coordinators of adaptation (the nervous system and the endocrine glands) are informed of this by the sum of the alarm signals arriving from all parts at any one time. When general stress is excessive the whole organism needs a rest; it cannot afford a struggle anywhere.

This dissertation supports Selye's tenets within the realm of exercise-induced muscle trauma, which is both localized to the musculature perturbed but also detectable systemically in the blood stream. Thus, generalized stress in the form of organism-wide disturbances may interact with localized stress in the form of acute disruption.

FUTURE DIRECTIONS

Future research should aim to replicate, improve and extend the methods utilized in this dissertation. The veracity of this research would be enhanced if other laboratories independently reached similar conclusions. Future research should systematically survey soreness perceptions in the period following fitness testing. Some individuals experienced soreness from the fitness testing at the first visit. Fortunately, there was minimal attrition due to soreness from fitness testing (one participant dropped out at the beginning of the E-RES workout). Those who did experience soreness from the first visit that subsequently healed may have experienced the "repeated bout effect" of protection against soreness. I selected a cycle test and minimized the number of sets on the leg press during fitness testing to minimize soreness.[31]

Experience with Intense Exercise

Prior exposure to this overload may be protective for experienced individuals. Our subjects likely had a wide variation in experience with resistance training and intense training of any type. Data from Fry (1999, pp. 156-157) suggest that more experienced weightlifters suffer much less disruption (in terms of testosterone to cortisol ratio) than less experienced weightlifters. These same individuals are also more likely to experience

[31] Interestingly, some of the participants reported that pressing isometrically provided relief from soreness, although this did not appear to be associated with stress. This phenomenon is consistent with tenets of the Wall-Melzack (1965) theory, which describe how acute/sharp and sudden pain is in conflict with more prolonged and dull pain. In essence, this is so because pain receptors, the X and Y interneurons, compete to send signals into the same afferent pathways that lead up the spine to the brain (see Sapolsky, 2004, pp. 189-192, for an overview of these processes). Indeed, isometric pressing can relieve chronic pain or soreness for up to several minutes and thus several interventions have been designed to take advantage of this natural phenomenon. This may also explain some of the benefits of massage.

supercompensation during periods of less training. This study does not generalize to the population investigated in the current study; however, it suggests a limitation that may need further investigation.

Health Behaviors and Other Factors

Sleep and nutrition, both adversely affected by the experience of stress, may mediate the recovery and stress relationship. Miller et al. (2004, as cited in Burns, 2006) found that after including sleep behavior in stress analysis, over 60% of the variability attributed to stress was eliminated. Health behaviors, along with other activities immediately after a vigorous workout, likely have a large effect on recovery from exercise. Alcohol, tobacco, and drug use should also be studied within this context (Clarkson & Reichsman, 1990). Certain potential moderators (or outcomes themselves) were not analyzed for their effect on recovery, including positive and negative affectivity and mental energy and fatigue.

How one deals with stress may have greater implications than the experience of stress itself. Stress resiliency, an enduring personality factor, is one such construct utilized to identify those protected from the nefarious effects of stress (Kumpfer, 1999). Garmezy (1991, p. 459) defines resiliency as "the capacity for recovery and maintained adaptive behavior that may follow initial retreat or incapacity upon initiating a stressful event." Difficulty in this line of research arises from the multifarious quantification of resiliency as a predictor, descriptor, and/or outcome of stress processes (Smith, Smoll, & Ptacek, 1990; Yi & Vitaliano, 2005). One may exhibit a history of stress resistance, thus being labeled retrospectively as "resilient" by the researcher, or one may subjectively rate oneself as resilient. Despite these difficulties, resiliency research has identified mechanisms responsible for both the experience of stress and pain during acute physical

stressors (Friborg et al., 2006). Therefore, this line of research is likely to shed light on stress-recovery processes.

Recent work from Temoshok, Wald, Synowksi, and Garzino-Demo (2008) on suppression of stress-related emotions and advancement of chronic disease may also provide a promising avenue for future research endeavors. In short, chronic physical adaptations (e.g., HIV progression) have been linked to these constructs, and thus, may be tied to adaptation from exercise. Research in this area, while valuable, is hindered by the difficulty in identifying true stress suppressors. Essentially, a great number of individuals react negatively to suppression surveys and become defensive. Research utilizing strong acute stressors, however, may help to identify those who suppress emotion under conditions of chronic stress (Repetti & Wood, 1997). Perhaps chronically stressed individuals muffle responses to acute stress as a strategy to ameliorate an ever-taxing level of chronic stress. If this is the case, more proximal mechanisms may be discovered which explain the stress and recovery relationship.

Interventions

Work also needs to be completed on the effects of mental health interventions on the stress-recovery relationship. Such interventions may focus on emotional disclosure (Petrie, Booth, Penebaker et al., 1995), cognitive-behavioral stress management (Davidson et al., 2003; Perna et al., 2003), or positive self-talk (Ievleva & Orlick, 1991), all of which have all been successful in previous investigations within the realm of exercise and/or sport. If successful stress management interventions were associated with enhanced recovery from intense exercise, then an extra layer of validity would be added to the results of this dissertation.

This strategy has already been utilized in research on stress, health, and injury. For instance, Perna et al. (2003) demonstrated that a structured stress management

program conducted by a professional psychologist resulted in enhanced health outcomes among a group of collegiate rowers. These individuals were assessed for frequency of injury and illness (defined as the total number of days that athletes sought medical attention), were restricted from activity by a medical practitioner, and self-reported days of injury and illness before these events. Furthermore, negative affect partially mediated this effect. Perhaps similar programs may be extended towards the goal of enhancing recovery and healing from exercise as opposed to preventing infirmity. Such an endeavor may accomplish this goal and improve general health concomitantly.

Bi-Directional Effects Research

Few mind-body (or body-mind) studies attempt to disentangle issues of bi-directionality. Does impaired recovery lead to the experience and perception of stress just as stress likely results in poor recovery?[32] Research on the stress - pain relationship has frequently relied on groups of individuals who experience chronic pain and correlate this disorder with self-reports of stress (Sapolsky, 2004). A cross-sectional design such as this is plagued with threats to validity. Sapolsky (2004, p. 161-164) notes that retrospective self-reports of stress are particularly problematic as individuals with chronic conditions often over-rate the experience of stress. This inflates the association between the condition and stress measures.

Prospective designs are an improvement over cross-sectional studies. Utilization of experimental designs similar to the current one would facilitate understanding of how pain (or soreness) affects perceptions of stress. Previous data suggest that changes in the experience of pain moderate perceived stress over long term periods (Latimer, Martin Ginis, & Hicks, 2004). In less cautious words, pain is stressful and when pain remits so does stress. Investigations which measure stress daily during a period of recovery would

[32] Kumpfer (1999, p 209) notes that children with poor physical strength frequently internalize this characteristic as poor self-worth and psychological weakness.

provide an ideal model for exploring these interactions on a smaller time frame. Furthermore, relating a daily stress construct to outcomes of interest (e.g., daily improvement in soreness, muscle function) would shed light on how the experience of transient stressors over short periods of time inhibit healing (see Gil, Carson, Porter et al., 2003).

Correlating muscle soreness, energy, fatigue, and function at each time point in a single analysis may demonstrate how these variables' recovery trajectories relate to each other over time. New statistical analyses, such as HLM (multivariate repeated measures), should be utilized to correlate these variables of interest at each point of recovery. This procedure prevents analyzing time at the hour because time must be quantified identically for all individuals. However, it retains flexibility in that missing data are permitted. This more sophistical multi-level analysis also automatically identifies the time at which groups differ for any given variable without having to recode for the intercept. It also increases statistical power, just as MANOVA is superior to univariate ANOVA in this regard.

CONCLUSION

Chronic stress/strain is indeed related to patterns of recovery from a strong physical stressor. I screened 210 weight-training students for perceptions of stress. Those scoring approximately a half standard deviation above or below the population mean for perceived stress were invited to complete a very strenuous bout of resistance training, which induced muscular micro-trauma. Thirty-one participants finished this workout and were followed for 4 days of post-exercise recovery. I found that self-reports of chronic stress and strain moderated both physical and psychological recovery from strenuous resistance exercise even after controlling for other factors (such as fitness and percent disruption) that may contribute to recovery. Furthermore, lower stress scores were

associated with recovery to baseline for maximal isometric force over two times more quickly than higher stress scores. The pattern of recovery for lower stress was even related to muscle function levels higher than baseline, in other words, a supercompensation response. These data may help researchers to understand the wide variability in the recovery from muscular damage seen after exercise with a heavy eccentric component. From a practical standpoint, those experiencing periods of unduly high stress may need to engage in exercise of lighter intensity or add a substantial amount of rest time before the next bout of vigorous exercise. This dissertation shifts the focus of stress research from the drift into disease and dysfunction to the road of recovery, restoration, and enhanced function. Future research needs to identify other factors that enhance recovery and contribute to positive growth.

Table 4.4 Pearson Product Correlations

	PSS: Online	PSS: 1st Visit	USQ	Neg Affect	Finals	PSS: Mean	Neg Affect: Mean	Phys Energy	Phys Fatigue	Mental Energy	Mental Fatigue	Soreness
PSS: 1st visit	.747 **											
USQ	.302	.594 **										
Neg Affect	.213	.487 **	.463 *									
Finals	.385 *	.339	.156	.343								
PSS: Mean	.884 **	.946 **	.561 **	.422 *	.417 *							
Neg Affect: Mean	.393 *	.602 **	.544 **	.810 **	.339	.572 **						
Phys Energy	-.323	-.460 *	-.298	-.438 *	-.354	-.461 *	-.622 **					
Phys Fatigue	.207	.291	.383 *	.590 **	.261	.275	.529 **	-.430 *				
Mental Energy	-.284	-.523 **	-.402 *	-.405 *	-.345	-.448 *	-.506 **	.674 **	-.165			
Mental Fatigue	.317	.357	.139	.313	-.046	.322	.337	-.087	.635 **	.085		
Soreness	.123	.134	.221	.014	-.053	.119	.172	-.317	.512 **	-.068	.515 **	
PRETIE-Q	-.136	-.321	-.107	-.150	.050	-.252	-.153	.324	.004	.515 **	.079	-.028

* Correlation is significant at the 0.05 level (2-tailed)
** Correlation is significant at the 0.01 level (2-tailed)

Table 4.5 Descriptives for stress/strain measures *

	Range		Mean (SD)		
Variable	Minimum	Maximum	Low Stress (n = 18)	High Stress (n = 13)	All
CES-D	0	27	6.1 (3.1)	19.5 (6.8)	11.7 (8.3)
USQ	7	50	23.9 (11.5)	28.0 (10.0)	25.5 (10.9)
PSS (online)	3	22	8.9 (3.3)	20.9 (1.1)	13.9 (6.6)
PSS (first visit)	3	27	11.1 (5.5)	20.2 (4.3)	14.9 (6.8)
PSS (mean)	5	24.5	11.5 (4.0)	20.8 (2.7)	15.4 (5.8)
Negative Affect	10	24	13.3 (3.3)	14.8 (3.0)	14.0 (3.3)

* Low vs high stress groupings determined by online PSS screening score.

Table 4.6 Physical and performance descriptives for the current sample (n = 31) and for a group of physical activity class students (n = 380)

Current Sample	Women		Men		All	
Variable	M	SD	M	SD	M	SD
Weight (kg)	62.003	14.198	75.013	12.842	71.236	14.326
Fat mass (kg)	19.127	9.706	16.040	9.585	16.936	9.563
Fat Free Mass (kg)	40.587	7.759	56.471	7.905	51.86	10.654
DXA Body Fat (%)	31.033	8.857	21.127	10.024	24.003	10.590
Heart Rate Reserve (bpm)	120.111	11.634	125.273	13.548	123.774	13.048
VO2max (l/min)	1.979	0.290	2.888	0.616	2.624	0.681
VO2max (ml/kg/min)	32.474	6.254	39.165	8.824	37.223	8.629
Leg Press 1-RM (kg)	160.054	42.743	277.252	58.749	243.227	76.338
Bench Press 1-RM (kg)	39.217	17.376	72.43	16.310	62.787	22.396
Jump Power (Watts)	2680.904	1258.799	4089.336	680.211	3680.437	1081.095
Max Jump Height (cm)	35.719	13.836	46.124	9.611	43.349	11.619
Max Cycling Power (watts)	1047.111	250.994	1445.810	307.099	1326.200	341.984
Max Isometric Force (kg)	216.770	74.032	324.256	92.752	293.051	99.716
Phys. Activity Class Sample						
	Women		Men		All	
Variable	M	SD	M	SD	M	SD
VO2max (ml/kg/min)	37.262	6.141	44.899	7.627	40.792	7.847
Leg Press 1-RM (kg)	180.549	60.805	332.495	89.614	250.972	106.986
Jump Power (Watts)	2658.251	627.759	4506.418	838.179	3541.102	1180.865

Table 4.7 Correlations between physical characteristics, USQ, and PSS at the first visit

	USQ	PSS at First Visit
Anthropometrics		
Mass	-.270	-.088
Fat Mass	.053	.240
Fat Free Mass	-.449	-.317
% Body Fat	.191	.360 *
Fitness		
Heart Rate Reserve	-.269	-.126
Absolute VO2max	-.146	-.187
Relative VO2max	.086	-.129
Leg Press 1-RM	-.236	-.078
Bench Press 1-RM	-.376 *	-.218
Jump Power	-.348	-.245
Jump Height	-.203	-.210
Max Cycling Power	-.259	-.142
Max Isometric Force	-.400 *	-.240
Workout Workload		
Workload (kg)	.076	.198
Workload/BdyMass (kg)	.401 *	.401 *
Workload/FFM (kg)	.433 *	.461 **
Repetitions	.469 **	.492 **
MIF force reduction	-.263	-.194
Peak HeartRate	-.076	-.131
Ave HR Over Last 6 Sets	.031	-.032

* Correlation is significant at the .05 level (2-tailed)

** Correlation is significant at the .01 level (2-tailed)

Table 4.8 HLM level 1 descriptives for four days of recovery

Variable	N	Mean	SD	Minimum	Maximum
Maximal isometric force (MIF, kg)	138	271.9	101.6	105.2	547.9
Peak jump height (cm)	99	44.3	11.3	24.1	69.9
Maximal cycling power (watts)	108	1350.6	335.6	740.0	2247.0
Physical energy (VAS)	136	50.8	19.0	2.3	97.4
Physical fatigue (VAS)	136	45.2	24.4	0.0	91.5
Soreness (VAS)	136	53.1	26.5	0.0	100.0
Hours	141	45.5	34.6	1.0	114.0
Hours awake	105	3.6	2.2	0.5	10.0
Hours out of bed	105	3.2	2.2	0.5	9.7
Daily stress	107	4.6	2.2	1.0	9.0
Mental energy (VAS)	136	53.8	19.5	6.2	95.8
Mental fatigue (VAS)	136	42.6	23.8	0.0	97.7
Muscle pain during MIF	137	2.7	2.4	0.0	10.0

Table 4.9 HLM level 1 descriptives for during the E-RES workout

Variable	N	Mean	SD	Minimum	Maximum
Load for set (kg)	404	134.22	59.05	20.40	321.85
Repetitions	404	10.54	2.82	1.00	24.00
Heart rate	354	138.09	28.32	59.00	191.00
RPE	300	8.14	2.55	0.50	10.00
Feeling	285	1.05	2.14	-5.00	5.00
Activation/Arousal	285	3.71	1.40	1.00	6.00
Muscular pain	344	4.72	3.60	0.00	15.00

Table 4.10 Relation of life event stress to four days of recovery for maximal isometric force (MIF; kg) *

Fixed Effect	Coefficient	se	t-ratio	df	p Value
Intercept, P0					
Base, B00	302.895	44.190	6.854	28	.000
USQ, B01	-2.506	1.600	-1.566	28	.128
Time slope, P1					
Base, B10	3.210	0.777	4.130	28	.000
USQ, B11	-0.066	0.028	-2.335	28	.027
Time squared slope, P2					
Base, B20	-0.024	0.007	-3.257	129	.002
USQ, B21	0.001	0.000	2.173	129	.031

Random Effect	Standard deviation	Variance component	df	χ^2	p Value
Intercept, R0	87.918	7729.660	28	339.221	.000
Time slope , R1	0.072	0.005	28	20.537	> .500
Level-1, E	32.842	1078.569			

* After eliminating effect of USQ on the intercept (P0), B11 improves to .010 and B21 improves to .011.

Table 4.11 Relation of life event stress to 4 days of recovery for maximal isometric force (MIF; kg) adjusting for significant covariates *

Fixed Effect	Coefficient	se	t-ratio	df	p Value
Intercept, P0					
Base, B00	235.176	15.342	15.329	28	.000
Muscular Fitness, B01	0.746	0.229	3.261	28	.003
Time slope, P1					
Base, B10	2.677	0.771	3.472	26	.002
USQ, B11	-0.060	0.027	-2.265	26	.032
MIF disruption, B12	1.320	0.471	2.804	26	.010
Finals, B13	-0.356	0.186	-1.914	26	.066
Time squared slope, P2					
Base, B20	-0.024	0.007	-3.608	127	.001
USQ, B21	0.001	0.000	2.333	127	.021

Random Effect	Standard deviation	Variance component	df	χ^2	p Value
Intercept, R0	78.037	6089.836	28	316.906	.000
Time slope , R1	0.082	0.007	26	11.430	> .500
Level-1, E	31.411	986.659			

* After eliminating the effect of Finals on the time slope (P1), B11 p Value improves to .024.

Table 4.12 Relation of stress/strain measures with physical energy in the four days after workout for USQ.

Fixed Effect	Coefficient	se	t-ratio	df	p Value
Intercept, P0					
Base, B00	44.884	6.985	6.426	28	.000
USQ, B01	-0.077	0.254	-0.303	28	.764
Time slope, P1					
Time slope, B10	0.379	0.098	3.877	129	.000
USQ, B11	-0.007	0.004	-2.090	129	.038

Random Effect	Standard deviation	Variance component	df	χ^2	p Value
Intercept, R0	8.986	80.755	28	73.021	.000
Level-1, E	14.799	219.007			

*B11 improves to .015 with removal of B01.

Table 4.13 Relation of USQ with physical fatigue in the four days after workout

Fixed Effect	Coefficient	se	t-ratio	df	p Value
Intercept, P0					
Base, B00	61.600	7.740	7.959	28	.000
USQ, B01	0.048	0.281	0.171	28	.866
Time slope, P1					
Time slope, B10	-0.594	0.115	-5.167	28	.000
USQ, B11	0.009	0.004	2.150	28	.040

Random Effect	Standard deviation	Variance component	df	χ^2	p Value
Intercept, R0	11.147	124.264	28	53.335	.003
Time slope , R1	0.113	0.013	28	37.722	.104
Level-1, E	15.015	225.454			

*When USQ is eliminated from the intercept equation (P0), the B11 p Value reduces to .015.

Table 4.14 Relation of USQ with soreness in the four days after workout

Fixed Effect	Coefficient	se	t-ratio	df	p Value
Intercept, P0					
Base, B00	61.557	3.832	16.064	29	.000
Time slope, P1					
Base, B10	-0.420	0.333	-1.261	28	.218
USQ, B11	0.027	0.012	2.332	28	.027
Time squared slope, P2					
Base, B20	-0.000	0.003	-0.044	128	.965
USQ, B21	-0.000	0.000	-1.962	128	.052

Random Effect	Standard deviation	Variance component	df	χ^2	p Value
Intercept, R0	12.656	160.164	29	53.340	.004
Time slope , R1	0.134	0.018	28	39	.073
Level-1, E	16.837	283.491			

Table 4.15 Relation of perceived stress (visit 1) to four days of recovery for maximal isometric force (MIF; kg)

Fixed Effect	Coefficient	se	t-ratio	df	p Value
Intercept, P0					
Base, B00	271.240	43.551	6.228	29	.000
PSS at V1, B01	-2.475	2.676	-0.925	29	.363
Time slope, P1					
Base, B10	3.500	0.666	5.254	29	.000
PSS at V1, B11	-0.138	0.042	-3.292	29	.003
Time squared slope, P2					
Base, B20	-0.027	0.006	-4.402	132	.000
PSS at V1, B21	0.001	0.000	3.202	132	.002

Random Effect	Standard deviation	Variance component	df	χ^2	p Value
Intercept, R0	93.912	8819.392	29	392.300	.000
Time slope , R1	0.098	0.010	29	23.554	> 0.500
Level-1, E	31.800	1011.017			

Table 4.16 Relation of perceived stress (mean) to four days of recovery for maximal isometric force (MIF; kg)

Fixed Effect	Coefficient	se	t-ratio	df	p Value
Intercept, P0					
Base, B00	277.068	50.630	5.472	29	.000
Mean PSS, B01	-2.760	3.089	-0.893	29	.379
Time slope, P1					
Base, B10	4.493	0.797	5.641	29	.000
Mean PSS, B11	-0.197	0.050	-3.979	29	.001
Time squared slope, P2					
Base, B20	-0.366	0.008	-4.855	132	.000
Mean PSS, B21	0.002	0.000	3.859	132	.000

Random Effect	Standard deviation	Variance component	df	χ^2	p Value
Intercept, R0	93.063	8660.660	29	411.793	.000
Time slope , R1	0.105	0.011	29	426.000	> 0.500
Level-1, E	31.142	969.844			

Table 4.17 Relation of perceived stress (mean) to four days of recovery for maximal isometric force (MIF; kg)

Fixed Effect	Coefficient	se	t-ratio	df	p Value
Intercept, P0					
Base, B00	281.429	50.717	5.549	29	.000
Mean PSS, B01	-3.185	3.093	-1.030	29	.312
Time slope, P1					
Base, B10	8.858	2.387	3.711	29	.001
Mean PSS, B11	-0.546	0.152	-3.599	29	.001
Time squared slope, P2					
Base, B20	-0.065	0.016	-4.032	130	.000
Mean PSS, B21	0.004	0.001	3.962	130	.000
Time LogN slope, P3					
Base, B30	-35.311	18.970	-1.861	130	.064
Mean PSS, B31	2.872	1.120	2.402	130	.018

Random Effect	Standard deviation	Variance component	df	χ^2	p Value
Intercept, R0	93.303	8705.458	29	437.309	.000
Time slope , R1	0.123	0.015	29	24.552	> .500
Level-1, E	30.259	915.585			

Table 4.18 Relation of perceived stress (average over all visits) to 4 days of recovery for maximal isometric force (MIF; kg) adjusting for significant covariates

Fixed Effect	Coefficient	se	t-ratio	df	p Value
Intercept, P0					
Base, B00	233.674	14.858	15.727	29	.000
Muscular Fitness, B01	0.765	0.209	3.666	29	.001
Time slope, P1					
Base, B10	8.101	2.286	3.544	27	.002
Mean PSS, B11	-0.507	0.145	-3.485	27	.002
MIF disruption, B12	1.408	0.442	3.183	27	.004
Finals, B13	-0.346	0.187	-1.852	27	.074
Time squared slope, P2					
Base, B20	-0.065	0.015	-4.241	128	.000
Mean PSS, B21	0.004	0.001	4.004	128	.000
Time LogN slope, P3					
Base, B30	-35.862	18.054	-1.986	128	.049
Mean PSS, B31	2.740	1.138	2.407	128	.018

Random Effect	Standard deviation	Variance component	df	χ^2	p Value
Intercept, R0	77.142	5950.940	29	380.381	.000
Time slope , R1	0.119	0.014	27	15.421	> .500
Level-1, E	28.841	831.850			

Table 4.19 Relation of perceived stress (first visit) to 4 days of recovery for maximal isometric force (MIF; kg) adjusting for significant covariates

Fixed Effect	Coefficient	se	t-ratio	df	p Value
Intercept, P0					
Base, B00	235.201	14.798	15.894	29	.000
Muscular Fitness, B01	0.782	0.207	3.772	29	.001
Time slope, P1					
Base, B10	2.874	0.655	4.389	27	.000
PSS at V1, B11	-0.126	0.039	-3.202	27	.004
MIF disruption, B12	1.428	0.446	3.204	27	.004
Finals, B13	-0.371	0.183	-2.035	27	.051
Time squared slope, P2					
Base, B20	-0.028	0.006	-4.876	130	.000
PSS at V1, B21	0.001	0.000	3.497	130	.001

Random Effect	Standard deviation	Variance component	df	χ^2	p Value
Intercept, R0	76.742	5889.340	29	343.277	.000
Time slope , R1	0.088	0.008	27	12.834	> .500
Level-1, E	30.144	908.672			

Table 4.20 Relation of perceived stress (average over all visits) to 4 days of recovery for vertical jump height (cm)

Fixed Effect	Coefficient	se	t-ratio	df	p Value
Intercept, P0					
Base, B00	48.176	6.513	7.397	28	.000
Mean PSS, B01	-0.454	0.407	-1.117	28	.274
Time slope, P1					
Base, B10	0.319	0.104	3.069	28	.005
Mean PSS, B11	-0.150	0.007	-2.209	28	.035
Time squared slope, P2					
Base, B20	-0.002	0.001	-3.011	93	.004
Mean PSS, B21	0.000	0.000	2.396	93	.019

Random Effect	Standard deviation	Variance component	df	χ^2	p Value
Intercept, R0	11.373	129.347	27	934.255	.000
Time slope , R1	0.043	0.002	27	86.027	.000
Level-1, E	1.490	2.219			

Table 4.21 Relation of perceived stress (average over all visits) to 4 days of recovery for vertical jump height (cm)

Fixed Effect	Coefficient	se	t-ratio	df	p Value
Intercept, P0					
Base, B00	40.931	2.092	19.565	25	.000
Muscular Fitness, B01	0.085	0.027	3.165	25	.004
Time slope, P1					
Base, B10	0.332	0.099	3.367	23	.003
Mean PSS, B11	-0.015	0.006	-2.428	23	.024
Jump disruption, B12	0.054	0.078	0.692	23	.496
Finals, B13	-0.023	0.021	-1.099	23	.284
Time squared slope, P2					
Base, B20	-0.002	0.001	-3.099	80	.003
Mean PSS, B21	0.000	0.000	2.486	80	.015

Random Effect	Standard deviation	Variance component	df	χ^2	p Value
Intercept, R0	9.937	98.737	24	639.985	.000
Time slope , R1	0.038	0.001	22	54.395	.000
Level-1, E	1.565	2.450			

Table 4.22 Relation of perceived stress at the first visit to feeling (pleasure) during the E-RES workout *

Fixed Effect	Coefficient	se	t-ratio	df	p Value
Intercept, P0					
Base, B00	3.895	0.674	5.776	25	.000
PSS at V1, B01	-0.112	0.040	-2.777	25	.011
Sets squared slope, P1					
Base, B10	-0.061	0.014	-4.313	25	.000
PSS at V1, B11	0.002	0.001	2.928	25	.008
Sets cubed slope, P2					
Base, B20	0.003	0.001	3.000	279	.003
PSS at V1, B21	-0.000	0.000	-2.473	279	.014

Random Effect	Standard deviation	Variance component	df	χ^2	p Value
Intercept, R0	1.385	1.918	25	391.616	.000
Time slope , R1	0.016	0.000	25	315.654	.000
Level-1, E	0.725	0.525			

* Linear time was not a significant functional form of time and was not modeled.

Table 4.23 Relation of perceived stress at the first visit to activation (arousal) during the E-RES workout *

Fixed Effect	Coefficient	se	t-ratio	df	p Value
Intercept, P0					
Base, B00	1.690	0.900	1.878	25	.072
PSS at V1, B01	0.101	0.053	1.892	25	.070
Sets squared slope, P1					
Base, B10	0.032	0.008	4.094	25	.000
PSS at V1, B11	-0.001	0.000	-2.624	25	.015
Sets cubed slope, P2					
Base, B20	0.002	0.000	4.077	279	.000
PSS at V1, B21	0.000	0.000	-2.128	279	.034

Random Effect	Standard deviation	Variance component	df	χ^2	p Value
Intercept, R0	1.872	3.503	25	1599.216	.000
Time slope , R1	0.009	0.000	25	615.442	.000
Level-1, E	0.487	0.237			

* Linear time was not a significant functional form of time and was not modeled.

* The intercept is recoded for the last set.

Table 4.24 Relation of perceived stress at the first visit to muscular pain during the E-RES workout *

Fixed Effect	Coefficient	se	t-ratio	df	p Value
Intercept, P0					
Base, B00	1.191	1.004	1.186	29	.246
PSS at V1, B01	0.020	0.062	0.327	29	.746
Sets squared slope, P1					
Base, B10	0.207	0.021	9.725	29	.000
PSS at V1, B11	-0.006	0.001	-5.169	29	.000
Sets cubed slope, P2					
Base, B20	-0.013	0.002	-8.421	338	.000
PSS at V1, B21	0.000	0.000	5.186	338	.000

Random Effect	Standard deviation	Variance component	df	χ^2	p Value
Intercept, R0	2.103	4.424	29	327.367	.000
Time slope , R1	0.022	0.000	29	225.999	.000
Level-1, E	1.357	1.841			

* Linear time was not a significant functional form of time and was not modeled.

Table 4.25 Relation of perceived stress at the first visit to RPE during the E-RES workout *

Fixed Effect	Coefficient	se	t-ratio	df	p Value
Intercept, P0					
Base, B00	4.128	0.592	6.971	25	.000
PSS at V1, B01	0.033	0.035	0.927	25	.363
Sets squared slope, P1					
Base, B10	0.191	0.025	7.742	25	.000
PSS at V1, B11	-0.003	0.001	-2.442	25	.022
Sets cubed slope, P2					
Base, B20	-0.013	0.002	-6.777	294	.000
PSS at V1, B21	0.000	0.000	2.732	294	.007

Random Effect	Standard deviation	Variance component	df	χ^2	p Value
Intercept, R0	0.719	0.516	25	41.289	.000
Time squared slope, R1	0.009	0.000	25	51.235	.000
Level-1, E	1.618	2.619			

* Linear time was not significant and was dropped from the analysis.

Table 4.26 Relation of stress/strain measures with physical energy in the four days after workout for PSS at the first

Fixed Effect	Coefficient	se	t-ratio	df	p Value
Model for relation of PSS at V1					
Intercept, P0					
Base, B00	34.987	6.802	5.144	29	.000
PSS at V1, B01	0.517	0.420	1.231	29	.229
Time slope, P1					
Time slope, B10	0.397	0.088	4.528	132	.000
PSS at V1, B11	-0.015	0.005	-2.648	132	.009

Random Effect	Standard deviation	Variance component	df	χ^2	p Value
Intercept, R0	10.629	112.981	29	96.269	.000
Level-1, E	14.467	209.306			

Model for relation of Mean PSS over all visits					
Intercept, P0					
Base, B00	34.356	7.985	4.303	29	.000
Mean PSS, B01	0.541	0.490	1.104	29	.279
Time slope, P1					
Time slope, B10	0.483	0.105	4.591	132	.000
Mean PSS, B11	-0.020	0.006	-3.003	132	.004

Random Effect	Standard deviation	Variance component	df	χ^2	p Value
Intercept, R0	10.632	113.048	29	97.420	.000
Level-1, E	14.323	205.134			

Table 4.27 Relation of perceived stress with physical fatigue in the four days after workout

Fixed Effect	Coefficient	se	t-ratio	df	p Value
Model for relation of PSS at V1					
Intercept, P0					
Base, B00	59.600	7.378	8.078	29	.000
PSS at V1, B01	0.168	0.455	0.370	29	.714
Time slope, P1					
Time slope, B10	-0.460	0.114	-4.042	29	.000
PSS at V1, B11	0.007	0.007	0.997	29	.327

Random Effect	Standard deviation	Variance component	df	χ^2	p Value
Intercept, R0	12.022	144.520	29	57.457	.002
Time slope , R1	0.145	0.021	29	43.675	.039
Level-1, E	14.933	222.982			

Model for relation of Mean PSS over all visits					
Intercept, P0					
Base, B00	63.46	8.731	7.269	29	.000
Mean PSS, B01	-0.089	0.535	-0.166	29	.869
Time slope, P1					
Time slope, B10	-0.520	0.136	-3.828	29	.001
Mean PSS, B11	0.011	0.008	1.283	29	.210

Random Effect	Standard deviation	Variance component	df	χ^2	p Value
Intercept, R0	12.130	147.146	29	57.454	.002
Time slope , R1	0.141	0.020	29	42.946	.046
Level-1, E	14.921	222.650			

Table 4.28 Relation of finals (average over four visits) to four days of recovery for maximal isometric force (MIF; kg)

Fixed Effect	Coefficient	se	t-ratio	df	p Value
Intercept, P0					
Base, B00	239.114	23.000	10.396	29	.000
Finals, B01	-14.946	38.725	-0.386	29	.702
Time slope, P1					
Base, B10	1.869	0.331	5.638	29	.000
Finals, B11	-1.048	0.620	-1.691	29	.101
Time squared slope, P2					
Base, B20	-0.011	0.003	-3.734	132	.000
Finals, B21	0.007	0.006	1.202	132	.232

Random Effect	Standard deviation	Variance component	df	χ^2	p Value
Intercept, R0	97.728	9550.710	29	399.475	.000
Time slope , R1	0.059	0.003	29	20.229	> .500
Level-1, E	32.766	1073.636			

Table 4.29 Relation of being in the finals period to 4 days of recovery for maximal cycling power (watts)

Fixed Effect	Coefficient	se	t-ratio	df	p Value
Intercept, P0					
Base, B00	1293.381	61.140	21.154	30	.000
Time squared slope, P1					
Base, B10	0.020	0.003	6.585	105	.000
Finals, B11	-0.013	0.006	-2.074	105	.040

Random Effect	Standard deviation	Variance component	df	χ^2	p Value
Intercept, R0	330.535	109253.219	30	1207.599	.000
Level-1, E	95.179	9059.120			

* Linear time was not a significant functional form of time and was not modeled.

Table 4.30 Relation of finals with physical energy in the four days after workout

Fixed Effect	Coefficient	se	t-ratio	df	p Value
Intercept, P0					
Base, B00	39.013	3.333	11.705	29	.000
Finals, B01	10.366	5.808	1.785	29	.084
Time slope, P1	0.217	0.050	4.309	29	.000
Time slope, B10	-0.112	0.091	-1.234	29	.227
Finals, B11					

Random Effect	Standard deviation	Variance component	df	χ^2	p Value
Intercept, R0	9.773	95.513	29	48.402	.013
Time slope , R1	0.110	0.012	29	38.891	.104
Level-1, E	14.313	204.849			

Table 4.31 Relation of finals with physical fatigue in the four days after workout.

Fixed Effect	Coefficient	se	t-ratio	df	p Value
Intercept, P0					
Base, B00	65.684	3.700	17.752	29	.000
Finals, B01	-11.056	6.420	-1.722	29	.095
Time slope, P1					
Time slope, B10	-0.429	0.052	-8.263	29	.000
Finals, B11	0.232	0.094	2.473	29	.020

Random Effect	Standard deviation	Variance component	df	χ^2	p Value
Intercept, R0	11.786	138.918	29	54.397	.003
Time slope , R1	0.111	0.012	29	36.708	.154
Level-1, E	14.780	218.461			

Table 4.32 Relation of finals with soreness in the four days after workout

Fixed Effect	Coefficient	se	t-ratio	df	p Value
Intercept, P0					
Base, B00	65.508	4.924	13.305	29	.000
Finals, B01	-13.293	8.592	-1.547	29	.132
Time slope, P1					
Base, B10	0.092	0.169	0.546	29	.589
Finals, B11	0.678	0.327	2.072	29	.047
Time squared slope, P2					
Base, B20	-0.005	0.002	-3.148	130	.002
Finals, B21	-0.005	0.003	-1.684	130	.094

Random Effect	Standard deviation	Variance component	df	χ^2	p Value
Intercept, R0	15.189	230.716	29	64.523	.000
Time slope , R1	0.127	0.016	29	40.723	.073
Level-1, E	16.240	263.726			

Table 4.33 HLM level 1 descriptives for the first hour of recovery

Variable	N	Mean	SD	Minimum	Maximum
Maximal isometric force (MIF, kg)	121	210.11	90.21	49.00	479.79
Physical energy (VAS)	117	35.69	21.44	1.96	86.60
Physical fatigue (VAS)	117	72.28	16.23	8.50	99.35
Soreness (VAS)	117	65.11	19.63	4.90	100.00
Mental energy	117	46.14	22.17	2.61	88.56
Mental fatigue	117	58.52	21.14	6.21	98.04
Muscle pain during MIF	117	3.56	2.81	0.00	11.00

Table 4.34 Relation of USQ (life event stress) to the first hour of recovery for maximal isometric force (MIF; kg)

Fixed Effect	Coefficient	se	t-ratio	df	p Value
Intercept, P0					
Base, B00	190.056	34.767	5.467	28	.000
USQ, B01	-0.936	1.255	-0.746	28	.462
Time slope, P1					
Base, B10	288.196	53.403	5.397	28	.000
USQ, B11	-5.169	1.939	-2.666	28	.013
Time squared slope, P2					
Base, B20	-170.100	46.069	-3.692	113	.001
USQ, B21	3.353	1.700	1.975	113	.050

Random Effect	Standard deviation	Variance component	df	χ^2	p Value
Intercept, R0	70.958	5035.033	28	479.964	.000
Time slope , R1	52.815	2789.371	28	124.410	.000
Level-1, E	21.115	445.839			

Table 4.35 Relation of USQ (life event stress) to first hour of recovery in maximal isometric force (MIF; kg) after adjusting for covariates

Fixed Effect	Coefficient	se	t-ratio	df	p Value
Intercept, P0					
Base, B00	164.461	12.101	13.591	28	.000
Muscular Fitness, B01	0.423	0.182	2.330	28	.027
Time slope, P1					
Base, B10	179.923	84.000	2.142	26	.042
USQ, B11	-6.376	1.818	-3.508	26	.002
Workload, B12	0.010	0.003	3.126	26	.005
Semester, B13	-44.907	17.082	-2.629	26	.015
Time squared slope, P2					
Base, B20	-19.953	64.763	-0.308	110	.758
USQ, B21	3.846	1.588	2.422	110	.017
Workload, B22	-0.009	0.003	-3.076	110	.003

Random Effect	Standard deviation	Variance component	df	χ^2	p Value
Intercept, R0	63.268	4002.805	28	434.872	.000
Time slope , R1	46.712	2181.960	26	105.392	.000
Level-1, E	19.849	393.962			

Table 4.36 Relation of perceived stress (first visit) to first hour of recovery in maximal isometric force (MIF; kg) after adjusting for covariates *

Fixed Effect	Coefficient	se	t-ratio	df	p Value
Intercept, P0					
Base, B00	164.155	11.707	14.022	29	.000
Muscular Fitness, B01	0.451	0.166	2.726	29	.011
Time slope, P1					
Base, B10	65.696	76.590	0.858	27	.399
PSS at V1, B11	-7.131	2.959	-2.410	27	.023
Workload, B12	0.012	0.003	3.665	27	.001
Semester, B13	-28.257	17.161	-1.647	27	.111
Time squared slope, P2					
Base, B20	29.858	61.909	0.482	112	.630
PSS at V1, B21	3.987	2.533	1.574	112	.118
Workload, B22	-0.009	0.003	-3.172	112	.002

Random Effect	Standard deviation	Variance component	df	χ^2	p Value
Intercept, R0	62.061	3851.623	29	411.764	.000
Time slope , R1	48.825	2383.890	27	109.948	.000
Level-1, E	20.416	416.818			

* Model is unaffected by utilization of mean perceived stress over 2 days.

B11 p Value associated with mean PSS changes to .025.

B21 p Value associated with mean PSS remains at .118.

Table 4.37 IL-1β, IL-6 and TNFα for low and high stress groups (means and standard deviations). Units are pg/ml.

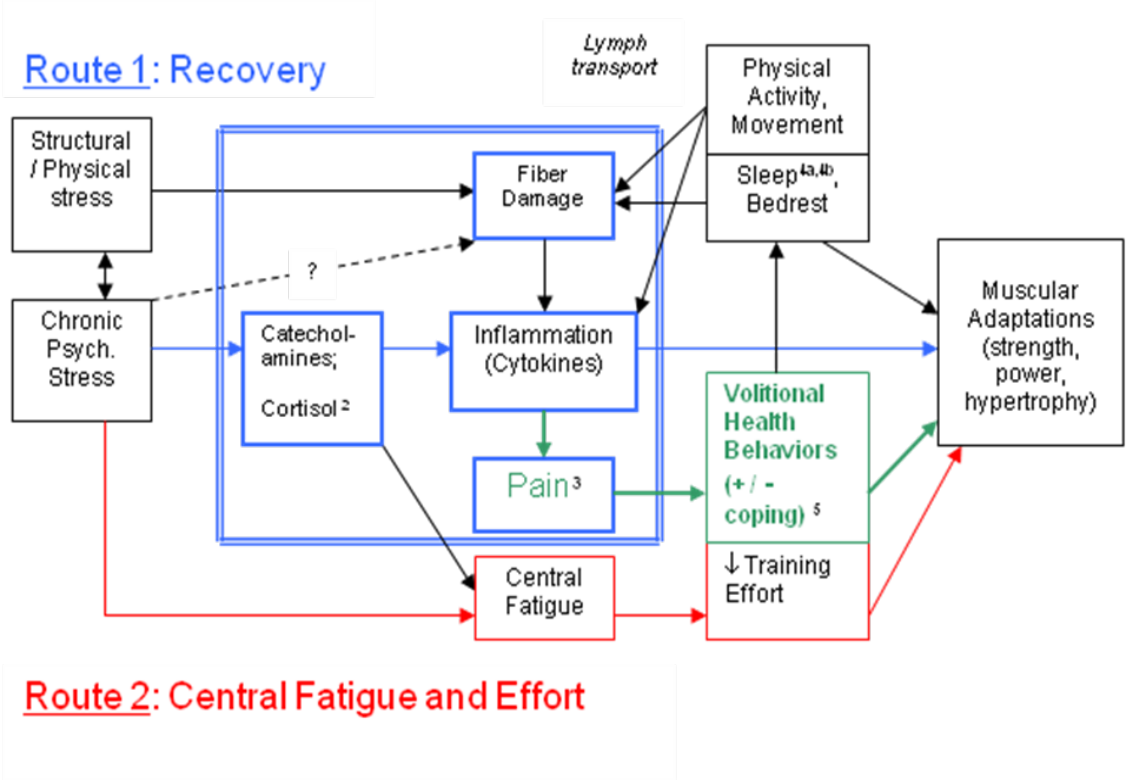
	Pre-Workout	24 hours	48 hours
IL-1 Beta			
Low stress	0.348 (0.035)	0.400 (0.091)	0.387 (0.044)
High stress	0.290 (0.058)	0.299 (0.044)	0.306 (0.042)
All	0.317 (0.056)	0.345 (0.085)	0.343 (0.059)
IL-6			
Low stress	0.465 (0.070)	0.565 (0.115)	0.570 (0.189)
High stress	0.569 (0.230)	0.679 (0.230)	0.561 (0.143)
All	0.521 (0.177)	0.626 (0.188)	0.565 (0.158)
TNF Alpha			
Low stress	2.978 (1.055)	4.583 (2.470)	3.215 (1.473)
High stress	2.647 (1.616)	3.536 (1.993)	2.803 (1.164)
All	2.800 (1.341)	4.019 (2.196)	2.993 (1.276)

Table 4.38 Relation of stress to IL-1β for linear time

Fixed Effect	Coefficient	se	t Ratio	df	p Value
Intercept, P0					
Base, B00	0.309	0.021	14.957	12	.000
Time slope, P1					
Base, B10	0.029	0.010	2.964	36	.006
Stress, B11	-0.030	0.010	-3.078	36	.004

Random Effect	Standard deviation	Variance component	df	X^2	p Value
Intercept, R0	0.036	0.001	12	35.419	.001
Level-1, E	0.043	0.002			

Appendix A – Proposed Model



Proposed Full Model to Explain the Stress/Muscle Adaptation Relationship

A key mediator in the stress and recovery relationship is inflammation (Glaser et al, 1999). Inflammation, controlled through the immune system, is compulsory for the needed repair from local or systemic physical damage. The inflammation response, however, may be most impaired or degraded by the experience of stress (Christian, Graham, Padgett, Glaser, & Kiecolt-Glaser, 2007; Smith, 2000). Cortisol, the primary glucocorticoid, is bi-directionally linked to inflammation (Dhabhar & McEwen, 2001). Dysregulation of inflammation is related to hyper-catabolism (Path et al., 1997) and may explain why glucocorticoids are released in excess after unremitting stress.

Alternative routes explain the impact of pain perception and health behaviors on recovery and physical adaptation. The experience of exorbitant pain may be an indicator of poor adaptation and may also be related to poorer sleep, less movement and poor choice of coping behaviors, including smoking, drug use, and alcohol consumption.

Route 1 (blue paths) demonstrates that stress may directly impair physical recovery through alterations in catecholamines (e.g., norepinephrine)/cortisol and inflammation.

Route 2 (red paths) demonstrates that life event stress may cause central fatigue (Davis & Bailey, 1997; Emerson, Kappenman, Ronnan, Renner & Summers, 2000; Smith, 2000) leading to decreased training effort.

The green paths show how pain may be instrumental in the life event stress/adaptation process by prompting positive and negative volitional health behaviors.

1. Damage due to eccentric exercise does not necessarily lead to an immediate inflammation response (Bruunsgaard et al., 1997 as cited in Pedersen et al., 1998; Nosaka & Clarkson, 1996a).
2. For evidence that catecholamines mediate the stress and cytokine relationship, see Johnson, Campisi, Sharkey et al., (2005). For evidence concerning cortisol, see Pedersen et al., (1998, p. 329) and Padgett et al., (1998).
3. Pain (DOMS) is related to inflammation but not damage to the muscle.
4. 4a. Sleep quality and duration; sleep indicators measured before physical stressor may impact extent of damage and inflammation.
5. 4b. Exercise may allay effects of damage. See Emery et al. (2005).
6. Christian et al. (2007) calls for health behavior measurements.

7. The relationship between stress hormones and inflammation is bi-directional. See Dhabhar & McEwen (2001), Malarkey & Mills (2006), Kunz-Ebrecht et al. (2003) and DeRijk et al. (1997).

Appendix B – PSS

PSS-10 scores are obtained by reversing the scores on the four positive items, e.g., 0=4, 1=3, 2=2, etc. and then summing across all 10 items. Items 4,5, 7, and 8 are the positively stated items.

P. S. S. - 10 Items: The questions in this scale ask you about your feelings and thoughts during the last month. In each case, please indicate by circling how often you felt or thought a certain way.

1. In the last month, how often have you been upset because of something that happened unexpectedly?

0=never 1=almost never 2=sometimes 3=fairly often 4=very often

2. In the last month, how often have you felt that you were unable to control the important things in your life?

0=never 1=almost never 2=sometimes 3=fairly often 4=very often

3. In the last month, how often have you felt nervous and "stressed"?

0=never 1=almost never 2=sometimes 3=fairly often 4=very often

4. In the last month, how often have you felt confident about your ability to handle your personal problems?

0=never 1=almost never 2=sometimes 3=fairly often 4=very often

5. In the last month, how often have you felt that things were going your way?

0=never 1=almost never 2=sometimes 3=fairly often 4=very often

6. In the last month, how often have you found that you could not cope with all the things that you had to do?

0=never 1=almost never 2=sometimes 3=fairly often 4=very often

7. In the last month, how often have you been able to control irritations in your life?

0=never 1=almost never 2=sometimes 3=fairly often 4=very often

8. In the last month, how often have you felt that you were on top of things?

0=never 1=almost never 2=sometimes 3=fairly often 4=very often

9. In the last month, how often have you been angered because of things that were outside of your control?

0=never 1=almost never 2=sometimes 3=fairly often 4=very often

10. In the last month, how often have you felt difficulties were piling up so high that you could not overcome them?

0=never 1=almost never 2=sometimes 3=fairly often 4=very often

Appendix C – Energy and Fatigue: How You Feel Right Now

Directions. This part of the questionnaire asks about your current feelings of energy and fatigue. We are interested in how you feel right now, even if it is different than how you usually feel. Therefore, it is important that you focus on how you feel right now at this moment in responding to each item. There are no right or wrong answers. Please be as honest and accurate as possible in your responses. Make a vertical line through each horizontal line below to indicate the intensity of your current feelings. If you have a complete absence of the feeling described then place a vertical mark at the left edge of the horizontal line. If your feelings are the strongest intensity that you have ever experienced then place a vertical mark at the right edge of the horizontal line. If your feelings are between these two extremes, then use the distance from the left edge to represent the intensity of your feelings.

Example:

I feel I have no energy ——————————— Strongest feelings of energy ever felt

How do you feel right now with regard to your capacity to perform your typical ***PHYSICAL ACTIVITIES***....

I feel I have no energy ——————————— Strongest feelings of energy ever felt

I feel no fatigue ——————————— Strongest feelings of fatigue ever felt

I feel I have no vigor ——————————— Strongest feelings of vigor ever felt

I feel no exhaustion ——————————— Strongest feelings of exhaustion ever felt

I feel I have no pep ——————————— Strongest feelings of pep ever felt

I have no feelings of being worn out ——————————— Strongest feelings of being worn out ever felt

I have no feelings of soreness ——————————— Strongest feelings of soreness ever felt

How do you feel right now with regard to your capacity to perform your typical ***MENTAL ACTIVITIES***

I feel I have no energy	———	Strongest feelings of energy ever felt
I feel no fatigue	———	Strongest feelings of fatigue ever felt
I feel I have no vigor	———	Strongest feelings of vigor ever felt
I feel no exhaustion	———	Strongest feelings of exhaustion ever felt
I feel I have no pep	———	Strongest feelings of pep ever felt
I have no feelings of being worn out	———	Strongest feelings of being worn out ever felt

Note to administrator - horizontal lines must be 10 cm in length, photocopying can change the length

Appendix D – Demographic Questionnaire

Date: _____

ID: _____

Please fill out the following information about yourself. Your confidentiality is ensured. This information will be locked and only accessed by authorized personnel. Please, ask the administrator of these assessments if you have any questions.

1	Gender (circle one)	Female	Male			
2	Age (YEARS)	_____				
3	Ethnicity (circle one)	African-American / Black	Asian / Pacific Islander	Caucasian / White	Hispanic / Latino / Latina	Other
4a	Do you work outside of your studies (circle one)	YES	NO			
4b	If 'YES' to question 4a, how many hours do you work per week (to the nearest hour)?	_____				
5	Do you or your parents take out student loans to support your studies?	YES	NO	I DON'T KNOW		
6	How many semesters have you taken this P.Ed.106C weightlifting course? (Circle one)	1	2	3	4	5

Appendix E – Health Questionnaire

Please answer the following questions to the best of your knowledge by checking yes, no or unknown. **This questionnaire is completely confidential** and will only be examined by the researchers involved in this study.

Section 1	YES	NO	Unknown
1 Has a doctor ever said that you have a heart condition and recommended only medically supervised physical activity?	_____	_____	_____
2 Do you have chest pain brought on by physical activity?	_____	_____	_____
3 Have you developed chest pain in the last month when not doing physical activity?	_____	_____	_____
4 Do you lose your balance because of dizziness or do you ever lose consciousness?	_____	_____	_____
5 Has a doctor ever recommended medication for your blood pressure or a heart condition?	_____	_____	_____
6 Are you aware, through your own experience, a doctor's advice, or any other physical reason that would prohibit you from engaging in physical activity?	_____	_____	_____
Section 2			
7 Is your blood cholesterol level >240 mg/dl?	_____	_____	_____
8 Do you have a close relative who has had a heart attack or sudden death before age 55 (father or brother) or age 65 (mother or sister)?	_____	_____	_____
Section 3			
9 Have you ever experienced pain or discomfort in the	_____	_____	_____

	chest, neck, jaw, arm, or other areas of your body that indicate lack of blood flow to the heart?			
10	Do you ever experience shortness of breath at rest or with mild physical activity?	_____	_____	_____
11	Do you ever experience shortness of breath while lying flat or wake up in the middle of the night with shortness of breath?	_____	_____	_____
12	Do you ever experience palpitations of your heart or a very rapid heart rate with mild exertion?	_____	_____	_____
13	Do you ever experience unusual fatigue or shortness of breath with usual daily activities?	_____	_____	_____
14	Do you ever experience pain in your legs while exercising that is relieved by rest?	_____	_____	_____
Section 4				
	Have you recently had:	YES	NO	Unknown
15	Muscle Trauma	_____	_____	_____
16	Muscle tears	_____	_____	_____
17	Edema (swelling) of ankles/joints	_____	_____	_____
18	Tendonitis	_____	_____	_____
19	Knee/joint problems of any kind	_____	_____	_____
Section 5				
	Have you ever had:			
20	Coronary heart disease, heart attack, coronary artery surgery	_____	_____	_____
21	Angina	_____	_____	_____
22	High blood pressure	_____	_____	_____
23	Peripheral vascular disease	_____	_____	_____

24	Stroke	_____	_____	_____
25	Heart Murmur	_____	_____	_____
26	Diabetes	_____	_____	_____
27	Thyroid problems	_____	_____	_____
28	Hepatitis	_____	_____	_____
29	Arthritis	_____	_____	_____
30	Gout	_____	_____	_____
31	Chronic and Severe Headaches	_____	_____	_____
32	Head injury or epilepsy	_____	_____	_____
33	Abdominal pain, hernia, or G.I. bleeding	_____	_____	_____
34	Kidney problems or discomfort when urinating	_____	_____	_____
35	Tendency to bleed or bruise easily	_____	_____	_____
36	Anemia	_____	_____	_____
37	Lung problems	_____	_____	_____
38	Liver problems	_____	_____	_____
39	Shingles	_____	_____	_____
		YES	NO	Unknown
40	Have you donated or lost a lot of blood from injury in the last two weeks?	_____	_____	_____

Section 6

Have you ever had:	YES	NO	Unknown
41 Blood clots	_____	_____	_____
42 Compartment syndrome	_____	_____	_____
43 Fasciotomy	_____	_____	_____
44 Heat stroke	_____	_____	_____
45 Heat stress/illness history	_____	_____	_____
46 Myoglobinuria	_____	_____	_____
47 Sickle Cell Trait	_____	_____	_____
48 Myalgia: Chronic muscle pain			

Section 7

Have you recently had:	YES	NO	Unknown
49 Cold or flu-like symptoms	_____	_____	_____
50 Other acute, short-term illness	_____	_____	_____
51 Soft tissue Injury	_____	_____	_____

52 How many times have you seen the doctor in the last 12 months? _____

53 How many colds or upper respiratory infections have you had in the last 12 months? _____

54 How many days have you missed work/class due to illness in the last year? _____

		YES	NO	Unknown
55	Can you think of *any other conditions* that would be aggravated by maximal exercise?	_____	_____	_____
56	Can you think of any other conditions that may impair your ability to fully train, adapt properly to your training, or perform fitness/ strength testing (e.g., neurological, neuromuscular problems)?	_____	_____	_____

57 If ‘YES” to either, explain:

_____

Section 8		YES	NO	Unknown
58	Bleeding gums	_____	_____	_____
59	Recent dental work	_____	_____	_____
Section 9: Medications		YES	NO	Unknown
60	Allergy	_____	_____	_____
61	Hormones	_____	_____	_____
62	Anti-depressants	_____	_____	_____
63	Anti-anxiolytic (anxiety)	_____	_____	_____
64	Anti-inflammatory	_____	_____	_____
65	Pain medications	_____	_____	_____
66	Antibiotics	_____	_____	_____
67	Sleep Aids (eg, Ambien, Lunesta)	_____	_____	_____

68 Please list all medications (prescription and over-the-counter) you are currently taking:

_____
_____
_____
_____
_____
_____

69 Please list all vitamins and supplements you are currently taking (list amount):

_____
_____

70 What is your normal caffeine intake? (list # of cups/ bottles/cans):

_____
_____

71 Please list all ergogenic aids (performance or sports enhancing), including those with stimulants (e.g., ephedrine):

_____
_____

	YES	NO
72 Have you had any abrupt changes in diet to either gain or lose weight?	_____	_____ _

Section 10: Women only	YES	NO	Unknown
73 Are you or have you been pregnant in the last month?	_____	_____	_____
74 Are you in the luteal / quiescent phase of your menstrual cycle?	_____	_____	_____
75 How many days has it been since the end of your last period?	_____	_____	_____

Appendix F – PRETIE-Q–TOL

		1 I totally disagree	2 I disagree	3 Neither agree or disagree	4 I agree	5 I strongly agree
1	Feeling tired during exercise is my signal to slow down or stop	1	2	3	4	5
2	During exercise, if my muscles begin to burn excessively or if I find myself breathing very hard, it is time for me to ease off.	1	2	3	4	5
3	While exercising, I try to keep going even after I feel exhausted.	1	2	3	4	5
4	I block out the feeling of fatigue when exercising.	1	2	3	4	5
5	I'd rather slow down or stop when a workout starts to get too tough.	1	2	3	4	5
6	Fatigue is the last thing that affects when I stop a workout; I have a goal and stop only when I reach it.	1	2	3	4	5
7	When my muscles start burning during exercise, I usually ease off some.	1	2	3	4	5
8	I always push through muscle soreness and fatigue when working out.	1	2	3	4	5

Appendix G – PAIN

Instructions:

“You are about to undergo an exercise test. The scale before you contains the numbers 0-10. You will use this scale to assess perceptions of pain in your legs during and after the exercise test. In this context, pain is defined as the intensity of hurt that you feel. Don't underestimate or overestimate the degree of hurt you feel, just try to estimate it as honestly and objectively as possible.

‘The numbers on the scale represent a range of pain intensity from very faint pain (number 1/2) to extremely intense pain-almost unbearable (number 10). When you feel no pain in your legs, you should respond with the number 0. If your legs feel extremely strong pain that is almost unbearable, you should respond with the number 10. If the pain is greater than 10, respond with the number that represents the pain intensity you feel in relation to 10. For example, if the pain is twice as intense as 10 give the number 20.

‘Repeatedly during the test you will be asked to rate the feelings of pain in your legs. When rating these pain sensations be sure to attend only to the specific sensations in your legs and not report other pains you may be feeling (e.g., seat discomfort).

‘It is very important that your ratings of pain intensity reflect only the degree of hurt you are feeling in your legs either during exercise or following exercise as pain perceptions are abating. Do not use your ratings as an expression of fatigue (i.e., inability of the muscle to produce force) or relief that the exercise task is completed.

In summary you will be asked to: (i) provide pain intensity ratings in your legs only; (ii) give ratings as accurately as possible; and (iii) not under- or overestimate the pain, but simply rate your pain honestly. You should use the verbal expressions to help rate your perceptions.”

No pain at all	Very faint pain (just Notice-able)	Weak pain	Mild pain	Moder-ate pain	Some-what strong pain	Strong pain	--	Very strong pain	--	--	Extremely intense pain (almost unbear-able)
0	0.5	1	2	3	4	5	6	7	8	9	10

Appendix H – RPE (Category-Ratio-10, Borg, 1998)

Instructions for participant: Rate your perception of exertion, that is, how heavy and strenuous the exercise feels to you. The perception of exertion depends mainly on the strain and fatigue A) over your entire body, B) in all of your lower body muscles and on your feeling of breathlessness or aches. 0 is like resting comfortably. Rate this set for exertion over the entire set of repetitions.

Rating	Descriptor
[0]	[Rest]
1	Very, Very Easy
2	Easy
3	Moderate
4	Somewhat Hard
5	Hard
6	-
7	Very Hard
8	-
9	-
10	Maximal

Appendix I – Feeling Scale (F.S.), Felt Arousal Scale

F.S.

Instructions: While participating in exercise it is quite common to experience changes in mood. Some individuals find exercise pleasurable, whereas others find it displeasurable. Additionally, feeling may fluctuate across time. That is, one might feel good and bad a number of times during exercise. Scientists have developed a scale to measure such responses.

+5	**+4**	**+3**	**+2**	**+1**	**0**	**-1**	**-2**	**-3**	**-4**	**-5**
Very GOOD		Good		Fairly Good	Neutral	Fairly Bad		Bad		Very BAD

F.A.S.

Instructions: Please note feelings during the period of time indicated by the experimenter (the task performance period), in terms of the following rating scale. Do this by circling a point corresponding to a number. By "arousal" here is meant how "worked up" you feel. You might experience high arousal in one of a variety of ways, for example, as excitement or anxiety or anger. Low arousal might also be experienced by you in one of a number of different ways, for example as relaxation or boredom or calmness.

1	**2**	**3**	**4**	**5**	**6**
Low Arousal					High Arousal

Appendix J – CES-Depression Inventory

Instructions: For each statement, please circle the number in the column that best describes how you have been feeling in the *past week.*

		Rarely or none of the time (less than 1 day)	Some or a little of the time (1-2days)	Occasionally or a moderate amount of the time (3-4 days)	Most or all of the time (5-7 days)
1	I was bothered by things that usually don't bother me.	0	1	2	3
2	I did not feel like eating; my appetite was poor.	0	1	2	3
3	I felt that I could not shake off the blues, even with the help from family or friends.	0	1	2	3
4	I felt that I was just as good as other people.	0	1	2	3
5	I had trouble keeping my mind on what I was doing.	0	1	2	3
6	I felt depressed.	0	1	2	3
7	I felt that everything I	0	1	2	3

	did was an effort.				
8	I felt hopeful about the future.	0	1	2	3
9	I thought my life had been a failure.	0	1	2	3
10	I felt fearful.	0	1	2	3
11	My sleep was restless.	0	1	2	3
12	I was happy.	0	1	2	3
13	I talked less than usual.	0	1	2	3
14	I felt lonely.	0	1	2	3
15	People were unfriendly.	0	1	2	3
16	I enjoyed life.	0	1	2	3
17	I had crying spells.	0	1	2	3
18	I felt sad.	0	1	2	3
19	I felt that people dislike me.	0	1	2	3
20	I could not get “going”.	0	1	2	3

Appendix K – PANAS: Positive and Negative Affect Schedule (Watson, Clark, & Tellegen, 1988)

Directions: This scale consists of a number of words that describe different feelings and emotions. Read each item and then mark the appropriate answer in the space next to that word. Indicate to what extent you feel ***right now***, that is, at the present moment. Use the following scale to record your answers.

1	2	3	4	5
Very slightly	a little	moderately	quite a bit	extremely or not at all

1. interested	____	11. irritable	____
2. distressed	____	12. alert	____
3. excited	____	13. ashamed	____
4. upset	____	14. inspired	____
5. strong	____	15. nervous	____
6. guilty	____	16. determined	____
7. scared	____	17. attentive	____
8. hostile	____	18. jittery	____
9. enthusiastic	____	19. active	____
10. proud	____	20. afraid	____

Appendix L – USQ: Undergraduate Stress Questionnaire (Crandall, Preisler, & Aussprung, 1992)

Has this stressful event happened to you at any time during the last MONTH? If so, check the box in the right column.

Item Number	Event	Occurrence
1.	Death (family member or friend)	☐
2.	Had a lot of tests	☐
3.	It's finals week	☐
4.	Applying to graduate school	☐
5.	Victim of a crime	☐
6.	Assignments in many or all classes due the same day	☐
7.	Breaking up with boy-/girlfriend	☐
8.	Found out boy-/girlfriend cheated on you	☐
9.	Lots of deadlines to meet	☐
10.	Property stolen	☐
11.	You have a hard upcoming week	☐
12.	Went into a test unprepared	☐
13.	Lost something (especially wallet)	☐
14.	Death of a pet	☐
15.	Did worse than expected on test	☐
16.	Had an interview	☐
17.	Had projects, research papers due	☐
18.	Did badly on a test	☐
19.	Parents getting a divorce	☐
20.	Dependent on other people	☐
21.	Having roommate conflicts	☐
22.	Car/bike broke down, flat tire, etc.	☐
23.	Got a traffic ticket	☐
24.	Missed your period and waiting	☐
25.	Coping with addictions	☐
26.	Thoughts about future	☐
27.	Lack of money	☐
28.	Dealt with incompetence at the Registrar's, Bursar's, Financial Aid, or Advising office	☐
29.	Thought about unfinished work	☐
30.	No sleep	☐
31.	Sick, injury	☐
32.	Had a class presentation	☐
33.	Applying for a job	☐

34.	Fought with boy-/girlfriend	☐
35.	Working while in school	☐
36.	Arguments, conflicts of values with friends	☐
37.	Bothered by having no social support of family	☐
38.	Performed poorly at a task	☐
39.	Can't finish everything you needed to do	☐
40.	Heard bad news	☐
41.	Had confrontation with an authority figure	☐
42.	Maintaining a long-distance boy-/girlfriend	☐
43.	Crammed for a test	☐
44.	Felt unorganized	☐
45.	Trying to decide on major	☐
46.	Feel isolated	☐
47.	Parents controlling with money	☐
48.	Couldn't find a parking space	☐
49.	Noise disturbed you while you were trying to study	☐
50.	Someone borrowed something without permission	☐
51.	Had to ask for money	☐
52.	Problems with a printer	☐
53.	Erratic schedule	☐
54.	Can't understand your professor	☐
55.	Trying to get into your major college	☐
56.	Registration for a class	☐
57.	Stayed up late writing a paper	☐
58.	Someone you expected to call did not	☐
59.	Someone broke a promise	☐
60.	Can't concentrate	☐
61.	Someone did a "pet peeve" of yours	☐
62.	Living with boy-/girlfriend	☐
63.	Felt need for transportation	☐
64.	Bad haircut	☐
65.	Job requirements changed	☐
66.	No time to eat	☐
67.	Felt some peer pressure	☐
68.	You have a hangover	☐
69.	Problems with your computer	☐
70.	Problem getting home from bar when drunk	☐
71.	Used a fake ID	☐
72.	No sex in a while	☐
73.	Someone cut ahead of you	☐
74.	Bank account overdrawn or credit card	☐

	maxed out	
75.	Visit from relative and entertaining them	☐
76.	Decision to have sex on your mind	☐
77.	Talked with a professor	☐
78.	Change of environment (new doctor, dentist, etc.)	☐
79.	Exposed to upsetting TV show, book, or movie	☐
80.	Got to class late	☐
81.	Holiday	☐
82.	Sat through a boring class	☐
83.	Favorite sporting team lost	☐

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Vita

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