NEUROMUSCULAR AND PERCEPTUAL ASPECTS OF
ECCENTRIC MUSCLE DAMAGE AND RECOVERY

by

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This thesis has been read by each member of the following supervisory committee and by majority vote has been found to be satisfactory.
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ABSTRACT

Isometric and isokinetic strength loss following damaging eccentric exercise ($E_{\text{dam}}$) has been widely studied but changes in functional (neuromuscular power) and perceptual (perceived effort) responses have received less attention. The purpose of this investigation was to quantify neuromuscular function during $E_{\text{dam}}$ and evaluate recovery of maximal power ($P_{\text{max}}$) and rating of perceived exertion (RPE) to a challenging submaximal task. Eleven trained cyclists (age: 30 ± 8 yrs; mass: 74 ± 8 kg, height: 176 ± 9 cm) performed eccentric cycling with their right leg ($E_{\text{dam}}$) and concentric cycling with their left leg (control) until total work was matched (-69 ± 5 kJ vs. 70 ± 5 kJ). Single-leg $P_{\text{max}}$ was assessed with inertial-load cycling on three different crank lengths (120, 170, and 220 mm). Single-leg RPE in response to a 90 s standardized submaximal cycling task (2 kp, 80 rpm) was assessed with a Borg scale. Biomechanical joint powers were calculated during maximal isokinetic cycling (120 rpm) using a force measuring pedal and instrumental spatial linkage system. All measures were assessed at six time points (baseline, 0, 24, 48, 72, 96 hr). One-way ANOVA indicated that the knee joint absorbed the majority of eccentric work during $E_{\text{dam}}$. Repeated measures ANOVA indicated that $P_{\text{max}}$ decreased (0-72 hr) on all three crank lengths and RPE increased (0-72 hr) in the damaged leg. Joint powers produced by the ankle, knee, and hip were not different following $E_{\text{dam}}$. This study is the first to quantify and control eccentric work. $P_{\text{max}}$ findings support previous reports but fail to identify the intrinsic mechanism that limits
maximal power recovery. The wide variation in joint powers may reflect differences in
the location of muscle damage and/or recovery strategies. Alterations in RPE appear to be
quite different compared to traditional soreness measures. From the results of this
investigation I conclude that $P_{\text{max}}$ and RPE are more functional recovery measures
compared to traditional strength and soreness measures.
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1. INTRODUCTION

Unaccustomed exercise can cause muscle damage that leads to muscle weakness and soreness. Muscles are particularly prone to becoming weak and sore after exercise in which they are actively lengthened (eccentric muscle actions). For example, running and strength training elicit significant eccentric actions and, consequently, an individual who begins a running or strength training program will likely experience muscle weakness and soreness in the days following the initial exercise. Occupational activities such as operating power hand tools for extended periods of time (e.g., factory work) can produce muscle damage as well (Sesto, Radwin, Best, & Richard, 2004; Sesto, Radwin, Block, & Best, 2005). In a series of investigations Clarkson and colleagues (Clarkson, Nosaka, & Braun, 1992; Newham, Jones, & Clarkson, 1987; Nosaka & Clarkson, 1992, 1996a, 1996b) have established that strength and soreness alterations associated with muscle damage are immediate, severe, and long lasting. Because of the wide range of recreational and occupational activities that can elicit eccentric muscle damage and the substantial time required for recovery it is imperative to understand the underlying mechanisms and resulting functional limitations associated with muscle damage. Such an understanding is an important first step toward developing effective treatments to minimize muscle damage and speed the recovery process.

Traditionally, investigators have used the reductions in maximal isometric (measured as force and/or joint torque) and isokinetic (measured as joint torque during
constant velocity movements) strength to quantify the magnitude of muscle damage and assess recovery. It is well documented in both human and animal muscle that maximal isometric strength is immediately reduced between 40-60% and remains reduced for several days (e.g., 10-14 days; Clarkson et al., 1992; Ingalls, Warren, Williams, Ward, & Armstrong, 1998; Jones, Allen, Talbot, Morgan, & Proske, 1997; Newham et al., 1987; Nosaka & Clarkson, 1992, 1996a, 1996b; Warren, Lowe, Hayes, Karwoski, Prior, & Armstrong, 1993). The trends for recovery of isokinetic strength are less obvious. In an extensive review, Bryne, Twist, and Eston (2004) emphasized that there were no meaningful differences in rate of isokinetic strength recovery across muscle actions (concentric vs. eccentric vs. isometric or across the velocity of movement (slower vs. faster)).

Although isometric and isokinetic strength measures are widely used to evaluate muscle damage and recovery, these measures may not represent muscle actions that occur during voluntary movement. During most repetitive voluntary movements, such as cycling, muscles must become excited, produce force while shortening, relax, and undergo passive or partially active lengthening before becoming excited again. During these repetitive movements, the time required for muscle excitation and relaxation (excitation/relaxation kinetics) and the velocity at which the muscle shortens (force-velocity kinetics) greatly influence neuromuscular function (Caiozzo & Baldwin, 1997; Martin, 2007; Martin, Brown, Anderson, & Spirduso, 2000). The influences of excitation/relaxation and force-velocity kinetics are greatly reduced during isometric and isokinetic movements. An alternative way to represent voluntary neuromuscular function in humans is through the use of a cycling model (Martin, 2007; Martin et al., 2000a;
Martin, Farrar, Wagner, & Spirduso, 2000; Martin, Lamb, & Brown, 2002; Martin, & Spirduso, 2001; Neptune & Kautz, 2001). Power (power = force x velocity) produced during cycling is influenced by the intrinsic muscle factors of excitation/relaxation and force-velocity kinetics (Martin, 2007), muscle fiber type (Hautier, Linossier, Belli, Lacour, & Arsac, 1996; Martin et al., 2000c; Martin, Wagner, & Coyle, 1997) and biomechanical factors (joint kinematics and kinetics; Broker & Gregor, 1994; Ericson, 1986, 1988a, 1988b; Ericson, Bratt, Nisell, Arborelius, & Ekholm, 1986; Reiser, Peterson, & Broker 2002), thus serving as a more functional alternative to isometric and isokinetic measures.

Surprisingly, the ability of damaged muscle to generate maximal power has received limited attention and to my knowledge, only four groups (Byrne & Eston, 2002b; Malm, Lenkei, & Sjodin, 1999; Sargeant & Dolan, 1987; Twist & Eston, 2005) have directly reported alterations in maximal cycling power. In general, maximal power was immediately reduced by approximately 15-20% and power was restored to baseline within one week (Byrne & Eston, 2002b; Sargeant & Dolan, 1987; Twist & Eston, 2005). Although there is a growing body of literature in maximal power recovery, information about the muscle damage process, underlying mechanisms, and submaximal recovery is not well documented.

In most investigations, much of the emphasis has been placed on understanding the recovery process rather than the damage process. The immediate reductions in maximal strength and power provide useful information regarding short-term recovery but they may not be the most effective method for quantifying the magnitude of muscle damage. Furthermore, investigators rarely describe the specific methods that were used to
control for and quantify the damaging eccentric work. Alternatively, measuring mechanical work (work = power/time) during the actual damaging exercise would provide a better method for quantifying eccentric work and ensuring that work was matched across participants and treatment groups. Information describing how muscles actually become damaged would be an important first step toward understanding the recovery process.

The underlying mechanisms that limit neuromuscular function in damaged muscle have been attributed to intrinsic disruption of actin-myosin cross bridge formation and calcium handling (Barash, Peters, Friden, Lutz, & Lieber, 2002; Lieber, Thornell, & Friden, 1996; Morgan, 1990; Warren, Ingalls, Lowe, & Armstrong, 2001; Warren et al., 1993), selective damage to type II muscle fibers (Friden, Sjostrom, & Ekblom, 1983), and glycogen depletion (Asp, Daugaard, Kristiansen, Kiens, & Richter, 1998; Costill et al., 1990; O’Reilly, Warhol, & Fielding, 1987). These mechanisms, however, have been determined in isolation using several different research models (animal vs. human and isometric vs. isokinetic) and no direct attempt has been made to evaluate these potential mechanisms while producing maximal power. As described earlier, power produced during cycling is influenced by intrinsic muscle factors, muscle fiber type, and biomechanical factors and thus may provide an investigative paradigm for determining the relative contributions of each potential mechanism. Identifying these contributions during cycling exercises that are more representative of voluntary movements would offer a unique way to determine if these potential mechanisms substantially impact voluntary neuromuscular function.
Although maximal power is an important functional aspect of recovery it is important to consider that most voluntary tasks are submaximal (e.g., sit to stance, walking, and stair climbing). Traditionally, investigators have used measures of muscle pain and delayed onset muscle soreness to assess perceptual responses associated with muscle damage. These assessments, however, are most often evaluated at rest, during single joint movements (e.g., flexing/extending a joint), and over short durations (e.g., 1-3 s). In contrast, a dynamic movement such as walking is not performed at rest, requires integration of several joint movements, may last for long durations, and can be challenging at times (e.g., climbing stairs). An alternative way to evaluate perceptual recovery would be to measure rating of perceived exertion (RPE; Borg, 1970). Rating of perceived exertion has been widely used during maximal and submaximal exercise to assess perceptual responses such as “how hard does the task feel” (Borg, Ljunggren, & Ceci, 1985; Gearhart, Becque, Palm, & Hutchins, 2005; Mengelkoch & Clark, 2006; Noble, Borg, Jacobs, Ceci, & Kaiser, 1983). To my knowledge, rating of perceived exertion has not been assessed following eccentric muscle damage and perceived exertion may exhibit a different recovery profile when compared to traditional soreness measures. For example, would muscle soreness associated with climbing one step be the same as the perceived exertion associated with climbing several flights of stairs? Establishing the time course of alterations in perceived exertion during challenging submaximal exercise of moderate duration (e.g., 90 s) would offer novel information regarding submaximal and perceptual recovery and compliment previous muscle pain and soreness findings.

In this investigation, my goals were to control and quantify the eccentric work associated with the damaging eccentric exercise, identify the mechanisms that limit
maximal power recovery, and incorporate measures of rating of perceived exertion to evaluate submaximal and perceptual recovery. Such information should provide a more complete understanding of muscle damage and recovery.

Purpose Statement

The purpose of this investigation was to quantify neuromuscular function during damaging eccentric exercise and evaluate recovery of maximal neuromuscular function and perceptual responses to a challenging submaximal task.

Research Questions

A total of six research questions were investigated in this study:

1. How do the ankle, knee, and hip joints absorb eccentric work during an acute bout of eccentric cycling exercise?
2. What is the time course of alterations in overall maximal power following an acute bout of eccentric cycling exercise?
3. To what extent does an acute bout of eccentric cycling exercise alter excitation/relaxation kinetics compared with alterations to force-velocity kinetics?
4. What are the time courses of alterations in biomechanical joint powers produced at the ankle, knee, and hip following an acute bout of eccentric cycling exercise?
5. Do the time courses of alterations in biomechanical joint powers at the ankle, knee, and hip parallel the time course of alterations for overall maximal power?
6. What is the time course of alterations in rating of perceived exertion to a challenging submaximal task following an acute bout of eccentric cycling exercise?
Research Hypotheses

The following hypotheses were tested in this investigation:

RQ #1: Biomechanical aspects of eccentric muscle damage

I hypothesized that the knee joint would absorb the majority of the eccentric work during an acute bout of eccentric cycling exercise. This was based on pilot data and that the knee joint produces substantial power during normal recumbent cycling (Reiser et al., 2002).

RQ #2: Time courses of alterations in overall maximal power

From pilot data and previous reports (Byrne & Eston, 2002b; Sargeant & Dolan, 1987; Twist & Eston, 2005) I hypothesized that overall maximal power in the damaged leg would be immediately reduced (15-25%) and power would be restored within 1 week.

RQ #3: Alterations in excitation/relaxation and force-velocity kinetics

This information was completely novel; thus there was no directional hypothesis.

RQ #4: Biomechanical aspects of power recovery

This information was completely novel; thus there was no directional hypothesis.

RQ #5: Overall power and joint power comparisons

This information was completely novel; thus there was no directional hypothesis.

RQ #6: Time course of alterations in rating of perceived exertion
Based on pilot data I expected that rating of perceived exertion in the damaged leg would increase during the initial days following damage (24-48 hr) and slowly recover over time.

**Significance**

The damage-recovery model developed in this study will be used in future investigations in which Neuromuscular Function Laboratory researchers will evaluate the extent to which protein-carbohydrate supplementation influences recovery. Quantifying and controlling eccentric work will ensure that eccentric work was matched across participants and groups and also help to develop working hypotheses concerning recovery. Information regarding the time course of alterations in overall maximal power and assessment of selective damage to type II muscle fibers will be important contributions to the limited work in these areas. Separating the contributions of excitation/relaxation and force-velocity kinetics will provide information about the intrinsic properties of damaged muscle and improve the understanding as to which mechanism limits power recovery. Biomechanical aspects of muscle damage and maximal power recovery may give a more complete picture of the functional limitations and time needed for recovery. Rating of perceived exertion in response to a challenging submaximal task will compliment other perceptual responses associated with recovery. Finally, because the exercises and measures used in this study are representative of voluntary movements, the results may be translated into practical recommendations for individuals who experience recreational and/or occupational related muscle damage.
Delimitations

The following delimitations were made for this investigation:

1. Participants in this study were trained cyclists currently living in Salt Lake City, UT.
2. Participants were between 19-44 years of age.
3. Participants were those willing to participate and who volunteered for the entire two week study (convenience sample).

Limitations

The following limitations were present for this investigation:

1. Participants may have felt inclined to answer in a socially desirable way to questions of a personal nature.
2. Results from this study were only generalizable to trained cyclists.
3. Maximal power tests were based on voluntary individual effort. Participants may not have put forth their best efforts during these tests. Week 1 “Practice Week” was designed so that participants had practice at putting forth a maximal effort.
4. There was no way to totally “blind” participants and testers to the eccentric or traditional concentric intervention group.
5. Travel time and distance to the facility may have limited those who would like to participate.

Assumptions

The following assumptions were made for this investigation:

1. Participants filled out the background questionnaires out to the best of their abilities and knowledge.
2. All participants experienced intense muscle damage.

3. All participants performed at maximum capabilities on power tests and were not influenced by the primary investigator.
2. REVIEW OF LITERATURE

In the initial portion of this chapter, I emphasize the symptoms of muscle damage that will be discussed and review the traditional strength measures used to evaluate muscle damage and recovery. Subsequently, I explain why these measures may not be representative of muscle actions that occur during voluntary movements. Next, I introduce maximal power as a preferable alternative for evaluating the functional limitations associated with muscle damage. I also identify the basic mechanisms that are responsible for strength loss. Finally, I describe how the damage-recovery model developed in this investigation was used to evaluate the potential mechanisms and factors that may influence the recovery time required to restore maximal power.

Symptoms of Muscle Damage

In this investigation the symptoms of weakness (reductions in isometric and isokinetic strength and power) and muscle soreness associated with muscle damage were the primary symptoms of interest. Other symptoms of muscle damage include, but are not limited to, elevated muscle enzymes (Brown, Child, Day, & Donnelly, 1997; Clarkson, Byrnes, McCormick, Turcotte, & White, 1986; Nosaka & Clarkson, 1992, 1996a, 1996b), stiffness and swelling (Nosaka & Clarkson, 1996b), and disruption of intracellular and extracellular structures (Friden et al., 1983; Friden, Kjorell, & Thornell, 1984; Lieber & Friden, 2002; Lieber et al., 1996).
Isometric Strength and Recovery

Investigators have used the reductions in maximal isometric muscle force or joint torque to quantify the magnitude of muscle damage and assess recovery. Clarkson and colleagues (Clarkson et al., 1992; Newham et al., 1987; Nosaka & Clarkson, 1992, 1996a, 1996b) have used an arm damage-recovery model to establish that isometric force in elbow flexor muscles is immediately reduced by 50-60% and force is slow to recover as deficits are still present at 10 days. Similarly, investigators who have used in vitro models have measured immediate isometric force deficits between 43-50% in damaged mammalian and amphibian muscle (Ingalls et al., 1998; Jones et al., 1997; Warren et al., 1993). Further, in mammalian muscle (mouse soleus) force was slow to recover as deficits still persisted at 14 days (Ingalls et al., 1998). Interestingly, alterations in isometric strength of isolated muscle closely parallel those alterations observed in human elbow flexor muscles. Thus, in human and animal muscles, reductions in isometric strength are immediate, severe, and long lasting.

Isokinetic Strength and Recovery

In addition to isometric strength measurements, maximal joint torque has been measured during single-joint movements performed at constant velocities (isokinetic). Isokinetic strength measures in damaged muscle are used to determine if reductions in joint torque are dependent upon the type of muscle action (concentric, isometric, eccentric) and/or the angular velocity of movement. Friden et al. (1983) reported no differences in recovery between isometric torque and concentric torque at slower velocities (1.57 and 3.14 rad/s) as isometric and concentric torques were reduced 7, 8, and 9% respectively 6 days after exercise. Concentric torque at higher velocities (5.24
rad/s) was slower to recover as torque was still reduced by 24% at 6 days. Some investigators (Eston, Finney, Baker, & Baltzopoulos, 1996; Golden & Dudley, 1992) have also reported slower recovery of joint torque at higher angular velocities whereas others (Deschenes et al., 2000; Gibala, MacDougall, Tarnopolsky, Stauber, & Elorriaga, 1995; Michaut, Pousson, & Babault, 2002) have reported slower recovery at lower angular velocities. The contrasting results are most likely dependent upon the specific damage-recovery model used (e.g., arm vs. leg), the amount and type of muscle mass damaged (e.g., elbow flexors vs. knee extensors), and dose-response relationships (Jamurtas et al., 2005). The strength and recovery inconsistencies may also be due to inter-participant variability as observed in biochemical markers of muscle damage such as creatine kinase (Nosaka & Clarkson, 1996b).

**Voluntary Movements**

During repetitive voluntary movements, the time required for muscle excitation and relaxation (excitation/relaxation kinetics) and the velocity at which the muscle shortens (force-velocity kinetics) influence neuromuscular function (Caiozzo & Baldwin, 1997; Martin, 2007; Martin et al., 2000a). The influences of excitation/relaxation and force-velocity kinetics are greatly reduced during isometric muscle actions. Similarly, the influence of excitation kinetics is also minimized during maximally activated isokinetic movements as sufficient time is provided to fully excite the muscle before it is allowed to shorten (e.g., preloaded). Furthermore, single-joint isokinetic movements are performed at constant velocities but repetitive voluntary movements require variations in muscle length and velocity trajectories (Neptune & Kautz, 2001).
Power and Recovery

Power (power = force x velocity) produced during cycling serves as a more functional measure because it is influenced by both excitation/relaxation and force-velocity kinetics (Martin, 2007), muscle fiber type (Hautier et al., 1996; Martin et al., 2000c) and biomechanical factors (joint kinematics and kinetics; Broker & Gregor, 1994; Ericson, 1986, 1988a, 1988b; Ericson et al., 1986; Reiser et al., 2002).

Three groups have used cycle ergometry ranging between 6-20 s to establish that maximal power was immediately reduced by approximately 15-20% (Byrne & Eston, 2002b; Sargeant & Dolan, 1987; Twist & Eston, 2005). Furthermore, Sargeant and Dolan (1987) and Byrne and Eston (2002b) observed that power was fully recovered within 5-7 days. In contrast, Malm and colleagues (1999) reported no changes in pre and post average power during a Wingate Anaerobic Test. The damage protocol employed may not have been sufficient to induce muscle damage (Byrne et al., 2004) and average power during a 30 s trial is also influenced by fatigue and/or anaerobic capacity. The only report of power recovery in isolated muscle was by Stevens (1996). The author focused on stimulation parameters associated with eccentric work and as a result the reductions in work (e.g., work = power x time) varied between 2-12%. Several groups have also indirectly evaluated power recovery in damaged muscles by measuring vertical jump (Byrne & Eston 2002a; Horita, Komi, Nicol, & Kyröläinen, 1999) and sprinting performance (Semark, Noakes, St. Clair Gibson, & Lambert, 1999; Twist & Eston, 2005).
Intrinsic disruptions

The underlying mechanism that limits neuromuscular function in damaged muscle remains an issue of ongoing debate. Morgan (1990) has proposed that weaker sarcomeres are overstretched during repetitive eccentric muscle actions and subsequently lie scattered along the length of the myofibril. This failure to reintegrate leads to fewer actin-myosin cross bridge formations and reductions in maximal isometric force ("Popping Sarcomere Theory"). Alternatively, Barash et al. (2002) and Lieber and colleagues (1996) have proposed that degradations of the cytoskeleton protein desmin are responsible for reductions in maximal isometric torque. Desmin is known to play a key role in lateral transmission of forces (Price & Sanger, 1983) and desmin disappearance occurs within minutes, thus it is the earliest documented structural change observed in muscle after damaging exercise (Lieber et al., 1996).

A major competing proposal is that the mechanism is not mechanical in nature; rather it is due to some impairment in the excitation-contraction coupling process (Warren et al., 2001; Warren et al., 1993). Warren and colleagues (1993, 2001) demonstrated that the majority of maximal isometric force loss in isolated muscle can be recovered with caffeine administration, which suggests some disturbance to the kinetics of calcium (Ca$^{2+}$) handling. These primary theories of muscle damage were developed based on measures of maximal isometric strength. Consequently, the relative contributions of calcium handling and actin-myosin cross bridge formation have been difficult to separate during repetitive voluntary movements. As described earlier, maximal power is influenced by both excitation/relaxation and force-velocity kinetics.
Excitation/relaxation kinetics account for calcium handling whereas force-velocity kinetics account for actin-myosin cross bridge formation and thus may provide an investigative paradigm for determining the relative contributions of each potential mechanism.

Selective muscle fiber type damage

Friden and colleagues (1983) have reported that an additional mechanism for the decline in force production involves selective damage to type II muscle fibers. More specifically, fast twitch glycolytic fibers are recruited initially because of the high eccentric forces and become fatigued. These fibers are unable to regenerate adenosine triphosphate (ATP) and go into a state of rigor resulting in mechanical disruption. Subsequently, these disrupted fibers produce less than optimal force, which is most noticeable during maximal voluntary movements.

Impaired glycogen metabolism

It is well documented that muscle glycogen resynthesis is impaired following damaging eccentric exercise (Asp et al., 1998; Costill et al., 1990; O’Reilly et al., 1987). Specifically, decreases in muscle glucose transporter GLUT-4 proteins are responsible for the delay in muscle glycogen resynthesis after eccentric exercise.

Damage-Recovery Model

The mechanisms described above are well accepted and investigators who have measured maximal power have speculated that these mechanisms contributed to the reduction in maximal power. Producing power is influenced by intrinsic muscle factors, muscle fiber type, glycogen content, and biomechanical factors. A damage-recovery
model that considers each of these influential factors would be more directly applicable to voluntary movements. Below I describe how to address each of these factors through the use of a human cycling.

**Intrinsic mechanisms**

In a series of studies, Martin and co-workers (Martin, 2007; Martin et al., 2000a; Martin & Spirduso, 2001) were able to separate the relative contributions of excitation/relaxation and force-velocity kinetics during cycling by altering the ergometer crank length during inertial-load cycling. Crank length alterations differentially affect pedaling rate and pedal speed. Pedaling rate, a measure of cycle frequency (e.g., rpm, Hz), serves as a surrogate measure of muscle excitation/relaxation kinetics and pedal speed (tangential velocity of pedal) serves as a surrogate measure of average muscle shortening velocity (force-velocity kinetics; Yoshihuku & Herzog, 1990). Alterations in crank length (e.g., 5-10 cm) make it possible to separate the effects of excitation/relaxation and force-velocity kinetics. In the context of muscle damage and recovery, if power produced on long cranks (emphasize pedal speed) was reduced to greater extent compared to shorter cranks, this would suggest that force-velocity kinetics and actin/myosin cross bridge formation limit recovery. Alternatively, if power produced on shorter cranks (emphasize pedaling rate) was reduced to greater extent compared to longer cranks, this would indicate that excitation/relaxation kinetics and calcium handling limit power recovery. Sargeant and Dolan (1987) measured power recovery at pedaling rates of 80 revolutions per minute (rpm) and 110 rpm and reported slower recovery at the higher pedaling rate. Increasing pedaling rate influences both excitation/relaxation and
force-velocity kinetics as they are linearly coupled, which makes it difficult to separate their relative contributions.

Muscle fiber type

Hautier and colleagues (1996) and later Martin and colleagues (2000c) reported that the optimal pedaling rate that elicits maximal power during inertial-load cycling serves as a surrogate measure for muscle fiber type distribution. On average humans produce maximal power near 120 rpm. A marathon runner with a majority of slower acting musculature in the lower body (thigh and shank) would produce maximal power at a lower pedaling rate (e.g., 110 rpm) whereas a 100 meter sprinter with faster acting musculature would produce power at a higher pedaling rate (e.g., 130 rpm). Based on Friden’s theory (Friden et al., 1983) of greater damage to type II muscle fibers following eccentric exercise, I hypothesized that following damaging eccentric exercise individuals would produce maximal power at a lower optimal pedaling rate compared to the undamaged state. Using inertial-load cycling it is possible to test Friden’s theory (1983) and this hypothesis.

Muscle glycogen content

The duration of the maximal inertial-load cycling trials (e.g., ~3 s) would emphasize metabolism of immediate phosphocreatine stores rather than glycogen metabolism. Additionally, the shorter duration of the inertial-load trials would reduce the possibility of fatigue as a confounding factor. Morgan, Gregory, and Proske (2004) have emphasized that it is often difficult to separate muscle damage from fatigue. Thus, inertial-load cycling would be advantageous for ruling out the possible confounding factors of glycogen depletion and fatigue.
Biomechanical factors

Previous investigators (Byrne & Eston, 2002b; Malm et al., 1999; Sargeant & Dolan, 2001; Twist & Eston, 2005) who measured power specifically measured the overall power produced and delivered to the ergometer crank. Cycling is a complex task and the power delivered to the crank is produced by muscles that span the ankle, knee, and hip (Broker & Gregor, 1994; Ericson, 1986, 1988a, 1988b; Ericson, et al., 1986; Reiser et al., 2002). The relative contributions of power produced by each joint with respect to overall power produced at the crank (biomechanical aspects) have been reported in healthy individuals during submaximal cycling only (Broker & Gregor, 1994; Ericson, 1986, 1988a, 1988b; Ericson et al., 1988; Reiser et al., 2002). Biomechanical aspects of power measured during the damaging exercise would indicate the magnitude of damage in each joint and serves as means to quantify the eccentric work. Rarely, have previous investigators reported how eccentric work was absorbed during the damaging exercise, controlled, and/or quantified, which may contribute to the variability in muscle damage and recovery. Subsequent biomechanical aspects of power measured during the recovery phase would establish the time course of alterations in power for each joint. These biomechanical analyses can be performed with the use of an instrumented force measuring pedal and motion capture system.

In the current investigation I have attempted to extend upon several previous investigations. The damage-recovery model developed in this investigation has been based primarily on those findings reported in healthy individuals producing maximal power. The mechanisms responsible for these findings may function differently during muscle damage and recovery which could result in very different final outcomes.
Additionally, I should emphasize that it would be difficult to directly implicate a specific intracellular mechanism based on a model that does not directly investigate these processes; rather this investigation depends on the validity of a model. This investigation offers an alternative approach to studying muscle damage and recovery and could lead to a greater understanding of the subject.
3. METHODS

In this chapter I explain the methodology used in this investigation. Specifically, I describe the participants who volunteered for the study, experimental procedures under which the investigation was conducted, instrumentation, and research design and analyses.

Participants

All exercise protocols and procedures were reviewed by the University of Utah Internal Review Board. The target population for this investigation was healthy individuals who engage in some form of acute eccentric exercise and experience muscle damage. A sample of trained cyclists was used for this investigation. A trained cyclist was defined as an individual who cycled on average of at least 5-hr per week. Trained cyclists are typically highly skilled at the cycling tasks used in this investigation and should produce reliable data thereby increasing the statistical power for any specific number of participants. Additionally, most trained cyclists engage in very little chronic eccentric training (e.g., weightlifting and running) and are not protected against eccentric damage.

All potential participants completed a background questionnaire to ensure that they had not engaged in any chronic eccentric training within the last 4 months. Potential participants who had previous chronic eccentric training in the past 4 months were not allowed to participate in the study. Previous eccentric training would have made it
difficult to ensure that the participants did in fact experience muscle damage. Verbal and
written explanations of the study were provided to each of the participants so that they
were fully aware of the time commitment and level of exercise intensity that this
investigation required. An informed consent form was completed, dated, and signed by
each participant.

A convenience sampling procedure was used to recruit trained cyclists who met
the inclusion criteria described. Trained cyclists between 19-44 years of age were
recruited through local cycling forums, flyers, websites (e.g., Craig's List), and by word of
mouth in the Salt Lake City area. Sample size for this study was estimated from the
repeated measures tables provided by Green (1990) and Stevens (2002). Twenty
sampling units \( n = 10 \)/treatment group were required assuming a large effect size and
power of \(.8\) (\(\alpha = .05\)). In this study participants performed eccentric exercise with
their right leg (treatment group) and concentric exercise their left leg (control group), thus
each participant served as his/her own control. Therefore a total of 10 participants would
be required to obtain the appropriate effect size and power \( n = 10 \)/treatment group; \( N = 20 \). Effect size and average correlation parameters required for sample size estimation
were calculated from pilot data \( n = 8 \)/group; \( N = 16 \) rather than data from previous
investigators (Byrne & Eston, 2002b; Sargeant & Dolan, 1987; Twist & Eston, 2005)
because the necessary information to estimate these parameters was not reported.

Previous investigators (Byrne & Eston, 2002b; Sargeant & Dolan, 1987; Twist & Eston,
2005) used sample sizes of 7, 4, and 10 and found significant alterations in power over
time. Based on the sample size estimation and previous literature a total of 11 participants
(n = 11/treatment group; N = 22) were recruited, which allowed for one participant dropout.

**Investigation Timeline**

This investigation took place over 2 consecutive weeks. Participants were required to report to the Neuromuscular Function Laboratory on eight occasions over the 2 weeks, which consumed approximately 12 hr of their time. Figure 3.1 provides an overview of the time line of the investigation.

**Practice Week Procedures**

**Week 1: Practice week (Monday, Wednesday, and Friday)**

Participants reported to the laboratory on 3 days for approximately 1 hr in order to become familiar with the laboratory and testing procedures. On the first day of the practice week participant height, mass, thigh length, shank length, and foot length were recorded. On each day participants performed a cycling warm-up for 5 min at a self-selected intensity on a cycle ergometer. Participants practiced submaximal single-leg cycling at a self-selected intensity for 5 min with each leg. The cycle ergometer used for submaximal single-leg cycling was fitted with bicycle-racing handlebars, cranks, pedals, and seat. Additionally, the ergometer was equipped with a 9.9 kg counterweight device (Figure 3.2). The counterweight device served to balance the system during single-leg cycling and allowed the participant to pedal with biomechanics similar to those during normal double-leg cycling. Participants also performed a standardized 90 s submaximal cycling trial at a challenging resistance load of approximately 2 kiloponds (kp) at 80 rpm with each leg. The combination of this pedaling rate and resistance load was equivalent to
pedaling at approximately 160 watts. During the final 10 s participants rated their perceived exertion by either pointing or verbally communicating the value that corresponded to their current exertion level. A resistance load that elicited a perceived exertion of 13-14 (e.g., “somewhat hard”) was established during the practice week and subsequently used throughout the duration of the investigation.

Participants performed multiple practice trials of maximal single-leg cycling (inertial-load trials). The cycle ergometer used for maximal single-leg cycling was fixed to the floor and fitted with bicycle-racing handlebars, cranks, pedals, and seat. To allow for smooth and safe maximal single-leg cycling, the ergometer was modified with a 9.9 kg counterweight device, foot stabilization platform, and constraint belt (Figure 3.3). Participants began from rest and sprinted maximally for approximately 3-4 s on verbal
command with standardized encouragement. Three maximal single-leg trials were performed on 170 mm crank lengths with the left and right leg for a total of six trials. With the right leg only, three single-leg cycling trials were performed on 120 mm and 220 mm crank lengths for an additional six trials. A separate cycle ergometer with similar single-leg modifications (as previously described) was used for these trials (Figure 3.4).

Participants performed three maximal single-leg cycling trials on an isokinetic ergometer at 120 rpm. Briefly, the isokinetic ergometer (Figure 3.5) was constructed using a Monark (Vansbro, Sweden) cycle ergometer frame and flywheel. The flywheel was driven by a 3750 watt direct current motor (Baldor Electric Company model CDP3605, Fort Smith, AR, USA) via pulleys and a belt. The motor was controlled by a speed controller equipped with regenerative braking capability (Monark model RG5500U, Glendale, CA, USA). When a participant cycled maximally and applied power to the ergometer, the motor acted as a generator and the generated current was dissipated by a resistor and heat sink built into the speed controller. The controller can,
Figure 3.3. Ergometer setup for maximal single-leg cycling. Smaller image shows details of the foot stabilization device (A) and protective barrier (B).

Figure 3.4. Ergometer setup for maximal single-leg cycling with different crank lengths. Smaller image shows details of chain tensioner used to allow for gear ratio changes (A).
therefore, maintain a specified pedaling rate while resisting power outputs of up to 3750 watts. For the single-leg trials participants sprinted maximally for approximately 3-4 s on verbal command with standardized encouragement.

The practice testing described was used so that participants would have sufficient time to become familiar with submaximal and maximal single-leg cycling. To summarize, the order of events for the practice week included:

**Week 1: Practice Week (Monday, Wednesday, Friday)**
1. 5 min warm-up of normal double-leg cycling at a self-selected intensity
2. 5 min submaximal single-leg cycling with each leg
3. 90 s standardized submaximal single-leg trial at 160 watts with RPE for each leg
4. 6 maximal single-leg trials on 170 mm cranks (3 trials/leg)
5. 3 maximal single-leg trials on 120 mm cranks (right leg only)
6. 3 maximal single-leg trials on 220 mm cranks (right leg only)
7. 3 maximal isokinetic single-leg trials at 120 rpm on 170 mm cranks (right leg only)
Experimental Week Procedures

Phase 1: Baseline

On the first day of the experimental week participants reported to the laboratory at their scheduled time and this visit lasted approximately 2 hr. Participants performed a cycling warm-up for 5 min at a self-selected intensity. Participants performed two maximal single-leg cycling trials with the right leg followed by two maximal single-leg cycling trials with their left leg (~3 s) with the standard 170 mm cranks. Right leg trials were performed first so that this leg had time to adequately recover for additional trials on different crank lengths. With the right leg only participants performed two maximal single-leg cycling trials on 120 mm cranks and on 220 mm cranks (~3 s). The order of crank length (120 or 220 mm) was counterbalanced. For each crank length, a short warm-up (~1-2 min) was included so that the participants could adjust to the crank length. Next participants performed one standardized 90 s submaximal cycling trial with each leg in a counterbalanced fashion and rated their perceived exertion during the final 10 s. Lastly, participants performed two maximal single-leg cycling trials on an isokinetic ergometer at a constant pedaling rate of 120 rpm (~3 s). To summarize, the order of events for the Phase 1: Baseline included:

Week 2: Experimental Week - Phase 1: Baseline
1. 5 min warm-up of normal double-leg cycling at a self-selected intensity
2. 4 maximal single-leg trials on 170 mm cranks (2 trials/leg)
3. 2 maximal single-leg trials on 120 mm cranks (right leg only)
4. 2 maximal single-leg trials on 220 mm cranks (right leg only)
5. 90 s standardized submaximal single-leg trial at 160 watts with RPE for each leg
6. 2 maximal isokinetic single-leg trials at 120 rpm on 170 mm cranks (right leg only)
Phase 2: Eccentric muscle damage

On the first day of the experimental week following the completion of Phase 1, single-leg eccentric cycling was used to elicit muscle damage followed by control exercise of normal single-leg cycling. For eccentric cycling participants were positioned in a recumbent seat and resisted the reverse moving pedals of an isokinetic ergometer at a constant pedaling rate of 60 rpm (Figure 3.6). A 9.9 kg counterweight was offset 90 degrees to balance the system and allow for smooth eccentric cycling (Figure 3.6a). A Shoberer Rad Masstechnik power meter (SRM) provided digital feedback to the participant regarding power absorption and allowed them to maintain a specified eccentric power during the trial (Figure 3.6b). Through pilot testing I determined that 40% of maximal single-leg (right) power (40% MSLP) for 5 min was sufficient to elicit substantial muscle damage. All participants attempted to resist 40% MSLP with their right leg until the total amount of prescribed eccentric work (kJ) was absorbed. Because eccentric cycling was a completely novel task, there was a wide variation in the time it took participants to absorb the prescribed amount of work (~5-10 min). The SRM power meter data were downloaded and stored on a computer and used to confirm total eccentric work and average eccentric power.

Following eccentric cycling, participants cycled with their left leg on a Velotron ergometer (Elite, Seattle, WA, USA) at an all out time trial effort (e.g., as hard as they could go) at a self-selected challenging resistance load until total concentric work matched total eccentric work (~7-10 min). The Velotron ergometer was equipped with a counterweight device and concentric work was calculated by dividing the average power
reported by the Velotron Software by the total time. To summarize, the order or events for the Phase 2: Eccentric Muscle Damage included:

**Week 2: Experimental Week-Phase 2: Eccentric Muscle Damage**
1. Single-leg (right) eccentric cycling at 60 rpm at 40% MSLP for approximately 5 min
2. Single-leg (left) control cycling at time trial pace until concentric work matched eccentric work

**Phase 3: Immediate recovery**

On the first day of the experimental week immediately following the completion of Phases 1 and 2, participants repeated the exact sequence of events they performed in Phase 1. To summarize, the order or events for the Phase 3: Immediate Recovery included:

**Week 2: Experimental Week-Phase 3: Immediate Recovery**
1. 5 min warm-up of normal double-leg cycling at a self-selected intensity
2. 4 maximal single-leg trials on 170 mm cranks (2 trials/leg)
3. 2 maximal single-leg trials on 120 mm cranks (right leg only)
4. 2 maximal single-leg trials on 220 mm cranks (right leg only)
5. 90 s standardized submaximal single-leg trial at 160 watts with RPE for each leg
6. 2 maximal isokinetic single-leg trials at 120 rpm on 170 mm cranks (right leg only)

**Phase 4: Long-term recovery**

The exact sequence of events that took place during Phase 1 was again repeated at 24, 48, 72, and 96 hr. These time points were selected based on the time course of alterations for overall power and rating of perceived exertion established from pilot data and previous literature (Byrne & Eston, 2002b; Sargeant & Dolan, 1987; Twist & Eston, 2005). All testing occurred at approximately the same time each day. During the experimental week participants were informed and reminded to refrain from additional exercise. The only exercises performed were the cycling exercises that took place during the laboratory visits. Participants were also asked to refrain from the following: massage therapy, cryotherapy, hot tubs, nutritional recovery products, and pharmacological treatments (e.g., ibuprofen). All participants were advised to adhere to their prescribed diet. To summarize, the order of events for the Phase 4: Long-Term Recovery included:

**Week 2: Experimental Week-Phase 4: Long-Term Recovery**
1. 5 min warm-up of normal double-leg cycling at a self-selected intensity
2. 4 maximal single-leg trials on 170 mm cranks (2 trials/leg)
3. 2 maximal single-leg trials on 120 mm cranks (right leg only)
4. 2 maximal single-leg trials on 220 mm cranks (right leg only)
5. 90 s standardized submaximal single-leg trial at 160 watts with RPE for each leg
6. 2 maximal isokinetic single-leg trials at 120 rpm on 170 mm cranks (right leg only)
Instrumentation

Maximal power

Maximal cycling power was measured using the inertial-load method, which determines maximal power across a range of pedaling rates (e.g., 60-180 rpm). The details of this method have been described by Martin and colleagues (1997). Briefly, resistance was provided solely by the moment of inertia of the flywheel. An infrared diode and detector were mounted on the ergometer frame. A slotted disc that consists of 14 slots spaced \( \pi/8 \) radians and one index slot spaced \( \pi/4 \) radians were located along the perimeter of the disk and the disk was mounted on the flywheel. The index slot allowed for identification and measurement of the angle between each individual slot to correct for machining tolerances in the slots. The surface of the disk reflected the infrared beam, and slots failed to reflect (interrupted) the infrared beam. The detector circuit was programmed to emit a square pulse at each interruption that enabled the angular position of the flywheel to be measured. With time and angular position known, it was possible to calculate angular velocity \( (\omega) \) and acceleration \( (\alpha) \) of the flywheel. Torque \( (T) \) delivered to the ergometer flywheel was averaged over each complete crank revolution and was calculated as the rate of change in angular momentum, \( T = I (\Delta \omega/\Delta t) \), where \( I \) is the flywheel moment of inertia \( (I = .96 \text{ kgm}^2) \). Power \( (P) \) averaged over one complete crank revolution was calculated as rate of change in kinetic energy, \( P = \frac{1}{2} I (\Delta \omega^2/\Delta t) \).

Maximum power was identified as the highest power for a complete revolution within the trial. This measurement technique has been shown to be highly reliable \( (ICC = .99) \) and precise with coefficient of variation values between 2-4\% (Martin et al., 1997). Additionally, Martin, Diedrich, and Coyle (2000) have determined that trained cyclists
produce valid and reliable maximal cycling power with a single day of practice. Finally, inertial-load cycling has been used previously to measure maximal power in single-leg cycling (Martin et al., 2002).

In-house reliability was assessed to determine the internal consistency and test-retest reliability of the maximal single-leg power values. Four participants performed two trials with each leg (right and left) on 3 separate days. Using the Statistical Package for the Social Sciences Software Version 13.0 (SPSS Inc., Chicago, IL) a mixed model repeated measures ANOVA indicated that the means of each leg were consistent for each day (Table 3.1). The mean trials of the right and left leg from Day 1, 2, and 3 were not different across days (Table 3.2). Coefficient of variations for all trials performed on the right and left legs were 4.3% and 3.8%, respectively. The reliability analysis suggested that maximal power data from single-leg cycling are reliable, stable, and precise.

Maximal power with different crank lengths

In addition to measuring maximal power on standard 170 mm crank lengths, maximal power was measured with 120 mm and 220 mm crank lengths. Adjustments to the gear ratio (GR) were made to ensure that the inertial-load resistance at the pedal (IL_p) was the same for both crank lengths as well as the standard 170 mm crank lengths. Specifically, \( IL_p = IL/CL \) where IL was the inertial-load \( (IL = \frac{1}{2} I x GR^2) \) and CL was crank length. Thus, alterations in GR changed IL and offset the effects of CL alterations.

The egrometer was equipped with a chain tensioner to allow for easy GR changes (Figure 3.4a). Altering crank length also influenced pedaling rate (PR) and pedal speed (PS). Pedaling rate (flywheel angular velocity) was calculated from the angular position and time data. Pedal speed was the tangential velocity of the pedal and was calculated
### Table 3.1
Assessment of Reliability-Internal Consistency

<table>
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<tr>
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<th>Internal Consistency</th>
<th></th>
<th></th>
<th>F&lt;sup&gt;a&lt;/sup&gt;</th>
<th>p</th>
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<tbody>
<tr>
<td></td>
<td>Cronbach's Alpha</td>
<td>95% CI</td>
<td>F&lt;sup&gt;a&lt;/sup&gt;</td>
<td>p</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Lower Bound</td>
<td>Upper Bound</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left Leg</td>
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<td></td>
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</tr>
<tr>
<td>Day 1</td>
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<td>Day 3</td>
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<td>.994</td>
<td>.01</td>
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<tr>
<td>Right Leg</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Day 1</td>
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<sup>a</sup>. a = .05

N = 4

### Table 3.2
Assessment of Reliability-Test-Retest

<table>
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<th>p</th>
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<tbody>
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<td></td>
<td>Cronbach's Alpha</td>
<td>95% CI</td>
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<td>F&lt;sup&gt;a&lt;/sup&gt;</td>
<td>p</td>
</tr>
<tr>
<td></td>
<td>Lower Bound</td>
<td>Upper Bound</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Left Leg</td>
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<td>.996</td>
<td>.73</td>
<td>.66</td>
</tr>
<tr>
<td>Right Leg</td>
<td>.884</td>
<td>.223</td>
<td>.992</td>
<td>.34</td>
<td>.45</td>
</tr>
</tbody>
</table>

<sup>a</sup>. a = .05

N = 4
from pedaling rate and crank length (\( PS = PR \text{ (rpm)} \times CL \text{ (m)} \times 2 \pi / 60 \)). Maximal
power was measured as previously described and optimal pedaling rate and pedal speed
that elicit maximal power were also reported (e.g., 120 mm crank lengths yield 800 watts
at 135 rpm and 1.7 m/s).

**Biomechanical joint powers**

Biomechanical joint powers were measured in Phases 1, 2, and 4 during maximal
isokinetic single-leg trials and in Phase 2 during eccentric cycling. The right pedal of the
isokinetic ergometer was equipped with two 3-component piezoelectric force transducers
(Kistler 9251: Kistler USA, Amherst, NY, USA), and the right pedal and crank were
equipped with digital position encoders (U.S. Digital model S5S-1024: Vancouver,
Washington, USA; Figure 3.6c). Normal and tangential pedal forces and pedal and crank
positions were recorded for 3 s at 240 Hz using Bioware Software Version 3.0 (Kistler
USA, Amherst, NY, USA). The normal and tangential pedal forces were resolved into
vertical and horizontal components using the pedal and crank position data.

Position of the anterior superior iliac spine (ASIS) were recorded with an
instrumented spatial linkage (ISL; Figure 3.7). Details of the ISL are described by Martin,
Elmer, Horscroft, Brown, and Shultz (2007). Briefly, the ISL consisted of a ground
anchored base, two aluminum segments, bearings, and digital encoders. The end of the
ISL segment was mounted to a threaded connector that was centered on the participant’s
ASIS and held in place with belt tension and double-sided tape (Figure 3.7b). Position
data from the digital encoders were also recorded using Bioware software. The ISL used
served as a cost-effective, accurate, and valid measure for two-dimensional kinematic
data within the typical range of motions for cycling (Martin et al., 2007).
Prior to the protocol, pedal, crank center, and individual greater trochanter and ASIS positions were determined by a one second static data collection of each participant using the ISL. During the exercise protocol ASIS and pedal and crank position coordinates were measured, which allowed sagittal plane leg segment positions to be determined. More specifically, the coordinates of the ASIS during the exercise protocol and the known distance between the ASIS and the greater trochanter from the static shot allowed for the position of the great trochanter to be inferred throughout the pedal cycle. In addition, crank angle, pedal angle, and the angle created between the pedal and the lateral malleolus allowed for the coordinates of the lateral malleolus to be determined throughout the pedal cycle. With the coordinates of the lateral malleolus and greater trochanter and the known lengths of the foot, shank, and thigh a triangle was formed with known sides. Using the law of cosines \( c^2 = a^2 + b^2 - 2ab\cos C \) the joint angles at the knee and hip were determined. From these data it was possible to calculate joint angular velocities and accelerations at the ankle, knee, and hip. Linear and angular velocities and
accelerations of the limb segments were determined by finite differentiation of position data with respect to time. Position data were filtered using a fourth order zero lag Butterworth Filter and a cutoff frequency was determined based on the recommendations provided by Winter (2005).

Segmental masses, moments of inertia, and location of centers of mass were estimated using the regression equations of de Leva (1996). Sagittal plane joint reaction forces and net joint moments at the ankle, knee, and hip were determined by using inverse dynamic techniques (Elftman, 1939). Joint powers were calculated as the product of net joint torques and joint angular velocities. Power transferred across the hip joint was calculated as the product of the hip joint reaction force and linear velocity. Calculated values for ankle, knee, and hip joint and hip transfer power were averaged over all the complete pedal cycles within the data collection interval.

Rating of perceived exertion

A Borg scale (Borg, 1970) was used to measure rating of perceived exertion during the 90 s standardized submaximal single-leg trial. The Borg scale has been used for over 40 years in various research settings and it has been shown to be both valid and reliable (Garcia, Wolff, & Bejma, 2003). The specific Borg scale used is displayed in Figure 3.8.

Research Design and Analysis

RQ #1: Biomechanical aspects of eccentric muscle damage

A paired t-test was used to confirm that total absolute eccentric work absorbed by the damaged leg matched total concentric work produced by the control leg ($\alpha = .05$).
Figure 3.8. Borg scale.

Descriptive statistics and one-way ANOVA (four levels) were used to determine if eccentric joint power absorption at the ankle, knee, hip, and across the hip (hip JRF) in the damaged leg was different during the damaging exercise ($\alpha = .05$). Subsequent Tukey post hoc tests were used to identify where the joint power differences occurred.

RQ #2: Time course of alterations in overall maximal power

A 2 (eccentric leg vs. control leg) x 4 (24, 48, 72, 96 hr) within factorial repeated measures ANOVA was used to analyze the time course of alterations in overall normalized maximal power. Power values were normalized as a percentage of baseline power (power produced in Phase 1). The 0-hr time point was not used in the analysis as muscle fatigue related to the damage protocol may have been present. Assumptions of sphericity were assessed using the Mauchly test of sphericity, with any violations
adjusted by use of Huynh-Feldt correction. Alpha was set at .05. If there were a main effect for leg then multiple paired t-tests were performed to determine how the legs differed at each time point ($\alpha = .05/4 = .01$). To address the interaction effect for leg x time, subsequent 2 (eccentric leg vs. control leg) x 2 (time points) within factorial repeated measures ANOVA were run to identify where the interaction existed ($\alpha = .05/3 = .02$). If main effect for time existed then separate additional one-way repeated measures ANOVA’s were run on each leg while the alpha level was adjusted ($\alpha = .05/2 = .025$) in order to reduce the probability of type I error.

**RQ #3: Alterations in excitation/relaxation and force-velocity kinetics**

A 3 (120 vs. 170 vs. 220 mm crank length) x 4 (24, 48, 72, 96 hr) within factorial repeated measures ANOVA was used to analyze alterations in normalized maximal power produced on 120, 170, and 220 mm crank lengths. Power values produced on each crank length were normalized as a percentage of baseline power (power measured in Phase 1). The 0-hr time point was not used in the analysis as muscle fatigue may have been present. Assumptions of sphericity were assessed using the Mauchly test of sphericity, with any violations adjusted by use of Huynh-Feldt correction. Alpha was set at .05.

**RQ #4: Biomechanical aspects of power recovery**

A 4 (ankle vs. knee vs. hip vs. hip JRF) x 4 (24, 48, 72, 96 hr) within factorial repeated measures ANOVA was used to analyze the time course of alterations in normalized maximal joint power. Joint power values were normalized as a percentage of baseline joint power (power measured in Phase 1). The 0-hr time point was not used in
RQ #5: Overall power and joint power comparisons

The time course of alterations for overall maximal power, biomechanical knee joint power, and biomechanical hip joint power were graphed to compare recovery profiles.

RQ #6: Time course of alterations in rating of perceived exertion

A 2 (eccentric leg vs. control leg) x 4 (24, 48, 72, 96 hr) within factorial repeated measures ANOVA was used to analyze the time course of alterations in normalized rating of perceived exertion values. RPE values were normalized as a percentage of baseline RPE (RPE measured in Phase 1). The 0-hr time point was not used in the analysis as muscle fatigue related to the damage protocol may have been present. Assumptions of sphericity were assessed using the Mauchly test of sphericity, with any violations adjusted by use of Huynh-Feldt correction. Alpha was set at .05. If there were a main effect for leg then multiple paired t-tests were performed to determine how the legs differed at each time point (\( \alpha = .05/4 = .01 \)). To address an interaction effect for leg x time, subsequent 2 (eccentric leg vs. control leg) x 2 (time points) within factorial repeated measures ANOVA were run to identify where the interaction existed (\( \alpha = .05/3 = .02 \)). If there were a main effect for time then separate additional one-way repeated measures ANOVA’s were run on each joint while the alpha level was adjusted (\( \alpha = .05/2 = .025 \)) in order to reduce the probability of type I error.
4. RESULTS AND DISCUSSION

In this chapter I first present the results of the data analyses and later discuss the results with respect to my research questions and relevant literature.

Analysis

The complete sample consisted of 11 participants, 10 males ($n = 10$) and one female ($n = 1$). All participants cycled a minimum of 5-hr per week and had a variety of cycling backgrounds which included competitive racing, recreational riding, and street commuting. Overall and subgroup demographics are presented in Table 4.1. Participants completed all aspects of the study; however, baseline biomechanical joint powers and eccentric biomechanical joint powers were not collected for one participant due to equipment malfunction. Thus, only this participant’s inertial load power and RPE data were used in the analysis.

Results

RQ #1: Biomechanical aspects of eccentric muscle damage

Table 4.2 displays eccentric work absorbed during the damage trial and concentric work produced during the control trial for each individual. A paired samples t-test did not reveal any differences between absolute eccentric work and concentric work ($t(9) = -0.959$, $p = 0.363$).

The one-way ANOVA that compared joint power absorption during the damaging
Table 4.1  
Descriptive Characteristics

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>All Cyclists</th>
<th>Racing Cyclists</th>
<th>Recreational Cyclists</th>
<th>Commuter Cyclists</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>11</td>
<td>5</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>Male</td>
<td>10</td>
<td>4</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>Female</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age&lt;sup&gt;a&lt;/sup&gt; (yrs)</td>
<td>30.5 ± 7.7</td>
<td>35.4 ± 7.3</td>
<td>28.5 ± 5.3</td>
<td>22.0 ± 2.8</td>
</tr>
<tr>
<td>Height&lt;sup&gt;a&lt;/sup&gt; (cm)</td>
<td>175.9 ± 8.6</td>
<td>171.5 ± 9.7</td>
<td>182.3 ± 5.5</td>
<td>174.3 ± 3.9</td>
</tr>
<tr>
<td>Mass&lt;sup&gt;a&lt;/sup&gt; (kg)</td>
<td>74.1 ± 8.2</td>
<td>71.5 ± 9.6</td>
<td>79.9 ± 4.8</td>
<td>69.2 ± 3.3</td>
</tr>
</tbody>
</table>

<sup>a</sup> Means ± standard deviation (SD)

exercise was significant, \( F(3, 36) = 13.0, p = .00 \). Subsequent Tukey HSD post hoc analyses indicated that knee joint power absorption was greater than ankle, hip, and hip JRF power absorption \( p = .00 \). The homogeneity of variance assumption for the one way ANOVA was violated. Kruskal-Wallis nonparametric test confirmed the parametric finding. Figure 4.1 illustrates the power absorption of each joint with respect to the overall crank power. The ankle, knee, hip, and hip JRF absorbed 12.3 ± 3.5, 114.5 ± 15.4, 31.8 ± 20.5, and 10.0 ± 1.8 watts, respectively. These values were approximately 7%, 68%, 19%, and 6% of overall total crank power, respectively. Figure 4.2 provides a descriptive analysis of instantaneous joint power absorption throughout a complete crank revolution. Because the participants were absorbing power, the crank revolution starts at 360 degrees (top dead center) and goes counterclockwise through 180 degrees (bottom dead center) until 0 degrees for one complete revolution. Thus, the graph is read from left
Table 4.2

Eccentric vs. Concentric Work

<table>
<thead>
<tr>
<th>Participant</th>
<th>Eccentric (Damaged Leg)</th>
<th>Concentric (Control Leg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>-67.9</td>
<td>69.8</td>
</tr>
<tr>
<td>B</td>
<td>-77.6</td>
<td>77.8</td>
</tr>
<tr>
<td>C</td>
<td>-56.4</td>
<td>62.5</td>
</tr>
<tr>
<td>D</td>
<td>-64.5</td>
<td>66.2</td>
</tr>
<tr>
<td>E</td>
<td>-81.8</td>
<td>81.0</td>
</tr>
<tr>
<td>F</td>
<td>-38.3</td>
<td>36.7</td>
</tr>
<tr>
<td>G</td>
<td>-67.4</td>
<td>71.6</td>
</tr>
<tr>
<td>H</td>
<td>-98.2</td>
<td>95.9</td>
</tr>
<tr>
<td>I</td>
<td>-60.0</td>
<td>59.2</td>
</tr>
<tr>
<td>J</td>
<td>-77.9</td>
<td>77.4</td>
</tr>
</tbody>
</table>
| Mean\(^a\) | -69.0 ± 5.0             | 69.8 ± 5.1               \\

\(^a\) Means ± standard error of the mean (SEM)

to right in a counterclockwise manner. Ankle power absorption occurred between 90-30 degrees, knee power absorption began at approximately 90 degrees and continued until 285 degrees, hip power absorption began at 45 degrees and continued until 315 degrees, and hip JRF power absorption occurred between 90-0 degrees.
Figure 4.1. Joint power absorption. Power absorbed in the damaged leg during single-leg eccentric cycling (60 rpm on 170 mm cranks). Joint powers were averaged over one complete crank revolution. Power values are negative because power is being absorbed rather than being produced. Group data are represented by means ± standard error of the mean (SEM). * indicates significantly different than ankle, hip, and hip JRF (α = .05).

RQ #2: Time courses of alterations in overall maximal power

The 2 (eccentric leg vs. control leg) x 4 (24, 48, 72, 96 hr) repeated measures ANOVA indicated a significant main effect for leg ($F(1) = 8.1, p = .02$) and time ($F(3) = 8.7, p = .00$) and a significant leg x time interaction effect ($F(3) = 12.3, p = .00$). The leg, time, and leg x time factors accounted for 44.7%, 46.5 and 55.1% of the variance in power, respectively. The normalized power values (produced on 170 mm cranks) were different between the damage and control legs at 24 and 48 hr ($p < .01$) and approaching significance at 72 hr ($p = .06$). Leg x time interaction effect existed between the 48- and 72-hr time points ($p < .02$) and was approaching significance between the 72- and 96-hr
Figure 4.2. Eccentric cycling biomechanics. Instantaneous power absorbed by the ankle, knee, hip, and across the hip (Hip JRF) in the damaged leg during single-leg eccentric cycling (60 rpm on 170 mm cranks). Joint powers were assessed over one complete crank revolution. Because the participants are absorbing power, the crank revolution starts at 360 deg (top dead center) and goes counterclockwise through 180 deg (bottom dead center) until 0 deg for one complete revolution. The graph is read from left to right. Group data are represented by means ± standard error of the mean (SEM).

time points ($p = .05$). At 24 hr, power was reduced by $9.1 \pm 2.3$ and remained reduced for 72 hr. At 96 hr, power was statistically increased when compared to 24 hr ($p < .025$; Figure 4.3). No significant changes in power were observed in the control leg.

Table 4.3 compares changes in the damaged and control legs pedaling rate, power, and torque characteristics from baseline to 24 hr (produced on 170 mm cranks). Figure 4.4 and Figure 4.5 illustrate the inertial-load power versus pedaling rate and torque versus pedaling rate relationships in the damaged leg at the baseline and 24-hr time points. A second order polynomial regression line (constrained to pass through the same origin) was fitted to the power-pedaling rate relationship.
Figure 4.3. Time course of alterations in maximal power. Power produced by the damaged and control legs (170 mm cranks). Group data are represented by means ± standard error of the mean (SEM). * indicates significantly different from 24 hr in the damaged leg (∝=.025).

Table 4.3

Baseline vs. 24-hr Changes

<table>
<thead>
<tr>
<th>Variable</th>
<th>Δ %</th>
<th>Damage Leg</th>
<th>Control Leg</th>
</tr>
</thead>
<tbody>
<tr>
<td>RPM_{opt}^{a}</td>
<td>0.0</td>
<td>.9</td>
<td></td>
</tr>
<tr>
<td>RPM_{max}^{b}</td>
<td>0.0</td>
<td>1.3</td>
<td></td>
</tr>
<tr>
<td>P_{max}^{c}</td>
<td>-9.3</td>
<td>2.4</td>
<td></td>
</tr>
<tr>
<td>T_{max}^{d}</td>
<td>-11.2</td>
<td>0.0</td>
<td></td>
</tr>
</tbody>
</table>

a. optimal pedaling rate for producing maximal power
b. maximal pedaling rate
c. maximal power
d. maximal torque
Figure 4.4. Baseline vs. 24-hr inertial-load power-pedaling rate relationships. Relationships are for the damaged leg only (170 mm cranks). Group data are represented by means ± standard error of the mean (SEM).

RQ #3: Alterations in excitation/relaxation and force-velocity kinetics

The 3 (120 vs. 170 vs. 220 mm crank length) x 4 (24, 48, 72, 96 hr) repeated measures ANOVA indicated a significant main effect for time ($F(3) = 11.59, p = .00$). The time factor accounted for 53.7% of the variance in power. Power (averaged across the three crank lengths) was reduced by $9.1 \pm 2.3\%$ at 24 hr and remained reduced at 48 hr. At 72 and 96 hr, power was statistically increased when compared to 24 hr ($p < .05$; Figure 4.6).

RQ #4: Biomechanical aspects of power recovery

The 4 (ankle vs. knee vs. hip vs. hip JRF) x 4 (24, 48, 72, 96 hr) repeated measures ANOVA revealed no significant findings for joint ($F(3) = .41, p = .75$), time ($F(3) = 1.18, p = .34$), or joint x time ($F(9) = 1.27, p = .26$).
Figure 4.5. Baseline vs. 24-hr inertial-load torque-pedaling rate relationships. Relationships are for the damaged leg only (170 mm cranks). Group data are represented by means ± standard error of the mean (SEM).

RQ #5: Overall power and joint power comparisons

Because there were no alterations in any of the joint powers no comparisons were made between joint powers and overall power produced at the crank.

RQ #6: Time course of alterations in rating of perceived exertion

The 2 (eccentric leg vs. control leg) x 4 (24, 48, 72, 96 hr) repeated measures ANOVA indicated a significant main effect for leg \( (F(1) = 11.72, p = .01) \) and time \( (F(3) = 4.06, p = .02) \). The leg and time factors accounted for 54.0% and 28.9% of the variance in RPE, respectively. The normalized RPE values were different between the damaged and control legs at 24 and 72 hr \( (p < .01) \) and approaching significance at 48 hr \( (p = .02) \).
Figure 4.6. Time course of alterations in maximal power produced on different crank lengths. Power produced on 120, 170, and 220 mm crank lengths in the damaged leg. Group data are represented by means ± standard error of the mean (SEM). * indicates significantly different from 24 hr (α = .05).

At 24 hr damaged leg RPE increased by 12.1 ± 2.4% and remained increased for 72 hr. At 96 hr RPE was statistically decreased (p < .025; Figure 4.9). No changes in RPE were observed in the control leg when compared to 24 hr.

Discussion

The purpose of the present investigation was to quantify neuromuscular function during damaging eccentric exercise and to evaluate recovery of maximal neuromuscular function and perceptual responses to a challenging submaximal task.
Biomechanical aspects of eccentric muscle damage

One of the novel findings in this investigation was that the knee joint absorbed the majority of the eccentric work (68%) during unaccustomed eccentric cycling (Figure 4.1). More specifically, knee power absorption began at approximately 90 degrees and continued until 285 degrees (counter clockwise), which suggests that knee extensor muscles were primarily active while the leg was being flexed (Figure 4.2). In contrast, the hip and ankle joints absorbed very little power and muscles spanning these joints were most likely damaged to a lesser extent. EMG data would be required to confirm the timing, duration, and magnitude of muscle activity throughout the crank cycle. Two participants relied heavily on absorbing eccentric work with their hip joints whereas the
other eight participants absorbed primarily using the knee. Interestingly, these two
participants chose to use eccentric hip extension to absorb the damaging work. Clearly,
these different absorption strategies (eccentric knee vs. hip extension) demonstrate intra-
participant variability that could be associated with learning the novel eccentric task
and/or single-leg coordination strategies.

Although there are no previous reports of eccentric cycling biomechanics, Reiser
et al. (2002) reported that the ankle, knee, hip, and hip transfer joints produced 11%,
55%, 25%, 8% of total crank power respectively during submaximal cycling in healthy
individuals who cycled in the recumbent position. In this investigation the ankle, knee,
hip, and hip transfer were absorbing power rather than producing power, however, their
relative contributions to the overall power at the crank (7%, 68%, 19%, 6%) support
those contributions reported by Reiser et al. (2002). This supports the notion that the
single-leg eccentric cycling in the recumbent position allowed for smooth cycling with
similar biomechanics of double-leg cycling.

In addition to eccentric power absorption, total mechanical work was also
matched between the damage and control exercises. This allowed for subsequent
comparisons to be made between the damaged and control legs in order to identify those
alterations that were specifically related to eccentric muscle damage. Without a true
mechanically matched control group confounding factors related to fatigue, mechanical
work, and the testing protocol cannot be removed. Of the previous groups who evaluated
maximal power, only Sargeant and Dolan (1987) included a control group but did not
match for mechanical work, intensity, or duration.
To my knowledge, this investigation was the first to include a mechanical matched control group combined with a biomechanical quantification of eccentric work. Based on the finding that the knee joint absorbed the majority of eccentric work, a working hypothesis was developed that power produced by the knee joint during maximal cycling would exhibit the greatest reduction and require the longest time to recover when compared to ankle and hip power production.

Maximal power and recovery

A confirmatory finding in this investigation was that maximal power was reduced by 9% at 24 hr which is close to the previous reductions of 10-18% (Byrne & Eston, 2002b; Sargeant & Dolan, 1987; Twist & Eston, 2005). The cycling protocol used in the current investigation was shorter (3 s) than those used in previous investigation (6-20 s) and likely was not influenced by fatigue. A nonfatiguing maximal power protocol may explain the slightly lower power reduction at 24 hr. Stevens (1996) and Byrne and Eston (2002a) reported immediate reductions of 2-12% in maximal work and 5-8% in vertical jump performance, respectively. These differences may be due to the fact that Stevens focused on stimulation parameters rather than muscle damage and recovery whereas jump performance (e.g., jump height) serves as an indirect measure of maximal power. In contrast, Malm and colleagues (1999) did not observe changes in immediate average Wingate power. Previous investigators (Byrne et al., 2004; Twist & Eston, 2005) have speculated that the damage protocol employed may not have been sufficient to induce muscle damage.

In this investigation maximal power remained reduced for several days and was eventually statistically restored at 96 hr (Figure 4.3). Sargeant and Dolan (1987) and later
Byrne and Eston (2002b) reported similar recovery time periods of 120-168 hr (5-7 days) and Twist and Eston (2005) measured reductions in maximal power at the conclusion of their 72-hr study. Although jump height is an indirect measure of maximal power, Byrne and Eston (2002a) observed prolonged reductions in maximal jump height that persisted for 72 hr. Despite the variations in damage and recovery protocols, the time course of alterations in maximal power are remarkably consistent across investigations. The small but growing body of literature in maximal power recovery would suggest that power is a more functional measure compared to traditional isometric and isokinetic strength. Even considering the wide variety of damage protocols used to elicit muscle damage, the overall trend is that immediate reductions in power are less severe and recovery is achieved in a timelier manner compared to traditional strength measures.

One advantage of the damage-recovery model used in this investigation was that each participant served as his/her own control. More specifically, each participant performed eccentric exercise with their right leg and mechanically matched concentric exercise with their left leg. The alterations in maximal power were specific to the eccentrically exercised leg as no alterations were detected in the control leg. The absence of alterations in the control leg likely rule out any central systemic effects associated with muscle damage. Alternatively, systemic effects could be related to the magnitude of power reductions induced by the damage protocol. To my knowledge, systemic effects related to muscle damage have not been considered in previous maximal power investigations.

The overall reductions in maximal power and isometric and isokinetic strength have been attributed to selective damage to type II muscle fibers in humans and animals.
(Byrne & Eston, 2002b; Friden et al., 1983; Frieden & Lieber, 1992; Lieber & Friden, 1988; Twist & Eston, 2005). Optimal pedaling rate during inertial-load cycling serves as a surrogate measure of muscle fiber type distribution (Hautier et al., 1996; Martin et al., 2000c). If type II fibers were selectively damaged optimal pedaling rate would most likely be reduced resulting in a leftward shift in the power-pedaling rate curve. The power-pedaling rate data collected in this investigation do not support the notion that type II muscle fibers were selectively damaged as no alterations were observed in optimal pedaling rate, power-pedaling rate relationship, or torque-pedaling rate relationship (Figure 4.4 and 4.5). Thus, additional mechanisms were explored to explain the alterations in maximal power.

Several groups have reported reductions in muscle glycogen levels following eccentric exercise (Asp et al., 1998; Costill et al., 1990; O'Reilly et al., 1987), which could also influence maximal power production and recovery. In the current investigation inertial-load cycling trials were approximately 3-4 s, which would emphasize a greater reliance on immediate phosphocreatine stores rather than glycogen metabolism (McArdle, Katch, & Katch 2001). Therefore, glycogen depletion was not likely responsible for the alterations in power. In contrast, the cycling protocols (6-20 s) used in previous investigations (Byrne & Eston, 2002b; Malm et al., 1999; Sargenat & Dolan, 1987; Twist & Eston, 2005) would have relied more on glycogen metabolism and possibly depleted glycogen stores.

In summary, eccentric muscle damage impacts dynamic neuromuscular function as maximal power is immediately reduced and slow to recover. These results add to the limited reports on maximal power recovery and help to emphasize that maximal power is
a more functional measure compared to traditional measures of isometric and isokinetic strength. Selective damage to type II muscle fibers was not likely responsible for the reduction in power and the power protocol used eliminated glycogen depletion as a possible contributor. The intrinsic mechanism, however, that limits maximal power recovery remains unknown and warrants further investigation.

Mechanism that limits maximal power recovery

A second attempt was made to identify the intrinsic mechanism that limits maximal power. Specifically, the ergometer crank length was manipulated (120 vs. 170 vs. 220 mm) to separate the relative influential contributions of force-velocity and excitation-relaxation kinetics (Caiozzo & Baldwin, 1997; Martin, 2007; Martin et al., 2000a; Martin et al., 2001; Neptune & Kautz, 2001). The crank length perturbation had no effect as maximal power did not differ between the three crank lengths at any time points (Figure 4.6). If alterations in excitation/relaxation kinetics were primarily responsible for the reduction in maximal power, then power produced on 120 mm cranks would have been compromised to a greater extent. Alternatively, if alterations in force-velocity kinetics were primarily responsible, then power produced on 220 mm cranks would have reduced to a greater extent.

Clearly, maximal single-leg cycling on novel short and long crank lengths would provide challenges for the participants and possibly influence the outcomes of this investigation. It is important to emphasize that trained cyclists produce valid and reliable maximal cycling power (double-leg) with a single day of practice (Martin et al., 2000b). Furthermore, inertial-load cycling has also been used to measure maximal power during single-leg cycling (Martin et al., 2002). Based on these previous investigations I chose to
counterweight inertial-load cycling (9.9 kg) and include a week of practice trials. Despite these efforts, it is possible that the 10 cm perturbation in crank length was not large enough to detect differences in maximal power. Additionally, single-leg coordination using novel crank lengths may have also influenced power production.

Several groups have offered theories that describe the intrinsic mechanism that limits neuromuscular function in damaged muscle as either mechanical in nature (Barash et al., 2002; Lieber et al., 1996; Morgan, 1990) or excitation related (Warren et al., 1993, 2001). As stated earlier, these theories were developed primarily on measures obtained during isometric and isokinetic movements, which might not be applicable to repetitive voluntary movements such as cycling. Sargeant and Dolan (1987) measured faster recovery in maximal power produced during 80 rpm compared to 110 rpm but offered no explanation for this improved recovery. The relative contributions of force-velocity and excitation/relaxation kinetics increase linearly with increasing pedaling rate, thus it is unknown which mechanism limited recovery. Morgan et al. (2004) explained that changes in unloaded shortening velocity and active number of sarcomeres in series (force-velocity kinetics) were responsible for the reduction in force during constant velocity shortening contractions in feline gastrocnemius muscle. Perhaps, expanding upon Morgan’s work by using in situ work loops (Stevens, 1996), would more clearly identify the mechanism.

In summary, a crank length perturbation of 10 cm had no effect on maximal power recovery suggesting that excitation/relaxation and force velocity/kinetics may have been altered in similar manners. Partial replication of this study could be performed using normal double-leg cycling crank length perturbations to rule out any single-leg
coordination issues. Although the mechanism that limits maximal power recovery remains unknown it raises the question of what other possible factors could explain the immediate and prolonged recovery (96 hr) in maximal power?

**Biomechanical aspects of recovery**

In a third attempt to identify the mechanism responsible for power reduction, biomechanical techniques were used to describe the extent to which the ankle, knee, and hip joints produced power during recovery. Because the knee joint absorbed the majority of eccentric work during the damaging exercise I expected the knee to exhibit the greatest reductions in power and require the most time for recovery. Joint powers, however, produced by the ankle, knee, hip, and across the hip did not differ during the recovery period (0-96 hr).

To my knowledge, there are no previous reports that describe the biomechanics of maximal cycling, thus, this investigation is the first to document the process of producing power at the joint level during maximal exercise. Several groups have reported the relative contributions of power produced by each joint with respect to overall power produced at the crank in healthy individuals during submaximal cycling (Broker & Gregor, 1994; Ericson, 1986, 1988a, 1988b; Ericson et al., 1988; Reiser et al., 2002). In these investigations power produced by each joint was influenced by the overall crank power manipulated by the investigators (e.g., 150 vs. 250 watts influenced ankle, knee, and hip contributions). In general, the knee is the dominant power producing joint during submaximal cycling but some individuals rely more heavily on hip power production (Broker & Gregor, 1994; Reiser et al., 2002)
Again, factors associated with maximal single-leg cycling and individual pedaling-power producing style may have influenced the results in this investigation and accounted for some of the variability. The cycling biomechanics collected during the baseline trials, however, were consistent and very similar to those of double-leg cycling (comparisons made to unpublished data from our lab). Although there were wide variations in the biomechanics of recovery at the ankle, knee, and hip, overall power produced at the crank was much more stable. Recently, Chang (2008) reported that a global strategy for whole limb control (leg length and orientation) was retained when walking with paralyzed or re-innervated feline ankle extensor muscles despite large changes in individual joint kinematics at the ankle, knee, and hip. Similarly, in this investigation, global recovery of overall power produced at the crank was achieved despite large individual changes in ankle, knee, and hip power. It is possible that the variability in joint powers may reflect differences in the location of muscle damage (damaged knee vs. hip extensors) and/or compensation strategies. Even though these data do not tell a clear story it may be more important to emphasize that the overall power at the crank was restored. The overall power produced at the crank is delivered to the environment and ultimately determines how an individual will move.

Perceptual recovery during submaximal exercise

Another novel finding in this investigation was that rating of perceived exertion (RPE; Borg, 1970) immediately increased, remained elevated for several days, and returned to baseline at 96 hr in the damaged leg (Figure 4.9). Interestingly, rating of perceived exertion exhibited a similar relative recovery profile to maximal power and was inversely related ($r = -.43$). At 24 hr alterations in perceived exertion and power were
12% and 9%, respectively, and full recovery for both measures was restored within 96 hr. The alterations in perceived exertion like those of maximal power were specific to the eccentrically exercised leg as no alterations in perceived exertion were detected in the control leg. Thus, information regarding perceived exertion compliments power rather nicely. Furthermore, because most voluntary tasks are submaximal, perceived exertion may perhaps be even more important than information regarding maximal power as few tasks are performed at maximal effort.

Although perceived exertion has been widely used to assess perceptual responses during maximal and submaximal exercise, no previous investigators have used rating of perceived exertion to assess recovery following damaging eccentric exercise. In an effort to compare perceived exertion responses with traditional muscle pain and soreness responses, I also evaluated muscle soreness in response to a dynamic wall squat (Kendall & Eston, 2002; Marginson & Eston, 2002; Twist & Eston, 2005). The muscle soreness findings support the extensive work in this area in which muscle pain and soreness are most elevated between 24-48 hr and very slow to recover (Clarkson et al., 1992; Jamurtas et al., 2005; Twist & Eston, 2005). Muscle soreness, unlike rating of perceived exertion, remained elevated for the entire duration of the investigation and did not recover. Rating of perceived exertion and muscle soreness were moderately related ($r = .58$).

Surprisingly, although the participants were very sore they were able to perform challenging submaximal exercise at only a moderately elevated perceived exertion level. Perceived exertion seems to be a more functional recovery measure than muscle pain soreness. One limitation when comparing perceived exertion and muscle soreness is that the submaximal exercise consisted primarily of active concentric muscle actions (normal
concentric cycling) whereas the muscle soreness exercise (dynamic wall squat) occurred during a movement that included both active concentric and eccentric muscle actions.
5. SUMMARY, CONCLUSION, AND RECOMMENDATIONS

In this chapter I summarize the findings of this investigation, draw conclusions, and offer recommendations for future research.

Summary

Biomechanical analysis of unaccustomed eccentric cycling indicated that the knee joint absorbed the majority of eccentric work, which is the first quantification of eccentric muscle damage during a multijoint movement. Eccentric muscle damage greatly influenced dynamic neuromuscular function as maximal power was immediately reduced and remained reduced for several days. There was no evidence of selective damaged to type II muscle fibers and excitation/relaxation and force velocity/kinetics did not preferentially limit recovery. Thus, the mechanisms responsible for the reductions in power are still not clear. Biomechanical aspects of recovery exhibited large variations and did not tell a clear story. Perceived exertion immediately increased and recovered within 96 hr whereas traditional muscle soreness did not recover. No central systemic effects related to muscle damage were observed as alterations in power or perceived exertion were not present in the control leg. These results add to the limited reports on maximal power recovery and help identify the functional limitations associated with muscle damage and recovery. Due to the wide range of recreational and occupational activities that can elicit eccentric muscle damage and the lengthy time needed for
recovery it is important to increase our understanding so that effective treatments can be developed to minimize muscle damage and speed the recovery process.

**Conclusion**

In conclusion, multijoint eccentric exercise induced extensive muscle damage and produced greater alterations in neuromuscular and perceptual responses compared to equally matched concentric exercise. Maximal power and perceived exertion provided more functional indications of recovery compared to traditional measures.

**Future Recommendations**

I recommend that future investigators continue to explore the perceptual responses associated with submaximal recovery following damaging exercise. Specifically, perceptual recovery during a standardized task that includes both an active concentric and eccentric phase, such as stair climbing/descending, could offer a more complete description. Assessments of profile of mood states (POMS) during recovery may be useful to identify mood related disturbances related to muscle damage. I also recommend partially replicating this investigation by using double-leg cycling in order to rule out coordination and learning factors related to single-leg cycling. Double-leg cycling should require less practice and possibly reduce coordination issues associated with eccentric cycling and inertial-load cycling with different crank lengths. The effects resulting from crank length manipulations may be more easily detected using double-leg cycling as well as alterations in joint powers. Incorporating electromyography techniques along with biomechanical measures would provide a more detailed quantification of muscle damage and recovery. Finally, I recommend the use of isolated muscle
preparations with work loops to elicit muscle damage and to evaluate recovery. Altering cycle frequencies and muscle strain length may help identify an underlying mechanism that limits recovery of maximal work and power.
REFERENCES


