Effect of hydrotherapy on recovery of muscle-damage and exercise-induced fatigue.

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Overview

Achieving adequate and appropriate recovery from exercise is essential in ensuring optimal performance during repeated bouts of exercise. The use of various recovery interventions has become popular in an attempt to enhance subsequent performance and accelerate post-exercise recovery. The application of various post-exercise hydrotherapy interventions has become increasingly popular, however, the majority of current recovery practices appear to be based largely on anecdotal evidence as opposed to rigorous scientific research or evidence based findings. Physiologically, various hydrotherapy protocols have been shown to affect the body via fluid shifts (interstitial to intravascular space), changes in blood flow and cardiovascular function, and reductions in oedema. The possible psychological effects of water immersion must also be considered, with athletes commonly reporting reduced sensations of fatigue and soreness following immersion. Current literature suggests both hydrostatic pressure and water temperature to be important factors influencing the success of hydrotherapy. The overall aim of the present thesis was to enhance current knowledge and understanding with regards to the physiological and performance effects of various forms of hydrotherapy, used as a post-exercise recovery intervention. Initially, four cold water immersion interventions were compared to active recovery, performed between two bouts of high intensity cycling in hot environmental conditions. Effectiveness of recovery was determined via performance in a subsequent exercise bout; in addition, core body temperature, lactate, and heart rate were recorded. The remaining studies were designed to investigate the effects of cold water immersion, hot water immersion, contrast water therapy, and passive recovery

(control) following exercise-induced fatigue and exercise-induced muscle damage. Rate of recovery was assessed through changes in performance, core body temperature, thigh girths, blood markers, and perceived exertion/soreness. The results of the combined studies indicate cold water immersion to be more effective than active recovery when performed immediately post-exercise between two bouts of high intensity cycling in hot environmental conditions. Additionally, both cold water immersion and contrast water therapy were effective in aiding recovery from exercise-induced fatigue and exercise-induced muscle damage. Performance variables indicated an improved maintenance or return of performance following these recovery protocols. The present studies have provided additional information to the limited knowledge base regarding the effect of post-exercise hydrotherapy interventions, specifically, the effect of such interventions on subsequent athletic performance. In conclusion, cold water immersion and contrast water therapy appear to be superior to hot water immersion, active recovery, and passive recovery following fatiguing and muscle damaging exercise. Functional and physiological recovery was enhanced following the use of these two recovery protocols.

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Dedication

I would like to dedicate this thesis to an amazing woman, my aunty, Denise Robinson who lost her gallant fight with cancer on January 13th 2006. I am so proud of you; you taught us the true meaning of courage. You are an inspiration, will always be in our hearts, and the beautiful memories of your life never forgotten.

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List of Abbreviations

CWI	Cold water immersion
CWT	Contrast water therapy
HWI	Hot water immersion
PAS	Passive recovery (control)
PPO	Peak power output
HR	Heart rate
RPE	Rating of perceived exertion
DOMS	Delayed onset muscle soreness
IL-6	Interleukin-6
CK	Creatine kinase
Mb	Myoglobin
LDH	Lactate dehydrogenase
ROM	Range of motion
bpm	Beats per minute (Heart Rate)
W	Watts
N	Newtons

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CHAPTER ONE

Introduction

1.0   Background

In recent years, the area of recovery, specifically an athlete's ability to regain physiological and psychological function following training and competition, has gained considerable interest. Recovery interventions are frequently performed in an attempt to accelerate recovery and optimize subsequent training and performance. By integrating recovery into a training program it is hoped training adaptations will be maximized through minimization of fatigue. Nowadays, many athletes' livelihoods are dependent on successful performances. Considerable pressure and competition has lead to athletes needing to train at greater levels to achieve success. Athletic training and competition regimes often require repetitive high intensity and/or high volume work loads. Intensive training often results in athletes being frequently exposed to muscle damage, swelling, energy depletion, increased risk of injury and overreaching, a depressed immune system, and cumulative fatigue. It is for these reasons that post-exercise recovery is growing in popularity, with the common aim of maximizing physiological and psychological recovery following intense training. Therefore, recovery has become an integral aspect of any elite athletic training program.

In an attempt to reduce recovery time and minimize post-exercise decrements in performance a variety of recovery interventions have been investigated, with varying degrees of success. More recently, various methods of hydrotherapy have proven popular and are often incorporated into an athlete's post-exercise regime. Despite hydrotherapy protocols becoming common practice, little scientific literature exists to support the use of these interventions.   More

specifically, the optimal mode of hydrotherapy, water temperature, duration of exposure, and frequency of treatment remain to be elucidated.

1.1 Statement of the problem

The purpose of the present thesis was to investigate the effects of various hydrotherapy interventions on the recovery of subsequent performance in hot environmental conditions, recovery of exercise-induced fatigue, and recovery of exercise-induced muscle damage.

1.2 Specific aims of the studies

1. Chapter Three: Effect of cold water immersion on repeat cycling performance and thermoregulation in the heat.

The purpose of this study was to investigate the effects of various cold water immersion protocols and active recovery on repeat cycling performance and thermoregulation in a hot environment.

2. Chapter Four: Effect of hydrotherapy on recovery from fatigue.

The purpose of this study was to investigate the effect of three different hydrotherapy interventions, specifically cold water immersion, hot water immersion, and contrast water immersion, on the recovery of exercise-induced fatigue and next day performance in trained cyclists.

3. Chapter Five: Effect of hydrotherapy on the signs and symptoms of delayed onset muscle soreness.

This chapter incorporates three independent studies designed to examine the difference between three hydrotherapy interventions (cold water immersion, hot water immersion, contrast water therapy) compared to passive recovery, on recovery following a controlled muscle-damaging exercise task. The functional and physical symptoms of delayed onset muscle soreness (DOMS) and recovery of performance were assessed.

CHAPTER TWO

Literature Review

2.0   Introduction

Elite athletes and coaches will seek any small advantage in their performance and preparation for competition.   Optimal recovery from training and competition may provide numerous potential benefits during repetitive high-level training and competition. Fatigue, in its various forms, can be a significant factor for athletes as it may cause a reduction in exercise performance and hasten termination of exercise. In addition, muscle damage has been shown to cause chronic pain, to decrease muscle function, and to limit the ability to train and compete at high levels (Weerapong, Hume, & Kolt, 2005).   Therefore, factors relating to exercise-induced fatigue and muscle damage, and the influence of popular hydrotherapy recovery interventions on these factors, will be critically reviewed here in an attempt to identify key findings and directions for further research.

2.1   Exercise in hot environmental conditions

Thermoregulation has been defined as a complex system involving physical, chemical, and behavioural processes that allow the maintenance of body temperatures within a restricted range under conditions of variable internal and external heat loads (Kaciuba-Uscilko & Grucza, 2001).   With the commencement of sub-maximal exercise, core body temperature gradually increases until heat production can be balanced with heat loss (Kay, Taaffe, & Marino, 1999), although depending on the exercise intensity, environmental conditions, and a range of other factors this is not always possible. Elite athletes tend to have an enhanced thermoregulatory capacity, in part because of an increased plasma volume, allowing more blood to be available to assist peripheral convective cooling.   Athletes also usually have an increased

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sweating response, that, while it may improve evaporative heat loss, also results in a progressive reduction in body water during exercise (Reilly, Drust, & Gregson, 2006). Effective thermoregulation prevents hyperthermia and assists in the maintenance of body water stores despite increased sweating, while allowing exercise to continue at a high level (Reilly *et al.*, 2006).

2.1.1 *Responses to exercise in a hot environment*

In a hot environment an athlete's exercise capacity is often reduced (Armada-da-Silva, Woods, & Jones, 2004). Compared to thermoneutral conditions, exercise in the heat often results in altered muscle metabolism and increases in core body temperature, perceived exertion, heart rate, and body water losses (Nielsen & Nybo, 2003; Nielsen, Savard, Richter, Hargreaves, & Saltin, 1990). In combination, these effects appear to reduce endurance performance (Nielsen, 1974; Nielsen & Nybo, 2003).

Exhaustion during endurance exercise has been closely associated with core body temperature reaching a critically high limit (Nielsen & Nybo, 2003). Multiple studies support the theory of a critical limiting temperature (~40°C) initiating a decline in performance or termination of exercise in a hot environment (Fuller, *et al.,* 1998; Gonzalez-Alonso *et al.*, 1999; MacDougall, Reddan, Layton, & Dempsey, 1974; Nielsen *et al.*, 1993; Nielsen, *et al.,* 1997). A high core body temperature has also been found to impair maximal muscle activation, resulting in a reduced force production and output (Nybo & Nielsen, 2001). These factors appear to indicate a regulatory failure of the central nervous system (Nielsen & Nybo, 2003).

Many studies have investigated the effect of precooling the body on performance in hot environments (Table 2.1). Precooling strategies involve reducing core body temperature prior to exercise (Marino, 2002) and are thought to enhance performance by increasing the overall capacity for heat storage, therefore reducing cardiovascular and thermoregulatory strain (Kay *et al.*, 1999). The time taken to reach the critical limiting temperature is also increased, allowing a longer period until exercise intensity can no longer be maintained (Marino, 2002). While current knowledge suggests that whole body precooling is beneficial to increase exercise capacity, no studies have examined the effect of post-exercise cooling on subsequent performance in a hot environment. Precooling may also be implemented during exercise e.g. half time of matches. While this is often viewed as a post-exercise recovery intervention, it also effectively acts as a precooling strategy prior to the subsequent exercise bout. This area should be further investigated as many sporting events require multiple performances within a short period of time.

Table 2.1. Summary of precooling studies including methods and outcomes (Marino, 2002; Page 90). * Oesophageal temperature; # rectal temperature; ^ tympanic temperature; T_c core temperature; ΔT_c change in core temperature; *rh* relative humidity.

Study	Precooling Method	Ex Protocol	Pre-ex T_c (°C)	ΔT_c at end ex	Ambient conditions	Outcome
Bergh & Ekblom (1979)	Swimming water temp 13-15°C	Arm & leg ex to exhaustion within 5-8 min	34.9 *	Not reported	20-22°C	Lowering T_c reduced physical performance
Schmidt & Brück (1981)	Cold air 0°C	Cycling with increasing workload to exhaustion	36.4 *	0.6°C	18°C	Increased time to exhaustion and work performed
Hessemer *et al.* (1984)	Cold air 0°C	60min work rate test	36.4 *	0.4°C	18°C	Increased work rate following precooling
Olschewski & Brück (1988)	Cold air 0°C	Cycling with increasing workload to exhaustion	36.9 *	0.4°C	18°C 50% *rh*	Increased endurance time following precooling
Kruk *et al.* (1991)	Cold air 5°C	Cycling at 50% VO_{2max} for 30 min	37.0° #	0.5°C	5°C	Precooling reduced exercise capacity in a cold environment
Lee & Haymes (1995)	Cold air 5°C	Running at 82% VO_{2max} to exhaustion	0.37 #	1.5°C	24°C 51-22% *rh*	Precooling increased exercise endurance and rate of heat storage
Booth *et al.* (1997)	Water immersion 23-24°C	30 min self paced treadmill running	36.7 #	2.2°C	31.6°C 60% *rh*	Increased distance run in 30 min by 304 m (+4%)
González-Alonso *et al.* (1999)	30 min water immersion	Cycling at 60% VO_{2max} to exhaustion	35.9 *	4.2°C	40°C 19% *rh*	Performance time increased. Exercise termination at identical T_c to control
Kay *et al.* (1999)	Water immersion 24°C	30 min cycling time trial	0 #	1.0°C	31.4°C 60.2% *rh*	Precooling the skin alone increased distance cycling by 0.9 km and increased rate of heat storage
Marsh & Sleivert (1999)	30 min water immersion	70 second cycling power test	36.4 #	0.1°C	29°C 80% *rh*	Mean 70 second power output increased following precooling by 2.7%
Booth *et al.* (2001)	Water immersion 24°C	35 min cycling at 60% VO_{2peak}	36.4 *	1.9°C	34.9°C 46.4% *rh*	Precooling had limited effect on muscle metabolism
Cotter *et al.* (2001)	Ice vest with and without thigh cooling + cold air 3°C	20 min cycling at 65% VO_{2peak} + 15 min work performance (35 min total)	36.8 *#	1.7°C	33°C	Precooling reduced physiological and psychophysical strain and increased endurance performance
Duffield *et al.* (2003)	Ice vest	80 min repeat sprint cycling	37.4 #	1.2°C	30°C	Precooling did not improve performance, perception of thermal load was reduced
Castle *et al.* (2006)	1) Ice vest 2) Cold water 3) Ice packs on upper legs	Intermittent sprint cycling protocol	1) ~ 36.5 2) ~ 36.0 3) ~ 36.5 #	1) 3.4°C 2) 3.5°C 3) 3.0°C	33.7°C 51.6% *rh*	Leg precooling provided the greater effect on performance than upper or whole body cooling
Yeargin (2008)	Ice vest (20 min)	Incremental step test on a treadmill	37.1°C ^	Not reported	30-32°C 50% *rh*	Precooling reduced physiological and psychophysical strain and improved performance

2.2 Exercise-Induced Fatigue

Fatigue, although well researched, is a complicated phenomenon with many underlying mechanisms that remain largely unknown. Fatigue has commonly been defined as a reduced capacity for force development (Fitts & Holloszy, 1976). However, this definition is now considered inappropriate as it does not acknowledge the possibility of low-frequency fatigue (impairments in excitation/contraction coupling, characterised by selective loss of force at low stimulation frequencies of 10-20Hz), occurring when the contractile response to low frequency stimulation is reduced, while at the same time the response to high frequency stimulation is unaffected (MacIntosh & Rassier, 2002). Therefore, fatigue may be better defined as "a response that is less than the expected or anticipated contractile response, for a given stimulation". This would allow changes in contractile performance and both low and high frequency fatigue to be identified (MacIntosh & Rassier, 2002).

Enoka (2002) has outlined nine processes that can be impaired during physical activity (Figure 2.1). These processes include: activation of the primary motor cortex, the central nervous system drive to the motor neurons, the muscles and motor units that are activated, neuromuscular propagation, excitation contraction coupling, the availability of metabolic substrates, the intracellular environment, the contractile apparatus and muscle blood flow (Enoka, 2002). The failure or reduced functional capacity of any of these processes may result in fatigue.
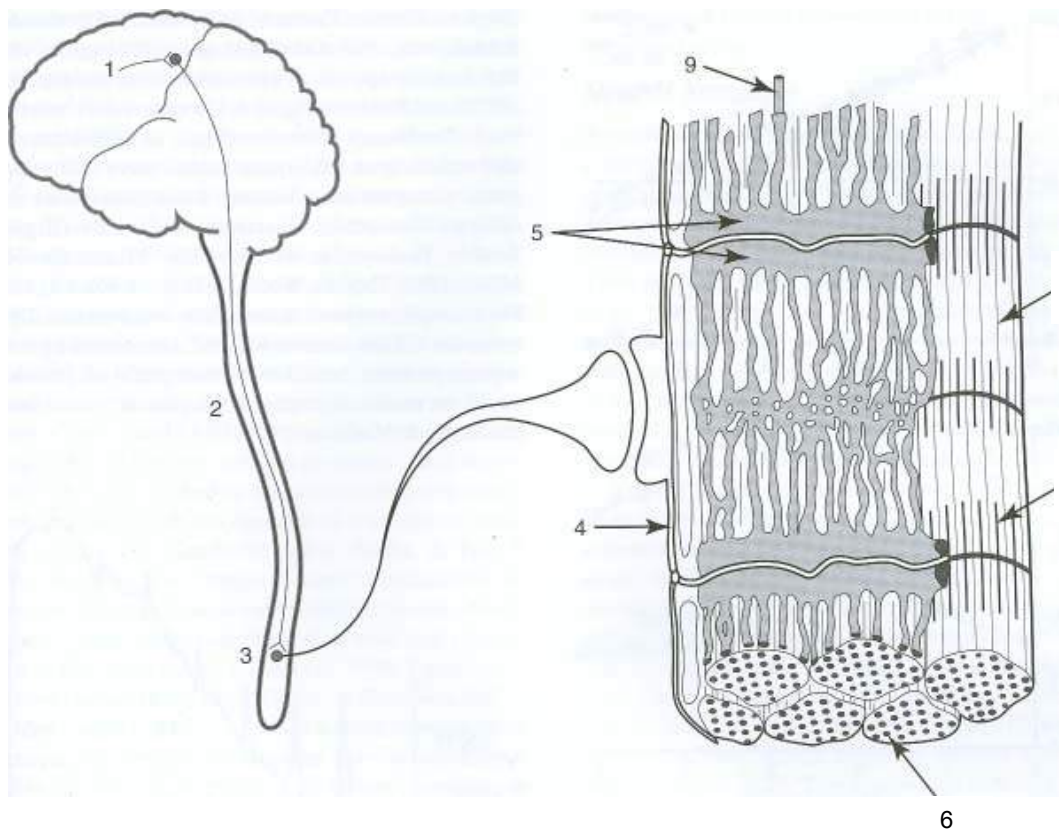
Figure 2.1. The locations of nine processes that may contribute to fatigue during physical activity (Enoka, 2002; Page 375)

1. Activation of the primary motor cortex
2. Central nervous system drive to motor neurons
3. Muscles and motor units that are activated
4. Neuromuscular propagation
5. Excitation-contraction coupling
6. Availability of metabolic substrates
7. Intracellular milieu
8. Contractile apparatus
9. Muscle blood flow

The causes of fatigue in animals and humans has been well-researched (MacIntosh & Rassier, 2002; McComas & White, 1996). However, there is limited research into the mechanisms by which muscle is restored to a pre-fatigued level. The ability to restore muscle to a pre-fatigued state, enabling maximal performance capabilities to be achieved again, is an essential component of sporting performance. Therefore, recovery interventions following fatigue-inducing exercise may play a critical role in subsequent performance.

2.2.1 Central Fatigue and Peripheral Fatigue

Fatigue can be of central or peripheral origin. Central fatigue occurs when the muscles are capable of a larger output than the central nervous system is able to manifest (MacIntosh & Rassier, 2002; McComas & White, 1996). It has been hypothesised that reductions in the power output of skeletal muscle are the result of altered efferent command from the brain (St Clair Gibson & Noakes, 2004). It has also been speculated that changes in the concentration of various neurotransmitters (e.g. increases in serotonin, and decreases in dopamine and acetylcholine) may result in fatigue (St Clair Gibson & Noakes, 2004). In contrast, peripheral fatigue occurs when the muscles are no longer capable of responding as they did prior to the exercise task that induced the fatigue (MacIntosh & Rassier, 2002). Peripheral fatigue is further defined as the changes that occur beyond the neuromuscular junction and are known as "local" factors (Edwards, 1983).

Initial theories of peripheral fatigue suggest that the depletion of adenosine triphosphate (ATP) and phosphocreatine (PCr) may contribute to fatigue.

However, while some studies have observed an association between concentrations of ATP and PCr at fatigue, it is not known if this contributes to fatigue or is simply a consequence of muscle contraction during exercise (Roberts & Smith, 1989).

Fatigue has also been found to coincide with muscle glycogen depletion. As exercise duration increases, the contribution of blood glucose to the total energy output increases, resulting in a reduction in muscle glycogen concentration (Fitts, 1994). The consumption of carbohydrate has been shown to maintain blood glucose concentration and spare endogenous sources, consequently delaying fatigue (Jeukendrup, 2004). However, the mechanism by which muscle glycogen specifically influences fatigue remains to be fully elucidated.

The accumulation of lactate and hydrogen ions ($H+$) during exercise has been suggested to result in a decline in maximal force generating capacity (Tesch, Sjodin, Thorstensson, & Karlsson, 1978). Lactate accumulation has been suggested to inhibit force production due to an increase in $H+$ concentration; occurring as the result of the dissociation of lactic acid into lactate and $H+$ (Roberts & Smith, 1989). Accumulation of $H+$ has been shown to negatively affect force generating capacity via the following:

1) inhibition of phosphofructokinase, which may slow glycolysis (Maclaren, Gibson, Parry-Billings, & Edwards, 1989; Sahlin, 1992),

2) stimulation of pain receptors (Brooks, Fahey, & White, 1996),

3) displacement of calcium from troponin, potentially slowing glycolysis (Brooks *et al.*, 1996; Maclaren *et al.*, 1989),

4) side effects such as nausea and disorientation (Brooks *et al.*, 1996),

5) a reduced release of free fatty acids into the circulation (Brooks *et al.*, 1996),

6) a reduction in cross-bridge attachments (Fitts, 1994),

7) inhibition of ATPase (Fitts, 1994), and

8) an inhibition of the generation of action potentials (Maclaren *et al.*, 1989).

Despite the varied nature of fatigue, future research must investigate the effects of recovery interventions on the reduction of exercise-induced fatigue and the facilitation of the recovery process. This process becomes particularly important when athletes are required to maintain or improve athletic performance during training or competition, multiple times a day and often on consecutive days.

2.3   Assessment strategies for monitoring cycling performance

In the present thesis, various forms of cycle ergometry were selected as the exercise mode chosen to induce fatigue. Therefore, assessment strategies for monitoring cycling performance will be discussed. The use of cycle ergometry has commonly been used to assess responses to exercise (Atkinson, Davison, Jeukendrup, & Passfield, 2003). More often than not, one of three tests is utilised throughout an investigation; either a graded exercise test, an anaerobic test, or a performance test.

Graded exercise tests incorporate a ramp protocol and have traditionally been implemented for laboratory based testing (Faria, Parker, & Faria, 2005b). They are frequently performed to determine lactate thresholds and related sub-maximal and maximal physiological variables (McNaughton, Roberts, & Bentley,

2006). Lactate threshold has been shown to be an important variable related to cycling performance and is often included in the assessment of an endurance athlete (Coyle, 1995; Coyle *et al.,* 1988; McNaughton *et al.,* 2006).

During competitive cycling events, athletes are often required to generate high power outputs for relatively short periods of time (e.g. climbing, sprinting, individual time trial). To assess this ability, anaerobic power tests are often utilized. In a recent review, Faria *et al.* (2005b) identified tests for anaerobic power to generally last 10-30 s with the cyclist remaining seated, generating a cadence of 50-140 rpm. Previous research has proven that repeated bouts of maximal effort and of short duration are fuelled predominately by ATP, derived from PCr degradation (Gaitanos *et al.,* 1993). However, the ability to maintain a high power output is determined by the extent to which homeostasis is restored during intermittent periods of recovery and is thought to be related to the oxygen-dependent recovery kinetics of PCr and inorganic phosphate (Glaister *et al.,* 2006; McLester, 1997; Tomlin & Wenger, 2001; Westerblad, Allen, & Lannergren, 2002).

Performance tests often take the form of a time trial performed on the cyclist's bicycle using a calibrated wind-braked cycle ergometer or on a Lode ergometer. Cyclists are instructed to generate the highest possible power output for a given period of time; often power output (e.g. 70-75% peak power output) is controlled for the initial period of the test, after which the cyclist is free to control both pedal cadence and force (Faria *et al.,* 2005b). Such protocols have also been successfully altered to include a series of sprint performances to mimic the nature of bicycle road races (Faria *et al.,* 2005b; Palmer, Noakes, & Hawley,

1997). In addition to laboratory based ergometry, SRM Training Systems (SRM, Schoberer Rad Meßtechnik, Germany) have frequently been used to calculate power output from torque and angular velocity. This technology involves the use of strain-gauges located between the crank axle and the chain ring. Their deformation is proportional to the torque generated by each pedal revolution (Faria, Parker, & Faria, 2005a). The SRM Training System can be adapted to either laboratory or field settings (Atkinson *et al.*, 2003) and can store all recorded data in its memory (e.g. power output, speed, distance, cadence, and HR).

Muscular fatigue can adversely affect cycling performance. Muscular fatigue of the lower body may also result in an altered cycling motion and therefore, altered muscle activation patterns (Raymond, Joseph, & Gabriel, 2005). The rate of voluntary force development during cycling has been shown to decrease with the presence of muscular fatigue (Bentley *et al.,* 2000; Lepers *et al.,* 2002). In many scientific investigations fatigue is identified by monitoring the rate of decline of power output (Raymond *et al.*, 2005). Although the exact cause of fatigue is often debated, fatigue resulting from repetitive high intensity, short duration cycling tasks may induce an acute decrease in force production via a reduction in the neural input to the muscle and in the efficiency of the contractile mechanism (Paavolainen *et al.,* 1999).

In conclusion, there are numerous ways to assess the affects of fatigue on cycling performance, with the most popular methods being graded exercise tests, anaerobic tests and performance tests (namely time trialling). While the relationship between performance in laboratory-based tests and performance in

the field (competition) has not been adequately investigated, laboratory measures can be reliable, valid, and repeatable when conducted well.

2.4   Delayed Onset Muscle Soreness (DOMS)

Delayed onset muscle soreness (DOMS) is the sensation of discomfort that often occurs within a few days of strenuous, unaccustomed exercise (Crenshaw, Thornell, & Friden, 1994; MacIntyre, Reid, & McKenzie, 1995). Delayed onset muscle soreness has been shown to be particularly prevalent after the performance of high-load lengthening (eccentric) contractions (Cleak & Eston, 1992; Gibala *et al.*, 2000) or high-intensity unaccustomed exercise (Crenshaw *et al.*, 1994). The intensity of physical symptoms usually peaks 48-72 h post-exercise (Clarkson & Sayers, 1999) and then progressively subside over a period of several days (Eston & Peters, 1999). However, functional symptoms, including a prolonged loss of force-generating capacity, can be significant for up to ten days (Clarkson & Sayers, 1999). This loss of muscle function can have significant consequences for athletic performance (Allen, Dumont, & MacIntyre, 2004; Byrne & Eston, 2002).

Cleak and Eston (1992) associated DOMS with muscle shortening, swelling, and a decrease in strength. Further consequences of exercise-induced muscle damage include a dull aching pain, increased muscle stiffness, decreased range of motion, increased metabolic rate, tenderness/soreness, and a prolonged loss of muscle function localised in the affected muscle (Eston & Peters, 1999; Weerapong *et al.*, 2005). The extent of muscle damage has been documented directly through analysis of biopsy samples (Rinard *et al.*, 2000). Muscle damage has also been indirectly examined by measuring losses

in both strength and range of motion as well as monitoring increases in blood levels of muscle proteins such as creatine kinase and myoglobin (Rinard *et al.*, 2000). In addition, perceptions of pain and girth measurements have also been examined.

2.4.1 Aetiology of Muscle Soreness

The aetiology of acute muscle soreness has been attributed to the combination of ischemia and the accumulation of metabolic by-products (Gulick & Kimura, 1996). The mechanisms by which the symptoms of DOMS occur have proven to be more mysterious and are likely to be multi-factorial. Several theories to identify the cause of DOMS have been proposed in the last decade. MacIntyre, Reid, and McKenzie (1995) suggest that initially a mechanical injury occurs, followed by a biochemical injury that may be responsible for changes that occur within the muscle following eccentric exercise. Evidence of cellular infiltrates in the muscle, such as neutrophils and macrophages and inflammatory mediators have also been reported (Figure 2.2) (MacIntyre *et al.*, 1995).
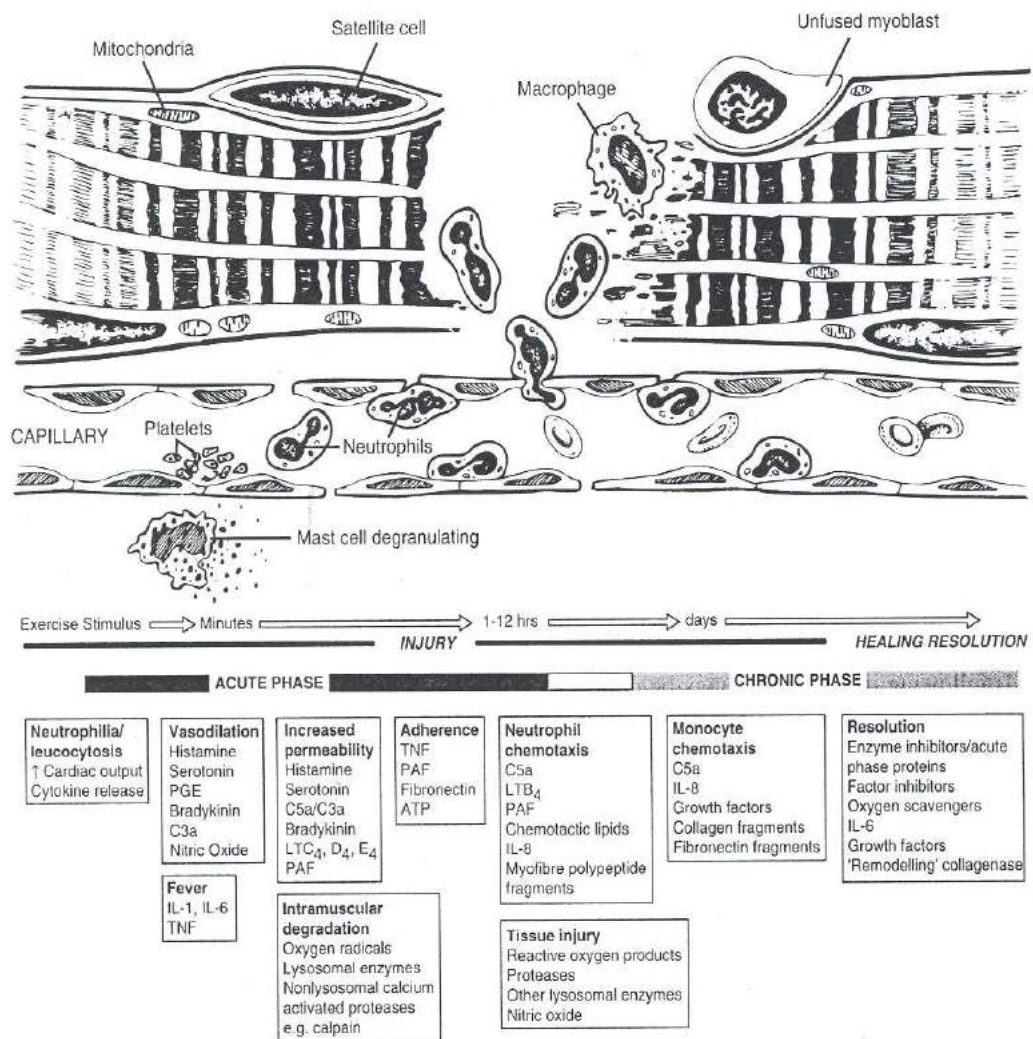
Figure 2.2. Possible sequence of events involving inflammation that occurs following a muscle injury (MacIntyre *et al.,* 1995; Page 27). ATP = adenosine triphosphate; IL = interleukin; LT = leukotriene; PAF = platelet activating factor; PGE = prostaglandin E; TNF = tumour necrosis factor.

In 1996, Gulick and Kimura (1996) identified six theories attempting to explain the cause of DOMS. These were lactic acid accumulation theory, muscle spasm, torn tissue, connective tissue, enzyme efflux, and tissue fluid theories. In addition to these, Clarkson and Sayers (1999) proposed that mechanical strain, disturbance of intracellular calcium homeostasis, and the inflammatory response may be factors responsible for muscular damage following eccentric exercise.

After unaccustomed exercise there is initially a significant disruption of sarcomeres within the muscle, followed by an inflammatory response (Connolly, Sayers, & McHugh, 2003; Stupka *et al.,* 2001), excitation-coupling failure (Balnave & Allen, 1995; Warren, Lowe, & Armstrong, 1999), local swelling (Clarkson & Sayers, 1999), increases in free calcium and sodium and decreases in pH (Yeung *et al.,* 2002). Figure 2.3 outlines the events associated with DOMS as well as interventions designed to target various aspects of the sequence (Connolly *et al.*, 2003). Eccentric exercise results in an injury to the cell membrane, which causes an inflammatory response resulting in the synthesis of prostaglandin and leukotriene (Connolly *et al.*, 2003) as well as the infiltration of neutrophils, neutrophil activation and the release of myocellular enzymes into the plasma (Fielding *et al.*, 2000). The inflammatory response is believed to be the cause of a second reduction in strength approximately two days after the initial damage (Faulkner, Brooks, & Opiteck, 1993; Horita *et al.,* 1996).

Leukotrienes → Neutrophils → Free Radicals

Injury to muscle membrane → Phosphlipase A_2 activation / Arachidonic acid metabolism → PGE_2 → **PAIN**
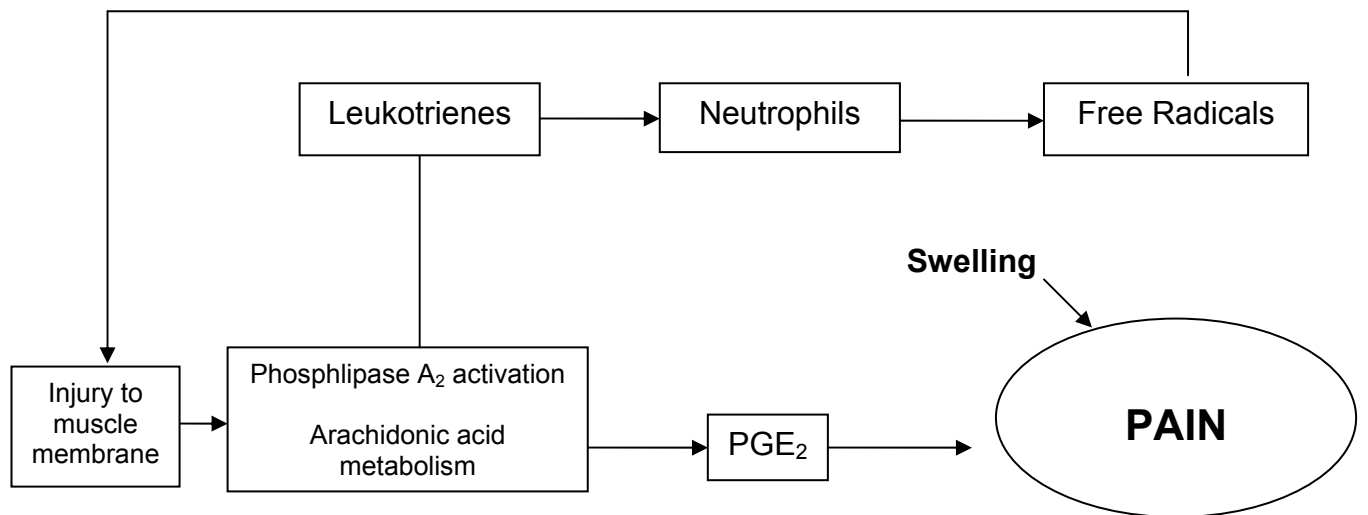
Swelling

I. Mechanical Damage   II. Inflammation and Swelling   III. Free Radical Proliferation

Interventions targeting potential mechanisms of injury

- Stretching
- Massage
- Warm-up
- Hyperbaric oxygen therapy
- Ultrasound
- E-Stimulation
- L-Carnitine
- Rest
- Light Exercise

- Anti-inflammatory medications
- Cryotherapy
- Ice Massage
- Homeopathic remedies
- Acupuncture
- Electromagnetic Shielding

- Vitamin E
- Vitamin C
- Ubiquinones
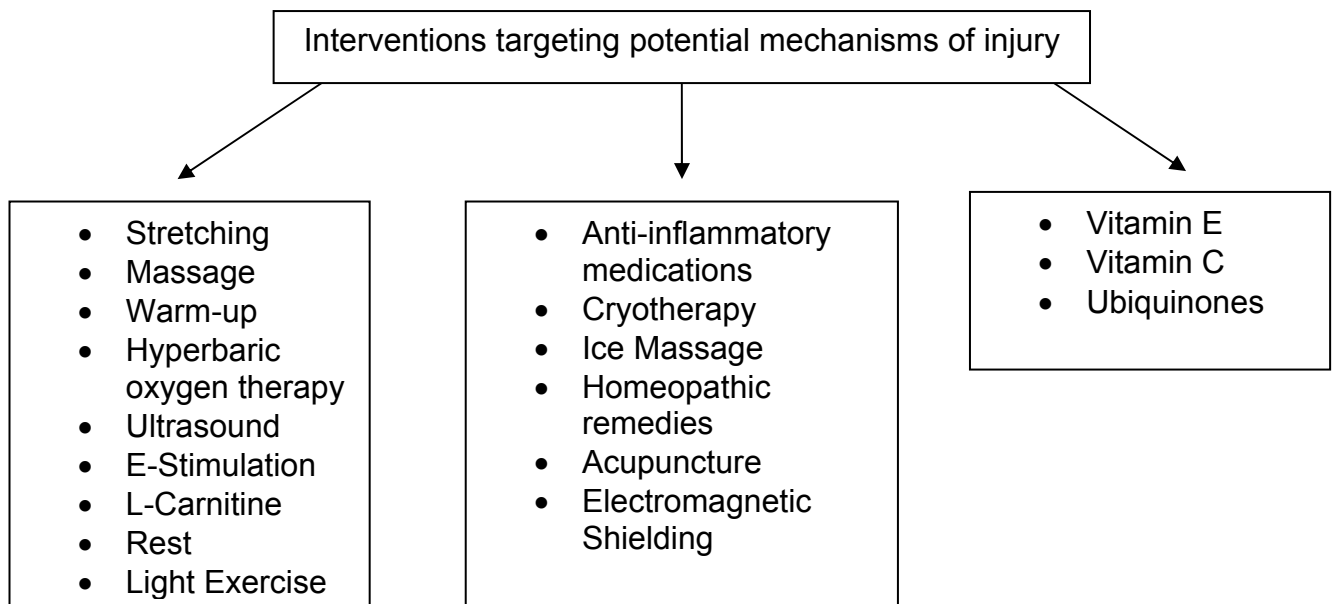
Figure 2.3. Schematic showing possible sequence of injury and treatment of DOMS (Connolly *et al.,* 2003; Page 198).

Connolly *et al.* (2003) identified that prostaglandin release causes a sensation of pain by sensitizing type III and IV pain afferents to the effects of chemical stimuli. Leukotrienes increase the vascular permeability and attract neutrophils to the site of damage. Swelling is the result of movement of cells and fluid from the bloodstream into the interstitial spaces and is also thought to contribute to the sensation of pain (Connolly *et al.*, 2003).

Damage to either the sarcoplasmic reticulum or the muscle membrane can increase intracellular calcium and trigger calcium-sensitive degradative pathways (Clarkson & Sayers, 1999). The sensation of muscle tenderness appears to be initiated by the loss of cellular calcium homeostasis (Clarkson *et al.,* 1986) resulting from the activity-induced disturbance of sarcomeres (Enoka, 1994). Damage to muscle fibres results in an inflammatory response that causes a transfer of fluid and cells to the damaged tissue (Clarkson & Sayers, 1999). Post-injury swelling then occurs as a result of the increased fluid (Clarkson & Sayers, 1999). MacIntyre *et al.* (1995) identified two sub-classifications of inflammation as acute and chronic. The first response of the body to injury is acute inflammation, characterised by a rapid change in blood flow or vascular permeability and the immigration of neutrophils and monocytes (MacAuley, 2001). The typical symptoms of this acute inflammatory reaction include redness, swelling, heat, and pain (MacIntyre *et al.*, 1995). The second response of the body to injury is chronic inflammation, characterised by the presence of lymphocytes and monocytes (MacIntyre *et al.*, 1995). The chronic inflammatory response is usually present 3-4 days after the initial muscle-damaging injury, and if the cause of injury is removed this response usually subsides within three to four weeks (MacIntyre *et al.*, 1995).

Immediately following intense eccentric exercise, individuals will usually experience problems controlling movements, a loss of force, increased tremor, and difficulty fully flexing and extending the affected limb (Jones & Round, 1997). While these experiences are generally not painful, over the next 6-12 h discomfort will begin to develop in the exercised muscles. The major sensation is one of muscle tenderness, a feeling similar to a bruise or sprain (Jones & Round, 1997). When in a state of rest and with no external pressure on the muscle, no discomfort is experienced, however external pressure and stretching can cause intense pain (Jones & Round, 1997). The precise mechanisms of how soreness develops and why there is a delay in the onset of soreness is poorly understood (Cheung, Hume, & Maxwell, 2003; Weerapong *et al.*, 2005). Along with local tenderness, a sensation of stiffness that limits the range of movement of the limb, is also experienced, as there are signs of oedema over the affected muscle (Jones & Round, 1997). There are many physiological and psychological responses to muscle soreness; Figure 2.4 demonstrates some of the delayed responses that occur in reaction to eccentric exercise (Evans & Cannon, 1991).
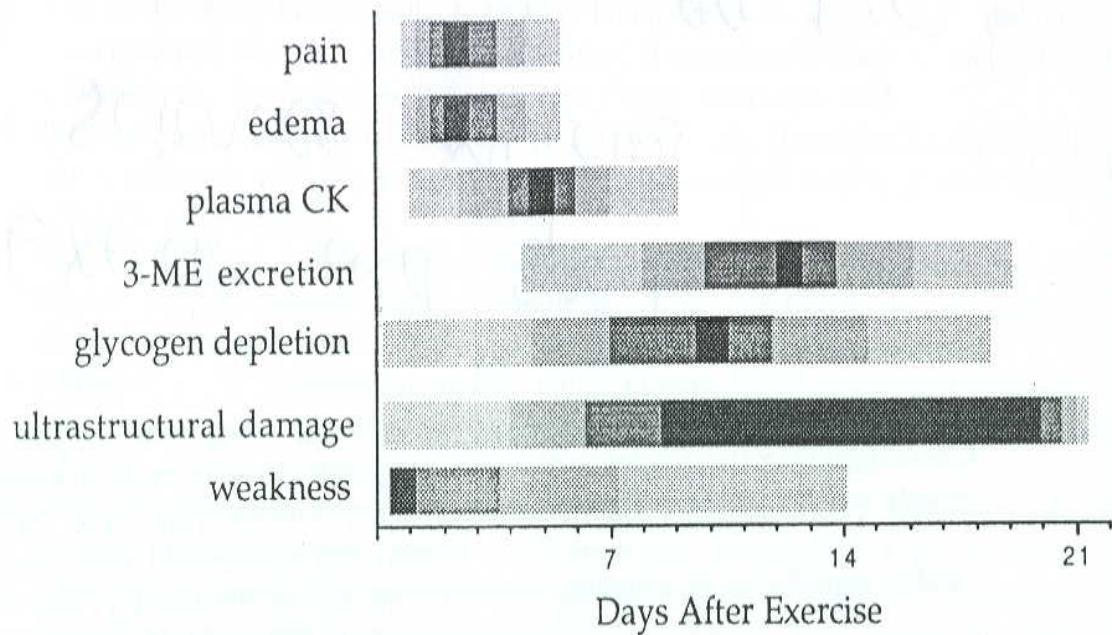
Figure 2.4. Delayed responses to eccentric exercise. Density of shading in each bar corresponds to the intensity of the response at the time indicated on the horizontal axis (Evans & Cannon, 1991; Page 100). Darker shading indicates the maximum intensity of the response while lighter shading indicates a lower intensity of the response.

2.4.2 Protocols

Many different eccentric muscle-damaging protocols have been used in the research of muscle soreness and DOMS (Cleak & Eston, 1992; Harrison *et al.*, 2001; Mair *et al.*, 1995; Sayers *et al.,* 1999). When investigating the effect of intense eccentric exercise on muscle soreness, swelling, stiffness, and strength loss, Cleak and Eston (1992) used a protocol consisting of 70 maximum voluntary contractions of the elbow flexors. Each contraction lasted for 3 s with a 12 s rest period between contractions. Harrison *et al.* (2001) used a protocol consisting of six sets of ten eccentric repetitions at 120% concentric one repetition maximum (1RM). Mair *et al.* (1995) used an eccentric exercise protocol that involved contractions at 150% of the participant's maximal voluntary generated force. Participants performed seven sets of ten eccentric contractions of the quadriceps femoris muscle group, each contraction lasting 1-2 s, with 15 s rest between contractions and 2-3 min rest between sets (Mair *et al.*, 1995). In all of these protocols, DOMS was successfully induced, with decreases in performance, swelling, and exercise related responses observed in various blood markers (Cleak & Eston, 1992; Harrison *et al.*, 2001; Mair *et al.*, 1995; Sayers *et al.*, 1999).

In summary, eccentric protocols consisting of 60-70 maximal contractions lasting 1-3 s with 12-15 s between repetitions and 2-3 min between sets, using maximal or supra-maximal loads of 100-150% maximal voluntary force/1RM have proved to be effective in the production of DOMS.

2.4.3 Adaptation to Eccentric Exercise

It is postulated that a single bout of eccentric exercise may have a prophylactic effect on muscle soreness, blood variables, and performance capabilities following a second bout of eccentric exercise (Brown, 1997; Byrnes & Clarkson, 1986; Mair *et al.*, 1995; Nosaka *et al.,* 2001). This has been referred to as the "repeated bout effect" (Nosaka & Clarkson, 1995). Although multiple theories have been proposed to explain the repeated bout effect, the specific mechanism/s have not yet been identified (Connolly, Reed, & McHugh, 2002). Figure 2.5 illustrates the three mechanisms that have been proposed to explain this phenomenon. These have neural, cellular and mechanical (the connective tissue theory) origins (Connolly *et al.*, 2002; McHugh *et al.*, 1999).

Potential neural adaptations include a change in motor unit recruitment following the initial bout of eccentric exercise that may limit the extent of subsequent damage (Golden & Dudley, 1992; McHugh *et al.*, 1999; Nosaka & Clarkson, 1995) and an increase in the synchrony of motor unit firing which may reduce myofibrillar stresses during a repeated bout (McHugh *et al.*, 1999; Pierrynowski, Tudus, & Plyley, 1987). The decreased motor unit activation associated with eccentric contractions may produce a learning effect and provide more efficient recruitment for a repeated bout (Golden & Dudley, 1992).

Theories of cellular adaptation include strengthening of the cell membrane, removal of weak fibres or sarcomeres after the initial damage and longitudinal addition of sarcomeres (McHugh *et al.*, 1999). An early study found pain and stiffness following an initial bout of eccentric exercise to be attributed to the shortening of contractile connective tissue (Newham, Jones, & Clarkson, 1987).

Adaptation to connective tissue has been proposed as a possible mechanism for decreased sensations of pain and stiffness following a repeated bout of eccentric exercise (McHugh *et al.*, 1999). In addition, there is indirect evidence to support the theory of connective tissue adaptation and the ability to protect against further muscle damage, the protective effect may be attributed to the ability of the connective tissue to disperse myofibrillar stresses (Lapier *et al.*, 1995). Furthermore, following damaging eccentric exercise, tissue repair may be characterised by a similar increase in intramuscular connective tissue, therefore protecting against any subsequent damage caused by repeated bouts (Lapier *et al.*, 1995; McHugh *et al.*, 1999).

The duration of the protective effect of eccentric exercise-induced muscle damage has been found to be variable (Clarkson, Nosaka, & Braun, 1992; Ebbeling & Clarkson, 1989; Nosaka *et al.*, 2001; Prou, 1999). Clarkson *et al.* (1992) found that the length of adaptation differed among measures; when the exercise regime was separated by six weeks, all measures (muscle soreness, muscle strength, range of motion, creatine kinase [CK]) resulted in a reduced response following the second exercise bout compared to the first. Additionally, after 10 weeks only CK and muscle shortening showed a reduction in response and after six months only CK response was reduced (Clarkson *et al.*, 1992). More recently, Nosaka *et al.* (2001) investigated whether indicators of muscle damage were reduced when a second exercise bout was performed six, nine, and 12 months after the first bout of damaging exercise. The results showed that the repeated bout effect for most measures appeared to last at least six months and was lost after nine to 12 months (Nosaka *et al.*, 2001). In contrast, Prou *et al.* (1999) found the first eccentric exercise task had no

prophylactic effect against muscle damage when the same exercise task was performed four weeks later. These results contradict the other findings reporting an adaptive process following a single session of eccentric exercise (Byrnes *et al.*, 1985; Nosaka & Clarkson, 1995).

In conclusion, the exact duration of the adaptive effect following an initial bout of eccentric exercise remains largely unknown, with the findings of the various studies contradicting one another. The phenomenon of such adaptation is important and should be taken into account for future research. Thus, it appears that the use of a cross-over design, ensuring participants are both familiar with and accustomed to resistance training and ensuring a substantial wash-out period between exercise tasks are effective ways of minimising the effect of the first bout of eccentric exercise (Viitasalo *et al.*, 1995). In addition, the implementation of a cross-over design also takes into account the individual variations in response to a given task; therefore, using each participant as their own control is ideal.

```
                    ┌─────────────────────┐
                    │ Initial bout of     │
                    │ eccentric exercise  │
                    └─────────────────────┘
                              │
                              ▼
                    ┌─────────────────────┐
                    │    Muscle Damage    │
                    └─────────────────────┘
                              │
                              ▼
                    ┌─────────────────────┐
                    │     Adaptation      │
                    └─────────────────────┘
```

Neural Theory

Increased motor unit activity

Increased slow-twitch fibre recruitment

Increased motor unit synchronisation

Connective Tissue Theory

Increased intramuscular connective tissue

Intermediated filament remodelling

Cellular Theory

Strengthening of cell membranes

Removal of weak fibres

Longitudinal addition of sarcomeres

```
                    ┌─────────────────────┐
                    │ Repeated bout of    │
                    │ eccentric exercise  │
                    └─────────────────────┘
                              │
                              ▼
                    ┌─────────────────────┐
                    │ Less muscle damage  │
                    └─────────────────────┘
```

Figure 2.5. Potential mechanisms which may explain the repeated bout effect following an initial bout of eccentric exercise (McHugh *et al.,* 1999; Page 168).

2.5 Treatment and Management Strategies

The majority of scientific research investigating recovery interventions has been based on models of DOMS, in which muscle damage is induced and recovery of performance monitored for effectiveness of the intervention. Numerous studies have examined the efficacy of methods to promote recovery from muscle-damaging exercise. Some of these interventions include compression garments (Ali, Caine, & Snow, 2007; Kraemer *et al.*, 2001), active recovery (Sayers, Clarkson, & Lee, 2000), hyperbaric oxygen therapy (Harrison *et al.*, 2001) and ibuprofen administration (Hasson *et al.*, 1993; Tokmakidis *et al.,* 2003). In addition, interventions such as massage (Tiidus & Shoemaker, 1995), stretching (Lund *et al.,* 1998), ultrasound (Plaskett, Tiidus, & Livingston, 1999) and nutritional supplementation (Hellsten *et al.,* 1997; Jakeman & Maxwell, 1993; Kaminski & Boal, 1992; Warren *et al.,* 1992) have been trialled with the aim of alleviating DOMS (Connolly *et al.*, 2003). More recently, post-exercise hydrotherapy interventions have been employed in an attempt to assist and accelerate recovery.

2.6 Hydrotherapy

Despite the widespread incorporation of hydrotherapy into athletes' post-exercise recovery regimes, information regarding these interventions is largely anecdotal. Some of the physiological responses to water immersion are well researched and understood, however, in terms of post-exercise recovery; the underlying mechanisms are poorly understood. The benefits to subsequent performance have not been clearly established. The human body responds to water immersion with changes in cardiac response, peripheral resistance, and changes in blood flow (Wilcock *et al.*, 2006). In addition, both hydrostatic

pressure and temperature of the immersion medium may influence the success of different hydrotherapy recovery interventions (Wilcock *et al.*, 2006).

Immersion of the body in water can result in an inward and upward displacement of fluid from the extremities to the central cavity due to hydrostatic pressure. As identified by Wilcock *et al.* (2006), the resulting displacement of fluid may bring about an increase in the translocation of substrates from the muscle. Therefore, post-exercise oedema may be lessened and muscle function maintained. In addition, another physiological response to water immersion is an increase in stroke volume, which has been shown to result in an increase in cardiac output (see Table 2.2). Peripheral resistance also decreases during head-out water immersion, indicating the presence of peripheral vasodilation (Arborelius *et al.,* 1972; Park, Choi, & Park, 1999; Weston *et al.,* 1987; Wilcock *et al.*, 2006; Yun, Choi, & Park, 2004).

While the effects of hydrostatic pressure exerted on the body during water immersion may be beneficial, the temperature of water the body is exposed to is also thought to influence the success of such recovery interventions. The main physiological effect of immersion in cold water is a reduction in blood flow due to peripheral vasoconstriction (Meeusen & Lievens, 1986). In contrast, immersion in hot water increases blood flow due to peripheral vasodilation (Bonde-Petersen, Schultz-Pedersen, & Dragsted, 1992; Knight & Londeree, 1980).

Table 2.2. Cardiac responses to thermoneutral immersion compared with non-immersion (Wilcock *et al.,* 2006; Page 755).

Study	Immersion Duration	Change in SV (%)	Change in HR (%)	Change in cardiac output (%)
Hip Level Immersion				
Farhi and Linnarsson (1977)	-	11.9 *	-3.9 *	14.0 *
Löllgen *et al.* (1981)	-	37.0 *	-5.7 *	29.2 *
Xiphoid Process Immersion				
Farhi and Linnarsson (1977)	-	64.2 *	-10.5 *	48.0 *
Löllgen *et al.* (1981)	-	67.1 *	-11.4 *	48.1 *
Bonde-Petersen *et al.* (1992)	15	38.7 *	-14.5	19.1 *
Gabrielsen *et al.* (2002)	10	50.8 *	-10.6 *	32.6 *
Gabrielsen *et al.* (2000)	10	-	-14.1 *	-
Watenpaugh *et al.* (2000)	30	-	-18.3 *	-
Weston *et al.* (1987)	15	50.0 *	-11.0 *	31.5 *
Head-Out Immersion				
Arborellius *et al.* (1972)	10	28.3 *	-303	28.9 *
Farhi and Linnarsson (1977)	-	79.1 *	-6.6 *	66.0 *
Löllgen *et al.* (1981)	-	79.5 *	-11.4 *	59.1 *
Gabrielsen *et al.* (2000)	10	-	-15.3 *	-
Johansen *et al.* (1997)	5	-	-6.9	-
	10	-	-8.6 *	-
	15	-	-8.6 *	-
Park *et al.* (1999)	30	54.7 *	-1.4 *	53.2 *
Shiraishi *et al.* (2002)	30	62.1 *	-8.6 *	52.4 *
Sramek *et al.* (2000)	10	-	-8.0 *	-
Yun *et al.* (2004)	20 (a)	52.5 *	-1.7	49.4 *
Yun *et al.* (2004)	20 (b)	56.4 *	-6.3	48.7 *
Yun *et al.* (2004)	20 (c)	95.3 *	-2.3	101.7 *

(* = $p < 0.05$) SV = Stoke Volume, HR = Heart Rate;
Yun *et al.* (2004) (a) subjects = breath-hold divers (mean age 55 y); (b) subjects = housewives (55 y); (c) subjects = housewives (22 y)

2.6.1   Cold Water Immersion

Cryotherapy (normally in the form of an ice-pack) is the most commonly used strategy for the treatment of acute soft tissue sports injuries, due to its ability to reduce the inflammatory response and to alleviate spasm and pain (Eston & Peters, 1999; Meeusen & Lievens, 1986; Merrick *et al.,* 1999).   Multiple physiological responses to various cooling methods have been observed, including a reduction in heart rate and cardiac output, and an increase in arterial blood pressure and peripheral resistance (Sramek *et al.,* 2000; Wilcock *et al.,* 2006).   Other responses include decreases in core and tissue temperature (Enwemeka *et al.,* 2002; Lee *et al.,* 1997; Merrick, Jutte, & Smith, 2003; Yanagisawa *et al.,* 2007), acute inflammation (Yanagisawa *et al.,* 2004), pain (Bailey *et al.,* 2007; Washington, Gibson, & Helme, 2000), and a better maintenance of performance (Burke *et al.,* 2000; Yeargin *et al.,* 2006). Merrick *et al.* (1999) also suggest that cryotherapy is an effective method for decreasing skin/muscle/intra-articular temperatures, inflammation, blood flow, muscle spasm, and pain.

The use of cryotherapy (cold treatment) in the treatment of muscle damage and exercise-induced fatigue has been investigated with varying findings. Eston and Peters (1999) investigated the effects of cold water immersion (of the exercised limb in 15°C for 15 min) on the symptoms of exercise-induced muscle damage following strenuous eccentric exercise. The muscle-damaging exercise consisted of eight sets of five maximal isokinetic contractions (eccentric and concentric) of the elbow flexors of the dominant arm (0.58 rad·s^{-1} and 60 s rest between sets). The measures used to assess the presence of exercise-induced muscle damage included plasma CK concentration, isometric

strength of the elbow flexors, relaxed arm angle, local muscle tenderness, and upper arm circumference. Eston and Peters (1999) found CK activity to be lower and relaxed elbow angle to be greater for the cold water immersion group on days two and three following the eccentric exercise, concluding that the use of cold water immersion may reduce the degree to which the muscle and connective tissue unit becomes shortened after strenuous eccentric exercise.

In a recent study, Bailey et al. (2007) investigated the influence of cold water immersion on indices of muscle damage. Cold water immersion (or passive recovery) was administered immediately following a 90 min intermittent shuttle run protocol; rating of perceived exertion (RPE), muscular performance (maximal voluntary contraction of the knee extensors and flexors) and blood variables were monitored prior to exercise, during recovery, and post-recovery for seven days. The authors concluded that cold water immersion is a highly beneficial recovery intervention, finding a reduction in muscle soreness, a reduced decrement of performance, and a reduction in serum myoglobin concentration one hour post-exercise (Bailey et al., 2007). However, further values across the seven day collection period were not cited and CK response was unchanged regardless of intervention. Lane and Wenger (2004) investigated the effects of active recovery, massage, and cold water immersion on repeated bouts of intermittent cycling separated by 24 h. Cold water immersion had a greater effect compared to passive recovery, active recovery, and massage on recovery between exercise bouts, resulting in enhanced subsequent performance. This is an important investigation as most studies in cold water immersion research have been conducted using muscle damage models or recovery from injury.

Despite these promising results, some studies have found negligible changes when investigating the recovery effects of cold water immersion (Paddon-Jones & Quigley, 1997; Sellwood *et al.,* 2007; Yamane *et al.*, 2006).

In a randomised controlled trial Sellwood *et al.* (2007) investigated the effect of ice-water immersion on DOMS. Following a leg extension exercise task (5 × 10 sets at 120% concentric 1RM) participants performed either 3 × 1 min water exposure separated by one minute in either 5°C or 24°C (control) water. Pain, swelling, muscle function (one-legged hop for distance), maximal isometric strength, and serum CK were recorded at baseline, 24, 48, and 72 h post-damage. The only significant difference observed between the groups was lower pain in the sit-to-stand test at 24 h post-exercise in the ice-water immersion group (Sellwood *et al.*, 2007). In accordance with Yamane *et al.* (2006) only the exercised limb was immersed at a temperature of 5°C. In this study, ice-water immersion was no more beneficial than tepid water immersion in the recovery from DOMS. Paddon-Jones and Quigley (1997) induced damage in both arms (64 eccentric elbow flexions), and then one arm was immersed in 5°C water for 5 × 20 min, with 60 min between immersions, while the other served as a control. No differences were observed between arms during the next six days for isometric and isokinetic torque, soreness, and limb volume (Paddon-Jones & Quigley, 1997). In the aforementioned studies, cold water immersion appeared to be an ineffective treatment, specifically when immersing an isolated limb in 5°C water.

Only one study has investigated the effect of cold water immersion on training adaptation. Yamane *et al.* (2006) investigated the influence of regular post-

exercise cold water immersion following cycling or handgrip exercise. Exercise tasks were completed 3-4 times per week for 4-6 weeks, with cooling protocols consisting of limb immersion in 5°C (leg) or 10°C (arm) water. The control group showed a significant training effect in comparison to the treatment group, with the authors concluding that cooling was ineffective in inducing molecular and humoral adjustments associated with specified training effects (e.g. muscle hypertrophy, increased blood supply, and myofibril regeneration).

Despite these findings, the majority of research supports the notion that cold water immersion is an effective treatment intervention for the reduction of symptoms associated with DOMS (Eston & Peters, 1999), repetitive high intensity exercise (Bailey *et al.*, 2007; Lane & Wenger, 2004), and muscle injury (Brukner & Khan, 1993). Despite indications of there being a positive benefit, little evidence has been reported on the effect of cold water immersion on subsequent performance. A more refined investigation into the individual components of a specific recovery protocol is needed to reveal the effect of varying the duration of exposure, the temperature, and the medium used, whether it be ice, air, or water.

2.6.2 *Hot Water Immersion*

The use of heat as a recovery tool has been recommended to increase the working capacity of athletes (Viitasalo *et al.*, 1995) and assist the rehabilitation of soft tissue injuries and athletic recovery (Brukner & Khan, 1993; Cornelius, Ebrahim, Watson, & Hill, 1992). The majority of hot water immersion protocols are performed in water greater than 37°C, resulting in a rise in muscle and core body temperature (Bonde-Petersen *et al.*, 1992; Weston *et al.*, 1987). The

physiological effects of immersion in hot water remain to be elucidated. One of the main physiological responses associated with exposure to heat is increased peripheral vasodilation, resulting in increased blood flow (Bonde-Petersen *et al.*, 1992; Wilcock *et al.*, 2006).

The effect of hot water immersion on subsequent performance is also poorly understood. Only one study has investigated the effect of hot water immersion on post-exercise recovery. Viitasalo *et al.* (1995) incorporated three 20 min warm (~37°C) underwater water-jet massages into the training week of 14 junior track and field athletes. The results indicated an enhanced maintenance of performance (assessed via plyometric drop jumps and repeated bounding) following the water treatment, indicating a possible reduction in DOMS. However, significantly higher CK and myoglobin concentrations were observed following the water treatment, suggesting either greater damage to the muscle cells or an increased leakage of proteins from the muscle into the blood. Viitasalo *et al.* (1995) concluded that combining underwater water-jet massage with intense strength training increases the release of proteins from the muscle into the blood, while enhancing the maintenance of neuromuscular performance.

However, there is a lack of supporting evidence for these findings and the use of hot water immersion for recovery has received minimal research attention. Despite the hypothesised benefits of this intervention, anecdotal evidence suggests that hot water immersion is not widely prescribed on its own or as a substitute for other recovery interventions. Additionally, speculation surrounds the possible effects, timing of recovery and optimal intervention category (e.g.

following which type or intensity of exercise), for the use of hot water immersion. Finally, there has been minimal focus on acute fatigue and performance.

2.6.3 Contrast Water Therapy

During contrast water therapy participants' alternate between heat exposure and cold exposure by immersion in warm and cold water respectively. It has frequently been used as a recovery intervention in sports medicine (Higgins & Kaminski, 1998) and is now commonly used within the sporting community. Although research investigating contrast water therapy as a recovery intervention for muscle soreness and exercise-induced fatigue is limited, several researchers have proposed possible mechanisms that may support its use. Higgins and Kaminski (1998) suggested that contrast water therapy can reduce oedema through a "pumping action" created by alternating peripheral vasoconstriction and vasodilation. Contrast water therapy may bring about other changes such as increased or decreased tissue temperature, increased or decreased blood flow, changes in blood flow distribution, reduced muscle spasm, hyperaemia of superficial blood vessels, reduced inflammation, and improved range of motion (Myrer, Draper, & Durrant, 1994). Active recovery has traditionally been considered a superior recovery intervention to passive recovery. Contrast water therapy may elicit many of the same benefits of active recovery, and may prove to be more beneficial, given the reduced energy demands required to perform it (Wilcock *et al.*, 2006).

Contrast water therapy has been found to effectively decrease post-exercise lactate levels (Coffey, Leveritt, & Gill, 2004; Hamlin, 2007; Morton, 2006;

Sanders, 1996). After a series of Wingate tests, it was found that blood lactate concentrations recovered at similar rates when using either contrast water therapy or active recovery protocols, and that, after passive rest blood lactate removal was significantly slower (Sanders, 1996). Coffey *et al.* (2004) investigated the effects of three different recovery interventions (active, passive and contrast water therapy) on four-hour repeated treadmill running performance. Contrast water therapy and active recovery reduced blood lactate concentration by similar amounts after high intensity running. In addition, contrast water therapy was associated with a perception of increased recovery. However, performance during the high intensity treadmill running task returned to baseline levels four hours after the initial exercise task regardless of the recovery intervention performed.

In a more recent study investigating the effect of contrast water therapy on the symptoms of DOMS and the recovery of explosive athletic performance, recreational athletes completed a muscle-damaging protocol on two separate occasions in a randomised cross-over design (Vaile, Gill, & Blazevich, 2007). The two exercise sessions differed only in recovery intervention (contrast water therapy or passive recovery/control). Following contrast water therapy, isometric force production was not significantly reduced below baseline levels throughout the 72 h data collection period, with reductions of approximately 4-10% observed. However, following passive recovery, peak strength was significantly reduced from baseline by 14.8 ± 11.4% (Vaile *et al.*, 2007). Strength was also restored more rapidly within the contrast water therapy group. In addition, thigh volume measured immediately following contrast water therapy was significantly less than that following passive recovery,

indicating lower levels of tissue oedema. These results indicate that symptoms of DOMS and restoration of strength are improved following contrast water therapy compared to passive recovery (Vaile *et al.*, 2007). However, Hamlin (2007) found contrast water therapy to have no beneficial effect on performance during repeated sprinting. Twenty rugby players performed two repeated sprint tests separated by one hour; between trials subjects completed either contrast water therapy or active recovery. While substantial decreases in blood lactate concentration and heart rate were observed following contrast water therapy, compared to the first exercise bout, performance in the second exercise bout was decreased regardless of intervention (Hamlin, 2007). Therefore, while contrast water therapy appears to be beneficial in the treatment of DOMS, it may not hasten the recovery of performance following high intensity repeated sprint exercise.

There is anecdotal support for the use of contrast water therapy throughout Australia and the world. Teams such as the Wallabies, Hockeyroos, Australian Swim Team, and Australian Cricket Team are currently using contrast water therapy. It was also utilised at the Athens 2004 Olympic Games in the Australian team recovery centre, and will be again at the Beijing 2008 Olympic and Paralympic Games. However, the physiological mechanisms underlying the reputed benefits remain unclear. Temperatures for contrast water therapy generally range from 10-15°C for cold water and 35-38°C for warm water. It is evident that contrast water therapy is being widely used; however, additional research needs to be conducted to clarify its optimal role and relative efficacy.

2.7   Summary

Although all three of these hydrotherapy interventions are being widely used for recovery from high intensity exercise there are few consistencies in the advice and methodology of such interventions. Future research should investigate the optimal water temperatures, duration of exposure, and the number and timing of rotations completed during the protocol.   In addition, the efficacy of hydrotherapy as a recovery tool for differing types of activity (e.g. strength vs. endurance, single day vs. multiple days) is also needed.   Comparisons between these three popular hydrotherapy interventions (cold water immersion, hot water immersion, and contrast water therapy) are also needed to establish the effectiveness of each.

While hydrostatic pressure is thought to play a role in the success of post-exercise hydrotherapy recovery interventions, there is little experimental support for this contention. At present, it is unclear if the benefit of water immersion is the result of pressure exerted on the body or if water temperature plays a substantial role in enhancing the recovery process.

2.8   Assessment strategies for monitoring DOMS

A wide range of symptoms are associated with DOMS, with a diversity of mechanisms proposed to account for these. The variety of methods used to assess DOMS reflects this complexity. Maximum voluntary contraction and isometric strength assessment, circumference measurement, blood analysis, and the assessment of pain have been widely used in the monitoring of muscle damage and its rate of recovery.

2.8.1 Performance Measures

Exercise-induced muscle damage is often quantified by measuring isometric maximal voluntary contraction (MVC), this being the primary means of determining muscle function following muscle-damaging exercise (Byrne & Eston, 2002; Warren *et al.*, 1999). In a review of human studies, Warren *et al.* (1999) found MVC was assessed in 50% of the reviewed studies, the third most frequently used tool, behind the assessment of soreness/pain and blood levels of myofibril proteins (e.g. CK).

Maximal voluntary contraction appears to be the best measure of muscle function change resulting from eccentric contractions (Warren *et al.*, 1999). Moreover, it appears to be a relatively accurate and reliable measure, suitable for determining muscle function in human studies (Warren *et al.*, 1999).

Although MVC is a common measure in DOMS studies, other factors must be considered when measuring MVC responses. For example, Byrne and Eston (2002) found that following exercise-induced muscle damage, strength loss was independent of the muscle action being performed. However, the limitation of muscle function was attenuated when the stretch-shortening cycle was used (e.g. vertical jump performance). Warren *et al.* (1999) also acknowledge the importance of joint angle in the assessment of MVC torque, as valid comparisons can be made both within and between individuals providing torque measurements are made at the same joint angle.

Another common variable measured in DOMS research that indicates the ability to produce force is the assessment of strength. When investigating the

effect of hyperbaric oxygen therapy on recovery of DOMS, Mekjavic *et al.* (2000) found the isometric strength of the elbow flexors decreased significantly from pre-exercise levels for both the treatment (47.8%) and the control groups (50.8%). Over the 10 day recovery period, there was no difference in the rate of recovery of muscle strength between the two groups, with isometric strength recovering to 62% and 61% of pre-exercise levels for the hyperbaric oxygen therapy and control groups respectively (Mekjavic *et al.,* 2000). Cleak and Eston (1992) assessed isometric strength of the elbow flexors through three 5 s maximal contractions separated by 30 s rest. A reduction in strength was found immediately following the eccentric exercise protocol, with maximum strength loss occurring 24 h later (46% of pre-exercise values). Isometric strength also remained 20% lower 11 days after exercise (Cleak & Eston, 1992).

In eight moderately active participants, Byrne and Eston (2002) found consistent reductions in strength have been observed over a four day period following an eccentric protocol involving the knee extensor muscles (Byrne & Eston, 2002). Reductions in strength were approximately 20% (one hour post-exercise), 25% (day one), 21% (two days), 15% (three days), 13% (four days), and 5% (seven days) lower than baseline measures (Byrne & Eston, 2002). Eston and Peters (1999) found similar isometric strength decrements of 23% (cryotherapy group) and 27% (control group) below baseline values. However, by 72 h post-exercise, isometric strength had increased by 10% from baseline for the cryotherapy group, whereas isometric strength for the control group was 14% lower than the baseline values. Additionally, the decline in squat jump performance was significantly higher compared to that in countermovement jump performance (91.6 $\pm$ 1.1% compared to 95.2 $\pm$ 1.3% of pre-exercise

levels); the overall relative decline in jump squat performance was also significantly higher than that in drop jump performance (91.6 ± 1.1% compared to 95.2 ± 1.4%) (Byrne & Eston, 2002).

Electrical stimulation applied during MVCs has demonstrated that motor unit activation is similar at times when muscles are pain-free (pre-exercise) and when they are experiencing DOMS (post-exercise) (Byrne & Eston, 2002; Gibala *et al.,* 1995; Newham *et al.*, 1987; Saxton & Donnelly, 1996). These results suggest that individuals are able to activate painful muscles fully during isometric MVCs following exercise-induced muscle damage (Byrne & Eston, 2002).

Research has clearly demonstrated the effect muscle damage has on performance, with maximal isometric force, MVC, and jump performance all showing significant decreases following muscle-damaging exercise, before gradually returning to pre-exercise values (Behm *et al.,* 2001; Byrne & Eston, 2002; Eston & Peters, 1999). In particular, isometric strength has been reported to decrease by 24-50% immediately post-exercise and decrease further by 24 h (19-46%) and 48 h post-exercise (9-19%) (Behm *et al.*, 2001; Cleak & Eston, 1992; Eston & Peters, 1999; Mekjavic *et al.*, 2000; Sayers *et al.*, 2000). Recovery of isometric strength is more varied by 72 h after exercise, with increases of 10% or decreases of 14% from baseline reported (Behm *et al.*, 2001; Cleak & Eston, 1992; Eston & Peters, 1999; Mekjavic *et al.*, 2000; Sayers *et al.*, 2000).

2.8.2 Circumference

Exercise often results in hyperemia-induced swelling of the muscle/s (Chleboun *et al.,* 1998). Under normal conditions this swelling usually subsides relatively quickly after the cessation of exercise. However, following muscle damage, swelling tends to have a delayed onset and duration of several days (Chleboun *et al.,* 1998). Circumference or girth measurements of the exercised limb have often been used to assess expansion or swelling (Brown, 1997; Chen & Hsieh, 2000; Chleboun *et al.,* 1998; Eston & Peters, 1999). Circumference is usually measured around the midpoint of the limb using an anthropometric tape (Eston & Peters, 1999; Nosaka & Clarkson, 1995). After eccentric exercise, an enlargement of the muscle has been documented by an increase in circumference (Clarkson *et al.,* 1992; Howell *et al.,* 1985; Nosaka & Clarkson, 1995). Cleak and Eston (1992) found the circumference at the musculotendinous distal junction of the arm to be greatest by day four, then to have subsided by 10 days post-exercise; at its greatest, the mean difference was 1.0 cm for the mid-belly and 1.8 cm (SD not reported by authors). Using circumference measurements, Mekjavic *et al.* (2000) found peak swelling following a muscle-damaging protocol occurred between three to five days after exercise. Similar results were reported by Eston and Peters (1999) who found that swelling increased from baseline measurements (26.7 $\pm$ 2.3 cm cryotherapy group; 29.8 $\pm$ 2.2 cm control group) on days two (27.3 $\pm$ 2.1 cm cryotherapy group *vs.* 30.4 $\pm$ 2.4 cm control group) and three (27.1 $\pm$ 2.2 cm cryotherapy group *vs.* 30.4 $\pm$ 2.7 cm control group) post-exercise.

Circumference measurements have provided information regarding changes in the muscle (e.g. enlargement) due to inflammation associated with muscle-

damaging exercise. Following muscle-damaging exercise, circumference has tended to peak between two and five days after exercise, then to subside to normal by 10 days post-activity (Cleak & Eston, 1992; Eston & Peters, 1999; Mekjavic *et al.*, 2000).

2.8.3 Range of Motion

Range of motion (ROM) has been defined as the arc over which a joint may operate and is determined by the mechanical properties of the skin, subcutaneous tissue, tendon, articular capsule, bone and muscle (Warren *et al.*, 1999). Muscle injury induced by eccentric exercise may cause an increase in muscle stiffness, defined as an increased resistance of the resting muscle to passive lengthening (Chleboun *et al.*, 1998; Howell, Chleboun, & Conatser, 1993). Chleboun *et al.* (1998) assessed the stiffness of the elbow flexors on 11 untrained female college students following eccentric exercise by measuring the slope of the passive torque-angle curve. Stiffness increased immediately post-exercise (59.9 ± 14.1%) and remained at or above this level for five days, before decreasing to pre-exercise levels over a period of seven to 11 days (Chleboun *et al.*, 1998).

The intensity of the fatiguing eccentric exercise appears to affect the degree of ROM lost following muscle damage. A study investigating the difference in the magnitude of muscle damage between maximal (100%) and sub-maximal (50%) eccentric loading found a significantly greater decrease in ROM (relaxed elbow joint angle) immediately following the maximal effort (14.3º) than that of the 50% effort (6.7º) (Nosaka & Newton, 2002). Furthermore, the recovery time was significantly shorter after 50% effort than after maximal effort. Flexed

elbow joint ROM was also measured, with similar responses found between groups (50% *vs.* maximal) immediately post-exercise (approximately 10º for both exercise groups). However, a further decrease in the ROM was found 48 h (-26.4 ± 4.9º) following the maximal exercise task, recovering to -14.6 ± 3.6º of pre-exercise levels after five days. In contrast, the ROM following 50% maximal exercise had begun to recover one day after exercise and was close to pre-exercise values after five days (3.6º ± 0.7º) (Nosaka & Newton, 2002).

Muscle stiffness and ROM have been reported to change (i.e. increased stiffness or decreased ROM) immediately post-exercise and for as long as 11 days post-exercise. However, these are not common measures of DOMS as Warren *et al.* (1999) reported ROM to be measured in only 19% of reviewed human studies, with only one study measuring full ROM.

2.8.4 Blood Variables

Many blood variables have been assessed in an attempt to monitor and quantify muscle damage and subsequent recovery from damage. For the purposes of this review, changes in the levels of creatine kinase (CK), myoglobin (Mb) and interleukin-6 (IL-6) will be reviewed in detail, as these have been most frequently reported. The enzyme lactate dehydrogenase (LDH) is also released into the bloodstream following muscle damage (Avela, Kyrolainen, & Komi, 1999; Knitter *et al.,* 2000) but its measurement in the scientific literature is less common. Increased LDH concentration following high intensity eccentric exercise has been suggested to reflect muscle damage (Brown, Day & Donnelly, 1999). However, there is much conflicting evidence regarding the time course and magnitude of change in LDH activity following

muscle damaging exercise (Athanasios *et al.,* 2005; Chen & Hsieh, 2001; Childs *et al.,* 2001). Therefore, CK, Mb and IL-6 responses as markers of muscle damage and post-exercise recovery will be concentrated upon in this section.

Creatine kinase concentration in the blood has been used extensively to assess muscle damage. Attention has focused on CK responses during exercise, mainly due to the relationship between CK response and damage (Viru & Viru, 2001). Most researchers agree that after muscle damage occurs, CK moves from the muscle cell into the interstitial fluid prior to entering the circulation via the lymphatic system (Hortobagyi & Denahan, 1989). Immediately following unaccustomed eccentric exercise, injury of the skeletal muscle fibers is evident from the disruption of the normal myofilament structures in some sarcomeres (Viru & Viru, 2001). Disruption of the muscle cell membrane is thought to be associated with the release of CK from the muscle tissue to the blood, indicating that muscle enzymes have leaked out of damaged muscle cells (Hortobagyi & Denahan, 1989). Therefore, muscle CK levels would be expected to decrease and blood CK levels to increase in response to exercise. Several studies have confirmed that this relationship occurs during DOMS, particularly following eccentric exercise (Byrnes *et al.*, 1985; Hyatt & Clarkson, 1998; Viru & Viru, 2001).

Numerous factors are considered to influence the dynamics of CK release, for example, the type of contraction performed can significantly influence CK response. Byrnes and Clarkson (1986) compared CK levels following eccentric, isometric and concentric exercise, reporting increased levels with a

longer time delay in the eccentric group. The quantity of CK released may also be related to the overall tension of the muscle(s) involved (Clarkson *et al.,* 1985). In addition, individual differences in numerous parameters, such as activity pattern, physical training status, body surface area, diurnal variations and core temperature response can all influence the release of CK (Hortobagyi & Denahan, 1989). These variations should be considered when interpreting changes in CK concentration over time and between studies as they are in other enzyme and hormone sampling procedures.

Eston and Peters (1999) investigated the effects of cold water immersion on the symptoms of exercise-induced muscle damage. Plasma CK activity was measured from a fingertip blood sample immediately before exercise and daily for three days post-exercise. There were no differences between the pre-test and the 24 h post-exercise CK activity levels in either the treatment or the control group. However, by 48 and 72 h post-exercise, CK activity in the control group was significantly higher, but in the treatment group had not changed significantly with time (Eston & Peters, 1999).

In a study investigating the effect of hyperbaric oxygen therapy as a treatment for muscle injury, Harrison *et al.* (2001) used a protocol consisting of six sets of ten eccentric repetitions at 120% concentric one repetition maximum (1RM), finding that serum CK increased significantly for all groups (P=0.0007). However, there was no significant difference found between groups (P=0.943) (Harrison *et al.*, 2001). When investigating the effect of compression sleeves of the treatment for DOMS, Kraemer *et al.* (2001) induced muscle-damage using a protocol consisting of two sets of 50 passive arm curls on a isokinetic

dynamometer (maximal eccentric muscle action superimposed every fourth passive repetition). Although CK levels were significantly elevated from baseline values in both the compression sleeve group and the control group ($P<0.05$), the control group showed a more dramatic increase in serum CK concentration at 72 h (~1350 U/L compared to ~480 U/L in the compression sleeve group). Byrne and Eston (2002) found significant elevations in CK activity one hour after exercise (barbell squats, 10 sets of 10 repetitions at 70% body mass) as well as one, two and three days post-exercise. Activity levels peaked one day after exercise, with values approximately 580% higher than pre-exercise levels. Furthermore, when investigating the effect of ibuprofen on DOMS and muscular performance, Tokmakidis *et al.* (2003) found that CK levels were significantly higher 48 h post-exercise (6 sets of 10 eccentric repetitions at 100% concentric 1RM) in the placebo group and that the ibuprofen treated group produced peak levels at 24 h (rather than 48 h).

In summary, CK levels change during DOMS with dramatic increases observed between 24 and 72 h post-exercise, followed by a gradual decline to baseline (pre-exercise) values. The measurement of CK concentration has been widely used in the assessment of muscle damage. The magnitude of change in CK concentration may be primarily affected by the type of contraction performed (Byrnes & Clarkson, 1986) and the tension output of the muscle(s) involved (Clarkson *et al.*, 1985).

Myoglobin (Mb) is another muscle protein frequently used as an indirect marker of damage in studies investigating exercise-induced muscle damage (Peake *et al.*, 2005; Pizza *et al.,* 1999; Pizza *et al.*, 1996; Pizza *et al.*, 1995; Sayers &

Clarkson, 2003). The release of Mb from the muscle may occur as a result of increased permeability of the myocellular membrane and/or increased permeability of the intramuscular vasculature (Cannon *et al.,* 1990; Peake *et al.,* 2005). The time course of the appearance of Mb in the blood differs from that of CK, possibly due to the differing routes of delivery into the circulation (Sayers & Clarkson, 2003). Myoglobin is a smaller protein than CK, allowing it a more direct route into the microvascular endothelium. Therefore, it appears in the blood at a faster rate than larger protein molecules (Mair, 1999; Sayers & Clarkson, 2003).

Following downhill running, Peake *et al.* (2005) observed an immediate post-exercise increase in plasma Mb concentration (1100%: ES = 4.6; P<0.01), and one hour later was even greater than baseline values (1800%: ES = 4.5; P<0.01). At 24 h post-exercise, the values had decreased, although they remained 100% higher than the baseline (ES = 1.9; P<0.01) (Peake *et al.,* 2005). Comparable responses in plasma Mb concentrations were observed immediately after a similar exercise protocol (30 min downhill running) with values returning to baseline levels 24 h post-exercise (Feasson *et al.,* 2002). Sayers and Clarkson (2003) investigated the effect of immobilization following an eccentric exercise protocol (eccentric contractions of the elbow flexors) to assess whether or not such an intervention decreased lymphatic transport, thereby blunting the post-exercise Mb response. Myoglobin levels were found to increase 24 h post-exercise and peak between 72 - 96 h after exercise, regardless of the intervention (Sayers & Clarkson, 2003).

In conclusion, Mb levels have frequently been utilized as an indirect marker of muscle damage. The majority of studies have observed myoglobin to have a faster release into the blood than CK due to being a smaller molecule.

Exercise-induced muscle damage has also been associated with acute inflammation. As a result, cytokines are released at the site of inflammation (Olschewski & Bruck, 1988; Peake *et al.*, 2005; Smith *et al.*, 2000). Cytokine production can be affected by a range of variables, including strenuous exercise and stress hormones, which can be characterised as pro- or anti-inflammatory. At the onset of inflammation there is an up-regulation of pro-inflammatory cytokines including Interleukin-6 (IL-6) (Cavaillon, 1994; Dinarello, 1997; Smith *et al.*, 2000), making this an important cytokine in the acute phase response (Ostrowski, Schjerling, & Pedersen, 2000). While IL-6 has traditionally been considered to be a pro-inflammatory cytokine, more recently it has been suggested to have an inflammation-controlling role, important for the return of homeostasis following inflammation (Ostrowski *et al.*, 2000; Tilg, Dinarello, & Mier, 1997; Xing *et al.*, 1998). Interleukin-6 also possesses some anti-inflammatory properties, being shown to inhibit the synthesis of Interleukin-1 (IL-1) and tumour necrosis factor-α (TNF-α), to suppress the production of macrophages, to protect against lung damage during pulmonary inflammation, and to increase inhibitors of matrix metalloproteases (Curfs, Meis, & Hoogkamp-Korstanje, 1997).

Peake *et al.* (2005) investigated the effect of exercise intensity and muscle damage on plasma cytokine changes. Their findings suggest that high-intensity running at 60% $\dot{V}O_{2max}$ for 60 min created a greater inflammatory response

than downhill running (-10% treadmill gradient at an intensity of 60% $\dot{V}O_{2max}$ for 45 min). In agreement with these findings, Suzuki *et al.* (2000) observed significant increases in IL-6 concentration immediately following endurance exercise (marathon distance race), with the post-exercise IL-6 concentrations showing a 100-fold increase from baseline values. Smith *et al.* (2000) found IL-6 to be significantly elevated at 12, 24, and 72 h following an eccentric bench press and leg curl exercise task.

In response to trauma and strenuous exercise, IL-6 appears to be the most consistently elevated cytokine; perhaps because it is produced early in the inflammatory phase, soon after IL-1 and TNF-α (Cannon, 2000). Interleukin-6 levels have been shown to peak at the end of a strenuous exercise bout , or within a few hours, before declining to baseline levels (Pedersen *et al.*, 1999). However, depending on the exercise task and protocol implemented, IL-6 has been shown to peak at varying times following exercise. Pedersen *et al.* (1999) suggest IL-6 values peak 0-2 h post-exercise with a gradual return to baseline levels, however, Smith *et al.* (2000) observed elevated levels of IL-6 at 12, 24, and 72 h post-exercise.

2.8.5 Perceptual Measures

Perceptual rating of soreness was the most commonly used method for the assessment of injury, being used in 73% of reviewed human studies; 12% of these used an objective means of assessing soreness while 63% made a subjective evaluation using either a visual analogue or numerical scale (Warren *et al.*, 1999).

Various methods have been used to assess the perception of intensity and duration of muscle soreness. For example, Cleak and Eston (1992) used a visual analogue scale (VAS) to measure soreness. The VAS consists of ten numerically rated descriptions of pain (Cleak & Eston, 1992). When assessing muscle soreness after intense eccentric exercise, a significant increase (P<0.01) in perceived soreness in the experimental arm 24 h after exercise was reported, with muscle soreness peaking after three days and subsiding by day eight (Cleak & Eston, 1992). Using the VAS, Mair *et al.* (1995) found that subjects' perception of soreness of the quadriceps muscles peaked at 24-48 h after eccentric exercise (rating 7.5 and 8.0 respectively) followed by a decline on subsequent days (rating 3.5 after four days). Harrison *et al.* (2001) found a significant increase in soreness after an eccentric exercise task, with perceived soreness values peaking two days post-exercise (5.7 and 7.0 on days one and two post-exercise) and returning to baseline 15 days post-exercise (6.3, 4.8, 1.5 and 1.0 for days 3, 4, 7, and 15 respectively). When investigating the effect of ibuprofen on DOMS, Tokmakidis *et al.* (2003) found a significant increase in muscle soreness at 24 and 48 h post-exercise, but was significantly lower for the ibuprofen group in comparison to the control group after 24 h (3.8 $\pm$ 1.3 compared with 5.5 $\pm$ 1.4) and 48 h (5.0 $\pm$ 1.6 compared with 5.9 $\pm$ 1.6).

The VAS is a simple tool to administer and is straightforward for participants to complete. Despite the abundance of such scales, it appears that soreness may have a poor correlation with changes in the magnitude and time-course of muscle function and post-exercise blood responses (Warren *et al.*, 1999). Nonetheless, soreness regularly occurs following the onset of contractile decrements within the muscle (Warren *et al.*, 1999).

Information gained from the assessment of individual responses to pain and soreness is essential to enable valuable information regarding the time course of soreness to be monitored. This is particularly important when assessing the effectiveness of an intervention. The most common method for assessing perception of pain is through participant completion of a pain assessment questionnaire. Pain assessment questionnaires are ideal in the sense that they take minimal time to administer and provide an insight into the psychological effects and time course of soreness.

2.8.6 Summary

There are various methods available for assessing an individual's response to DOMS. Circumference measurements provide an objective assessment of swelling and oedema changes within the thigh, while the collection of blood samples can be used to monitor various blood markers following exercise and muscle damage. Isometric strength measurements are commonly used in the assessment of muscle damage following exercise, while pain assessment questionnaires allow individuals to specify current perceptions on a numerical scale ranging from no pain to extreme pain. Perceptual responses to pain and soreness can be further investigated through the use of localized pressure where persons are required to indicate when the sensation of pressure becomes uncomfortable or painful. All of these assessments of pain and soreness allow valuable insight into the pattern and time course of muscle damage as well as essential information regarding the effectiveness of various recovery interventions.

2.9   Significance/influence on athletic performance

The use of recovery interventions, in particular, the use of hydrotherapy techniques is a topical issue. However, there is insufficient evidence to allow firm conclusions on their effectiveness and little quality research has been conducted in this field. Thus, athletes and trainers have been unable to make informed decisions about which recovery intervention might be most appropriate for use. While various hydrotherapy techniques are commonly used as post-exercise recovery interventions, there seems to be a trend of "following others" rather than reasoned recovery selections being made. Further research must be performed to enable coaches and athletes alike to base their choice of recovery intervention on scientific evidence.

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CHAPTER THREE

Paper One

Effect of cold water immersion on repeat cycling performance and thermoregulation in the heat.

Abstract

To assess the effect of cold water immersion (CWI) and active recovery (ACT) on thermoregulation and repeat cycling performance in the heat, ten well-trained male cyclists completed five trials, each separated by one week. Each trial consisted of a 30 min exercise task (E1), one of five 15 min recoveries (intermittent CWI in 10°C, 15°C and 20°C water, continuous CWI in 20°C water and ACT), followed by 40 min passive recovery, before repeating the 30 min exercise task (E2). Recovery strategy effectiveness was assessed via changes in total work in E2 compared to E1. Following ACT a 4.1 ± 1.8% decrease in total work ($P<0.001$) was completed in E2 when compared to E1. However, no significant differences in total work (E2 vs. E1) were found between any of the CWI protocols. Core and skin temperature, blood lactate, heart rate, rating of thermal sensation, and rate of perceived exertion were recorded. During E1 and E2 there were no significant differences in lactate concentration between interventions, however, post ACT lactate concentration was significantly lower ($P<0.05$; 2.0 ± 0.8 mmol.L^{-1}) compared to all CWI protocols. All CWI protocols were effective in reducing thermal strain and were more effective in maintaining subsequent high intensity cycling performance in comparison to ACT.

Introduction

Cryotherapy is a commonly used post-exercise recovery strategy in a variety of sports and is thought to be effective when core temperature is significantly increased (Hadad, Rav-Acha, Heled, Epstein, & Moran, 2004) or for the treatment of inflammation, spasm and pain (Eston & Peters, 1999; Meeusen & Lievens, 1986; Merrick *et al.*, 1999). While various forms of cryotherapy, including cold water immersion (CWI) have been suggested to be effective treatments to decrease metabolism, inflammation, blood flow, pain, and skin, muscle and intra-articular temperatures, as well as increase tissue stiffness (Merrick *et al.*, 1999), the specific effects of CWI on the recovery profile and subsequent performance of athletes has not been studied thoroughly. Cold water immersion has been used to treat cases of hyperthermia and heat stroke as it creates a thermal gradient between the skin and the environment which is 25 times that of air (Hadad *et al.*, 2004). Despite a lack of scientific research and understanding about its effects, performing CWI as a recovery strategy following high intensity exercise has become increasingly popular.

In addition to the use of CWI as a post-exercise recovery strategy, it has also been investigated as a cooling intervention prior to physical activity (precooling). Intense exercise in hot environmental conditions can raise core temperature by up to one degree every five to seven minutes of exercise (Kay *et al.*, 1999). When core body temperature exceeds 39°C the ability to maintain maximal muscle activation may become impaired and eventually result in the premature termination of exercise (Gonzalez-Alonso *et al.*, 1999; Marino, 2002; Nielsen *et al.*, 1993). Additionally, similar muscle and core temperatures have been observed at the point of fatigue suggesting that fatigue primarily responds to

signals initiating in the active muscles and internal organs as well as the central nervous system (Gonzalez-Alonso *et al.*, 1999). Whole body precooling is thought to enhance the safe temperature margin between the operating temperature and the critical limiting temperature (Marino, 2002), and therefore may enhance athletic performance in hot environments.

Active recovery (ACT) is anecdotally reported to be one of the most commonly performed post-exercise recovery strategies; therefore, active recovery serves as an ideal control. While ACT has been shown to enhance the removal of lactate (Bonen & Belcastro, 1976; Gupta, Goswami, Sadhukhan, & Mathur, 1996; Hayashi *et al.*, 2004; Taoutaou *et al.*, 1996), the effect of ACT on subsequent performance remains inconclusive, with some studies suggesting ACT can result in the maintenance of performance (Bogdanis, Nevill, Lakomy, Graham, & Louis, 1996; Monedero & Donne, 2000; Signorile, Ingalls, & Tremblay, 1993; Thiriet *et al.*, 1993), while others suggest subsequent performance is not maintained or enhanced by ACT (Watson & Hanley, 1986; Weltman & Regan, 1983). The conflicting findings may be attributed to differences in methodologies, exercise protocols/modalities, and markers of recovery. Whole body CWI performed between exercise bouts may enhance recovery, however, depending on timing, may also provide a precooling stimulus for the next exercise performance.

To our knowledge, the effect of whole body CWI on repeat cycling performance and thermoregulation in the heat has not been researched. Furthermore, the effect of various water temperatures and durations of exposure have not been investigated. CWI appears to be an effective recovery strategy for reducing

symptoms associated with muscle soreness (Eston & Peters, 1999) and fatigue (Lane & Wenger, 2004) as well as an effective method of precooling prior to exercise (Kay *et al.,* 1999; Lee & Haymes, 1995; Marsh & Sleivert, 1999). Therefore, it seems appropriate to investigate the effects of various CWI protocols on physiological responses to exercise in the heat and cycling performance repeated within a short duration of time. However, it is important to ensure a comparison of the CWI interventions with the commonly implemented practise of ACT. While the effect of cooling provides a greater allowance for heat storage during exercise as well as reducing both cardiovascular and thermoregulatory strain (Kay *et al.*, 1999), in a hot environment ACT may have the opposite effect. Therefore, the purpose of the present study was to investigate the effects of CWI and ACT on repeated cycling performance and thermoregulation in a hot environment.

Methods

Subjects

Ten well-trained male cyclists volunteered to participate in this study. Their mean ± standard deviation (*s)* age, height, body mass, $\dot{V}O_{2peak}$ and sum of seven skinfolds were 32 ± 5 years, 181.0 ± 4.7 cm, 71.6 ± 5.9 kg, 70.7 ± 7.9 $ml.kg^{-1}.min^{-1}$ and 53.6 ± 18.6 mm, respectively. Subjects were informed of any risks and provided written informed consent. The study was approved by the Australian Institute of Sport Research Ethics Committee.

Experimental design

Initially, subjects completed an incremental cycling test on a cycle ergometer (Lode, Groningen, Netherlands) in order to establish each individual's peak

power output (PPO) and $\dot{V}O_{2peak}$. In addition, as subjects were not heat acclimatised, each individual completed two familiarisation trials prior to the commencement of testing. The subjects had access to a fan at all times throughout the study, with self-selected fan settings maintained at those selected in each subject's familiarisation session. The identical cycle exercise tasks (E1 and E2) consisted of a five minute warm-up period (60 sec at each of the following intensities: 125 W, 150 W, 175 W, 200 W, 75% PPO), 15 min at a workload equal to 75% PPO followed immediately by a 15 min time-trial (Jeukendrup, Saris, Brouns, & Kester, 1996). Subjects had access to time information and were required to produce as much work as possible in that timeframe, but no other information or encouragement was provided. Immediately following E1 a standardised cool down was completed (five minutes at 40% $\dot{V}O_{2peak}$) (McAinch *et al.*, 2004) followed by one of five 15 min recovery strategies and 40 min of passive recovery. Passive recovery consisted of the subject remaining seated in a temperature-controlled chamber in an attempt to replicate real life exposure in athletic settings. One hour after the cessation of the initial exercise task (E1) (including five minute cool down, 15 min recovery strategy and 40 min passive rest) subjects were required to repeat the initial 30 min exercise task (E2) (Figure 1). In a randomised crossover design subjects completed a total of five trials, each separated by one week. The typical error of measurement for total work completed was relatively consistent throughout treatments (0.9-1.3%). All testing sessions were conducted in a temperature-controlled chamber in which ambient temperature and relative humidity were maintained at (mean ± *s*) 34.0 ± 0.2°C and 39.4 ± 1.5%, respectively. During all five trials, a carbohydrate beverage (Gatorade; 6% carbohydrate content) was supplied at 3 ml.kg^{-1} of body mass,

and consumed during the first 15 min of the exercise task (E1 and E2) as well as 15 ml.kg^{-1}, consumed throughout the one hour recovery period between exercise bouts. Subjects performed each exercise trial at the same time of day, additionally, body mass was recorded prior to each trial to ensure body mass was stable throughout the duration of the study.

| 5min warm up | 15min@75% PPO 15min time trial (E1) in heat | 5min warm down | **15min Recovery Strategy** 1) Active 2) 10°C CWI 3) 15°C CWI 4) 20°C CWI 5) 20°C+ CWI | 40min passive recovery in heat | 5min warm up | 15min@75% PPO 15min time trial (E2) in heat |
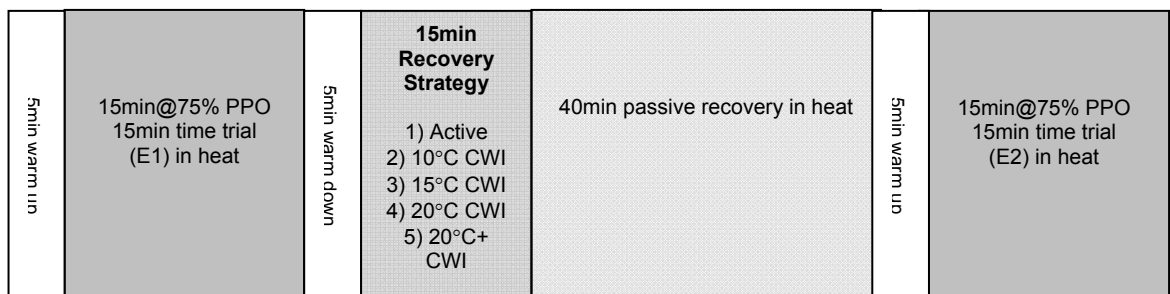
Figure 1. Events of a single testing session, including a five min warm-up, 30 min exercise task (E1) (15 min fixed intensity at 75% PPO followed by a 15 min time trial), five min warm-down, one of five 15 min recovery strategies followed by 40 min passive recovery seated in a temperature-controlled chamber before repeating the exercise task (E2).

Recovery Strategies

Immediately post-exercise, subjects performed five minutes of cycling at an intensity of 40% $\dot{V}$ O$_{2peak}$ (McAinch *et al.*, 2004) followed by one of five recovery strategies:

1) Subjects immersed their entire body (excluding the neck and head) while seated in *10°C* water in an inflatable bath for one minute, followed by two minutes out of the bath, repeated five times (five cycles = 15 min). For all CWI protocols, mean ± *s* air temperature and relative humidity was 29.2 ± 1.4°C and 58.0 ± 2.1% respectively.

2) Subjects immersed their entire body (excluding the neck and head) while seated in *15°C* water in an inflatable bath for one minute, followed by two minutes out of the bath, repeated five times (five cycles = 15 min).

3) Subjects immersed their entire body (excluding the neck and head) while seated in *20°C* water in an inflatable bath for one minute, followed by two minutes out of the bath, repeated five times (five cycles = 15 min).

4) Subjects immersed their entire body (excluding the neck and head) while seated in *20°C* water in an inflatable bath for 15 min (continuous exposure).

5) Subjects cycled continuously at 40% $\dot{V}$ O$_{2peak}$ (McAinch *et al.*, 2004) for 15 min (active recovery) (air temperature of 31.1 ± 2.6°C and 48.0 ± 4.2% relative humidity).

Water temperature was maintained through the addition of ice and continuously monitored using a thermometer.

Performance Assessment – Total Work

The effectiveness of each recovery strategy in maintaining or improving total work during the two 15 min time trials occurred by comparing the total work

measured during E2 and E1. Recovery and performance following the CWI and ACT recovery strategies was also assessed through the measurement of lactate concentration, ratings of perceived exertion, and ratings of perceived thermal comfort.

Mean Body Temperature ($\overline{T}_b$)

Core temperature was monitored with a disposable rectal probe (Monatherm, Mallinckrodt Medical, St Louis, MO, USA) inserted at least 12 cm beyond the anal sphincter prior to testing (O'Brien *et al.*, 2000; Zhang & Tokura, 1999). Skin temperatures were monitored through the use of skin thermistors (Grant Instruments Ltd, Cambridgeshire) attached to the left side of the body at four sites (chest, forearm, quadriceps and calf) using adhesive tape. Rectal and skin temperatures were recorded every five minutes throughout the testing session (exercise and recovery) from an Eight-Channel Digital Thermometer (Zentemp 5000, Zencor Pty Ltd, Victoria, Australia). Rectal (T_{core}) and skin temperatures ($\overline{T}_{sk}$) were then used to calculate mean skin temperature according to the equation established by Ramanathan (Ramanathan, 1964). Mean body temperature ($\overline{T}_b$) was also calculated by methods described by Schmidt and Bruck (1981).

$$\overline{T}_{sk} = 0.3 \times (T_{Chest} + T_{Forearm}) + 0.2 \times (T_{Thigh} + T_{Calf})$$

Equation 1. Equation for the calculation of Mean Skin Temperature ($\overline{T}_{sk}$)

(Ramanathan, 1964)

$$\overline{T}_b = 0.87\, T_{core} + 0.13\, \overline{T}_{sk}$$

Equation 2. Equation for the calculation of the Mean Body Temperature ($\overline{T}_b$) (Schmidt & Bruck, 1981).

The typical error of measurement for skin temperature was 0.13°C (0.45% TEM), repeat tests of core temperature had an intra-class correlation of 0.86, with a typical error of 0.11°C (0.30% TEM).

Blood Lactate Concentration

Blood lactate concentration was measured via a capillary earlobe sample and analysed with a Lactate-Pro (Shiga, Japan). During both E1 and E2, blood lactate was measured immediately pre-exercise, at the end of the 15 min at a fixed load for E1 and E2 (75% PPO), and at the end of each of the 30 min exercise tasks. In addition, blood lactate was analysed immediately following the 15 min recovery period. Typical error of measurement for blood lactate was 0.1 mmol.L^{-1} (<5 mmol.L^{-1}) and 0.4 mmol.L^{-1} (5-10 mmol.L^{-1}).

Rating of Perceived Exertion (RPE)

Subjects rated their perceived exertion on a scale of six (no exertion at all) to 20 (maximal exertion) (Noble, Borg, Jacobs, Ceci, & Kaiser, 1983) every five minutes throughout the fixed intensity phase of the exercise task. Subjects were familiarised with the RPE scale during pre-testing.

Thermal Sensation Scale

Subjects rated their perceived thermal comfort on a scale of zero (unbearably cold) to eight (unbearably hot) (Young, Sawka, Epstein, Decristofano, & Pandolf, 1987) every five minutes throughout the entire testing session.

Heart Rate (HR)

A Polar heart rate monitor (Polar Electro Oy, Finland) was fitted to the subject for the duration of the testing session. Heart rate was recorded every five minutes throughout both E1 and E2, as well as during the one hour recovery period between the exercise tasks.

Statistical Analysis

Data are reported as mean ± s unless otherwise stated. A repeated measures analysis of variance (ANOVA) was used and post-hoc pairwise comparisons conducted to ascertain any significant changes (P <0.05) between selected change scores or means. Percentage change was calculated via log transformation to allow the assessment of changes relative to individual responses rather than absolute values; additionally, log transformation applied more uniformity to all subjects than raw units. In addition, 95% confidence intervals (CI) (defining the likely range of the true value in the population from which the sample was drawn) for mean scores and differences between means were also calculated and presented where appropriate. Cohen's effect sizes using pre-test s to standardise effects were also calculated to describe any trends in the data. Statistical analyses were conducted using SPSS computer software (Version 12.0, SPSS Inc, Illinois, USA).

Results

Performance

When ACT was performed between the two exercise bouts a 4.1 ± 1.8% decrease (P <0.001) in total work (kJ) was recorded in the second exercise (E2) bout when compared to the first (E1) (Figure 2). Absolute values of total work (log transformed kJ) completed are presented in Table 1. However, all CWI protocols resulted in the maintenance of performance in comparison to ACT, as they achieved significantly lower percentage differences in work completed from E1 to E2 (P<0.05). There were no significant differences (P>0.05) found among the temperature or temporal variations of CWI, as all four CWI protocols produced statistically similar improvements over ACT (Figure 2).

Mean Body Temperature ($\overline{T}_b$)

From the completion of E1 and the end of recovery there was a significant difference in $\overline{T}_b$ of 2.6-3.9°C (95% CI) between ACT and intermittent CWI in 10°C water (Figure 3). Additionally, there was a significant difference in $\overline{T}_b$ of 2.2-3.2°C (15°C; 95% CI), 1.6-1.6°C (20°C; 95% CI) and 1.9-1.9°C (20°C+; 95% CI) respectively in the other CWI conditions. Between E1 and E2 there was a difference in $\overline{T}_b$ of 0.9-1.4°C (10°C; 95% CI), 0.7-1.6 (15°C; 95% CI), 0.5-1.1 (20°C; 95%CI), and 0.6-1.3 (20°C+; 95% CI) between the CWI and ACT treatments. Additionally, the thermal effect of each recovery intervention was demonstrated immediately post recovery with $\overline{T}_b$ of 34.6 ± 0.6°C (10°C CWI), 35.3 ± 0.6°C (15°C CWI), 36.5 ± 0.5°C (20°C CWI), 36.1 ± 0.2°C (20°C+ continuous CWI), and 38.2 ± 0.4°C (ACT). Therefore, a significant reduction in $\overline{T}_b$ was observed immediately post all CWI recovery interventions and all CWI protocols resulted in a significant $\overline{T}_b$ reduction compared to ACT.
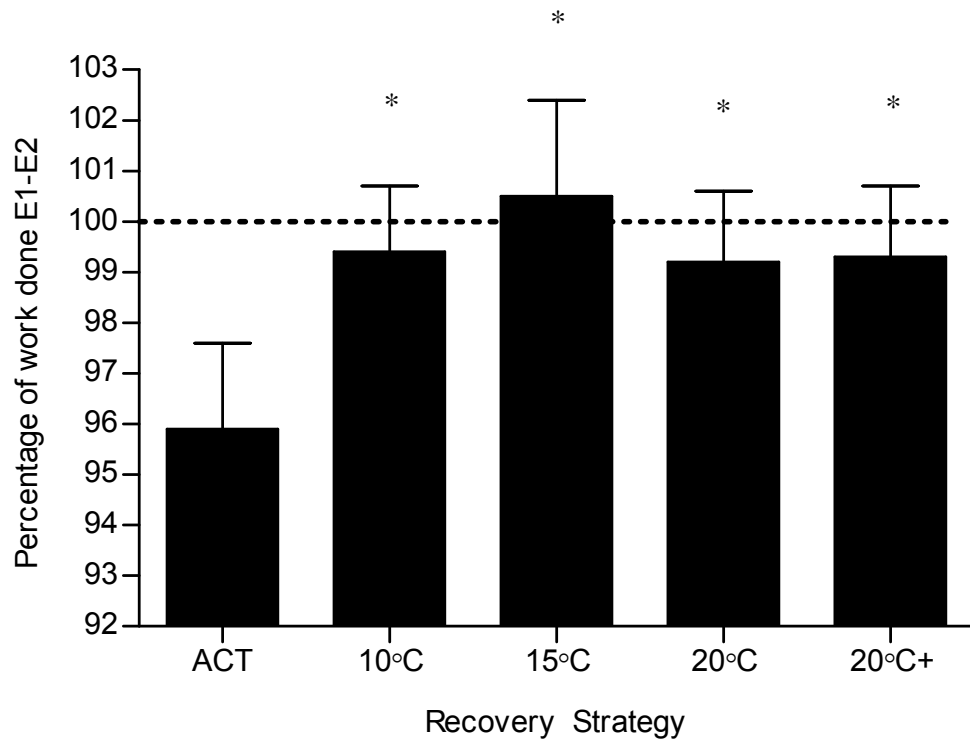
Figure 2. Work done (mean ± *s*) in the second exercise bout (E2) relative to the first (E1) as a percentage. Dashed line indicates E1=E2. ACT = Active recovery; *10°C, 15°C, 20°C* = temperature of cold water in intermittent CWI recoveries; *20°C+* = continuous CWI recovery in water of this temperature.

* Indicates a significant maintenance/improvement in performance compared to ACT (P <0.05).

Table 1. Log transformed absolute values of total work (kJ) completed during the first 30 min exercise task (E1) and the subsequent 30 min exercise task (E2) performed one hour after E1.

Recovery Condition	E1	E2
Intermittent CWI in 10°C	498 ± 48	495 ± 46
Intermittent CWI in 15°C	498 ± 47	500 ± 46
Intermittent CWI in 20°C	500 ± 44	495 ± 47
Continuous CWI in 20°C	502 ± 47	499 ± 48
Active Recovery	503 ± 42	481 ± 38
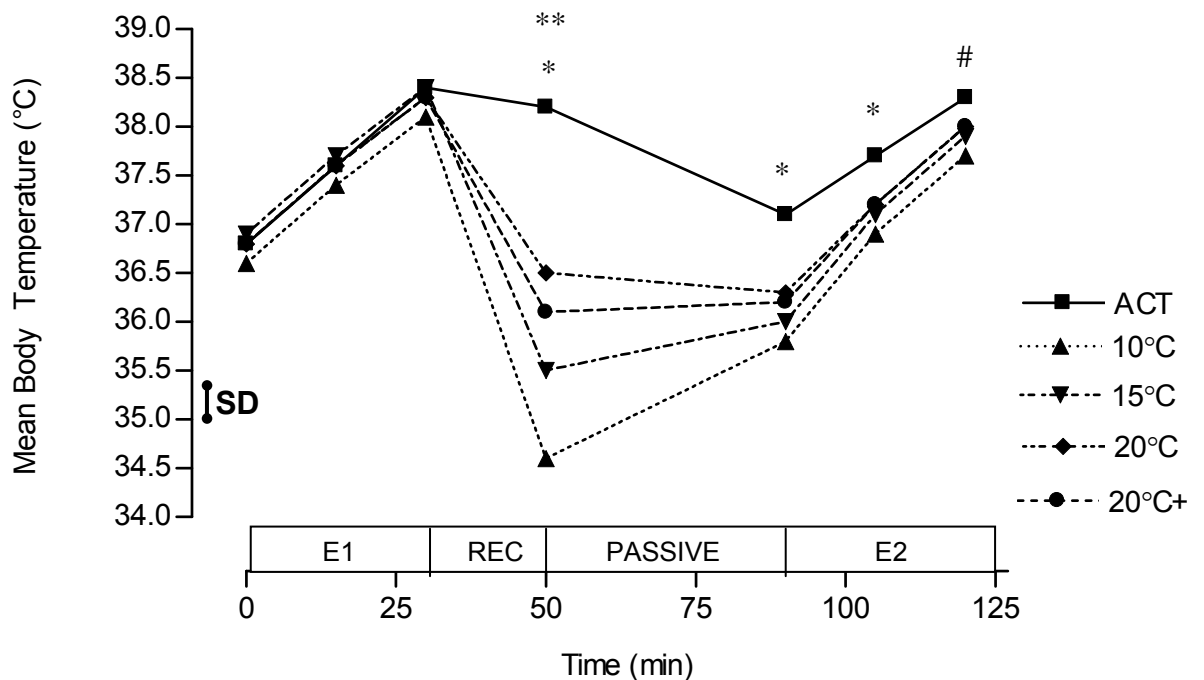
Figure 3. Changes in mean body temperature (°C) (mean) during E1, five min active cool down followed by a 15 min recovery strategy, 40 min passive rest, and E2. ACT = Active recovery; *10°C, 15°C, 20°C* = temperature of cold water in intermittent CWI recoveries; *20°C* + = continuous CWI recovery in water of this temperature.

** Indicates a significant difference ($P<0.01$) between ACT and all four CWI interventions.

\# Indicates a significant difference ($P<0.05$) between ACT vs. 10°C, 15°C and 20°C+ CWI recovery interventions.

* Indicates a significant difference ($P<0.05$) between all four CWI recovery interventions

Blood Lactate Concentration

There were no significant differences between recovery treatments during E1 or E2, however, immediately post ACT blood lactate concentration was significantly lower ($P<0.05$) than that observed immediately post all CWI interventions (Figure 4).

Rating of Perceived Exertion (RPE)

Rating of perceived exertion at the mid-point of exercise during E1 and E2 was significantly lower following intermittent CWI in 10°C ($P<0.05$; 2.4-5.7 95% CI) and 15°C ($P<0.05$; 0.3-1.4 95% CI) water as well as continuous CWI in 20°C water (20°C+) ($P<0.05$; 0.6-2.2 95% CI) when compared to ACT. However, intermittent CWI in 20°C water did not result in a reduced perception of effort when compared to ACT ($P>0.05$; 0.1-1.5 95% CI). When RPE was compared at the end point of both exercise bouts none of the CWI interventions significantly reduced ($P>0.05$) perceived exertion when compared to ACT (10°C -0.3-0.8; 15°C -0.4-1.0; 20°C 0.2-1.0; 20°C+ -0.9-0.5).
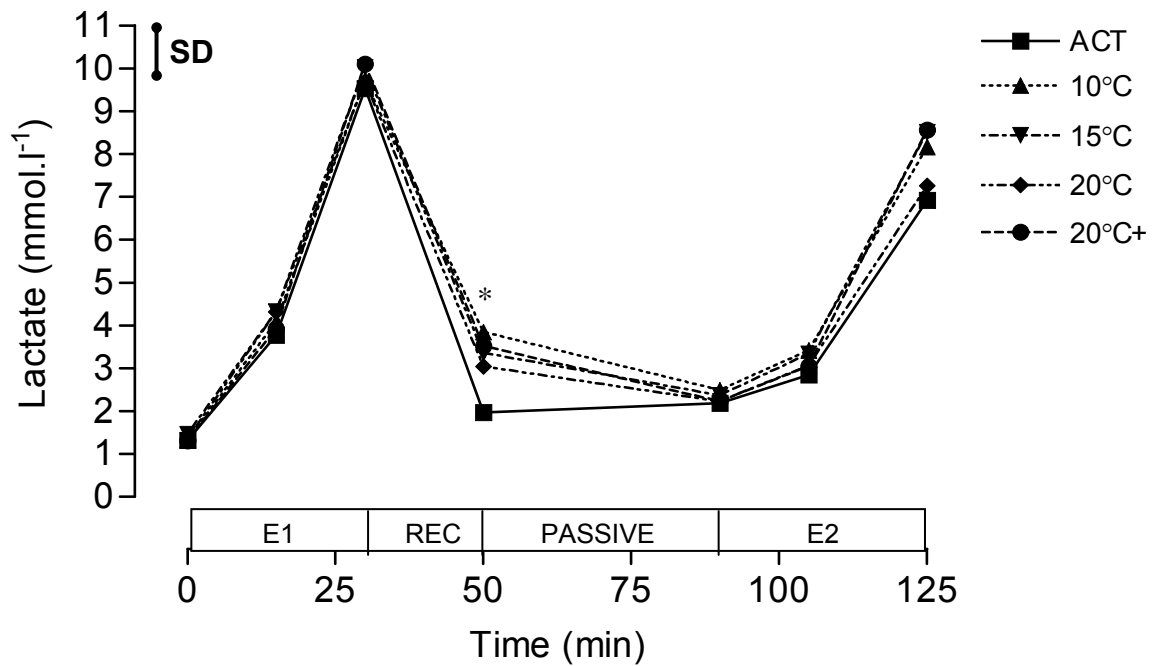
Figure 4. Changes in mean ± *s* blood lactate concentration (mmol.L^{-1}) during E1, five min active cool down followed by a 15 min recovery strategy, 40 min passive rest, and E2. ACT = Active recovery; *10°C, 15°C, 20°C* = temperature of cold water in intermittent CWI recoveries; *20°C+* = continuous CWI recovery in water of this temperature.

* Indicates a significant difference (*P*<0.05) between ACT and all four CWI interventions.

Thermal Sensation Scale

Following ACT, subjects rating of perceived thermal comfort immediately post-recovery, pre-E2, mid-E2, and end-E2 time points were significantly higher than those following all CWI protocols (Figure 5). Further, immediately post-recovery thermal comfort was rated significantly lower in 10°C versus 15°C and 20°C respectively, as well as for 20°C+ versus 20°C. In addition, immediately pre-E2 (95 min) thermal comfort ratings were also significantly lower for 10°C versus 15°C, 20°C and 20°C+ respectively, as well as for 15°C versus 20°C+.

Heart Rate (HR)

During both exercise bouts (E1 and E2), there were no significant differences ($P>0.05$) in heart rate (HR) response between any of the recovery interventions. However, not-surprisingly, immediately post-ACT HR was significantly higher ($P<0.001$; 128 ± 7 bpm) than all CWI interventions (86 ± 12 bpm, 10°C; 80 ± 7 bpm, 15°C; 81 ± 12 bpm, 20°C; 81 ± 9 bpm, 20°C+). Interestingly, this significantly reduced HR following CWI compared to ACT (87 ± 11 bpm) was still evident after 40 min passive rest in the heat (10°C 74 ± 13 bpm; 15°C 69 ± 8 bpm; 20°C+ 71 ± 8 bpm), with the exception of intermittent 20°C CWI (80 ± 6 bpm).
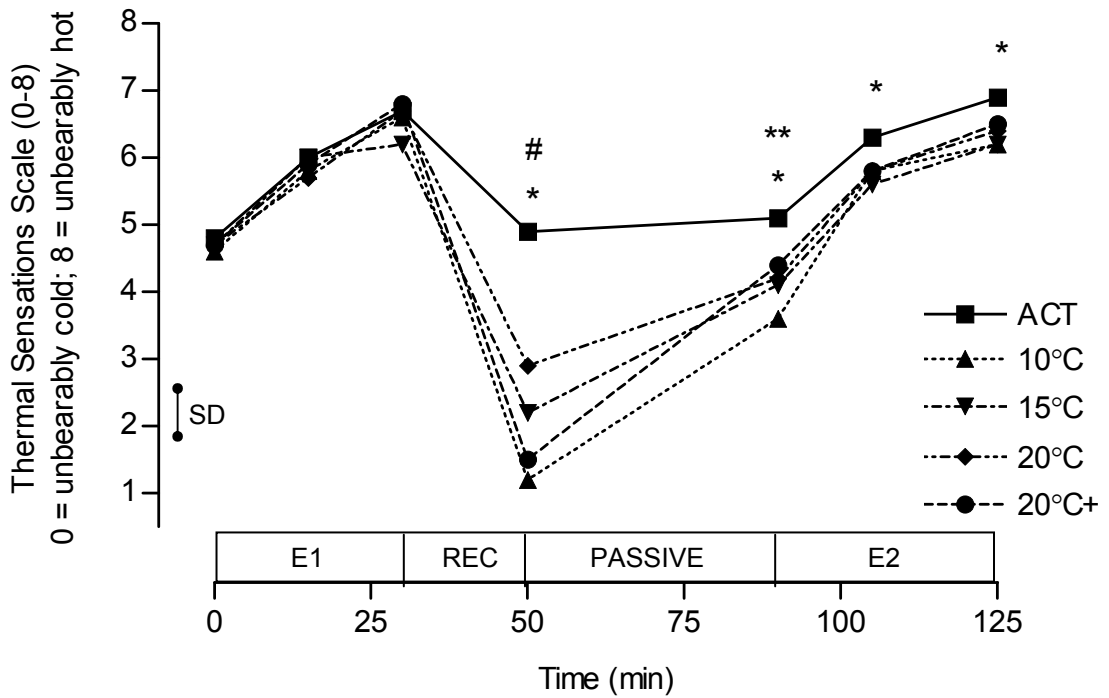
Figure 5. Changes in mean ± *s* perceived thermal comfort during E1, five min active cool down followed by a 15 min recovery strategy, 40 min passive rest, and E2. ACT = (Active recovery); *10°C, 15°C, 20°C* = temperature of cold water in intermittent CWI recoveries; *20°C+* = continuous CWI recovery in water of this temperature.

* Indicates a significant difference (*P*<0.05) between ACT and all four CWI interventions.

# indicates a significant difference between (*P*<0.05) CWI protocols 10°C vs. 15°C, 10°C vs. 20°C, and 20°C vs. 20°C+.

** Indicates a significant difference between (*P*<0.01) CWI protocols 10°C vs. 15°C, 10°C vs. 20°C, 10°C vs. 20°C +, and 15°C vs. 20°C.

Discussion

The main finding of the present study was that all CWI protocols were effective in reducing thermal strain and were more effective in maintaining subsequent high intensity cycling performance in comparison to ACT. Indeed, no significant differences in total work (E2 vs. E1) were found between any of the CWI protocols, and during E1 and E2 there were no significant differences in lactate concentration between interventions.

The use of CWI as a post-exercise recovery intervention has become increasingly popular and is emerging as an effective post-exercise method of both cooling and enhancing recovery (Eston & Peters, 1999; Lane & Wenger, 2004; Merrick *et al.*, 1999; Yanagisawa *et al.*, 2004). Previously, CWI has been used as a precooling method prior to exercise in an attempt to improve performance in hot and humid environmental conditions. Various studies have shown CWI (Eston & Peters, 1999; Merrick *et al.*, 1999) and precooling (Hayashi *et al.*, 2004; Kay *et al.*, 1999; Marsh & Sleivert, 1999) to be effective, providing positive results for recovery and/or subsequent performance.

While the effect of precooling has been investigated, we are not aware of any studies that have investigated the effect of such an intervention on subsequent exercise bouts. The present study used the CWI intervention as a post-exercise recovery (post-cooling), rather than a pre-exercise (precooling) strategy. The results of this study suggest that the use of CWI of varying temperatures and exposures assisted in an enhanced ability to maintain performance when compared to ACT. Other studies have observed similar findings, reporting various precooling strategies to similarly enhance

performance (Armada-da-Silva *et al.*, 2004; Lee & Haymes, 1995; Marsh & Sleivert, 1999). Lee and Haymes (Lee & Haymes, 1995) found a significantly (*P*<0.01) longer average exercise duration (at 82% $\dot{V}O_{2max}$) following precooling compared to control. Their precooling protocol consisted of a 30 min exposure to 5°C air (hypothermic) as opposed to 24°C air (thermocomfortable); in addition to a prolonged exercise time, heat storage during the exercise bout was greater (*P*<0.01) following the hypothermic exposure. The authors concluded that precooling resulted in an increased exercise endurance capability, enhanced heat storage capacity, and less strain on both metabolic and cardiovascular systems (Lee & Haymes, 1995). Similarly, Marsh and Sleivert (1999) found precooling to be effective on a single bout of short term high intensity cycle performance, observing significant increases of 3.3 ± 2.7% for a performance test following precooling compared to no precooling. In addition, the results of the present study are also in agreement with findings by Hessemer et al. (Hessemer *et al.,* 1984) who observed a 6.8% increase in mean work rate during a one hour exercise period compared to a control. Therefore, the current findings of an increased ability to produce work following a post-cooling intervention compared to a control are in agreement with previous studies in this area. The findings of this study support CWI as an effective recovery intervention resulting in a maintenance of subsequent performance significantly greater than that observed following ACT. It is important to note that this study implemented CWI as a post-exercise recovery strategy (post-cooling) as opposed to a targeted pre-exercise precooling intervention. Therefore, while the aforementioned studies are important in understanding the possible mechanism behind our observed maintenance of performance following CWI, the two cannot be directly compared.

A consistent finding within this study was that there were significant reductions in $\overline{T}_b$ following all CWI protocols (intermittent CWI in 10°C, 15°C and 20°C water, and continuous CWI in 20°C water), suggesting changes in blood distribution occurred, likely to be from the peripheral circulation to the central circulation (Marsh & Sleivert, 1999). Indeed, it has been suggested that a critical limiting temperature results in the termination or decline of exercise performance and this is thought to occur due to a reduced efferent command to the skeletal muscle via the central nervous system (Marino, 2004; Nielsen *et al.*, 1990; Nybo & Nielsen, 2001). In addition, a reduction in core temperature appears to provide a superior capacity for heat storage, which may ordinarily be limited by exercise intensity, body size, metabolic heat production and also environmental conditions (Marino, 2002). However, recent findings (Marino, Lambert, & Noakes, 2004; Tatterson, Hahn, Martin, & Febbraio, 2000) suggest, alternatively, that there may be an anticipatory response that occurs during exercise allowing individuals to ensure the maintenance of homeostasis (Marino, 2004) and that it is this anticipatory response that prevents the attainment of lethal hyperthermia as opposed to the attainment of a critically high core temperature. Whether or not the effect of lowering core temperature via cooling affects the intensity of pacing due to core temperature at the onset of exercise being reduced and therefore enabling an enhancement and/or maintenance of performance can only be speculated upon at this time. The results of the present study indicate all CWI protocols effectively enhanced the maintenance of repeat performance when compared to ACT; suggesting that the reduction in core temperature observed prior to the second exercise bout was beneficial, supporting the notion of an anticipatory regulatory response to exercise in the heat.

A decreased heart rate following precooling strategies has been observed (Hayashi *et al.*, 2004; Marsh & Sleivert, 1999; Olschewski & Bruck, 1988; Wilson *et al.*, 2002) and the results of the present study support such findings. In the present study, heart rate was significantly reduced during 40 min of passive rest in the heat following all CWI protocols compared to ACT of the same duration. No significant differences were observed during the second exercise bout; however, it is important to note that more work was completed following all CWI strategies which may have masked any such effect. Marsh and Sleivert (1999) suggest cooling interventions may result in a decrease in peripheral blood flow, causing an increase in central blood volume and therefore, enhance blood delivery to the working muscles. This increase in central blood flow may be beneficial for subsequent performance and therefore may have played a role in the subjects' ability to maintain performance more successfully following CWI compared to ACT. The hydrostatic pressure applied to the body during immersion in water may not only improve the return of fluid from muscle to blood but also increase blood volume causing an increased stroke volume and cardiac output resulting in an increase of blood flow throughout the body (Wilcock *et al.*, 2006). Therefore, hydrostatic pressure appears to influence a number of physiological responses that may improve recovery from sustained high intensity exercise. Marsh and Sleivert (1999) also identify that an increase in central blood volume may provide greater blood availability to the muscle during exercise. In addition, it may increase the clearance of metabolic by-products from the muscle. However, the results of this study do not support this contention, with no significant differences observed in blood lactate concentration between any of the recovery interventions during exercise.

The present study demonstrated a significant reduction in perceived exertion (RPE) during the mid-point of the second exercise task (E2) following intermittent CWI in 10°C and 15°C water as well as continuous CWI in 20°C water (20°C+). Not surprisingly, no significant differences were found in RPE between interventions at the end of E2 as individuals were near exhaustion at this time point and all subjects were required to complete as much work as possible in the 15 min time trial in each of the exercise bouts. A reduced endurance capability during exercise in the heat has been associated with a higher rating of perceived exertion when compared to similar exercise performance in thermocomfortable conditions (Galloway & Maughan, 1997). Amada-da-Silva et al. (2004) found a significant increase in RPE at the end of a 14 min cycling exercise following passive heating compared to control. The RPE is thought to be affected via changes in the central nervous system as well as factors such as perception of pain and thermal discomfort (Armada-da-Silva *et al.*, 2004).

The present study found that a CWI intervention performed between two high intensity exercise bouts helped to maintain repeat performance in hot environmental conditions when compared to ACT. A reduction in $\overline{T}_b$ and heart rate following all CWI protocols may have resulted in a decrease in peripheral blood flow and therefore produced a greater volume of blood available centrally or to working muscles (Lee & Haymes, 1995; Marsh & Sleivert, 1999). Indeed, the magnitudes of these temperature reductions were related to the temperature of the cold water used in the protocols (e.g. lower water temperatures resulted in the greatest reductions in $\overline{T}_b$). The reductions in $\overline{T}_b$ may also alter or allow improvements in thermoregulation via greater

temperature gradients, producing a larger margin prior to the previously reported critical temperature being reached. Finally, the neural effects (Meeusen & Lievens, 1986) of cooling and the likely effects of anticipation, pacing ability and less inhibition of skeletal muscles have all been suggested following cooling.

The findings of the present study support the use of CWI in various sports at times when two training sessions a day may be performed in hot environmental conditions, and during prolonged competitions where opportunities exist for CWI (e.g. half time). Whilst this study did not observe a significant performance enhancement, the maintenance of performance during maximal efforts separated by only one hour may be crucial in many sports (e.g. cycling, rowing).

While the results of the present study are promising, the area of CWI for post-exercise recovery is one that needs to be researched further. Future research should attempt to investigate alternative modes of exercise, varying temperatures and durations of CWI as well as the effect of such an intervention in thermocomfortable conditions.

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CHAPTER FOUR

Paper Two

Effect of hydrotherapy on recovery from fatigue

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Running title: Water immersion and cycling performance

Key Words: Water Immersion, Recovery, Performance

Abstract

The present study investigated the effects of three hydrotherapy interventions on next day performance recovery following strenuous training. Twelve cyclists completed four experimental trials differing only in 14 min recovery intervention: cold water immersion (CWI), hot water immersion (HWI), contrast water therapy (CWT), or passive recovery (PAS). Each trial comprised five consecutive exercise days of 105 min duration, including 66 maximal effort sprints. Additionally, subjects performed a total of 9 min sustained effort (time trial - TT). After completing each exercise session athletes performed one of four recovery interventions (randomly assigned to each trial). Performance (average power), core temperature, heart rate (HR), and rating of perceived exertion (RPE) were recorded throughout each session. Sprint (0.1-2.2%) and TT (0.0-1.7%) performance were enhanced across the five day trial following CWI and CWT, when compared to HWI and PAS. Additionally, differences in rectal temperature were observed between interventions immediately and 15 min post recovery, however, no significant differences were observed in HR or RPE regardless of day of trial/intervention. Overall, CWI and CWT appear to improve recovery from high intensity cycling when compared to HWI and PAS, with athletes better able to maintain performance across a five day period.

Introduction

In elite cycling events, athletes require the ability to maintain a consistently high level of performance. This is especially important in stage racing where cyclists are required to produce demanding and consistent performances on multiple days. However, when athletes are required to perform on consecutive days the ability to recover well, referring to a period of both physiological and psychological restoration and regeneration becomes very important.

Anecdotal evidence suggests that the sport of cycling has traditionally favoured a recovery profile consisting predominantly of massage and nutritional replacement strategies. However, the use of various recovery interventions and in particular the use of post-exercise hydrotherapy has become increasingly popular (Cochrane, 2004; Wilcock, Cronin, & Hing, 2006). Nonetheless, there is insufficient evidence to reach firm conclusions and little quality research has been conducted in this field, particularly following fatigue-inducing exercise. Despite limited scientific evidence the use of cold water immersion (CWI), hot water immersion (HWI), and contrast water therapy (CWT) as post-exercise recovery interventions have become common practice within many athletic settings (Cochrane, 2004; Wilcock *et al.*, 2006).

Cryotherapy has been recognised as the most commonly used treatment for acute soft tissue sports injuries and in different forms has also been utilised as a post-exercise recovery intervention. Additionally, CWI has been shown to be effective in the treatment of muscle damage and inflammation, or when significant increases in core temperature may affect performance (Eston & Peters, 1999; Marino, 2002; Merrick *et al.*, 1999). In a recent review, it was

concluded that apart from an analgesic effect, there appears to be limited scientific evidence to suggest any enhancement in post-exercise recovery from muscle damage by CWI (Cheung *et al.*, 2003). However, the effect of CWI on repeat high intensity exercise performance has not been fully elucidated.

Hot water immersion (HWI) is a thermotherapeutic intervention in which the body is immersed in water exceeding 36°C (Wilcock *et al.*, 2006). Very little scientific evidence exists to support the use of HWI as a post-exercise recovery intervention; specifically, the effect of HWI on physiological variables and subsequent performance are largely unknown.

Contrast water therapy (CWT) utilizes both cold and hot water immersion and has become a very popular post-exercise recovery intervention (Cochrane, 2004; Wilcock *et al.*, 2006). Similar to CWI, CWT has been shown to be an effective treatment strategy for muscle damage, soreness and inflammation and to enhance the recovery of performance (Vaile *et al.*, 2007). Additionally, CWT has been associated with an increase in the perception of recovery and a quicker reduction of lactate post-exercise (Coffey *et al.*, 2004). However, further research must be conducted to confirm the potential recovery benefits of CWT and to investigate possible mechanisms and protocols.

While the aforementioned recovery interventions have been shown to be effective in various settings or following specific types of exercise (e.g. eccentric exercise) the effect of such interventions on repeated days of high intensity cycling exercise remains largely unknown. Due to the current popularity of post-exercise hydrotherapy (Cochrane, 2004; Wilcock *et al.*, 2006) this is an

area that needs to be investigated further. Therefore, the purpose of the present study was to investigate the effect of three different hydrotherapy techniques (CWI, HWI, CWT) on the recovery of exercise-induced fatigue and next day performance.

Methods

Twelve endurance trained male cyclists volunteered to participate in this study. Their mean ± standard deviation age, height, body mass, $\dot{V}O_{2max}$ and sum of seven skin folds were 32.2 ± 4.3 years, 176.6 ± 4.5 cm, 68.8 ± 7.2 kg, 68.8 ± 3.6 ml.kg-1.min-1 and 58.2 ± 16.9 mm, respectively. Prior to involvement, all subjects were informed of the study requirements and risks and provided informed written consent. Subjects were required to refrain from any strenuous exercise 24 h prior to testing, perform no additional strenuous activity during the five day data collection period, and abstain from caffeine (24 h) and alcohol (48 h) prior to and throughout the five day testing period. The study was approved by the Australian Institute of Sport Research Ethics Committee.

Experimental Design

Prior to participation all subjects completed a $\dot{V}O_{2max}$ test on a cycle ergometer (Lode, Groningen, Netherlands) as well as three familiarisation sessions, including protocols and procedures identical to the five day trial, to minimise any learning effect in the first week of the study. On four separate occasions separated by nine days (randomised crossover design), each subject completed a fatigue-inducing cycle protocol on five consecutive days, each followed by one of four recovery interventions (CWI, HWI, CWT, or passive recovery [PAS, control]) (Figure 1). Recovery intervention remained the same for each day of

the five day trial. Subjects' were required to complete a training and food diary throughout the eight week period, in which training during 'off' weeks was matched for volume and intensity throughout the study and to ensure food intake throughout the testing weeks remained consistent.
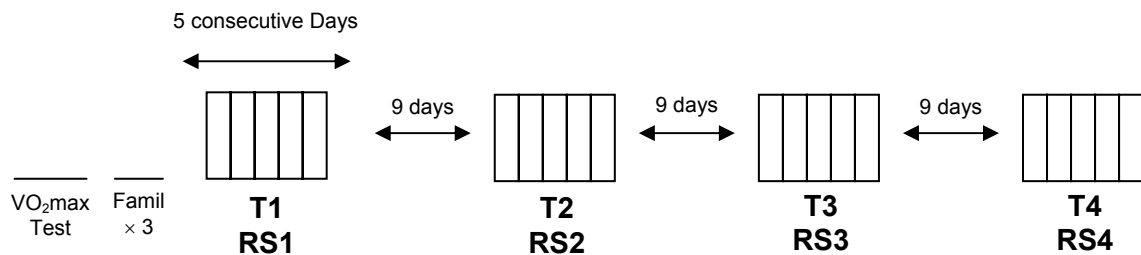
Figure 1. Experimental design indicating preliminary testing ($\dot{V}O_2$max test), familiarisation of testing protocol, and four trials (T1-T4) consisting of the exercise task, performed on five consecutive days. Each session was followed by one of four recovery interventions (RS1-RS4; randomised crossover design).

Fatigue-Inducing Protocol

Subjects completed a 10 min self-paced warm up followed by 3 × 3 s sprints at a perceived intensity of 70%, 80% and 90% of maximum effort respectively. The main exercise task completed daily for five consecutive days totalled approximately 105 min in duration, consisting of 66 maximal effort sprints of 5-15 s duration with specific work to rest ratios of 1:6, 1:3, 1:1 (Martin *et al.*, 2005). Additionally, a total of 9 min of sustained effort was incorporated into the protocol consisting of 2 × 2 min and one 5 min time trial (TT) (Figure 2). This protocol was used in an attempt to simulate the demands of cycling races and to provide an indication of repeat performance capabilities; and has previously been used in our laboratory with elite cyclists. Typical error of this protocol was 17.4 Watts and 2.1%. Throughout each testing session of the five day trial, strong verbal encouragement and support was given to the subjects by the same person and in the same fashion each day, remaining consistent for the duration of the study.

The 'work' phase of the interval session involved a maximal effort while the 'rest' phase involved the subjects cycling at an intensity of 40-50% of their individual peak power output. During the TT efforts the subjects were instructed to complete as much work as possible in the specified time (2 or 5 min effort).

10 min warm up

Set 1 – 12 × 5 s; 1:6 (Work:Rest)

Set 2 – 12 × 5 s; 1:3 (W:R)

Set 3 – 12 × 5 s; 1:1 (W:R)

4 min ACT – *2 min TT* – 4 min ACT

Set 4 – 6 × 10 s; 1:6 (W:R)

Set 5 – 6 × 10 s; 1:3 (W:R)

Set 6 – 6 × 10 s; 1:1 (W:R)

4 min ACT – *2 min TT* – 4 min ACT

Set 7 – 4 × 15 s; 1:6 (W:R)

Set 8 – 4 × 15 s; 1:3 (W:R)

Set 9 – 4 × 15 s; 1:1 (W:R)

5 min ACT – *5 min TT* – 5 min ACT

Recovery Intervention (CWI, HWI, CWT, or PAS) – 14 min

Figure 2. Breakdown of the high intensity exercise task performed daily for five consecutive days. Athletes performed 5 min active recovery between sets 1-2, 2-3, 4-5, 5-6, 7-8, and 8-9 (Martin *et al.*, 2005). ACT = Active Recovery.

Recovery Interventions

Immediately post-exercise, subjects completed a 5 min cycling warm down at approximately 40% of individual peak power output followed by one of four recovery interventions. The same recovery intervention was performed for all five days of each trial. *Cold Water Immersion (CWI):* Subjects immersed their entire body (excluding the neck and head) in a plunge pool set at 15°C for 14 min. *Hot Water Immersion (HWI):* Subjects immersed their entire body (excluding the neck and head) in a spa bath set at 38°C for 14 min (no jets). *Contrast Water Therapy (CWT):* Subjects immersed their entire body (excluding the neck and head) alternating between cold (15°C one min) and hot (38°C one min) water for a total of 14 min (seven cycles). Transition time between hot and cold water baths was approximately 5 s. *Passive Recovery/Control (PAS):* Subjects remained seated with minimal movement for 14 min.

Recovery Assessment

Performance Assessment

Subjects repeated the fatigue-inducing cycle exercise protocol on five consecutive days; peak and average power output and total work completed were recorded via an SRM powermeter (SRM, Schoberer Rad Meßtechnik, Jülich, Germany) fitted to each subjects bicycle, which was in turn mounted on a windtrainer (Kinetic fluid trainers, Kurt Kinetic, Jordan, MN, USA). This allowed data to be collected throughout each of the testing sessions to establish performance, with reduction in power and/or work completed over a session and between days being indicative of fatigue.

Core Temperature

Core temperature was monitored via disposable rectal probe (Monatherm, Mallinckrodt Medical, St Louis, MO, USA) inserted at least 12 cm beyond the anal sphincter prior to testing (O'Brien *et al.,* 2000; Zhang & Tokura, 1999). Core temperature (Zentemp 5000, Zencor Pty Ltd, Victoria, Australia) was recorded pre- and post-exercise, pre- and post-recovery as well as 15 min post recovery.

Heart Rate (HR)

Heart rate was monitored using a Polar heart rate (Kempele, Finland) monitor fitted to the subject for the duration of the testing session. Heart rate was recorded at the end of each sprint set, as well as pre and post recovery.

Rating of Perceived Exertion (RPE)

Subjects rated their perceived exertion on a scale of zero (no exertion at all) to ten (maximal exertion) (Noble *et al.,* 1983) at the end of each sprint set and at the end of the 5 min TT which concluded the exercise session.

Statistical Analysis

Mean effects were calculated for each intervention (significant changes $p < 0.05$) and 95% confidence limits were estimated with a spreadsheet (Batterham & Hopkins, 2005) via the unequal-variances t statistic computed for change scores between pre- and post-tests of the four groups. Each subject's change score was expressed as a percentage of baseline score via analysis of log-transformed values, in order to reduce bias arising from non-uniformity of error.

Results

Sprint Performance

When CWI and CWT was performed following the high intensity exercise bout on five consecutive days there was a significantly ($p<0.01$) better maintenance/improvement of average power on days four and five compared to PAS (Figure 3). However, there were no significant differences ($p>0.05$) between other treatments on any of the five days. Across the five exercise days average power was decreased by 1.7 - 4.9% following PAS, and 0.6 - 3.7% following HWI, while improvements of 0.5 - 2.2% were observed following CWT and 0.1 -1.4% following CWI.

Time Trial (TT) Performance

Absolute values of overall total work (kJ) completed during the 9 min of TT performance are presented in Table 1. Although no significant differences were observed between treatments on day one of each exercise week, it is acknowledged that both CWI and CWT groups produced marginally less total work compared to HWI and PAS. Following PAS, TT performance (average power) had decreased by 3.8% during the five consecutive days of high intensity exercise. However, in comparison, CWT and CWI resulted in a significantly better maintenance of performance compared to PAS ($p<0.05$) (Figure 4). Across the five exercise days performance was decreased by 2.6-3.8% following PAS, in contrast, performance was maintained and slightly improved following CWI (range of 0.1 - 1.0%) and CWT (0.0 - 1.7%); with performance ranging from an improvement of 1.5% to a decrease of 3.4% throughout the five day period following HWI. While there were no significant differences ($p>0.05$) observed in average power between CWT and CWI or HWI

and CWI, there were significant differences observed between HWI and PAS (p=0.02) on day three and between CWT and HWI (p=0.01) on day four of exercise.
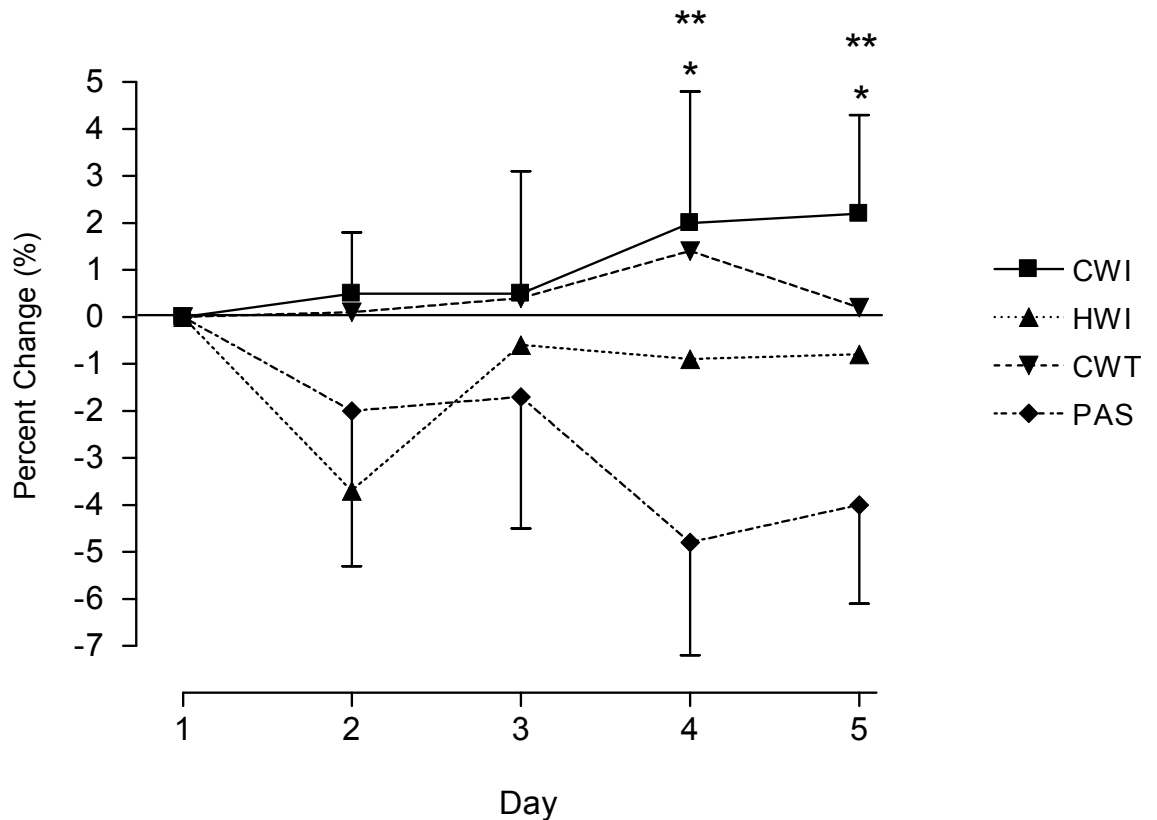
Figure 3. Changes in sprint performance (average power; percent change from baseline/day one) on five consecutive days of high intensity cycle exercise.

* Indicates a significant difference (p<0.05) between CWI and PAS.

** Indicates a significant difference (p<0.05) between CWT and PAS.

Table 1. Absolute values of total work (kJ) completed during the totalled nine minutes of time trial performed daily on five consecutive days.

* indicates a significant difference (p<0.05) between the stated intervention (CWI or CWT) and both HWI and PAS.

	Work (kJ)				
Recovery	Day 1	Day 2	Day 3	Day 4	Day 5
CWI	157 ± 20	158 ± 21	159 ± 21	161 ± 21*	160 ± 20*
HWI	159 ± 21	158 ± 22	161 ± 23	153 ± 21	156 ± 22
CWT	157 ± 21	160 ± 22	160 ± 20	162 ± 22*	161 ± 20*
PAS	161 ± 20	160 ± 20	157 ± 22	155 ± 22	155 ± 22
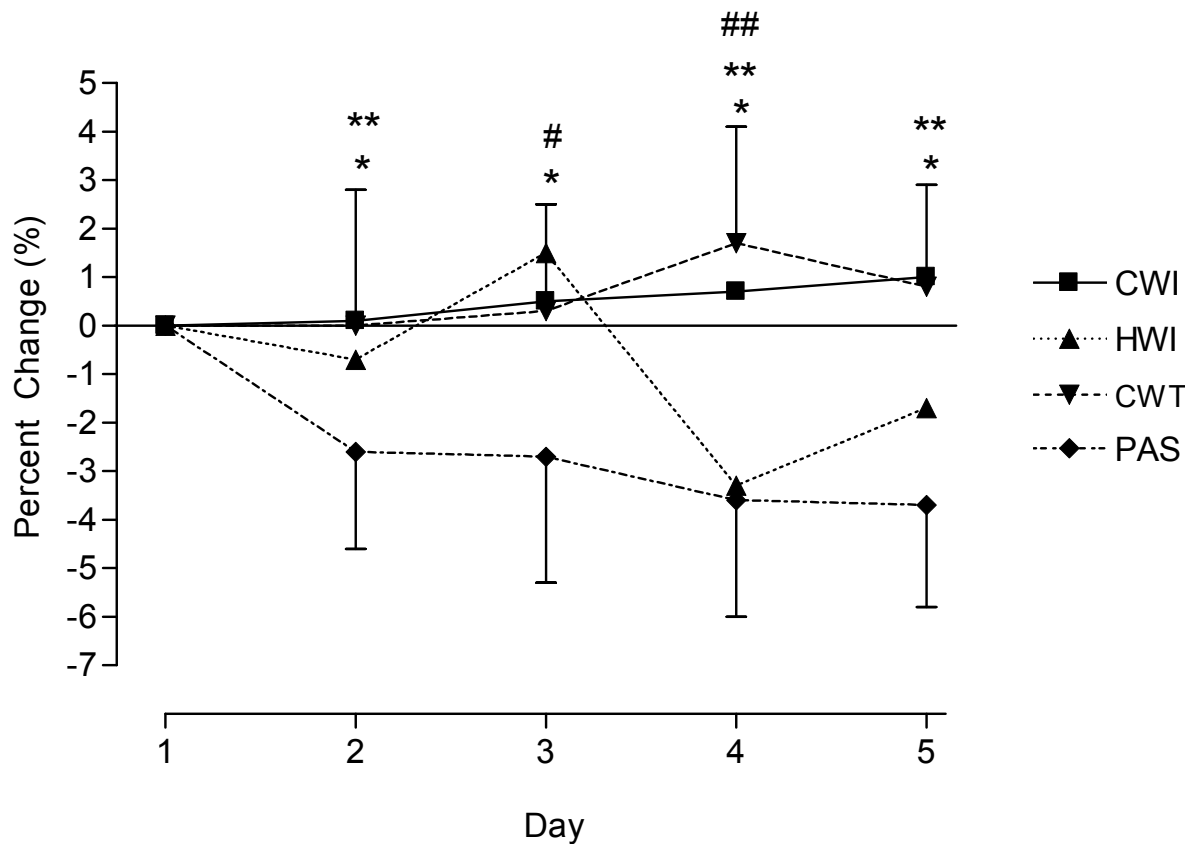
Figure 4. Changes in time trial performance (average power; percent change from baseline/day one) on five consecutive days of high intensity cycle exercise.
* Indicates a significant difference (p<0.05) between CWT and PAS.
** Indicates a significant difference (p<0.05) between CWI and PAS.
# Indicates a significant difference (p<0.05) between HWI and PAS.
Indicates a significant difference (p<0.05) between CWT and HWI.

Rectal Core Temperature

No significant differences ($p > 0.05$) in rectal temperature were observed between groups at baseline (pre-exercise) or immediately post-exercise (Figure 5). Average pre-exercise rectal temperature regardless of intervention group or day of exercise was 37.3 ± 0.2°C with an average rectal temperature of 38.5 ± 0.2°C at the completion of the high-intensity exercise task. Immediately post-recovery rectal temperature was 37.3 ± 0.2°C (CWI), 37.6 ± 0.2°C (HWI), 37.5 ± 0.2°C (CWT), and 37.4 ± 0.2°C (PAS). Significant differences ($P < 0.02$) were observed between HWI vs. CWI and PAS, as well as CWI vs. CWT. However, 15 min post-recovery rectal temperature was 36.8 ± 0.2°C (CWI), 37.7 ± 0.2°C (HWI) and 37.3 ± 0.2°C (CWT and PAS). Significant differences ($p < 0.03$) were observed between HWI vs. CWI, CWT and PAS, as well as CWI vs. CWT and PAS.

Heart Rate (HR)

Heart rate recorded immediately post-TT (5 min) on days 1-5 were 174, 175, 175, 169, 170 bpm respectively following PAS; 173, 173, 174, 174, 169 bpm following CWI; 173, 171, 172, 167, 167 bpm following HWI; and 174, 171, 169, 172, 172 bpm following CWT. While there were no significant differences in HR immediately post-TT, there were some noteworthy effects sizes (ES) suggesting there may be some small to large effects in HR response during exercise between interventions. For example, post-TT HR tended to be lower on days 2-5 following HWI when compared to PAS representing a moderate effect (ES > 0.6). In addition, when compared to PAS, HR tended to be higher on day 4 and day 5 following CWT (ES = 0.6), and a similar but larger effect was evident on day 4 following CWI (ES = 1.2).
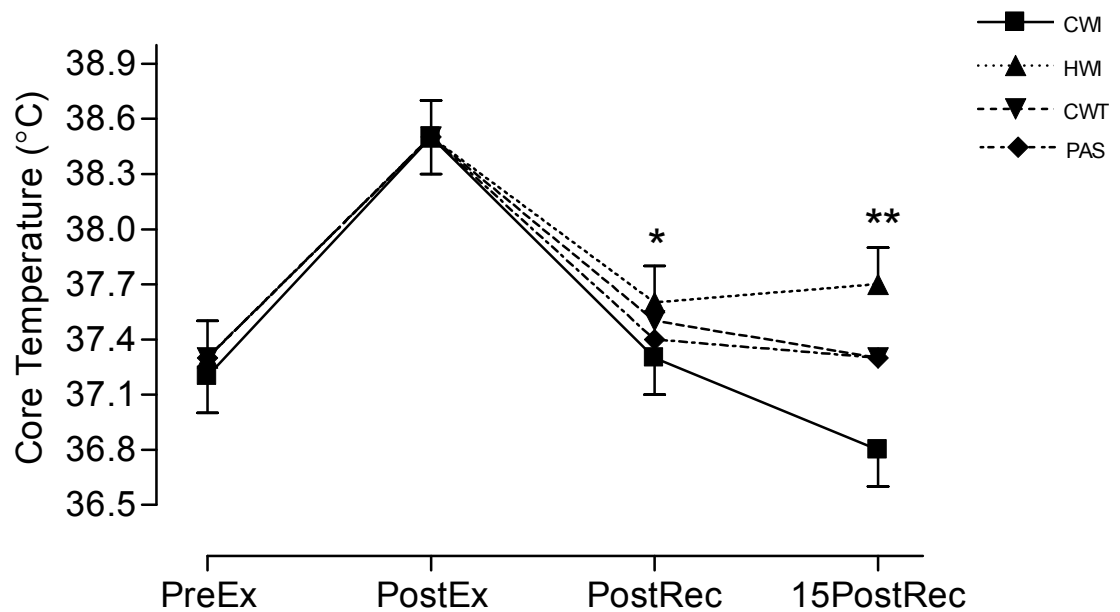
Figure 5. Changes in core temperature recorded pre and post-exercise, immediately post recovery (CWI, HWI, CWT, or PAS), and 15 min post recovery. Values represent the average core temperature at the given time points across the five day trial for each individual intervention.

* Indicates significant differences (p<0.02) between HWI vs. CWI and PAS, CWI vs. CWT.

** Indicates a significant difference (p<0.03) between HWI vs. CWI, CWT and PAS, CWI vs. CWT and PAS.

Rating of Perceived Exertion (RPE)

There were no significant differences or changes in the subjects' perception of exertion throughout the exercise protocol regardless of recovery intervention or day of trial. The average RPE reported throughout the study (independent of intervention and day of trial) was observed to be between 8 and 9, on a scale of 0-10 with 10 being maximal exertion.

Discussion

The main finding of the present study was that both CWI and CWT significantly better maintained performance compared to HWI and PAS throughout the five consecutive days of testing. Sprint performance was maintained and slightly improved following CWI and CWT (0.5 - 2.2% and 0.1 - 1.4% respectively) with similar trends observed throughout TT performances (CWI 0.1 - 1.0%; CWT 0.0 - 1.7%). In contrast, following HWI and PAS a decline of 0.6 - 3.7% and up to 3.4 and 3.8% throughout sprint performance and 1.7% and 3.8% throughout TT performance, respectively. Interestingly, the effects of hydrotherapy appeared to be more pronounced during TT performance compared to sprint performance, with some of the hydrotherapy interventions proving to be significantly better than PAS from the second day of exercise as opposed to just the fourth and fifth day of exercise in sprint performance. These are noteworthy findings in regard to athlete workloads, as while recovery is generally considered an important aspect of training and competition, inappropriate and/or inadequate recovery may result in a decrease in performance ability (Cochrane, 2004; Halson & Jeukendrup, 2004).

In a similar randomised cross over design Lane and Wenger (2004) investigated the effect of CWI, active recovery and massage on repeat cycling performance separated by 24 h. Following the completion of a cycle sprint protocol, subjects performed one of four 15 min recovery interventions (CWI, active recovery, massage, or passive recovery/control) then 24 h after the first exercise session the cycle sprint protocol was repeated. When active recovery, CWI, and massage were performed between exercise bouts the ability to maintain power in the second exercise bout was significantly enhanced. The authors concluded that CWI, active recovery and massage can all facilitate recovery between two high intensity exercise bouts separated by 24 h (Lane & Wenger, 2004). However, CWI provided the only improvement in performance, suggesting it was the most beneficial of the three treatments. The results of the present study are in agreement with the findings of Lane and Wenger (2004), in that CWI also provided a maintenance and slight improvement in performance over consecutive days.

Despite the significant improvements in performance following CWI and CWT, there were no significant changes in RPE or HR throughout the study, regardless of treatment. Due to the nature of the maximal effort exercise task, it is not surprising that perceptions of the effort and HR were not significantly different throughout the study. Therefore, the findings of this study suggest that during maximal effort exercise, measures of HR and RPE may not be sensitive enough for assessing physiological or psychological responses to tasks of this nature. This may become more apparent during sub-maximal exercise.

In a recent review (Wilcock *et al.*, 2006), it was noted that a major contraindication of CWT and its subsequent research is the concurrent exposure to both hot and cold water, and that outcomes have not been compared to either hot or cold water immersion protocols independently. Therefore, recovery interventions in the present study were selected to investigate the isolated effect of immersion in hot and cold water individually as well as when alternated (CWT), with duration of water exposure (14 min) identical regardless of intervention. Results suggest that intermittent exposure to both hot and cold water of prescribed temperatures and duration should not be a concern from a physiological or performance viewpoint. Indeed, the present results support the use of CWT as a post-exercise recovery intervention to supplement recovery from high intensity repeat cycling. Additionally, there is an increasing body of knowledge to support the use of CWT as a recovery intervention and while not all studies have found a performance benefit, it has not been found harmful or detrimental in any way (Coffey *et al.*, 2004; Vaile *et al.*, 2007).

There is limited scientific research into the physiological mechanisms by which various post-exercise hydrotherapy interventions may be advantageous. However, the effects of CWI in various temperatures has been investigated, with physiological responses including reductions in core and tissue temperature (Enwemeka *et al.*, 2002; Myrer, Measom, Durrant, & Fellingham, 1997), decreased heart rate and cardiac output, increased peripheral resistance and arterial blood pressure (Sramek *et al.*, 2000), fluid shifts (Enwemeka *et al.*, 2002; Wilcock *et al.*, 2006), and reduced swelling (Cochrane, 2004; Smith, 1991) being reported. Data from the present study support previous findings of

a reduction in core temperature following CWI (Marsh & Sleivert, 1999). While a reduced HR was not observed in the present study, it must be noted that the effectiveness of recovery was assessed 24 h post-recovery. In addition, reduced perceptions of pain have also been observed (Smith, 1991). Due to the nature of the exercise task, requiring maximal effort, subjects RPE remained unchanged throughout the five day exercise period. However, a sub-maximal exercise component to the protocol may have resulted in a different finding.

Unlike CWI, research investigating the effects of water immersion in hot temperatures is limited. However, HWI has been suggested to increase heart rate, cardiac output, and peripheral vasodilation, resulting in increased tissue temperatures (Bonde-Petersen *et al.*, 1992; Wilcock *et al.*, 2006). The present study observed significantly higher post-recovery core temperatures following HWI when compared to CWI, CWT, and PAS. The inflammatory response and swelling are also exacerbated following the application of heat (Cote, Prentice, Hooker, & Shields, 1988; Wilcock *et al.*, 2006), and while performance appeared to be compromised in this study following HWI, specific mechanisms must be further investigated. Contrast water therapy protocols utilise the effects of both hot and cold water exposure and therefore may enhance recovery by increasing blood flow, circulation, lactate removal, and range of motion, and by decreasing the inflammatory response, pain, stiffness and the effects of exercise-induced muscle damage (Wilcock *et al.*, 2006). In the present study, performance appeared to be better maintained following the use of CWT, however, a more mechanistic approach in future studies may provide further information regarding the process by which CWT may be effective.

In addition to temperature aspects, the effects of hydrostatic pressure during water immersion may be an important aspect of the success of hydrotherapy as a recovery intervention. The pressure applied to the body during water immersion may cause a displacement of fluid from the extremities, increasing central blood volume (Arborelius *et al.*, 1972; Lollgen, von Nieding, Koppenhagen, Kersting, & Just, 1981; Wilcock *et al.*, 2006). Although this beneficial effect of hydrostatic pressure is evident, regardless of temperature, all interventions in the present study (CWI, HWI, CWT) were performed in identical conditions with pressure exerted and duration of water exposure identical. Therefore, although hydrostatic pressure may contribute to the effectiveness of hydrotherapy, temperature must also play an integral role, with findings suggesting CWI and CWT to be more beneficial than HWI. Physiologically, continuous HWI may be more detrimental than exposure to cooler water due to the increase in cardiovascular strain.

Research into the recovery from repetitive bouts of training or competition provides valuable knowledge regarding the adaptation to specific stress. Such information is essential if the purpose of a particular training session or competition regime is to produce improved performances. The results from the present study are unique and novel from a training perspective. Data examining recovery must be gathered in applied and practical environments to allow transfer of results to the 'real world' of athletic performance. The current findings suggest that CWI and CWT are useful interventions for maintaining and even slightly improving consecutive daily cycling performance in both TT and repetitive sprint performance. Whether such results are from a reduction in cumulative fatigue or an accelerated adaptation is unknown. Indeed, the

precise mechanism/s by which this occurs is unclear and requires further investigation.

Practical applications

The results of the present study suggest that CWI and CWT may be beneficial recovery interventions following and between events such as track cycling where the task requires short maximal efforts, as well as longer events such as stage races where the task requires continuous high intensity efforts on successive days.

Future research

Future scientific research should be conducted to further investigate the effect of hydrotherapy techniques, following high intensity exercise, particularly when repeat performances are required. The present study suggests CWI and CWT to be promising recovery interventions; however, future studies must be conducted to enhance the body of knowledge and understanding of hydrotherapy and its associated mechanisms.

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CHAPTER FIVE

Paper Three

Effect of hydrotherapy on the signs and symptoms of delayed onset muscle soreness

Abstract

This study independently examined the effects of three hydrotherapy interventions on the physiological and functional symptoms of DOMS. Strength trained males (n = 38) completed two experimental trials separated by eight months in a randomised crossover design; one trial involved passive recovery (PAS, control), the other a specific hydrotherapy protocol for 72 h post-exercise; either: 1) cold water immersion (CWI: n = 12) 2) hot water immersion (HWI: n = 11) or 3) contrast water therapy (CWT: n = 15). For each trial subjects performed a DOMS-inducing leg press protocol followed by PAS or one of the hydrotherapy interventions for 14 min. Weighted squat jump, isometric squat, perceived pain, thigh girths, and blood variables were measured prior to, immediately after, then 24, 48 and 72 h post-exercise. Squat jump performance and isometric force recovery was significantly enhanced (P<0.05) at 24, 48 and 72 h post-exercise following CWT and at 48 and 72 h post-exercise following CWI when compared to PAS. Isometric force recovery was also greater (P<0.05) at 24, 48, and 72 h post-exercise following HWI when compared to PAS. Perceived pain was only improved (P<0.01) following CWT at 24, 48 and 72 h post-exercise. Overall, CWI and CWT were found to be effective in reducing the physiological and functional deficits associated with DOMS, including improved recovery of isometric force and dynamic power and a reduction in localised oedema. While HWI was effective in the recovery of isometric force, it was an ineffective for recovery of all other markers compared to PAS.

Introduction

Delayed onset muscle soreness (DOMS) is a well documented phenomenon, often occurring as the result of unaccustomed or high intensity eccentric exercise (Connolly *et al.,* 2003; MacIntyre *et al.,* 1995). Associated symptoms include muscle shortening, increased passive stiffness, swelling, decreases in strength and power, localised soreness, and disturbed proprioception (Proske & Morgan, 2001). Symptoms will often present within 24 h post-exercise and typically subside after 3-4 days (Clarkson & Sayers, 1999). Elite athletes are often susceptible to muscle damage due to muscles being regularly subjected to repetitive, high intensity contractions (Allen *et al.*, 2004).

Recently, the use of various forms of hydrotherapy as post-exercise recovery interventions, such as cold water immersion (CWI), hot water immersion (HWI), and contrast water therapy (CWT) have gained popularity and are now common practice within elite sporting environments (Cochrane, 2004; Vaile *et al.,* 2007). However, such recovery interventions are being employed despite a lack of scientific investigation and evidence regarding their potential benefits and/or mechanisms by which they may work.

Various forms of cryotherapy have been shown to produce multiple physiological responses, including decreased swelling (Yanagisawa *et al.*, 2004), tissue temperatures (Enwemeka *et al.*, 2002), heart rate (HR) and cardiac output (Sramek *et al.*, 2000), enhanced creatine kinase clearance (Eston & Peters, 1999) and analgesic effects, resulting in altered perceptions of pain and discomfort (Bailey *et al.*, 2007). However, there appear to be conflicting conclusions regarding the effect of CWI on performance, with some

studies suggesting beneficial effects (Bailey *et al.*, 2007; Burke *et al.*, 2000; Lane & Wenger, 2004) and others indicating negligible changes (Isabell, Durrant, Myrer, & Anderson, 1992; Paddon-Jones & Quigley, 1997; Sellwood *et al.*, 2007; Yamane *et al.*, 2006). In contrast, despite limited research in the area, HWI affects the body differently, resulting in increased HR, cardiac output and tissue temperatures and may enhance the inflammatory response (Wilcock *et al.*, 2006). Contrast water therapy (CWT) incorporates the combined effect of both CWI and HWI with athletes alternating between them for a set period of time. While there is limited research investigating the physiological effects of CWT and its role on return/maintenance of performance following damage or exercise-induced fatigue, current knowledge suggests CWT to be a promising recovery intervention (Coffey *et al.*, 2004; Gill, Beaven, & Cook, 2006; Vaile *et al.*, 2007). However, Wilcock *et al.* (2006) have recently criticised CWT, suggesting potential contraindications to be the unknown effects of exposure to both hot and cold water as well as the effect of CWT on tissue oedema accumulation.

Consequently, the present studies set out to examine the effect of the three hydrotherapy interventions (CWI, HWI, and CWT) in comparison to a passive rest recovery following a controlled muscle-damaging exercise task, ensuring identical durations of recovery, water exposure and temperatures were maintained. Functional and physical symptoms of DOMS and the recovery of performance were assessed.

Methods

Subjects

A total of 38 strength trained males completed two experimental trials separated by eight months in a randomised crossover design; one trial involved passive recovery (PAS, control), the other a specific hydrotherapy protocol. Subjects were randomly assigned to one of three groups differing only in recovery hydrotherapy intervention: 1) cold water immersion (CWI, 15°C, n = 12), 2) hot water immersion (HWI, 38°C, n = 11) or 3) contrast water therapy (CWT, 15°C/38°C, n = 15). These interventions were selected using water temperatures and durations similar to those used in common practice and to ensure identical durations of water exposure. The physical and functional symptoms of DOMS were monitored throughout a 72 h follow-up period and compared to pre-exercise values. After an eight month washout period, the subjects completed the exercise task with the alternate (hydrotherapy or PAS) recovery protocol.

Experimental Design

On two separate occasions (eight months apart; hydrotherapy vs. PAS) subjects completed a muscle-damaging protocol (MDP) consisting of seven sets of ten eccentric repetitions on a leg press machine. Previously it has been demonstrated that a single bout of eccentric exercise can have a prophylactic effect on not only muscle soreness, but also blood responses and performance capabilities after a second bout of eccentric exercise performed within a few weeks (Brown, 1997; Byrnes & Clarkson, 1986; Mair *et al.*, 1995; Nosaka *et al.*, 2001). Therefore, it was important to consider this effect and control for it by utilising a crossover design and selecting athletes who were both familiar and

accustomed to resistance training (Viitasalo *et al.*, 1995). A substantial wash-out period of eight months was chosen to minimise the effect of the first session of eccentric exercise (athletes were required to continue exercising as per usual and not perform any specific eccentric training). Nosaka et al. (2001) investigated the duration of the protective effect of eccentric exercise-induced muscle damage, concluding that the repeated bout effect for most measures appeared to last at least six months.

Two weeks prior to both trials (separated by eight months) subjects completed a comprehensive familiarisation session to determine maximal strength in the form of one repetition maximum (1RM) on the leg press machine and isometric squat 1RM to establish squat jump load (30% isometric squat) (Nosaka & Newton, 2002). Additionally, subjects were familiarised with squat jump and isometric squat protocols until no further learning/improvement was apparent (this was achieved by a maximum of three independent familiarisation sessions). Following each testing session, and once a day for 72 h post-exercise, subjects performed one of two recovery interventions (hydrotherapy or PAS). Prior to participation, all subjects were informed of the requirement and risks associated with the study and provided informed written consent. The study was approved by the Australian Institute of Sport Research Ethics Committee.

Procedures

The DOMS-inducing exercise protocol consisted of 5 × 10 eccentric bi-lateral leg press contractions with a load of 120% of one repetition maximum (1-RM [concentric]) followed by 2 × 10 at a load of 100% 1-RM. The aforementioned

protocol was chosen as eccentric strength has been shown to be approximately 20-60% greater than concentric strength and similar protocols have been successfully employed to induce DOMS (Hortobagyi & Katch, 1990). During each eccentric contraction, the load was resisted with both legs from full knee extension to a 90° knee angle (Vaile *et al.*, 2007) with contractions lasting 3-5 s in duration. After the completion of each eccentric repetition the load was raised by an electrical winch. Subjects completed one contraction every 15 s and had a 3 min rest period between sets (Nosaka & Newton, 2002; Vaile *et al.*, 2007).

Recovery Interventions

Following each testing session, and once a day for 72 h post-exercise, subjects performed one of two recovery interventions (hydrotherapy or PAS). All subjects completed PAS recovery and one of the other three hydrotherapy interventions (subjects wore shorts during the hydrotherapy intervention and shorts/t-shirt during PAS). These were: 1) Passive recovery/control (PAS) whereby subjects were seated with minimal movement for 14 min. 2) Cold water immersion (CWI) where subjects immersed their entire body (excluding head and neck) in 15°C water for 14 min. 3) Hot water immersion (HWI) where subjects immersed their entire body (excluding head and neck) in 38°C water for 14 min. 4) Contrast water therapy (CWT) where subjects immersed their entire body (excluding head and neck) and alternated between cold water exposure (15°C one min) and hot water exposure (38°C one min) water for a total of 14 min (seven cycles). Subjects were required to transfer between the hot and cold baths in less than 5 s to ensure maximal duration of water

exposure. Recovery was performed immediately following the post-exercise testing session, then 24, 48, and 72 h post-exercise.

Outcome measures

The effects of the exercise task and subsequent recovery were assessed though the measurement of isometric squat force, squat jump performance, blood markers (creatine kinase [CK], myoglobin [Mb], interleukin-6 [IL-6], lactate dehydrogenase [LDH]), thigh circumference and perceived muscle soreness. Measures were recorded pre-exercise, and immediately post-exercise, as well as 24, 48 and 72 h post-exercise.

Recovery Assessment

Isometric Squat (Peak Force)

The production of vertical ground reaction forces were measured via force platform (Kistler Instrumenté, Switzerland) and assessed through an isometric squat performed against an immovable bar on a Smith Machine. On each occasion, subjects performed thee trials, each separated by 3 min, with the best effort (indicated by peak vertical force) used to represent the subject's isometric squat force. The squat was performed in an identical position each time, with foot placement recorded for each individual and maintained throughout all testing sessions to ensure a straight line from the temporo-mandibular joint to the lateral malleolus with the subject in a standing position (Blazevich, Gill, & Newton, 2002; Vaile et al., 2007). The protocol used to assess isometric force was found to have an ICC = 0.97 and TEM = 2.9%.

Squat Jump (Peak Power)

Subjects were required to perform squat jumps (separated by 2 min) on a Smith machine which was loaded to a combined weight equivalent of 30% of their isometric squat force. The best of the three attempts was recorded for analysis. Subjects were instructed to lower the weighted bar to a 90° knee angle, pause for 2 s, and then jump upward for maximum height (Vaile *et al.*, 2007). Peak power was measured using a GymAware system (Kinetic Performance, Australia). When assessed on 10 subjects this peak power protocol was acceptably reliable (ICC = 0.94, TEM = 6.1%).

Blood Markers

Venous blood samples were collected pre-exercise and at each of the four post-exercise time-points. Each blood sample (8mL) was collected from a superficial forearm vein using standard venipuncture techniques.   All samples were collected directly into serum separator collection tubes (Greiner Bio-one; Frickenhausen, Germany) and serum separated by centrifugation at 4000 rpm for 5 min. Serum samples were stored frozen at -80 °C until analysis. Creatine Kinase (CV 0.6%) and LDH (CV 0.8%) concentrations were determined using a Hitachi 911 automated clinical chemistry analyzer (Roche Diagnostics Corporation; Indianapolis, IN, USA) and commercially available reagents (Roche Diagnostics Corporation; IN, USA). Myoglobin (CV 2.6%) and IL-6 (CV 3.5%) concentrations were determined using an Immulite 1000 (Diagnostics Products Corporation, CA, USA) solid-phase chemiluminescent enzyme immunoassay system and commercially available assay kits (Diagnostics Products Corporation, CA, USA).

Thigh Circumference

A non-stretch anthropometric measuring tape (Lufkin, USA) was used to measure circumference at three sites on the upper leg: above-knee, mid-thigh, and sub-gluteal. Measurement sites were marked with a permanent marker to ensure re-test reliability (0, 24, 48 and 72 h). Circumference measurements were taken as an indicator of acute changes in thigh volume (Brown, 1997; Chen & Hsieh, 2000; Chleboun et al., 1998; Eston & Peters, 1999), likely to occur from osmotic fluid shifts or inflammation, which has often been associated with muscle-damage and eccentric exercise (Fielding et al., 2000). For the purposes of presentation, mid-thigh girth was selected for representation of all upper leg measurements (above-knee, mid-thigh, and sub-gluteal) as it closely resembled changes throughout all of the measured sites. When 10 subjects were tested and re-tested using identical methodology as used in the present study the reliability of these measurements was ICC = 1.00; TEM = 0.1%.

Perceived Soreness

A visual analogue scale (VAS; 0-10) was used to assess the subjects perceived soreness whereby they were required to rank their perception of soreness on a scale of zero to 10, with zero being 'normal' and 10 being 'extremely sore'. This method has been used previously as a non-invasive way to monitor changes in perceived pain following muscle damaging protocols (Cleak & Eston, 1992; Harrison et al., 2001; Vaile et al., 2007). Prior to reporting their VAS ranking, subjects were required to perform a standardised half squat to ensure all subjects were experiencing the same movement/sensation.

Statistical Analysis

Each part of the present study (CWI vs. PAS; HWI vs. PAS; CWT vs. PAS) was independently analysed. Mean effects were calculated using a spreadsheet via the unequal-variances t statistic computed for change scores between pre- and post-tests of the two groups (Batterham & Hopkins, 2005). Each subject's change score was expressed as a percentage of baseline score via analysis of log-transformed values, in order to reduce bias arising from non-uniformity of error. Baseline values (for all variables) from the two trials eight months apart were also compared, with no significant difference observed over time.

Results

Performance Measures

Isometric Squat

No differences were observed between any intervention at baseline or immediately post-exercise (P>1.3) (Table 1). However, change in isometric squat performance (% change from baseline) was significantly less at 24, 48, and 72 h post exercise following both HWI (-12.8, -10.1, -3.2%; P<0.05; Figure 1b) compared to PAS (-17.0, -16.0, -9.8%) and CWT (-10.3, -7.4, -2.8%; P<0.01; Figure 1c) compared to PAS (-17.3, -14.0, -11.5%). Additionally, at 48 and 72 h post-exercise change in isometric squat performance from baseline was significantly less following CWI (-7.3, -4.3%; P<0.05; Figure 1a) when compared to PAS (-15.7, -11.7%).

Weighted Squat Jump

Compared to PAS, change in peak power performance (% change from baseline) was significantly less at 48 (P=0.01) and 72 (P=0.03) h post-exercise

following CWI (Figure 2a) and 24, 48, and 72 h post-exercise following CWT ($P<0.01$; Figure 2c). However, HWI did not positively influence the recovery of squat jump performance compared to PAS (Table 1). Production of peak power 72 h post-exercise was significantly reduced below baseline by 8.2 ± 4.1% following HWI and 7.7 ± 3.2% following PAS; no differences were observed between HWI and PAS ($P>0.05$; Figure 2b) at any time point.

Table 1. Descriptive statistics (mean ± SD) for dependent variables for each intervention and its independent control (CWT vs. PAS, CWI vs. PAS, and HWI vs. PAS). Note: Where appropriate statistics were completed using log transformed values. * Significant difference between specified hydrotherapy treatment and PAS

Variable	CWT	vs.	PAS	CWI	vs.	PAS	HWI	vs.	PAS
Squat Jump (Peak Power W)									
Baseline	3938 ± 871		3969 ± 879	4158 ± 945		4170 ± 947	3902 ± 303		3900 ± 277
0 h post ex	3328 ± 806		3479 ± 792	3547 ± 1033		3564 ± 878	3446 ± 351		3382 ± 278
24 h post ex	3675 ± 741 *		3389 ± 750	3735 ± 872		3577 ± 878	3459 ± 389		3401 ± 416
48 h post ex	3805 ± 821 *		3473 ± 755	3939 ± 877*		3507 ± 795	3487 ± 455		3460 ± 370
72 h post ex	3937 ± 808 *		3659 ± 795	4080 ± 914 *		3857 ± 846	3593 ± 409		3606 ± 356
Isometric Squat (Peak Force N)									
Baseline	2068 ± 446		2066± 469	2110 ± 472		2089 ± 443	1592 ± 262		1916 ± 350
0 h post ex	1733 ± 320		1750 ± 389	1748 ± 424		1734 ± 420	1929 ± 295		1597 ± 271
24 h post ex	1857 ± 405 *		1711 ± 396	1877 ± 418		1792 ± 401	1685 ± 286 *		1598 ± 342
48 h post ex	1923 ± 457 *		1783 ± 424	2077 ± 465 *		1769 ± 412	1735 ± 272 *		1617 ± 329
72 h post ex	2018 ± 477 *		1833 ± 436	2074 ± 487 *		1859 ± 463	1868 ± 291 *		1724 ± 290
Mid-Thigh Circumference (cm)									
Baseline	56.2 ± 4.5		56.1 ± 4.5	56.7 ± 3.7		56.6 ± 3.4	57.3 ± 3.8		57.4 ± 3.7
0 h post ex	56.8 ± 4.6		56.7 ± 4.6	57.4 ± 3.8		57.1 ± 3.3	57.8 ± 3.8		57.9 ± 3.7
24 h post ex	56.4 ± 4.5 *		56.9 ± 4.7	57.1 ± 3.8 *		57.6 ± 3.2	58.1 ± 3.9		58.1 ± 3.8
48 h post ex	56.3 ± 4.6 *		56.9 ± 4.7	56.9 ± 3.8 *		57.4 ± 3.3	57.9 ± 3.9		58.0 ± 3.7
72 h post ex	56.3 ± 4.5 *		56.7 ± 4.7	56.9 ± 3.8 *		57.1 ± 3.3	57.6 ± 3.8		57.8 ± 3.8
Creatine Kinase (U/L)									
Baseline	176 ± 76		245 ± 220	223 ± 222		189 ± 45	199 ± 241		143 ± 105
0 h post ex	229 ± 147		218 ± 168	203 ± 175		193 ± 156	269 ± 411		165 ± 105
24 h post ex	736 ± 1115		737 ± 361	231 ± 182 *		570 ± 263	312 ± 242		402 ± 255
48 h post ex	416 ± 589		361 ± 318	211 ± 259		263 ± 174	225 ± 221 *		748 ± 1694
72 h post ex	359 ± 433		271 ± 234	204 ± 343 *		296 ± 290	151 ± 57		169 ± 86
Lactate Dehydrogenase (U/L)									
Baseline	271 ± 72		218 ± 107	236 ± 82		207 ± 61	261 ± 87		256 ± 93
0 h post ex	280 ± 87		246 ± 98	227 ± 95		208 ± 52	278 ± 85		272 ± 103
24 h post ex	291 ± 132		270 ± 123	194 ± 65		194 ± 69	271 ± 90		269 ± 97
48 h post ex	264 ± 117		230 ± 92	177 ± 71		204 ± 89	260 ± 69		280 ± 68
72 h post ex	254 ± 109		247 ± 112	183 ± 68		219 ± 75	254 ± 83		267 ± 77
Myoglobin (ng/mL)									
Baseline	44.1 ± 22.3		47.8 ± 38.4	36.4 ± 17.8		27.2 ± 7.71	35.6 ± 22.8		27.3 ± 7.7
0 h post ex	95.4 ± 76.6		116.2 ± 101.1	60.7 ± 30.1		67.5 ± 24.9	65.1 ± 44.3		74.8 ± 68.1
24 h post ex	67.2 ± 51.1		69.5 ± 54.9	44.9 ± 25.4		38.5 ± 13.3	39.8 ± 23.2		47.3 ± 22.7
Interleukin-6 (pg/mL)									
Baseline	1.5 ± 0.6		1.7 ± 0.7	3.6 ± 3.9		2.6 ± 2.3	1.7 ± 1.1		1.7 ± 1.1
0 h post ex	2.2 ± 0.7		2.6 ± 1.1	4.5 ± 6.8		3.4 ± 3.2	2.3 ± 1.3		2.3 ± 1.3
24 h post ex	1.5 ± 0.9		1.9 ± 1.0	3.7 ± 6.3		2.8 ± 2.5	1.7 ± 0.9		1.7 ± 0.9
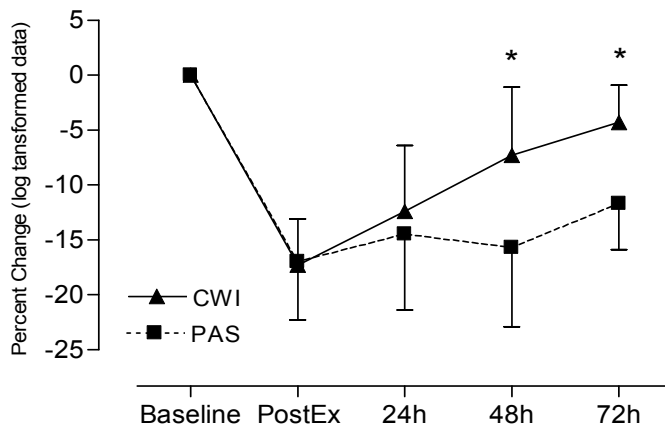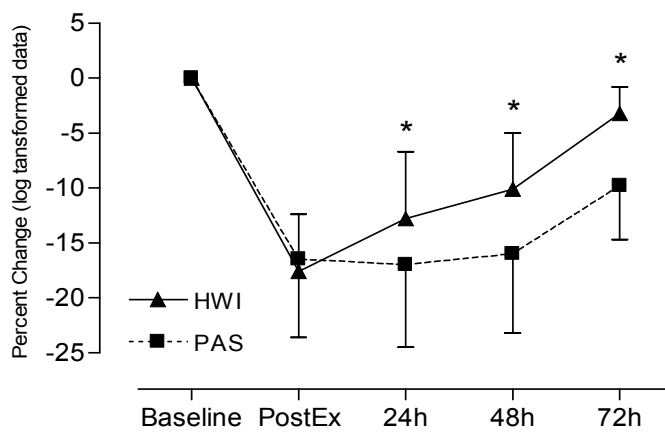
Figure 1a

Figure 1b
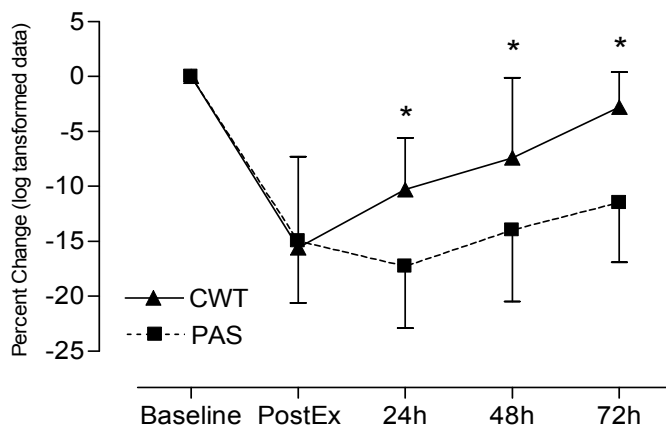
Figure 1c

Figure 1. Percent change in isometric squat performance (peak force) following CWI (1a), HWI (1b), and CWT (1c). Performance was assessed pre and post muscle-damaging exercise as well as 24, 48, and 72 h post-exercise. * Indicates a significant difference between hydrotherapy intervention and PAS.
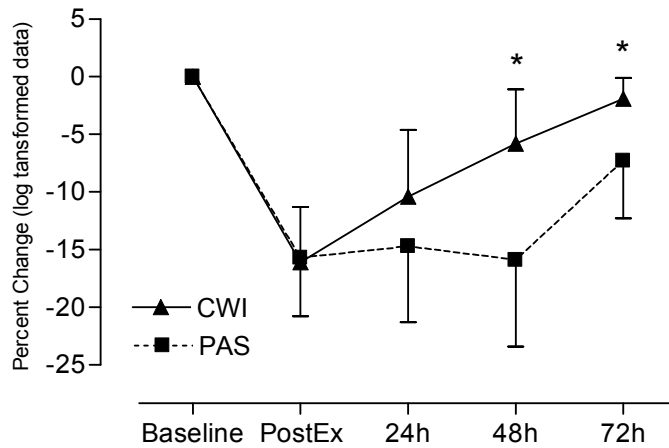
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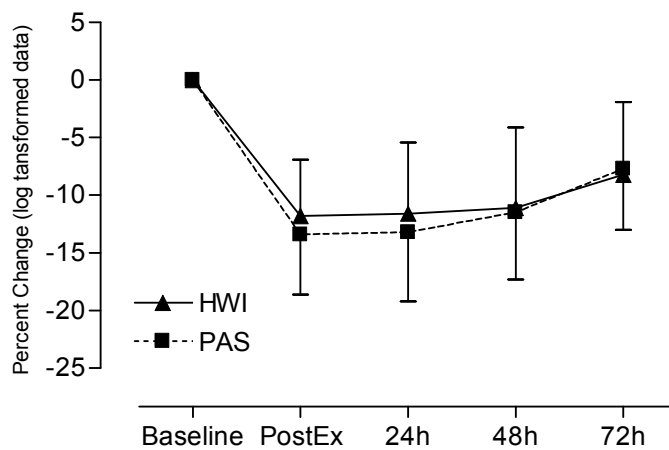
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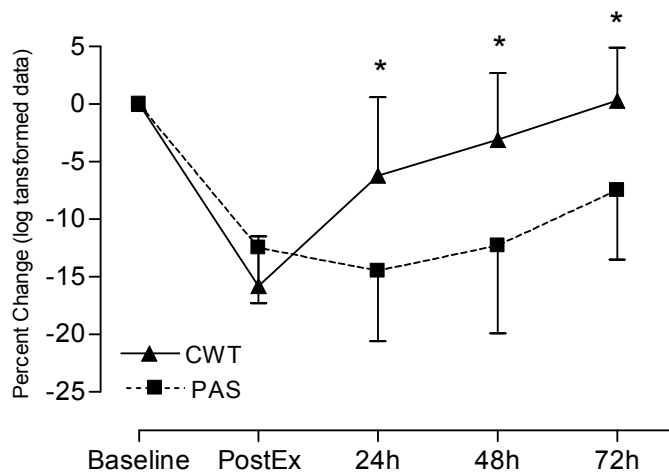
Figure 2c

Figure 2. Percent change in squat jump performance (peak power) following CWI (2a), HWI (2b), and CWT (2c). Performance was assessed pre and post muscle-damaging exercise as well as 24, 48, and 72 h post-exercise.

* Indicates a significant difference between hydrotherapy intervention and PAS

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Mid-Thigh Girth

Mid-thigh girth was significantly reduced at 24, 48 and 72 h post-exercise following CWI ($P<0.03$; Figure 3a) and CWT interventions ($P<0.01$; Figure 3c) compared to PAS (Table 1). However, HWI was not effective ($P>0.05$; Figure 3b) in reducing thigh volume compared to PAS.

Blood Variables

Significant reductions in [CK] were observed 24 ($P=0.03$) and 72 ($P=0.04$) h post-exercise following CWI, and 48 h ($P=0.04$) post-exercise following HWI when compared to PAS. However, none of the three hydrotherapy interventions influenced post-exercise changes of Mb, IL-6, or LDH.

Perceived Pain (VAS)

Perception of pain was only reduced at 24, 48, and 72 h post-exercise following CWT ($P<0.01$) compared to PAS (Figure 4c). Both CWI and HWI ($P>0.05$) were ineffective in reducing perceptions of pain following intense eccentric exercise (Figure 4a/b).
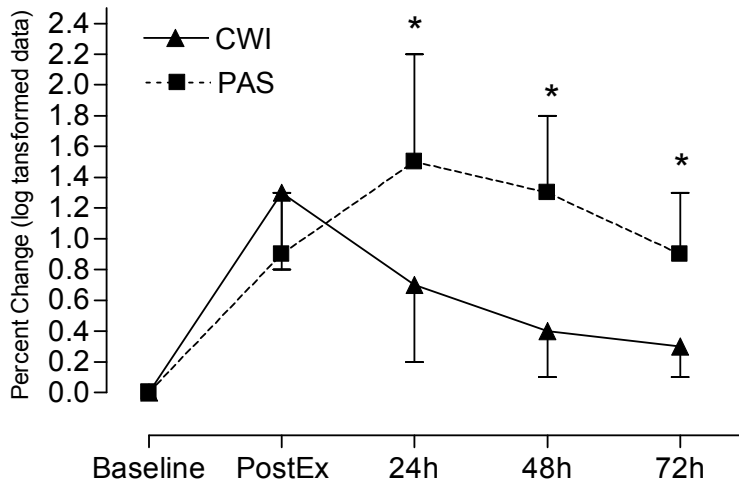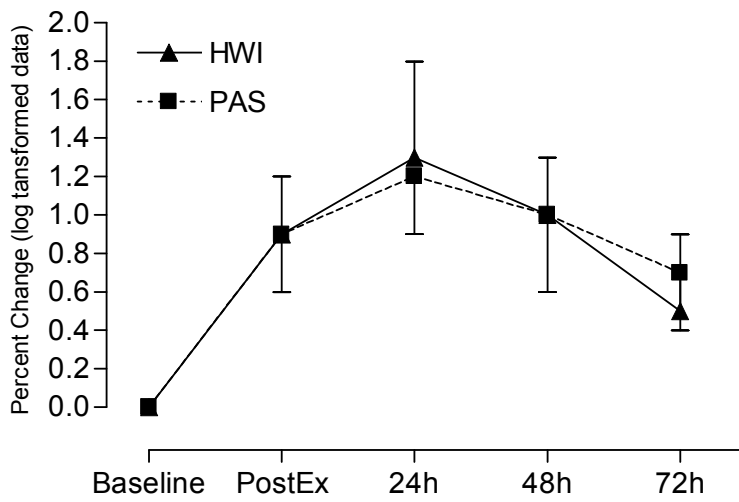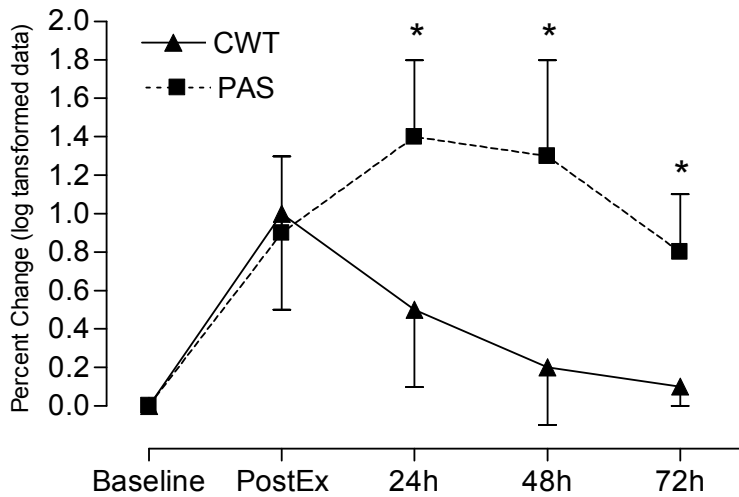
Figure 3. Percent change in mid-thigh circumference following CWI (3a), HWI (3b), and CWT (3c). Circumference was assessed pre and post muscle-damaging exercise as well as 24, 48, and 72 h post-exercise.

* Indicates a significant difference between hydrotherapy intervention and PAS.
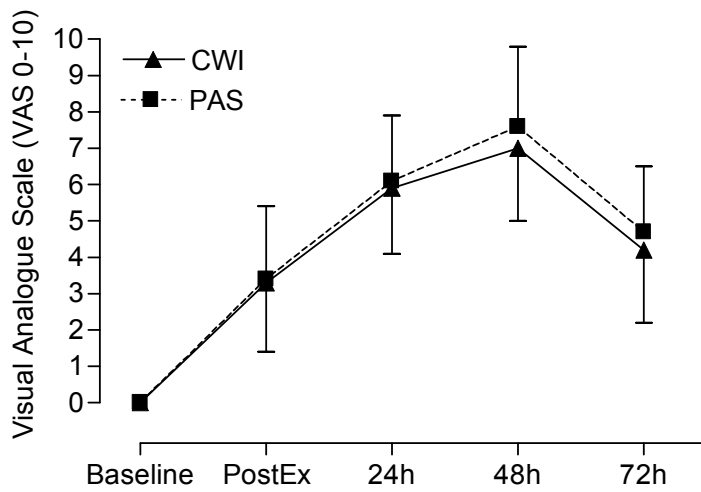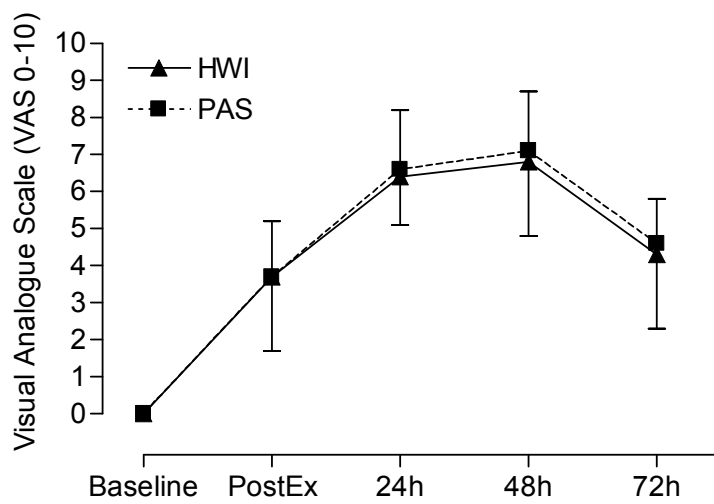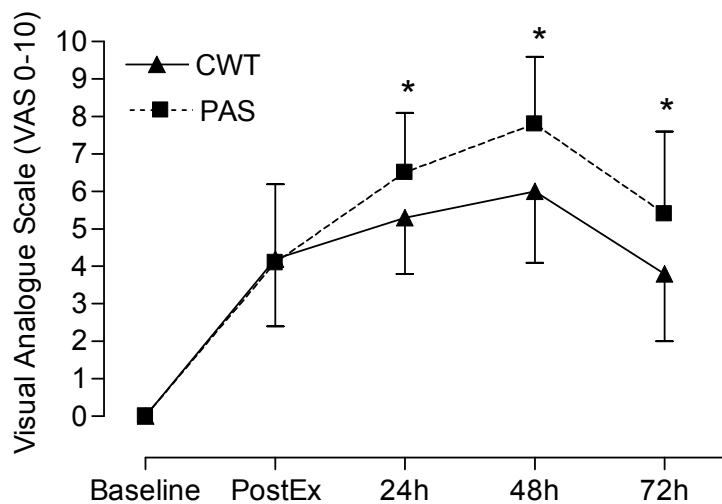
Figure 4. Perception of pain (CWI 4a), HWI (4b), and CWT (4c). The visual analogue scale was completed immediately post muscle-damaging exercise as well as 24, 48, and 72 h post-exercise.

* Indicates a significant difference between hydrotherapy intervention and PAS

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Discussion

The main findings of the present studies were that following DOMS-inducing exercise, all three hydrotherapy interventions (CWI, HWI, and CWT) improved the recovery of isometric force compared to PAS throughout the first 72 h post-exercise. However, compared to PAS, only CWI and CWT significantly enhanced the recovery of dynamic power (squat jump), while HWI appeared to have no effect on return of power, following a similar trend to PAS. In addition to enhancing the recovery of athletic performance, CWI and CWT (but not HWI) significantly reduced the degree of post-exercise swelling when compared to PAS.

To the authors knowledge, the present studies are the first to independently investigate three commonly prescribed post-exercise hydrotherapy interventions ensuring identical exercise mode and intensity, duration of water exposure and water temperature (CWI 15°C, HWI 38°C, CWT 38°C/15°C). The mechanism by which such interventions may be effective remains largely unknown. However, there are multiple theories surrounding the effectiveness of water immersion.

The effect of hydrostatic pressure exerted on the body during water immersion is becoming more defined. The compressive effect of immersion in water is thought to create a displacement of fluids from the periphery to the central cavity. This results in multiple physiological changes, including increases in substrate transport and cardiac output as well as a reduction in peripheral resistance (Hinghofer-Szalkay, Harrison, & Greenleaf, 1987; Wilcock *et al.*, 2006). Full body (head out) water immersion, as prescribed in the present

studies, has been shown to increase central blood volume (Hinghofer-Szalkay *et al.*, 1987; Johansen, Jensen, Pump, & Norsk, 1997; Wilcock *et al.*, 2006) and increase extracellular fluid volume via intracellular-intravascular osmotic gradients. Such changes may increase the removal of waste products with the potential of enhancing recovery from exercise. Although the present studies observed post-exercise increases in the blood markers analysed, the only post-exercise reductions observed between interventions was in CK response at 24 and 72 h post-exercise following CWI and 48 h post HWI compared to PAS. In the present study, compared to PAS, CWI and CWT were effective in reducing swelling of the thigh following muscle damaging exercise. This result indicates a possible increase in the re-absorption of interstitial fluid resulting in reduced oedema (Vaile *et al.*, 2007). Similar to the effects of compression garments (Bernhardt & Anderson, 2005; Doan *et al.*, 2003; Kraemer *et al.*, 2001), hydrostatic pressure has been shown to increase the pressure gradient between the interstitial compartment of the legs and the intravascular space (Wilcock *et al.*, 2006). In addition, the reduction of post-exercise oedema may not only improve the contractile functions within the muscle but also decrease the chances of secondary damage to the tissues that may result from cellular infiltration (Wilcock *et al.*, 2006). However, immersion in hot water did not have the same effect despite identical exposure time and water depth. Therefore, in addition to hydrostatic pressure, water temperature appears to play a role in overall recovery following damaging exercise.

The main physiological effects resulting from immersion in cold water appear to be localised vasoconstriction and decreased blood flow that may reduce oedema (Meeusen & Lievens, 1986). The effect of cold application though

various mediums has been shown to stimulate an analgesic effect, resulting in a decreased perception of pain (Cheung *et al.*, 2003; Meeusen & Lievens, 1986). While the results of the present study do not indicate an altered perception of pain compared to PAS, it must be noted that pain ratings were taken prior to immersion on each of the testing occasions. Therefore, while subjects may have experienced an acute analgesic effect immediately post-CWI, any such effect had diminished 24 h post-recovery.

Not surprisingly, immersion in hot water has been shown to demonstrate opposite physiological effects on the body; including an increase in blood flow, HR, and cardiac output, and a decrease in peripheral resistance (Wilcock *et al.*, 2006). Benefits such as decreased muscle spasm, stiffness and increased range of motion have also been reported following the application of heat (Kaul & Herring, 1994; Prentice, 1999). However, to the author's knowledge, no study has investigated the isolated effects of hot water immersion on the recovery of muscle damage in a controlled environment. The present study found HWI to be beneficial only through enhanced recovery of isometric force in comparison to PAS. When a specific movement (squat jump) was performed requiring dynamic power HWI did not appear to provide any improvement in return of performance to baseline levels. However, in comparison to PAS, CWT enhanced the recovery of both isometric force production and squat jump performance. The combined effects of alternating between hot and CWI appears to be more beneficial than when the interventions are prescribed as an isolated exposure. However, despite a growing body of knowledge, the physiological effects arising from CWT remain largely unknown. Contrast water therapy has been suggested to be an effective post-exercise intervention due to

increased lactate clearance (Cochrane, 2004), decreased oedema (Vaile *et al.*, 2007), increased blood flow (Cochrane, 2004), increased stimulation of the central nervous system and reduced metabolic rate (Coffey *et al.*, 2004; Hamlin, 2007; Vaile *et al.*, 2007). Myrer et al. (1994) and Higgins and Kaminski (1998) proposed one of the main effects of CWT to be a pumping action stimulated by vasodilation and vasoconstriction of the blood vessels. No study has observed any form of vasodilation or vasoconstriction during or following CWT (Higgins & Kaminski, 1998; Myrer *et al.*, 1994), however, this has only been assessed via intramuscular temperature measures, with results indicating no significant changes following various applications of CWT. Measures of blood flow using Doppler ultrasound (or similar) procedures may help to improve knowledge regarding the potential effects of CWT on muscle blood flow.

The present series of studies has contributed to the limited knowledge base investigating the effects of, and mechanisms underlying, three popular hydrotherapy interventions. The findings indicate CWI and CWT to be effective in minimising the physiological and functional deficits associated with DOMS, when compared to PAS. While HWI was effective in the recovery of isometric force, it was ineffective for recovery of all other markers compared to PAS.

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CHAPTER SIX

Thesis summary and future directions

6.1 Thesis Summary

The use of various post-exercise recovery interventions has become widespread and common amongst high-performance athletes. More specifically, the use of hydrotherapy techniques including cold water immersion, hot water immersion, and contrast water therapy are commonly prescribed and utilised post-exercise. However, there are limited research studies investigating the performance effect and potential mechanisms of such interventions. The findings of the majority of research conducted in the area of post-exercise recovery appear contradictory, in part, because numerous exercise tasks and recovery protocols have been implemented, thus limiting comparisons between investigations. Additionally, immersion temperatures, duration of exposure, and level of immersion have been inconsistently applied. Therefore, a series of studies were conducted in an attempt to further understand the performance effects of the aforementioned interventions, enable a comparison between interventions using controlled randomised crossover experimental designs, and investigate some of the possible mechanisms underlying their use.

A summary of findings from this thesis is presented in Table 1. Study one (Chapter Three) examined the effects of post-exercise cold water immersion compared to active recovery when performed between two exercise bouts in hot environmental conditions. Four full body (excluding head and neck) cold water immersion protocols were investigated, differing in either water temperature or exposure time. Subsequent performance was better maintained following all of the cold water immersion protocols. While no significant differences were observed between the cold water interventions, subsequent performance following an active recovery protocol was reduced by 4.1 ± 1.8% (mean ± SD).

However, post-recovery lactate was significantly lower (2.0 ± 0.8 mmol.L^{-1}) following active recovery compared to all cold water immersion protocols. It was concluded that subsequent performance was significantly enhanced when cold water immersion was utilised between two exercise bouts in the heat, compared to active recovery.

Study two (Chapter Four) investigated the effect of cold water immersion, hot water immersion, contrast water therapy and passive recovery (control) on the recovery of exercise-induced fatigue throughout a five day exercise period. Daily post-exercise cold water immersion and contrast water therapy were more effective in maintaining sprint and time trial performance across the five day period, compared to hot water immersion and passive recovery.

Study three (Chapter Five) examined the effect of cold water immersion, hot water immersion, contrast water therapy, and passive recovery on the recovery of the functional and physiological deficits associated with exercise-induced muscle damage (DOMS). In agreement with study two (Chapter Four), when compared to their independent control (passive recovery), cold water immersion, and contrast water therapy were more beneficial than hot water immersion or passive recovery in accelerating recovery from DOMS.

The collective results of the present thesis (Table 1) suggest cold water immersion (15°C water for 14 min) and contrast water therapy (38°C for one minute followed by 15°C for one minute repeated seven times) to be beneficial recovery interventions following muscle-damaging resistance exercise and fatigue-inducing cycling exercise. However, apart from return of isometric

strength, hot water immersion was no more beneficial in reducing recovery time or enhancing return of performance than passive recovery (control). In addition, cold water immersion protocols of varying temperature (10°C, 15°C, 20°C intermittent exposure and 20°C continuous exposure for 15 min) appear to be more beneficial than a 15 min active recovery (40% PPO) when implemented between two 30 min exercise bouts separated by one hour, performed in hot environmental conditions (34°C).

While it was difficult in the present series of studies to investigate all possible mechanisms of each individual intervention as well as establish changes in performance, some mechanisms associated with hydrotherapy treatment can be speculated upon given the current findings. For example, the effect of hydrostatic pressure has been reported to potentially be a significant contributor to the success of various hydrotherapy techniques. Interestingly, the results of the present studies, which implemented identical levels of immersion (full body) and duration, indicate that while hydrostatic pressure may play a role, water temperature must be an important contributing factor, considering the variations in findings between protocols utilising different temperatures. The present studies observed significant differences (i.e. improved return and maintenance of performance and a reduction in swelling) between cold water immersion and contrast water therapy, when compared to hot water immersion. Collectively, this series of studies attempted to ensure hydrostatic pressure was consistently applied in order to isolate possible water temperature effects. It is evident from current findings that water temperature is a critical component and therefore requires consideration when implementing post-exercise hydrotherapy interventions. Several of the physiological responses to and possible

mechanisms of hot water immersion, cold water immersion, and contrast water therapy, as previously acknowledged in the literature are presented in Table 2. Some of the proposed mechanisms appear to be supported by findings from this series of studies.

The present series of studies contribute to the limited knowledge base of controlled scientific research investigating the area of post-exercise recovery. An additional advantage of the present research is the use of controlled randomised crossover experimental designs. To the author's knowledge, no previous research in the recovery area has systematically attempted to compare the effects of cold water immersion, hot water immersion and contrast water therapy to each other and to an independent control. This is an important consideration, given the current lack of research into the area of contrast water therapy. It is hoped that the present research can improve understanding of the performance effects of such interventions and lead to a more holistic and mechanistic approach to future research.

Table 1. Summary of findings from the present thesis.

Variable	Hot Water Immersion	Cold Water Immersion	Contrast Water Therapy
Performance	Negative effect on subsequent cycling performance and return of performance following muscle-damage (squat jump). Beneficial effect on return of isometric strength.	Beneficial effect on subsequent cycling performance and return of performance following muscle-damage (isometric squat and squat jump)	Beneficial effect on subsequent cycling performance and return of performance following muscle-damage (isometric squat and squat jump)
Swelling	No effect on swelling	Beneficial effect, reduction of swelling	Beneficial effect, reduction of swelling
Core Body Temperature	Increase in core body temperature (above baseline)	Decrease in core body temperature (below baseline)	Stabilisation of core body temperature (baseline)
RPE	Did not affect perceptions of exertion	Did not affect perceptions of exertion In hot conditions, RPE was reduced at sub-maximal workloads	Did not affect perceptions of exertion
HR	No change in exercise HR 24 h post-immersion	No change in exercise HR 24 h post immersion	No change in exercise HR 24 h post-immersion
Thermal Sensation	N/A	Reduced perceptions of thermal strain during exercise	N/A
Blood Variables	Possible effect - reduction in CK 48 h post exercise. No effect for any other blood variable measured	Possible effect – reduction in CK 24 and 72 h post-exercise. No effect for any other blood variable measured	No effect

Table 2. Physiological responses to and possible mechanisms of hot water immersion, cold water immersion, and contrast water therapy.

Hot Water Immersion	Cold Water Immersion	Contrast Water Therapy
Increased blood flow (vasodilation) (Bonde-Petersen *et al.*, 1992)	Reduction in blood flow – potential to reduce acute inflammation (Barcroft & Edholm, 1943; Eston & Peters, 1999)	Enhanced circulation and blood flow (Cochrane, 2004; Wilcock *et al.*, 2006)
Increased cardiovascular strain (Nagasawa *et al.*, 2001).	Reduction in heart rate and cardiac output (Sramek *et al.*, 2000; Weston *et al.*, 1987)	Enhanced lactate removal (Hamlin, 2007; Wilcock *et al.*, 2006)
Decreased blood pressure due to rapid vasodilation (Wilcock *et al.*, 2006)	Increased arterial pressure and peripheral resistance (Bonde-Petersen *et al.*, 1992; Weston *et al.*, 1987)	Potential neurological recovery of the peripheral nervous system via reducing sympathetic activity (Cochrane, 2004)
Heat exposure may cause an inflammatory response and exacerbate swelling (Cote *et al.*, 1988; Magness, Garrett, & Erickson, 1970)		

6.2   Practical Applications

Current knowledge and understanding of hydrotherapy recovery interventions can be used to implement a recovery program. While it is acknowledged that further research is required to confirm such applications, the following recommendations are based on current scientific information.

- Where possible, full body immersion (excluding head and neck) should be implemented. More often than not exercise tasks involve the majority of the body; therefore, a full body recovery application is ideal. Partial immersion of the body (particularly in cold water) may limit changes and result in a redistribution of blood flow, therefore reducing some of the potential and proven benefits of water immersion. Additionally, partial immersion reduces the hydrostatic pressure exerted on the body and may reduce the effectiveness of the hydrotherapy intervention.

- Recovery interventions should aim to be practical and time efficient. Hydrotherapy interventions of 10-15 min duration appear to be effective.

- There is much conjecture regarding the optimal water temperature for various hydrotherapy protocols and little consistency between research investigations, often leading to contradictory findings. However, current knowledge suggests water temperatures of 10-15°C (cold) and 38-42°C (hot) to be effective. If athletes are performing a continuous cold water immersion protocol it is recommended to use a slightly warmer temperature (e.g. 15°C).   This is perceptually more comfortable (enhancing compliance), has been shown to effectively lower core body temperature, and enhance the recovery of performance in certain settings. However, if an athlete is performing an intermittent cold water

immersion protocol, a cooler temperature (e.g. 10-12°C) may be more effective given the shorter exposure time.

- An important outcome of hydrotherapy may be to reduce post-exercise core body temperature. Investigations into contrast water therapy have indicated that a 1:1 (hot:cold) ratio may be ideal in stabilising core temperature following exercise. In addition, isolated hot water immersion (e.g. spa 38-42°C) has been shown to increase core temperature; therefore it is currently recommended that protocols should avoid inclusion of more hot water exposure than cold water exposure.

- It is important to recognise individual responses to various recovery interventions. Not every athlete will respond in the same way, and this should be acknowledged, particularly in team sport environments where a group of athletes often perform the same recovery protocol, regardless of game time, position, physiological status, body mass and composition.

6.3   Future Research Directions

The findings of the present series of studies bring forth several considerations for future research. As identified throughout this thesis, there is a paucity of current scientific research in the area of post-exercise hydrotherapy. Specifically:

1. There is a lack of consistency in the administered protocols, such that while it is important for a variety of recovery protocols to be investigated, there should be some attempt to allow comparisons between past and future research. Currently, exercise protocols, recovery modes, water temperatures, durations and exposure level, as well as methods of determining recovery protocol effectiveness are widely varied between

studies, making meaningful comparisons difficult. Research should be conducted utilising similar exercise models incorporating exercise-induced fatigue or muscle-damage, whilst varying only the hydrotherapy intervention protocol. It is important to design studies that are compatible with regard to the levels of muscle damage or fatigue commonly experienced by athletes so that comparisons and practical applications of research data can be more easily made. Additionally, the use of hydrotherapy protocols should allow for comparisons of water temperature, duration, and exposure level when designing an investigation.

2. Future research should also attempt to establish the effect and importance of the timing of post-exercise recovery exposure (i.e. is immediately post-exercise more advantageous than one hour post-exercise?). The ideal timing of a specified recovery intervention could be assessed by using a randomised cross over experimental design (differing only in spacing of post-exercise recovery) and examining changes in post-exercise physiological responses and subsequent performance. The use of MRI and/or ultrasound could be valuable tools for assessing changes within the affected muscles.

3. In addition to determining the effectiveness of various recovery interventions, examining the potential mechanisms by which such interventions may or may not be effective should be undertaken. Possible mechanisms to be investigated include the effect of post-exercise hydrotherapy interventions on changes in blood flow, hormone

release, muscle oxygenation, tissue oedema, and skin, muscle, and core temperatures.

4. While the acute effects of the specified interventions have been investigated within this thesis, the effects of chronic long term exposure remains to be elucidated. Future research should examine the potential effects of repetitive long term use of hydrotherapy interventions by implementing a controlled training regime over a set period of time (e.g. five months), with selected recovery interventions employed at specific intervals across the study. The effect of the specified recovery intervention may be established via changes in performance across the selected training period, identifying any improvements or decrements in performance. Additionally, physiological variables such as those outlined above (point 3) could also be investigated.

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APPENDICES

Visual Analogue Scale

Perceived Soreness Questionnaire

Rate the intensity of soreness that you feel on the below scale where zero indicates no soreness (normal) and 10 indicates extreme soreness. Also, add any comments at the bottom of the page explaining the feeling of soreness that you are experiencing (if any).

10   Extremely Sore

9

8   Very Sore

7

6

5   Sore

4

3   Uncomfortable

2

1

0   Normal

Comments (general feelings and perceptions):

……………………………………………………………………………………………

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Thermal sensations scale

0.0	**Unbearably Cold**
1.0	**Very Cold**
2.0	**Cold**
3.0	**Cool**
4.0	**Comfortable**
5.0	**Warm**
6.0	**Hot**
7.0	**Very Hot**
8.0	**Unbearably Hot**

Informed Consent

Project Title:
Principal Researcher: Jo Vaile

This is to certify that I, _____ hereby agree to participate as a volunteer in a scientific investigation as an authorised part of the research program of the Australian Sports Commission under the supervision of Jo Vaile.

The investigation and my part in the investigation have been defined and fully explained to me and I understand the explanation. A copy of the procedures of this investigation and a description of any risks and discomforts has been provided to me and has been discussed in detail with me.

- I have been given an opportunity to ask whatever questions I may have had and all such questions and inquiries have been answered to my satisfaction.

- I understand that I am free to deny any answers to specific items or questions in interviews or questionnaires.

- I understand that I am free to withdraw consent and to discontinue participation in the project or activity at any time.

- I understand that any data or answers to questions will remain confidential with regard to my identity.

- I certify to the best of my knowledge and belief, I have no physical or mental illness or weakness that would increase the risk to me of participating in this investigation.

- I am participating in this project of my own free will and I have not been coerced in any way to participate.

Signature of Participant: _____ Date: ___/___/___

I, the undersigned, was present when the study was explained to the participant/s in detail and to the best of my knowledge and belief it was understood.

Signature of Researcher: _____ Date: ___/___/___

Raw data – Chapter Three

Performance (kJ)

Subject	10°C E1	10°C E2	15°C E1	15°C E2	20°C E1	20°C E2	20°C+ E1	20°C+ E2	ACT E1	ACT E2
1	510.5	513.2	515.6	525.2	520.8	517.7	525.0	521.5	521.2	509.0
2	425.7	421.4	415.6	429.8	437.1	425.9	436.5	426.9	443.0	415.7
3	524.3	519.5	519.7	525.4	512.2	522.9	525.7	531.0	530.8	510.5
4	509.4	492.9	508.1	504.1	514.4	503.4	507.2	494.5	502.9	483.0
5	436.0	438.0	443.7	436.7	438.1	434.9	442.3	432.5	450.8	445.9
6	575.9	576.3	562.8	558.8	556.6	547.5	576.6	572.7	552.9	523.9
7	440.4	441.6	439.4	437.4	436.3	430.4	437.8	439.7	439.0	428.3
8	533.7	526.9	540.7	531.8	542.9	535.3	543.8	538.0	550.7	520.4
9	540.0	532.2	545.0	545.8	540.3	534.7	537.0	535.7	535.3	510.8
10	488.0	492.1	486.9	504.4	496.6	501.4	489.0	496.9	504.0	475.1
Mean	498.4	495.4	497.8	499.9	499.5	495.4	502.1	498.9	503.1	482.3
SD	47.5	46.3	47.3	45.5	43.9	44.6	46.7	47.9	41.6	37.7

Mean Body Temperature (°C)

Raw Data		Trials								Effects								
Rec	Subject	0	15	30	50	90	105	120		15-0	50-0	90-0	15-105	30-50	30-90	30-120	50-90	90-105
ACT	1	36.5	37.6	38.5	37.9	36.9	37.6	38.4		1.1	1.4	0.4	0.0	0.6	1.7	0.1	1.0	-0.7
ACT	2	36.4	37.1	37.6	37.9	36.8	37.3	37.7		0.6	1.4	0.3	-0.2	-0.2	0.9	0.0	1.1	-0.5
ACT	3	36.4	37.2	38.2	38.2	37.0	37.6	38.3		0.8	1.8	0.6	-0.4	0.0	1.2	-0.2	1.2	-0.6
ACT	4	37.1	37.7	38.2	37.8	36.8	37.3	37.5		0.5	0.7	-0.3	0.4	0.4	1.4	0.7	1.0	-0.5
ACT	5	37.1	37.8	38.5	38.1	37.4	37.8	38.4		0.7	1.0	0.2	0.0	0.4	1.1	0.1	0.8	-0.4
ACT	6	36.4	37.5	38.6	38.7	37.5	37.9	38.7		1.1	2.3	1.1	-0.5	-0.1	1.1	-0.1	1.2	-0.4
ACT	7	36.9	38.1	38.9	38.7	37.2	38.0	39.0		1.2	1.8	0.4	0.0	0.2	1.7	0.0	1.5	-0.8
ACT	8	36.8	37.7	38.3	38.0	37.0	37.7	38.1		0.8	1.2	0.2	0.0	0.3	1.3	0.3	1.0	-0.6
ACT	9	36.9	37.8	38.2	37.6	37.0	37.9	38.4		0.9	0.7	0.1	-0.1	0.6	1.2	-0.2	0.6	-0.9
ACT	10	37.0	37.8	38.6	38.6	37.1	37.7	38.5		0.8	1.6	0.1	0.1	0.0	1.5	0.2	1.5	-0.5
	mean	36.8	37.6	38.4	38.2	37.1	37.7	38.3	mean	0.9	1.4	0.3	-0.1	0.2	1.3	0.1	1.1	-0.6
	SD	0.3	0.3	0.3	0.4	0.2	0.3	0.4	SD	0.2	0.5	0.4	0.2	0.3	0.3	0.2	0.3	0.2
10°C	1	36.8	38.0	38.8	34.9	36.0	37.2	38.1		1.1	-1.9	-0.8	0.8	3.9	2.8	0.6	-1.1	-1.2
10°C	2	36.3	37.2	37.8	33.7	35.5	36.7	37.3		0.9	-2.6	-0.8	0.5	4.1	2.3	0.5	-1.8	-1.2
10°C	3	36.2	36.8	37.6	35.6	36.0	36.5	37.2		0.6	-0.6	-0.1	0.3	2.0	1.6	0.4	-0.4	-0.5
10°C	4	36.3	37.3	37.8	34.1	35.6	36.5	37.3		0.9	-2.2	-0.7	0.7	3.7	2.1	0.4	-1.5	-0.9
10°C	5	36.8	36.8	37.4	35.4	36.3	37.1	37.8		0.0	-1.4	-0.5	-0.3	2.0	1.0	-0.5	-1.0	-0.7
10°C	6	36.5	37.2	38.1	34.8	36.2	36.9	37.7		0.7	-1.7	-0.3	0.3	3.3	1.9	0.4	-1.4	-0.7
10°C	7	37.0	38.2	38.8	34.6	35.6	36.9	38.1		1.1	-2.4	-1.5	1.2	4.1	3.2	0.6	-0.9	-1.3
10°C	8	36.6	37.6	38.3	34.5	35.8	36.9	37.6		1.0	-2.1	-0.8	0.8	3.7	2.5	0.7	-1.3	-1.1
10°C	9	37.0	37.5	38.0	34.1	35.6	36.7	37.3		0.5	-2.9	-1.4	0.7	3.9	2.4	0.6	-1.5	-1.1
10°C	10	37.0	37.9	38.6	34.7	35.8	37.2	38.4		0.9	-2.3	-1.2	0.6	3.9	2.8	0.2	-1.1	-1.4
	mean	36.6	37.4	38.1	34.6	35.8	36.9	37.7	mean	0.8	-2.0	-0.8	0.6	3.5	2.3	0.4	-1.2	-1.0
	SD	0.3	0.5	0.5	0.6	0.3	0.3	0.4	SD	0.3	0.7	0.4	0.4	0.8	0.6	0.3	0.4	0.3
15°C	1	37.0	37.9	39.0	35.0	36.3	37.3	38.1		0.9	-2.0	-0.7	0.6	4.0	2.7	0.9	-1.3	-1.0
15°C	2	37.1	37.9	38.2	35.0	36.1	37.3	37.9		0.8	-2.1	-1.0	0.6	3.2	2.1	0.3	-1.1	-1.2
15°C	3	36.3	37.4	38.1	36.3	36.4	37.3	38.0		1.0	0.0	0.1	0.1	1.9	1.7	0.1	-0.2	-0.9
15°C	4	37.0	37.5	38.3	35.5	35.4	36.8	37.5		0.5	-1.5	-1.6	0.7	2.8	2.9	0.8	0.1	-1.4
15°C	5	36.9	37.6	38.4	35.9	37.2	37.0	37.7		0.7	-1.0	0.3	0.5	2.5	1.2	0.7	-1.3	0.2
15°C	6	36.8	37.4	38.4	36.0	36.1	36.8	37.5		0.6	-0.8	-0.7	0.6	2.4	2.3	0.9	-0.1	-0.7
15°C	7	36.9	38.0	38.8	35.9	35.6	37.5	38.3		1.1	-1.0	-1.3	0.5	2.8	3.1	0.5	0.3	-1.8
15°C	8	36.7	37.4	38.1	36.1	36.3	36.9	37.7		0.7	-0.7	-0.4	0.5	2.1	1.8	0.5	-0.3	-0.6
15°C	9	36.7	37.4	37.9	34.6	35.3	36.6	37.3		0.7	-2.1	-1.4	0.9	3.3	2.6	0.6	-0.8	-1.2
15°C	10	37.3	38.0	38.7	34.7	35.6	37.7	38.8		0.7	-2.7	-1.8	0.3	4.0	3.1	-0.2	-0.9	-2.2
	mean	36.9	37.7	38.4	35.5	36.0	37.1	37.9	mean	0.8	-1.4	-0.8	0.5	2.9	2.3	0.5	-0.5	-1.1
	SD	0.3	0.3	0.3	0.6	0.6	0.4	0.4	SD	0.2	0.8	0.7	0.2	0.7	0.6	0.3	0.6	0.7

20°C	1	36.7	37.3	38.0	35.9	35.9	36.6	37.5		0.6	-0.8	-0.8	0.7	2.1	2.2	0.6	0.1	-0.8
20°C	2	36.4	37.4	38.0	36.5	36.1	37.2	37.7		0.9	0.0	-0.3	0.1	1.5	1.8	0.2	0.3	-1.1
20°C	3	36.3	37.5	38.3	36.0	36.5	37.3	38.2		1.1	-0.3	0.2	0.2	2.2	1.8	0.1	-0.5	-0.8
20°C	4	36.7	37.5	38.0	35.9	36.3	37.1	37.7		0.7	-0.9	-0.5	0.4	2.1	1.7	0.3	-0.4	-0.9
20°C	5	36.7	37.6	38.4	36.9	36.7	37.7	38.4		0.9	0.2	-0.1	-0.1	1.4	1.7	-0.1	0.3	-1.0
20°C	6	36.8	37.6	38.4	37.0	36.6	37.2	38.0		0.8	0.2	-0.2	0.3	1.4	1.9	0.5	0.4	-0.7
20°C	7	37.0	38.1	38.9	36.7	36.2	37.5	38.3		1.1	-0.3	-0.8	0.7	2.2	2.7	0.6	0.5	-1.2
20°C	8	36.9	38.1	38.8	36.8	36.5	37.3	37.9		1.2	-0.1	-0.5	0.8	2.1	2.4	0.9	0.3	-0.8
20°C	9	36.8	37.5	38.0	35.9	35.9	37.4	37.9		0.7	-0.9	-0.9	0.1	2.1	2.1	0.0	0.0	-1.5
20°C	10	37.3	37.8	38.6	37.3	36.6	36.4	38.6		0.5	0.0	-0.6	1.3	1.3	2.0	0.0	0.7	0.2
mean		36.8	37.6	38.3	36.5	36.3	37.2	38.0	mean	0.9	-0.3	-0.4	0.4	1.8	2.0	0.3	0.2	-0.9
SD		0.3	0.3	0.4	0.5	0.3	0.4	0.4	SD	0.2	0.4	0.4	0.4	0.4	0.3	0.3	0.4	0.5

20°C+	1	36.6	37.7	38.4	35.6	35.6	37.1	38.1		1.0	-1.0	-1.0	0.6	2.8	2.8	0.4	0.0	-1.5
20°C+	2	36.4	37.2	37.7	36.0	35.7	36.8	37.4		0.8	-0.4	-0.7	0.4	1.7	2.0	0.3	0.3	-1.1
20°C+	3	36.5	37.1	37.9	36.2	36.7	37.2	37.9		0.6	-0.3	0.2	0.0	1.7	1.2	0.0	-0.5	-0.4
20°C+	4	37.2	37.9	38.3	36.1	36.1	37.2	37.8		0.7	-1.1	-1.1	0.7	2.2	2.3	0.6	0.0	-1.1
20°C+	5	36.8	37.4	38.2	36.3	36.8	37.5	38.1		0.6	-0.5	-0.1	-0.1	1.9	1.4	0.1	-0.5	-0.7
20°C+	6	36.6	37.3	38.3	36.3	35.6	36.6	37.8		0.7	-0.3	-1.0	0.7	2.0	2.7	0.4	0.7	-1.0
20°C+	7	36.9	37.9	38.8	36.1	36.0	37.5	38.3		1.1	-0.7	-0.8	0.5	2.6	2.7	0.5	0.1	-1.5
20°C+	8	36.9	37.8	38.5	36.2	36.4	37.7	38.5		0.9	-0.8	-0.5	0.1	2.4	2.1	0.1	-0.3	-1.3
20°C+	9	36.8	37.6	38.2	36.0	36.3	37.1	37.8		0.8	-0.8	-0.5	0.5	2.2	1.9	0.4	-0.3	-0.8
20°C+	10	37.2	38.0	38.7	36.4	36.4	37.2	38.1		0.8	-0.9	-0.8	0.8	2.4	2.3	0.6	0.0	-0.8
mean		36.8	37.6	38.3	36.1	36.2	37.2	38.0	mean	0.8	-0.7	-0.6	0.4	2.2	2.1	0.3	0.0	-1.0
SD		0.3	0.3	0.3	0.2	0.4	0.3	0.3	SD	0.2	0.3	0.4	0.3	0.4	0.5	0.2	0.4	0.3

Blood Lactate (mmol.L^{-1})

Raw Data		Trials								Effects								
Rec	Subject	0	15	30	50	90	105	120		15-0	50-0	90-0	15-105	30-50	30-90	30-120	50-90	90-105
ACT	1	1.1	3.1	8.9	1.6	1.9	1.9	15.3		2.0	-7.8	7.3	-13.4	-6.4	-0.5	-6.4	-0.3	0.0
ACT	2	1.4	3.9	11.2	2.0	2.2	3.4	5.0		2.5	-9.8	9.2	-2.8	6.2	-0.6	6.2	-0.2	-1.2
ACT	3	1.2	4.7	9.2	1.6	2.2	3.4	7.0		3.5	-8.0	7.6	-4.8	2.2	-0.4	2.2	-0.6	-1.2
ACT	4	1.7	1.2	5.8	1.3	1.6	1.6	3.2		-0.5	-4.1	4.5	-1.6	2.6	0.4	2.6	-0.3	0.0
ACT	5	1.4	6.8	9.4	2.9	2.4	3.7	6.4		5.4	-8.0	6.5	-4.0	3.0	-1.5	3.0	0.5	-1.3
ACT	6	1.1	2.4	8.2	1.8	2.6	2.1	5.1		1.3	-7.1	6.4	-2.5	3.1	-0.7	3.1	-0.8	0.5
ACT	7	1.3	5.7	13.1	3.7	2.2	4.3	9.3		4.4	-11.8	9.4	-7.1	3.8	-2.4	3.8	1.5	-2.1
ACT	8	1.4	3.0	10.8	1.3	3.4	3.0	6.9		1.6	-9.4	9.5	-3.5	3.9	0.1	3.9	-2.1	0.4
ACT	9	1.3	3.9	11.3	1.7	2.1	3.3	6.6		2.6	-10.0	9.6	-4.5	4.7	-0.4	4.7	-0.4	-1.2
ACT	10	1.3	3.0	7.4	1.8	1.2	1.8	4.4		1.7	-6.1	5.6	-3.2	3.0	-0.5	3.0	0.6	-0.6
	mean	1.3	3.8	9.5	2.0	2.2	2.9	6.9	mean	2.5	-8.2	7.6	-4.7	2.6	-0.7	2.6	-0.2	-0.7
	SD	0.2	1.6	2.1	0.8	0.6	0.9	3.4	SD	1.7	2.2	1.8	3.4	3.4	0.8	3.4	1.0	0.9
10°C	1	1.1	3.8	8.4	2.9	3.0	3.6	5.9		2.7	-7.3	5.5	-2.9	2.5	-1.8	2.5	-0.1	-0.6
10°C	2	1.4	6.3	10.9	3.7	3.2	5.2	7.8		4.9	-9.5	7.2	-4.6	3.1	-2.3	3.1	0.5	-2.0
10°C	3	0.9	4.0	8.9	4.4	2.4	3.0	8.1		3.1	-8.0	4.5	-5.7	0.8	-3.5	0.8	2.0	-0.6
10°C	4	1.9	1.4	8.8	2.9	1.8	1.6	4.0		-0.5	-6.9	5.9	-2.2	4.8	-1.0	4.8	1.1	0.2
10°C	5	1.7	7.8	11.2	4.4	3.1	5.7	9.3		6.1	-9.5	6.8	-6.2	1.9	-2.7	1.9	1.3	-2.6
10°C	6	1.4	2.9	12.7	2.4	2.6	2.4	12.2		1.5	-11.3	10.3	-9.6	0.5	-1.0	0.5	-0.2	0.2
10°C	7	1.0	4.7	11.0	4.1	2.2	4.3	10.3		3.7	-10.0	6.9	-8.1	0.7	-3.1	0.7	1.9	-2.1
10°C	8	1.4	3.4	9.0	4.1	3.0	3.4	7.7		2.0	-7.6	4.9	-4.7	1.3	-2.7	1.3	1.1	-0.4
10°C	9	1.1	3.3	10.1	4.6	1.8	2.8	8.7		2.2	-9.0	5.5	-6.9	1.4	-3.5	1.4	2.8	-1.0
10°C	10	1.4	3.2	6.1	4.9	1.8	2.2	7.6		1.8	-4.7	1.2	-5.8	-1.5	-3.5	-1.5	3.1	-0.4
	mean	1.3	4.1	9.7	3.8	2.5	3.4	8.2	mean	2.8	-8.4	5.9	-5.7	1.6	-2.5	1.6	1.4	-0.9
	SD	0.3	1.8	1.9	0.8	0.6	1.3	2.3	SD	1.8	1.9	2.3	2.2	1.7	1.0	1.7	1.1	1.0
15°C	1	1.1	3.9	9.6	3.9	2.8	3.8	8.3		2.8	-8.5	5.7	-5.5	1.3	-2.8	1.3	1.1	-1.0
15°C	2	1.7	4.7	9.2	2.0	2.6	3.4	9.0		3.0	-7.5	7.2	-6.4	0.2	-0.3	0.2	-0.6	-0.8
15°C	3	2.0	5.2	9.3	2.8	2.0	3.8	8.9		3.2	-7.3	6.5	-6.9	0.4	-0.8	0.4	0.8	-1.8
15°C	4	1.7	1.8	8.4	2.7	2.1	1.7	6.2		0.1	-6.7	5.7	-4.1	2.2	-1.0	2.2	0.6	0.4
15°C	5	1.6	7.6	11.3	3.6	2.8	5.2	5.3		6.0	-9.7	7.7	-2.5	6.0	-2.0	6.0	0.8	-2.4
15°C	6	1.4	4.0	11.0	3.4	2.7	3.8	11.1		2.6	-9.6	7.6	-8.4	-0.1	-2.0	-0.1	0.7	-1.1
15°C	7	0.9	5.3	12.6	4.0	2.2	4.1	10.0		4.4	-11.7	8.6	-7.8	2.6	-3.1	2.6	1.8	-1.9
15°C	8	1.9	3.9	9.9	3.6	2.6	3.0	10.3		2.0	-8.0	6.3	-7.7	-0.4	-1.7	-0.4	1.0	-0.4
15°C	9	1.2	2.8	10.8	4.2	1.9	2.3	9.0		1.6	-9.6	6.6	-7.1	1.8	-3.0	1.8	2.3	-0.4
15°C	10	1.1	4.0	8.1	3.4	2.0	2.3	7.0		2.9	-7.0	4.7	-5.0	1.1	-2.3	1.1	1.4	-0.3
	mean	1.5	4.3	10.0	3.4	2.4	3.3	8.5	mean	2.9	-8.6	6.7	-6.1	1.5	-1.9	1.5	1.0	-1.0
	SD	0.4	1.6	1.4	0.7	0.4	1.0	1.8	SD	1.6	1.6	1.1	1.9	1.9	1.0	1.9	0.8	0.9
20°C	1	0.9	5.7	13.1	2.8	2.8	4.0	8.6		4.8	-12.2	10.3	-5.8	4.5	-1.9	4.5	0.0	-1.2
20°C	2	1.4	5.4	10.4	2.8	2.0	3.9	7.3		4.0	-9.0	7.6	-5.3	3.1	-1.4	3.1	0.8	-1.9
20°C	3	1.1	5.2	8.0	2.1	2.3	3.6	8.1		4.1	-6.9	5.9	-5.8	-0.1	-1.0	-0.1	-0.2	-1.3
20°C	4	1.6	1.3	6.2	3.0	1.8	2.1	4.1		-0.3	-4.6	3.2	-2.3	2.1	-1.4	2.1	1.2	-0.3
20°C	5	1.4	6.0	8.2	4.3	2.9	3.1	4.8		4.6	-6.8	3.9	-1.9	3.4	-2.9	3.4	1.4	-0.2
20°C	6	1.6	3.8	10.8	3.1	2.2	3.0	7.4		2.2	-9.2	7.7	-5.2	3.4	-1.5	3.4	0.9	-0.8
20°C	7	1.3	5.1	11.1	2.3	2.6	3.8	9.8		3.8	-9.8	8.8	-7.2	1.3	-1.0	1.3	-0.3	-1.2
20°C	8	1.9	2.8	9.3	4.0	2.7	2.4	7.6		0.9	-7.4	5.3	-4.9	1.7	-2.1	1.7	1.3	0.3
20°C	9	1.1	3.9	9.6	2.7	1.2	2.3	7.0		2.8	-8.5	6.9	-5.8	2.6	-1.6	2.6	1.5	-1.1
20°C	10	1.2	3.9	10.2	3.3	1.8	2.4	7.9		2.7	-9.0	6.9	-6.1	2.3	-2.1	2.3	1.5	-0.6
	mean	1.4	4.3	9.7	3.0	2.2	3.1	7.3	mean	3.0	-8.3	6.7	-5.0	2.4	-1.7	2.4	0.8	-0.8
	SD	0.3	1.5	1.9	0.7	0.5	0.7	1.7	SD	1.7	2.1	2.2	1.7	1.3	0.6	1.3	0.7	0.6

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20°C +	1	1.2	3.1	12.6	2.9	3.3	2.9	11.6		1.9	-11.4	9.7	-8.3	1.0	-1.7	1.0	-0.4	0.4
20°C +	2	1.6	5.3	10.4	3.2	2.3	4.4	7.5		3.7	-8.8	7.2	-5.2	2.9	-1.6	2.9	0.9	-2.1
20°C +	3	1.2	5.6	11.1	4.0	2.7	4.6	11.1		4.4	-9.9	7.1	-8.4	0.0	-2.8	0.0	1.3	-1.9
20°C +	4	1.4	1.6	8.7	3.4	1.9	1.6	4.6		0.2	-7.3	5.3	-2.7	4.1	-2.0	4.1	1.5	0.3
20°C +	5	1.4	4.8	6.7	3.3	2.2	2.6	4.9		3.4	-5.3	3.4	-2.7	1.8	-1.9	1.8	1.1	-0.4
20°C +	6	1.3	2.9	12.6	3.7	2.6	2.4	11.1		1.6	-11.3	8.9	-8.5	1.5	-2.4	1.5	1.1	0.2
20°C +	7	1.2	5.7	12.2	3.7	1.9	4.4	9.9		4.5	-11.0	8.5	-8.0	2.3	-2.5	2.3	1.8	-2.5
20°C +	8	1.4	3.6	9.8	3.9	1.9	3.3	10.3		2.2	-8.4	5.9	-8.4	-0.5	-2.5	-0.5	2.0	-1.4
20°C +	9	1.6	4.0	10.1	4.0	1.9	2.9	7.9		2.4	-8.5	6.1	-6.0	2.2	-2.4	2.2	2.1	-1.0
20°C +	10	0.8	2.7	6.8	3.1	1.6	1.4	6.7		1.9	-6.0	3.7	-5.1	0.1	-2.3	0.1	1.5	0.2
	mean	1.3	3.9	10.1	3.5	2.2	3.1	8.6	mean	2.6	-8.8	6.6	-6.3	1.5	-2.2	1.5	1.3	-0.8
	SD	0.2	1.4	2.2	0.4	0.5	1.1	2.6	SD	1.4	2.2	2.1	2.3	1.4	0.4	1.4	0.7	1.1

Heart Rate (bpm)

Raw Data		Trials								Effects		90-0	15-105	30-50	30-90	30-120	50-90	90-105
Rec	Subject	0	15	30	50	90	105	120		15-0	50-0							
ACT	1	69	168	191	137	91	165	189		99	68	22	3	54	100	2	46	-74
ACT	2	70	154	185	122	99	163	179		84	52	29	-9	63	86	6	23	-64
ACT	3	69	163	180	130	82	165	180		94	61	13	-2	50	98	0	48	-83
ACT	4	64	149	183	116	70	153	175		85	52	6	-4	67	113	8	46	-83
ACT	5	88	164	172	122	106	164	172		76	34	18	0	50	66	0	16	-58
ACT	6	79	161	185	136	93	169	187		82	57	14	-8	49	92	-2	43	-76
ACT	7	71	164	178	132	84	173	187		93	61	13	-9	46	94	-9	48	-89
ACT	8	67	156	178	123	73	160	176		89	56	6	-4	55	105	2	50	-87
ACT	9	76	171	185	130	80	170	179		95	54	4	1	55	105	6	50	-90
ACT	10	71	165	178	127	92	166	175		94	56	21	-1	51	86	3	35	-74
	mean	72	162	182	128	87	165	180	mean	89	55	15	-3	54	95	2	41	-78
	SD	7	7	5	7	11	6	6	SD	7	9	8	4	7	13	5	12	11
10°C	1	68	175	182	95	105	165	186		107	27	37	10	87	77	-4	-10	-60
10°C	2	65	156	176	98	84	155	176		91	33	19	1	78	92	0	14	-71
10°C	3	68	155	178	71	74	160	179		87	3	6	-5	107	104	-1	-3	-86
10°C	4	65	158	181	75	74	153	176		93	10	9	5	106	107	5	1	-79
10°C	5	98	166	173	88	74	158	175		68	-10	-24	8	85	99	-2	14	-84
10°C	6	67	152	182	88	76	151	185		85	21	9	1	94	106	-3	12	-75
10°C	7	72	165	187	72	60	167	186		93	0	-12	-2	115	127	1	12	-107
10°C	8	74	171	185	104	64	165	185		97	30	-10	6	81	121	0	40	-101
10°C	9	70	172	190	74	63	171	187		102	4	-7	1	116	127	3	11	-108
10°C	10	69	164	179	94	65	164	180		95	25	-4	0	85	114	-1	29	-99
	mean	72	163	181	86	74	161	182	mean	92	14	2	3	95	107	0	12	-87
	SD	10	8	5	12	13	7	5	SD	11	15	18	5	14	16	3	15	16
15°C	1	68	169	183	84	66	173	183		101	16	-2	-4	99	117	0	18	-107
15°C	2	69	154	169	74	71	154	176		85	5	2	0	95	98	-7	3	-83
15°C	3	71	161	178	72	69	165	173		90	1	-2	-4	106	109	5	3	-96
15°C	4	61	158	188	70	57	156	186		97	9	-4	2	118	131	2	13	-99
15°C	5	85	167	177	82	88	166	176		82	-3	3	1	95	89	1	-6	-78
15°C	6	69	161	182	91	66	157	184		92	22	-3	4	91	116	-2	25	-91
15°C	7	85	162	187	72	61	160	186		77	-13	-24	2	115	126	1	11	-99
15°C	8	69	167	184	81	66	162	184		98	12	-3	5	103	118	0	15	-96
15°C	9	62	172	193	78	69	170	190		110	16	7	2	115	124	3	9	-101
15°C	10	66	169	185	87	72	164	180		103	21	6	5	98	113	5	15	-92
	mean	71	164	183	80	69	163	182	mean	94	9	-2	1	102	114	1	10	-94
	SD	8	6	7	7	8	6	5	SD	10	12	9	3	9	13	4	9	9
20°C	1	67	163	185	89	82	161	178		96	22	15	2	96	103	7	7	-79
20°C	2	61	158	183	90	77	163	183		97	29	16	-5	93	106	0	13	-86
20°C	3	74	170	176	74	87	167	181		96	0	13	3	102	89	-5	-13	-80
20°C	4	65	149	182	62	80	155	183		84	-3	15	-6	120	102	-1	-18	-75
20°C	5	72	169	176	91	86	170	185		97	19	14	-1	85	90	-9	5	-84
20°C	6	76	157	178	99	82	154	177		81	23	6	3	79	96	1	17	-72
20°C	7	63	168	184	74	67	168	183		105	11	4	0	110	117	1	7	-101
20°C	8	64	167	182	63	78	160	183		103	-1	14	7	119	104	-1	-15	-82
20°C	9	70	175	192	82	82	176	192		105	12	12	-1	110	110	0	0	-94
20°C	10	70	168	178	82	74	163	177		98	12	4	5	96	104	1	8	-89
	mean	68	164	182	81	80	164	182	mean	96	12	11	1	101	102	-1	1	-84
	SD	5	8	5	12	6	7	4	SD	8	11	5	4	14	9	4	12	9
20°C+	1	57	167	187	80	69	163	187		110	23	12	4	107	118	0	11	-94
20°C+	2	71	159	180	74	64	161	181		88	3	-7	-2	106	116	-1	10	-97
20°C+	3	82	164	179	70	84	169	182		82	-12	2	-5	109	95	-3	-14	-85
20°C+	4	69	158	186	69	71	159	181		89	0	2	-1	117	115	5	-2	-88
20°C+	5	94	172	182	95	82	165	174		78	1	-12	7	87	100	8	13	-83
20°C+	6	75	156	183	88	74	154	183		81	13	-1	2	95	109	0	14	-80
20°C+	7	71	166	188	77	60	170	188		95	6	-11	-4	111	128	0	17	-110
20°C+	8	71	166	181	84	68	164	183		95	13	-3	2	97	113	-2	16	-96
20°C+	9	70	173	187	80	66	175	187		103	10	-4	-2	107	121	0	14	-109
20°C+	10	71	163	180	93	70	159	177		92	22	-1	4	87	110	3	23	-89
	mean	73	164	183	81	71	164	182	mean	91	8	-2	1	102	113	1	10	-93
	SD	10	6	3	9	8	6	4	SD	10	11	7	4	10	10	3	11	10

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Thermal Sensations Scale
(0 = unbearably cold, 10 = unbearably hot)

Raw Data		Trials								Effects								
Rec	Subject	0	15	30	50	90	105	120		15-0	50-0	90-0	15-105	30-50	30-90	30-120	50-90	90-105
ACT	1	4	6	8	5	5	6	8		2	1	1	0	-1	3	0	0	-1
ACT	2	4	5	6	4	4	6	7		1	0	0	-1	0	2	-1	0	-2
ACT	3	5	7	8	7	6	8	8		2	2	1	-1	-2	2	0	2	-2
ACT	4	4	6	7	4	4	6	7		2	0	0	0	0	3	1	0	-2
ACT	5	5	6	7	5	6	7	7		1	0	1	-1	0	1	0	-1	-1
ACT	6	5	6	6	5	5	5	7		1	1	1	1	-1	1	-1	0	0
ACT	7	6	6	7	5	6	6	7		0	-2	0	0	2	1	0	-2	0
ACT	8	5	5	6	5	6	6	6		0	-1	1	-1	1	-1	-1	-2	1
ACT	9	5	7	8	6	6	8	8		2	1	1	-1	-1	2	0	0	-2
ACT	10	5	6	7	4	4	7	8		1	-1	-1	-1	3	3	-1	0	-3
	mean	5	6	7	5	5	6	7	mean	1	0	0	0	0	2	0	0	-1
	SD	1	1	1	1	1	1	1	SD	1	1	1	0	1	1	0	1	1
10°C	1	4	5	6	2	4	5	6		1	-3	0	0	3	2	0	-3	-1
10°C	2	4	5	7	2	4	5	6		1	-3	-1	0	3	4	2	-2	-2
10°C	3	5	7	8	3	4	7	8		2	-3	-2	-1	3	4	0	-1	-4
10°C	4	4	6	7	0	3	6	6		2	-4	-1	-1	4	4	1	-3	-3
10°C	5	5	6	7	1	5	7	7		1	-4	0	-1	4	2	-1	-4	-2
10°C	6	4	5	6	1	4	5	6		1	-3	0	0	3	2	1	-3	-1
10°C	7	6	6	6	1	3	6	6		1	-5	-3	1	5	3	0	-2	-3
10°C	8	5	6	6	1	3	5	4		1	-4	-2	2	4	3	2	-2	-2
10°C	9	5	7	8	2	3	7	8		2	-3	-2	0	3	5	0	-1	-4
10°C	10	4	6	7	1	4	6	7		2	-4	0	0	4	3	0	-4	-2
	mean	5	6	7	1	4	6	6	mean	1	-3	-1	0	3	3	0	-2	-2
	SD	1	1	1	1	1	1	1	SD	0	1	1	1	1	1	1	1	1
15°C	1	4	6	2	4	4	5	6		2	0	0	2	0	-2	-4	0	-1
15°C	2	4	5	6	1	4	5	5		2	-3	1	1	3	2	2	-4	-1
15°C	3	5	8	8	5	5	7	8		3	0	0	1	0	4	1	0	-2
15°C	4	5	6	7	2	4	6	7		1	-4	-1	0	4	3	-1	-3	-2
15°C	5	5	6	7	2	5	7	7		1	-4	0	-1	4	2	0	-4	-2
15°C	6	5	5	6	2	4	5	6		1	-3	-1	0	3	2	1	-2	-1
15°C	7	6	7	7	2	4	6	6		1	-4	-2	1	4	3	1	-2	-2
15°C	8	5	5	5	2	4	6	5		0	-3	-1	-1	3	1	0	-2	-2
15°C	9	5	7	8	3	4	6	8		2	-2	-1	1	2	4	0	-1	-3
15°C	10	5	7	8	1	4	6	7		2	-5	-1	1	5	4	1	-4	-2
	mean	5	6	6	2	4	6	6	mean	1	-3	-1	0	3	2	0	-2	-2
	SD	1	1	2	1	0	1	1	SD	1	2	1	1	2	2	2	1	1
20°C	1	4	5	7	2	4	5	7		1	-2	0	0	2	3	0	-2	-1
20°C	2	4	5	6	2	4	6	6		1	-2	0	-1	2	2	0	-2	-2
20°C	3	5	6	7	3	4	7	8		2	-2	-1	-1	2	3	-1	-1	-3
20°C	4	4	6	7	4	4	6	6		2	0	0	0	0	3	1	0	-2
20°C	5	5	6	7	3	6	6	7		1	-2	1	0	2	2	1	-3	-1
20°C	6	4	6	7	4	4	5	6		2	0	0	1	0	3	1	0	-1
20°C	7	6	6	7	3	4	6	6		0	-4	-3	1	4	4	1	-1	-2
20°C	8	5	5	6	4	4	6	5		0	-1	-1	-1	1	2	1	0	-2
20°C	9	5	7	8	3	5	7	8		2	-3	0	0	3	3	0	-3	-2
20°C	10	5	6	7	2	4	6	7		1	-3	-1	0	3	3	0	-2	-2
	mean	5	6	7	3	4	6	6	mean	1	-2	0	0	2	2	0	-1	-2
	SD	1	1	1	1	1	1	1	SD	1	1	1	0	1	1	0	1	1
20°C+	1	4	5	7	2	4	5	7		1	-2	0	0	2	3	0	-2	-1
20°C+	2	4	6	7	2	4	6	7		2	-3	0	0	3	3	1	-3	-2
20°C+	3	5	7	7	2	5	7	8		2	-3	0	-1	3	2	-1	-3	-2
20°C+	4	5	6	7	1	4	6	7		2	-4	-1	0	4	3	0	-3	-2
20°C+	5	5	6	7	2	6	6	7		1	-4	1	0	4	2	0	-4	-1
20°C+	6	5	6	6	3	4	6	6		1	-2	-1	0	2	2	0	-2	-2
20°C+	7	6	6	7	1	4	6	6		1	-5	-2	1	5	3	1	-3	-2
20°C+	8	5	6	7	2	5	5	5		1	-3	0	1	3	2	2	-3	-1
20°C+	9	5	7	8	1	5	7	8		2	-5	0	1	5	3	0	-5	-2
20°C+	10	5	6	7	1	5	6	6		1	-4	-1	1	4	2	1	-4	-1
	mean	5	6	7	2	4	6	7	mean	1	-3	0	0	3	2	0	-3	-1
	SD	0	1	0	1	1	1	1	SD	0	1	1	0	1	1	1	1	1

Rating of Perceived Exertion
(6 = no exertion at all, 20 = maximal exertion)

Raw Data		Trials							Effects					
Rec	Subject	0	15	30	90	105	120		15-0	90-0	15-105	30-120	105-90	120-105
ACT	1	13	15	20	15	15	20		2	2	0	0	0	-5
ACT	2	9	13	20	10	15	19		4	1	-6	1	-5	-4
ACT	3	13	15	20	13	16	20		2	0	-5	0	-3	-4
ACT	4	12	15	19	13	16	20		3	1	-5	-1	-3	-4
ACT	5	13	15	19	13	16	19		2	0	-4	0	-3	-3
ACT	6	13	13	20	13	14	20		0	0	-7	0	-1	-6
ACT	7	13	15	20	15	17	20		2	2	-5	0	-2	-3
ACT	8	9	12	19.5	11	15	20		3	2	-8	-0.5	-4	-5
ACT	9	14	17	19	16	19	20		3	2	-3	-1	-3	-1
ACT	10	13	15	19	13	17	19		2	0	-4	0	-4	-2
	mean	12	15	20	13	16	20	mean	2	1	-5	0	-3	-4
	SD	2	1	1	2	1	1	SD	1	1	2	1	2	2
10°C	1	12	15	18	13	15	18		3	1	0	0	-2	-3
10°C	2	9	13	19	10	13	19		4	1	0	0	-3	-6
10°C	3	12	14.5	20	13	17	20		2.5	1	-2.5	0	-4	-3
10°C	4	10	13	20	11	15	19		3	1	-2	1	-4	-4
10°C	5	13	15	19	13	17	19		2	0	-2	0	-4	-2
10°C	6	12	13	20	12	13	20		1	0	0	0	-1	-7
10°C	7	13	14	20	15	14	20		1	2	0	0	1	-6
10°C	8	9	13	19	10	14	19		4	1	-1	0	-4	-5
10°C	9	12	17	19	12	17	20		5	0	0	-1	-5	-3
10°C	10	13	15	19	12	14	18		2	-1	1	1	-2	-4
	mean	12	14	19	12	15	20	mean	3	1	-1	0	-3	-4
	SD	2	1	1	2	2	1	SD	1	1	1	1	2	2
15°C	1	13	14	19	12	14	19		1	-1	1	0	7	5
15°C	2	8	13	17	12	14	17		5	4	6	-1	5	3
15°C	3	12	14	19.5	12	15.5	20		2	0	3.5	-1.5	7.5	4
15°C	4	12	15	20	13	15	20		3	1	3	0	7	5
15°C	5	12	16	19	12	16	19		4	0	4	0	7	3
15°C	6	12	12	20	11	13	20		0	-1	1	-1	9	7
15°C	7	14	14	20	16	15	20		0	2	1	-1	4	5
15°C	8	8	12	20	9	14	19		4	1	6	-2	11	6
15°C	9	11	16	19	12	16	20		5	1	5	0	7	3
15°C	10	13	15	19	12	15	17		2	-1	2	0	7	4
	mean	12	14	19	12	15	19	mean	3	1	3	-1	7	5
	SD	2	1	1	2	1	1	SD	2	2	2	1	2	1
20°C	1	13	14	20	13	14	20		1	0	1	0	7	6
20°C	2	9	13	19	12	15	19		4	3	6	-2	7	4
20°C	3	11	14	19	11	15	19.5		3	0	4	-1	8	4
20°C	4	11	14	20	11	16	20		3	0	5	-2	9	4
20°C	5	12	16	19	13	16	19		4	1	4	0	6	3
20°C	6	12	13	19	12	12	18		1	0	0	1	7	7
20°C	7	14	15	20	16	17	20		1	2	3	-2	4	3
20°C	8	9	12	19	12	14	19		3	3	5	-2	7	5
20°C	9	13	17	19	14	17	19		4	1	4	0	5	2
20°C	10	13	14	19	13	14	17		1	0	1	0	6	5
	mean	12	14	19	13	15	19	mean	3	1	3	-1	7	4
	SD	2	2	1	2	2	1	SD	1	1	2	1	1	2
20°C+	1	13	14	20	11	13	20		1	-2	0	1	9	7
20°C+	2	9	14	19	11	14	20		5	2	5	0	8	5
20°C+	3	13	13	19.5	11	15	20		0	-2	2	-2	8.5	4.5
20°C+	4	11	15	20	13	15	20		4	2	4	0	7	5
20°C+	5	13	16	19	12	16	19		3	-1	3	0	7	3
20°C+	6	13	13	20	13	13	20		0	0	0	0	7	7
20°C+	7	14	15	20	16	15	20		1	2	1	0	4	5
20°C+	8	9	12	17	9	13	19		3	0	4	-1	8	4
20°C+	9	12	17	19	12	17	19		5	0	5	0	7	2
20°C+	10	11	15	18	11	14	18		4	0	3	1	7	4
	mean	12	14	19	12	15	20	mean	3	0	3	0	7	5
	SD	2	2	1	2	1	1	SD	2	2	2	1	1	2

Raw data – Chapter Four

Sprint Performance (W)

Raw Data		Trials (Days 1-5)						Effects			
Rec	Subject	1	2	3	4	5		1-2	1-3	1-4	1-5
PAS	1	499.2	495.8	508.0	486.1	499.9		-3.3	8.8	-13.1	0.8
PAS	2	600.0	603.6	611.7	604.5	606.1		3.6	11.7	4.5	6.1
PAS	3	667.6	653.9	670.1	670.8	569.0		-13.7	2.5	3.2	-98.6
PAS	4	480.0	481.1	487.6	491.9	443.7		1.1	7.6	11.9	-36.3
PAS	5	519.4	420.6	430.1	426.6	480.4		-98.8	-89.3	-92.8	-39.0
PAS	6	657.7	720.4	681.9	626.2	626.2		62.8	24.2	-31.5	-31.5
PAS	7	618.3	619.1	598.6	573.5	602.0		0.8	-19.6	-44.7	-16.3
PAS	8	607.0	605.0	598.0	595.0	601.0		-2.0	-9.0	-12.0	-6.0
PAS	9	555.0	520.0	535.0	543.0	548.0		-35.0	-20.0	-12.0	-7.0
PAS	10	651.2	648.7	640.6	535.8	631.0		-2.4	-10.6	-115.3	-20.2
PAS	11	525.0	509.3	525.0	529.1	520.0		-15.7	0.0	4.1	-5.0
PAS	12	675.0	673.8	672.5	640.5	643.3		-1.2	-2.5	-34.5	-31.7
	mean	587.9	579.3	579.9	560.3	564.2	mean	-8.7	-8.0	-27.7	-23.7
	SD	69.7	91.6	81.8	71.5	65.4	SD	36.3	28.7	39.9	28.0
CWT	1	464.1	476.2	478.7	493.6	469.3		12.1	14.6	29.5	5.1
CWT	2	623.3	621.3	616.8	624.5	620.7		-2.0	-6.4	1.2	-2.5
CWT	3	675.0	686.7	680.0	685.5	677.6		11.7	5.0	10.5	2.6
CWT	4	478.6	517.2	519.4	480.5	433.5		38.6	40.8	1.9	-45.1
CWT	5	611.1	592.4	574.7	581.0	599.2		-18.8	-36.4	-30.2	-12.0
CWT	6	630.0	628.0	643.4	638.9	628.0		-2.0	13.4	8.9	-2.0
CWT	7	593.5	576.8	587.2	580.1	622.9		-16.7	-6.3	-13.4	29.4
CWT	8	658.6	642.4	640.0	654.5	658.2		-16.1	-18.6	-4.0	-0.4
CWT	9	551.8	542.2	554.2	554.1	559.2		-9.6	2.4	2.3	7.4
CWT	10	654.9	645.4	649.4	662.6	645.0		-9.5	-5.5	7.7	-9.9
CWT	11	549.6	540.0	542.4	564.0	560.2		-9.6	-7.1	14.4	10.6
CWT	12	569.3	587.3	591.1	635.7	607.3		18.0	21.8	66.4	38.0
	mean	588.3	588.0	589.8	596.2	590.1	mean	-0.3	1.5	7.9	1.8
	SD	68.1	60.9	59.4	65.5	73.9	SD	17.2	19.9	23.5	20.8
HWI	1	501.3	510.9	489.9	482.2	531.3		9.6	-11.4	-19.0	30.0
HWI	2	626.1	620.9	622.0	632.1	623.3		-5.2	-4.1	5.9	-2.8
HWI	3	676.3	602.1	651.0	690.4	707.6		-74.2	-25.3	14.1	31.3
HWI	4	496.0	498.9	491.9	485.6	484.8		3.0	-4.1	-10.4	-11.1
HWI	5	629.8	548.5	605.9	596.8	573.8		-81.3	-24.0	-33.0	-56.0
HWI	6	614.1	601.5	611.0	610.5	609.0		-12.5	-3.1	-3.6	-5.1
HWI	7	600.4	518.5	608.0	539.2	568.9		-81.9	7.6	-61.2	-31.5
HWI	8	590.0	615.8	636.4	621.8	623.9		25.9	46.4	31.9	33.9
HWI	9	490.3	477.1	489.9	503.8	515.3		-13.3	-0.4	13.5	24.9
HWI	10	633.8	676.0	640.4	649.8	572.7		42.2	6.6	16.0	-61.1
HWI	11	550.0	484.9	523.3	534.5	535.3		-65.1	-26.7	-15.5	-14.7
HWI	12	646.8	649.3	648.2	653.4	648.1		2.4	1.4	6.6	1.2
	mean	587.9	567.0	584.8	583.3	582.8	mean	-20.9	-3.1	-4.6	-5.1
	SD	63.5	68.5	65.8	71.4	62.7	SD	43.4	19.7	25.3	32.5
CWI	1	501.0	510.0	518.4	514.0	501.4		9.0	17.4	13.0	0.4
CWI	2	624.4	629.1	628.0	633.8	636.3		4.7	3.6	9.4	11.9
CWI	3	660.7	666.5	665.3	684.3	690.3		5.8	4.6	23.6	29.6
CWI	4	459.2	460.0	465.4	511.5	472.3		0.8	6.3	52.4	13.1
CWI	5	585.8	577.4	555.9	595.7	605.5		-8.4	-29.9	9.9	19.7
CWI	6	640.0	651.7	657.1	626.6	653.2		11.7	17.2	-13.4	13.2
CWI	7	605.0	618.0	608.0	616.0	613.0		13.0	3.0	11.0	8.0
CWI	8	613.0	625.4	629.6	633.1	670.4		12.4	16.6	20.1	57.3
CWI	9	531.4	531.2	554.3	553.5	554.7		-0.2	22.9	22.2	23.4
CWI	10	654.0	655.0	648.4	647.7	661.9		1.0	-5.6	-6.3	7.9
CWI	11	545.0	542.0	537.3	551.5	546.9		-3.0	-7.7	6.5	1.9
CWI	12	685.5	675.6	672.1	663.1	656.8		-9.9	-13.4	-22.4	-28.6
	mean	592.1	595.1	595.0	602.6	605.2	mean	3.1	2.9	10.5	13.1
	SD	69.5	69.9	66.9	57.4	70.8	SD	7.8	15.2	19.3	20.1

Time Trial Performance (W)

Raw Data		Trials (Days 1-5)						Effects			
Rec	Subject	1	2	3	4	5		1-2	1-3	1-4	1-5
PAS	1	262.3	251.3	242.3	239.5	251.2		-11.0	-20.0	-22.8	-11.1
PAS	2	354.2	353.5	348.4	353.2	355.9		-0.7	-5.8	-1.0	1.8
PAS	3	372.2	355.3	358.2	352.0	362.2		-16.9	-14.0	-20.2	-10.1
PAS	4	265.3	260.7	247.2	246.1	241.0		-4.6	-18.0	-19.2	-24.3
PAS	5	283.3	277.5	275.0	276.5	286.8		-5.7	-8.3	-6.8	3.5
PAS	6	421.8	420.0	453.7	432.3	432.3		-1.8	31.8	10.5	10.5
PAS	7	289.1	275.6	256.9	276.8	258.5		-13.5	-32.2	-12.3	-30.6
PAS	8	234.3	230.0	228.0	232.0	232.1		-4.3	-6.3	-2.3	-2.2
PAS	9	239.4	236.0	247.0	235.2	234.3		-3.4	7.6	-4.2	-5.2
PAS	10	333.6	330.0	333.0	317.0	305.0		-3.6	-0.7	-16.7	-28.6
PAS	11	260.8	243.9	257.6	245.4	248.1		-16.9	-3.2	-15.4	-12.7
PAS	12	317.6	310.0	304.6	307.5	304.1		-7.7	-13.0	-10.1	-13.5
	mean	302.8	295.3	296.0	292.8	292.6	mean	-7.5	-6.8	-10.0	-10.2
	SD	57.9	58.9	66.5	62.0	62.7	SD	5.7	15.9	9.7	12.9
CWT	1	245.8	251.2	248.0	249.9	251.6		5.4	2.2	4.1	5.8
CWT	2	352.0	353.5	354.1	359.8	357.8		1.5	2.1	7.8	5.8
CWT	3	371.2	375.5	378.3	374.2	362.5		4.2	7.1	3.0	-8.7
CWT	4	250.0	251.6	254.8	248.0	247.0		1.6	4.8	-2.0	-3.0
CWT	5	313.0	315.0	305.4	315.2	323.9		2.0	-7.5	2.2	10.9
CWT	6	420.0	442.2	428.6	457.1	428.8		22.2	8.6	37.1	8.8
CWT	7	268.3	255.8	266.1	275.2	269.8		-12.5	-2.2	6.9	1.5
CWT	8	235.8	236.5	231.6	242.0	249.0		0.7	-4.2	6.2	13.2
CWT	9	248.8	254.0	264.7	256.8	254.8		5.2	15.8	7.9	6.0
CWT	10	320.0	315.4	318.9	326.7	327.0		-4.6	-1.1	6.7	7.0
CWT	11	274.5	267.3	269.5	266.4	265.7		-7.3	-5.1	-8.1	-8.8
CWT	12	294.4	283.3	285.4	293.7	283.0		-11.1	-9.0	-0.7	-11.4
	mean	299.5	300.1	300.5	305.4	301.8	mean	0.6	1.0	5.9	2.3
	SD	57.7	62.8	59.5	65.1	58.2	SD	9.2	7.3	10.9	8.3
HWI	1	238.4	241.6	241.3	227.7	242.9		3.2	3.0	-10.7	4.5
HWI	2	356.5	360.6	363.4	359.2	351.2		4.1	6.9	2.7	-5.3
HWI	3	364.6	369.0	376.9	359.0	355.9		4.4	12.3	-5.6	-8.7
HWI	4	252.1	247.8	240.1	228.8	223.7		-4.2	-11.9	-23.3	-28.3
HWI	5	304.2	305.6	314.3	308.6	283.6		1.4	10.1	4.4	-20.6
HWI	6	426.2	418.2	446.2	441.7	439.9		-8.0	20.0	15.4	13.7
HWI	7	274.5	245.5	278.8	271.8	267.8		-29.0	4.2	-2.8	-6.7
HWI	8	238.0	244.0	256.7	233.8	244.8		6.0	18.7	-4.2	6.8
HWI	9	241.5	248.1	242.3	237.2	244.9		6.7	0.8	-4.3	3.5
HWI	10	307.0	323.6	315.8	317.5	324.0		16.6	8.8	10.5	17.0
HWI	11	260.3	246.2	244.9	200.0	243.3		-14.1	-15.4	-60.3	-17.0
HWI	12	321.1	313.9	329.8	311.4	311.0		-7.1	8.7	-9.7	-10.1
	mean	298.7	297.0	304.2	291.4	294.4	mean	-1.7	5.5	-7.3	-4.3
	SD	59.8	61.0	66.2	71.4	64.2	SD	11.8	10.6	19.5	13.8
CWI	1	250.2	250.0	249.0	246.0	251.0		-0.1	-1.2	-4.2	0.8
CWI	2	359.0	358.0	366.3	367.4	366.2		-1.0	7.3	8.4	7.2
CWI	3	371.0	373.3	370.3	364.1	365.8		2.3	-0.7	-6.9	-5.2
CWI	4	252.6	252.0	251.0	252.0	257.0		-0.6	-1.6	-0.6	4.4
CWI	5	313.0	313.0	314.0	315.0	313.7		0.0	1.0	2.0	0.7
CWI	6	418.0	419.0	418.1	416.0	413.3		1.0	0.1	-2.0	-4.7
CWI	7	251.8	251.4	273.9	259.8	253.0		-0.3	22.2	8.0	1.2
CWI	8	249.0	250.0	253.4	248.8	258.4		1.0	4.4	-0.2	9.4
CWI	9	258.0	257.0	255.8	256.0	261.0		-1.0	-2.2	-2.0	3.0
CWI	10	325.0	325.3	309.7	336.3	333.1		0.3	-15.3	11.3	8.1
CWI	11	253.0	252.3	253.6	259.2	255.0		-0.7	0.6	6.2	2.0
CWI	12	319.0	324.0	319.0	322.0	325.8		5.0	0.0	3.0	6.8
	mean	301.6	302.1	302.8	303.6	304.4	mean	0.5	1.2	1.9	2.8
	SD	58.2	58.8	57.0	58.0	56.4	SD	1.7	8.5	5.6	4.7

Total Work (kJ)

Raw Data		Trials (Days 1-5)						Effects			
Rec	Subject	1	2	3	4	5		1-2	1-3	1-4	1-5
PAS	1	141.6	135.7	130.8	129.3	135.7		-5.9	-10.8	-12.3	-6.0
PAS	2	191.3	190.9	188.1	190.7	192.2		-0.4	-3.1	-0.5	1.0
PAS	3	201.0	191.9	193.4	190.1	195.6		-9.1	-7.6	-10.9	-5.4
PAS	4	143.3	140.8	133.5	132.9	130.1		-2.5	-9.7	-10.4	-13.1
PAS	5	153.0	149.9	153.4	149.3	154.9		-3.1	0.4	-3.7	1.9
PAS	6	227.8	240.0	245.0	233.5	233.5		12.2	17.2	5.7	5.7
PAS	7	156.1	148.8	138.7	149.5	139.6		-7.3	-17.4	-6.6	-16.5
PAS	8	126.5	139.6	128.8	125.3	125.3		13.1	2.3	-1.2	-1.2
PAS	9	129.3	134.5	133.4	127.0	126.5		5.2	4.1	-2.3	-2.8
PAS	10	180.2	178.7	179.8	171.2	164.7		-1.5	-0.4	-9.0	-15.4
PAS	11	140.8	131.7	139.1	132.5	134.0		-9.1	-1.7	-8.3	-6.9
PAS	12	171.5	167.4	164.5	166.1	164.2		-4.1	-7.0	-5.5	-7.3
	mean	163.5	162.5	160.7	158.1	158.0	mean	-1.0	-2.8	-5.4	-5.5
	SD	31.3	32.7	35.3	33.5	33.9	SD	7.5	8.8	5.2	6.9
CWT	1	132.7	135.7	138.3	134.9	135.9		2.9	5.5	2.2	3.1
CWT	2	187.8	190.9	191.2	194.3	193.2		3.0	3.4	6.4	5.4
CWT	3	200.5	202.8	204.3	202.1	195.8		2.3	3.8	1.6	-4.7
CWT	4	122.6	135.8	137.6	127.5	127.3		13.2	15.0	4.9	4.7
CWT	5	169.0	174.8	164.9	170.2	174.9		5.8	-4.1	1.2	5.9
CWT	6	225.6	238.8	231.4	246.9	231.5		13.2	5.8	21.3	6.0
CWT	7	144.9	138.1	143.7	148.6	145.7		-6.8	-1.2	3.7	0.8
CWT	8	127.3	127.7	125.1	138.9	143.0		0.4	-2.3	11.6	15.7
CWT	9	134.4	137.2	142.9	138.7	137.6		2.8	8.5	4.3	3.2
CWT	10	165.9	170.3	172.2	176.4	176.6		4.4	6.3	10.5	10.7
CWT	11	148.2	144.3	145.5	143.9	143.5		-3.9	-2.7	-4.4	-4.7
CWT	12	159.0	153.0	154.1	158.6	152.8		-6.0	-4.8	-0.4	-6.1
	mean	159.8	162.4	162.6	165.1	163.2	mean	2.6	2.8	5.2	3.3
	SD	31.7	34.1	31.8	35.1	31.4	SD	6.4	6.0	6.7	6.4
HWI	1	128.7	130.5	130.3	123.0	131.2		1.7	1.6	-5.8	2.4
HWI	2	192.5	194.7	196.2	194.0	189.6		2.2	3.7	1.5	-2.9
HWI	3	196.9	199.3	203.6	193.9	192.2		2.4	6.7	-3.0	-4.7
HWI	4	136.1	133.8	129.7	123.5	120.8		-2.3	-6.4	-12.6	-15.3
HWI	5	164.3	165.0	169.7	166.6	153.1		0.7	5.5	2.4	-11.1
HWI	6	230.2	225.8	241.0	238.5	237.5		-4.3	10.8	8.3	7.4
HWI	7	148.3	132.6	150.5	146.7	144.6		-15.7	2.3	-1.5	-3.6
HWI	8	128.5	131.8	138.6	126.3	132.2		3.2	10.1	-2.3	3.7
HWI	9	130.4	134.0	130.8	128.1	132.3		3.6	0.4	-2.3	1.9
HWI	10	165.8	174.7	170.6	171.5	175.0		9.0	4.8	5.7	9.2
HWI	11	140.5	132.9	132.2	108.0	131.4		-7.6	-8.3	-32.6	-9.2
HWI	12	173.4	169.5	178.1	168.1	167.9		-3.9	4.7	-5.2	-5.4
	mean	161.3	160.4	164.3	157.3	159.0	mean	-0.9	3.0	-3.9	-2.3
	SD	32.3	32.9	35.7	38.6	34.7	SD	6.4	5.7	10.5	7.5
CWI	1	135.1	135.0	129.0	128.8	131.4		-0.1	-6.0	-6.3	-3.7
CWI	2	190.0	193.3	197.8	198.4	197.7		3.3	7.8	8.4	7.7
CWI	3	201.6	201.6	200.0	196.6	197.5		0.0	-1.6	-4.9	-4.0
CWI	4	136.4	127.5	128.4	134.3	127.6		-8.9	-8.0	-2.1	-8.8
CWI	5	169.0	167.9	165.5	170.1	169.4		-1.1	-3.5	1.1	0.4
CWI	6	207.9	220.1	225.8	224.7	223.2		12.2	17.9	16.8	15.3
CWI	7	135.9	135.8	147.9	140.3	135.2		-0.2	12.0	4.3	-0.8
CWI	8	126.1	132.5	136.8	134.4	139.5		6.4	10.8	8.3	13.5
CWI	9	132.7	133.8	138.1	138.3	141.0		1.1	5.4	5.6	8.3
CWI	10	176.6	175.7	167.2	181.6	179.9		-1.0	-9.4	5.0	3.2
CWI	11	137.2	136.2	136.9	140.0	135.1		-0.9	-0.3	2.8	-2.1
CWI	12	165.5	165.7	168.5	170.4	175.9		0.2	2.9	4.9	10.4
	mean	159.5	160.4	161.8	163.2	162.8	mean	0.9	2.3	3.7	3.3
	SD	29.4	31.7	31.8	31.8	32.1	SD	5.0	8.6	6.3	7.7

Core Temperature (°C) - Pre-Exercise

Raw Data		Trials (Days 1-5)						Effects			
Rec	Subject	1	2	3	4	5		1-2	1-3	1-4	1-5
PAS	1	37.2	37.1	37.3	37.1	37.2		-0.1	0.1	-0.1	0.0
PAS	2	37.6	37.5	37.4	37.5	37.4		-0.1	-0.2	-0.1	-0.2
PAS	3	37.7	37.2	37.4	37.3	37.4		-0.5	-0.3	-0.4	-0.3
PAS	4	37.2	37.2	37.1	37.0	37.1		0.0	-0.1	-0.2	-0.1
PAS	5	37.2	37.3	37.1	37.2	37.0		0.1	-0.1	0.0	-0.2
PAS	6	37.2	37.1	37.5	37.6	37.7		-0.1	0.3	0.4	0.5
PAS	7	37.7	37.5	37.5	37.5	37.5		-0.2	-0.2	-0.2	-0.2
PAS	8	37.2	37.3	36.9	37.3	37.0		0.1	-0.3	0.1	-0.2
PAS	9	37.0	37.0	37.2	36.9	37.0		0.0	0.2	-0.1	0.0
PAS	10	37.4	37.4	37.4	36.9	37.2		0.0	0.0	-0.5	-0.2
PAS	11	37.1	37.3	37.3	37.3	37.3		0.2	0.2	0.2	0.2
PAS	12	37.4	37.4	37.6	37.2	37.4		0.0	0.2	-0.2	0.0
	mean	37.3	37.3	37.3	37.2	37.3	mean	-0.1	0.0	-0.1	-0.1
	SD	0.2	0.2	0.2	0.2	0.2	SD	0.2	0.2	0.2	0.2
CWT	1	37.4	37.2	37.0	37.1	37.4		-0.2	-0.4	-0.3	0.0
CWT	2	37.5	37.4	37.4	37.5	37.4		-0.1	-0.1	0.0	-0.1
CWT	3	37.4	37.2	37.2	37.4	37.2		-0.2	-0.2	0.0	-0.2
CWT	4	37.2	37.1	37.1	37.4	37.2		-0.1	-0.1	0.2	0.0
CWT	5	37.2	37.0	37.4	37.1	37.4		-0.2	0.2	-0.1	0.2
CWT	6	37.6	37.7	37.6	37.8	37.5		0.1	0.0	0.2	-0.1
CWT	7	37.4	37.3	37.7	37.5	37.2		-0.1	0.3	0.1	-0.2
CWT	8	37.1	37.2	37.3	37.4	37.5		0.1	0.2	0.3	0.4
CWT	9	37.1	36.8	37.1	37.0	36.8		-0.3	0.0	-0.1	-0.3
CWT	10	37.3	37.1	37.4	37.3	37.1		-0.2	0.1	0.0	-0.2
CWT	11	37.3	37.5	37.1	37.2	37.2		0.2	-0.2	-0.1	-0.1
CWT	12	37.2	37.6	37.5	37.3	37.2		0.4	0.3	0.1	0.0
	mean	37.3	37.3	37.3	37.3	37.3	mean	0.0	0.0	0.0	0.0
	SD	0.2	0.3	0.2	0.2	0.2	SD	0.2	0.2	0.2	0.2
HWI	1	37.1	37.3	37.0	37.1	37.2		0.2	-0.1	0.0	0.1
HWI	2	37.5	37.6	37.5	37.4	37.5		0.1	0.0	-0.1	0.0
HWI	3	37.1	37.5	37.3	37.3	37.4		0.4	0.2	0.2	0.3
HWI	4	37.2	37.2	37.1	37.0	37.1		0.0	-0.1	-0.2	-0.1
HWI	5	37.5	37.4	37.3	37.5	37.3		-0.1	-0.2	0.0	-0.2
HWI	6	37.6	37.6	37.6	37.7	37.7		0.0	0.0	0.1	0.1
HWI	7	37.6	37.5	37.4	37.3	37.3		-0.1	-0.2	-0.3	-0.3
HWI	8	37.2	37.5	37.4	37.1	37.2		0.3	0.2	-0.1	0.0
HWI	9	36.7	37.1	37.3	36.9	36.9		0.4	0.6	0.2	0.2
HWI	10	37.4	37.2	37.3	37.2	37.2		-0.2	-0.1	-0.2	-0.2
HWI	11	37.3	37.5	37.0	37.1	37.6		0.2	-0.3	-0.2	0.3
HWI	12	37.3	37.5	37.3	37.2	37.3		0.2	0.0	-0.1	0.0
	mean	37.3	37.4	37.3	37.2	37.3	mean	0.1	0.0	-0.1	0.0
	SD	0.3	0.2	0.2	0.2	0.2	SD	0.2	0.2	0.2	0.2
CWI	1	37.0	37.1	37.0	37.1	37.2		0.1	0.0	0.1	0.2
CWI	2	37.2	37.1	37.4	37.2	37.4		-0.1	0.2	0.0	0.2
CWI	3	37.2	37.0	37.2	37.2	37.4		-0.2	0.0	0.0	0.2
CWI	4	37.4	37.3	37.4	37.5	37.3		-0.1	0.0	0.1	-0.1
CWI	5	37.2	37.2	37.3	37.2	37.1		0.0	0.1	0.0	-0.1
CWI	6	37.2	37.3	37.4	37.2	37.0		0.1	0.2	0.0	-0.2
CWI	7	37.6	37.3	37.7	37.7	37.5		-0.3	0.1	0.1	-0.1
CWI	8	37.1	37.2	37.2	37.1	37.2		0.1	0.1	0.0	0.1
CWI	9	36.8	36.9	37.1	37.0	36.9		0.1	0.3	0.2	0.1
CWI	10	37.2	37.1	37.4	37.4	37.4		-0.1	0.2	0.2	0.2
CWI	11	37.4	37.5	37.2	37.4	37.3		0.1	-0.2	0.0	-0.1
CWI	12	37.1	37.5	37.3	37.2	37.5		0.4	0.2	0.1	0.4
	mean	37.2	37.2	37.3	37.3	37.3	mean	0.0	0.1	0.1	0.1
	SD	0.2	0.2	0.2	0.2	0.2	SD	0.2	0.1	0.1	0.2

Core Temperature (°C) - Post-Exercise

Raw Data		Trials						Effects			
Rec	Subject	1	2	3	4	5		1-2	1-3	1-4	1-5
PAS	1	38.2	38.1	38.3	37.8	38.2		-0.1	0.1	-0.4	0.0
PAS	2	37.9	38.4	38.5	38.0	38.2		0.5	0.6	0.1	0.3
PAS	3	38.7	38.8	38.4	38.5	38.4		0.1	-0.3	-0.2	-0.3
PAS	4	39.0	38.6	38.3	39.1	38.5		-0.4	-0.7	0.1	-0.5
PAS	5	38.0	38.4	38.6	38.3	38.1		0.4	0.6	0.3	0.1
PAS	6	39.0	39.0	38.9	38.8	38.7		0.0	-0.1	-0.2	-0.3
PAS	7	38.9	38.5	38.6	38.5	38.7		-0.4	-0.3	-0.4	-0.2
PAS	8	38.6	38.7	38.4	38.3	38.3		0.1	-0.2	-0.3	-0.3
PAS	9	38.4	38.4	38.5	38.3	38.2		0.0	0.1	-0.1	-0.2
PAS	10	37.8	38.5	38.6	37.9	38.2		0.7	0.8	0.1	0.4
PAS	11	38.6	38.4	38.6	38.5	38.5		-0.2	0.0	-0.1	-0.1
PAS	12	39.0	38.7	38.4	39.0	38.6		-0.3	-0.6	0.0	-0.4
	mean	38.5	38.5	38.5	38.4	38.4	mean	0.0	0.0	-0.1	-0.1
	SD	0.4	0.2	0.2	0.4	0.2	SD	0.4	0.5	0.2	0.3
CWT	1	38.1	38.2	38.2	38.2	38.4		0.1	0.1	0.1	0.3
CWT	2	38.6	38.6	38.8	38.7	38.8		0.0	0.2	0.1	0.2
CWT	3	38.4	38.5	38.3	38.2	38.6		0.1	-0.1	-0.2	0.2
CWT	4	38.4	38.5	38.6	38.4	38.5		0.1	0.2	0.0	0.1
CWT	5	38.3	38.3	38.6	38.3	38.6		0.0	0.3	0.0	0.3
CWT	6	39.2	39.1	39.2	39.2	39.0		-0.1	0.0	0.0	-0.2
CWT	7	38.8	38.5	39.0	38.6	38.5		-0.3	0.2	-0.2	-0.3
CWT	8	38.1	38.4	38.5	38.2	38.0		0.3	0.4	0.1	-0.1
CWT	9	38.4	38.3	38.3	38.4	38.2		-0.1	-0.1	0.0	-0.2
CWT	10	38.5	38.3	38.4	38.2	38.1		-0.2	-0.1	-0.3	-0.4
CWT	11	38.6	38.5	39.0	38.6	38.7		-0.1	0.4	0.0	0.1
CWT	12	38.9	38.7	39.0	38.3	38.6		-0.2	0.1	-0.6	-0.3
	mean	38.5	38.5	38.7	38.4	38.5	mean	0.0	0.1	-0.1	0.0
	SD	0.3	0.2	0.3	0.3	0.3	SD	0.2	0.2	0.2	0.3
HWI	1	38.1	38.3	38.2	38.1	38.3		0.2	0.1	0.0	0.2
HWI	2	38.7	38.6	38.8	38.8	38.7		-0.1	0.1	0.1	0.0
HWI	3	38.6	38.6	38.7	38.5	38.3		0.0	0.1	-0.1	-0.3
HWI	4	38.2	38.4	38.5	38.4	38.5		0.2	0.3	0.2	0.3
HWI	5	38.0	38.5	38.6	38.0	38.3		0.5	0.6	0.0	0.3
HWI	6	38.7	37.8	38.9	39.0	39.2		-0.9	0.2	0.3	0.5
HWI	7	38.6	38.7	38.5	38.6	38.7		0.1	-0.1	0.0	0.1
HWI	8	38.4	38.4	38.5	38.3	38.1		0.0	0.1	-0.1	-0.3
HWI	9	38.2	38.3	38.5	38.2	38.4		0.1	0.3	0.0	0.2
HWI	10	38.5	38.3	38.3	38.1	38.2		-0.2	-0.2	-0.4	-0.3
HWI	11	38.6	38.5	39.0	38.6	38.7		-0.1	0.4	0.0	0.1
HWI	12	38.6	38.7	38.6	38.7	38.6		0.1	0.0	0.1	0.0
	mean	38.4	38.4	38.6	38.4	38.5	mean	0.0	0.2	0.0	0.1
	SD	0.2	0.2	0.2	0.3	0.3	SD	0.3	0.2	0.2	0.3
CWI	1	38.3	38.3	38.5	38.3	38.5		0.0	0.2	0.0	0.2
CWI	2	38.7	38.8	38.8	38.7	38.8		0.1	0.1	0.0	0.1
CWI	3	38.6	38.3	38.6	38.4	38.7		-0.3	0.0	-0.2	0.1
CWI	4	38.3	38.5	38.4	38.5	38.5		0.2	0.1	0.2	0.2
CWI	5	38.6	38.0	38.3	38.5	38.3		-0.6	-0.3	-0.1	-0.3
CWI	6	38.7	38.8	38.6	39.3	39.2		0.1	-0.1	0.6	0.5
CWI	7	38.6	38.3	38.6	38.8	38.8		-0.3	0.0	0.2	0.2
CWI	8	38.4	38.5	38.4	38.4	38.7		0.1	0.0	0.0	0.3
CWI	9	38.2	37.9	38.4	38.2	38.2		-0.3	0.2	0.0	0.0
CWI	10	38.3	37.9	38.2	38.1	38.2		-0.4	-0.1	-0.2	-0.1
CWI	11	38.5	38.9	38.6	38.6	38.6		0.4	0.1	0.1	0.1
CWI	12	38.8	38.6	38.6	38.6	38.9		-0.2	-0.2	-0.2	0.1
	mean	38.5	38.4	38.5	38.5	38.6	mean	-0.1	0.0	0.0	0.1
	SD	0.2	0.3	0.2	0.3	0.3	SD	0.3	0.2	0.2	0.2

Core Temperature (°C) - Post-Recovery

Raw Data		Trials					Effects		
Rec	Subject	1	2	3	4		1-2	1-3	1-4
PAS	1	36.9	36.7	37.1	37.1		-0.2	0.2	0.2
PAS	2	37.2	37.3	37.3	37.2		0.1	0.1	0.0
PAS	3	37.5	37.5	37.5	37.4		0.0	0.0	-0.1
PAS	4	37.4	37.5	37.4	37.5		0.1	0.0	0.1
PAS	5	37.3	37.6	37.4	37.3		0.3	0.1	0.0
PAS	6	37.5	37.8	37.5	37.7		0.3	0.0	0.2
PAS	7	37.8	37.5	37.6	37.4		-0.3	-0.2	-0.4
PAS	8	37.7	37.7	37.3	37.6		0.0	-0.4	-0.1
PAS	9	37.3	37.3	37.3	37.2		0.0	0.0	-0.1
PAS	10	37.3	37.1	37.2	37.0		-0.2	-0.1	-0.3
PAS	11	36.9	37.4	37.5	37.1		0.5	0.6	0.2
PAS	12	37.3	37.3	37.3	37.6		0.0	0.0	0.3
	mean	37.3	37.4	37.4	37.3	mean	0.1	0.0	0.0
	SD	0.3	0.3	0.1	0.2	SD	0.2	0.2	0.2
CWT	1	37.2	37.3	37.2	37.2		0.1	0.0	0.0
CWT	2	37.5	37.6	37.5	37.6		0.1	0.0	0.1
CWT	3	37.6	37.6	37.3	37.4		0.0	-0.3	-0.2
CWT	4	37.4	37.5	37.4	37.5		0.1	0.0	0.1
CWT	5	37.5	37.5	37.4	37.7		0.0	-0.1	0.2
CWT	6	38.1	38.0	37.9	38.0		-0.1	-0.2	-0.1
CWT	7	37.4	37.3	37.5	37.8		-0.1	0.1	0.4
CWT	8	37.4	37.6	37.6	37.6		0.2	0.2	0.2
CWT	9	37.5	36.9	36.9	37.3		-0.6	-0.6	-0.2
CWT	10	37.3	37.2	37.4	37.3		-0.1	0.1	0.0
CWT	11	37.6	37.8	37.5	37.7		0.2	-0.1	0.1
CWT	12	37.5	37.5	37.7	37.5		0.0	0.2	0.0
	mean	37.5	37.5	37.4	37.6	mean	0.0	-0.1	0.1
	SD	0.2	0.3	0.3	0.2	SD	0.2	0.2	0.2
HWI	1	37.1	37.4	37.4	37.3		0.3	0.3	0.2
HWI	2	37.8	37.8	37.7	37.7		0.0	-0.1	-0.1
HWI	3	37.6	37.8	37.6	37.8		0.2	0.0	0.2
HWI	4	37.6	37.4	37.6	37.6		-0.2	0.0	0.0
HWI	5	37.4	37.5	38.1	37.7		0.1	0.7	0.3
HWI	6	37.4	37.6	37.7	37.8		0.2	0.3	0.4
HWI	7	37.8	37.7	37.3	37.5		-0.1	-0.5	-0.3
HWI	8	37.8	37.8	37.8	37.7		0.0	0.0	-0.1
HWI	9	37.3	37.4	37.6	37.5		0.1	0.3	0.2
HWI	10	37.4	37.7	37.3	37.3		0.3	-0.1	-0.1
HWI	11	37.6	37.8	37.5	37.4		0.2	-0.1	-0.2
HWI	12	37.4	37.7	37.4	37.5		0.3	0.0	0.1
	mean	37.5	37.6	37.6	37.6	mean	0.1	0.1	0.1
	SD	0.2	0.2	0.2	0.2	SD	0.2	0.3	0.2
CWI	1	37.2	37.0	37.1	37.3		-0.2	-0.1	0.1
CWI	2	37.2	37.2	37.1	37.1		0.0	-0.1	-0.1
CWI	3	37.3	37.2	37.5	37.2		-0.1	0.2	-0.1
CWI	4	37.3	37.5	37.3	37.3		0.2	0.0	0.0
CWI	5	37.6	37.1	37.5	37.4		-0.5	-0.1	-0.2
CWI	6	37.4	37.7	37.5	38.0		0.3	0.1	0.6
CWI	7	37.3	37.0	37.0	37.3		-0.3	-0.3	0.0
CWI	8	37.7	37.6	37.6	37.5		-0.1	-0.1	-0.2
CWI	9	36.6	37.1	37.0	36.6		0.5	0.4	0.0
CWI	10	37.0	37.1	36.9	37.1		0.1	-0.1	0.1
CWI	11	37.4	37.5	37.5	37.2		0.1	0.1	-0.2
CWI	12	37.4	37.0	37.3	37.4		-0.4	-0.1	0.0
	mean	37.3	37.3	37.3	37.3	mean	0.0	0.0	0.0
	SD	0.3	0.3	0.2	0.3	SD	0.3	0.2	0.2

Core Temperature (°C) - 15 min Post-Recovery

Raw Data		Trials					Effects		
Rec	Subject	1	2	3	4		1-2	1-3	1-4
PAS	1	36.8	36.7	37.1	36.8		-0.1	0.3	0.0
PAS	2	37.3	37.3	37.2	37.3		0.0	-0.1	0.0
PAS	3	37.5	37.5	37.4	37.2		0.0	-0.1	-0.3
PAS	4	37.2	37.1	37.2	37.2		-0.1	0.0	0.0
PAS	5	37.1	37.3	37.3	37.2		0.2	0.2	0.1
PAS	6	37.4	37.6	37.4	37.5		0.2	0.0	0.1
PAS	7	37.6	37.4	37.5	37.4		-0.2	-0.1	-0.2
PAS	8	37.5	37.5	37.1	37.4		0.0	-0.4	-0.1
PAS	9	37.2	37.3	37.2	37.1		0.1	0.0	-0.1
PAS	10	37.2	37.1	37.3	36.9		-0.1	0.1	-0.3
PAS	11	36.9	37.3	37.4	37.0		0.4	0.5	0.1
PAS	12	37.3	37.2	37.4	37.4		-0.1	0.1	0.1
	mean	37.3	37.3	37.3	37.2	mean	0.0	0.0	-0.1
	SD	0.2	0.2	0.1	0.2	SD	0.2	0.2	0.2
CWT	1	37.2	37.2	36.9	36.8		0.0	-0.3	-0.4
CWT	2	37.3	37.3	37.4	37.4		0.0	0.1	0.1
CWT	3	37.5	37.5	37.2	37.3		0.0	-0.3	-0.2
CWT	4	37.1	37.0	37.2	37.2		-0.1	0.1	0.1
CWT	5	37.5	37.2	37.2	37.5		-0.3	-0.3	0.0
CWT	6	38.1	38.0	37.8	37.8		-0.1	-0.3	-0.3
CWT	7	37.4	37.1	37.4	37.1		-0.3	0.0	-0.3
CWT	8	37.2	37.3	37.4	37.6		0.1	0.2	0.4
CWT	9	37.1	36.6	36.6	36.9		-0.5	-0.5	-0.2
CWT	10	37.3	37.2	37.3	37.2		-0.1	0.0	-0.1
CWT	11	37.6	37.7	37.3	37.7		0.1	-0.3	0.1
CWT	12	37.5	37.3	37.6	37.5		-0.2	0.1	0.0
	mean	37.4	37.3	37.3	37.3	mean	-0.1	-0.1	-0.1
	SD	0.3	0.3	0.3	0.3	SD	0.2	0.2	0.2
HWI	1	37.2	37.4	37.6	37.7		0.2	0.4	0.5
HWI	2	37.6	37.7	37.7	37.6		0.1	0.1	0.0
HWI	3	37.8	38.0	37.4	37.9		0.2	-0.4	0.1
HWI	4	37.8	37.8	37.7	37.8		0.0	-0.1	0.0
HWI	5	37.7	37.7	37.9	37.6		0.0	0.2	-0.1
HWI	6	37.6	37.8	37.9	38.0		0.2	0.3	0.4
HWI	7	37.9	37.7	37.4	37.6		-0.2	-0.5	-0.3
HWI	8	37.9	37.9	37.7	37.8		0.0	-0.2	-0.1
HWI	9	37.5	37.5	37.4	37.6		0.0	-0.1	0.1
HWI	10	37.5	37.7	37.6	37.4		0.2	0.1	-0.1
HWI	11	37.7	37.9	37.5	37.6		0.2	-0.2	-0.1
HWI	12	37.7	37.7	37.5	37.6		0.0	-0.2	-0.1
	mean	37.7	37.7	37.6	37.7	mean	0.1	-0.1	0.0
	SD	0.2	0.2	0.2	0.2	SD	0.1	0.3	0.2
CWI	1	36.0	36.5	36.2	36.2		0.5	0.2	0.2
CWI	2	37.0	37.0	36.9	36.9		0.0	-0.1	-0.1
CWI	3	37.1	36.9	36.9	36.9		-0.2	-0.2	-0.2
CWI	4	36.9	36.8	36.9	36.7		-0.1	0.0	-0.2
CWI	5	37.2	36.9	37.2	36.9		-0.3	0.0	-0.3
CWI	6	37.0	37.3	37.1	37.5		0.3	0.1	0.5
CWI	7	36.9	36.6	36.6	36.9		-0.3	-0.3	0.0
CWI	8	37.5	37.3	36.4	37.1		-0.2	-1.1	-0.4
CWI	9	36.5	36.7	36.7	36.2		0.2	0.2	-0.3
CWI	10	36.8	36.6	36.5	36.9		-0.2	-0.3	0.1
CWI	11	36.9	37.4	37.2	37.0		0.5	0.3	0.1
CWI	12	36.7	36.5	36.5	36.4		-0.2	-0.2	-0.3
	mean	36.9	36.9	36.8	36.8	mean	0.0	-0.1	-0.1
	SD	0.4	0.3	0.3	0.4	SD	0.3	0.4	0.3

Rating of Perceived Exertion
(0 = no exertion at all, 10 = maximal exertion)

Raw Data		Trials (Days 1-5)				
Treatment	Subject	1	2	3	4	5
PAS	1	9	9	9	8	9
PAS	2	9	9	9	8	10
PAS	3	8	8	8	9	9
PAS	4	8	9	8	8	9
PAS	5	8	8	8	8	8
PAS	6	8	8	8	8	8
PAS	7	8	8	8	8	8
PAS	8	8	9	9	9	9
PAS	9	8	8	8	9	9
PAS	10	8	8	8	8	8
PAS	11	9	8	9	8	9
PAS	12	8	9	9	9	9
	mean	8	8	8	8	9
	SD	0.4	0.4	0.4	0.3	0.3

CWT	1	8	9	9	9	9
CWT	2	8	9	8	9	9
CWT	3	8	9	8	8	9
CWT	4	9	9	8	8	9
CWT	5	8	8	8	8	8
CWT	6	8	8	8	8	8
CWT	7	8	8	8	8	8
CWT	8	9	9	9	8	9
CWT	9	8	8	8	9	8
CWT	10	8	8	8	9	8
CWT	11	8	8	8	9	9
CWT	12	8	8	9	8	8
	mean	8	8	8	8	8
	SD	0.3	0.3	0.3	0.3	0.3

HWI	1	9	9	8	9	9
HWI	2	9	9	9	9	9
HWI	3	9	9	9	9	9
HWI	4	9	9	9	8	9
HWI	5	8	8	8	8	8
HWI	6	8	8	8	8	8
HWI	7	8	8	8	8	8
HWI	8	9	9	9	9	9
HWI	9	8	8	8	8	9
HWI	10	8	9	8	9	9
HWI	11	8	9	9	9	8
HWI	12	8	8	8	8	9
	mean	8	8	8	8	9
	SD	0.3	0.4	0.3	0.4	0.3

CWI	1	8	8	9	9	9
CWI	2	9	8	9	8	9
CWI	3	8	9	9	8	8
CWI	4	8	9	9	8	8
CWI	5	8	8	8	8	8
CWI	6	8	8	8	8	8
CWI	7	8	8	8	8	8
CWI	8	8	9	8	8	9
CWI	9	8	8	8	8	8
CWI	10	8	8	8	9	9
CWI	11	8	9	9	9	9
CWI	12	8	8	8	8	8
	mean	8	8	8	8	8
	SD	0.3	0.3	0.4	0.2	0.3

Heart Rate (bpm) - Sprint

Raw Data		Trials (Days 1-5)						Effects			
Treatment	Subject	1	2	3	4	5		1-2	1-3	1-4	1-5
PAS	1	171	172	173	166	168		1.1	2.7	-5.2	-2.7
PAS	2	169	171	171	168	167		2.0	2.0	-1.0	-2.0
PAS	3	170	166	167	165	165		-3.7	-3.4	-4.6	-5.3
PAS	4	170	170	171	168	169		0.0	1.0	-2.0	-1.0
PAS	5	161	158	166	159	157		-3.0	4.6	-2.7	-4.4
PAS	6	164	165	163	164	162		0.3	-1.1	-0.7	-2.0
PAS	7	173	164	167	167	171		-8.6	-5.6	-5.7	-2.3
PAS	8	162	165	167	166	161		3.1	4.3	3.7	-1.6
PAS	9	169	169	165	161	165		-0.4	-4.1	-8.4	-4.0
PAS	10	164	164	165	161	155		0.0	1.3	-2.9	-8.4
PAS	11	187	186	184	183	185		-1.1	-2.2	-3.7	-1.2
PAS	12	169	181	170	164	162		11.8	1.1	-5.4	-6.7
	mean	169	169	169	166	166	mean	0.1	0.1	-3.2	-3.5
	SD	7	8	6	6	8	SD	4.8	3.3	3.1	2.4
CWT	1	169	170	167	164	167		1.1	-2.4	-5.1	-2.2
CWT	2	168	171	170	171	170		3.0	2.0	3.0	2.0
CWT	3	168	168	165	165	165		-0.2	-3.3	-3.0	-3.0
CWT	4	171	173	170	171	172		2.0	-1.0	0.0	1.0
CWT	5	163	162	163	157	161		-0.2	0.2	-5.6	-2.0
CWT	6	172	167	166	165	160		-5.6	-6.6	-7.2	-11.9
CWT	7	175	172	173	171	167		-3.0	-1.7	-4.6	-8.4
CWT	8	156	161	161	161	164		5.6	5.0	5.0	8.0
CWT	9	165	164	162	166	160		-1.6	-3.1	1.1	-5.1
CWT	10	159	158	156	166	147		-1.2	-2.8	7.2	-12.3
CWT	11	181	184	180	180	179		3.2	-0.3	-0.6	-2.1
CWT	12	174	169	176	165	178		-5.2	1.7	-8.9	4.2
	mean	168	168	167	167	166	mean	-0.2	-1.0	-1.6	-2.7
	SD	7	7	7	6	9	SD	3.4	3.0	5.0	6.1
HWI	1	169	170	169	168	170		1.6	0.2	-0.8	1.3
HWI	2	167	170	172	171	171		3.0	5.0	4.0	4.0
HWI	3	169	173	171	166	167		4.1	1.8	-3.0	-1.9
HWI	4	170	171	172	170	170		1.0	2.0	0.0	0.0
HWI	5	172	170	170	164	163		-2.0	-2.7	-8.6	-9.0
HWI	6	169	169	168	163	163		0.6	-1.3	-5.7	-5.4
HWI	7	164	157	165	162	162		-7.3	0.2	-2.4	-2.6
HWI	8	157	160	159	159	155		2.9	2.7	1.9	-1.9
HWI	9	160	159	162	160	166		-0.9	2.1	-0.1	5.9
HWI	10	160	167	162	158	154		7.0	2.3	-2.4	-6.3
HWI	11	169	187	183	177	181		17.8	13.6	8.2	12.1
HWI	12	166	163	163	163	165		-2.3	-2.1	-2.2	-0.3
	mean	166	168	168	165	166	mean	2.1	2.0	-0.9	-0.3
	SD	5	8	6	6	7	SD	6.1	4.3	4.4	5.7
CWI	1	169	170	171	169	165		1.1	2.0	0.0	-3.6
CWI	2	168	170	171	170	169		2.0	3.0	2.0	1.0
CWI	3	168	169	170	165	165		1.3	1.7	-2.9	-2.6
CWI	4	170	171	172	170	170		1.0	2.0	0.0	0.0
CWI	5	159	162	163	162	157		2.9	3.7	2.4	-2.1
CWI	6	168	167	166	167	164		-0.1	-1.9	-0.4	-3.1
CWI	7	172	171	168	163	164		-1.2	-4.8	-9.4	-8.8
CWI	8	164	162	165	160	164		-2.2	0.9	-3.7	0.3
CWI	9	168	165	166	166	163		-2.7	-1.6	-1.8	-4.6
CWI	10	159	159	156	152	156		0.7	-3.0	-6.4	-2.4
CWI	11	179	185	185	182	185		6.6	6.3	2.9	5.8
CWI	12	166	166	166	179	162		0.1	0.0	12.9	-4.8
	mean	167	168	168	167	165	mean	0.8	0.7	-0.4	-2.1
	SD	5	7	7	8	7	SD	2.5	3.1	5.5	3.6

Heart Rate (bpm) - Time Trial

Raw Data		Trials (Days 1-5)							Effects			
Treatment	Subject	1	2	3	4	5			1-2	1-3	1-4	1-5
PAS	1	170	175	170	164	168			5.0	0.0	-6.0	-2.0
PAS	2	170	176	175	174	170			6.0	5.0	4.0	0.0
PAS	3	168	169	160	162	160			1.0	-8.0	-6.0	-8.0
PAS	4	171	175	176	169	170			4.0	5.0	-2.0	-1.0
PAS	5	178	183	187	178	179			5.0	9.0	0.0	1.0
PAS	6	173	174	174	170	170			1.0	1.0	-3.0	-3.0
PAS	7	179	168	172	170	172			-11.0	-7.0	-9.0	-7.0
PAS	8	170	162	171	160	162			-8.0	1.0	-10.0	-8.0
PAS	9	175	175	175	164	164			0.0	0.0	-11.0	-11.0
PAS	10	164	171	173	165	168			7.0	9.0	1.0	4.0
PAS	11	191	193	190	185	190			2.0	-1.0	-6.0	-1.0
PAS	12	174	175	172	170	170			1.0	-2.0	-4.0	-4.0
	mean	174	175	175	169	170	mean		1.1	1.0	-4.3	-3.3
	SD	7	8	8	7	8	SD		5.5	5.4	4.6	4.4
CWT	1	170	176	169	170	175			6.0	-1.0	0.0	5.0
CWT	2	172	171	169	175	174			-1.0	-3.0	3.0	2.0
CWT	3	170	161	168	165	168			-9.0	-2.0	-5.0	-2.0
CWT	4	171	170	168	170	170			-1.0	-3.0	-1.0	-1.0
CWT	5	180	180	175	178	183			0.0	-5.0	-2.0	3.0
CWT	6	177	174	168	172	171			-3.0	-9.0	-5.0	-6.0
CWT	7	173	168	170	174	167			-5.0	-3.0	1.0	-6.0
CWT	8	169	159	168	167	164			-10.0	-1.0	-2.0	-5.0
CWT	9	161	162	158	170	172			1.0	-3.0	9.0	11.0
CWT	10	167	165	165	168	150			-2.0	-2.0	1.0	-17.0
CWT	11	197	198	185	185	188			1.0	-12.0	-12.0	-9.0
CWT	12	175	172	170	172	185			-3.0	-5.0	-3.0	10.0
	mean	174	171	169	172	172	mean		-2.2	-4.1	-1.3	-1.3
	SD	9	11	6	5	10	SD		4.4	3.3	5.1	8.1
HWI	1	172	168	170	166	167			-4.0	-2.0	-6.0	-5.0
HWI	2	179	176	175	174	170			-3.0	-4.0	-5.0	-9.0
HWI	3	165	165	175	160	152			0.0	10.0	-5.0	-13.0
HWI	4	180	175	176	169	170			-5.0	-4.0	-11.0	-10.0
HWI	5	180	180	174	180	170			0.0	-6.0	0.0	-10.0
HWI	6	173	174	175	169	171			1.0	2.0	-4.0	-2.0
HWI	7	170	170	171	170	177			0.0	1.0	0.0	7.0
HWI	8	160	155	159	154	156			-5.0	-1.0	-6.0	-4.0
HWI	9	175	174	172	170	171			-1.0	-3.0	-5.0	-4.0
HWI	10	159	165	162	150	150			6.0	3.0	-9.0	-9.0
HWI	11	194	193	190	187	185			-1.0	-4.0	-7.0	-9.0
HWI	12	166	160	168	160	165			-6.0	2.0	-6.0	-1.0
	mean	173	171	172	167	167	mean		-1.5	-0.5	-5.3	-5.8
	SD	10	10	8	10	10	SD		3.3	4.4	3.1	5.5
CWI	1	170	172	177	173	172			2.0	7.0	3.0	2.0
CWI	2	172	176	174	174	168			4.0	2.0	2.0	-4.0
CWI	3	170	165	171	171	152			-5.0	1.0	1.0	-18.0
CWI	4	172	175	175	173	170			3.0	3.0	1.0	-2.0
CWI	5	180	184	183	188	178			4.0	3.0	8.0	-2.0
CWI	6	173	173	172	173	172			0.0	-1.0	0.0	-1.0
CWI	7	173	172	175	173	167			-1.0	2.0	0.0	-6.0
CWI	8	168	162	165	168	169			-6.0	-3.0	0.0	1.0
CWI	9	175	174	174	169	172			-1.0	-1.0	-6.0	-3.0
CWI	10	161	162	156	158	156			1.0	-5.0	-3.0	-5.0
CWI	11	193	196	198	195	194			3.0	5.0	2.0	1.0
CWI	12	165	167	164	169	162			2.0	-1.0	4.0	-3.0
	mean	173	173	174	174	169	mean		0.5	1.0	1.0	-3.3
	SD	8	10	10	9	11	SD		3.3	3.4	3.5	5.2

Raw data – Chapter Five

Cold water immersion vs. Passive Recovery – **Isometric Squat Performance (N)**

Raw Data		Trials						Effects			
Treatment	Subject	Pre	Post	24	48	72		Post-Pre	24-Pre	48-Pre	72-Pre
PAS	1	2060.6	1606.3	1924.5	1898.0	1905.7		-454.3	-136.1	-162.6	-154.9
PAS	2	1743.8	1493.3	1216.3	1287.4	1344.8		-250.5	-527.5	-456.4	-399.0
PAS	3	1724.4	1584.6	1441.6	1475.8	1591.3		-139.8	-282.8	-248.6	-133.1
PAS	4	1759.9	1328.5	1537.6	1546.2	1510.3		-431.4	-222.3	-213.7	-249.6
PAS	5	2534.0	2211.2	1910.1	1673.0	1756.8		-322.8	-623.9	-861.0	-777.2
PAS	6	2606.8	2142.5	2469.7	2546.0	2704.1		-464.3	-137.1	-60.8	97.3
PAS	7	1834.5	1517.2	1406.4	1327.8	1325.7		-317.3	-428.1	-506.7	-508.8
PAS	8	2989.6	2591.4	2310.9	2419.0	2563.4		-398.2	-678.7	-570.6	-426.2
PAS	9	2451.6	2172.6	2281.4	2151.5	2365.2		-279.0	-170.2	-300.1	-86.4
PAS	10	1872.7	1313.6	1559.1	1724.7	1741.8		-559.1	-313.6	-148.0	-130.9
PAS	11	1601.9	1423.8	1576.7	1432.4	1520.8		-178.1	-25.2	-169.5	-81.1
PAS	12	1887.1	1531.9	1874.0	1753.6	1987.5		-355.2	-13.1	-133.5	100.4
	mean	2088.9	1743.1	1792.4	1769.6	1859.8	mean	-345.8	-296.6	-319.3	-229.1
	SD	443.1	420.3	401.6	413.0	463.8	SD	123.2	223.8	234.5	257.4
CWI	1	2091.5	1768.9	2017.6	1987.4	2026.8		-322.6	-73.9	-104.1	-64.7
CWI	2	1593.0	1204.2	1281.5	1336.7	1457.5		-388.8	-311.5	-256.3	-135.5
CWI	3	1676.4	1411.4	1548.6	1572.1	1664.8		-265.0	-127.8	-104.3	-11.6
CWI	4	1821.6	1406.4	1687.1	1838.6	1814.7		-415.2	-134.5	17.0	-6.9
CWI	5	2580.2	2185.9	2092.2	2357.4	2527.6		-394.3	-488.0	-222.8	-52.6
CWI	6	2729.2	2213.9	2237.9	2595.7	2697.1		-515.3	-491.3	-133.5	-32.1
CWI	7	1796.9	1455.4	1639.6	1716.2	1727.7		-341.5	-157.3	-80.7	-69.2
CWI	8	3038.5	2627.3	2703.7	2901.0	3011.6		-411.2	-334.8	-137.5	-26.9
CWI	9	2441.3	2003.2	2325.6	2401.8	2449.1		-438.1	-115.7	-39.5	7.8
CWI	10	1901.0	1593.7	1824.2	1889.7	1968.6		-307.3	-76.8	-11.3	67.6
CWI	11	1696.7	1443.9	1338.6	1574.2	1580.1		-252.8	-358.1	-122.5	-116.6
CWI	12	1958.8	1673.2	1829.2	1914.1	1963.7		-285.6	-129.6	-44.7	4.9
	mean	2110.4	1749.0	1877.2	2007.1	2074.1	mean	-361.5	-233.3	-103.4	-36.3
	SD	472.0	424.3	419.0	465.7	487.8	SD	78.9	155.1	80.4	56.0

Hot water immersion vs. Passive Recovery – **Isometric Squat Performance (N)**

Raw Data		Trials					Effects				
Treatment	Subject	Pre	Post	24	48	72		Post-Pre	24-Pre	48-Pre	72-Pre
PAS	1	1677.2	1452.1	1435.5	1411.6	1565.2		-225.1	-241.7	-265.6	-112.0
PAS	2	1412.5	1252.0	1266.8	1023.0	1330.3		-160.5	-145.7	-389.5	-82.2
PAS	3	1989.7	1538.4	1235.4	1549.2	1636.4		-451.3	-754.3	-440.5	-353.3
PAS	4	1716.5	1581.2	1571.2	1530.2	1578.1		-135.3	-145.3	-186.3	-138.4
PAS	5	2130.9	1786.4	1766.5	1762.9	1827.1		-344.5	-364.4	-368.0	-303.8
PAS	6	1852.3	1558.6	1496.2	1719.0	1734.5		-293.7	-356.1	-133.3	-117.8
PAS	7	1679.3	1224.7	1422.3	1392.4	1471.2		-454.6	-257.0	-286.9	-208.1
PAS	8	2487.1	1944.3	2093.6	2014.9	2119.8		-542.8	-393.5	-472.2	-367.3
PAS	9	2025.1	1787.8	1734.2	1772.7	1905.3		-237.3	-290.9	-252.4	-119.8
PAS	10	1611.4	1385.6	1276.1	1392.7	1506.3		-225.8	-335.3	-218.7	-105.1
PAS	11	2490.8	2060.3	2280.4	2218.4	2287.6		-430.5	-210.4	-272.4	-203.2
	mean	1771.2	1481.0	1479.7	1496.8	1599.0	mean	-290.2	-291.6	-274.5	-172.3
	SD	601.4	479.0	524.0	521.6	513.0	SD	161.5	183.8	131.8	120.7
HWI	1	1692.1	1464.5	1576.9	1493.3	1622.1		-227.6	-115.2	-198.8	-70.0
HWI	2	1559.0	1217.2	1473.5	1458.5	1520.5		-341.8	-85.5	-100.5	-38.5
HWI	3	1993.1	1600.6	1342.2	1425.7	1917.6		-392.5	-650.9	-567.4	-75.5
HWI	4	1756.6	1556.8	1701.0	1679.0	1652.1		-199.8	-55.6	-77.6	-104.5
HWI	5	2080.9	1749.2	1876.3	1769.3	2103.4		-331.7	-204.6	-311.6	22.5
HWI	6	1974.0	1646.8	1706.9	1793.7	1893.2		-327.2	-267.1	-180.3	-80.8
HWI	7	1653.6	1222.8	1470.8	1631.6	1643.8		-430.8	-182.8	-22.0	-9.8
HWI	8	2414.7	1986.5	2190.9	2241.3	2288.8		-428.2	-223.8	-173.4	-125.9
HWI	9	2064.1	1664.5	1592.0	1829.3	2052.6		-399.6	-472.1	-234.8	-11.5
HWI	10	1644.9	1402.1	1435.3	1576.7	1547.6		-242.8	-209.6	-68.2	-97.3
HWI	11	2387.4	2005.7	2170.7	2187.6	2312.7		-381.7	-216.7	-199.8	-74.7
	mean	1796.3	1477.7	1561.2	1606.0	1726.7	mean	-318.6	-235.1	-190.3	-69.6
	SD	539.6	469.7	508.8	517.3	564.5	SD	99.7	168.8	143.1	53.6

Contrast Water Therapy vs. Passive Recovery – **Isometric Squat Performance (N)**

Raw Data		Trials						Effects			
Treatment	Subject	Pre	Post	24	48	72		Post-Pre	24-Pre	48-Pre	72-Pre
PAS	1	1010.6	921.2	877.3	840.9	897.7		-89.4	-133.3	-169.7	-112.9
PAS	2	2060.1	1664.2	1661.7	1798.7	1794.0		-395.9	-398.4	-261.4	-266.1
PAS	3	1589.6	1381.2	1423.9	1516.3	1413.8		-208.4	-165.7	-73.3	-175.8
PAS	4	2326.9	2007.2	2018.8	1987.2	2004.6		-319.7	-308.1	-339.7	-322.3
PAS	5	2710.5	2264.2	2234.9	2412.7	2499.6		-446.3	-475.6	-297.8	-210.9
PAS	6	2047.0	1731.7	1683.4	1787.7	1823.6		-315.3	-363.6	-259.3	-223.4
PAS	7	2341.2	2018.4	2128.1	2171.2	2257.8		-322.8	-213.1	-170.0	-83.4
PAS	8	1538.8	1377.0	1103.7	1224.7	1235.8		-161.8	-435.1	-314.1	-303.0
PAS	9	2230.3	1928.7	1817.6	1894.9	1933.8		-301.6	-412.7	-335.4	-296.5
PAS	10	1889.4	1562.7	1426.1	1454.3	1646.9		-326.7	-463.3	-435.1	-242.5
PAS	11	2798.6	2421.3	2150.2	2199.8	2290.0		-377.3	-648.4	-598.8	-508.6
PAS	12	2437.7	1876.7	2050.7	2272.5	2351.5		-561.0	-387.0	-165.2	-86.2
PAS	13	1946.9	1795.7	1662.9	1609.7	1702.4		-151.2	-284.0	-337.2	-244.5
PAS	14	1749.6	1501.4	1486.1	1567.3	1652.7		-248.2	-263.5	-182.3	-96.9
PAS	15	2314.6	1786.7	1941.9	2013.8	1996.4		-527.9	-372.7	-300.8	-318.2
	mean	2066.1	1749.2	1711.2	1783.4	1833.4	mean	-316.9	-355.0	-282.7	-232.7
	SD	469.6	375.2	396.4	424.5	436.0	SD	133.9	132.6	127.6	114.1
CWT	1	1142.7	1097.7	1027.3	1062.7	1173.1		-45.0	-115.4	-80.0	30.4
CWT	2	2055.1	1600.2	1942.3	1926.2	1965.2		-454.9	-112.8	-128.9	-89.9
CWT	3	1631.4	1393.3	1512.9	1564.2	1651.6		-238.1	-118.5	-67.2	20.2
CWT	4	2266.2	1928.7	2066.9	2132.1	2235.6		-337.5	-199.3	-134.1	-30.6
CWT	5	2773.1	2050.1	2353.5	2638.1	2759.3		-723.0	-419.6	-135.0	-13.8
CWT	6	2011.3	1745.4	1789.1	1850.3	2034.6		-265.9	-222.2	-161.0	23.3
CWT	7	2339.6	2033.4	2268.5	2378.7	2524.1		-306.2	-71.1	39.1	184.5
CWT	8	1542.1	1374.5	1301.4	1428.8	1415.1		-167.6	-240.7	-113.3	-127.0
CWT	9	2273.5	1932.7	1970.1	1920.8	2052.9		-340.8	-303.4	-352.7	-220.6
CWT	10	1941.7	1667.8	1628.9	1461.0	1578.8		-273.9	-312.8	-480.7	-362.9
CWT	11	2689.4	2374.6	2511.2	2624.3	2690.1		-314.8	-178.2	-65.1	0.7
CWT	12	2454.1	1891.8	2136.8	2357.5	2494.4		-562.3	-317.3	-96.6	40.3
CWT	13	1916.3	1743.9	1811.7	1823.7	1862.9		-172.4	-104.6	-92.6	-53.4
CWT	14	1663.7	1487.3	1520.8	1587.4	1593.0		-176.4	-142.9	-76.3	-70.7
CWT	15	2327.4	1684.1	2013.7	2093.3	2240.4		-643.3	-313.7	-234.1	-87.0
	mean	2068.5	1733.7	1857.0	1923.3	2018.1	mean	-334.8	-211.5	-145.2	-50.4
	SD	445.9	320.5	405.1	457.2	477.7	SD	187.9	103.3	127.0	125.5

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Cold Water Immersion vs. Passive Recovery – **Squat Jump Performance (W)**

Raw Data		Trials						Effects			
Treatment	Subject	Pre	Post	24	48	72		Post-Pre	24-Pre	48-Pre	72-Pre
PAS	1	3269.4	2917.8	3121.8	3188.9	3037.2		-351.6	-147.6	-80.5	-232.2
PAS	2	3265.7	2189.9	2119.4	2103.9	2897.6		-1075.8	-1146.3	-1161.8	-368.1
PAS	3	4526.0	3731.5	3952.0	3598.1	3851.2		-794.5	-574.0	-927.9	-674.8
PAS	4	2830.2	2092.9	2440.3	2650.9	2756.8		-737.3	-389.9	-179.3	-73.4
PAS	5	5561.0	4643.2	4276.7	3573.1	4768.5		-917.8	-1284.3	-1987.9	-792.5
PAS	6	3664.5	3113.6	3431.4	3452.2	3556.9		-550.9	-233.1	-212.3	-107.6
PAS	7	3768.5	3291.8	3270.3	3379.4	3711.7		-476.7	-498.2	-389.1	-56.8
PAS	8	5865.3	5606.9	5167.6	5123.9	5575.6		-258.4	-697.7	-741.4	-289.7
PAS	9	4930.9	4449.5	4629.7	4669.3	4586.2		-481.4	-301.2	-261.6	-344.7
PAS	10	3611.8	3298.1	3008.4	3139.5	3243.6		-313.7	-603.4	-472.3	-368.2
PAS	11	4613.7	3675.8	3963.4	3674.1	4369.2		-937.9	-650.3	-939.6	-244.5
PAS	12	4136.0	3767.5	3548.8	3541.2	3936.9		-368.5	-587.2	-594.8	-199.1
	mean	4170.3	3564.9	3577.5	3507.9	3857.6	mean	-605.4	-592.8	-662.4	-312.6
	SD	947.0	1001.0	878.1	795.7	846.1	SD	276.9	338.7	540.2	225.2
CWI	1	3188.1	2900.7	3208.7	3197.8	3210.9		-287.4	20.6	9.7	22.8
CWI	2	3145.9	2206.3	2849.4	2916.2	2971.3		-939.6	-296.5	-229.7	-174.6
CWI	3	4493.6	3826.1	3974.3	4121.4	4362.1		-667.5	-519.3	-372.2	-131.5
CWI	4	2994.9	2001.6	2901.1	2890.6	3000.0		-993.3	-93.8	-104.3	5.1
CWI	5	5484.8	4533.2	4468.9	4938.5	5270.0		-951.6	-1015.9	-546.3	-214.8
CWI	6	3722.9	3269.0	3037.6	3500.9	3682.5		-453.9	-685.3	-222.0	-40.4
CWI	7	3727.6	3259.9	3369.3	3652.6	3728.6		-467.7	-358.3	-75.0	1.0
CWI	8	5976.2	5786.5	5812.0	5792.1	5834.8		-189.7	-164.2	-184.1	-141.4
CWI	9	4977.1	4474.8	4681.2	4912.8	5049.3		-502.3	-295.9	-64.3	72.2
CWI	10	3766.0	3321.9	3546.2	3698.6	3724.8		-444.1	-219.8	-67.4	-41.2
CWI	11	4460.7	3548.9	3369.6	3842.3	4308.6		-911.8	-1091.1	-618.4	-152.1
CWI	12	3960.5	3435.6	3604.3	3814.2	3820.1		-524.9	-356.2	-146.3	-140.4
	mean	4158.2	3547.0	3735.2	3939.8	4080.3	mean	-611.2	-423.0	-218.4	-77.9
	SD	945.2	1033.7	872.2	877.3	914.2	SD	276.1	347.7	197.7	91.9

Hot Water Immersion vs. Passive Recovery – **Squat Jump Performance (W)**

Raw Data		Trials						Effects			
Treatment	Subject	Pre	Post	24	48	72		Post-Pre	24-Pre	48-Pre	72-Pre
PAS	1	3463.4	3087.7	3216.4	3221.8	3279.2		-375.7	-247.0	-241.6	-184.2
PAS	2	3676.6	2986.4	3271.2	3415.9	3401.7		-690.2	-405.4	-260.7	-274.9
PAS	3	3676.9	3224.0	2690.1	2957.6	2990.3		-452.9	-986.8	-719.3	-686.6
PAS	4	3979.2	3209.1	2993.9	3131.7	3693.8		-770.1	-985.3	-847.5	-285.4
PAS	5	3890.3	3752.1	3466.2	3266.4	3560.4		-138.2	-424.1	-623.9	-329.9
PAS	6	4052.7	3709.1	3571.6	3320.3	3955.6		-343.6	-481.1	-732.4	-97.1
PAS	7	4272.1	3387.0	3527.8	3745.1	3891.7		-885.1	-744.3	-527.0	-380.4
PAS	8	3986.7	3568.7	3636.9	3947.3	3913.8		-418.0	-349.8	-39.4	-72.9
PAS	9	3976.7	3612.0	4075.6	3793.1	3721.9		-364.7	98.9	-183.6	-254.8
PAS	10	3587.3	3067.8	3012.6	3185.8	3175.0		-519.5	-574.7	-401.5	-412.3
PAS	11	4344.5	3598.6	3952.2	4081.8	4089.5		-745.9	-392.3	-262.7	-255.0
	mean	3900.6	3382.0	3401.3	3460.6	3606.6	mean	-518.5	-499.3	-440.0	-294.0
	SD	277.0	278.5	416.8	369.8	356.2	SD	226.4	317.8	264.9	167.4
HWI	1	3516.7	3161.2	3483.2	3457.7	3245.9		-355.5	-33.5	-59.0	-270.8
HWI	2	3758.2	2883.1	3602.1	3796.6	3620.3		-875.1	-156.1	38.4	-137.9
HWI	3	3589.1	3119.7	2863.7	2834.2	2959.1		-469.4	-725.4	-754.9	-630.0
HWI	4	4028.9	3394.3	3349.1	3646.8	3637.5		-634.6	-679.8	-382.1	-391.4
HWI	5	3790.6	3501.4	3149.2	2820.2	3206.1		-289.2	-641.4	-970.4	-584.5
HWI	6	4288.2	3968.6	3868.3	3925.3	4001.5		-319.6	-419.9	-362.9	-286.7
HWI	7	4199.3	3292.2	3661.3	3548.8	3663.5		-907.1	-538.0	-650.5	-535.8
HWI	8	4123.6	3787.4	3962.8	4042.8	4066.8		-336.2	-160.8	-80.8	-56.8
HWI	9	4049.4	3518.3	3716.4	3662.9	3785.0		-531.1	-333.0	-386.5	-264.4
HWI	10	3408.3	3979.3	2769.1	2816.2	3145.6		571.0	-639.2	-592.1	-262.7
HWI	11	4165.8	3304.9	3631.2	3800.3	4197.1		-860.9	-534.6	-365.5	31.3
	mean	3901.6	3446.4	3459.7	3486.5	3593.5	mean	-455.2	-442.0	-415.1	-308.2
	SD	302.9	351.1	389.6	455.7	409.0	SD	413.1	239.4	310.1	212.6

Contrast Water Therapy vs. Passive Recovery – **Squat Jump Performance (W)**

Raw Data		Trials						Effects			
Treatment	Subject	Pre	Post	24	48	72		Post-Pre	24-Pre	48-Pre	72-Pre
PAS	1	1968.3	1669.5	1819.8	1798.8	2038.0		-298.8	-148.5	-169.5	69.7
PAS	2	3592.1	3118.9	2741.1	2865.1	2918.9		-473.2	-851.0	-727.0	-673.2
PAS	3	3423.6	2895.7	3180.4	3290.9	3314.2		-527.9	-243.2	-132.7	-109.4
PAS	4	5106.3	4334.1	3921.1	3824.7	4510.7		-772.2	-1185.2	-1281.6	-595.6
PAS	5	4722.6	3875.9	3525.7	4295.5	4581.4		-846.7	-1196.9	-427.1	-141.2
PAS	6	3741.9	3108.7	3127.3	3421.3	3344.7		-633.2	-614.6	-320.6	-397.2
PAS	7	5485.1	5003.4	4996.2	5164.7	5251.9		-481.7	-488.9	-320.4	-233.2
PAS	8	4211.6	3891.4	3747.8	3861.7	3812.0		-320.2	-463.8	-349.9	-399.6
PAS	9	3559.8	3252.2	2828.6	2897.8	3305.0		-307.6	-731.2	-662.0	-254.8
PAS	10	3853.5	3332.7	3719.9	3461.1	3662.7		-520.8	-133.6	-392.4	-190.8
PAS	11	4615.0	4268.5	4025.8	3906.7	3928.1		-346.5	-589.2	-708.3	-686.9
PAS	12	4598.1	3859.6	3759.8	3426.9	4268.8		-738.5	-838.3	-1171.2	-329.3
PAS	13	4172.4	3735.7	3756.7	3754.3	3823.7		-436.7	-415.7	-418.1	-348.7
PAS	14	3138.1	2995.9	2539.9	2940.3	2939.5		-142.2	-598.2	-197.8	-198.6
PAS	15	3356.5	2857.6	3154.1	3181.8	3199.1		-498.9	-202.4	-174.7	-157.4
	mean	3969.7	3480.0	3389.6	3472.8	3659.9	mean	-489.7	-580.0	-496.9	-309.7
	SD	878.9	792.4	750.5	755.5	795.3	SD	195.5	338.2	352.0	214.6
CWT	1	2117.7	1985.7	2292.6	2269.1	2223.4		-132.0	174.9	151.4	105.7
CWT	2	3547.1	2461.0	3203.7	3018.6	3276.6		-1086.1	-343.4	-528.5	-270.5
CWT	3	3242.2	2623.9	3152.8	3179.6	3286.4		-618.3	-89.4	-62.6	44.2
CWT	4	4995.4	3744.9	4011.2	4633.1	4701.6		-1250.5	-984.2	-362.3	-293.8
CWT	5	4743.5	3943.5	4504.5	4691.3	4698.4		-800.0	-239.0	-52.2	-45.1
CWT	6	3949.0	3150.8	3559.6	3608.2	3727.9		-798.2	-389.4	-340.8	-221.1
CWT	7	5433.1	5057.2	5217.3	5436.6	5446.2		-375.9	-215.8	3.5	13.1
CWT	8	4181.9	3903.8	3996.7	4165.1	4161.3		-278.1	-185.2	-16.8	-20.6
CWT	9	3479.8	3101.9	3408.9	3443.5	3586.1		-377.9	-70.9	-36.3	106.3
CWT	10	4080.2	3485.0	4038.6	3978.9	4186.4		-595.2	-41.6	-101.3	106.2
CWT	11	4631.1	3944.8	4039.3	4408.1	4588.6		-686.3	-591.8	-223.0	-42.5
CWT	12	4686.3	4037.2	4367.8	4303.2	4597.1		-649.1	-318.5	-383.1	-89.2
CWT	13	3706.8	3205.5	3492.6	3620.9	3878.3		-501.3	-214.2	-85.9	171.5
CWT	14	3249.7	2970.9	2977.2	3412.3	3535.9		-278.8	-272.5	162.6	286.2
CWT	15	3032.8	2301.2	2865.1	2905.7	3161.3		-731.6	-167.7	-127.1	128.5
	mean	3938.4	3327.8	3675.2	3804.9	3937.0	mean	-610.6	-263.2	-133.5	-1.4
	SD	871.3	806.7	741.3	821.2	808.2	SD	304.7	264.8	197.7	165.7

Cold Water Immersion vs. Passive Recovery – **Mid Thigh Circumference (cm)**

Raw Data		Trials						Effects			
Treatment	Subject	Pre	Post	24	48	72		Post-Pre	24-Pre	48-Pre	72-Pre
PAS	1	54.3	54.8	55.2	55.0	55.2		0.5	0.9	0.7	0.9
PAS	2	48.1	48.8	49.6	49.1	48.8		0.7	1.5	1.0	0.7
PAS	3	55.6	56.2	57.1	57.4	56.2		0.6	1.5	1.8	0.6
PAS	4	58.5	59.0	59.7	59.7	59.2		0.5	1.2	1.2	0.7
PAS	5	58.2	59.1	59.6	59.5	59.0		0.9	1.4	1.3	0.8
PAS	6	61.5	61.9	61.6	61.6	61.5		0.4	0.1	0.1	0.0
PAS	7	57.8	57.8	58.9	58.8	58.3		0.0	1.1	1.0	0.5
PAS	8	60.4	61.1	61.3	61.1	61.0		0.7	0.9	0.7	0.6
PAS	9	56.4	56.7	57.2	56.5	57.1		0.3	0.8	0.1	0.7
PAS	10	55.2	55.8	56.1	56.2	55.4		0.6	0.9	1.0	0.2
PAS	11	56.1	56.8	56.6	56.2	56.2		0.7	0.5	0.1	0.1
PAS	12	57.1	57.6	57.8	58.1	58.0		0.5	0.7	1.0	0.9
	mean	56.6	57.1	57.6	57.4	57.2	mean	0.5	1.0	0.8	0.6
	SD	3.4	3.4	3.2	3.3	3.3	SD	0.2	0.4	0.5	0.3
CWI	1	54.9	55.3	55.1	55.1	55.0		0.4	0.2	0.2	0.1
CWI	2	47.0	47.6	47.4	47.2	47.0		0.6	0.4	0.2	0.0
CWI	3	55.3	55.9	55.6	55.4	55.4		0.6	0.3	0.1	0.1
CWI	4	59.8	60.2	60.0	59.9	59.9		0.4	0.2	0.1	0.1
CWI	5	58.0	58.9	58.5	58.1	58.2		0.9	0.5	0.1	0.2
CWI	6	61.7	62.4	62.8	61.9	61.9		0.7	1.1	0.2	0.2
CWI	7	57.7	58.2	58.0	57.9	57.8		0.5	0.3	0.2	0.1
CWI	8	60.8	61.6	61.2	61.1	60.9		0.8	0.4	0.3	0.1
CWI	9	56.5	57.9	56.7	56.6	56.6		1.4	0.2	0.1	0.1
CWI	10	55.0	55.9	55.7	55.3	55.1		0.9	0.7	0.3	0.1
CWI	11	56.2	57.1	57.0	57.1	57.0		0.9	0.8	0.9	0.8
CWI	12	57.3	57.8	57.3	57.3	57.4		0.5	0.0	0.0	0.1
	mean	56.7	57.4	57.1	56.9	56.9	mean	0.7	0.4	0.2	0.2
	SD	3.8	3.8	3.8	3.8	3.8	SD	0.3	0.3	0.2	0.2

Hot Water Immersion vs. Passive Recovery – **Mid Thigh Circumference (cm)**

Raw Data		Trials						Effects			
Treatment	Subject	Pre	Post	24	48	72		Post-Pre	24-Pre	48-Pre	72-Pre
PAS	1	54.3	54.6	54.8	54.7	54.6		0.3	0.5	0.4	0.3
PAS	2	56.5	57.1	57.0	57.1	56.7		0.6	0.5	0.6	0.2
PAS	3	53.3	53.9	54.1	53.8	53.6		0.6	0.8	0.5	0.3
PAS	4	55.7	56.3	56.1	56.3	55.9		0.6	0.4	0.6	0.2
PAS	5	61.3	61.6	61.7	61.5	61.4		0.3	0.4	0.2	0.1
PAS	6	57.8	58.1	58.6	58.0	58.0		0.3	0.8	0.2	0.2
PAS	7	59.0	59.3	59.6	59.9	59.8		0.3	0.6	0.9	0.8
PAS	8	62.9	63.6	63.4	63.1	63.1		0.7	0.5	0.2	0.2
PAS	9	61.2	61.9	62.4	62.5	62.1		0.7	1.2	1.3	0.9
PAS	10	50.8	51.3	51.5	51.4	51.2		0.5	0.7	0.6	0.4
PAS	11	58.6	59.4	59.8	59.2	59.3		0.8	1.2	0.6	0.7
	mean	57.4	57.9	58.1	58.0	57.8	mean	0.5	0.7	0.6	0.4
	SD	3.7	3.7	3.7	3.7	3.8	SD	0.2	0.3	0.3	0.3
HWI	1	53.2	53.7	53.9	53.6	53.5		0.5	0.7	0.4	0.3
HWI	2	56.3	56.6	56.9	56.9	56.7		0.3	0.6	0.6	0.4
HWI	3	53.5	53.9	53.9	53.7	53.6		0.4	0.4	0.2	0.1
HWI	4	55.9	56.3	56.5	56.3	56.0		0.4	0.6	0.4	0.1
HWI	5	61.0	61.8	62.1	61.8	61.4		0.8	1.1	0.8	0.4
HWI	6	58.4	58.7	59.6	58.9	58.2		0.3	1.2	0.5	-0.2
HWI	7	59.0	59.5	60.1	60.0	60.0		0.5	1.1	1.0	1.0
HWI	8	62.7	63.0	62.9	62.7	62.8		0.3	0.2	0.0	0.1
HWI	9	61.0	61.7	62.1	62.0	61.4		0.7	1.1	1.0	0.4
HWI	10	50.6	51.2	51.2	51.0	50.9		0.6	0.6	0.4	0.3
HWI	11	58.7	59.4	59.3	59.5	58.9		0.7	0.6	0.8	0.2
	mean	57.3	57.8	58.0	57.9	57.6	mean	0.5	0.7	0.6	0.3
	SD	3.8	3.8	3.9	3.9	3.8	SD	0.2	0.3	0.3	0.3

Contrast Water Therapy vs. Passive Recovery – **Mid Thigh Circumference (cm)**

Raw Data		Trials						Effects			
Treatment	Subject	Pre	Post	24	48	72		Post-Pre	24-Pre	48-Pre	72-Pre
PAS	1	44.6	44.9	45.1	44.9	44.7		0.3	0.5	0.3	0.1
PAS	2	54.5	55.2	55.3	55.1	55.0		0.7	0.8	0.6	0.5
PAS	3	54.4	54.7	55.1	54.6	54.4		0.3	0.7	0.2	0.0
PAS	4	55.8	56.7	56.9	57.4	57.3		0.9	1.1	1.6	1.5
PAS	5	59.0	59.8	59.9	59.7	59.4		0.8	0.9	0.7	0.4
PAS	6	54.1	54.7	54.7	54.9	54.5		0.6	0.6	0.8	0.4
PAS	7	58.6	59.2	59.1	59.0	58.7		0.6	0.5	0.4	0.1
PAS	8	55.5	55.9	56.1	56.0	55.8		0.4	0.6	0.5	0.3
PAS	9	57.4	57.8	58.3	58.5	57.9		0.4	0.9	1.1	0.5
PAS	10	56.4	57.2	57.2	57.4	57.1		0.8	0.8	1.0	0.7
PAS	11	66.0	66.4	66.9	67.2	67.0		0.4	0.9	1.2	1.0
PAS	12	61.1	61.9	62.3	61.9	61.8		0.8	1.2	0.8	0.7
PAS	13	55.1	55.4	56.3	55.9	55.6		0.3	1.2	0.8	0.5
PAS	14	53.8	54.2	54.1	54.0	53.9		0.4	0.3	0.2	0.1
PAS	15	56.6	56.9	57.2	57.3	57.1		0.3	0.6	0.7	0.5
	mean	56.2	56.7	57.0	56.9	56.7	mean	0.5	0.8	0.7	0.5
	SD	4.5	4.6	4.7	4.7	4.7	SD	0.2	0.3	0.4	0.4
CWT	1	44.7	44.9	45.1	44.8	44.9		0.2	0.4	0.1	0.2
CWT	2	54.6	56.0	54.8	54.7	54.7		1.4	0.2	0.1	0.1
CWT	3	54.2	54.8	54.5	54.4	54.3		0.6	0.3	0.2	0.1
CWT	4	56.2	57.1	56.6	56.4	56.3		0.9	0.4	0.2	0.1
CWT	5	59.2	59.6	59.4	59.3	59.2		0.4	0.2	0.1	0.0
CWT	6	54.3	54.6	55.1	54.4	54.4		0.3	0.8	0.1	0.1
CWT	7	58.4	59.3	58.6	58.5	58.5		0.9	0.2	0.1	0.1
CWT	8	55.7	56.1	55.8	55.6	55.8		0.4	0.1	-0.1	0.1
CWT	9	57.2	58.0	57.7	57.1	57.2		0.8	0.5	-0.1	0.0
CWT	10	56.3	56.7	56.8	56.6	56.4		0.4	0.5	0.3	0.1
CWT	11	66.2	66.9	66.4	66.2	66.3		0.7	0.2	0.0	0.1
CWT	12	60.9	61.6	61.1	61.4	61.1		0.7	0.2	0.5	0.2
CWT	13	54.9	55.3	54.8	54.8	54.8		0.4	-0.1	-0.1	-0.1
CWT	14	53.7	53.9	53.7	53.8	53.8		0.2	0.0	0.1	0.1
CWT	15	56.7	56.9	56.8	56.7	56.7		0.2	0.1	0.0	0.0
	mean	56.2	56.8	56.5	56.3	56.3	mean	0.6	0.3	0.1	0.1
	SD	4.5	4.7	4.5	4.5	4.5	SD	0.3	0.2	0.2	0.1

Cold Water Immersion vs. Passive Recovery – **VAS (0 = no pain, 10 = extremely sore)**

Raw Data		Trials						Effects			
Treatment	Subject	Pre	Post	24	48	72		post-pre	24-pre	48-Pre	72-Pre
PAS	1	0	4	5	8	3		4	5	8	3
PAS	2	0	3	9	9	6		3	9	9	6
PAS	3	0	0	5	10	7		0	5	10	7
PAS	4	0	7	5	7	2		7	5	7	2
PAS	5	0	0	8	10	5		0	8	10	5
PAS	6	0	2	2	3	2		2	2	3	2
PAS	7	0	5	7	8	5		5	7	8	5
PAS	8	0	4	7	8	6		4	7	8	6
PAS	9	0	5	7	6	4		5	7	6	4
PAS	10	0	4	7	9	7		4	7	9	7
PAS	11	0	4	6	9	6		4	6	9	6
PAS	12	0	3	5	4	3		3	5	4	3
	mean	0	3	6	8	5	mean	3	6	8	5
	SD	0	2	2	2	2	SD	2	2	2	2
CWI	1	0	4	3	5	2		4	3	5	2
CWI	2	0	1	8	10	7		1	8	10	7
CWI	3	0	4	5	8	6		4	5	8	6
CWI	4	0	7	5	6	3		7	5	6	3
CWI	5	0	1	3	6	3		1	3	6	3
CWI	6	0	2	5	8	2		2	5	8	2
CWI	7	0	1	2	3	1		1	2	3	1
CWI	8	0	4	6	7	3		4	6	7	3
CWI	9	0	5	5	5	2		5	5	5	2
CWI	10	0	3	7	8	5		3	7	8	5
CWI	11	0	5	6	9	7		5	6	9	7
CWI	12	0	3	7	6	5		3	7	6	5
	mean	0	3	5	7	4	mean	3	5	7	4
	SD	0	2	2	2	2	SD	2	2	2	2

Hot Water Immersion vs. Passive Recovery – **VAS (0 = no pain, 10 = extremely sore)**

Raw Data		Trials						Effects			
Treatment	Subject	Pre	Post	24	48	72		Post-Pre	24-Pre	48-Pre	72-Pre
PAS	1	0	3	6	4	3		3	6	4	3
PAS	2	0	3	6	8	5		3	6	8	5
PAS	3	0	3	5	5	4		3	5	5	4
PAS	4	0	3	8	8	7		3	8	8	7
PAS	5	0	5	5	8	4		5	5	8	4
PAS	6	0	7	6	6	4		7	6	6	4
PAS	7	0	4	6	8	6		4	6	8	6
PAS	8	0	4	10	9	5		4	10	9	5
PAS	9	0	2	5	8	5		2	5	8	5
PAS	10	0	2	7	6	3		2	7	6	3
PAS	11	0	5	9	9	5		5	9	9	5
	mean	0	4	7	7	5	mean	4	7	7	5
	SD	0	1	2	2	1	SD	1	2	2	1
HWI	1	0	3	5	3	2		3	5	3	2
HWI	2	0	1	6	5	3		1	6	5	3
HWI	3	0	3	4	6	4		3	4	6	4
HWI	4	0	7	9	8	8		7	9	8	8
HWI	5	0	5	7	8	6		5	7	8	6
HWI	6	0	6	7	6	5		6	7	6	5
HWI	7	0	5	7	8	7		5	7	8	7
HWI	8	0	5	7	4	3		5	7	4	3
HWI	9	0	2	6	9	5		2	6	9	5
HWI	10	0	2	5	6	3		2	5	6	3
HWI	11	0	2	8	9	2		2	8	9	2
	mean	0	4	6	7	4	mean	4	6	7	4
	SD	0	2	1	2	2	SD	2	1	2	2

Contrast Water Therapy vs. Passive Recovery – **VAS (0 = no pain, 10 = extremely sore)**

Raw Data		Trials					Effects			
Treatment	Subject	Pre	Post	24	48	72		Post-Pre	24-Pre	48-Pre	72-Pre
PAS	1	0	2	6	9	3		2	6	9	3
PAS	2	0	4	5	6	4		4	5	6	4
PAS	3	0	3	5	7	5		3	5	7	5
PAS	4	0	9	6	5	2		9	6	5	2
PAS	5	0	6	9	9	8		6	9	9	8
PAS	6	0	1	6	9	4		1	6	9	4
PAS	7	0	6	8	9	8		6	8	9	8
PAS	8	0	5	8	8	6		5	8	8	6
PAS	9	0	5	8	10	7		5	8	10	7
PAS	10	0	3	7	10	9		3	7	10	9
PAS	11	0	3	5	9	7		3	5	9	7
PAS	12	0	5	8	9	7		5	8	9	7
PAS	13	0	5	3	6	4		5	3	6	4
PAS	14	0	2	6	4	4		2	6	4	4
PAS	15	0	2	8	6	4		2	8	6	4
	mean	0	4	6	8	5	mean	4	6	8	5
	SD	0	2	2	2	2	SD	2	2	2	2
CWT	1	0	3	5	7	2		3	5	7	2
CWT	2	0	4	4	5	3		4	4	5	3
CWT	3	0	3	5	7	3		3	5	7	3
CWT	4	0	8	5	4	3		8	5	4	3
CWT	5	0	4	6	9	8		4	6	9	8
CWT	6	0	2	4	6	2		2	4	6	2
CWT	7	0	1	6	5	4		1	6	5	4
CWT	8	0	3	5	4	3		3	5	4	3
CWT	9	0	3	8	7	7		3	8	7	7
CWT	10	0	7	8	9	6		7	8	9	6
CWT	11	0	3	5	7	5		3	5	7	5
CWT	12	0	5	8	8	5		5	8	8	5
CWT	13	0	2	4	5	3		2	4	5	3
CWT	14	0	3	5	4	3		3	5	4	3
CWT	15	0	3	3	3	2		3	3	3	2
	mean	0	4	5	6	4	mean	4	5	6	4
	SD	0	2	1	2	2	SD	2	1	2	2

Cold Water Immersion vs. Passive Recovery – **Creatine Kinase (U/L)**

Raw Data		Trials						Effects			
Treatment	Subject	Pre	Post	24	48	72		Post-Pre	24-Pre	48-Pre	72-Pre
PAS	1	130	130	238	183	165		0	108	53	35
PAS	2	72	51	49	48	76		-21	-23	-24	4
PAS	3	82	91	157	141	71		9	75	59	-11
PAS	4	96	90	162	178	172		-6	66	82	76
PAS	5	188	203	400	496	1056		15	212	308	868
PAS	6	151	182	1047	502	401		31	896	351	250
PAS	7	42	62	115	49	62		20	73	7	20
PAS	8	152	214	255	110	134		62	103	-42	-18
PAS	9	153	131	176	116	83		-22	23	-37	-70
PAS	10	57	61	139	72	67		4	82	15	10
PAS	11	149	172	350	464	502		23	201	315	353
PAS	12	126	144	157	125	157		18	31	-1	31
	mean	117	128	270	207	246	mean	11	154	91	129
	SD	46	57	264	175	291	SD	23	243	146	262
CWI	1	848	648	552	722	627		-200	-296	-126	-221
CWI	2	47	53	61	48	55		6	14	1	8
CWI	3	71	83	179	128	47		12	108	57	-24
CWI	4	198	211	143	92	83		13	-55	-106	-115
CWI	5	78	74	104	120	62		-4	26	42	-16
CWI	6	263	277	509	84	215		14	246	-179	-48
CWI	7	121	57	87	54	48		-64	-34	-67	-73
CWI	8	79	129	94	75	76		50	15	-4	-3
CWI	9	61	57	73	44	61		-4	12	-17	0
CWI	10	272	281	306	204	156		9	34	-68	-116
CWI	11	311	364	471	789	1192		53	160	478	881
CWI	12	328	209	200	178	147		-119	-128	-150	-181
	mean	223	204	232	212	231	mean	-20	9	-12	8
	SD	223	175	182	259	343	SD	74	137	171	285

Hot Water Immersion vs. Passive Recovery – **Creatine Kinase (U/L)**

Raw Data		Trials						Effects			
Treatment	Subject	Pre	Post	24	48	72		Post-Pre	24-Pre	48-Pre	72-Pre
PAS	1	414	426	633	5845	184		12	219	5431	-230
PAS	2	202	121	129	202	107		-81	-73	0	-95
PAS	3	122	247	844	419	327		125	722	297	205
PAS	4	92	119	471	254	278		27	379	162	186
PAS	5	87	130	390	212	125		43	303	125	38
PAS	6	39	56	158	107	71		17	119	68	32
PAS	7	47	57	68	104	63		10	21	57	16
PAS	8	138	146	266	194	147		8	128	56	9
PAS	9	207	235	283	183	123		28	76	-24	-84
PAS	10	131	138	436	256	177		7	305	125	46
PAS	11	100	149	745	454	263		49	645	354	163
	mean	144	166	402	748	170	mean	22	259	605	26
	SD	105	105	255	1694	87	SD	48	249	1605	130

HWI	1	378	404	542	263	247		26	164	-115	-131
HWI	2	873	1476	833	837	232		603	-40	-36	-641
HWI	3	120	157	212	147	120		37	92	27	0
HWI	4	47	78	102	109	124		31	55	62	77
HWI	5	120	129	167	128	119		9	47	8	-1
HWI	6	54	68	233	76	73		14	179	22	19
HWI	7	52	77	104	76	128		25	52	24	76
HWI	8	125	94	242	166	137		-31	117	41	12
HWI	9	183	211	187	142	122		28	4	-41	-61
HWI	10	118	127	631	387	232		9	513	269	114
HWI	11	129	140	184	152	131		11	55	23	2
	mean	200	269	312	226	151	mean	69	113	26	-49
	SD	241	411	242	222	58	SD	178	147	94	208

Contrast Water Therapy vs. Passive Recovery – **Creatine Kinase (U/L)**

Raw Data		Trials						Effects			
Treatment	Subject	Pre	Post	24	48	72		Post-Pre	24-Pre	48-Pre	72-Pre
PAS	1	48	1	108	98	82		-47	60	50	34
PAS	2	191	303	739	424	364		112	548	233	173
PAS	3	115	290	891	420	356		175	776	305	241
PAS	4	113	121	1275	344	444		8	1162	231	331
PAS	5	158	157	446	250	104		-1	288	92	-54
PAS	6	202	295	1688	636	586		93	1486	434	384
PAS	7	62	169	163	83	71		107	101	21	9
PAS	8	111	106	154	146	139		-5	43	35	28
PAS	9	238	233	502	243	190		-5	264	5	-48
PAS	10	166	208	496	276	145		42	330	110	-21
PAS	11	246	269	664	331	150		23	418	85	-96
PAS	12	266	243	223	128	110		-23	-43	-138	-156
PAS	13	744	957	1176	801	385		213	432	57	-359
PAS	14	231	166	2290	1195	874		-65	2059	964	643
PAS	15	283	212	404	105	150		-71	121	-178	-133
	mean	212	249	748	365	277	mean	37	536	154	65
	SD	164	212	625	307	227	SD	86	600	274	251
CWT	1	109	115	122	112	104		6	13	3	-5
CWT	2	144	218	485	291	199		74	341	147	55
CWT	3	193	198	648	429	349		5	455	236	156
CWT	4	308	619	1136	412	565		311	828	104	257
CWT	5	88	87	158	124	88		-1	70	36	0
CWT	6	182	323	1719	795	425		141	1537	613	243
CWT	7	155	168	120	119	117		13	-35	-36	-38
CWT	8	124	134	152	106	178		10	28	-18	54
CWT	9	163	202	1016	809	779		39	853	646	616
CWT	10	103	118	254	147	269		15	151	44	166
CWT	11	222	248	386	275	318		26	164	53	96
CWT	12	173	167	219	133	177		-6	46	-40	4
CWT	13	126	133	189	133	77		7	63	7	-49
CWT	14	353	447	4225	2282	1681		94	3872	1929	1328
CWT	15	217	232	133	98	57		15	-84	-119	-160
	mean	177	227	731	418	359	mean	50	553	240	182
	SD	74	142	1075	568	417	SD	83	1020	518	366

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Cold Water Immersion vs. Passive Recovery – **Myoglobin (ng/mL)**

Raw Data		Trials					Effects	
Treatment	Subject	Pre	Post	24			Post-Pre	24-Pre
PAS	1	27.1	79.9	49.9			52.8	22.8
PAS	2	15.4	23.9	14.1			8.5	-1.3
PAS	3	22.6	57.3	37.2			34.7	14.6
PAS	4	31.5	86.7	41.8			55.2	10.3
PAS	5	23.9	78.6	57.6			54.7	33.7
PAS	6	23.8	124.0	53.4			100.2	29.6
PAS	7	34.5	68.2	42.7			33.7	8.2
PAS	8	29.9	48.9	50.7			19.0	20.8
PAS	9	21.5	49.5	31.3			28.0	9.8
PAS	10	21.0	53.1	23.9			32.1	2.9
PAS	11	30.0	76.0	25.3			46.0	-4.7
PAS	12	44.9	63.3	34.4			18.4	-10.5
	mean	27.2	67.5	38.5		mean	40.3	11.4
	SD	7.7	24.9	13.3		SD	24.2	13.6

							Effects	
CWI	1	77.8	99.8	119			22.0	41.2
CWI	2	21.6	78.6	57.6			57.0	36.0
CWI	3	18.4	34.4	33.1			16.0	14.7
CWI	4	34.0	38.9	33.7			4.9	-0.3
CWI	5	22.3	36.7	34.3			14.4	12.0
CWI	6	56.5	68.7	50.4			12.2	-6.1
CWI	7	33.2	41.0	37.2			7.8	4.0
CWI	8	26.4	33.5	40.3			7.1	13.9
CWI	9	26.9	29.9	28.0			3.0	1.1
CWI	10	27.4	67.2	22.3			39.8	-5.1
CWI	11	38.3	129.0	49.6			90.7	11.3
CWI	12	54.2	71.1	34.1			16.9	-20.1
	mean	36.4	60.7	45.0		mean	24.3	8.6
	SD	17.8	31.0	25.4		SD	26.1	17.3

Hot Water Immersion vs. Passive Recovery – **Myoglobin (ng/mL)**

Raw Data		Trials			Effects		
Treatment	Subject	Pre	Post	24		Post-Pre	24-Pre
PAS	1	27.7	28.4	108.0		0.7	80.3
PAS	2	40.1	45.4	38.4		5.3	-1.7
PAS	3	18.8	187.0	55.1		168.2	36.3
PAS	4	33.9	224.0	56.7		190.1	22.8
PAS	5	30.5	73.8	47.7		43.3	17.2
PAS	6	15.9	57.2	27.2		41.3	11.3
PAS	7	24.3	33.8	25.8		9.5	1.5
PAS	8	36.0	51.4	50.1		15.4	14.1
PAS	9	27.6	61.9	35.3		34.3	7.7
PAS	10	27.7	58.6	44.1		30.9	16.4
PAS	11	17.8	1.2	32.3		-16.6	14.5
	mean	27.3	74.8	47.3	mean	47.5	20.0
	SD	7.7	68.0	22.7	SD	67.8	22.4

HWI	1	38.6	63.9	39.8		25.3	1.2
HWI	2	97.6	157.0	102.0		59.4	4.4
HWI	3	45.2	20.6	24.1		-24.6	-21.1
HWI	4	23.0	62.8	34.3		39.8	11.3
HWI	5	34.2	45.8	24.6		11.6	-9.6
HWI	6	32.6	142.0	30.6		109.4	-2.0
HWI	7	19.7	43.8	27.9		24.1	8.2
HWI	8	43.3	68.3	48.2		25.0	4.9
HWI	9	19.2	36.5	21.9		17.3	2.7
HWI	10	18.6	44.5	56.2		25.9	37.6
HWI	11	19.9	30.5	28.1		10.6	8.2
	mean	35.6	65.1	39.8	mean	29.4	4.2
	SD	22.9	44.3	23.2	SD	33.5	14.4

Contrast Water Therapy vs. Passive Recovery – **Myoglobin (ng/mL)**

Raw Data		Trials					Effects	
Treatment	Subject	Pre	Post	24			Post-Pre	24-Pre
PAS	1	18.5	23.1	16.5			4.6	-2.0
PAS	2	22.1	141.0	50.6			118.9	28.5
PAS	3	14.7	56.0	20.7			41.3	6.0
PAS	4	32.7	148.0	74.5			115.3	41.8
PAS	5	46.8	65.5	59.3			18.7	12.5
PAS	6	37.1	129.0	110.0			91.9	72.9
PAS	7	50.5	56.0	38.2			5.5	-12.3
PAS	8	34.1	28.7	23.8			-5.4	-10.3
PAS	9	46.9	98.3	77.2			51.4	30.3
PAS	10	24.3	84.1	38.3			59.8	14.0
PAS	11	82.1	205.0	116.0			122.9	33.9
PAS	12	47.5	75.5	73.7			28.0	26.2
PAS	13	173.0	435.0	116.0			262.0	-57.0
PAS	14	36.3	127.0	208.0			90.7	171.7
PAS	15	41.4	73.4	23.0			32.0	-18.4
	mean	47.2	116.4	69.7		mean	69.2	22.5
	SD	38.5	100.9	51.5		SD	68.8	51.2
CWT	1	31.9	33.2	21.4			1.3	-10.5
CWT	2	29.2	88.2	28.5			59.0	-0.7
CWT	3	28.6	32.9	26.8			4.3	-1.8
CWT	4	77.0	121.0	135.0			44.0	58.0
CWT	5	26.2	59.8	65.6			33.6	39.4
CWT	6	49.7	239.0	106.0			189.3	56.3
CWT	7	28.9	58.2	26.0			29.3	-2.9
CWT	8	24.9	33.9	31.2			9.0	6.3
CWT	9	31.3	107.0	113.0			75.7	81.7
CWT	10	16.7	50.3	22.1			33.6	5.4
CWT	11	63.3	118.0	76.5			54.7	13.2
CWT	12	55.6	71.3	73.1			15.7	17.5
CWT	13	51.5	52.9	51.4			1.4	-0.1
CWT	14	93.9	296.0	195.0			202.1	101.1
CWT	15	55.2	74.9	27.6			19.7	-27.6
	mean	44.3	95.8	66.6		mean	51.5	22.4
	SD	21.9	76.2	51.2		SD	62.6	36.8

Cold Water Immersion vs. Passive Recovery – **Interleukin-6 (pg/mL)**

Raw Data		Trials		
Treatment	Subject	Pre	Post	24
PAS	1	4.48	5.32	2.63
PAS	2	1.73	1.14	0.48
PAS	3	9.43	12.70	9.99
PAS	4	2.48	4.25	3.51
PAS	5	2.04	3.58	3.33
PAS	6	1.45	1.96	1.79
PAS	7	1.55	1.24	1.12
PAS	8	2.12	1.65	3.96
PAS	9	1.52	1.34	0.77
PAS	10	1.12	1.74	1.36
PAS	11	1.15	3.15	1.74
PAS	12	2.45	2.75	2.82
	mean	2.63	3.40	2.79
	SD	2.32	3.21	2.53

Effects		
	Post-Pre	24-Pre
	0.84	-1.85
	-0.59	-1.25
	3.27	0.56
	1.77	1.03
	1.54	1.29
	0.51	0.34
	-0.31	-0.43
	-0.47	1.84
	-0.18	-0.75
	0.62	0.24
	2.00	0.59
	0.30	0.37
mean	0.78	0.17
SD	1.17	1.07

		Trials		
HWI	1	8.99	12.30	8.28
HWI	2	2.05	1.63	2.32
HWI	3	14.00	23.90	22.80
HWI	4	1.35	1.72	1.50
HWI	5	0.78	1.00	1.19
HWI	6	2.24	1.59	1.46
HWI	7	1.24	1.32	1.12
HWI	8	1.70	2.45	0.75
HWI	9	1.67	1.14	1.05
HWI	10	3.18	2.48	1.46
HWI	11	3.00	2.27	1.96
HWI	12	3.33	2.48	1.20
	mean	3.63	4.52	3.76
	SD	3.91	6.83	6.33

	Post-Pre	24-Pre
	3.31	-0.71
	-0.42	0.27
	9.90	8.80
	0.37	0.15
	0.22	0.41
	-0.65	-0.78
	0.08	-0.12
	0.75	-0.95
	-0.53	-0.62
	-0.70	-1.72
	-0.73	-1.04
	-0.85	-2.13
mean	0.90	0.13
SD	3.06	2.84

Hot Water Immersion vs. Passive Recovery – **Interleukin-6 (pg/mL)**

Raw Data		Trials		
Treatment	Subject	Pre	Post	24
PAS	1	2.28	1.37	3.59
PAS	2	6.85	9.45	8.01
PAS	3	8.49	3.53	1.97
PAS	4	3.45	1.80	2.51
PAS	5	2.63	6.62	2.43
PAS	6	1.06	1.75	2.20
PAS	7	1.15	0.91	1.09
PAS	8	0.52	0.90	1.43
PAS	9	0.17	1.09	0.84
PAS	10	1.02	0.54	0.83
PAS	11	0.96	1.77	1.30
	mean	2.60	2.70	2.38
	SD	2.71	2.82	2.05

Effects		
	Post-Pre	24-Pre
	-0.91	1.31
	2.60	1.16
	-4.96	-6.52
	-1.65	-0.94
	3.99	-0.20
	0.69	1.14
	-0.24	-0.06
	0.38	0.91
	0.92	0.67
	-0.48	-0.19
	0.81	0.34
mean	0.10	-0.22
SD	2.30	2.21

	Subject	Pre	Post	24
HWI	1	1.44	1.62	1.70
HWI	2	1.84	3.27	2.57
HWI	3	2.23	1.81	2.99
HWI	4	1.48	3.30	1.96
HWI	5	4.50	5.26	2.97
HWI	6	1.08	1.41	0.83
HWI	7	1.44	1.42	1.49
HWI	8	2.70	2.71	1.44
HWI	9	0.54	0.41	0.84
HWI	10	0.61	1.95	0.51
HWI	11	1.66	2.36	1.00
	mean	1.77	2.32	1.66
	SD	1.10	1.30	0.87

	Post-Pre	24-Pre
	0.18	0.26
	1.43	0.73
	-0.42	0.76
	1.82	0.48
	0.76	-1.53
	0.33	-0.26
	-0.02	0.05
	0.01	-1.26
	-0.12	0.31
	1.34	-0.10
	0.70	-0.66
mean	0.55	-0.11
SD	0.73	0.76

Contrast Water Therapy vs. Passive Recovery – **Interleukin-6 (pg/mL)**

Raw Data		Trials				Effects	
Treatment	Subject	Pre	Post	24		Post-Pre	24-Pre
PAS	1	1.69	2.48	2.00		0.79	0.31
PAS	2	1.29	4.42	1.19		3.13	-0.10
PAS	3	0.75	0.66	0.32		-0.09	-0.43
PAS	4	1.84	3.96	1.24		2.12	-0.60
PAS	5	0.71	1.45	2.49		0.74	1.78
PAS	6	1.13	1.93	1.77		0.80	0.64
PAS	7	1.78	2.99	2.59		1.21	0.81
PAS	8	1.80	2.58	3.06		0.78	1.26
PAS	9	1.60	3.01	1.09		1.41	-0.51
PAS	10	1.50	1.61	1.61		0.11	0.11
PAS	11	0.71	1.96	2.77		1.25	2.06
PAS	12	2.52	1.66	1.13		-0.86	-1.39
PAS	13	2.40	4.18	2.94		1.78	0.54
PAS	14	1.70	2.85	0.95		1.15	-0.76
PAS	15	3.29	3.47	4.11		0.18	0.82
	mean	1.65	2.61	1.95	mean	0.97	0.30
	SD	0.71	1.09	1.02	SD	0.96	0.97
CWT	1	1.95	1.54	1.70		-0.41	-0.25
CWT	2	0.77	2.71	1.43		1.94	0.66
CWT	3	1.32	1.23	0.25		-0.09	-1.07
CWT	4	1.42	1.85	0.93		0.43	-0.49
CWT	5	1.12	2.43	0.39		1.31	-0.73
CWT	6	1.59	3.23	1.73		1.64	0.14
CWT	7	1.33	2.12	1.07		0.79	-0.26
CWT	8	1.10	3.07	1.22		1.97	0.12
CWT	9	2.43	3.29	3.21		0.86	0.78
CWT	10	1.02	2.15	1.22		1.13	0.20
CWT	11	1.70	1.22	0.88		-0.48	-0.83
CWT	12	1.71	1.83	1.35		0.12	-0.36
CWT	13	3.01	2.66	2.76		-0.35	-0.25
CWT	14	1.14	1.97	1.27		0.83	0.13
CWT	15	1.56	1.69	3.38		0.13	1.82
	mean	1.54	2.20	1.52	mean	0.65	-0.03
	SD	0.58	0.68	0.93	SD	0.83	0.72

Cold Water Immersion vs. Passive Recovery – **Lactate Dehydrogenase (U/L)**

Raw Data		Trials						Effects			
Treatment	Subject	Pre	Post	24	48	72		Post-Pre	24-Pre	48-Pre	72-Pre
PAS	1	179	181	148	167	192		2	-31	-12	13
PAS	2	171	149	148	147	273		-22	-23	-24	102
PAS	3	231	225	171	268	181		-6	-60	37	-50
PAS	4	211	162	172	294	301		-49	-39	83	90
PAS	5	272	262	187	196	227		-10	-85	-76	-45
PAS	6	280	306	352	346	366		26	72	66	86
PAS	7	74	133	88	50	83		59	14	-24	9
PAS	8	170	218	179	114	229		48	9	-56	59
PAS	9	245	215	215	241	200		-30	-30	-4	-45
PAS	10	177	180	185	139	118		3	8	-38	-59
PAS	11	291	270	299	315	241		-21	8	24	-50
PAS	12	182	201	178	174	226		19	-4	-8	44
	mean	207	209	194	204	220	mean	2	-13	-3	13
	SD	61	52	70	89	76	SD	32	41	48	62
CWI	1	228	281	161	168	213		53	-67	-60	-15
CWI	2	155	153	125	121	178		-2	-30	-34	23
CWI	3	208	223	180	197	125		15	-28	-11	-83
CWI	4	308	293	266	266	277		-15	-42	-42	-31
CWI	5	162	149	150	196	107		-13	-12	34	-55
CWI	6	328	375	328	145	210		47	0	-183	-118
CWI	7	148	64	131	68	78		-84	-17	-80	-70
CWI	8	142	152	132	123	138		10	-10	-19	-4
CWI	9	171	161	172	102	196		-10	1	-69	25
CWI	10	334	308	205	246	197		-26	-129	-88	-137
CWI	11	321	355	284	309	316		34	-37	-12	-5
CWI	12	335	216	198	186	166		-119	-137	-149	-169
	mean	237	228	194	177	183	mean	-9	-42	-59	-53
	SD	82	96	66	71	68	SD	50	46	61	64

Hot Water Immersion vs. Passive Recovery – **Lactate Dehydrogenase (U/L)**

Raw Data		Trials						Effects			
Treatment	Subject	Pre	Post	24	48	72		Post-Pre	24-Pre	48-Pre	72-Pre
PAS	1	392	395	409	361	196		3	17	-31	-196
PAS	2	226	126	135	230	173		-100	-91	4	-53
PAS	3	286	311	336	299	285		25	50	13	-1
PAS	4	136	159	167	138	304		23	31	2	168
PAS	5	220	313	312	286	319		93	92	66	99
PAS	6	199	234	220	253	217		35	21	54	18
PAS	7	103	110	116	222	133		7	13	119	30
PAS	8	339	335	302	345	361		-4	-37	6	22
PAS	9	384	408	381	369	350		24	-3	-15	-34
PAS	10	281	283	302	272	269		2	21	-9	-12
PAS	11	247	322	288	301	339		75	41	54	92
	mean	256	272	270	280	268	mean	17	14	24	12
	SD	93	103	98	68	77	SD	49	47	44	94

Treatment	Subject	Pre	Post	24	48	72		Post-Pre	24-Pre	48-Pre	72-Pre
HWI	1	396	438	421	319	391		42	25	-77	-5
HWI	2	182	261	211	218	153		79	29	36	-29
HWI	3	303	310	310	311	306		7	7	8	3
HWI	4	136	223	131	137	141		87	-5	1	5
HWI	5	313	324	279	293	261		11	-34	-20	-52
HWI	6	209	227	342	193	209		18	133	-16	0
HWI	7	125	143	177	172	156		18	52	47	31
HWI	8	285	185	166	279	268		-100	-119	-6	-17
HWI	9	348	386	346	339	365		38	-2	-9	17
HWI	10	300	278	337	333	284		-22	37	33	-16
HWI	11	275	283	264	270	265		8	-11	-5	-10
	mean	261	278	271	260	254	mean	17	10	-1	-7
	SD	87	86	91	70	83	SD	50	61	34	22

Contrast Water Therapy vs. Passive Recovery – **Lactate Dehydrogenase (U/L)**

Raw Data		Trials					Effects			
Treatment	Subject	Pre	Post	24	48	72		Post-Pre	24-Pre	48-Pre	72-Pre
PAS	1	156	317	352	315	311		161	196	159	155
PAS	2	278	353	309	271	298		75	31	-7	20
PAS	3	220	250	271	239	242		30	51	19	22
PAS	4	111	106	306	139	252		-5	195	28	141
PAS	5	168	154	133	170	150		-14	-35	2	-18
PAS	6	352	404	411	356	464		52	59	4	112
PAS	7	56	140	131	91	89		84	75	35	33
PAS	8	334	339	324	309	317		5	-10	-25	-17
PAS	9	234	190	232	156	169		-44	-2	-78	-65
PAS	10	38	243	200	211	215		205	162	173	177
PAS	11	334	321	377	313	141		-13	43	-21	-193
PAS	12	202	190	182	162	196		-12	-20	-40	-6
PAS	13	288	335	328	283	278		47	40	-5	-10
PAS	14	345	238	460	347	442		-107	115	2	97
PAS	15	168	115	225	106	141		-53	57	-62	-27
	mean	219	246	283	231	247	mean	27	64	12	28
	SD	103	95	98	89	108	SD	81	74	70	96
CWT	1	303	293	289	277	312		-10	-14	-26	9
CWT	2	278	319	305	283	296		41	27	5	18
CWT	3	270	294	306	271	243		24	36	1	-27
CWT	4	298	337	292	152	290		39	-6	-146	-8
CWT	5	180	138	101	118	144		-42	-79	-62	-36
CWT	6	381	409	427	433	403		28	46	52	22
CWT	7	138	138	120	119	117		0	-18	-19	-21
CWT	8	338	352	369	306	178		14	31	-32	-160
CWT	9	205	219	249	195	244		14	44	-10	39
CWT	10	239	261	236	220	229		22	-3	-19	-10
CWT	11	294	314	317	339	317		20	23	45	23
CWT	12	208	186	194	164	255		-22	-14	-44	47
CWT	13	274	271	298	311	125		-3	24	37	-149
CWT	14	390	415	638	523	492		25	248	133	102
	15	280	279	249	250	155		-1	-31	-30	-125
	mean	272	282	293	264	253	mean	10	21	-8	-18
	SD	70	84	127	113	105	SD	23	71	62	74