THE EFFECT OF HYDROTHERAPY ON RECOVERY AND PERFORMANCE DURING HIGH INTENSITY EXERCISE

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ABSTRACT

THE EFFECT OF HYDROTHERAPY ON RECOVERY AND PERFORMANCE DURING HIGH INTENSITY EXERCISE

Athletes use a wide range of interventions to promote recovery from strenuous exercise, but few data are available regarding the efficacy of such practices. OBJECTIVE: To examine the effectiveness of commonly used interventions [Rest, light exercise (AR), contrast therapy (CT) and cryotherapy (CR)] during recovery between bouts of intense exercise. We tested the hypothesis that hydrotherapy interventions (CT and CR) would induce favorable physiological and/or psychological alterations such that performance would be improved versus AR and Rest. METHODS: In Study I, 12 active men (25-35 yrs; VO_{2peak} = 46±3 $ml \cdot kg^{-1} \cdot min^{-1}$; mean±SD) performed 5 consecutive days of HI exercise (4-6 bouts x 30 sec 'all out' Wingate Tests, with 4-min recovery, each day). After each training session, subjects either rested for 20 min (CON, n=6) or completed a CT protocol (n=6) that consisted of alternating cold (10°C) and hot (40°C) tubs using a 4x2:3 min ratio. Performance measures [Peak (Wmax) and mean (Wmean) power, VO_{2peak}, and a 250 kJ Time Trial (TT)] were assessed before and after the HIT. In Study II, 9 active men (29±6 yr, VO_{2peak} = 44±8 $ml \cdot kg^{-1} \cdot min^{-1}$) performed 3 exercise trials separated by 1 wk. Each trial consisted of 3 x 50 KJ time trials (~100-120%VO_{2peak}) with a different 20-min recovery period [CON, AR (cycling @ 50W) or CR (cold tub @ 10°C)] between rides each week. Venous blood samples were obtained after each recovery period, and analyzed for lactate, interleukin-6, neutrophils, and lymphocytes. Questionnaires designed to assess exercise preparedness were also completed daily in

both studies. RESULTS: In Study I, Wmax and TT performance improved after 5 d of HI exercise (time effect, $P<0.05$), but there were no differences between groups (Wmax - CT: Post: 1310±45 vs Pre: 1215±86; CON: Post: 1343±54 vs Pre: 1220±74 W: TT - CT: Post: 15.8±0.6 vs Pre: 16.7±0.7; Rest: Post: 18.1±1.0 vs Pre: 18.8±1.2 min, means±SEM). In Study II, TT performance averaged 118±10 sec for bout 1 and was 8% and 14% slower during bouts 2 (128±11 sec) and 3 (134±11 sec), respectively, with no difference between treatments (Time effect, $P\leq0.05$). Blood lactate was lower after AR compared to CR and Rest, and neutrophils and lymphocytes were higher and lower respectively ($P\leq0.05$), after CR (8.7±1.3 and 1.4±0.2 x 10^9cells/L) versus AR (7.1±1.0 and 1.6±0.1) and Rest (6.7±0.7 and 1.6±0.1). With respect to the psychological measurements, the CT and CR groups in both studies reported feeling more revitalized after each treatment session and greater preparedness for subsequent exercise (Treatment effect, $P\leq.05$). CONCLUSIONS: Exercise performance during repeated bouts of intense cycling was not influenced by the type of recovery intervention employed, either during a single session or over the course of a 5 d training session. CR caused greater perturbations in blood immune markers and most notably, hydrotherapy interventions created the perception that subjects were better prepared for subsequent exercise.

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CHAPTER 1

THE EFFECT OF HYDROTHERAPY ON RECOVERY AND PERFORMANCE DURING HIGH INTENSITY EXERCISE

REVIEW OF LITERATURE

1.1 INTRODUCTION

The most common training model used by coaches and athletes is based on the "Overload Principle" or "Physical Stress Theory"(Baechle and Earle, 2000). An essential component of the model is that high intensity physical exercise creates a disturbance in cellular homeostasis. This disturbance then acts as a stimulus that initiates physiological responses in order to restore homeostasis and induce training adaptations (Kuipers, 1998; Väänänen, 2004). To push performance capacity to the limit of tolerance, therefore, relatively high amounts of intense training must be performed. Although the "optimal" amount of high-intensity training remains unclear, athletes are generally inclined to do "too much". When exercise and the associated disturbance in homeostasis are not followed by adequate recovery, athletes may be subjected to excess muscular fatigue and/or injury (Esperson et. al., 1990; Bruunsgaard et. al., 1997; McKenzie, 1999).

The recovery process, therefore, is of particular importance in sports where an athlete may have to compete or train more than once in a single day. This may include sports such as track and field, swimming, cycling, wrestling, and rowing. Along with daily sport-specific training sessions, many top athletes also engage in strength, flexibility, and other

adjunct forms of training in an effort to improve performance. Finally, many competition seasons are relatively short which means athletes must quickly attain peak fitness and maintain fitness for extended periods of time. The cumulative effect of the combined training places a huge physical and psychological strain on the athlete, which in some individuals may lead to "over-training" or "over-reaching".

Over-training is characterized by persistent fatigue, poor performance, changes in mood state and neuroendocrine factors, and frequent illness that do not immediately resolve with extended periods of rest (MacKinnon, 2000; Smith, 2000; Armstrong and VanHeest, 2002). These adverse changes reflect the body's inability to adapt to the cumulative stress of daily, intense exercise training without adequate rest and recovery. Over-reaching, on the other hand, describes a qualitatively similar state to over-training but is less severe and resolves quicker (Armstrong and VanHeest, 2002). The incidence of over-reaching seems to be more prevalent in sports and is easier to induce in studies by employing a short-term, intensified training model. Researchers have reported that 30-100% of athletes exhibit some symptoms of over-reaching after intensified training (MacKinnon, 2000; Smith et al., 2000; Armstrong and VanHeest, 2002). Along with sudden increases in training volume and intensity, other causative factors include heavy competition schedules, lack of periodization or programmed recovery, monotonous training schedules, and high levels of stress in daily life (MacKinnon, 2000). The most common symptoms of over-training or over-reaching are summarized in Table 1.1.1.

	Signs and Symptoms of Over-training and Over-reaching
1.	Performance decrements and reduced ability to perform high intensity training,
2.	High ratings of fatigue,
3.	Decreases in maximal heart rate,
4.	Changes in blood lactate variables such as blood lactate threshold or concentrations during maximal exercise,
5.	Neuroendocrine changes such as reduced excretion of norepinephrine and changes in testosterone:cortisol ratios,
6.	Changes in athletes self-reported indicators of "well-being" including pain, fatigue, mood state and motivation.

Table 1.1.1 Signs and Symptoms of over-training and over-reaching (adapted from MacKinnon, 2000)

Intensified training is essential to performance enhancement. While the effects of a single bout of exercise and moderate long-term physical training are quite well documented, the effects of daily, repeated high intensity training and repeated loading are unknown. It is not surprising that fatigue and injury are always persistent threats. It seems those factors that could ultimately restrict performance are often overlooked in the pursuit of excellence. Perhaps a greater understanding of the negative cellular changes that occur with high intensity training is required. The recovery aspect of the training cycle is also frequently overlooked. The recovery process in sport plays an essential role in determining subsequent athletic performance. Depending on the amount and severity of stress during training, it could take several days and even weeks to regain power outputs necessary for high intensity exercise or competition. During the competition season, however, this time is not available. In this regard, practice often precedes principle as athletes and coaches find ways to "speed up the process". Although having a long history of use in sports medicine, many of these recovery interventions remain insufficiently researched and understood. The following literature review focuses on the metabolic disturbances associated with high intensity exercise training and the possible

influence on fatigue, injury, illness, and mental health. Finally it reviews the limited body of literature associated with recovery from high-intensity exercise. By better understanding the effects of training and the best way to recover, athlete potential can be maximized while minimizing any detrimental effects.

1.2 BIOCHEMISTRY OF HIGH INTENSITY EXERCISE

High intensity training (HIT) can be broadly defined as repeated bouts of short to moderate duration exercise (10 sec. to 5 min.) completed at an intensity that is greater than the anaerobic threshold (Laursen and Jenkins, 2002). Bouts are separated by brief periods of low-intensity work or inactivity that allow a partial but not a full recovery. The purpose is to repeatedly stress the physiological system to a greater extent than that which is actually required during the activity (Laursen and Jenkins, 2002). While the cardiovascular and skeletal muscle adaptations to endurance (aerobic) exercise have been extensively studied and validated (Abernethy et al., 1990), recent studies have demonstrated significant improvements in both peak power and mean power (MacDougall et al., 1998; Burgomaster et al., 2005) and maximal aerobic power (Parra et al., 2000) after sprint interval or high intensity training. HIT also offers the convenience of significant improvements in maximal aerobic capacity in relatively short amounts of time (Parra et al., 2000; Rodas et al., 2000) and more closely replicates the intermittent nature of many sport demands. In many sports, bouts of interval sprint training have been used as an adjunct to the regular training both pre-season and in-season. It is still

uncertain, however, what affect the long-term use of HIT will have on the physical and mental "well-being" of the athlete.

During strenuous or high intensity exercise a discrepancy develops between the demand for energy and energy provision from oxidative metabolism. This results in a depletion of Phosphocreatine (PCr) and a large accumulation of lactate (La) as the muscle is forced to rely on non-oxidative sources of energy (Gupta et. al., 1996; Bogdanis et. al. 1995, 1996). Intracellular acidosis associated with elevated levels of lactic acid is the most popular hypothesis to explain muscular fatigue. This is due to altered excitation-contraction (EC) coupling in the myofibril, as hydrogen ions (H^+) compete for Calcium (Ca^{++}) binding sites on troponin and interfere with Ca^{++} release and uptake by the sarcoplasmic reticulum (SR) (Roberts and Smith, 1998). Favero et al. (1995) studied the influence of H^+ and decreased pH on Ca^{++} release from the SR in white rabbit skeletal muscle. Decreasing pH from 7.1 to 6.5 resulted in a 50 % decrease in Ca^{++} released from the sarcoplasmic reticulum. These findings were also found in human subjects after high intensity exercise (Bogdanis et al., 1995; Bangsbo et al., 1996; Hargreaves et al., 1998). Bogdanis et. al. (1995) studied the recovery of PCr and pH after repeated bouts of high intensity cycling using variable rest periods between bouts. PCr was depleted quickly (~10s) but generally recovered quickly with a mean half recovery time of 56.6s. Muscle pH and [La], however remained acidic, even after 6 minutes. Following a bout of strenuous exercise, therefore, the exercising muscles must restore their PCr stores and acid-base balance, thereby preparing the tissue for subsequent physical challenges. If full metabolic recovery is not attained, fatigue and/or muscle injury may result.

1.3 MECHANISMS OF INJURY DURING HIGH INTENSITY EXERCISE

It is widely accepted that training and competition results in microtrauma to muscle and connective tissue (McCully, 1986; Kuipers, 1994; Bruunsgaard et. al., 1997). Intense exercise results in a mild inflammatory response, which stimulates healing and positive adaptations through a process referred to as "adaptive microtrauma". With repeated high volume, high intensity training and limited recovery, however, the trauma induced may negatively impact on performance. High intensity exercise, in untrained subjects, has been associated with increased levels of myofibre enzymes in plasma, ultra-structural damage of the muscle fibres, and an acute inflammatory response leading to edema, infiltration by inflammatory cells, and muscle soreness 24-48 hours following exercise (Newham et al., 1983; Kuipers, 1994). This inflammatory reaction has also been correlated with an increase in circulating levels of plasma cytokines, more specifically, Interleukin-6 (IL-6). For example, Bruunsgaard et al. (1997) reported exercise-induced increases in serum IL-6 and creatine kinase (CK), after 30 minutes of high-intensity cycling, which were positively correlated to muscle damage. Although increased levels of circulating cytokines have predominantly been described after exercise involving an eccentric component (Northoff et al., 1994), concentric exercise also induces cytokine production (Ullum et al., 1994; Weinstock et al., 1997; Ostrowski et al., 1998; Nieman et al., 1998, 2001). Increased IL-6, therefore, may be a strong indicator that the level of training is too intense.

Cytokines are soluble hormone-like proteins. However, in contrast to hormones, which are synthesized by specific endocrine tissues, cytokines are produced by a variety

of cells such as immune cells, endothelial cells, and fat-storing cells (Pedersen, 2000). As changes in some cytokines (IL-1 and TNFα) after exercise tend to be subtle, IL-6 seems to be the one cytokine that provides the most reliable results, being elevated shortly after strenuous exercise and tissue damage (Espersen et al., 1990; Bruunsgaard et al., 1997; Suzuki et al., 1999, 2000). (Fig. 1.3.1).

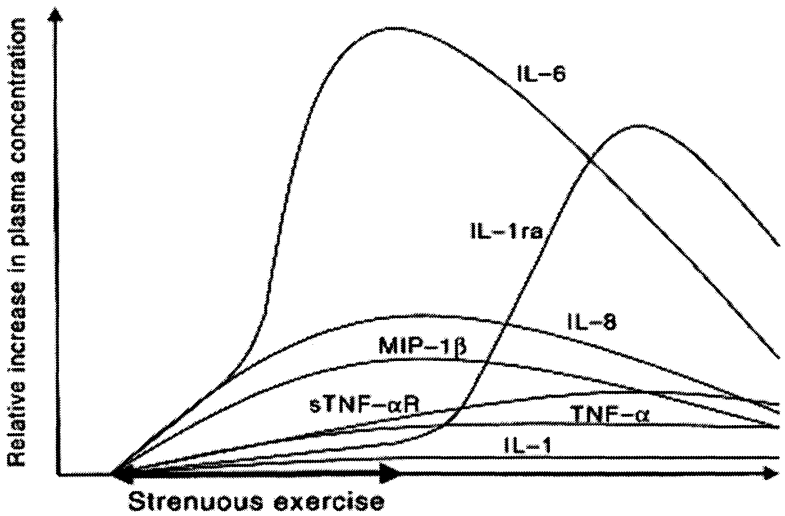

Figure 1.3.1 – Schematic representation of the changes in a number of cytokines in response to strenuous exercise (Pedersen, 2000)

The local response to tissue stress is the production of IL-6, which is released to the site of inflammation. Once established, IL-6 has the capacity to stimulate surrounding cells (paracrine) or themselves (autocrine), which may lead to further IL-6 production and amplification of the inflammatory response (Pedersen, 2000). IL-6, however, has been classified as both a pro-inflammatory and anti-inflammatory cytokine. Along with the inflammatory response, IL-6 facilitates the release of lymphocytes, neutrophils, monocytes, and other cells that participate in the clearance of the antigens and promote tissue healing (Pedersen, 2000). IL-6 also appears to be the primary inducer

of the hepatocyte-derived acute phase proteins, which have anti-inflammatory properties (Dinarello, 1997). Despite the ever-increasing number of studies on exercise and cytokines, it is still not understood if the increased levels of IL-6 after high intensity activities are positive or negative.

1.4 IMMUNE RESPONSE TO HIGH INTENSITY EXERCISE

The immune system is not an organ system but a collection of disease fighting cells, which serve to recognize, attack, and destroy elements that are foreign to the body. Illness results when the immune system fails to neutralize invading agents. This system is essentially divided into two broad functions: Innate (natural and non-specific), and Acquired (adaptive and specific) (Gleeson et al., 2004). Infectious agents enter the body and immediately activate the Innate system. This "first line of defense" restricts microorganism entry into the body and consists of physical barriers (skin, epithelial lining), chemical barriers (pH of body fluids), and phagocytic cells (leukocytes - neutrophils) (Gleeson et al., 2004). Failure of this system activates the Acquired system. As part of this latter system, monocytes or macrophages ingest and present foreign materials (antigens) to the lymphocytes. This is followed by clonal proliferation of T and B lymphocytes that enable the body to recognize and act should the body be re-infected with similar agents (Gleeson et al., 2004). Central to the activation and regulation of these immune functions is the production of cytokines and the influence of stress hormones such as cortisol and catecholamines (Smith, 1995). (Fig. 1.4.1)

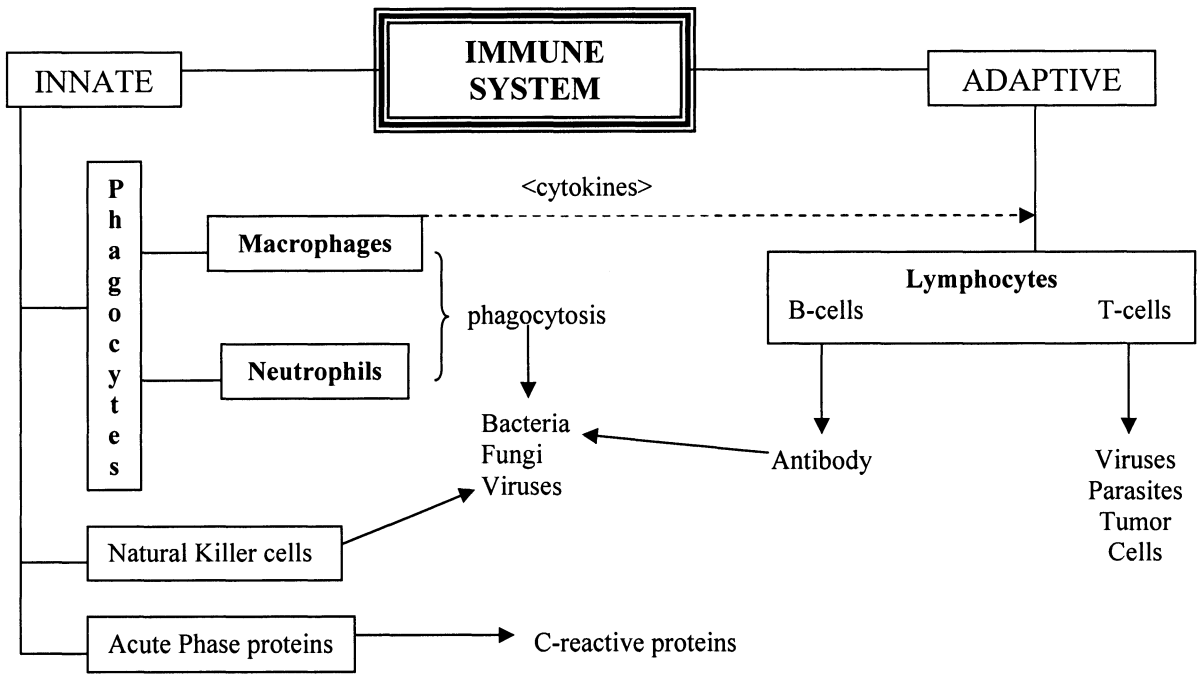

Figure 1.4.1 – Simple schematic of the major components of the adaptive and innate arms of the immune system and their targets. Although not in detail, cytokines and other humoral mediators released from most immune cells play a major role in stimulating the immune response. (adapted from Smith, 1995)

Exercise has been found to have a profound effect on the interaction between the endocrine and immune systems. When challenged with a physical task, the body responds through a series of integrated changes in function that involve most, if not all, of its physiological systems (Väänäanen, 2004). These responses are mediated by both neural and humeral mechanisms related to the function of the autonomic nervous system: the activation of the hypothalamus, sympathetic nervous system, and stress hormone secretion (Väänäanen, 2004). The magnitude of the response, however, is directly related to the intensity of the physical activity. For example, moderate exercise (40-60% VO_{2peak}) appears to stimulate the immune system (Sharp and Koutedakis, 1992; Shepherd and Shek, 1998a, 1998b; MacKinnon, 1999), whereas extremely intense and prolonged

exercises have been associated with immune suppression and increased susceptibility to illness (Nieman et al., 1994; Pedersen et al., 1998; Nieman, 2000). (Table 1.4.1)

Cell	% in Circulation	Normal Levels	Function	Response to Brief Intense Exercise (%Δ)
Granulocyte	60-70%			
Neutrophil	90%	3.0-5.55 cells 10^9/L	Phagocyte	Increase 30-150%
Eosinophil	2.5%	0.05-0.25 cells 10^9/L		
Basophil	0.2%	0.02 cells 10^9/L		
Lymphocyte	20-25%	1.0-2.5 cells 10^9/L	Lyphocyte activation	Increase 100-200% decrease below resting post exs.
T Cells	60-75%	1.0-2.5 cells 10^9/L		
B Cells	5-15%	0.3 cells 10^9/L		
NK Cells	10-20%	0.1-0.5 cells 10^9/L		
Monocyte	10-15%	0.15-0.60 cells 10^9/L	Phagocyte Cytokine Prod. Antigen presentation	Increase 0-20%

Table 1.4.1 – Circulating Leukocytes and their response to a single bout of brief intense exercise [>45 min] (adapted from Mackinnon, 1999)

The immune disturbances that follow a single bout of exhausting exercise or chronic overexertion are similar in many ways to the inflammatory response induced by trauma or infection. However, both the magnitude and duration of the response are much smaller than those associated with clinical sepsis as the primary trigger with exercise is sub-clinical muscle damage (Shephard and Shek, 1998a). Following a single bout of high intensity exercise, substances, including pro-inflammatory cytokines (IL-6) and stress hormones such as catecholamines, cortisol and growth hormone, are released from injured muscle cells and initiate an inflammatory response (Yamada et al., 2002). These substances regulate the rapid migration of leukocytes, neutrophils, and later monocytes, into areas of damaged muscle cells and other metabolically active tissue in order to

initiate repair. In the 15-60 minute period after the cessation of exercise, the initial increase in leukocytes (excluding neutrophils which continue to increase) subsequently falls below resting values (Nieman et al., 1994; Wigernaes et al., 2000, 2001). Only physical activity of significant intensity and/or duration produces this decrease during recovery. With acute exercise bouts, the increased levels of epinephrine also stimulate a rapid rise in lymphocytes. When exercise intensity is reduced and the levels of epinephrine diminish, these levels return to normal or depressed states (Nieman et al., 1994). Longer lasting increases in cortisol after high intensity exercise also perpetuate the decrease in lymphocyte levels post exercise (Nieman, 2000). Increases in blood leukocytes with intense exercise and subsequent decrease upon cessation, therefore, represent an important aspect of the inflammatory and immunosuppressive process.

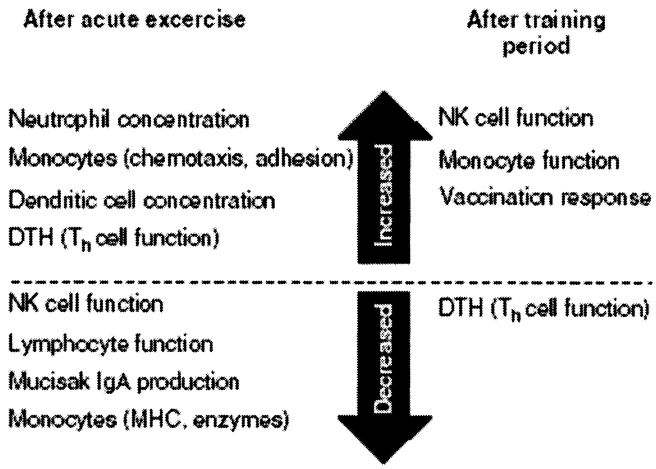

Figure 1.4.2 – Summary of immunological alterations with physical exercise demonstrating both increased and decreased immune function (Malm, 2004)

The above immune parameters have been investigated repeatedly (Espersen et al., 1990; Robson et al., 1999; Wigernaes et al., 2000, 2001; Yamada et al., 2002) and

demonstrate fairly consistent changes under acute and chronic exercise conditions. The actual clinical relevance with regard to illness and performance, however, remains unclear. Whether athletes are more susceptible to acquiring infection is also controversial, but increasing evidence suggests that competitive athletes may be at a greater risk than people who exercise at a moderate rate (Shepherd and Shek, 1998a).

1.5 PSYCHOLOGICAL ASPECTS OF EXERCISE PERFORMANCE

There is a widespread belief that exercise is associated with improvements in mental health including mood state, self-efficacy, psychological 'well-being', and self esteem. Numerous studies have investigated the mood enhancing properties of exercise and have shown that exercise can indeed have a positive influence on mood state. Mood state has been defined as, "the enduring but not permanent emotional predisposition to feel and react or behave in a certain way" (Väänänen, 2004). It involves such affective states as anger/hostility, tension/anxiety, depression/dejection, vigor/activity, fatigue/inertia, and confusion/bewilderment. Simply stated, it is the "feel better" phenomenon often associated with activity. In a review of exercise and mood state by R. Yeung (1996), 85% of the studies cited reported at least some degree of improved mood on a wide variety of measures following varying modes, intensities, and volumes of exercise. As little as 20-40 minutes of aerobic activity resulted in an improved state of anxiety and mood that persisted for several hours (Raglin, 1990; Scully et al., 1998). In some cases, however, exercise may result in negative psychological changes. For example, it has been found that elevations in training volume are associated with

increases in mood disturbances, and this relationship follows a dose-response pattern (Raglin, 1990). Yeung (1996) also found studies that reported worsened mood state, which were correlated with either extremely competitive or intense training levels, or unusual conditions during exercise such as heat stress. That is, mood disturbance became progressively worse as training intensity increased. Steptoe and Bolton (1988) found that low intensity exercise (25W) produced modest improvements in mood as measured by the Profile of Mood States (POMS), while higher intensity exercise (100W) increased negative mood states. Other researchers, however, have not found any effect of intensity on mood states (Felts, 1989; Pronk et al., 1995). Regardless, it does appear that very high and low intensities of exercise are not optimal for psychological benefit.

Disturbance of an athlete's mood state is also one of the key components of the over-training or over-reaching state of training. Symptoms include depression, anxiety, difficulty in concentrating, and irritability (Smith, 2000; Armstrong and VanHeest, 2002). The Profile of Mood State (POMS) test is most frequently used to measure alterations in psychopathologic parameters. This test was developed to measure six identifiable mood or affective states: Tension-Anxiety; Depression-Dejection; Anger-Hostility; Vigor-Activity; Fatigue-Inertia; and Confusion-Bewilderment. Although developed to measure mood states in psychiatric populations, this test has proved to be a sensitive measure of the effects of various experimental manipulations including exercise. Morgan (1981) in his work with the POMS, found that the athlete differs from non-athletes on a variety of psychological states. Athletes' profiles tend to follow the "Iceberg Profile" in that they tend to have lower scores in Tension, Depression, Fatigue, and Confusion and higher

scores in Vigor than non-athletes. In one study by Kowal et al. (1978), military recruits who participated in an eight-week basic training displayed significant drops in Tension, Depression, Fatigue and Confusion, and increases in Vigor. The POMS has also been used to show differences in mood states between elite and non-elite athletes, to predict success, to measure effectiveness of training, and to monitor over-training. During the over-trained or over-reached state of training, the athlete's rating of perceived exertion (RPE) is increased at a given workload and the POMS scores are negatively altered (Armstrong and VanHeest, 2002). The use of POMS, however, has been criticized as it was initially validated for use only in clinical populations and it includes only one positive mood dimension (Scully et al., 1998). Despite potential limitations, this test for Mood State changes in exercise has been cited in over 600 studies from 1975 to present.

The main objective for generating a positive psychological perspective is to improve performance, and it could be argued that perceived-self confidence, or self-efficacy, is a better predictor of success than mood state. Self-efficacy is the sense of success that an athlete feels he or she embodies or controls. Someone who is highly self-efficacious does not doubt his or her ability to succeed at a given task. Self-efficacy and "Preparedness for Exercise" tests may reflect whether an individual's psychological arousal is at an optimal level for success. Limited attention, however, has been given to the mechanisms by which self-efficacy influences the actual experience of physical activity. Rudolph and McAuley (1996) were two of the first to study self-efficacy and exercise and found that lower exercise self-efficacy was associated with higher levels of perceived exertion during actual exercise bouts. This conclusion was supported by the

results of a recent study by Pender et al. (2002). The authors assessed 103 adolescent girls for perceived exertion and self-efficacy during 20-minute bouts of cycling exercise at 60% VO_{2peak}. Pre-exercise efficacy was found to be an important factor influencing perceptions of exertion during exercise and post-exercise efficacy. Lower ratings of efficacy consistently resulted in higher ratings of perceived exertion. Using efficacy and preparedness scores, therefore, may allow coaches to better predict if athletes are ready to perform in repeated exercise trials.

There is strong support for the existence of acute mood benefits from a single bout of exercise. In some circumstances, however, intense training or over-reaching may result in negative psychological outcomes. Little is known about the specific factors that contribute to these mood effects but parameters such as duration, intensity, and mode seem to play a significant role. Hormonal influences associated with over-training cannot be discounted. Self-efficacy may also play an important role in exercise, as it seems to correlate well with levels of perceived exertion. In all likelihood, both physiological and psychological factors are involved in mood disturbance during over-reaching training.

1.6 THERAPEUTIC INTERVENTIONS FOR RECOVERY

The goals of athletic training are to enhance and optimize performance. To push physiological capacity to the limits of tolerance, however, relatively high amounts of intense training must take place, and it is usually up to the team therapist to deal with the side effects of "maximizing performance". Complaints of fatigue and "dead legs" are a common occurrence. Consequently, athletes and medical professionals often experiment with many

different interventions for recovery between intense bouts of exercise despite the fact there are very few studies validating their benefits. Three commonly used recovery interventions are: (1) Active recovery or light exercise, (2) Hydrotherapy or the use of cryotherapy and (3) Contrast baths.

1.6.1 Active Recovery

Recovery from exercises that produce high levels of lactate is not complete until lactate levels are reduced to normal levels in both blood and skeletal muscle. This is especially important during conditions with multiple bouts of high-intensity physical activity and short periods of recovery. In general, 25 minutes of rest/recovery is required following maximal exercise to remove one half of the accumulated lactate, and over one hour to remove 95% (Karlsson and Saltin, 1971). Lactate is cleared from blood and muscle more rapidly, however, when light exercise is performed during recovery (Belcastro and Bonen, 1975; Bangsbo et al., 1994; Ahmaidi et al., 1996). Furthermore, lactate can be used as a fuel by many tissues after its conversion to pyruvate and oxidization. This accounts for the majority of the lactate removed during recovery. Several organs are capable of using lactate for fuel, although skeletal muscle (type I > type II) is the major organ most involved in this process (Karlsson and Saltin, 1971). The exercise intensity that produces the fastest or optimal rate of removal has been calculated to be between 30-45% of VO_{2max}, which corresponds to an oxygen consumption of 1.0-1.5 $L.min^{-1}$ or 15-20 $ml.kg.min^{-1}$ (Belcastro and Bonen, 1975) This estimate is based on untrained subjects who worked on a cycle ergometer. With well-trained subjects the

exercise intensity is closer to 50-65% VO_{2max} (Belcastro and Bonen, 1975). This discrepancy is based on individual fitness levels as the higher the state of training (increased mitochondrial density and blood perfusion) the higher the recovery intensity. Light exercise in moderately trained individuals, therefore, at 40-60% VO_{2max} will reduce blood lactate levels ~50% after only 10 min (Belcastro and Bonen, 1975). Active exercise is also beneficial for resynthesizing high-energy phosphates, replenishing oxygen in the blood and muscle, and restoring body fluid homeostasis (Bangsbo et al., 1996; Dupont et al., 2004). Recent research has also shown that active recovery at 50% VO_{2peak} for 15 minutes significantly limited the rapid and possibly harmful reduction in leukocytes after exercise (Wigernaes et al., 2000, 2001). Active recovery, therefore, not only speeds up recovery in terms of removal of metabolic by-products, it may also be effective in limiting immunosuppression after high-intensity exercise.

1.6.2 Cryotherapy (Cold-Bath)

Many athletes utilize cryotherapy as a recovery intervention in hopes of countering the "harmful" effects of exercise. Cryotherapy is the therapeutic application of any substance to the body that results in the withdrawal of heat from the body, thereby lowering tissue temperature (Knight, 1995). Strategies include ice or cold-pack application, ice massage and cold-water baths. It is believed that by decreasing tissue temperature, cryotherapy can diminish pain, tissue metabolism, and muscle spasm, minimizing the inflammatory process and thereby aiding recovery after soft-tissue trauma

(Edwards et al., 1952; Knight, 1995; Enwemeka et al., 2002). The clinical application of cryotherapy is largely based on empirical, clinical evidence but the physiological mechanisms are not well understood.

Cold application results in an immediate and rapid decline in temperature of the surface tissues and a slower decrease in deeper tissue temperature (Knight, 1995). Cryotherapy modalities do not actually transfer cold to the tissues because cold is not transferable. Instead, tissues warm the cold modalities by losing heat to them. Because heat transfer is unidirectional, cold modalities work by absorbing heat from their immediate environment, particularly from the tissues being treated (Knight, 1995; Merrick et al., 2003). Similarly, deep tissues are cooled by losing heat to more superficial tissues (Knight, 1995; Merrick et al., 2003). This transfer of heat from one tissue to another is dependent on many factors including the size of the contact area, the relative masses of the tissue, the temperature gradient between the target tissue and the cooling agent, and the type of cooling modality applied (Knight, 1995; Merrick et al., 2003). The magnitude of the temperature change in deep tissue, therefore, is dependent on the magnitude of the cold application and the amount of heat removed from the body.

To optimize the therapeutic effects of cryotherapy, an optimal tissue temperature reduction of 10°-15°C may be necessary (Bugaj, 1975; Knight, 1976; MacAuley, 2001). A reduction in skin temperature to 14°C will achieve local analgesia (Bugaj, 1975), whereas, lowering tissue temperatures by 10-15°C will suppress tissue metabolism (Knight, 1976). The degree of temperature change, however, is dependant on the method and duration of application, the initial temperature of the tissue, and the depth of the

target tissue (MacAuley, 2001; Myrer et al., 2001). Evidence from a recent systematic review suggested that 10 min of ice pack treatment was the most effective duration for cooling injured animal tissue and healthy human tissue to therapeutic levels (MacAuley, 2001). The effectiveness of this particular protocol has not been tested on injured human subjects. Another application technique, mostly used in research because of the ability to control temperature, is immersion in cold water (Meeusen and Lievens, 1986, MacAuley, 2001, Bleakley et al., 2004). Cold baths absorb heat through conduction and may absorb through convection if the water is moving. For these reasons cold baths may be better than ice or gel packs. For example, in a study by Knight et al. (1981) it was found that the application of 10°C water baths for 10 min. was more effective than commercial cold packs in reducing forearm skin temperature from 32°C to 12°C. After 10 min., the commercial cold packs had warmed to the point where they were extracting less heat from the arm than the circulation restored. In a similar study by Myrer et al. (1998) 20 min of leg immersion in a 10°C bath resulted in a significant reduction in intramuscular temperature, with a prolonged reduction in temperature post treatment. The ideal form of cryotherapy for therapeutic benefit, therefore, may be the use of 10°C cold baths for durations in excess of 10 minutes.

Another important effect of cold application is decreased oxidative metabolism, which results in a decreased need for oxygen. Hypothermia reduces cellular energy needs, thereby reducing the tissue requirements for oxygen. Rao et al. (1976) found significantly higher levels of ATP, creatine phosphate, and glycogen in canine hearts maintained at 5°C during 1 hour of restricted blood flow than in hearts maintained at

normal temperatures. This indicates fewer breakdown of high-energy phosphate bonds, apparently because of a reduced demand for energy. Knight (1976) further suggested that lowering tissue temperature should protect the cells that survived the initial muscle damage from secondary hypoxic injury. Consequently, the total number of cells damaged would be reduced, possibly leading to a reduction in edema. It is possible that the amount of damage or the rate of damage to muscle cells might be reduced by cooling. In a study by Eston and Peters (1999), cold-water immersion after strenuous eccentric exercise was found to reduce the rate of post-exercise damage, as creatine kinase efflux was significantly lower in the cryotherapy group (4.72 $IU.1^{-1}$) versus the control group (5.99). Tissue injury, and the inflammatory process, also induces a rapid sequence of immune reactions. Reactions to control this process can be excessive resulting in a phase of immunosuppression when the body is more vulnerable to opportunistic infections (Niemen, 2000). Cold treatments may attenuate these responses. If the inflammatory process is controlled, the immune reactions should also be controlled, thus closing the "window" of immunosuppression. Rhind et al. (2001) suggested that although exhausting exercise may have increased monocytic activity, cold exposure might have increased the production of anti-inflammatory cytokines, thus limiting the severity of the host inflammatory response. Shephard and Shek (1998c) also found that although cold exposure increased plasma concentrations of some inflammatory cytokines, these changes probably helped restore immune responsiveness. Furthermore, cold exposure helps reduce the normal exercise-induced increases in core body temperature, and thus the stress associated with a given intensity of exercise, so that normal exercise-induced

changes in immune function are decreased (Shephard and Shek, 1998c). Perhaps by controlling the effects of tissue injury, cryotherapy limits the activity of neuroendocrine hormones thus facilitating the healing process while limiting immune stress. Unfortunately, the number of studies in the area of cryotherapy and immune function with exercise is limited.

The use of cryotherapy or cold baths has also been used to maintain physical performance. Muscle temperature remains in a relatively narrow range during resting conditions (35-37°C) but increases rapidly during vigorous exercise (Drust et al., 2005). Heat production during exercise may increase muscle temperature by as much as 5-6°C (Drust et al., 2005). When muscle temperatures increase, the body must adjust rapidly by increasing heat loss in order to maintain homeostasis and ideal function (Drust et al., 2005). Cryotherapy results in conductive heat loss and may assist other body mechanisms in reducing muscle temperature to a more appropriate level (Knight, 1995). In one study, 3-min intervals of cryotherapy by baseball pitchers between innings resulted in a significantly higher number of innings pitched with increased velocity and no alterations in accuracy (Verducci, 1997). The same author also found that interval cryotherapy between weight-pulling sets was associated with increased work, velocity, and power (Verducci, 2000). Similarly, studies by Sargeant (1987), Rogers and Albrechtsen (2003), and Fowles et al. (2003) used various forms of cryotherapy between bouts of exercise and found power and performance were maintained. In all these studies, however, the exact mechanisms were not investigated. From a performance perspective, cold also seems to have a positive effect on ratings of perceived exertion

(RPE). Nelson et al. (1991) found that RPE was lowered and self-efficacy enhanced during exercise in cold (-10° and 8°C) compared to thermoneutral environments (26°C). Maw et al. (1993) found 30 min of cycling was perceived to be harder in hot conditions compared to neutral, with subjects complaining of greater discomfort. These responses were reversed in the cold group indicating that during equal intensity exercise, cold elicited greater psychophysical comfort and less physiological strain. Despite the different methodologies in the studies by Maw et al. (1993) and Nelson et al. (1991), RPE was lower and comfort sensations more positive as temperature conditions were lowered. With less perceived effort and greater comfort, athletes using cold tubs may be able to work harder during repeated exercise bouts, which should allow for improved performance.

Finally, much of the therapeutic benefit of cryotherapy may be from neurological changes. Local cooling affects almost every component of the neuromuscular complex, including the motor neuron pool, the muscle bundle and its afferents, the myoneural junction and the extrafusal fibers of the muscle (Meeusen and Lievens, 1986). Specifically, cold decreases motor and sensory nerve conduction velocity. The decrease in nerve conduction velocity has been measured in human median (Abramson et al., 1966), ulnar (Henrikson, 1956; Abramson et al., 1966), and tibial (Halar et al., 1980) nerves. This decrease is thought to be linear with velocity decreasing 1.4 – 2.6 m/s/degree (Henrikson, 1956; Abramson et al., 1966; Lee et al., 1976). This progressive fall in conduction velocity is in proportion to the temperature of the tissues, rather than the local circulation (Abramson et al., 1966). Halar et al. (1976) found 20 min of cold-

water immersion (18.3°C) of the lower leg resulted in a 7.4°C decrease in skin temperature and a 6.4m/s decrease in tibial nerve velocity. Adequate cooling can reduce pain as well as nerve conduction. Clinical, as well as experimental, research on pain and the pain threshold indicates that pain reduction occurs after cooling the skin to temperatures of around 10-15°C (Travell, 1952; Bugaj, 1975). It is believed breaking the pain cycle by surface analgesia produces a shower of impulses on the central nervous system making the receptors resistant to pain impulses (Travell, 1952). By reducing pain, spasm and neural inhibition, athletes should be able to engage in earlier and more aggressive training or exercise. Cryotherapy is a versatile modality that has been used in the immediate and rehabilitative phases of physical training but the basis for its clinical application still requires further investigation.

1.6.3 Contrast Therapy

Contrast therapy, using cold and hot tubs for physical recovery, is in frequent use by many organizations including the National Football and Hockey Leagues. The theory is simple in that the alternating application of hot and cold water, generally applied to distal extremities, can be used to increase blood flow. This "vascular exercise", of alternating constriction and dilation of local blood vessels, is believed to increase peripheral circulation and aids in the removal of injury debris that has accumulated in areas of tissue stress (Prentice, 1990). It has been further proposed to relieve stiffness and pain, reduce necrotic cells, aid healing, reduce inflammation, and

improve range of motion (Myrer et al., 1994, 1997). Many athletes and coaches believe that contrast baths speed up recovery from intense training.

A single protocol has not been established as the "gold" standard for contrast therapy. Traditionally, contrast therapy has started and ended with heat. Others have recommended starting with heat and ending with cold to minimize the possibility of swelling (Prentice, 1990). Still others have suggested the opposite, to begin and end with cold (Bell and Horton, 1987). The ratio of minutes in cold to heat is also variable. The most accepted ratios appear to be 2 or 3 to 1 (heat to cold) with a total treatment time of 20 to 30 minutes (Woodmansey et al., 1938; Bell and Horton, 1987; Prentice, 1990; Knight 1995). The reported temperature range of the bath water is 10-15° for the cold water and 35-40° for the hot water (Woodmansey et al., 1938; Prentice, 1990; Knight, 1995). Actual research to substantiate these parameters, however, is lacking.

The theoretical rationale for contrast therapy, and more specifically contrast baths, can be questioned from a basic physiological perspective. From a vascular standpoint, any changes in blood flow to the area treated would be superficial (skin), not at the intramuscular level. In studies by Myrer et al. (1994, 1997), contrast therapy was found to be incapable of producing any significant physiological effect on the intramuscular tissue temperature 1 cm below the skin and subcutaneous tissue. From a rehabilitation perspective, the need is for lymphatic pumping not vascular pumping and any changes in vascular pumping that may result from contrast therapy are hundreds of times less than that which would occur using other modalities such as exercise (Knight, 1995).

Furthermore, research has not yet been done to support the conclusion that contrast baths assist in recovery from exercise. So, why is the use of contrast therapy so prevalent in high-level sport? Are there true benefits from this modality or is this just another "training room miracle cure"? Perhaps the benefits of this modality are mainly psychological.

1.7 PURPOSE AND HYPOTHESES

It is generally accepted that intense physical activity results in muscle injury, production of lactate, and other metabolic/endocrine substances, which result in muscle fatigue and decrements in performance. Furthermore, high intensity training and over-reaching has been found to negatively affect psychological health. Interestingly enough, all these adverse effects of exercise can be reversed with adequate rest and recovery. With short seasons and intense competition schedules, however, the time for rest and recovery is overlooked. Athletes, therefore, are using many different therapeutic interventions for recovery between intense bouts of exercise. As reflected in the literature, there are few studies validating their benefits. Using a model of repeated intense exercise with various physiological and performance markers should clarify the position of four commonly used interventions. The purpose of this research, therefore, is to determine which intervention – Rest, Active Recovery (AR), Cryotherapy (CR) or Contrast Baths (CT) – is most effective in maintaining physical performance during repeated bouts of intense exercise. We hypothesize that hydrotherapy (cryotherapy and contrast baths) used for recovery between bouts of high intensity cycle exercise will

improve physical performance and blood markers for exercise stress (IL-6, neutrophils, lymphocytes). Despite the effect on performance, however, we propose that only active recovery will have an effect on blood lactate. Psychological scores for pain, physical exertion and preparedness for exercise will also improve with hydrotherapy.

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CHAPTER 2

TESTING THE WATER: ARE THE EFFECTS OF CONTRAST THERAPY MORE PSYCHOLOGICAL THAN PHYSIOLOGICAL?

2.1 ABSTRACT

Contrast therapy (CT), or the alternating use of hot and cold tubs, is frequently used by athletes in an attempt to speed recovery from intense exercise. CT is purported to induce "vascular pumping" of peripheral blood vessels, promote the flow of nutrients to exercised muscles and accelerate the removal of "waste products." Despite its popularity, limited data are available to validate the proposed benefits of this intervention. PURPOSE: We examined the effect of CT during recovery from daily high-intensity interval training (HIT) on exercise performance and psychological measures related to exercise preparedness. METHODS: Twelve active men (25-35 yrs; VO_{2peak} = 46±3 ml·kg^{-1}·min^{-1}) performed 5 consecutive days of HIT (4-6 bouts x 30 s 'all out' Wingate Tests, with 4-min recovery). Following each HIT session, subjects either rested for 20 min (Control; CON, n=6) or completed a CT protocol (n=6) that consisted of alternating cold (10°C) and hot (40°C) tubs using a 4x2:3 min ratio. Peak (Wmax) and mean (Wmean) power during a 30-s Wingate test, VO_{2peak} and time to complete a 250 kJ Time Trial (TT) were assessed before and after the HIT. A Profile of Mood States (POMS) and questionnaires designed to assess exercise preparedness were also completed daily. RESULTS: Wmax increased by ~9% after HIT (main effect, $P<0.05$), but there was no significant difference between groups (CT: Post: 1310±45 vs. Pre: 1215±86; CON: Post: 1343±54 vs. Pre: 1220±74 W, means±SEM). TT performance also improved after HIT (main effect, $P<0.05$)

with no significant difference between groups (CT: Post: 15.8±0.6 vs Pre: 16.7±0.7; CON: Post: 18.1±1.0 vs Pre: 18.8±1.2 min). Wmean and VO_{2peak} were unchanged after HIT. There were no significant differences in POMS subscale scores; however, the CT group reported feeling more revitalized after each treatment session and having greater preparedness for subsequent exercise. CONCLUSION: 5 d of HIT (~15 min total exercise time) increased peak anaerobic power and decreased the time required to complete a defined bout of work. CT created the perception that subjects were better prepared for exercise, but did not further enhance performance.

2.2 INTRODUCTION

In an effort to improve performance many athletes are incorporating higher training volumes and intensities into their training schedules. One of the current trends in physical conditioning for sport is the use of high intensity training (HIT) or sprint interval training (SIT) (Laursen and Jenkins, 2002). Recent studies have demonstrated significant improvements in both peak power and mean power (Linossier et al., 1997; MacDougall et al., 1998) and maximal aerobic power (Parra et al., 2000) after sprint interval training. In many sports, bouts of sprint training have been used as an adjunct to the regular training both pre-season and in-season to improve conditioning. With higher intensity training, however, there may be a cost. The cumulative effect of the combined training may create a physical and psychological strain on the athlete. Complaints of fatigue and "dead legs" are common yet the athlete is still expected to maintain certain standards of fitness and performance.

The use of contrast baths or contrast therapy (CT) is one strategy used by athletes despite the fact that little evidence is available to support its validity as a therapeutic modality (Knight, 1995; Fu et al., 1997; Eston and Peters, 1999). The theory for contrast therapy is simple in that the alternating application of hot and cold water, generally applied to distal extremities, can be used to increase blood flow. This "vascular exercise" of alternating constriction and dilation of local blood vessels is believed to increase peripheral circulation, which aids in removing injury debris that accumulates in areas of tissue stress (Cote et al., 1988; Prentice, 1990). However, the theoretical rationale for hydrotherapy, more specifically contrast therapy, can be questioned from a basic physiological perspective. From a vascular perspective, any

changes in blood flow to the area treated would be superficial, not at the intramuscular level (Myrer et al., 1994). From a rehabilitation perspective, the need is for lymphatic pumping not vascular pumping and any changes in vascular pumping that may result from contrast baths are hundreds of times less than that which would occur using other modalities such as light exercise (Knight and Londeree, 1980; Knight, 1995). Furthermore, research has not yet been done to support the conclusion that contrast therapy assists in recovery from exercise. Are there true benefits from this modality as a recovery intervention, or is this just another "training room miracle cure"? Perhaps the benefits of this modality are mainly psychological.

The purpose of the present investigation was to examine the effects of contrast therapy (CT) on performance during high intensity sprint training. We tested the hypothesis that contrast baths applied during recovery after bouts of high intensity sprint training would increase anaerobic work capacity and time trial performance during subsequent bouts of intense aerobic cycling. In addition, we sought to determine whether CT would affect psychological measures associated with exercise preparedness.

2.3 METHODS

2.3.1 Subjects

Twelve (12) men with a mean age, height and weight of 28.4±6 yr, 179.1±3 cm, and 86.3±5 kg (mean±SD), respectively, volunteered to take part in the experiment. Subjects were recruited via word of mouth and posters placed around the McMaster University campus. The subjects were recreationally active but not specifically trained in any particular sporting event. The experimental procedures and

potential risk factors were fully explained to the subjects prior to beginning the study, and all subjects provided written, informed consent. The McMaster University/Hamilton Health Science Corporation Research Ethics Board approved the experimental protocol.

2.3.2 Pre-experimental procedures

Following familiarization with all testing procedures and equipment, subjects performed a series of baseline performance and psychological tests prior to the experimental training protocol. Each baseline test was conducted on a separate day with ≥24 h between tests. All subjects initially underwent a progressive exercise test to determine their peak oxygen uptake (VO_{2peak}). VO_{2peak} was determined via gas measurements taken from a mouthpiece attached to an online gas collection system (Moxus modular oxygen uptake system, AEI technologies, Pittsburg, PA) during an incremental cycle ergometer protocol. Volitional fatigue was used to determine the end of the test. Peak power, mean power, fatigue index and anaerobic capacity were determined during a 30-second bout of maximal sprint effort against a constant workload equivalent to 0.075 kg/kg body mass (i.e., a Wingate Test). Subjects also performed a simulated 10 km "time trial", whereby they were instructed to complete a standardized bout of work (250kJ) in as fast a time as possible. Pace was self-selected throughout the test, which lasted between 15 to 30 minutes depending on the fitness level and motivation of the subject. All testing and training were conducted using the same electronically braked cycle ergometer (Lode BV, Excalibur Sport V2.0, The Netherlands)(Fig. 2.3.a)

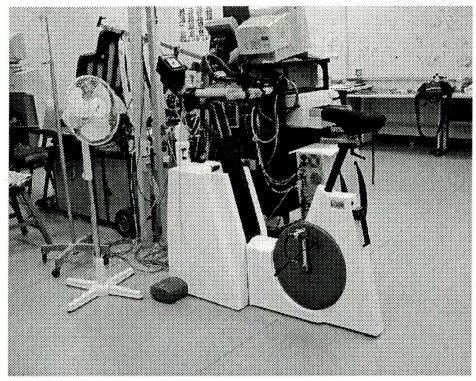


Figure 2.3.a – Electronically braked cycle ergometer used for all physiological testing and training (Lode BV, Excalibur Sport V2.0, The Netherlands).

To assess for psychological status, subjects in both groups completed three mini questionnaires [Profile of mood states (POMS), self-efficacy, and preparedness for exercise scale]. The questionnaires focused on mood, or affective state, feelings of well-being and preparedness for exercise. The latter two questionnaires were developed and designed to suit the present study with the assistance of Dr. Kathleen Martin Ginis (Associate Professor of Exercise Psychology – McMaster University).

2.3.3 Experimental Protocol

Subjects were randomly assigned to one of two training groups (n=6 /group). Both groups performed the same training protocol but one group (CT) was subjected to a 20 min contrast bath protocol after each training bout whereas the other acted as a control (CON) and rested quietly for the same amount of time. Prior to commencing each training session, all three psychological questionnaires were administered. Subjects were instructed to refrain from exercise, alcohol, and caffeine consumption for 24 hours prior to their testing days. They were also asked to record their diet for the day before testing so that it could be duplicated prior to each subsequent training session. Only water was consumed before, during and after the trials.

2.3.3a Training. Training was initiated 3 days following baseline testing and consisted of 5 sessions of sprint interval training over a 5-day period. Each session consisted of repeated 30 s "all out" efforts on an electronically braked cycle ergometer (Lode, BV) against a resistance equivalent to 0.075 kg/kg body mass (i.e., a Wingate Test). Peak power, mean power and a fatigue index were determined using an on-line data acquisition system (Wingate Software version 1.11, Lode BV). Subjects were verbally encouraged to continue pedaling as fast as possible throughout the 30 sec test. During the 4 min recovery period between tests, subjects remained on the bike and either rested or were permitted to cycle at a low cadence and resistance (<50rpm @ <50 W) in order to reduce venous pooling in the legs and feelings of nausea. The number of Wingate Tests increased from 4 to 6 over the 5 training sessions. (Fig. 2.3.c)

2.3.3b Treatment. Within fifteen minutes following each training session subjects completed 1 of 2 recovery interventions. For the contrast therapy group, 2 large hydrotherapy tubs in the McMaster Sports Medicine Clinic were used (Ferno Performance Pools, model 160, Ottawa, ON)(Fig. 2.3.b.). One was maintained "hot" (38-43°C) and the other "cold" (10-16°C). Subjects started in the cold tub for 2 minutes followed by the hot for 3 minutes. This was repeated 4 times for a total treatment time of twenty minutes. The CON group rested quietly in the laboratory for twenty minutes after each training session.

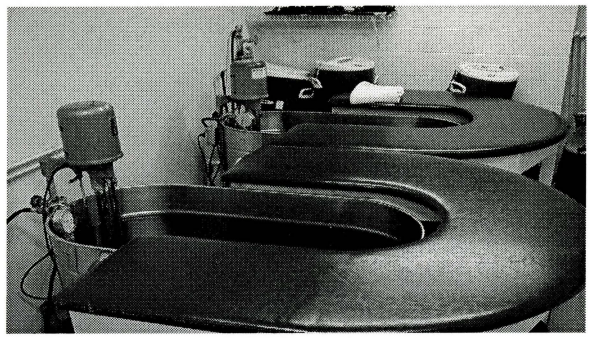
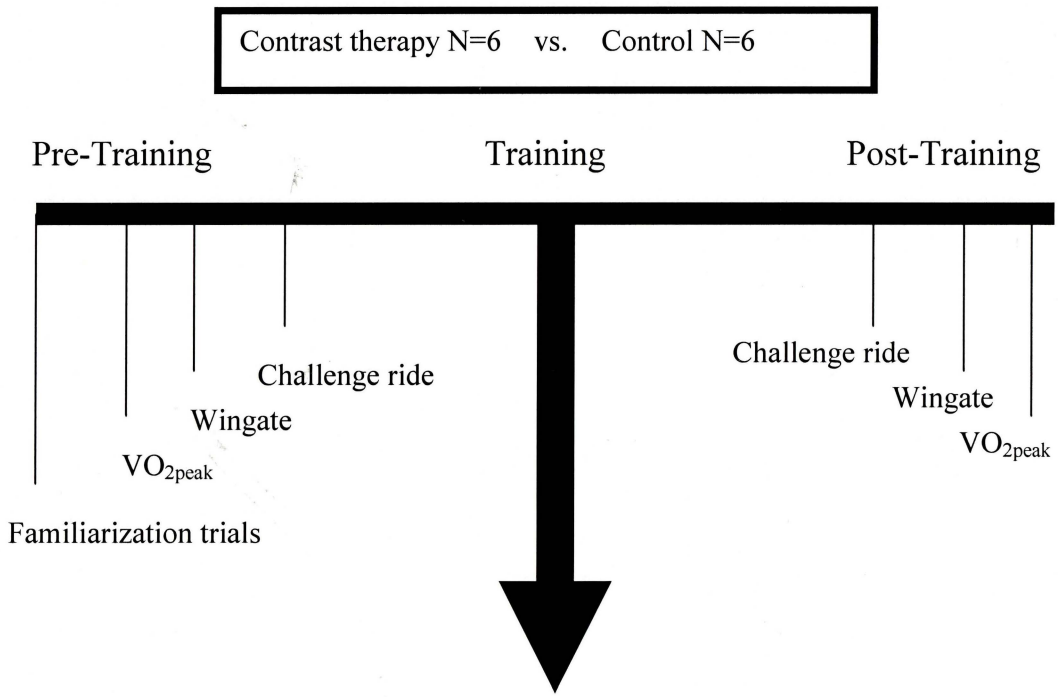


Figure 2.3.b – Hot and Cold hydrotherapy tubs used for contrast therapy protocol.

2.3.4 Post-experimental procedures

All baseline performance and psychological tests were re-administered 3 days following the final training session. The nature of the post-testing measurements was identical in all respects to the baseline test. (Fig. 2.3.c.)



Mon	Tue	Wed	Thur	Fri
-Psych. tests -4 Wingates (4 min rest btw bouts)	-Psych. tests -4 Wingates (4 min rest btw bouts)	-Psych. tests -5 Wingates (4 min rest btw bouts)	-Psych. tests -5 Wingates (4 min rest btw bouts)	-Psych. Tests -6 Wingates (4 min rest btw bouts)

Figure 2.3.c – Schematic design of experimental protocol

2.3.5 Statistical Analysis

The independent variable was the type of recovery (CON or CT) between training sessions. Dependent variables were VO_{2peak}, Peak anaerobic power, Mean anaerobic power, 250 kJ time trial performance, POMS, self-efficacy and PREP. The results were analyzed using SPSS (11.5 for Windows) statistical software. Performance and psychological data were analyzed using a 2-factor (time x condition) repeated measures analysis of variance. A Bonferroni calculation was used to compare main effects. The level of statistical significance was set at $P \leq 0.05$. All data are presented as means±SEM.

2.4 RESULTS

2.4.1 Time Trial Performance.

The mean improvement for the CT group was 56 s (6%) compared to baseline [Post: 15.8±0.6 vs Pre: 16.7±0.7 min] whereas the control group improved 42 sec (4%)[Post: 18.1±1.0 vs Pre: 18.8±1.2 min]. Both groups improved performance compared to the respective pre-training values however there was no difference between groups (main effect for time, $P \leq 0.05$). (Fig. 2.4.1)

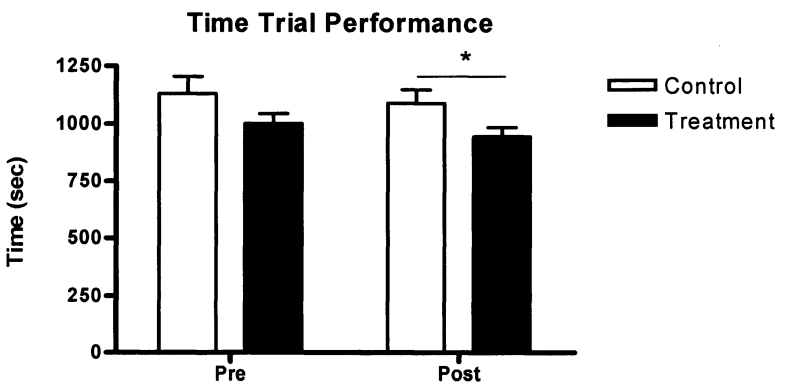


Figure. 2.4.1. Time trial performance before and after a 1-week sprint training protocol for control persons and subjects using contrast baths for recovery after training. Values are means±SEM (*time effect, $P \leq 0.05$).

2.4.2 Aerobic Capacity.

VO_{2peak} was unchanged after training (CT- Post: 46.5±1.1 vs. Pre- 45.4±1.5 and CON- Post: 46.8±1.4 vs. Pre- 47.0±1.1 $ml/kg^{-1}/min^{-1}$). (Fig. 2.4.2)

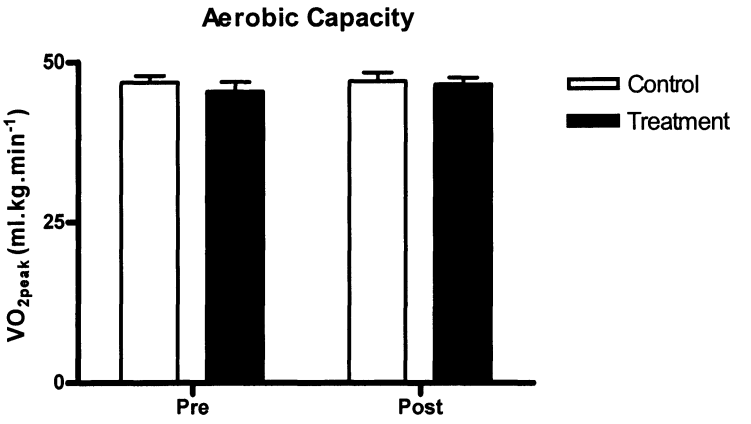


Figure 2.4.2 Peak oxygen uptake before and after a 1-week sprint training protocol for control persons and subjects using contrast baths for recovery after training. Values are means±SEM.

2.4.3 Anaerobic Capacity.

Peak power output improved 9% after the sprint interval training (time effect, $P \leq 0.05$), but there was no significant difference between groups (CT- Post: 1310±45 vs. Pre: 1215±86; CON- Post: 1343±54 vs. Pre: 1220±74 W). Mean power output was not significantly different after training for either group (CT- Post: 778±25 vs. Pre: 750±37; CON- Post: 740±23 vs. Pre: 724±21 W). (Fig. 2.4.3)

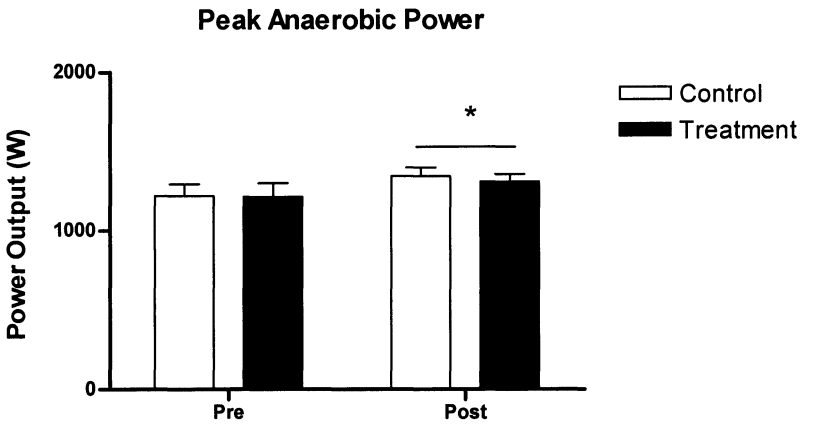


Figure. 2.4.3 Peak power output before and after a 1-week sprint training protocol for control persons and subjects using contrast baths for recovery after training. Values are means±SEM (*time effect, P ≤0.05).

2.4.4 Psychological Data.

Self-Efficacy scores improved throughout the training sessions with the biggest improvements in Cycling [Pre: 69±2% vs Post: 82±2%], and Running [Pre: 74±2% vs. Post: 79±2%](time effect, $P \leq 0.05$). Perception of strength, however, was consistently higher in the CON group [Pre: 99±2% vs Post: 84±2%](group effect, $P \leq 0.05$). Preparedness for Exercise answers were similar in both groups except for the question, "Does the treatment make your legs feel better?". For this question the CT group reported feeling more revitalized after each treatment session [CT: 8.5±0.4 vs. CON: 4.8±0.4) and better prepared for subsequent exercise (group effect, $P \leq 0.05$). In general there were no significant differences in POMS subscale scores throughout this study. Although there was a lack of statistical significance, some interesting patterns were noted. Despite the high intensity nature of the training, all scores for Depression, Fatigue and Tension improved from pre- to post-training regardless of group [Depression: Pre: 4.7±0.5 vs. Post: 1.0±0.5, Fatigue: Pre: 9.3±0.6 vs. Post:

4.1±0.6, and Tension: Pre: 9.5±0.5 vs. Post: 4.7±0.5). Scores for Vigor, however, were better in the CT group [Pre: 12.8±0.6 vs. Post:15.7±0.6] and worse in the CON group [16±0.6 vs. 13.3±0.6] after the training sessions. (Table 2.4.1)

		HYDRO		CONTROL	
		Pre	**Post**	**Pre**	**Post**
POMS	**Vigor**	12.8±0.1	15.7±0.1	16.0±0.1	13.3±0.1
	Depression	4.7±0.5	1.8±0.5	4.8±0.5	0.2±0.5
	Fatigue	9.6±0.6	5.0±0.6	9.0±0.6	3.2±0.6
	Tension	9.7±0.5	4.2±0.5	9.3±0.5	5.3±0.5
Self-Efficacy	**Cycling**	72.3±2%	81.7±2%†	66.4±2%	82.9±2%†
	Stairs	88.7±2%	91.3±2%	93.3±2%	98.3±2%
	Strength	81.7±2%	89.6±2%	96.7±2%*	99.6±2%*
	Jumping	73.8±4%	80.7±4%	87.7±4%	91.2±4%
	Running	68.7±2%	75.3±2%†	79.2±2%	83±2%†
Preparedness	**Exs. Ability**	7.7±0.2	7.4±0.2	9.3±0.2	8.3±0.2
	Leg Feeling	3.7±0.3	1.9±0.3	3.5±0.3	1.9±0.3
	Leg Pain	2.5±0.2	1.6±0.2	1±0.2	1±0.2
	ADL's	1±0.2	0.7±0.2	0.2±0.2	0.1±0.2
	Treatment effect	7.5±0.4*	9.0±0.4*	4.6±0.4	4.7±0.4

Table 2.4.1 Psychological data before and after a 1-week sprint training protocol for control persons and subjects using contrast baths for recovery after training. Values are mean±SEM (* group effect, †time effect - $P \leq 0.05$)

2.5 DISCUSSION

2.5.1 Main Findings

The present study examined the effect of two recovery interventions, contrast therapy (CT) and passive recovery (CON), on aerobic power, peak and mean anaerobic power, time trial performance and psychological measures related to mood state and prepared for exercise. The main finding from this study was five days of high intensity sprint training (~15 min total exercise time) increased peak anaerobic power (Wmax- increased ~9%) and decreased the time required to complete a defined bout of work (TT- decreased ~5%). Although contrast therapy did not further enhance

performance, it did create the perception that subjects were better prepared for exercise.

2.5.2 Contrast Therapy and Performance

Practice has preceded principle in many of the treatment protocols used in the sport medicine field. A lack of scientific understanding surrounds the use of contrast therapy, which is a therapeutic intervention used by athletes to recover from high intensity exercise. To our knowledge only one recent, controlled study specifically examined the physiological effects of CT. Myrer et al. (1994) proposed that for most of the physiological effects attributed to contrast therapy to occur, substantial fluctuations in tissue temperature must be produced with the alternations from hot to cold. It was concluded that contrast therapy was incapable of producing any major physiological effects as temperature alterations from hot to cold did not produce any significant intramuscular changes, statistically or clinically (Myrer et al., 1994). From an athletic perspective, we are unaware of any studies examining the effects of contrast baths on performance.

Although performance improvements have been noted after consecutive days of SIT (Parra et al., 2000), greater improvements have been found when 1-2 days of rest were introduced between training sessions. Burgomaster et al. (2005) found that 6 sessions of SIT (4-7 Wingates/session) performed over 14 days increased muscle oxidative potential and doubled endurance time to fatigue during cycling at ~80% VO_{2peak}. Would recovery interventions have a similar effect as rest during consecutive training sessions? In our study, although performance increases were observed after 5 consecutive days of SIT, no difference was found between treatments. Our data do not support, or refute, the

previous theories that contrast baths reduce intramuscular temperature or induce the vascular changes necessary for "physiological recovery" after strenuous exercise. Unfortunately we were not able to use subcutaneous microprobes to directly assess these parameters. Research investigating the interaction between performance and the independent use of hot or cold is more evident. In one study, appearing in abstract form, cold-water immersion (10 min @ 8°C) following intense running improved subsequent performance in submaximal exercise (Fowles et al., 2003). Rogers and Albrechtsen (2003) also found that cold-water immersion (9 min @ 10°C) between bouts of high intensity sprint exercise was effective for improving both peak anaerobic and relative peak anaerobic power. Similar studies by Verducci (1997, 2000) also found power and performance were maintained after the application of cryotherapy. It seems the use of cold therapy, more than contrast, is important for recovery from intense exercise.

2.5.3 Contrast Therapy and Psychological Changes

There is a widespread belief that exercise is associated with improvements in mental health including mood state, self-efficacy, psychological 'well-being', and self esteem (Yeung, 1996; Scully et al., 1998). Elevations in training volume and intensity, however, have also been associated with increases in mood disturbances (Raglin, 1990). In the present study, we studied the effect of SIT and contrast therapy on profile of mood state (POMS), self-efficacy (SE) and preparedness for exercise (PREP). Over the successive sessions of intense exercise, most scores for POMS and SE improved regardless of intervention. This is in contrast to the research of Steptoe

and Bolton (1988), who found that low intensity exercise (25W) produced modest improvements in mood as measured by the POMS, while higher intensity exercise (100W) increased negative mood states. Perhaps while some subjects may find the physical discomfort of high intensity exercise distressing, other subjects may enjoy the feeling of tiredness from a "good workout'. Additionally, subjects may experience a greater sense of accomplishment or self-efficacy after completing a difficult session of SIT. Exercise of high intensity but low volume, therefore, may be a positive stimulus for psychological well-being. Another important finding from this study was that the use of CT created the perception that subjects were better prepared for exercise. Over the course of training, POMS scores for vigor improved with CT yet worsened with the CON. Similarly, the CT group reported feeling more revitalized and ready for exercise after treatment than the CON group. Perhaps the use of alternating hot and cold hydrotherapy provides an analgesic effect? Although Myrer et al. (1994) found no physiological effect of contrast therapy on intramuscular tissue 1cm below the skin, they did support an influence on the superficial layer of fat and skin. At lower temperatures, the rate of firing of pain and temperature sensory receptors located in the skin – the superficial tissue most affected by cold therapy- is diminished, thus reducing the sensation of pain (Bugaj, 1975; Meeusen and Lievens, 1986; Enwemeka et al. 2002). Similar findings have also been reported with the application of heat (Knight, 1995). With less feeling of pain and heightened psychological well-being athletes may feel greater self-efficacy when commencing subsequent bouts of exercise. Little is known, however, about the specific factors that may contribute to these psychological effects.

2.6 CONCLUSION

Despite the fact that sprint interval training improves peak anaerobic power and endurance time trials, the use of contrast therapy as a form of recovery after training does not appear to further improve performance. The use of contrast therapy, however, did create the perception that subjects were better prepared to exercise. With regards to the mechanisms proposed to mediate these effects, the role of self-efficacy and analgesia from the hot and cold cannot be discounted. In all likelihood both psychological and physiological factors are involved. Perhaps the use of contrast therapy during high intensity exercise will allows athletes to train harder and in the long term, training effects may be more evident. Further studies, therefore, are warranted to substantiate the effects of long-term use of this modality in recovery from intense training in the athletic population.

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CHAPTER 3

REST, LIGHT EXERCISE OR CRYOTHERAPY: WHAT IS THE MOST EFFECTIVE WAY TO RECOVER BETWEEN REPEATED BOUTS OF INTENSE EXERCISE?

3.1 ABSTRACT

The goal of athletic training is to enhance performance. However, chronic high volumes of intense exercise can lead to muscle fatigue, injury and performance decrements. Athletes use a wide range of interventions to promote recovery from strenuous exercise, but few data are available regarding the efficacy of such practices. OBJECTIVE: To examine the effectiveness of three commonly used interventions [Rest, light exercise (AR) and cryotherapy (CR)] during recovery between bouts of intense exercise. We tested the hypothesis that CR and AR would induce favorable metabolic, immunological and psychological alterations such that performance would be improved versus Rest. METHODS: After extensive familiarization, 9 'recreationally active' men (29±6 yr, VO_{2peak} = 44±8 ml/kg/min; mean±SD) performed 3 exercise trials separated by 1 week. Each trial consisted of 3 x 50 KJ "challenge rides" (time trials at an intensity equivalent to ~100-120%VO_{2peak}) with a 20-min recovery period between rides. In a randomized manner, a different recovery intervention was applied between rides each week: Rest (rest supine); AR (cycling @ 50W) or CR (cold tub @ 10°C). Venous blood samples were obtained from an indwelling forearm catheter at rest and after each recovery period, and analyzed for lactate, interleukin-6, neutrophils, and lymphocytes. Questionnaires designed to assess pain, ratings of perceived exertion, and preparedness for exercise were

completed after each recovery period. RESULTS: Time trial performance averaged 118±10 s (mean±SEM) for bout 1 and was 8% and 14% slower during bouts 2(128±11 s) and 3 (134±11 s), respectively, with no difference between treatments (Time effect, $P \leq 0.05$). All blood markers increased after exercise compared to baseline (Time effect, $P \leq 0.05$). There were no differences between treatments with the exception of lactate, which was lower after bout 3 in AR (3.5±0.4 mmol/L) vs Rest (4.3±0.4) and CR (4.1±0.2). In addition, neutrophils and lymphocytes were higher and lower respectively ($P \leq 0.05$), after CR (8.7±1.3 and 1.4±0.2 x 10^9cells/L) versus AR (7.1±1.0 and 1.6±0.1) and Rest (6.7±0.7 and 1.6±0.1). Subjects also reported a higher preparedness for exercise score after CR (6.0±0.7/10) versus AR (4.8±0.9) and Rest (2.8±0.6) (Treatment effect, $P \leq .05$). CONCLUSIONS: The type of recovery intervention did not affect the decline in exercise performance during repeated bouts of intense cycling. Blood lactate was lower after AR, whereas, CR caused greater perturbations in blood immune markers and created the perception that subjects were better prepared for subsequent exercise.

3.2 INTRODUCTION

A common training model used by coaches and athletes is based on the "Overload Principle" or "Physical Stress Theory" (Baechle and Earle, 2000). An essential component of the model is that high intensity physical exercise creates a disturbance in cellular homeostasis. This disturbance then acts as a stimulus that initiates physiological responses in order to restore homeostasis and induce training adaptations (Kuipers, 1994,1998; Väänänen, 2004). To push performance to the limit of tolerance, therefore, relatively high amounts of intense training must be performed. Although the "optimal" amount of high-intensity training remains unclear, athletes are generally inclined to do "too much". When intense exercise and the associated disturbance in homeostasis are not followed by adequate recovery, athletes may experience excess muscular fatigue, soft tissue injury, and/or immune compromise (Espersen et al., 1990; Bruunsgaard et al., 1997; McKenzie, 1999; Yamada et al., 2002).

During strenuous or high intensity exercise a discrepancy may develop between the demand for energy and energy provision from oxidative metabolism. This results in a depletion of phosphocreatine (PCr) and a large accumulation of lactate [La] as the muscle is forced to rely on non-oxidative sources of energy (Bogdanis et al. 1995, 1996; Gupta et al., 1996). Intracellular acidosis associated with elevated levels of lactic acid is the most popular hypothesis to explain muscular fatigue, as it is believed the excess hydrogen ions (H^+) interferes with the muscle contraction process (Roberts and Smith, 1998). Following a bout of strenuous exercise, therefore, PCr stores and acid-base balance must be restored in exercised muscles, thereby preparing the tissues for subsequent physical challenges. If

full metabolic recovery is not attained, fatigue and/or muscle injury may result. It is currently believed that a period of active recovery, as opposed to passive recovery, enhances the rate of lactate removal (Ahmaidi et al., 1996; Bogdanis et al., 1996; Hudson et al., 1999; Sairyo et al., 2003). Low intensity exercise (<50% VO_{2max}) during the recovery phase after exercise is thought to enhance the rate of lactate uptake and oxidation by the previously active muscles (Belcastro and Bonen, 1975). Few studies, however, have assessed the effect of other recovery interventions, such as cold therapy, on blood lactate clearance.

Along with metabolic changes, it is also widely accepted that high intensity training and competition results in microtrauma to muscle and connective tissue (McCully, 1986; Kuipers, 1994; Bruunsgaard et al., 1997). With repeated high volume, high intensity training and limited recovery, the trauma induced may negatively impact training and performance. Several muscle injury markers have been proposed to estimate the extent of muscle damage but all have their limitations. One common method is to measure changes in venous blood concentrations of muscle-specific enzymes, such as creatine kinase (CK) and lactate dehydrogenase (LDH); however, these are at best qualitative markers of structural damage (Komulainen et al., 1995). It is also well established that strenuous exercise causes an increase in blood concentrations of various pro-inflammatory cytokines (Pedersen, 2000; Suzuki et al., 2000; Toft et al., 2002). After high intensity exercise, cytokines (IL-1, IL-6, TNFα) are produced locally in the damaged muscle (Ostrowski et al., 2000). As changes in IL-1 and TNFα after exercise tend to be subtle, IL-6 is one cytokine that provides the most observable changes, being

elevated shortly after strenuous exercise and tissue damage (Espersen et al., 1990; Bruunsgaard et al., 1997; Suzuki et al., 1999, 2000). Once established, IL-6 has the capacity to stimulate surrounding cells (paracrine) or themselves (autocrine), which leads to further IL-6 production and amplification of the inflammatory response (Pedersen, 2000). The majority of available data, however, have been obtained following prolonged exercise (Ullum et al., 1994; Suzuki et al., 1999, 2000) and it remains to be confirmed whether short-duration intensive exercise also causes rapid systemic cytokine release. Furthermore, few studies have examined the cytokine response to different recovery interventions after exercise.

The production of pro-inflammatory cytokines has also been associated with alterations in immune activity after high-intensity exercise. Following a single bout of high intensity exercise, substances, including pro-inflammatory cytokines (IL-6) and stress hormones such as catecholamines, cortisol and growth hormone, increase in response to injured muscle cells, which perpetuate the inflammatory response (Cox and Gauldie, 1997; Suzuki et al., 1999, 2000; Yamada et al., 2002). These substances regulate the rapid migration of leukocytes, neutrophils and later monocytes, into areas of damaged muscle cells and other metabolically active tissue in order to initiate repair. In the 15-60 minute period after the cessation of exercise, the initial increase in leukocytes (excluding neutrophils which continue to increase) subsequently falls below resting values (MacKinnon, 1999). This immediate decrease in leukocytes post exercise has been mediated by active recovery. Wigernaes et al. (2000, 2001) found that active recovery (15min @ 50% VO_{2max}) as opposed to rest recovery prevented the initial fall in

white blood cell count after strenuous endurance exercise. In addition, cold therapy may also influence immune activity. Limited evidence suggests leukocyte mobilization can be modulated by cold exposure during intense exercise (Jansky et al., 1996; Shephard and Shek, 1998c; Brenner et al., 1999; Rhind et al., 2001). However, the impact of cold therapy as a recovery intervention between bouts of high intensity exercise has not been explored.

It is generally accepted that intense physical activity results in muscle injury, production of lactate, and other metabolic/endocrine substances, which result in muscle fatigue, decrements in performance and possible immunosupression. Interestingly enough, all these adverse effects of exercise can be reversed with adequate rest and recovery. With short seasons and intense competition schedules, however, the time needed for rest and recovery is overlooked. Athletes, therefore, use many different therapeutic interventions for recovery between intense bouts of exercise.

The primary purpose of this investigation was to examine the effect of Rest, Active recovery (AR) and Cryotherapy (CR) on time trial performance. Many athletic events require athletes to complete a fixed amount of work in as short a time as possible (ie. a fixed distance race). Thus, for our study this type of performance test is preferable to other traditional tests such as prolonged exercise to exhaustion or extremely short high intensity activities (<10sec.). Secondly, we also examined changes in the proposed blood markers for muscle fatigue (blood lactate), systemic inflammation (IL-6), and immunosuppression (leukocytes - lymphocytes, neutrophils) at several time points throughout testing. Our intention was to clarify the metabolic effects of the recovery

interventions that have previously only been speculated upon. Finally, we also studied the potential psychological effects of these interventions. We hypothesized that CR and AR used for recovery between bouts of high intensity cycle training would induce favorable metabolic, immunological and psychological alterations such that performance will be improved versus Rest.

3.3 METHODS

3.3.1 Subjects

Nine healthy men with a mean age, height and weight of 29±5.8 y, 1.80±7.8 m and 96.9±13.2 kg respectively (mean±SD) , volunteered for the study. Participants were recruited via word of mouth and posters placed around the McMaster University campus. All were "recreationally active" although one played university rugby. None of the participants suffered from any illnesses or had been taking any medication for at least 4 weeks prior to, or during, the experimental period. The experimental procedure and potential risk factors were thoroughly explained prior to beginning the study, and all participants provided written, informed consent. McMaster University and the Hamilton Health Sciences Corporation Research Ethics Boards approved the experimental protocol.

3.3.2 Pre-experimental Procedures

Following familiarization with all testing procedures and equipment, participants performed a series of baseline performance tests (over 2 days) prior to the experimental training protocol. All subjects underwent a progressive exercise test on an electrically

braked cycle ergometer (Lode BV, Excalibur Sport V2.0, The Netherlands) to determine their peak aerobic power (VO_{2peak}). Peak oxygen uptake was determined via gas measurements taken from a mouthpiece attached to an online gas collection system (Moxus modular oxygen uptake system, AEI technologies, Pittsburg, PA) during the incremental cycle ergometer protocol. Volitional fatigue was used to determine the end of the test. VO_{2peak} was 44.4±8.0 $ml.kg^{-1}.min^{-1}$ (mean±SD). Subjects also performed a simulated "time trial" race, whereby they were instructed to complete a standardized bout of work (50kJ) in as fast a time as possible. Pace was self-selected throughout the test, which lasted between 2 to 5 min depending on the fitness level and motivation of the subject. Subjects rested 20 min as per the training protocol and repeated the trial a second time. Subjects were instructed to refrain from caffeine the day of testing, and strenuous exercise and alcohol consumption for 24 hours prior to their testing trial days. They were also asked to record their diet for the day before testing so that it could be duplicated prior to each subsequent training session.

3.3.3 Experimental Protocol

Each subject participated in 3 exercise trials separated by 1 week. All cycling trials were carried out on the electrically braked cycle ergometer (Lode BV, Excalibur Sport V2.0, The Netherlands) used for pre-testing. Upon arrival at the laboratory, a 20 ga. catheter was inserted into an anticubital vein and a resting blood sample was obtained. Prior to the exercise trials, a standard 10 min warm up at 50 W was performed. In each trial subjects performed three 50 KJ challenge rides (cycling at ~100-120%VO_{2peak})

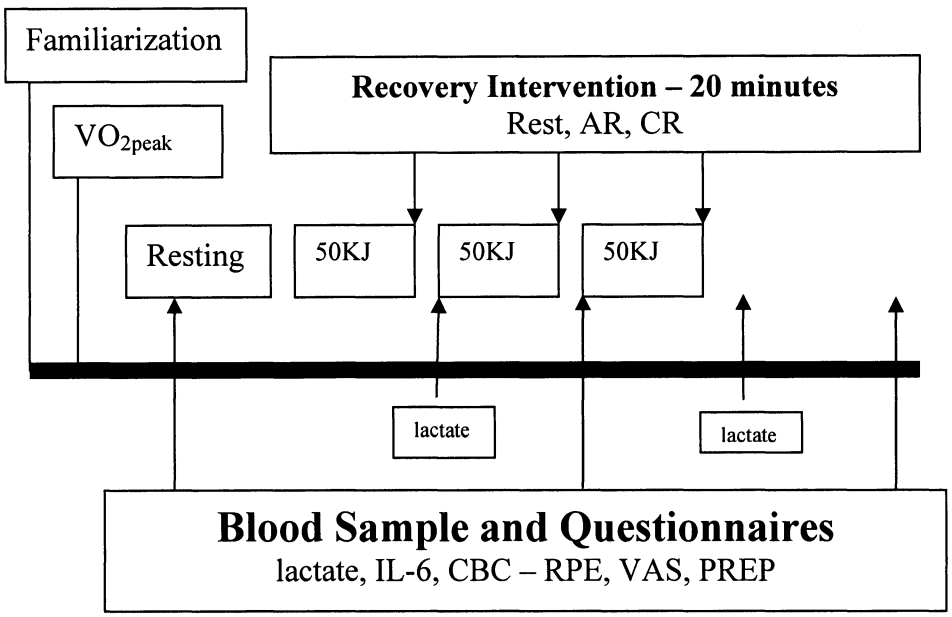
separated by a 20 minute recovery intervention Recovery interventions were randomized as to order for each week based on a latin square counter balance, with all subjects participating in all training/treatments over a 3 week period.

A	B	C
B	C	A
C	A	B

Table 3.3.1 – Latin square counter balance used to randomize the order of therapeutic interventions for each group (Streiner and Norman, 1986)

Recovery interventions included resting supine on a bed for 10 minutes (Rest), pedaling on a cycle ergometer for 10 min at 50W (AR), and sitting in a cold bath (10°C) for 10 min (CR). 10 additional minutes were allowed for transition time from the training room to the recovery room and back.

During the exercise protocol blood samples were collected immediately after each of the recovery interventions and 1 hour following the final recovery intervention. To gauge psychological status for exercise, subjects completed a Visual Analogue Scale (VAS) for general pain and a "Preparedness for Exercise" (PREP) questionnaire prior to commencing each bout of exercise. The Borg scale for ratings of perceived exertion (RPE) was completed immediately upon finishing each exercise bout. The same experimental protocol was used for each trial. (See Fig. 3.3.1)



Pre	Week 1	Week 2	Week 3
Testing & Familiarization	Training – 3 bouts Random intervention	Training – 3 bouts Random intervention	Training – 3 bouts Random intervention

Figure 3.3.1 – Schematic of training and experimental protocol

3.3.4 Blood Analyses

All blood samples were measured in duplicate. To avoid interassay variation, all samples were analyzed in one batch at the end of the study, with the exception of hematological measures, which were performed on the day of the collection.

3.3.4a Blood lactate

Blood samples (2 ml) for lactate determination were collected in Vacutainer tubes containing heparin and placed immediately on ice. Plasma was obtained via centrifugation (3000rpm for 5 min at 4°C). The plasma was then stored at -20°C until analysis. 50 nml of plasma was combined with 250 nml of perchloric acid (PCA),

vortexed and centrifuged (4500 rpm for 5 min). The PCA extract was subsequently analyzed for lactate, using an enzymatic assay adapted for fluorometry (Hitachi model F-2500, Hitachi Instruments, Japan) as described by Passoneau and Lowry (1993).

3.3.4b IL-6

Blood samples (5 ml) for IL-6 determination were collected in Vacutainer tubes containing fluoride/ethylenediaminetetraacetic acid (fluoride/EDTA) and centrifuged (3000rpm for 5m at 4°C). Plasma was stored at -20°C until analyzed by a commercially available enzyme-linked immunoassay kit. Plasma concentrations of IL-6 were determined with a High Sensitivity ELISA (Quantikine HS [HS600B] – R&D systems, Minnesota). The sensitivity of this assay, as reported by the manufacturer, is 0.039 pg/ml. Concentrations were calculated by comparison to a calibration curve established in the same measurement.

3.3.4c Hematology

Blood samples (5 ml) for complete blood count (CBC) determination were collected in vacutainer tubes containing fluoride/ethylenediaminetetraacetic acid (fluoride/EDTA) and placed immediately on ice. Whole blood treated with EDTA was analyzed for total leukocytes, neutrophils, lymphocytes, Hb and hematocrit with an automated Coulter counter by the clinical hematology group at McMaster University.

3.3.5 Statistical Analyses

This study was performed in a randomized crossover design. The independent variable was the type of recovery (REST, AR or CR) between exercise bouts. Dependent variables involved all blood variables analyzed: Blood Lactate, IL-6, total Leukocytes

and subsets (neutrophils and lymphocytes); performance (50 kJ time trials); and psychological parameters (RPE, VAS and PREP).

The results were analyzed using SPSS (v.11.5 for Windows) statistical software. All plasma variables were adjusted for plasma volume differences (Dill and Costill, 1974). Blood samples and performance data were analyzed using a 2-factor (time x condition) repeated measures analysis of variance. A Bonferroni test was used to compare main effects. A Pearson Product Moment Correlation was used to determine if a relationship existed between IL-6, leukocytes, neutrophils, and lymphocytes. The level of statistical significance was set at $P \leq 0.05$. All data are presented as means±SEM.

3.4 RESULTS

3.4.1 Time Trial Performance

Time trial performance averaged 118±10 s (mean±SEM) for bout 1 and was 8% and 14% slower during bouts 2 (128±11 s) and 3 (134±11 s), respectively, with no difference between treatments (main effect for time, $P \leq 0.05$). (Table 3.4.1.)

	Bout 1	*Bout 2*	*Bout 3*
Cryotherapy	115.4±9.5	*129.4±12.7	*134.9±12.3
Active Recovery	120.0±10.8	*128.4±10.0	*135.3±9.6
Rest	119.8±10.8	*126.9±10.7	*134.2±9.7

Table 3.4.1 – Performance time in seconds over successive bouts of high intensity exercise in subjects using cryotherapy, active and rest recovery interventions between the bouts of exercise. Values are means±SEM (*time effect, $P \leq 0.05$).

3.4.2 Blood Lactate

Following each supramaximal bout of exercise, blood lactate [La] was increased above resting concentrations in all three interventions. [La] at rest averaged 0.52±0.1 mmol/kg, and increased to 3.92±0.3, 4.13±0.3, and 3.99±0.3 mmol/kg after bouts 1, 2, and 3 respectively (main effect for time, P≤0.05). Although [La] remained unchanged for CR and Rest over the 3 bouts and AR decreased 14% from bout 1 to bout 3, this was not statistically significant. Regardless of group, [La] returned to near resting levels (1.03±0.1 mmol/kg) one hour after completing the final recovery intervention. (Fig. 3.4.1)

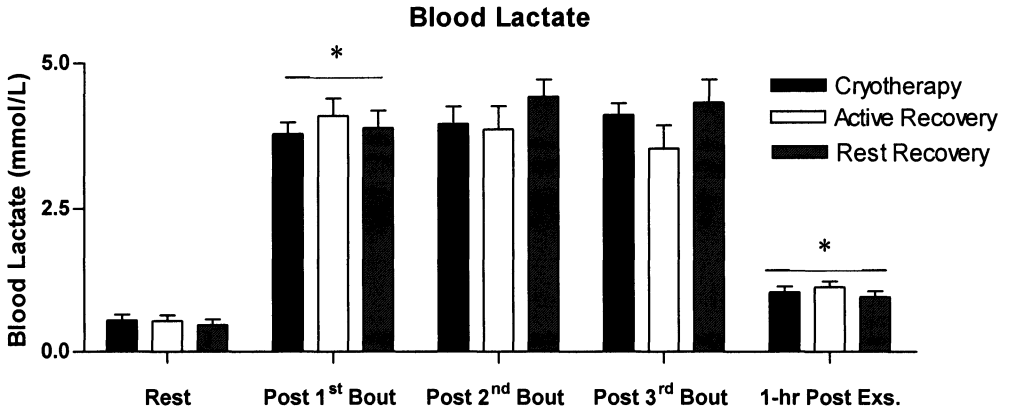


Figure 3.4.1 – Blood lactate concentrations before, during and 1 hour after bouts of high intensity exercise in subjects using cryotherapy, active and rest recovery interventions between the bouts of exercise. Values are means±SEM (*main effect for time, P ≤0.05).

3.4.3 IL-6

The resting values of circulating plasma IL-6 were similar in all three groups; CR: 1.64±0.3, AR:1.46±0.2 and Rest:1.97±0.6 pg/ml. IL-6 levels increased significantly from an average of 1.69±0.5 to 2.38±0.8 pg/ml after 2 bouts of exercise with no difference observed between groups (main effect for time, P≤0.05). During the hour after the final

recovery intervention, IL-6 levels continued to increase to a peak value of 3.98±1.2 pg/ml, 40% higher than during exercise (main effect for time, P≤0.05). Although CR levels were higher than AR or Rest in the final sample, 3.98±1.2 vs. 3.30±0.5 and 2.85±0.6 pg/ml respectively, the changes were not statistically significant. (Table 3.4.2)

	Rest	*Bout 2*	*1 Hour Post*
Cryotherapy	1.64±0.3	2.30±0.4*	3.98±1.2*
Active Recovery	1.46±0.2	2.24±0.3*	3.30±0.5*
Rest	1.97±0.6	2.61±0.8*	2.85±0.6*

Table 3.4.2 – IL-6 levels (pg/ml) before, during and 1 hour after bouts of high intensity exercise in subjects using cryotherapy, active and rest recovery interventions between the bouts of exercise. Values are means±SEM (*main effect for time, P ≤0.05).

3.4.4 Leukocytes

Intensive exercise caused total leukocyte concentrations to increase. Concentrations after 2 bouts were 10.59±0.7 (CR), 9.08±0.9 (AR) and 9.64±1.0 (Rest) x 10^9 cells/L. In the hour after the final intervention these numbers remained consistent with the CR numbers 22% higher than AR or Rest (main effect for group, P≤0.05)(Fig. 3.4.2a). In the leukocyte subsets a similar trend was observed. In general we observed an exercise-induced neutrocytosis in all groups (Fig.3.4.2b). For CR, circulating neutrophil counts increased from 5.13±0.4 x 10^9 cells/L during exercise to 8.71±1.3 x 10^9 cells/L one-hour post (ie. 140% above baseline)(main effect for group, P≤0.05). In contrast, AR and Rest increased on average 54% from 4.46±0.4 to 6.87±0.9 x 10^9 cells/L. The increase in lymphocyte count in response to exercise was similar for all groups [CR: 4.14±0.4, AR: 3.62±0.6, Rest: 3.60±0.5 x 10^9 cells/L] namely an 83% increase from

resting levels (main effect for time, P≤0.05). Reductions in circulating lymphocyte concentrations during the hour after the final intervention were not significantly different between CR, AR and Rest. However, there was a trend for a steeper decline in the CR group (66%) as compared to the AR (57%) and Rest (56%) groups. For all groups, lymphocyte count one hour after the final intervention was 27% below baseline (main effect for group x time, P≤0.05)(Fig. 3.4.2c.).

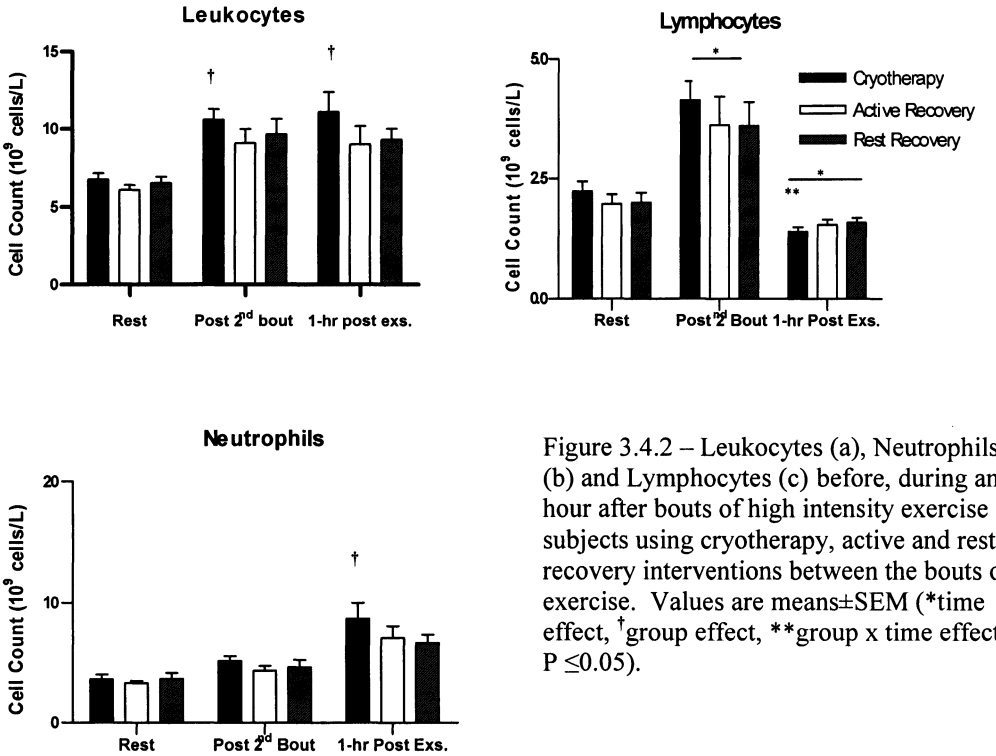


Figure 3.4.2 – Leukocytes (a), Neutrophils (b) and Lymphocytes (c) before, during and 1 hour after bouts of high intensity exercise in subjects using cryotherapy, active and rest recovery interventions between the bouts of exercise. Values are means±SEM (*time effect, †group effect, **group x time effect, P ≤0.05).

3.4.5 Correlations

Pearson's correlation coefficients were used to test whether the subjects with the largest IL-6 responses also had the most marked leukocyte changes. No significant correlations were found for IL-6 with leukocytes, neutrophils, or lymphocytes.

3.4.6 Psychological Data

General scores for pain as determined by a visual analogue scale (VAS) increased 270% across the trials (main effect for time, $P \leq 0.05$). Although there was no effect for group, CR scores were on average consistently lower than AR and Rest after each bout (12.2±3.1 vs. 14.4±4.0 and 17.4±3.9 respectively). Similar results were found with the Borg scale for ratings of perceived exertion (RPE). Although all group ratings increased 6% over the three bouts, CR scores were consistently lower (CR-17.0±o.4, AR-17.5±0.5, Rest-17.7±0.5). Finally, subjects perceived CR to result in more complete recovery as indicated by a significantly higher group mean for question 5 on the preparedness for exercise questionnaire asking "does this recovery intervention make you better prepared to exercise?"(main effect for group, $P \leq 0.05$).(Table 3.4.3)

		CRYO	*ACTIVE*	*REST*
VAS	Bout 1	8.8±1.8	10.2±2.7	14.8±3.8
	Bout 2	12.5±3.1*	15.5±4.3*	18.1±4.5*
	Bout 3	15.4±4.3*	17.6±4.6*	19.3±4.8*
RPE	Bout 1	16.4±0.4	16.9±0.5	17.1±0.4
	Bout 2	17.1±0.3	17.8±0.5	17.8±0.5
	Bout 3	17.6±0.6	17.7±0.5	18.2±0.6
Preparedness	Ability to Exs.	7.0±0.5	6.4±0.6	5.9±0.6
	Feeling in legs	3.6±0.7	3.5±06	4.1±0.6
	Pain in legs	1.4±0.4	1.4±0.4	2.0±0.6
	ADL's	1.4±0.4	1.4±0.4	1.4±0.4
	Treatment effect	6.0±0.7†	4.8±0.9	2.8±0.6

Table 3.4.3 Psychological data after repeated bouts of high intensity exercise in subjects using cryotherapy, active and rest recovery interventions between the bouts of exercise. Values are means±SEM (main effects for *time, and †group, P ≤0.05).

3.5 DISCUSSION

3.5.1 Main Findings

The present study examined the impact of three recovery interventions, Cryotherapy (CR), Active Recovery (AR) and Rest on time trial performance, blood lactate, IL-6 and selected markers of immune activity including leukocytes, neutrophils, and lymphocytes. The principal finding from this study was that the type of recovery intervention did not affect the decline in exercise performance during repeated bouts of intense cycling. Secondly, although blood lactate levels were slightly reduced after AR (no sig.), all groups (CR, AR and Rest) returned to resting levels 1 hour after the final intervention. Finally, the use of CR caused greater perturbations in blood immune markers and created the perception that subjects were better prepared for subsequent exercise. This work confirms that brief repeated bouts of intense cycle exercise can induce acute changes in blood markers of systemic stress. Additionally, despite the fact that performance was unaffected, the use of recovery interventions may play a role in attenuating the metabolic effects of strenuous exercise.

3.5.2 Performance

Contrary to our hypothesis no specific recovery intervention impacted physical performance during this study. Despite the use of recovery interventions, time trial performance were on average 8% and 14% slower during bouts 2 and 3 respectively with no difference between treatment groups. McAinch et al. (2004) identified similar findings after studying active recovery (15 min cycling at 40% VO_{2max}) between bouts of

intense aerobic exercise (20-min cycling at 75% VO_{2max}). Despite the fact that blood lactate levels were lower in AR versus Rest (main effect for group, $P<0.05$), work performed in Bout 2 was less than that performed in Bout 1, regardless of intervention. Paddon-Jones and Quigley (1997) assessed the effect of cryotherapy (20 mins @ 5 °C – cold bath) on muscle soreness and strength following eccentric elbow flexor exercises. No significant differences between the control and immersed arms were found for any variable. This does not necessarily mean that recovery interventions are ineffective. In another study, 3-minute intervals of cryotherapy (ice packs) by baseball pitchers on pitching arms between innings resulted in a significantly higher number of innings pitched with increased velocity and no alterations in accuracy (Verducci, 1997). The same author also found that interval cryotherapy (ice packs) on shoulders, between weight-pulling sets, was associated with increased work, velocity and power (Verducci, 2000). Similarly, studies by Rogers and Albrechtsen, (2003), Fowles et al., (2003) and Sargeant (1987) used various forms of cryotherapy between bouts of exercise and found power and performance were maintained. In all these field studies, however, the exact mechanisms for improvement were not investigated.

3.5.3 Blood Lactate response

During strenuous exercise (>75% VO_{2peak}) blood lactate concentrations [La] reflect the imbalance between anaerobic metabolism for energy production and use (Parra et al., 2000). The resulting increase in plasma [La] and low intramuscular pH play an important role in the development of muscular fatigue. Following a bout of strenuous exercise,

therefore, the exercising muscles must reduce [La] and restore acid-base balance, thereby preparing the tissue for subsequent physical challenges. Almost all investigations agree that optimal lactate clearance occurs by engaging in light-moderate activity (40-60% VO_{2max} x 10-20 min) during recovery (Belcastro and Bonen, 1975; Monedero and Donne, 2000; McAinch et al., 2004). In our study subjects performed active recovery on a stationary bike for 10 min at 50W (<50% VO_{2peak}). This form of recovery resulted in a 14% drop in [La] from bout 1 to bout 3. Despite the decrease in [La], however, there was no improvement in performance. This finding is similar to the results of McAinch et al. (2004), who found plasma [La] were higher in Rest (supine rest-10min) than AR (10min cycle @ ~40% VO_{2max}) during the recovery period between bouts of exercise. Despite this fact, work performed in subsequent bouts was less than the first bout, with no difference between interventions. Other studies (Gupta et al., 1996; Hudson et al., 1999; Monedero and Donne, 2000; Sairyo et al., 2003) used similar active recovery intensities and durations between repeated bouts of intense exercise, with similar findings in [La] reduction compared to Rest.

In two studies, however, AR was not more effective than PR in [La] recovery. Bogdanis et al. (1996) and Ahmaidi et al. (1996) used 4-5 minutes recovery interventions (cycling @ 30-40% VO_{2peak} vs. Rest) between bouts of 6-30s maximal sprints. No significant differences were found between interventions for venous blood lactate in either study. Perhaps the short duration of the active recovery was insufficient for significant changes in [La]. In most studies, decreases in blood lactate during active recovery were found only when the AR was prolonged (>10min), which allows more

time for lactate "uptake" by other tissues and muscles (Belcastro and Bonen, 1975; Mondero and Donne, 2000; McAinch et al., 2004).

Only one study has looked at the effects of hydrotherapy on blood lactate recovery after intense exercise. Hudson et al. (1999) used 20 min of running in water (33°C) at ~30% VO_{2peak} after 3 x 45 s bouts of supramaximal sprints (treadmill @ 8.6mph, 3% grade). The reduction in blood lactate concentration became significantly lower than Rest at the 8 min mark of recovery and remained significantly lower for a further 20 min. [La] was not significantly different from AR at any point in recovery. In our study, CR had no effect on [La], which increased 9% from bout 1 to 3, and was significantly higher than the AR intervention. It is difficult to compare these two studies, however, as the duration of recovery times, the type of hydrotherapy, and the temperature of the water were significantly different. It would seem active recovery following repeated bouts of high intensity exercise is more effective at decreasing [La] compared to passive recovery or cryotherapy. This form of recovery, however, is only evident in the recovery periods between and immediately after training, as 1 hour post exercise, all [La] regardless of recovery intervention, returned to resting levels.

3.5.4 IL-6 response

Plasma IL-6 concentrations are found to be elevated in a variety of conditions where system homeostasis is threatened or compromised. Recent findings have also demonstrated that strenuous exercise is a powerful inducer of elevated concentrations of cytokines (Espersen et al., 1990; Ostrowski et al., 1998; Suzuki et al., 1999). The results

of our study confirm the existence of a relationship between plasma IL-6 concentration and short duration, high intensity exercise. After 2 bouts of intense exercise plasma IL-6 increased in all groups by an average of 41% from resting levels and a further 60% in the 1h post exercise. This suggests concentric cycling at a high intensity and short duration is a factor in determining the increase in plasma concentration of IL-6. Ullum et al. (1994) found IL-6 levels increased 63% from resting levels during intense cycling (1 h @ 75% VO_{2peak}). In this study, however, IL-6 levels returned to resting levels 1 h after exercise. Perhaps intensity is more important than duration for stimulating IL-6 concentrations during concentric cycling exercises, as the IL-6 levels in our study continued to increase in the hour after the cessation of exercise.

The actual mechanisms responsible for the release of cytokines during and after exercise are not fully understood. It has been suggested that the release of pro-inflammatory cytokines represents an inflammatory reaction, presumably initiated by damage to skeletal muscle (Bruunsgaard et al., 1999; Toft et al., 2002). MacIntyre et al. (2001) found IL-6, neutrophils, and myosin heavy chain fragments levels were elevated up to 6 hours following 300 eccentric repetitions of quadriceps muscle on an isokinetic dynamometer. Based on these changes, and complaints of muscle soreness, it was suggested a relationship existed between damage to the contractile proteins and inflammation. In another study however (Ostrowski et al., 2000), no association was found between peak concentrations of IL-6 and creatine kinase (CK), after a 40km marathon. Furthermore, we found similar elevations in IL-6 and neutrophils after

concentric cycling without complaints of pain or muscle soreness. An increased level of IL-6 seems more related to systemic stress than inflammation.

Stress hormones are released in response to exercise stress without the presence of tissue damage and inflammation (Chrousos, 1995). Suzuki et al. (1999) found 90 min of cycling (90 W), 3 days in a row, increased circulating stress hormones and cytokines, which were positively correlated with peripheral neutrophilia. These changes were well in advance of increases in serum CK and myoglobin (Mb). These changes have also been found in stressful activities that do not involve exercise. Jansky et al. (1996) found the use of stress-inducing cold-water immersions (14 °C for 1 h), over a six-week period (3x/week), elevated blood concentrations of stress hormones and cytokines (IL-2). Zhu et al. (1996) also found boosted secretion of IL-1, TNFα and IL-6 after 5 min of swimming in water at 10°C (2 sessions/day). Although not statistically significant, we found IL-6 concentrations to be on average 30% higher in the CR group than AR and Rest. The increased mobilization of IL-6 in our study is probably a function of the cold stress itself. Subjectively, severe cold is one of the more unpleasant body stressors (Shephard and Shek, 1998c). If cold exposure is sufficient to stress the individual, there is likely an increased production of both catecholamines and cortisol (Shephard and Shek, 1998c). Therefore, this release of hormones from CR during recovery may contribute to the release of cytokines during or after intense exercise.

3.5.5 Immune System Response

Researchers believe the immune system is enhanced by moderate and regular physical exercise (Sharp and Koutedakis, 1992; Shephard and Shek, 1998a, 1998b; MacKinnon, 1999; Pedersen et al., 2000). Intense activity, however, causes inhibition of several functions of the immune system in the recovery phase following exercise. In contrast to moderate exercise, high intensity training is followed by a period of immunosuppression during which there is increased opportunity for pathogens and illness (Pedersen et al., 1998; Nieman, 2000). During exercise, Nieman et al. (1994) found that high intensity exercise (>75% VO_{2peak}) was associated with large increases in circulating neutrophils and plasma cortisol, and greater falls in lymphocytes compared with the same duration of moderate intensity exercise. In the present study, all immune markers followed a similar pattern. Neutrophils increased during exercise for all groups and continued to increase an average of 114%, from resting levels, in the hour after cessation of activity. Lymphocytes increased on average 83% during the exercise bouts but fell 27% lower than resting levels in the post-exercise period. Despite differences in exercise mode, intensity and duration, many different studies have replicated these findings (Nieman et al., 1994; Robson et al., 1999; Yamada et al., 2002; Timmons et al., 2004). The difference, however, is that few have studied the effect of recovery interventions on leukocyte activity.

Although white blood cell counts (leukocytes) increase after high intensity/duration exercise, during the 15-60 min period following they tend to fall significantly, often below resting levels (MacKinnon, 1999). This decrease in leukocytes may be prevented

by recovery interventions. In two separate studies by Wigernaes et al. (2000; 2001) active recovery (15 min @ 50% VO_{2max}) during the first 15 minutes after high intensity (80% VO_{2max}) and moderate duration exercise (60 min), counteracted the post-exercise fall in leukocytes as compared to rest recovery. In our study, leukocyte levels after AR (10 min of cycling @ 50 W) were not significantly different from Rest. Interesting to note, however, is that the leukocyte levels after 1 h post exercise in the Wigernaes study (2001) were similar for both AR and Rest groups. This finding closely reflects that found in our study. Unfortunately, there was no blood sampling in our study for the time interval 0-60 min post final bout of exercise. Samples taken after AR and Rest during exercise, however, were not significantly different. In contrast, leukocytes and their subsets were higher with the use of cold therapy (CR) for recovery as compared to AR and Rest.

Although all groups in this study followed a similar trend for immune markers during and after high intensity exercise, only the CR group demonstrated a significant difference. With neutrophils, although increasing an average of 114 % in all groups post exercise (from resting levels), the CR group experienced the greatest increase (143%) compared to the AR and Rest groups (100%). For the lymphocyte subset, the CR group had the largest increase during exercise but the greatest swing post exercise of 66% vs. 56% and 57% from mid-exercise levels. Despite the decrease in lymphocytes, there was an overall 5% increase in leukocytes with CR and a 1-4% decrease in AR and Rest respectively. Jansky et al. (1996) found that a single cold-water immersion (14°C for 1 h) created a small but significant increase in plasma monocytes and lymphocytes, and

concluded it was the stress-inducing effect of cold-water immersion that activated the immune system. This study, however, failed to look at the effect of cold immersion with exercise. Brenner et al. (1999) examined the immunological responses to cold exposure with and without the pre-treatment effect of exercise. Subsequent cold exposures induced a leukocytosis with increases in neutrophils and lymphocytes. Pretreatment with exercise further augmented this response. It was concluded that acute cold exposure had an immunoenhancing effect, and pretreatment exercise enhanced this response. If cold exposure is sufficiently severe to stress the individual, there is also an increased activation of the endocrine system and release of catecholamines and cortisol. Vigorous exercise has also been found to increase secretion of these endocrine markers (Shephard and Shek, 1998c; Brenner et al., 1999), and an interaction between the two stimuli is likely to have further impact. Catecholamines have been found to stimulate the rapid demargination of lymphocytes, and increased cortisol concentrations encourage the increased migration of neutrophils from bone marrow into circulation (Shephard and Shek, 1998c). In theory, therefore, the combination of cold exposure and exercise could alter the "mix" of stimuli resulting in alterations in immune activity. The biological significance of these changes on health, however, remains to be determined.

3.5.6 Psychological Changes

There is strong support for the existence of acute mood benefits from a single bout of exercise. In some circumstances, however, intense training or over-reaching may result in negative psychological outcomes (Yeung, 1996). Little is known about the

specific factors that contribute to these mood effects but parameters such as duration, intensity and mode may play a significant role. Hormonal influences associated with over-training cannot be discounted. Limited attention, however, has been given to the mechanisms by which recovery interventions influence psychological well-being and physical performance. In the present study, we studied the effect of recovery interventions on preparedness for exercise (PREP), perceived exertion (RPE) and perceptions of pain (VAS). Over the successive bouts of intense exercise, scores for VAS and RPE increased, regardless of intervention. Despite this fact, however, ratings for perceived exertion and pain were consistently lower, and preparedness for exercise higher, in the CR group. Hudson et al. (1999) also found ratings of perceived exertion to be lower after active recovery in water (running @ 30% VO_{2max} in a 33 °C pool). In another study (Stacey et al., 2004), despite the fact that recovery interventions had no effect on physical performance, the use of contrast baths created the perception that subjects were better prepared for subsequent bouts of exercise. It was believed the use of alternating hot and cold hydrotherapy created an analgesic effect. Although Myrer et al. (1994) found no physiological effect of contrast therapy on intramuscular tissue 1 cm below the skin, they did support an influence on the superficial layer of fat and skin. At lower temperatures, the rate of firing of pain and temperature sensory receptors located in the skin – the superficial tissue most affected by cold therapy- is diminished, thus reducing the sensation of pain (Bugaj, 1975; Meeusen and Lievens, 1986; Knight, 1995; Enwemeka et al. 2002). With less feeling of pain and heightened psychological well-being athletes may feel greater self-efficacy when commencing subsequent bouts of

exercise. Little is known, however, about the specific factors that may contribute to these psychological effects. In all likelihood, both physiological and psychological factors are involved in the psychological disturbance associated with over-reaching training, but the use of CR provides a greater perception of recovery.

3.6 CONCLUSION

The main finding from this study is that the use of various commonly used recovery interventions, Rest, Active recovery (AR), and Cryotherapy (CR), did not affect the decline in physical performance during repeated bouts of intense exercise. Although active recovery appeared more effective in reducing blood lactate during exercise, [La] for all groups returned to normal levels one hour following the final intervention. Finally, the use of cryotherapy was more effective than active or passive recovery in perturbating blood immune markers (neutrophils and lymphocytes). However, the exact nature and cause of the complex phenomenon of exercise immunosuppression and recovery remains obscure. Further research is needed to explore the source and trigger of cytokine release and subsequent alterations in immune activity following intense exercise and training. Despite the limited evidence supporting recovery interventions from a physiological perspective, the use of cryotherapy is effective in creating the perception that subjects are better prepared for subsequent exercise. Simply stated, it is the "feel better" phenomenon associated with cold therapy, which may allow athletes to train harder, and in the long term, improve performance through normal physiological adaptation.

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CHAPTER 4

THE EFFECT OF HYDROTHERAPY ON RECOVERY AND PERFORMANCE DURING HIGH INTENSITY EXERCISE

SUMMARY

4.1 SPRINT INTERVAL TRAINING EFFECTS

In an effort to improve performance many athletes are incorporating higher training volumes and intensities into their training schedules. One of the current trends in physical conditioning for sport is the use of high intensity training (HIT) or sprint interval training (SIT). HIT can be broadly defined as repeated bouts of exercise lasting less than 3 minutes, completed at an intensity that is greater than the anaerobic threshold (Laursen and Jenkins, 2002). Through repeated stress on the physiological systems associated with exercise, adaptations occur which allow for greater peak work capacity. Many have studied this form of training (Linossier et al., 1997; MacDougall et al., 1998; Parra et al., 2000) and reported significant improvements in peak power and mean power, maximal aerobic power, and endurance performance. Burgomaster et al. (2005) demonstrated an increase in muscle oxidative potential and improved endurance capacity during cycling (~80% VO_{2peak}) after 6 sessions of SIT (4-7 Wingate tests/session) performed over 14 days. In our first study, although performance increases were noted for peak power and time trial performance, no differences were found for mean power and VO_{2peak}. Our improvements were also not to the same magnitude as previous studies. This may be due to the fact that we only

trained our subjects for one week and did not allow enough time for recovery between training sessions. Parra et al. (2000) found performance did not improve with SIT unless enough time for recovery was provided between bouts of intense exercise. This highlights the importance of time for recovery as muscle fibres experience fatigue and/or injury when training and rest are not matched.

This state of training stress associated with HIT is sometimes referred to as "over-reaching". With an appropriate period of recovery it is anticipated a "supercompensation" effect will occur, with subsequent positive adaptations. Without adequate recovery, however, this form of training may induce greater tissue stress, muscular fatigue and injury (Laursen and Jenkins, 2002). As a consequence, athletes often experience acute feelings of fatigue and decreases in performance, even after a single intense training session. High intensity exercise, in untrained subjects, has been associated with increased levels of myofibre enzymes in plasma, ultra-structural damage of the muscle fibres and an acute inflammatory response leading to edema, infiltration by inflammatory cells and muscle soreness 24-48 hours following exercise (Newham et al., 1983; Kuipers, 1994). Although we did not measure muscle enzymes for tissue damage directly, subjects in both studies complained of fatigue and muscle soreness after exercise sessions. Besides fatigue and tissue injury, if this imbalance between training and recovery is allowed to persist, further physiological alterations may also result including alterations in endocrine and metabolic function, changes in mood state, decreases in performance capacity and immunosuppression (Halson and Jeukendrup, 2004).

4.2 FATIGUE AND BLOOD LACTATE

During strenuous or high intensity exercise a discrepancy develops between the demand for energy and energy provision from oxidative metabolism. This results in a depletion of phosphocreatine (PCr) and a large accumulation of lactate as the muscle is forced to rely on non-oxidative sources of energy (Bogdanis et al., 1995, 1996; Gupta et al., 1996). Although there are many different variables involved in metabolic fatigue, intracellular acidosis due to elevated levels of lactic acid is the most popular hypothesis to explain muscular fatigue. Elevated concentrations of hydrogen ions (H^+) and inorganic phosphate (Pi) interfere with the excitation-contraction coupling at the muscle fiber level limiting muscle function (Lattier et al., 2004). It is generally accepted that removal of lactic acid from muscles and blood is essential for enhancing recovery and for successful resumption of subsequent exercise. This is particularly important when athletes must compete on more than one occasion in a single day. Light exercise at 40-60% VO_{2max} has been found to reduce blood lactate levels ~50% after only 10 min (Belcastro and Bonen, 1975). In our second study, subjects performed active recovery on a stationary bike for 10 min at 50W (<50% VO_{2peak}). This resulted in a 14% drop in [La] from bout 1 to bout 3 when compared to other recovery interventions. Despite the reduction in [La], however, there was no improvement in performance. Therefore, active recovery following repeated bouts of high intensity exercise is more effective at decreasing [La] as compared to passive recovery or cryotherapy. This recovery, however, is only evident in the recovery periods between and immediately after training, as 1 hour post exercise all [La], regardless of recovery intervention, returns to resting

levels. Perhaps the benefits of recovery interventions lie beyond muscular fatigue due to intracellular acidosis.

4.3 CYTOKINE RESPONSE TO HIGH INTENSITY TRAINING

The local response to infection or injury is the production of cytokines that are released at the site of inflammation or tissue stress. Recent studies have demonstrated that high intensity exercise is also a powerful inducer of elevated cytokine concentrations (Espersen et al., 1990; Suzuki et al., 1999; Ostrowski et al., 1998, 2000). This form of strenuous exercise induces an increase in the proinflammatory cytokines TNFα and IL-1β and a dramatic increase in the inflammation responsive cytokine IL-6 (Pedersen, 2000). IL-6 levels have been known to increase up to 100-fold immediately after exercise, generally peaking after 1-1.5 hours (Suzuki et al., 1999; Yamada et al., 2002). Although increased levels of circulating cytokines have predominantly been described after exercise involving an eccentric component (Northoff et al., 1994), concentric exercise also induces cytokine production (Ullum et al.,1994; Weinstock et al.,1997; Nieman et al., 1998, 2001). The results of our study confirm the existence of a relationship between plasma IL-6 concentration and exercise intensity. After 2 bouts of intense exercise serum IL-6 increased in all groups by an average of 41% from resting levels. These concentrations increased another 60% in the 1h post exercise suggesting cycling at a high intensity and short duration is a factor in determining the increase in plasma concentrations of IL-6. The actual mechanisms responsible for the release of cytokines during and after exercise, however, are not fully understood.

It has been suggested that the release of pro-inflammatory cytokines represents an inflammatory reaction, presumably initiated by damage to skeletal muscle (Bruunsgaard et al., 1997; Toft et al. 2002). However a recent study (Ostrowski et al. 2000) found no association between peak concentrations of IL-6 and creatine kinase (CK), an indicator of tissue damage, after a 40 km marathon. Given the fact that IL-6, more than any other cytokine, is produced in large amounts after HIT and that it has growth factor abilities, it is likely that IL-6 also plays a beneficial role. Although it is generally regarded as a pro-inflammatory cytokine, it may play a contrary role in preventing further damage and activating the healing/repair process. Some of IL-6's biological activities include stimulation of the endocrine system with the subsequent release of cortisol, a general anti-inflammatory hormone, and glucocorticoids that further inhibit the inflammatory process (Moldoveanu et al., 2001). Il-6, in association with other anti-inflammatory cytokines (IL-4, IL-10), also attenuates production of the inflammatory cytokines TNFα and IL-1β (Moldoveanu et al., 2001). Finally, IL-6 facilitates the influx of phagocytic cells that participate in the clearance of damaged tissue, facilitate healing, and provoke higher immune responses (MacKinnon, 1999). Therefore, although IL-6 concentrations increase in response to the stresses of high intensity exercise and generally function as intercellular messengers to stimulate the inflammatory process, they can also evoke particular biological activities that promote healing and recovery.

4.4 EXERCISE INTENSITY AND IMMUNOSUPPRESSION

There is a growing belief that very strenuous physical activity can cause substantial subclinical injury, initiating an excessive inflammatory reaction and immunosuppression (Pedersen et al., 1998; Shephard and Shek, 1998b). The mechanisms underlying the exercise-induced immune changes are multi-factorial and support the existence of pathways between the immune, nervous and endocrine systems. Tissue stress from high intensity exercise induces a rapid but complex sequence of immune reactions. The immediate acute response leads to the release of various inflammatory mediators, such as cytokines, which in turn regulate the rapid infiltration of neutrophils and monocytes to the area of injury (Shephard and Shek, 1998b; Nieman et al., 2001). The extent of phagocytic infiltration and cytokine activation is proportional to the extent of the local tissue stress (Shephard and Shek, 1998b). Concentrations of endocrine hormones also increase in response to acute exercise stress. Adrenaline and to a lesser extent noradrenaline are believed to be responsible for the acute rise in lymphocyte counts whereas catecholamines and growth hormones in combination are responsible for the increase in neutrophil activity (Pedersen et al., 1998). Furthermore, the increased cortisol concentrations are thought to be responsible for the elevated neutrophil concentrations and depressed lymphocyte concentrations found post exercise (Pedersen et al., 1998). It is during this period of immunosuppression post exercise that there is believed to be an "open window" of opportunity for pathogens and illness (Pedersen et al., 1998).

In our second study, all immune markers followed a similar pattern of change. Neutrophils increased during exercise for all groups and continued to increase an average

of 114% from resting levels, in the one-hour after cessation of activity. Lymphocytes increased on average 83% during the exercise bouts but fell 27% lower than resting levels in the post-exercise period. A relationship between high intensity training and acute immunosuppression appears evident. The biological significance of this suppression, however, has not been established. In a review of the epidemiology of exercise immunology, Shephard (2000) failed to find a cause and effect relationship between exercise-induced immunosuppression and illness. In vitro alterations in immune function, therefore, do not necessitate clinical relevance but they do stress the importance of sufficient recovery.

4.5 THERAPEUTIC INTERVENTIONS FOR RECOVERY

When intense exercise and the associated disturbance in homeostasis are not followed by adequate recovery, athletes may experience excess muscular fatigue, soft tissue injury, and/or immune suppression (Espersen et al., 1990; Bruunsgaard et al., 1997; Yamada et al., 2002). Interestingly enough, all these adverse effects of exercise can be reversed with adequate rest and recovery. With short seasons and intense competition schedules, however, the time for rest and recovery is often overlooked. Athletes, therefore, are using many different therapeutic interventions for recovery between intense bouts of exercise. As reflected in the literature, there are few studies validating their benefits (Knight, 1995; Fu et al., 1997; Eston and Peters, 1999,). We tested the hypothesis that hydrotherapy (CR and CT) used for recovery between bouts of high intensity cycle training would have a positive effect on physical performance and blood

markers for exercise stress (IL-6, leukocytes - neutrophils, lymphocytes). Despite the effect on performance, however, we proposed that only active recovery would have an effect on blood lactate. Psychological scores for pain, physical exertion and preparedness for exercise would also improve with hydrotherapy.

In our contrast therapy study, although performance increases were noted for peak power and time trial performance, no difference was found between the treatment and control groups. In the only study found that assessed the physiological effects of contrast baths, Myrer et al. (1994) found the alternating use of hot and cold ineffective in inducing the temperature and thus vascular changes necessary for "physiological recovery". Similarly, in our second study, no specific recovery interventions (CR, AR, Rest) were found to impact on physical performance. Time trial performances were on average 8% and 14% slower during bouts 2 and 3 respectively, regardless of the intervention. The lack of improvement in performance was not expected in our second study, as other studies have found marked increases in both anaerobic power and endurance performance after cold therapy (Verducci, 1997, 2000; Fowles et al., 2003; Rogers and Albrechtsen, 2003). This variation may be due to the fact that recovery times in these studies were significantly longer than those used in our study, thus allowing for increased metabolic recovery. Despite this difference, subjects still found hydrotherapy (contrast and cold therapy) to be the preferred recovery intervention between bouts of intense exercise in both of our studies.

There is strong support for the existence of acute mood benefits from a single bout of exercise. In some circumstances, however, intense training or over-reaching may

result in negative psychological outcomes (Yeung, 1996). In our cryotherapy study (CR), we analyzed the effect of three different recovery interventions on preparedness for exercise (PREP), ratings of perceived exertion (RPE) and perceptions of pain (VAS). As expected, scores for VAS and RPE were progressively worse over successive bouts of intense exercise, regardless of intervention. Despite a lack of statistical significance, however, ratings for perceived exertion and pain were consistently lower in the CR group. Similarly, ratings of preparedness for exercise were higher in the CR group. Not unlike cryotherapy, the use of contrast baths (CT) in our first study also created the perception that subjects were better prepared for exercise. Over the course of training, POMS scores for vigor improved with CT yet worsened with the CON. Similarly, the CT group reported feeling more revitalized and ready for exercise after treatment than the CON group. Perhaps the use of cryotherapy and alternating hot/cold hydrotherapy provides an analgesic effect? Although Myrer et al. (1994) found no physiological effect of contrast therapy on intramuscular tissue 1cm below the skin, they did support an influence on the superficial layer of fat and skin. At lower temperatures, the rate of firing of pain and temperature sensory receptors located in the skin – the superficial tissue most affected by cold therapy- is diminished, thus reducing the sensation of pain (Bugaj, 1975; Meeusen and Lievens, 1986; Knight, 1995; Enwemeka et al. 2002). Similar findings have also been reported with the application of heat (Knight, 1995). With less feeling of pain and heightened psychological well-being athletes may feel greater self-efficacy when commencing subsequent bouts of exercise.

Another interesting finding in our cryotherapy study was the effect of cold baths on the immune response. Although all treatment groups in this study produced a similar trend with immune markers during and after high intensity exercise, only the CR group demonstrated a significant difference. Neutrophils increased 143% after exercise in the CR group as compared to 100% in the AR and Rest groups. The CR group also had the largest increase in lymphocytes during exercise (4.1±0.4 versus 3.6±0.6, 10^9 cells/L) and the greatest swing post exercise, decreasing 66% vs. 57% from mid-exercise levels. In a related study, Jansky et al. (1996) found that a single cold-water immersion (14 °C for 1h) created a small but significant increase in plasma monocytes and lymphocytes, and surmised the stress-inducing effect of cold-water immersion was responsible for the immune system activation. This study, however, failed to look at the effect of cold immersion with exercise. Brenner et al. (1999) examined the immunological responses to cold exposure with and without the pre-treatment effect of exercise. Subsequent cold exposures induced a leukocytosis with increases in neutrophils and lymphocytes. Pretreatment with exercise further augmented this response. It was concluded that acute cold exposure had an immunoenhancing effect, and pretreatment exercise enhanced this response. The clinical significance of these findings, however, requires further evaluation.

4.6 FUTURE DIRECTIONS

Exercise immunology is a complex subject where results can be potentially influenced by many biological and technical variables, some unrelated to exercise.

Furthermore, the effects of exercise (intensity/volume) on immune variables may not be explainable due to a lack of fundamental knowledge. For instance, the exact mechanisms underlying the apparent activation of leukocytes (neutrophils/lymphocytes) are questionable. Stress hormones, Cytokines (IL-6, IL-1β, and TNFα), and hemodynamic changes have all been implicated but are relatively inconsistent and unexplained (Pedersen et al., 1998; Shephard and Shek, 1998b; MacKinnon, 1999; Nieman, 2001). As such, research guidelines need to be standardized to control extraneous variables, including subject selection protocols (age, gender, diet, health), specimen collection times (circadian rhythm), and blood collection protocols. The significance of the immune system findings should also be re-evaluated, as it is still uncertain whether exercise-induced changes are related to alterations in immune function (Shephard, 2000). The roles of cytokines in inflammation and muscle/tissue repair are also unclear (Bruunsgaard et al., 1997; Ostrowski et al., 2000; Toft et al. 2002). Are the exercise-increased levels of IL-6 helpful or harmful? Laboratory protocols for the analysis of cytokines need to be improved (sensitivity and specificity) with more pro- and anti-inflammatory cytokines measured in a single assay. With a greater variety of cytokines available, trends during exercise and recovery may become more evident.

From the results of these studies it also seems obvious more research is necessary to explain the benefits of hydrotherapy (CT and CR) as a recovery intervention during high intensity exercise. Although there is no evidence of performance changes, subjects still perceive CT and CR to be more effective than Rest or AR. With limited evidence of metabolic changes, more emphasis should be placed on neuromuscular changes. There is

some evidence of sensory changes with cold therapy (Bugaj, 1975; Meeusen and Lievens, 1986; Knight, 1995; Enwemeka et al. 2002,) but the conclusions are still speculative. Analgesia and pain relief are believed to be the results of local cooling on most components of the neuromuscular complex, including the motor neuron pool, muscle spindles and their afferents, skin afferents, myoneural junctions, and extrafusal fibers (Meeusen and Lievens, 1986). Research focusing on the effects of CT and CR on intramuscular temperature, intra- and extra- muscular blood flow, and sensory nerve conduction may clarify the analgesia question. Finally, there is a greater need for more clinical studies. Most of the research to date, including the two studies in this thesis, has focused on performance over the short term (Parra et al., 2000; Brenner et al., 1999; Suzuki et al., 1999; Burgomaster et al., 2005). Clinical trials are necessary to test the use of recovery interventions, including CR and CT, during long-term training. If the benefits are more related to preparedness for exercise, perceived exertion and sensations of pain, training adaptations related to performance and the immune system may be more evident after a full training cycle.

4.7 CONCLUSION

An essential component of the sprint-training model is that repeated bouts of high intensity physical exercise create a disturbance in cellular homeostasis. This disturbance then acts as a stimulus to initiate physiological responses that restore homeostasis and induce training adaptation. Without adequate rest and recovery after HIT, however, those training adaptations will never be realized. The recovery process, therefore, is essential to determining subsequent athletic performance. The main finding from these two studies is that the use of various commonly used recovery interventions – active recovery (AR), cryotherapy (CR) and contrast baths (CT) – are no more effective than rest in improving physical performance during repeated bouts/sessions of intense exercise. From a metabolic perspective, although AR appeared more effective in reducing blood lactate during intense exercise, no recovery intervention was found to be more effective one hour following the final intervention. Another key finding is that the use of contrast and cold therapy created the perception that subjects were better prepared to exercise. With regards to the mechanisms proposed to mediate these effects, the role of self-efficacy and analgesia from the hot and cold cannot be discounted. In all likelihood both psychological and physiological factors are involved. Finally, although maximal exercise is strenuous on both the endocrine and immune systems, the use of cryotherapy seems to have a stimulating effect. The exact nature and effect of this phenomenon on exercise immunosuppression and recovery, however, remains obscure. The "feel good" phenomenon associated with cold therapy, therefore, should not be discounted. With increased feelings of self-efficacy, lower ratings of perceived exertion and potential

immunoenhancing and tissue healing effects, this form of recovery intervention may enable athletes to train longer and harder with less detrimental effects. Further studies are required to assess the long-term effects (>3 weeks) of this intervention in both high-volume and high-intensity training programs. Perhaps this "practice" will eventually become "principle".

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APPENDIX A

REB Approval Form

STUDENT REVIEW FORM

RECEIVED JUN 2 - 2003

(Please type)

This form along with the "Application for Review by Research Ethics Board" form should be submitted 4 weeks before commencing data collection, and must be approved and returned to student or research tutor before data collection begins.

REB # 03-211(5)

Name of Student/Researcher: **Doug Stacey BHScPT** Phone Number: **905-527-5454**

Name of Supervisor/Research Tutor: **Martin Gibala Ph.D** Phone Number: **905-525-9140 extension 23591**

Signature of Supervisor _____

Graduate Program **Masters of Science in Kinesiology**

Title of Study: **The Effects of Hydrotherapy on Recovery and Performance During high Intensity Training.**

APPROVED

Proposed Start of Data Collection **June/July 2003**

DATE May 27/03

Review Form Route

1. Register Application with C.S.D., 3N8 — Registered — 9 Date May 28/03
   Signed Dorothy Agnew
2. M. Pierrynowski/M. Townsend — Approved — 9 Date 30 May 2003
   Signed M Pierrynowski
3. Chair, Research Ethics Board — Approved — 9 Date June 5/03.
   Signed J Crowe/sm
4. Submitted to REB for information — Date May 30/03.

A copy of the form must be sent to C.S.D., 3N8, once completed.

NOTE: **If a signatory is unable to approve the project, he/she should contact the student or the research tutor as soon as possible to discuss the prohibiting factor(s).**

Should you require assistance in completing the form, please contact: Dorothy Agnew, CSD, X22469 or e-mail agnewdo@fhs.mcmaster.ca

S:\CSD Forms\student.wpd (Revised January 2002)

APPENDIX B

Subject Information and Consent Forms

McMaster University

Exercise Metabolism Research Group
Department of Kinesiology EMRG Laboratory:
Ivor Wynne Centre, Room A103
1280 Main Street West
Hamilton, Ontario, Canada
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Phone: 905-525-9140
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EXERCISE METABOLISM RESEARCH GROUP (EMRG)
DEPARTMENT OF KINESIOLOGY, MCMASTER UNIVERSITY

CONSENT TO PARTICIPATE IN RESEARCH

You are asked to participate in a research study being conducted by the investigators listed below at McMaster University, Hamilton, Ontario. Prior to your participation, you are asked to read and complete this form and the accompanying form, which outline the purpose, procedures, and risks associated with the study, and also provide other essential information regarding your rights and responsibilities as a subject. The accompanying form is the "Subject Screening Questionnaire." All experimental testing and training will be conducted in the EMRG Laboratory, Ivor Wynne Centre, Room A103.

LIST OF PRIMARY INVESTIGATORS
Name Campus Address Daytime Phone Number
Dr. Martin Gibala Kinesiology, AB122, 905-525-9140 ext. 23591
Dr. Kathleen Martin Ginis, Kinesiology A 103, 905-525-9140 ext. 23574
Mr. Doug Stacey BHScPT 905-527-5454

PROJECT TITLE

"The Effects of Hydrotherapy on Recovery and Performance During High Intensity Training"

PURPOSE OF THE STUDY

The use of hydrotherapy is already well in use in the sporting community yet there are no scientific studies available to validate its use. It is anticipated this study will shed some light on its benefits or lack thereof. The primary purpose of the proposed investigation is to examine the effects of contrast baths on performance during high intensity sprint training. We will also study the potential psychological effects of this intervention. We will assess performance through aerobic ($VO2_{peak}$), anaerobic (peak power, mean power) and aerobic endurance capacity (challenge ride) measurements. Pre-exercise questionnaires (Profile of Mood States, Preparedness for Exercise, Self-Efficacy) will be used to measure thoughts and feelings of the exercise/training.

DESCRIPTION OF TESTING AND EXPERIMENTAL PROCEDURES

Following routine medical screening and several familiarization visits to the laboratory (in order to become oriented with testing procedures and equipment), you will be required to make a total of 11 visits to the laboratory over a period of approximately 2 weeks. Specifically, the study will consist of 3 pre-training (baseline) experimental exercise tests, 5 training sessions and 5 hydrotherapy sessions (depending on which training group you are assigned to), and 3 tests during the post-training phase (see below for summary of complete schedule). You will also be required to fill out 3 questionnaires prior to commencing each training session and 3 questionnaires prior to the first test post-training.

OVERVIEW: LABORATORY VISITS, TESTING AND TRAINING PHASES:

(A) PRE-TRAINING PHASE: 3 *tests will be conducted over 3 days:*

1. VO_2peakTest (~ 1 hour)
2. Repeated Wingate Test (~ 35 min)
3. Challenge ride (~ 15-60 min)

(B) TRAINING PHASE: *5 lab visits (60 min each) over 5 days (Monday – Friday)*

(C) POST-TRAINING PHASE: *3 tests will be conducted over 3 days:*

1. Challenge ride (~ 15-60 min)
2. Repeated Wingate Test (~ 30 min)
3. VO_2peakTest (~ 1 hour)

VO_2peak Test. This test involves cycling on a stationary bike (cycle ergometer) at progressively higher workloads while the amount of oxygen taken up by your body is determined from a mouthpiece connected to a gas analyzer. You will perform a VO_2peak test prior to and following the sprint-training protocol.

Wingate Test: Single and Repeated. A Wingate test is a 30-second "all out" sprint exercise test on a cycle ergometer and is used to determine your maximal power output. You will perform repeated Wingate tests prior to and following the sprint-training protocol, 4 x 30 sec bouts of maximal cycling will be performed with 4 min of recovery in between each bout.

Exercise Performance Trial (Challenge Ride) - You will perform a simulated "time trial" race, whereby you will be instructed to complete a standardized bout of work (250kJ) in as fast a time as possible. Pace will be self-selected throughout the test, which should last between 15 and 30 minutes depending on your fitness level and motivation. You will perform this time trial prior to and following the sprint-training protocol.

Pre-Exercise Bout Questionnaires. Both groups will complete three mini questionnaires (Profile of Mood States [POMS], Self-Efficacy, and Preparedness for Exercise Scale). The questionnaires will focus on mood, or affective state, feelings of well-being and preparedness for exercise.

Training Protocol. The training protocol will consist of 5 days of sprint interval training. You will be randomly assigned to one of two training groups. Both groups will be subjected to the same training protocol but one group will follow a 2:3 (cold: hot) contrast bath protocol after each training bout (McMaster Sports Medicine Clinic), while the other will relax in a quiet area for the same amount of time. The sprint training program will begin with four intervals per session at the start of the week and progress to six intervals per session by the end of

the week. Recovery intervals will be four minutes in duration. Training will be performed on a cycle ergometer whereby one interval will be the equivalent to one Wingate test (30 sec all-out sprint cycle test against a constant resistance). The resistance will be set at 7.5% of your body mass throughout.

Post-Testing Procedures Following the training protocol, you will perform a series of three post-training tests (i.e., VO_2peak test, Repeated Wingate test, Performance Trial). These tests will be identical in all respects to the first series of tests described above.

DESCRIPTION OF POTENTIAL RISKS AND DISCOMFORTS

The potential risks of the proposed study are physical and relate to the sprint training and the contrast baths used for recovery. Most of the potential risks for contrast baths relate to the cryotherapy aspect of the baths. Although there are some conditions that preclude cryotherapy (eg. Rheumatoid diseases, coronary artery disease, and open sore) it is doubtful they will be present in our testing population. During the actual treatment session you may experience feelings of aching, burning, "pins and needles" or numbness with the cold bath. These may change to similar feelings of aching, burning and warmth with the transition to the hot bath. All sensations will be temporary and should resolve by the end of the treatment session.

The sprint training sessions will be brief but intense in nature. In addition to the stress associated with vigorous physical activity, you may experience severe local muscle fatigue/ache in your thigh muscles, nausea and/or light- headedness upon cessation of exercise. We will provide instruction regarding proper cool-down procedures (e.g. 'active recovery' whereby you continue to cycle against a light resistance) in order to try and minimize these symptoms. Some local muscle discomfort/fatigue may also be present throughout the training but this should not limit activities of daily living.

REMUNERATION

You will receive an honorarium of $100.00 in order to compensate for your time commitment and effort. Remuneration is normally provided within one week following completion of the study.

PROVISION OF CONFIDENTIALITY

Any information that is obtained in connection with this study will remain confidential, and appropriate measures will be taken by all investigators to ensure privacy. The results from this study will be used for educational purposes and may be published in scientific journals, presented at scientific meetings or disseminated using other appropriate methods. Regardless of presentation format, subjects will not be identified by name, and your personal data will only be identified by a code number. Upon completion of the study, you will have access to your own data and the group data for your own interest.

PARTICIPATION AND WITHDRAWAL

You can choose whether to be in this study or not. If you volunteer to be in this study, you may withdraw at any time without consequences of any kind. You may exercise the option of removing your data from the study. You may also refuse to answer any questions, which you do not want to and still remain in the study. The investigators also reserve the right to withdraw you from this research project if circumstances arise which

warrant doing so. Should you withdraw from the study prior to its completion; a partial honorarium payment will be made based on the relative proportion of the study, which was completed.

RIGHTS OF RESEARCH PARTICIPANTS

You may withdraw your consent at any time and discontinue participation without penalty. You are not waiving any legal claims, rights or remedies because of your participation in this research study. The nature of the exercise stresses and treatment procedures to be employed in this study have been approved by the Hamilton Health Sciences Corporation / McMaster University Research Ethics Board.

If you have any questions regarding your rights as a research participant you may contact the Hamilton Health Sciences Patient Relations Specialist at 905-521-2100, Ext. 75240.

SIGNATURE OF RESEARCH PARTICIPANT/LEGAL REPRESENTATIVE

I have read and understand the information provided for the study as described herein and in the accompanying form entitled "Subject Screening Questionnaire." My questions have been answered to my satisfaction, and I agree to participate in this study. I have been given a signed copy of this form.

_____

Name of Participant

_____

Name of Legal Representative (if applicable)

_____ _____

Signature of Participant or Legal Representative Date

SIGNATURE OF INVESTIGATOR

In my judgment, the participant is voluntarily and knowingly giving informed consent and possesses the legal capacity to give informed consent to participate in this research study.

_____ _____

Signature of Investigator Date

APPENDIX C

Raw Data

TESTING THE WATERS: ARE THE EFFECTS OF HYDROTHERAPY MORE PSYCHOLOGICAL THAN PHYSIOLOGICAL?

Challenge ride time trial (seconds)

Challenge Time (sec)	*control*	*group*	*treatment*	*group*
	pre	post	pre	post
SO1	821	859		
S10	1083	1074		
S04	1269	1105		
S06	1168	1063		
S08	1098	1126		
S11	1338	1302		
S03			1126	916
S07			848	793
S05			923	902
S02			1076	1003
S09			998	992
S12			1039	1061
Means	1130	1088	1002	945
SD	180	142	102	95
SEM	74	58	42	39

VO_{2peak} ($ml.kg^{-1}.min^{-1}$)

VO_{2peak} $ml.kg.min^{-1}$	*control*	*group*	*treatment*	*group*
	pre	post	pre	post
SO1	48.2	47.9		
S10	51.3	51.4		
S04	44.0	44.5		
S06	45.2	46.4		
S08	45.4	49.5		
S11	46.7	42.1		
S03			46.5	47.5
S07			51.5	51.0
S05			45.2	44.3
S02			43.4	45.2
S09			45.7	47.1
S12			40.1	44.1
Means	46.8	47.0	45.4	46.5
SD	2.6	3.4	3.8	2.6
SEM	1.1	1.4	1.5	1.1

Peak Anaerobic Power (W)

Peak Power (W)	*control group*		*treatment group*	
	pre	post	pre	post
SO1	972	1345		
S10	1126	1124		
S08	1279	1313		
S04	1199	1358		
S06	1520	1535		
S11	1223	1384		
S03			1112	1281
S07			1600	1503
S05			1227	1340
S02			1235	1179
S09			975	1307
S12			1140	1247
Means	1219.8	1343.2	1214.8	1309.5
SD	181.3	132.4	211.0	109.7
SEM	74	54	86	45

Mean Anaerobic Power (W)

Mean Power (W)	*control group*		*treatment group*	
	pre	post	pre	post
SO1	699.9	749.9		
S10	674.4	673.9		
S08	700.2	724.3		
S04	736.9	692.4		
S06	819.7	828.2		
S11	713.4	770.9		
S03			752.8	809.2
S07			882.1	884
S05			686.2	741.9
S02			797.7	747.2
S09			619.7	709
S12			760.2	773.6
Means	724.1	739.9	749.8	777.5
SD	51.1	56.1	90.4	62.0
SEM	21	23	37	25

Psychological Data (mean±SEM)

		Treatment	*Group*	*Control*	*Group*
		Pre	Post	Pre	Post
POMS	Vigor	12.8±0.1	15.7±0.1	16.0±0.1	13.3±0.1
	Depression	4.7±0.5	1.8±0.5	4.8±0.5	0.2±0.5
	Fatigue	9.6±0.6	5.0±0.6	9.0±0.6	3.2±0.6
	Tension	9.7±0.5	4.2±0.5	9.3±0.5	5.3±0.5
Self-Efficacy	Cycling	72.3±2%	81.7±2%	66.4±2%	82.9±2%
	Stairs	88.7±2%	91.3±2%	93.3±2%	98.3±2%
	Strength	81.7±2%	89.6±2%	96.7±2%	99.6±2%
	Jumping	73.8±4%	80.7±4%	87.7±4%	91.2±4%
	Running	68.7±2%	75.3±2%	79.2±2%	83±2%
Preparedness	Ex. Ability	7.7±0.2	7.4±0.2	9.3±0.2	8.3±0.2
	Leg feeling	3.7±0.3	1.9±0.3	3.5±0.3	1.9±0.3
	Leg Pain	2.5±0.2	1.6±0.2	1±0.2	1±0.2
	ADL's	1±0.2	0.7±0.2	0.2±0.2	0.1±0.2
	Treatment effect	7.5±0.4	9.0±0.4	4.6±0.4	4.7±0.4

APPENDIX D

Statistical Tables

REPEATED MEASURE ANOVA'S

BONFERRONI PAIRWISE COMPARISONS

TESTING THE WATERS: ARE THE EFFECTS OF HYDROTHERAPY MORE PSYCHOLOGICAL THAN PHYSIOLOGICAL?

1. Challenge Ride Time (sec)
Two-Factor Repeated Measure ANOVA (Pre/Post x Group)

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	163462.234(a)	2	81731.117	16.691	.001
Intercept	15021.889	1	15021.889	3.068	.114
TIME	101541.901	1	101541.901	20.737	.001
GROUP	7606.662	1	7606.662	1.553	.244
Error	44070.432	9	4896.715		
Total	12602734.000	12			
Corrected Total	207532.667	11			

a R Squared = .788 (Adjusted R Squared = .740)

Pairwise Comparisons

Measure: **TIME**

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.(a)	95% Confidence Interval for Difference(a)	
					Lower Bound	Upper Bound
1	2	49.250	23.288	.061	-2.640	101.140
2	1	-49.250	23.288	.061	-101.140	2.640

Based on estimated marginal means

a Adjustment for multiple comparisons: Bonferroni.

2. Peak Anaerobic Power (W)
Two-Factor Repeated Measure ANOVA (Pre/Post x Group)

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	66469.754(a)	2	33234.877	3.532	.074
Intercept	178069.679	1	178069.679	18.923	.002
TIME	63069.420	1	63069.420	6.702	.029
GROUP	3004.179	1	3004.179	.319	.586
Error	84692.913	9	9410.324		
Total	21261084.000	12			
Corrected Total	151162.667	11			

a R Squared = .440 (Adjusted R Squared = .315)

Pairwise Comparisons

Measure: **TIME**

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.(a)	95% Confidence Interval for Difference(a)	
					Lower Bound	Upper Bound
1	2	-109.000(*)	43.034	.030	-204.885	-13.115
2	1	109.000(*)	43.034	.030	13.115	204.885

Based on estimated marginal means

* The mean difference is significant at the .05 level.

a Adjustment for multiple comparisons: Bonferroni.

3. Mean Anaerobic Power (W)
Two-Factor Repeated Measure ANOVA (Pre/Post x Group)

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	26662.307(a)	2	13331.154	9.582	.006
Intercept	7912.067	1	7912.067	5.687	.041
TIME	22432.300	1	22432.300	16.124	.003
GROUP	1273.080	1	1273.080	.915	.364
Error	12521.242	9	1391.249		
Total	6946843.570	12			
Corrected Total	39183.549	11			

a R Squared = .680 (Adjusted R Squared = .609)

4. VO_{2peak} ($ml.kg^{-1}.min^{-1}$)
Two-Factor Repeated Measure ANOVA (Pre/Post x Group)

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	47.309(a)	2	23.655	4.856	.037
Intercept	12.633	1	12.633	2.593	.142
TIME	46.724	1	46.724	9.592	.013
GROUP	.672	1	.672	.138	.719
Error	43.841	9	4.871		
Total	26303.878	12			
Corrected Total	91.151	11			

a R Squared = .519 (Adjusted R Squared = .412)

5. POMS
Two-Factor Repeated Measure ANOVA (Day x Group)

TENSION

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	Hypothesis	2568.056	1	2568.056	54.344	.000
	Error	472.556	10	47.256(a)		
GROUP	Hypothesis	34.722	1	34.722	.735	.411
	Error	472.556	10	47.256(a)		
ID(GROUP)	Hypothesis	472.556	10	47.256	6.151	.000
	Error	384.111	50	7.682(b)		
DAY	Hypothesis	307.778	5	61.556	8.013	.000
	Error	384.111	50	7.682(b)		
GROUP * DAY	Hypothesis	36.778	5	7.356	.957	.453
	Error	384.111	50	7.682(b)		

a MS(ID(GROUP))
b MS(Error)

DEPRESSION

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	Hypothesis	364.500	1	364.500	5.678	.038
	Error	642.000	10	64.200(a)		
GROUP	Hypothesis	84.500	1	84.500	1.316	.278
	Error	642.000	10	64.200(a)		
ID(GROUP)	Hypothesis	642.000	10	64.200	9.573	.000
	Error	335.333	50	6.707(b)		
DAY	Hypothesis	127.333	5	25.467	3.797	.005
	Error	335.333	50	6.707(b)		
GROUP * DAY	Hypothesis	34.333	5	6.867	1.024	.414
	Error	335.333	50	6.707(b)		

VIGOR

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	Hypothesis	13068.056	1	13068.056	292.278	.000
	Error	447.111	10	44.711(a)		
GROUP	Hypothesis	24.500	1	24.500	.548	.476
	Error	447.111	10	44.711(a)		
ID(GROUP)	Hypothesis	447.111	10	44.711	2.428	.019
	Error	920.889	50	18.418(b)		
DAY	Hypothesis	62.778	5	12.556	.682	.639
	Error	920.889	50	18.418(b)		
GROUP * DAY	Hypothesis	162.667	5	32.533	1.766	.137
	Error	920.889	50	18.418(b)		

a MS(ID(GROUP))
b MS(Error)

FATIGUE

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	Hypothesis	3930.889	1	3930.889	63.254	.000
	Error	621.444	10	62.144(a)		
GROUP	Hypothesis	162.000	1	162.000	2.607	.137
	Error	621.444	10	62.144(a)		
ID(GROUP)	Hypothesis	621.444	10	62.144	3.558	.001
	Error	873.222	50	17.464(b)		
DAY	Hypothesis	199.778	5	39.956	2.288	.060
	Error	873.222	50	17.464(b)		
GROUP * DAY	Hypothesis	74.667	5	14.933	.855	.518
	Error	873.222	50	17.464(b)		

a MS(ID(GROUP))
b MS(Error)

6. Self-Efficacy
Two-Factor Repeated Measure Anova (Day x Group)

CYCLING

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	Hypothesis	371881.253	1	371881.253	307.835	.000
	Error	12080.556	10	1208.056(a)		
GROUP	Hypothesis	67.087	1	67.087	.056	.818
	Error	12080.556	10	1208.056(a)		
ID(GROUP)	Hypothesis	12080.556	10	1208.056	10.590	.000
	Error	5703.715	50	114.074(b)		
DAY	Hypothesis	2287.944	5	457.589	4.011	.004
	Error	5703.715	50	114.074(b)		
GROUP * DAY	Hypothesis	601.069	5	120.214	1.054	.397
	Error	5703.715	50	114.074(b)		

STAIRS

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	Hypothesis	589969.531	1	589969.531	492.432	.000
	Error	11980.729	10	1198.073(a)		
GROUP	Hypothesis	1188.281	1	1188.281	.992	.343
	Error	11980.729	10	1198.073(a)		
ID(GROUP)	Hypothesis	11980.729	10	1198.073	11.685	.000
	Error	5126.563	50	102.531(b)		
DAY	Hypothesis	844.531	5	168.906	1.647	.165
	Error	5126.563	50	102.531(b)		
GROUP * DAY	Hypothesis	159.115	5	31.823	.310	.904
	Error	5126.563	50	102.531(b)		

a MS(ID(GROUP))
b MS(Error)

STRENGTH

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	Hypothesis	604083.681	1	604083.681	955.335	.000
	Error	6323.264	10	632.326(a)		
GROUP	Hypothesis	3901.389	1	3901.389	6.170	.032
	Error	6323.264	10	632.326(a)		
ID(GROUP)	Hypothesis	6323.264	10	632.326	19.510	.000
	Error	1620.486	50	32.410(b)		
DAY	Hypothesis	222.569	5	44.514	1.373	.250
	Error	1620.486	50	32.410(b)		
GROUP * DAY	Hypothesis	111.111	5	22.222	.686	.636
	Error	1620.486	50	32.410(b)		

a MS(ID(GROUP))
b MS(Error)

JUMPING

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	Hypothesis	503205.120	1	503205.120	396.253	.000
	Error	12699.078	10	1269.908(a)		
GROUP	Hypothesis	3269.709	1	3269.709	2.575	.140
	Error	12699.078	10	1269.908(a)		
ID(GROUP)	Hypothesis	12699.078	10	1269.908	30.187	.000
	Error	2103.389	50	42.068(b)		
DAY	Hypothesis	222.393	5	44.479	1.057	.395
	Error	2103.389	50	42.068(b)		
GROUP * DAY	Hypothesis	150.471	5	30.094	.715	.615
	Error	2103.389	50	42.068(b)		

a MS(ID(GROUP))
b MS(Error)

RUNNING

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	Hypothesis	356449.389	1	356449.389	57.693	.000
	Error	61784.097	10	6178.410(a)		
GROUP	Hypothesis	806.681	1	806.681	.131	.725
	Error	61784.097	10	6178.410(a)		
ID(GROUP)	Hypothesis	61784.097	10	6178.410	110.701	.000
	Error	2790.590	50	55.812(b)		
DAY	Hypothesis	959.392	5	191.878	3.438	.010
	Error	2790.590	50	55.812(b)		
GROUP * DAY	Hypothesis	67.726	5	13.545	.243	.942
	Error	2790.590	50	55.812(b)		

a MS(ID(GROUP))
b MS(Error)

7. Preparedness for Exercise
Two-Factor Repeated Measure Anova (Day x Group)

Question 1 – Ability to Exercise

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	Hypothesis	4017.067	1	4017.067	417.122	.000
	Error	96.304	10	9.630(a)		
GROUP	Hypothesis	16.245	1	16.245	1.687	.223
	Error	96.304	10	9.630(a)		
ID(GROUP)	Hypothesis	96.304	10	9.630	5.800	.000
	Error	83.026	50	1.661(b)		
DAY	Hypothesis	26.513	5	5.303	3.193	.014
	Error	83.026	50	1.661(b)		
GROUP * DAY	Hypothesis	6.065	5	1.213	.730	.604
	Error	83.026	50	1.661(b)		

a MS(ID(GROUP))
b MS(Error)

Question 2 – Energy in Legs

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	Hypothesis	722.000	1	722.000	29.196	.000
	Error	247.297	10	24.730(a)		
GROUP	Hypothesis	.980	1	.980	.040	.846
	Error	247.297	10	24.730(a)		
ID(GROUP)	Hypothesis	247.297	10	24.730	16.824	.000
	Error	73.497	50	1.470(b)		
DAY	Hypothesis	26.237	5	5.247	3.570	.008
	Error	73.497	50	1.470(b)		
GROUP * DAY	Hypothesis	7.310	5	1.462	.995	.431
	Error	73.497	50	1.470(b)		

a MS(ID(GROUP))

Question 3 – Pain in Legs

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	Hypothesis	193.061	1	193.061	19.878	.001
	Error	97.124	10	9.712(a)		
GROUP	Hypothesis	22.557	1	22.557	2.322	.158
	Error	97.124	10	9.712(a)		
ID(GROUP)	Hypothesis	97.124	10	9.712	7.643	.000
	Error	63.535	50	1.271(b)		
DAY	Hypothesis	5.445	5	1.089	.857	.517
	Error	63.535	50	1.271(b)		
GROUP * DAY	Hypothesis	2.889	5	.578	.455	.808
	Error	63.535	50	1.271(b)		

a MS(ID(GROUP))
b MS(Error)

Question 4 – Ability to Complete ADL's

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	Hypothesis	39.014	1	39.014	7.710	.020
	Error	50.601	10	5.060(a)		
GROUP	Hypothesis	12.836	1	12.836	2.537	.142
	Error	50.601	10	5.060(a)		
ID(GROUP)	Hypothesis	50.601	10	5.060	5.209	.000
	Error	48.569	50	.971(b)		
DAY	Hypothesis	3.176	5	.635	.654	.660
	Error	48.569	50	.971(b)		
GROUP * DAY	Hypothesis	1.704	5	.341	.351	.879
	Error	48.569	50	.971(b)		

a MS(ID(GROUP))
b MS(Error)

Question 5 – Effectiveness of Treatment

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	Hypothesis	2630.788	1	2630.788	70.303	.000
	Error	374.206	10	37.421(a)		
GROUP	Hypothesis	207.948	1	207.948	5.557	.040
	Error	374.206	10	37.421(a)		
ID(GROUP)	Hypothesis	374.206	10	37.421	36.086	.000
	Error	41.479	40	1.037(b)		
DAY	Hypothesis	5.256	4	1.314	1.267	.299
	Error	41.479	40	1.037(b)		
GROUP * DAY	Hypothesis	4.433	4	1.108	1.069	.385
	Error	41.479	40	1.037(b)		

a MS(ID(GROUP))
b MS(Error)

APPENDIX E

Psychological Tests

POMS

SELF-EFFICACY

PREPAREDNESS FOR EXERCISE

TESTING THE WATERS: ARE THE EFFECTS OF HYDROTHERAPY MORE PSYCHOLOGICAL THAN PHYSIOLOGICAL?

Profile of Mood States

Date:_____ **Subject ID:**_____

Profile of Mood States (POMS)
Hydrotherapy study

Below is a list of words that describe feelings people have. Please read each one carefully, then fill in **ONE** space under the answer to the right, which best describes **HOW YOU HAVE BEEN FEELING THE PAST 24 HOURS.**

The numbers refer to these Phrases:

0- Not at all
1- A little
2- Moderately
3- Quite a bit
4- Extremely

	Not at all				Extremely
	0	1	2	3	4
1. Tense	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
2. Worn out	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
3. Unhappy	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
4. Lively	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
5. Sorry for thing done	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
6. Shaky	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
7. Listless	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
8. Sad	☐	☐	☐	☐	☐

	Not at all				Extremely
	0	1	2	3	4
9. Active	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
10. On edge	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
11. Blue	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
12. Energetic	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
13. Panicky	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
14. Hopeless	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
15. Relaxed	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
16. Unworthy	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
17. Uneasy	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
18. Restless	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
19. Fatigued	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
20. Discouraged	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
21. Nervous	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
22. Lonely	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
23. Miserable	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
24. Cheerful	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
25. Exhausted	☐	☐	☐	☐	☐

	Not at all				Extremely
	0	1	2	3	4
26. Anxious	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
27. Gloomy	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
28. Desperate	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
29. Sluggish	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
30. Helpless	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
31. Weary	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
32. Alert	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
33. Full of Pep	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
34. Worthless	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
35. Carefree	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
36. Terrified	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
37. Guilty	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
38. Vigorous	☐	☐	☐	☐	☐
	Not at all				Extremely
	0	1	2	3	4
39. Bushed	☐	☐	☐	☐	☐

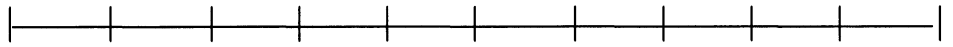
Make sure you have answered every item

Self-Efficacy

Date:_____ **Subject ID:**_____

Self-Efficacy Questionnaire
Hydrotherapy Study

Please indicate below how confident you are that you can successfully carry out **EACH** of the activities listed below, **RIGHT NOW**. Use a percentage based on the following scale.



0 10% 20% 30% 40% 50% 60% 70% 80% 90% 100%

Not at all Confident **A little Confident** **Moderately Confident** **Very Confident** **Extremely Confident**

Self-efficacy for walking

I believe that I can walk:

1) for 10 minutes at a moderately fast pace without stopping. _____
2) for 20 minutes at a moderately fast pace without stopping. _____
3) for 30 minutes at a moderately fast pace without stopping. _____
4) for 45minutes at a moderately fast pace without stopping. _____
5) for 60 minutes at a moderately fast pace without stopping. _____

Self-efficacy for stair climbing

I believe that I can climb:

6) 10 stairs without stopping. _____
7) 20 stairs without stopping. _____
8) 40 stairs without stopping. _____
9) 60 stairs without stopping. _____
10) 100 stairs without stopping _____

Self-efficacy for strength

I believe that I can :

11) leg press 50 lbs, 10 times without stopping. _____
12) extend my knee 10 times with 10 pounds without stopping._____
13) leg press 100 lbs, 10 times without stopping. _____
14) extend my knee 10 times with 25 pounds without stopping._____
15) leg press 150 lbs, 10 times without stopping. _____
16) extend my knee 10 times with 40 pounds without stopping._____

Self-efficacy for jumping

I believe that I can jump:

17) on two legs for 1 minute without stopping. _____
18) on two legs for 5 minute without stopping. _____
19) on two legs for 10 minute without stopping. _____
20) on one leg for 1 minute without stopping. _____
21) on one leg for 5 minute without stopping. _____

Self-efficacy for running

I believe that I can run, as fast as possible,:

22) for 50 meters without stopping. _____
23) for 100 meters without stopping. _____
24) for 400 meters without stopping. _____
25) for 800 meters without stopping. _____
26) for 1200 meters without stopping. _____

Self-efficacy for cycling

I believe that I can perform:

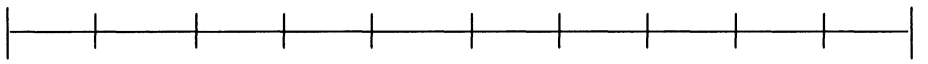
27) 1 Wingate without stopping. _____
28) 4 Wingate's, with a 4 minute rest between each. _____
29) 6 Wingate's, with a 4 minute rest between each. _____
30) 8 Wingate's, with a 4 minute rest between each. _____
31) 10 Wingate's, with a 4 minute rest between each. _____

Preparedness for Exercise

Date:_____ **Subject ID:**_____

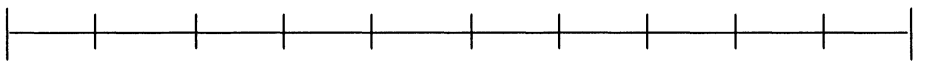
Preparedness for Exercise Scale
Hydrotherapy study

Please answer the following questions by placing a mark on the scale, following the question, that best describes how you feel **RIGHT NOW**.



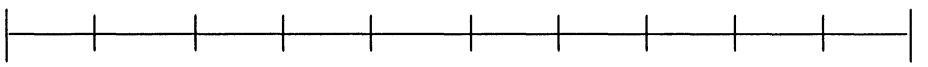
0 1 2 3 4 5 6 7 8 9 10
Not at all — a little — moderately — a lot — Extremely

1. Do you feel physically ABLE to exercise?



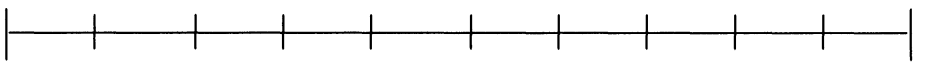
1 1 2 3 4 5 6 7 8 9 10
Not at all — a little — moderately — a lot — Extremely

2. How would you describe your legs?



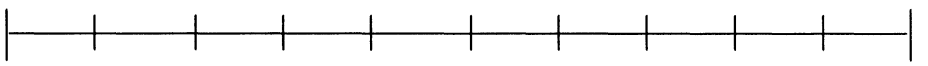
2 1 2 3 4 5 6 7 8 9 10
Energized — a little tired — Tired — Very Tired — "Dead"

3. Are you experiencing any PAIN in your legs related to exercise?



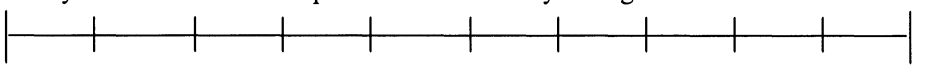
3 1 2 3 4 5 6 7 8 9 10
Not at all — a little — moderately — a lot — Extremely

4. Do you find it difficult to engage in your usual activities of daily living at this time?



4 1 2 3 4 5 6 7 8 9 10
Not at all — a little — moderately — a lot — Extremely

5. Do you find the treatment post-exercise makes your legs feel Better?



5 1 2 3 4 5 6 7 8 9 10
Not at all — a little — moderately — a lot — Extremely

APPENDIX F

REB Approval Form

McMaster University

Hamilton Health Sciences
EXEMPLARY CARE • ACADEMIC EXCELLENCE

RESEARCH ETHICS BOARD

August 6, 2004

PROJECT NUMBER: **04-224**

PROJECT TITLE: "Testing the Water: What is the most effective way to recover between bouts of intense exercise"

PRINCIPAL INVESTIGATOR: Dr. Martin Gibala

As you are aware your study was presented at the July 20, 2004 Research Ethics Board meeting where it received ***final*** approval from the full Research Ethics Board. The final approval is based on the revised documents submitted in your e-mail dated July 20, 2004. The submission, including the Consent Form and Information Sheet was found to be acceptable on both ethical and scientific grounds.

We are pleased to issue final approval for the above-named study for a period of 12 months from the date of this letter. Continuation beyond that date will require further review and renewal of REB approval. Any changes or amendments to the protocol or consent form must be approved by the Research Ethics Board.

We wish to advise the Research Ethics Board operates in compliance with ICH Good Clinical Practice Guidelines and the Tri-Council Policy Statement.

Investigators in the Project should be aware that they are responsible for ensuring that a complete consent form is inserted in the patient's health record. In the case of invasive or otherwise risky research, the investigator might consider the advisability of keeping personal copies.

A condition of approval is that the physician most responsible for the care of the patient is informed that the patient has agreed to enter the study. Any failure to meet this condition means that Research Ethics Board approval for the project has been withdrawn.

PLEASE QUOTE THE ABOVE-REFERENCED PROJECT NUMBER ON ALL FUTURE CORRESPONDENCE.

Sincerely,

J. Holland /dm

F. Jack Holland, MD, FRCP, FRCP(C)
Chair, Research Ethics Board

/dm

All correspondence should be addressed to the REB Chair and forwarded to:
REB Secretary, Henderson Campus, 90 Wing, Room #1
711 Concession Street, Hamilton ON L8V 1C3
Telephone: 905-527-4322, ext. 42013
Fax: 905-574-5645

APPENDIX G

Subject Information and Consent Forms

McMaster University

Exercise Metabolism Research Group
Department of Kinesiology EMRG Laboratory:
Ivor Wynne Centre, Room A103
1280 Main Street West
Hamilton, Ontario, Canada
L8S 4K1

Phone: 905-525-9140
ext. 27037
Dr. MJ Gibala: ext. 23591
Dr. MJ MacDonald: ext. 23580
Dr. SM Phillips: ext. 24465
Fax: 905-523-4025

EXERCISE METABOLISM RESEARCH GROUP (EMRG)
DEPARTMENT OF KINESIOLOGY, MCMASTER UNIVERSITY

CONSENT TO PARTICIPATE IN RESEARCH

You are asked to participate in a research study being conducted by the investigators listed below at McMaster University, Hamilton, Ontario. Prior to your participation, you are asked to read and complete this form and the accompanying form, which outline the purpose, procedures, and risks associated with the study, and also provide other essential information regarding your rights and responsibilities as a subject. The accompanying form is the "Subject Screening Questionnaire." All experimental testing and training will be conducted in the EMRG Laboratory, Ivor Wynne Centre, Room A103.

LIST OF PRIMARY INVESTIGATORS
Name Campus Address Daytime Phone Number
Dr. Martin Gibala Kinesiology, AB122, 905-525-9140 ext. 23591
Brian Timmons, Kinesiology, Evel - 305 , 905-525-9140 ext. 77259
Mr. Doug Stacey BHScPT 519-858-8393

PROJECT TITLE
Rest, Light Exercise or Cryotherapy: What is the Most Effective Way to Recover Between Repeated Bouts of Intense Exercise?

PURPOSE OF THE STUDY
The use of hydrotherapy as a form of recovery is already well in use in the sporting community yet there are no scientific studies available to validate its use. It is anticipated this study will shed some light on its benefits or lack thereof. The primary purpose of the proposed investigation is to examine the effects of cold baths on performance during repeated high intensity cycling bouts. Blood sample will be analyzed for markers that indicate fatigue and inflammation. We will also study the potential psychological effects of this intervention. We will assess performance through Aerobic endurance capacity (challenge ride) measurements. Pre-bout questionnaires (Visual analogue scale for pain, Borg scale for rating perceived exertion and Preparedness for Exercise) will be used to measure feelings towards the exercise/training.

DESCRIPTION OF TESTING AND EXPERIMENTAL PROCEDURES

Following routine medical screening and 2 familiarization visits to the laboratory (in order to become oriented with testing procedures and equipment), you will be required to make a total of 3 further visits to the laboratory over a period of approximately 3 weeks. Specifically, the study will consist of 2 pre-training (baseline) experimental exercise tests and 3 training sessions (Total of 5 visits). In each trial you will perform three challenge rides which involves stationary cycling at a high intensity (~90%VO_{2peak}) for 2-3 minutes. Each bout will be separated by a 20 minute recovery intervention Recovery interventions will be randomized as to order for each week and will include: Passive Recovery (PR) – rest stationary in a chair for 20 minutes; Active Recovery- pedal on a stationary bike for 10 min at a very low intensity (40% VO_{2peak}) + 10 min transition time; and Cryotherapy (Cryo) – sit in cold tub (10^0C) for 10 minutes (2min:1min rest interval) + 10 min transition time. Questionnaires (3) will be completed prior to commencing each training bout. You will also provide blood samples during the exercise protocol. Blood samples will be drawn from an indwelling catheter in the hand or forearm for analysis before, during and after the exercise bouts. The blood catheter will only remain in the hand or arm for the duration of the training bout.

OVERVIEW: LABORATORY VISITS, TESTING AND TRAINING PHASES:
*(**A) PRE-TRAINING PHASE: 2** tests will be conducted over 2 days:*
1. VO_{2peak} Test (~ 1 hour)
2. Challenge ride 2 x 50KJ with 20 min rest (~ 45 min)

***(B) TRAINING PHASE:** 3 lab visits (90 - 120 min each) over 3 weeks (1 day per week)*
Challenge ride 3 x 50 KJ with 20 min rest.

VO_{2peak} Test. This test involves cycling on a stationary bike (cycle ergometer) at progressively higher workloads while the amount of oxygen taken up by your body is determined from a mouthpiece connected to a gas analyzer. You will perform a VO_2max test prior to the high intensity cycling protocol.
Exercise Performance Trial (Challenge Ride) - You will perform a simulated "time trial" race, whereby you will be instructed to complete a standardized bout of work (50kJ) in as fast a time as possible. Pace will be self-selected throughout the test, which should last between 2 to 5 minutes depending on your fitness level and motivation. You will perform this time trial twice during familiarization with a 20 minute rest between bouts. During the exercise sessions, you will perform 3 time trials separated by a 20 minute recovery intervention.
Blood Samples – During the exercise bouts Blood samples will be drawn prior to the first exercise bout, before the second exercise bout, immediately after the final bout and 1 hour after completing the final bout (4 total).
Pre-Exercise Bout Questionnaires. You will complete three mini questionnaires (Visual analogue scale for pain, Borg scale for perceived exertion, and a Preparedness for Exercise Scale). The questionnaires will focus on feelings of well-being and preparedness for exercise.

Experimental Exercise Trial. Upon arrival at the laboratory, a catheter will be inserted into a forearm vein for blood sampling. The details of the blood sampling procedures and associated risks are thoroughly described on the attached forms entitled "Description of

Invasive Medical Procedures." You will perform 3 exercise trials separated by 1 week. You will refrain from any high intensity or strenuous exercise between each exercise session. In each trial you will perform three 50 KJ challenge rides (high intensity cycling at ~90%VO_{2peak}) separated by a 20 minute recovery intervention Recovery interventions will be randomized as to order for each week and will include: Passive Recovery (PR) – rest stationary in a chair for 20 minutes; Active Recovery- pedal on a stationary bike for 10 min at a low intensity (40% VO_{2peak}) + 10 min transition time; and Cryotherapy (Cryo) – sit in cold tub (10^0C) for 10 minutes (2min:1min rest interval) + 10 min transition time. Prior to training you will rest for 10 minutes and a blood sample will be drawn from an indwelling catheter in the hand or forearm for blood analysis. You will then perform the exercise protocol as randomly determined for that session. Blood samples will again be drawn prior to the second exercise bout, immediately after the final bout and 1 hour after completing the final bout. To gauge psychological status for exercise, you will complete a Visual Analogue Scale for pain and a Preparedness for Exercise questionnaire prior to commencing each bout of exercise. A Borg scale for rating perceived exertion will be completed immediately upon finishing each exercise bout.

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DESCRIPTION OF POTENTIAL RISKS AND DISCOMFORTS

Please refer to the attached form entitled "Description of Invasive Medical Procedures" for a complete description of the invasive medical procedures to be performed during the study and the potential risks and discomforts associated with these procedures.

Any other potential risks associated with this proposed study are physical and relate to the cycle training and the cold bath used for recovery. Although there are some conditions that preclude cryotherapy (eg. Rheumatoid diseases, coronary artery disease, and open sore) it is doubtful they will be present in our testing population. During the actual treatment session you may experience feelings of aching, burning, "pins and needles" or numbness with the cold bath. All sensations will be temporary and should resolve by the end of the treatment session.

The cycle training sessions will be brief but intense in nature. In addition to the stress associated with vigorous physical activity, you may experience severe local muscle fatigue/ache in your thigh muscles, nausea and/or light- headedness upon cessation of exercise. Some local muscle discomfort/fatigue may also be present throughout the training but this should not limit activities of daily living.

REMUNERATION

ou will receive an honorarium of $100.00 in order to compensate for your time commitment and effort. Remuneration is normally provided within one week following completion of the study.

PROVISION OF CONFIDENTIALITY

Any information that is obtained in connection with this study will remain confidential, and appropriate measures will be taken by all investigators to ensure privacy. The results from this study will be used for educational purposes and may be published in scientific journals, presented at scientific meetings or disseminated using other appropriate methods. Regardless of presentation format, subjects will not be identified by name, and your personal data will only be identified by a code number.

Upon completion of the study, you will have access to your own data and the group data for your own interest.

PARTICIPATION AND WITHDRAWAL

You can choose whether to be in this study or not. If you volunteer to be in this study, you may withdraw at any time without consequences of any kind. You may exercise the option of removing your data from the study. You may also refuse to answer any questions, which you do not want to and still remain in the study. The investigators also reserve the right to withdraw you from this research project if circumstances arise which warrant doing so. Should you withdraw from the study prior to its completion, a partial honorarium payment will be made based on the relative proportion of the study, which was completed.

RIGHTS OF RESEARCH PARTICIPANTS

You may withdraw your consent at any time and discontinue participation without penalty. You are not waiving any legal claims, rights or remedies because of your participation in this research study. The nature of the exercise stresses and treatment procedures to be employed in this study have been approved by the Hamilton Health Sciences Corporation / McMaster University Research Ethics Board.
If you have any questions regarding your rights as a research participant you may contact the Hamilton Health Sciences Patient Relations Specialist at 905-521-2100, Ext. 75240.

SIGNATURE OF RESEARCH PARTICIPANT/LEGAL REPRESENTATIVE

I have read and understand the information provided for the study as described herein and in the accompanying form entitled "Subject Screening Questionnaire." My questions have been answered to my satisfaction, and I agree to participate in this study. I have been given a signed copy of this form.

_____
Name of Participant

_____
Name of Legal Representative (if applicable)

_____ _____
Signature of Participant or Legal Representative Date

SIGNATURE OF INVESTIGATOR

In my judgment, the participant is voluntarily and knowingly giving informed consent and possesses the legal capacity to give informed consent to participate in this research study.

_____ _____
Signature of Investigator Date

McMaster University

Exercise Metabolism Research Group
Department of Kinesiology EMRG Laboratory:
Ivor Wynne Centre, Room A103
1280 Main Street West
Hamilton, Ontario, Canada
L8S 4K1

Phone: 905-525-9140
ext. 27037
Dr. MJ Gibala: ext. 23591
Dr. MJ MacDonald: ext. 23580
Dr. SM Phillips: ext. 24465
Fax: 905-523-4025

EXERCISE METABOLISM RESEARCH GROUP (EMRG)
DEPARTMENT OF KINESIOLOGY, MCMASTER UNIVERSITY

DESCRIPTION OF INVASIVE MEDICAL PROCEDURES

The study in which you are invited to participate involves several invasive medical procedures. Prior to your involvement in the study, you are asked to read this form which outlines the potential medical risks inherent to these procedures. In addition, you must also complete the "Subject Screening Questionnaire" which is designed to identify any medical reason which might preclude your participation as a subject.

Venous Catheterization and Blood Sampling

A small Teflon catheter will be inserted into a forearm vein with the assistance of a small needle, which is subsequently removed. The discomfort of this procedure is transient and is very similar to having an injection by a needle, or when donating blood. Once the needle is removed there should be no sensation from the catheter. During the course of the experiment, blood will be drawn periodically from the catheter. In any one experiment the total blood loss is typically less than 100 ml, which is approximately 1/6 of the blood removed during a donation to a blood bank. It is not enough of a blood loss to affect your physical performance in any way. After each blood sample has been taken, the catheter is "flushed" with a sterile saline solution in order to prevent blood from clotting in the catheter. This is a salt solution that is very similar in composition to your own blood and it will not affect you. Following removal of the catheter, pressure will be placed on the site in order to minimize bleeding and facilitate healing.

Potential Risks

The insertion of a venous catheter for blood sampling is a common medical practice and involves minimal risk provided proper precautions are taken. The catheter is inserted under completely sterile conditions, however there is a theoretical risk of infection. There is also chance of bleeding if adequate pressure is not maintained upon removal of the catheter. This may cause some minor discomfort and could result in bruising/skin discoloration which could last up to a few weeks. There is also the remote risk that trauma to the vessel wall could result in the formation of a small blood clot,

which could travel through the bloodstream and become lodged in a smaller vessel. However, we have never experienced such a complication in our laboratory after several thousand venous catheter placements.

APPENDIX H

Raw Data

REST, LIGHT EXERCISE OR CRYOTHERAPY: WHAT IS THE MOST EFFECTIVE WAY TO RECOVER BETWEEN REPEATED BOUTS OF INTENSE EXERCISE?

Time Trial Performance (sec)

ID	ORDER	TTA1	TTA2	TTA3	TTB1	TTB2	TTB3	TTC1	TTC2	TTC3
1	1	110	118	125	119	114	133	106	126	133
2	1	75	92	107	80	90	101	88	89	95
3	1	125	125	132	127	134	150	123	127	137
4	2	119	127	128	118	133	131	123	134	141
5	2	116	123	122	116	138	129	121	131	138
6	2	89	87	92	93	94	95	91	93	91
7	3	171	210	220	195	191	190	197	200	191
8	3	96	115	127	102	119	131	101	114	137
9	3	138	168	161	130	143	158	128	128	145
	mean	115.4	129.4	134.9	120.0	128.4	135.3	119.8	126.9	134.2
	SD	28.4	38.1	37.0	32.5	30.1	28.7	32.5	32.0	29.2
	SEM	9.5	12.7	12.3	10.8	10.0	9.6	10.8	10.7	9.7

Blood Lactate (mmol/L)

ID	ORDER	BLA0	BLA1	BLA2	BLA3	BLA4	BLB0	BLB1	BLB2	BLB3	BLB4	BLC0	BLC1	BLC2	BLC3	BLC4
1	1	0.43	3.9	4.03	4	1	0.35	3.2	3.5	3.8	0.9	0.31	4.6	4.5	4.4	0.9
2	1	0.44	4	4.73	4.3	1.2	0.53	4.9	5.1	5.2	1.2	0.61	3.8	4.1	4.3	1
3	1	0.46	3.4	3.83	4	0.8	0.36	4.1	4	3.3	0.8	0.52	3.9	4.7	4.4	0.8
4	2	1.24	3.1	4.08	3.7	1.1	0.71	5.3	3.9	4	1	0.71	3.8	4.1	4	0.5
5	2	0.2	3	2.53	3.3	0.8	0.64	3.7	2.2	2.4	0.5	0.24	2.8	3.5	3.4	0.6
6	2	0.66	3.1	3.56	4.3	1.1	0.97	3	2.7	1.3	1.1	0.47	4	4.7	5	1.5
7	3	0.61	4.6	4.16	4.4	0.7	0.27	3.5	4.3	4.3	1.3	0.34	4.4	5.2	5.3	0.8
8	3	0.54	4.9	5.32	5.4	1.4	0.49	3.3	3.5	2.9	2	0.37	2.7	2.8	2.3	1.1
9	3	0.38	4	3.28	3.6	1.3	0.53	5.8	5.5	4.6	1.4	0.7	4.9	6.2	5.8	1.4
	Mn	0.55	3.78	3.95	4.11	1.04	0.54	4.09	3.86	3.53	1.13	0.47	3.88	4.42	4.32	0.96
	SD	0.29	0.68	0.81	0.61	0.24	0.21	1.01	1.05	1.20	0.42	0.17	0.74	0.98	1.04	0.34
	SEM	0.1	0.2	0.3	0.2	0.1	0.1	0.3	0.4	0.4	0.1	0.1	0.3	0.3	0.4	0.1

IL-6 (pg/L)

ID	ORDER	IL6A0	IL6A1	IL6A2	IL6B0	IL6B1	!L6B2	IL6C0	IL6C1	IL6C2
1	1	1.82	3.15	5.99	1.84	3.34	4.68	1.86	2.54	2.77
2	1	0.67	0.98	0.47	0.58	1.58	4.51	0.45	0.79	1.21
3	1	1.89	2.37	3.58	1.35	2.16	2.28	1.79	1.92	1.90
4	2	1.77	2.32	3.13	2.34	2.26	5.09	2.35	3.54	5.78
5	2	0.50	1.00	7.49	0.42	1.70	2.55	0.81	1.31	1.91
6	2	1.31	1.37	0.05	1.66	1.32	1.15	0.84	0.83	1.36
7	3	2.93	3.91	5.34	2.53	3.21	4.52	6.49	8.38	5.80
8	3	1.27	2.00	10.14	1.05	1.41	1.40	1.00	1.36	1.85
9	3	2.57	3.56	-0.33	1.40	3.22	3.56	2.16	2.78	3.09
	Mean	1.64	2.30	3.98	1.46	2.24	3.30	1.97	2.61	2.85
	SD	0.80	1.08	3.59	0.72	0.82	1.50	1.82	2.36	1.77
	SEM	0.3	0.4	1.2	0.2	0.3	0.5	0.6	0.8	0.6

Leukocytes (10^9 cells/L)

ID	ORDER	LKCSA0	LKCSA1	LKCSA2	LKCSB0	LKCSB1	LKCSB2	LKCSC0	LKCSC1	LKCSC2
1	1	5.9	10.7	11.7	5.9	9.4	4.9	6.9	11.5	9.3
2	1	6.4	11.1	17.2	7.7	10.7	16	5.8	8.7	11.3
3	1	4.9	8	10.1	4.9	7.1	9	5.6	7.6	9.3
4	2	6	7.7	5.8	4.4	5.6	4.1	5.4	6.4	5.7
5	2	6.3	8.4	5.6	5.8	7.4	7.4	4.7	6	7.6
6	2	7.5	11.7	11.1	6.2	8.9	9	7	10.9	9.9
7	3	7.7	11.9	10	6.3	9.5	7.6	7.5	10.5	8.3
8	3	7.6	11.5	15.2	6.6	7.9	12.1	8.1	9.6	12.1
9	3	8.3	14.3	13.1	6.9	15.2	10.7	7.5	15.6	10.3
	Mean	6.73	10.59	11.09	6.08	9.08	8.98	6.50	9.64	9.31
	SD	1.10	2.17	3.86	1.00	2.75	3.65	1.16	2.96	1.94
	SEM	0.4	0.7	1.3	0.3	0.9	1.2	0.4	1.0	0.7

Lymphocytes (10^9 cells/L)

ID	ORDER	LYMA0	LYMA1	LYMA2	LYMB0	LYMB1	LYMB2	LYMC0	LYMC1	LYMC2
1	1	2.3	4.5	1.6	1.9	3.8	1.8	3	5.6	2.1
2	1	2.5	4.7	1.2	2.6	4.4	1.7	2.1	3.4	1.7
3	1	2	3.1	1.3	2	3.3	1.2	1.9	2.8	1.4
4	2	2.8	2.8	1.7	1.7	2.4	1.7	2	2.8	1.7
5	2	1.8	3.4	1.7	1.8	2.9	1.6	2.1	2.9	1.7
6	2	1.4	3.4	1	1.4	2.2	1.6	1.3	3.2	1.4
7	3	2.3	4.4	1.4	2.1	3.4	1.3	1.8	2.9	1.3
8	3	1.9	4	1.3	1.6	2.1	1.5	1.2	2	1.4
9	3	3.2	7	1.5	2.7	8.1	1.6	2.7	6.8	1.7
	Mean	2.24	4.14	1.41	1.98	3.62	1.56	2.01	3.60	1.60
	SD	0.55	1.26	0.24	0.44	1.84	0.19	0.58	1.55	0.25
	SEM	0.2	0.4	0.1	0.2	0.6	0.1	0.2	0.5	0.1

Neutrophils (10^9 cells/L)

ID	ORDER	NA0	NA1	NA2	NB0	NB1	NB2	NC0	NC1	NC2
1	1	2.8	5	9.1	3.1	4.3	6.5	2.6	3.9	6
2	1	2.8	4.5	14.5	3.8	4.5	13	2.2	3.5	7.7
3	1	2.2	3.9	8.1	2.4	3.4	7.2	3	3.8	7.3
4	2	2.4	3.8	3.2	1.9	2.1	1.9	2.5	2.4	2.9
5	2	3.7	4	3	3.4	3.6	5.1	1.9	2.2	5.1
6	2	5.1	7	9.2	3.8	5.4	6.6	4.9	6	7.8
7	3	4.5	6.2	7.7	3.4	4.8	5.5	5	6.2	6
8	3	4.8	6.1	13.1	4.1	5	9.4	6.5	7	10
9	3	4	5.7	10.5	3.5	5.7	8.5	4.1	6.5	7.1
	Mean	3.59	5.13	8.71	3.27	4.31	7.08	3.63	4.61	6.66
	SD	1.08	1.17	3.89	0.71	1.12	3.09	1.57	1.83	1.99
	SEM	0.4	0.4	1.3	0.2	0.4	1.0	0.5	0.6	0.7

Borg scale for ratings of perceived exertion (RPE)

ID	ORDER	RPE A1	RPE A2	RPE A3	RPE B1	RPE B2	RPE B3	RPE C1	RPE C2	RPE C3
1	1	16	17	18	18	17	17	17	17	18
2	1	19	17	18	20	20	19	18	17	19
3	1	17	18	18	17	18	17	16	17	17
4	2	17	18	17	18	18	17	18	19	19
5	2	15	15	14	15	15	15	17	15	14
6	2	15	18	19	15	18	19	16	18	19
7	3	17	17	20	17	18	20	19	20	20
8	3	15	17	18	16	17	18	16	19	20
9	3	17	17	16	16	19	17	17	18	18
	Mean	16.44	17.11	17.56	16.89	17.78	17.67	17.11	17.78	18.22
	SD	1.33	0.93	1.74	1.62	1.39	1.50	1.05	1.48	1.86
	SEM	0.4	0.3	0.6	0.5	0.5	0.5	0.4	0.5	0.6

Visual Analog Scale (VAS)

ID	ORDER	VASA0	VASA1	VASA2	VASA3	VASB0	VASB1	VASB2	VASB3	VASC0	VASC1	VASC2	VASC3
1	1	0	15	26	30	0	13	25	33	0	12	22	27
2	1	0	12	6	19	2	19	26	20	4	29	16	7
3	1	2	8	6	6	2	3	4	3	0	5.5	7	7
4	2	16	12	16	15.5	0	3	8	14	2	9	13	19
5	2	19	9	11.5	10	14	18	12	13	30	33	29	23
6	2	2	14	24	38.5	1	23	44	44	2	21.5	38	44
7	3	0	0	0	0	0	3	2.5	12	6	5	4	5
8	3	0	9	19	18	1	7	15	19	17	18	34	36
9	3	6	0	4	2	0	2.5	3	0	0	0	0	6
	Mean	5.00	8.78	12.50	15.44	2.22	10.17	15.50	17.56	6.78	14.78	18.11	19.33
	SD	7.38	5.49	9.24	12.75	4.49	8.18	13.87	13.83	10.22	11.35	13.52	14.34
	SEM	2.5	1.8	3.1	4.3	1.5	2.7	4.6	4.6	3.4	3.8	4.5	4.8

Preparedness for Exercise – Question 1

ID	ORDER	Prep1-A0	Prep1-A1	Prep1-A2	Prep1-A3	Prep1-B0	Prep1-B1	Prep1-B2	Prep1-B3	Prep1-C0	Prep1-C1	Prep1-C2	Prep1-C3
1	1	9	6	4	4	8	7	3	5	8	5	4	4
2	1	8	7.4	6	5	8	7	6	6.6	8	8	7	5.8
3	1	10	8	9	9	10	9	8	8	10	8	7	7
4	2	8	8	8.2	7.8	8	7.2	5.8	6.1	7	6	4.9	7
5	2	6	7	6	7	7	7	7	7	7	6	6	7
6	2	9.4	8.4	7.2	5.5	8	2.8	5.5	4.9	8.6	4.4	3.7	3.8
7	3	8	5	5	3	5	6	4	2	6	4	4	2
8	3	10	9	8.5	5.4	9	7.8	5	4.8	6.3	3	4.4	0.8
9	3	9	5	5	5.4	8	5	5	8	8	7.8	6.7	7.1
	Mean	8.60	7.09	6.54	5.79	7.89	6.53	5.48	5.82	7.66	5.80	5.30	4.94
	SD	1.26	1.46	1.76	1.86	1.36	1.78	1.49	1.90	1.23	1.85	1.38	2.40
	SEM	0.4	0.5	0.6	0.6	0.5	0.6	0.5	0.6	0.4	0.6	0.5	0.8

Preparedness for Exercise – Question 2

ID	ORDER	Prep2-A0	Prep2-A1	Prep2-A2	Prep2-A3	Prep2-B0	Prep2-B1	Prep2-B2	Prep2-B3	Prep2-C0	Prep2-C1	Prep2-C2	Prep2-C3
1	1	1	5	7	7	1	4	5	5	2	7	8	3
2	1	1	3	2.5	3.7	1.5	2	3.3	4	3	3	3.2	3
3	1	1	3	2	1	0	2	2	2	1	3	5	5
4	2	1.5	1.5	2.5	1.8	1	3.1	4.5	5.8	1.8	3.4	4.1	3.7
5	2	5	3	2	2	2	6	3	3	5	5	5	3
6	2	1.7	3.3	2.4	5.3	7.4	3.7	7.2	5.3	2.8	4.8	5.4	6.4
7	3	3	4	5	6	3	3	5	7	3	5	5	8
8	3	1	7	8	8.8	1	2	5	6.2	2.7	3.2	6.3	8.8
9	3	0.7	5	5.2	4.6	1	3	2.2	2	0.9	2	2.9	2.9
	Mean	1.77	3.87	4.07	4.47	1.99	3.20	4.13	4.48	2.47	4.04	4.99	4.87
	SD	1.39	1.60	2.30	2.60	2.19	1.28	1.66	1.83	1.25	1.53	1.55	2.33
	SEM	0.5	0.5	0.8	0.9	0.7	0.4	0.6	0.6	0.4	0.5	0.5	0.8

Preparedness for Exercise – Question 3

ID	ORDER	Prep3-A0	Prep3-A1	Prep3-A2	Prep3-A3	Prep3-B0	Prep3-B1	Prep3-B2	Prep3-B3	Prep3-C0	Prep3-C1	Prep3-C2	Prep3-C3
1	1	1	2	3	2	0	2	2	3	1	2	3	3
2	1	0.5	1.3	2	3	0.4	1.6	3	3	3	3	3.7	2
3	1	0	1	1	1	0	1	1	1	0	1	1	1
4	2	1.2	0.9	1.3	2.4	0	0.6	0.6	0.8	0.1	0.7	0.7	3.2
5	2	1	2	2	2	2	1	1	1	5	2	3	1
6	2	0.6	2.4	4.5	5.2	0.9	2.2	5.2	4.8	1.6	3.6	4.9	5.8
7	3	0	0	1	1	0	1	1	2	0	0.5	0.4	0.5
8	3	0.2	1	1.7	2.2	0.2	1.5	3.2	4.3	1.5	2.2	4.8	5.6
9	3	0	0	0	0	0	0	0	0	0	0	0.1	0
	Mean	0.50	1.18	1.83	2.09	0.39	1.21	1.89	2.21	1.36	1.67	2.40	2.46
	SD	0.48	0.85	1.30	1.47	0.68	0.69	1.64	1.66	1.71	1.20	1.89	2.13
	SEM	0.2	0.3	0.4	0.5	0.2	0.2	0.6	0.6	0.6	0.4	0.6	0.7

Preparedness for Exercise – Question 4

ID	ORDER	Prep4-A0	Prep4-A1	Prep4-A2	Prep4-A3	Prep4-B0	Prep4-B1	Prep4-B2	Prep4-B3	Prep4-C0	Prep4-C1	Prep4-C2	Prep4-C3
1	1	0	3	3	2	0	2	5	4	0	2	2	2
2	1	0	0	1	4	0	2.4	3.2	2.7	0	0	1.9	1.3
3	1	0	1	1	1	0	1	2	1	0	3	4	4
4	2	0	0	0.1	0.3	0	0	0.4	0.8	0	1.6	0.8	1.7
5	2	2	2	2.5	2	0	0	0	0	1	1	1	1
6	2	0.2	0.3	0.5	0.3	0.5	0.4	1.3	2.6	0.1	0.6	0.5	0.5
7	3	0	1	3	4	0	1	3	4	0	0	1	4
8	3	0	1	1	4	0	1.5	3.2	3.4	1.3	2.5	3.9	5.3
9	3	0	4	2.9	2.9	0	0	3	0	0	0	0.6	0.6
	Mean	0.24	1.37	1.67	2.28	0.06	0.92	2.34	2.06	0.27	1.19	1.74	2.27
	SD	0.66	1.38	1.17	1.54	0.17	0.90	1.58	1.63	0.51	1.14	1.35	1.73
	SEM	0.2	0.5	0.4	0.5	0.1	0.3	0.5	0.5	0.2	0.4	0.5	0.6

Preparedness for Exercise – Question 5

ID	ORDER	Prep5-A0	Prep5-A1	Prep5-A2	Prep5-A3	Prep5-B0	Prep5-B1	Prep5-B2	Prep5-B3	Prep5-C0	Prep5-C1	Prep5-C2	Prep5-C3
1	1	0	3	2	2	0	4	4	4	0	6	5	5
2	1	0	3	4	3	0	1	1	3	0	3	0.9	1
3	1	0	8	8	9	0	8	7	8	0	5	4	5
4	2	0	7.8	7.8	6.9	0	7.8	7.3	6.8	0	5.1	4.4	3.8
5	2	0	7	6	7.6	0	5	6	6	0	1	0	1
6	2	0	5.4	4.4	5.8	0	6.8	4.4	4.4	0	1.4	2.6	2.6
7	3	0	7	6	5	0	2	1	1	0	3	2	2
8	3	0	7	6.3	6.9	0	1.3	1.5	4.5	0	1.2	0.6	0.4
9	3	0	7	8	7.9	0	8	8.1	8	0	5	3	2.3
	Mean	0.00	6.13	5.83	6.01	0.00	4.88	4.48	5.08	0.00	3.41	2.50	2.57
	SD	0.00	1.92	2.05	2.31	0.00	2.93	2.81	2.34	0.00	1.93	1.77	1.71
	SEM	0.0	0.6	0.7	0.8	0.0	1.0	0.9	0.8	0.0	0.6	0.6	0.6

APPENDIX I

Statistical Tables

REPEATED MEASURE ANOVA'S

BONFERRONI PAIRWISE COMPARISONS

REST, LIGHT EXERCISE OR CRYOTHERAPY: WHAT IS THE MOST EFFECTIVE WAY TO RECOVER BETWEEN REPEATED BOUTS OF INTENSE EXERCISE?

1. Time Trial Performance (sec)
Two-Factor Repeated Measure Anova (Time x Group)

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
TIME	Sphericity Assumed	3683.136	2	1841.568	41.673	.000
	Greenhouse-Geisser	3683.136	1.461	2520.137	41.673	.000
	Huynh-Feldt	3683.136	2.000	1841.568	41.673	.000
	Lower-bound	3683.136	1.000	3683.136	41.673	.001
TIME * ORDER	Sphericity Assumed	539.901	4	134.975	3.054	.060
	Greenhouse-Geisser	539.901	2.923	184.710	3.054	.087
	Huynh-Feldt	539.901	4.000	134.975	3.054	.060
	Lower-bound	539.901	2.000	269.951	3.054	.122
GROUP	Sphericity Assumed	25.580	2	12.790	.190	.829
	Greenhouse-Geisser	25.580	1.564	16.360	.190	.778
	Huynh-Feldt	25.580	2.000	12.790	.190	.829
	Lower-bound	25.580	1.000	25.580	.190	.678
GROUP * ORDER	Sphericity Assumed	525.235	4	131.309	1.954	.166
	Greenhouse-Geisser	525.235	3.127	167.955	1.954	.188
	Huynh-Feldt	525.235	4.000	131.309	1.954	.166
	Lower-bound	525.235	2.000	262.617	1.954	.222
TIME * GROUP	Sphericity Assumed	128.642	4	32.160	.611	.658
	Greenhouse-Geisser	128.642	1.829	70.330	.611	.546
	Huynh-Feldt	128.642	3.467	37.101	.611	.637
	Lower-bound	128.642	1.000	128.642	.611	.464
TIME * GROUP * ORDER	Sphericity Assumed	566.988	8	70.873	1.347	.269
	Greenhouse-Geisser	566.988	3.658	154.990	1.347	.312
	Huynh-Feldt	566.988	6.935	81.760	1.347	.279
	Lower-bound	566.988	2.000	283.494	1.347	.329

Pairwise Comparisons

Measure: **Time**

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.(a)	95% Confidence Interval for Difference(a)	
					Lower Bound	Upper Bound
1	2	-9.852(*)	1.481	.002	-14.721	-4.983
	3	-16.407(*)	2.293	.001	-23.946	-8.869
2	1	9.852(*)	1.481	.002	4.983	14.721
	3	-6.556(*)	1.539	.016	-11.615	-1.496
3	1	16.407(*)	2.293	.001	8.869	23.946
	2	6.556(*)	1.539	.016	1.496	11.615

Based on estimated marginal means

* The mean difference is significant at the .05 level.

a Adjustment for multiple comparisons: Bonferroni.

2. Blood Lactate (mmol/L)

Two-Factor Repeated Measure Anova (Time x Group)

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
GROUP	Sphericity Assumed	.767	2	.384	.253	.781
	Greenhouse-Geisser	.767	1.794	.428	.253	.758
	Huynh-Feldt	.767	2.000	.384	.253	.781
	Lower-bound	.767	1.000	.767	.253	.633
GROUP * ORDER	Sphericity Assumed	.625	4	.156	.103	.979
	Greenhouse-Geisser	.625	3.587	.174	.103	.972
	Huynh-Feldt	.625	4.000	.156	.103	.979
	Lower-bound	.625	2.000	.313	.103	.904
TIME	Sphericity Assumed	337.866	4	84.466	344.998	.000
	Greenhouse-Geisser	337.866	1.617	208.926	344.998	.000
	Huynh-Feldt	337.866	2.864	117.952	344.998	.000
	Lower-bound	337.866	1.000	337.866	344.998	.000
TIME * ORDER	Sphericity Assumed	4.930	8	.616	2.517	.038
	Greenhouse-Geisser	4.930	3.234	1.524	2.517	.117
	Huynh-Feldt	4.930	5.729	.861	2.517	.064
	Lower-bound	4.930	2.000	2.465	2.517	.161
GROUP * TIME	Sphericity Assumed	4.528	8	.566	1.781	.104
	Greenhouse-Geisser	4.528	2.609	1.736	1.781	.196
	Huynh-Feldt	4.528	6.333	.715	1.781	.126
	Lower-bound	4.528	1.000	4.528	1.781	.230
GROUP * TIME * ORDER	Sphericity Assumed	3.866	16	.242	.760	.720
	Greenhouse-Geisser	3.866	5.217	.741	.760	.596
	Huynh-Feldt	3.866	12.666	.305	.760	.692
	Lower-bound	3.866	2.000	1.933	.760	.508

Pairwise Comparisons

Measure: **TIME**

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.(a)	95% Confidence Interval for Difference(a)	
					Lower Bound	Upper Bound
1	2	-3.393(*)	.130	.000	-3.957	-2.830
	3	-3.553(*)	.137	.000	-4.146	-2.960
	4	-3.467(*)	.143	.000	-4.086	-2.849
	5	-.523(*)	.077	.005	-.856	-.190
2	1	3.393(*)	.130	.000	2.830	3.957
	3	-.160	.080	.939	-.507	.187
	4	-.074	.087	1.000	-.451	.303
	5	2.870(*)	.176	.000	2.110	3.631
3	1	3.553(*)	.137	.000	2.960	4.146
	2	.160	.080	.939	-.187	.507
	4	.086	.075	1.000	-.239	.411
	5	3.030(*)	.179	.000	2.259	3.801
4	1	3.467(*)	.143	.000	2.849	4.086
	2	.074	.087	1.000	-.303	.451
	3	-.086	.075	1.000	-.411	.239
	5	2.944(*)	.190	.000	2.122	3.767
5	1	.523(*)	.077	.005	.190	.856
	2	-2.870(*)	.176	.000	-3.631	-2.110
	3	-3.030(*)	.179	.000	-3.801	-2.259
	4	-2.944(*)	.190	.000	-3.767	-2.122

3. IL-6 (pg/L)

Two-Factor Repeated Measure Anova (Time x Group)

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
GROUP	Sphericity Assumed	.767	2	.384	.253	.781
	Greenhouse-Geisser	.767	1.794	.428	.253	.758
	Huynh-Feldt	.767	2.000	.384	.253	.781
	Lower-bound	.767	1.000	.767	.253	.633
GROUP * ORDER	Sphericity Assumed	.625	4	.156	.103	.979
	Greenhouse-Geisser	.625	3.587	.174	.103	.972
	Huynh-Feldt	.625	4.000	.156	.103	.979
	Lower-bound	.625	2.000	.313	.103	.904
TIME	Sphericity Assumed	337.866	4	84.466	344.998	.000
	Greenhouse-Geisser	337.866	1.617	208.926	344.998	.000
	Huynh-Feldt	337.866	2.864	117.952	344.998	.000
	Lower-bound	337.866	1.000	337.866	344.998	.000
TIME * ORDER	Sphericity Assumed	4.930	8	.616	2.517	.038
	Greenhouse-Geisser	4.930	3.234	1.524	2.517	.117
	Huynh-Feldt	4.930	5.729	.861	2.517	.064
	Lower-bound	4.930	2.000	2.465	2.517	.161
GROUP * TIME	Sphericity Assumed	4.528	8	.566	1.781	.104
	Greenhouse-Geisser	4.528	2.609	1.736	1.781	.196
	Huynh-Feldt	4.528	6.333	.715	1.781	.126
	Lower-bound	4.528	1.000	4.528	1.781	.230
GROUP * TIME * ORDER	Sphericity Assumed	3.866	16	.242	.760	.720
	Greenhouse-Geisser	3.866	5.217	.741	.760	.596
	Huynh-Feldt	3.866	12.666	.305	.760	.692
	Lower-bound	3.866	2.000	1.933	.760	.508

Pairwise Comparisons

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.(a)	95% Confidence Interval for Difference(a)	
					Lower Bound	Upper Bound
1	2	-3.393(*)	.130	.000	-3.957	-2.830
	3	-3.553(*)	.137	.000	-4.146	-2.960
	4	-3.467(*)	.143	.000	-4.086	-2.849
	5	-.523(*)	.077	.005	-.856	-.190
2	1	3.393(*)	.130	.000	2.830	3.957
	3	-.160	.080	.939	-.507	.187
	4	-.074	.087	1.000	-.451	.303
	5	2.870(*)	.176	.000	2.110	3.631
3	1	3.553(*)	.137	.000	2.960	4.146
	2	.160	.080	.939	-.187	.507
	4	.086	.075	1.000	-.239	.411
	5	3.030(*)	.179	.000	2.259	3.801
4	1	3.467(*)	.143	.000	2.849	4.086
	2	.074	.087	1.000	-.303	.451
	3	-.086	.075	1.000	-.411	.239
	5	2.944(*)	.190	.000	2.122	3.767
5	1	.523(*)	.077	.005	.190	.856
	2	-2.870(*)	.176	.000	-3.631	-2.110
	3	-3.030(*)	.179	.000	-3.801	-2.259
	4	-2.944(*)	.190	.000	-3.767	-2.122

4. Leukocytes (10^9 cells/L)

Two-Factor Repeated Measure Anova (Time x Group)

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
GROUP	Sphericity Assumed	28.783	2	14.391	5.566	.019
	Greenhouse-Geisser	28.783	1.312	21.942	5.566	.040
	Huynh-Feldt	28.783	2.000	14.391	5.566	.019
	Lower-bound	28.783	1.000	28.783	5.566	.056
GROUP * ORDER	Sphericity Assumed	1.974	4	.494	.191	.939
	Greenhouse-Geisser	1.974	2.624	.752	.191	.879
	Huynh-Feldt	1.974	4.000	.494	.191	.939
	Lower-bound	1.974	2.000	.987	.191	.831
TIME	Sphericity Assumed	201.342	2	100.671	11.911	.001
	Greenhouse-Geisser	201.342	1.570	128.219	11.911	.004
	Huynh-Feldt	201.342	2.000	100.671	11.911	.001
	Lower-bound	201.342	1.000	201.342	11.911	.014
TIME * ORDER	Sphericity Assumed	37.126	4	9.281	1.098	.401
	Greenhouse-Geisser	37.126	3.141	11.821	1.098	.400
	Huynh-Feldt	37.126	4.000	9.281	1.098	.401
	Lower-bound	37.126	2.000	18.563	1.098	.392
GROUP * TIME	Sphericity Assumed	6.879	4	1.720	2.263	.092
	Greenhouse-Geisser	6.879	2.718	2.531	2.263	.124
	Huynh-Feldt	6.879	4.000	1.720	2.263	.092
	Lower-bound	6.879	1.000	6.879	2.263	.183
GROUP * TIME * ORDER	Sphericity Assumed	12.728	8	1.591	2.094	.077
	Greenhouse-Geisser	12.728	5.436	2.341	2.094	.115
	Huynh-Feldt	12.728	8.000	1.591	2.094	.077
	Lower-bound	12.728	2.000	6.364	2.094	.204

Pairwise Comparisons

Measure: **GROUP**

(I) GROUP	(J) GROUP	Mean Difference (I-J)	Std. Error	Sig.(a)	95% Confidence Interval for Difference(a)	
					Lower Bound	Upper Bound
1	2	1.426(*)	.382	.029	.171	2.681
	3	.985	.318	.063	-.060	2.030
2	1	-1.426(*)	.382	.029	-2.681	-.171
	3	-.441	.573	1.000	-2.323	1.441
3	1	-.985	.318	.063	-2.030	.060
	2	.441	.573	1.000	-1.441	2.323

Based on estimated marginal means

* The mean difference is significant at the .05 level.

a Adjustment for multiple comparisons: Bonferroni.

Pairwise Comparisons

Measure: **TIME**

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.(a)	95% Confidence Interval for Difference(a)	
					Lower Bound	Upper Bound
1	2	-3.333(*)	.605	.005	-5.323	-1.343
	3	-3.356(*)	.764	.014	-5.866	-.845
2	1	3.333(*)	.605	.005	1.343	5.323
	3	-.022	.964	1.000	-3.190	3.146
3	1	3.356(*)	.764	.014	.845	5.866
	2	.022	.964	1.000	-3.146	3.190

Based on estimated marginal means
* The mean difference is significant at the .05 level.
a Adjustment for multiple comparisons: Bonferroni.

5. Lymphocytes (10^9 cells/L)
Two-Factor Repeated Measure Anova (Time x Group)

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
GROUP	Sphericity Assumed	.765	2	.383	1.527	.257
	Greenhouse-Geisser	.765	1.660	.461	1.527	.261
	Huynh-Feldt	.765	2.000	.383	1.527	.257
	Lower-bound	.765	1.000	.765	1.527	.263
GROUP * ORDER	Sphericity Assumed	1.253	4	.313	1.250	.342
	Greenhouse-Geisser	1.253	3.319	.377	1.250	.346
	Huynh-Feldt	1.253	4.000	.313	1.250	.342
	Lower-bound	1.253	2.000	.626	1.250	.352
TIME	Sphericity Assumed	75.369	2	37.684	22.506	.000
	Greenhouse-Geisser	75.369	1.054	71.491	22.506	.003
	Huynh-Feldt	75.369	1.514	49.779	22.506	.000
	Lower-bound	75.369	1.000	75.369	22.506	.003
TIME * ORDER	Sphericity Assumed	7.589	4	1.897	1.133	.387
	Greenhouse-Geisser	7.589	2.108	3.599	1.133	.383
	Huynh-Feldt	7.589	3.028	2.506	1.133	.387
	Lower-bound	7.589	2.000	3.794	1.133	.382
GROUP * TIME	Sphericity Assumed	1.499	4	.375	2.300	.088
	Greenhouse-Geisser	1.499	2.164	.693	2.300	.137
	Huynh-Feldt	1.499	4.000	.375	2.300	.088
	Lower-bound	1.499	1.000	1.499	2.300	.180
GROUP * TIME * ORDER	Sphericity Assumed	.625	8	.078	.480	.859
	Greenhouse-Geisser	.625	4.328	.144	.480	.763
	Huynh-Feldt	.625	8.000	.078	.480	.859
	Lower-bound	.625	2.000	.313	.480	.641

Pairwise Comparisons

Measure: **TIME**

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.(a)	95% Confidence Interval for Difference(a)	
					Lower Bound	Upper Bound
1	2	-1.711(*)	.379	.012	-2.958	-.464
	3	.556(*)	.118	.010	.169	.942
2	1	1.711(*)	.379	.012	.464	2.958
	3	2.267(*)	.463	.008	.745	3.789
3	1	-.556(*)	.118	.010	-.942	-.169
	2	-2.267(*)	.463	.008	-3.789	-.745

Based on estimated marginal means

* The mean difference is significant at the .05 level.

a Adjustment for multiple comparisons: Bonferroni.

6. Neutrophils (10^9 cells/L)

Two-Factor Repeated Measure Anova (Time x Group)

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
GROUP	Sphericity Assumed	14.194	2	7.097	5.243	.023
	Greenhouse-Geisser	14.194	1.532	9.264	5.243	.036
	Huynh-Feldt	14.194	2.000	7.097	5.243	.023
	Lower-bound	14.194	1.000	14.194	5.243	.062
GROUP * ORDER	Sphericity Assumed	8.058	4	2.014	1.488	.267
	Greenhouse-Geisser	8.058	3.064	2.630	1.488	.281
	Huynh-Feldt	8.058	4.000	2.014	1.488	.267
	Lower-bound	8.058	2.000	4.029	1.488	.299
TIME	Sphericity Assumed	226.030	2	113.015	28.622	.000
	Greenhouse-Geisser	226.030	1.115	202.709	28.622	.001
	Huynh-Feldt	226.030	1.645	137.410	28.622	.000
	Lower-bound	226.030	1.000	226.030	28.622	.002
TIME * ORDER	Sphericity Assumed	51.475	4	12.869	3.259	.050
	Greenhouse-Geisser	51.475	2.230	23.082	3.259	.100
	Huynh-Feldt	51.475	3.290	15.646	3.259	.066
	Lower-bound	51.475	2.000	25.737	3.259	.110
GROUP * TIME	Sphericity Assumed	10.858	4	2.714	5.605	.002
	Greenhouse-Geisser	10.858	1.490	7.289	5.605	.033
	Huynh-Feldt	10.858	2.529	4.294	5.605	.011
	Lower-bound	10.858	1.000	10.858	5.605	.056
GROUP * TIME * ORDER	Sphericity Assumed	13.547	8	1.693	3.497	.008
	Greenhouse-Geisser	13.547	2.979	4.547	3.497	.064
	Huynh-Feldt	13.547	5.057	2.679	3.497	.026
	Lower-bound	13.547	2.000	6.773	3.497	.098

Pairwise Comparisons

Measure: **GROUP**

(I) GROUP	(J) GROUP	Mean Difference (I-J)	Std. Error	Sig.(a)	95% Confidence Interval for Difference(a)	
					Lower Bound	Upper Bound
1	2	.926(*)	.262	.037	.065	1.787
	3	.844	.277	.068	-.066	1.755
2	1	-.926(*)	.262	.037	-1.787	-.065
	3	-.081	.394	1.000	-1.378	1.215
3	1	-.844	.277	.068	-1.755	.066
	2	.081	.394	1.000	-1.215	1.378

Based on estimated marginal means
* The mean difference is significant at the .05 level.
a Adjustment for multiple comparisons: Bonferroni.

Pairwise Comparisons

Measure: **TIME**

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.(a)	95% Confidence Interval for Difference(a)	
					Lower Bound	Upper Bound
1	2	-1.189(*)	.179	.002	-1.776	-.602
	3	-3.985(*)	.650	.003	-6.121	-1.849
2	1	1.189(*)	.179	.002	.602	1.776
	3	-2.796(*)	.651	.015	-4.935	-.657
3	1	3.985(*)	.650	.003	1.849	6.121
	2	2.796(*)	.651	.015	.657	4.935

7. BORG scale for ratings of perceived exertion (RPE)
Two-Factor Repeated Measure Anova (Time x Group)

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
TIME	Sphericity Assumed	14.543	2	7.272	2.659	.111
	Greenhouse-Geisser	14.543	1.408	10.333	2.659	.135
	Huynh-Feldt	14.543	2.000	7.272	2.659	.111
	Lower-bound	14.543	1.000	14.543	2.659	.154
TIME * ORDER	Sphericity Assumed	7.309	4	1.827	.668	.626
	Greenhouse-Geisser	7.309	2.815	2.596	.668	.585
	Huynh-Feldt	7.309	4.000	1.827	.668	.626
	Lower-bound	7.309	2.000	3.654	.668	.547
GROUP	Sphericity Assumed	6.099	2	3.049	5.744	.018
	Greenhouse-Geisser	6.099	1.945	3.135	5.744	.019
	Huynh-Feldt	6.099	2.000	3.049	5.744	.018
	Lower-bound	6.099	1.000	6.099	5.744	.054
GROUP * ORDER	Sphericity Assumed	9.531	4	2.383	4.488	.019
	Greenhouse-Geisser	9.531	3.891	2.450	4.488	.020
	Huynh-Feldt	9.531	4.000	2.383	4.488	.019
	Lower-bound	9.531	2.000	4.765	4.488	.064
TIME * GROUP	Sphericity Assumed	.938	4	.235	.458	.766
	Greenhouse-Geisser	.938	2.280	.411	.458	.666
	Huynh-Feldt	.938	4.000	.235	.458	.766
	Lower-bound	.938	1.000	.938	.458	.524
TIME * GROUP * ORDER	Sphericity Assumed	4.099	8	.512	1.000	.461
	Greenhouse-Geisser	4.099	4.560	.899	1.000	.448
	Huynh-Feldt	4.099	8.000	.512	1.000	.461
	Lower-bound	4.099	2.000	2.049	1.000	.422

Pairwise Comparisons

Measure: **GROUP**

(I) GROUP	(J) GROUP	Mean Difference (I-J)	Std. Error	Sig.(a)	95% Confidence Interval for Difference(a)	
					Lower Bound	Upper Bound
1	2	-.407	.203	.274	-1.074	.259
	3	-.667(*)	.181	.031	-1.263	-.070
2	1	.407	.203	.274	-.259	1.074
	3	-.259	.210	.786	-.948	.430
3	1	.667(*)	.181	.031	.070	1.263
	2	.259	.210	.786	-.430	.948

Based on estimated marginal means

* The mean difference is significant at the .05 level.

a Adjustment for multiple comparisons: Bonferroni.

8. Visual Analog Scale (VAS)

Two-Factor Repeated Measure Anova (Time x Group)

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
TIME	Sphericity Assumed	2571.081	3	857.027	4.873	.012
	Greenhouse-Geisser	2571.081	1.133	2269.561	4.873	.061
	Huynh-Feldt	2571.081	1.684	1526.880	4.873	.037
	Lower-bound	2571.081	1.000	2571.081	4.873	.069
TIME * ORDER	Sphericity Assumed	340.801	6	56.800	.323	.916
	Greenhouse-Geisser	340.801	2.266	150.417	.323	.759
	Huynh-Feldt	340.801	3.368	101.195	.323	.829
	Lower-bound	340.801	2.000	170.400	.323	.736
GROUP	Sphericity Assumed	372.097	2	186.049	1.862	.198
	Greenhouse-Geisser	372.097	1.847	201.485	1.862	.202
	Huynh-Feldt	372.097	2.000	186.049	1.862	.198
	Lower-bound	372.097	1.000	372.097	1.862	.221
GROUP * ORDER	Sphericity Assumed	211.431	4	52.858	.529	.717
	Greenhouse-Geisser	211.431	3.694	57.243	.529	.705
	Huynh-Feldt	211.431	4.000	52.858	.529	.717
	Lower-bound	211.431	2.000	105.715	.529	.614
TIME * GROUP	Sphericity Assumed	110.495	6	18.416	1.336	.267
	Greenhouse-Geisser	110.495	2.382	46.392	1.336	.298
	Huynh-Feldt	110.495	5.372	20.570	1.336	.273
	Lower-bound	110.495	1.000	110.495	1.336	.292
TIME * GROUP * ORDER	Sphericity Assumed	195.144	12	16.262	1.180	.333
	Greenhouse-Geisser	195.144	4.764	40.966	1.180	.365
	Huynh-Feldt	195.144	10.743	18.164	1.180	.339
	Lower-bound	195.144	2.000	97.572	1.180	.370

Pairwise Comparisons

Measure: **TIME**

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.(a)	95% Confidence Interval for Difference(a)	
					Lower Bound	Upper Bound
1	2	-6.574	2.458	.221	-16.070	2.922
	3	-10.704	4.557	.343	-28.308	6.901
	4	-12.778	5.488	.353	-33.978	8.423
2	1	6.574	2.458	.221	-2.922	16.070
	3	-4.130	2.624	1.000	-14.266	6.007
	4	-6.204	3.606	.817	-20.135	7.727
3	1	10.704	4.557	.343	-6.901	28.308
	2	4.130	2.624	1.000	-6.007	14.266
	4	-2.074	1.158	.741	-6.549	2.401
4	1	12.778	5.488	.353	-8.423	33.978
	2	6.204	3.606	.817	-7.727	20.135
	3	2.074	1.158	.741	-2.401	6.549

Based on estimated marginal means

a Adjustment for multiple comparisons: Bonferroni.

9. Preparedness for Exercise (Prep)
Two-Factor Repeated Measure Anova (Time x Group)

Preparedness for Exercise – Question 1 – Ability to Exercise

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
TIME	Sphericity Assumed	104.737	3	34.912	13.889	.000
	Greenhouse-Geisser	104.737	2.132	49.135	13.889	.001
	Huynh-Feldt	104.737	3.000	34.912	13.889	.000
	Lower-bound	104.737	1.000	104.737	13.889	.010
TIME * ORDER	Sphericity Assumed	12.280	6	2.047	.814	.573
	Greenhouse-Geisser	12.280	4.263	2.880	.814	.545
	Huynh-Feldt	12.280	6.000	2.047	.814	.573
	Lower-bound	12.280	2.000	6.140	.814	.487
GROUP	Sphericity Assumed	21.046	2	10.523	2.542	.120
	Greenhouse-Geisser	21.046	1.228	17.145	2.542	.152
	Huynh-Feldt	21.046	1.896	11.101	2.542	.124
	Lower-bound	21.046	1.000	21.046	2.542	.162
GROUP * ORDER	Sphericity Assumed	6.451	4	1.613	.390	.812
	Greenhouse-Geisser	6.451	2.455	2.628	.390	.729
	Huynh-Feldt	6.451	3.792	1.701	.390	.803
	Lower-bound	6.451	2.000	3.225	.390	.693
TIME * GROUP	Sphericity Assumed	3.442	6	.574	.804	.573
	Greenhouse-Geisser	3.442	3.014	1.142	.804	.508
	Huynh-Feldt	3.442	6.000	.574	.804	.573
	Lower-bound	3.442	1.000	3.442	.804	.404
TIME * GROUP * ORDER	Sphericity Assumed	11.135	12	.928	1.301	.260
	Greenhouse-Geisser	11.135	6.027	1.847	1.301	.306
	Huynh-Feldt	11.135	12.000	.928	1.301	.260
	Lower-bound	11.135	2.000	5.568	1.301	.339

Pairwise Comparisons

Measure: **TIME**

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.(a)	95% Confidence Interval for Difference(a)	
					Lower Bound	Upper Bound
1	2	1.574	.410	.052	-.011	3.159
	3	2.274(*)	.429	.011	.618	3.930
	4	2.530(*)	.580	.029	.289	4.771
2	1	-1.574	.410	.052	-3.159	.011
	3	.700	.220	.115	-.151	1.551
	4	.956	.444	.451	-.761	2.673
3	1	-2.274(*)	.429	.011	-3.930	-.618
	2	-.700	.220	.115	-1.551	.151
	4	.256	.427	1.000	-1.395	1.906
4	1	-2.530(*)	.580	.029	-4.771	-.289
	2	-.956	.444	.451	-2.673	.761
	3	-.256	.427	1.000	-1.906	1.395

Based on estimated marginal means
* The mean difference is significant at the .05 level.
a Adjustment for multiple comparisons: Bonferroni.

Preparedness for Exercise – Question 2 – Energy in Legs

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
TIME	Sphericity Assumed	106.516	3	35.505	11.371	.000
	Greenhouse-Geisser	106.516	1.798	59.240	11.371	.003
	Huynh-Feldt	106.516	3.000	35.505	11.371	.000
	Lower-bound	106.516	1.000	106.516	11.371	.015
TIME * ORDER	Sphericity Assumed	32.796	6	5.466	1.751	.167
	Greenhouse-Geisser	32.796	3.596	9.120	1.751	.212
	Huynh-Feldt	32.796	6.000	5.466	1.751	.167
	Lower-bound	32.796	2.000	16.398	1.751	.252
GROUP	Sphericity Assumed	8.672	2	4.336	2.321	.141
	Greenhouse-Geisser	8.672	1.848	4.692	2.321	.146
	Huynh-Feldt	8.672	2.000	4.336	2.321	.141
	Lower-bound	8.672	1.000	8.672	2.321	.178
GROUP * ORDER	Sphericity Assumed	34.187	4	8.547	4.574	.018
	Greenhouse-Geisser	34.187	3.697	9.248	4.574	.021
	Huynh-Feldt	34.187	4.000	8.547	4.574	.018
	Lower-bound	34.187	2.000	17.093	4.574	.062
TIME * GROUP	Sphericity Assumed	2.894	6	.482	.303	.931
	Greenhouse-Geisser	2.894	3.008	.962	.303	.823
	Huynh-Feldt	2.894	6.000	.482	.303	.931
	Lower-bound	2.894	1.000	2.894	.303	.602
TIME * GROUP * ORDER	Sphericity Assumed	14.686	12	1.224	.769	.677
	Greenhouse-Geisser	14.686	6.017	2.441	.769	.605
	Huynh-Feldt	14.686	12.000	1.224	.769	.677
	Lower-bound	14.686	2.000	7.343	.769	.504

Pairwise Comparisons

Measure: **TIME**

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.(a)	95% Confidence Interval for Difference(a)	
					Lower Bound	Upper Bound
1	2	-1.630(*)	.370	.027	-3.060	-.199
	3	-2.322(*)	.580	.043	-4.564	-.080
	4	-2.530(*)	.581	.029	-4.774	-.286
2	1	1.630(*)	.370	.027	.199	3.060
	3	-.693	.362	.623	-2.089	.704
	4	-.900	.574	1.000	-3.119	1.319
3	1	2.322(*)	.580	.043	.080	4.564
	2	.693	.362	.623	-.704	2.089
	4	-.207	.340	1.000	-1.522	1.107
4	1	2.530(*)	.581	.029	.286	4.774
	2	.900	.574	1.000	-1.319	3.119
	3	.207	.340	1.000	-1.107	1.522

Based on estimated marginal means

* The mean difference is significant at the .05 level.

a Adjustment for multiple comparisons: Bonferroni.

Preparedness for Exercise – Question 3 – Pain in Legs

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
TIME	Sphericity Assumed	37.972	3	12.657	5.451	.008
	Greenhouse-Geisser	37.972	1.167	32.536	5.451	.049
	Huynh-Feldt	37.972	1.760	21.580	5.451	.026
	Lower-bound	37.972	1.000	37.972	5.451	.058
TIME * ORDER	Sphericity Assumed	1.865	6	.311	.134	.990
	Greenhouse-Geisser	1.865	2.334	.799	.134	.903
	Huynh-Feldt	1.865	3.519	.530	.134	.955
	Lower-bound	1.865	2.000	.933	.134	.877
GROUP	Sphericity Assumed	7.456	2	3.728	3.622	.059
	Greenhouse-Geisser	7.456	1.240	6.014	3.622	.092
	Huynh-Feldt	7.456	1.924	3.876	3.622	.061
	Lower-bound	7.456	1.000	7.456	3.622	.106
GROUP * ORDER	Sphericity Assumed	3.133	4	.783	.761	.570
	Greenhouse-Geisser	3.133	2.479	1.264	.761	.527
	Huynh-Feldt	3.133	3.847	.814	.761	.567
	Lower-bound	3.133	2.000	1.566	.761	.508
TIME * GROUP	Sphericity Assumed	1.307	6	.218	.494	.809
	Greenhouse-Geisser	1.307	1.962	.666	.494	.619
	Huynh-Feldt	1.307	3.877	.337	.494	.735
	Lower-bound	1.307	1.000	1.307	.494	.509
TIME * GROUP * ORDER	Sphericity Assumed	3.076	12	.256	.581	.843
	Greenhouse-Geisser	3.076	3.924	.784	.581	.680
	Huynh-Feldt	3.076	7.754	.397	.581	.779
	Lower-bound	3.076	2.000	1.538	.581	.588

Pairwise Comparisons

Measure: **TIME**

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.(a)	95% Confidence Interval for Difference(a)	
					Lower Bound	Upper Bound
1	2	-.604	.282	.458	-1.694	.487
	3	-1.293	.527	.297	-3.328	.742
	4	-1.504	.639	.340	-3.970	.963
2	1	.604	.282	.458	-.487	1.694
	3	-.689	.286	.317	-1.795	.417
	4	-.900	.379	.330	-2.362	.562
3	1	1.293	.527	.297	-.742	3.328
	2	.689	.286	.317	-.417	1.795
	4	-.211	.205	1.000	-1.001	.579
4	1	1.504	.639	.340	-.963	3.970
	2	.900	.379	.330	-.562	2.362
	3	.211	.205	1.000	-.579	1.001

Based on estimated marginal means

a Adjustment for multiple comparisons: Bonferroni.

Preparedness for Exercise – Question 4 – Ability to Complete ADL's

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
TIME	Sphericity Assumed	65.587	3	21.862	22.492	.000
	Greenhouse-Geisser	65.587	1.344	48.786	22.492	.001
	Huynh-Feldt	65.587	2.169	30.234	22.492	.000
	Lower-bound	65.587	1.000	65.587	22.492	.003
TIME * ORDER	Sphericity Assumed	20.590	6	3.432	3.530	.017
	Greenhouse-Geisser	20.590	2.689	7.658	3.530	.071
	Huynh-Feldt	20.590	4.339	4.746	3.530	.035
	Lower-bound	20.590	2.000	10.295	3.530	.097
GROUP	Sphericity Assumed	.036	2	.018	.005	.995
	Greenhouse-Geisser	.036	1.796	.020	.005	.992
	Huynh-Feldt	.036	2.000	.018	.005	.995
	Lower-bound	.036	1.000	.036	.005	.946
GROUP * ORDER	Sphericity Assumed	4.299	4	1.075	.302	.871
	Greenhouse-Geisser	4.299	3.592	1.197	.302	.854
	Huynh-Feldt	4.299	4.000	1.075	.302	.871
	Lower-bound	4.299	2.000	2.149	.302	.750
TIME * GROUP	Sphericity Assumed	3.866	6	.644	1.115	.373
	Greenhouse-Geisser	3.866	3.172	1.219	1.115	.370
	Huynh-Feldt	3.866	6.000	.644	1.115	.373
	Lower-bound	3.866	1.000	3.866	1.115	.332
TIME * GROUP * ORDER	Sphericity Assumed	6.649	12	.554	.959	.503
	Greenhouse-Geisser	6.649	6.344	1.048	.959	.481
	Huynh-Feldt	6.649	12.000	.554	.959	.503
	Lower-bound	6.649	2.000	3.324	.959	.435

Pairwise Comparisons

Measure: **TIME**

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.(a)	95% Confidence Interval for Difference(a)	
					Lower Bound	Upper Bound
1	2	-.970(*)	.172	.008	-1.633	-.308
	3	-1.730(*)	.136	.000	-2.256	-1.203
	4	-2.011(*)	.329	.005	-3.281	-.741
2	1	.970(*)	.172	.008	.308	1.633
	3	-.759(*)	.109	.003	-1.179	-.340
	4	-1.041	.399	.241	-2.581	.499
3	1	1.730(*)	.136	.000	1.203	2.256
	2	.759(*)	.109	.003	.340	1.179
	4	-.281	.324	1.000	-1.534	.971
4	1	2.011(*)	.329	.005	.741	3.281
	2	1.041	.399	.241	-.499	2.581
	3	.281	.324	1.000	-.971	1.534

Based on estimated marginal means

* The mean difference is significant at the .05 level.

a Adjustment for multiple comparisons: Bonferroni.

Preparedness for Exercise – Question 5 – Effectiveness of Treatment

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
TIME	Sphericity Assumed	3.897	2	1.948	3.036	.086
	Greenhouse-Geisser	3.897	1.163	3.351	3.036	.123
	Huynh-Feldt	3.897	1.750	2.227	3.036	.096
	Lower-bound	3.897	1.000	3.897	3.036	.132
TIME * ORDER	Sphericity Assumed	.268	4	.067	.104	.979
	Greenhouse-Geisser	.268	2.326	.115	.104	.925
	Huynh-Feldt	.268	3.500	.077	.104	.969
	Lower-bound	.268	2.000	.134	.104	.902
GROUP	Sphericity Assumed	138.282	2	69.141	9.860	.003
	Greenhouse-Geisser	138.282	1.911	72.379	9.860	.003
	Huynh-Feldt	138.282	2.000	69.141	9.860	.003
	Lower-bound	138.282	1.000	138.282	9.860	.020
GROUP * ORDER	Sphericity Assumed	52.789	4	13.197	1.882	.178
	Greenhouse-Geisser	52.789	3.821	13.816	1.882	.183
	Huynh-Feldt	52.789	4.000	13.197	1.882	.178
	Lower-bound	52.789	2.000	26.395	1.882	.232
TIME * GROUP	Sphericity Assumed	2.836	4	.709	1.141	.361
	Greenhouse-Geisser	2.836	2.566	1.105	1.141	.357
	Huynh-Feldt	2.836	4.000	.709	1.141	.361
	Lower-bound	2.836	1.000	2.836	1.141	.327
TIME * GROUP * ORDER	Sphericity Assumed	5.157	8	.645	1.037	.437
	Greenhouse-Geisser	5.157	5.131	1.005	1.037	.432
	Huynh-Feldt	5.157	8.000	.645	1.037	.437
	Lower-bound	5.157	2.000	2.579	1.037	.410

Pairwise Comparisons

Measure: **GROUP**

(I) GROUP	(J) GROUP	Mean Difference (I-J)	Std. Error	Sig.(a)	95% Confidence Interval for Difference(a)	
					Lower Bound	Upper Bound
1	2	1.181	.648	.354	-.947	3.310
	3	3.167(*)	.783	.020	.591	5.742
2	1	-1.181	.648	.354	-3.310	.947
	3	1.985	.725	.101	-.397	4.368
3	1	-3.167(*)	.783	.020	-5.742	-.591
	2	-1.985	.725	.101	-4.368	.397

Based on estimated marginal means

* The mean difference is significant at the .05 level.

a Adjustment for multiple comparisons: Bonferroni.

Pairwise Comparisons

Measure: **TIME**

(I) TIME	(J) TIME	Mean Difference (I-J)	Std. Error	Sig.(a)	95% Confidence Interval for Difference(a)	
					Lower Bound	Upper Bound
1	2	.537(*)	.091	.003	.237	.837
	3	.256	.275	1.000	-.647	1.158
2	1	-.537(*)	.091	.003	-.837	-.237
	3	-.281	.243	.870	-1.079	.516
3	1	-.256	.275	1.000	-1.158	.647
	2	.281	.243	.870	-.516	1.079

Based on estimated marginal means

* The mean difference is significant at the .05 level.

a Adjustment for multiple comparisons: Bonferroni.

Correlations – IL-6 to Leukocytes - Cryotherapy

		IL6A0	IL6A1	IL6A2	LEUKA0	LEUKA1	LEUKA2
IL6A0	Pearson Correlation	1	.942(**)	.257	.218	.153	-.247
	Sig. (2-tailed)	.	.000	.195	.274	.445	.214
	N	27	27	27	27	27	27
IL6A1	Pearson Correlation	.942(**)	1	.353	.152	.230	-.214
	Sig. (2-tailed)	.000	.	.071	.449	.248	.285
	N	27	27	27	27	27	27
IL6A2	Pearson Correlation	.257	.353	1	.023	-.058	-.195
	Sig. (2-tailed)	.195	.071	.	.910	.772	.331
	N	27	27	27	27	27	27
LEUKA0	Pearson Correlation	.218	.152	.023	1	.400(*)	.266
	Sig. (2-tailed)	.274	.449	.910	.	.039	.180
	N	27	27	27	27	27	27
LEUKA1	Pearson Correlation	.153	.230	-.058	.400(*)	1	.518(**)
	Sig. (2-tailed)	.445	.248	.772	.039	.	.006
	N	27	27	27	27	27	27
LEUKA2	Pearson Correlation	-.247	-.214	-.195	.266	.518(**)	1
	Sig. (2-tailed)	.214	.285	.331	.180	.006	.
	N	27	27	27	27	27	27

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

Correlations – IL-6 to Leukocytes – Active

		IL6B0	IL6B1	IL6B2	LEUKB0	LEUKB1	LEUKB2
IL6B0	Pearson Correlation	1	.577	.413	-.445	-.118	-.629
	Sig. (2-tailed)	.	.104	.269	.230	.762	.069
	N	9	9	9	9	9	9
IL6B1	Pearson Correlation	.577	1	.634	-.089	.433	-.443
	Sig. (2-tailed)	.104	.	.067	.821	.244	.232
	N	9	9	9	9	9	9
IL6B2	Pearson Correlation	.413	.634	1	-.054	.127	-.272
	Sig. (2-tailed)	.269	.067	.	.890	.745	.479
	N	9	9	9	9	9	9
LEUKB0	Pearson Correlation	-.445	-.089	-.054	1	.715(*)	.805(**)
	Sig. (2-tailed)	.230	.821	.890	.	.031	.009
	N	9	9	9	9	9	9
LEUKB1	Pearson Correlation	-.118	.433	.127	.715(*)	1	.446
	Sig. (2-tailed)	.762	.244	.745	.031	.	.228
	N	9	9	9	9	9	9
LEUKB2	Pearson Correlation	-.629	-.443	-.272	.805(**)	.446	1
	Sig. (2-tailed)	.069	.232	.479	.009	.228	.
	N	9	9	9	9	9	9

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

Correlations – IL-6 to Leukocytes - Passive

		IL6C0	IL6C1	IL6C2	LEUKC0	LEUKC1	LEUKC2
IL6C0	Pearson Correlation	1	.993(**)	.811(**)	.323	.191	-.360
	Sig. (2-tailed)	.	.000	.008	.397	.623	.341
	N	9	9	9	9	9	9
IL6C1	Pearson Correlation	.993(**)	1	.856(**)	.286	.152	-.409
	Sig. (2-tailed)	.000	.	.003	.456	.696	.274
	N	9	9	9	9	9	9
IL6C2	Pearson Correlation	.811(**)	.856(**)	1	.078	-.015	-.679(*)
	Sig. (2-tailed)	.008	.003	.	.841	.970	.044
	N	9	9	9	9	9	9
LEUKC0	Pearson Correlation	.323	.286	.078	1	.757(*)	.579
	Sig. (2-tailed)	.397	.456	.841	.	.018	.102
	N	9	9	9	9	9	9
LEUKC1	Pearson Correlation	.191	.152	-.015	.757(*)	1	.473
	Sig. (2-tailed)	.623	.696	.970	.018	.	.198
	N	9	9	9	9	9	9
LEUKC2	Pearson Correlation	-.360	-.409	-.679(*)	.579	.473	1
	Sig. (2-tailed)	.341	.274	.044	.102	.198	.
	N	9	9	9	9	9	9

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

Correlations – IL-6 to Lymphocytes – Cryotherapy

		LYMA0	LYMA1	LYMA2	IL6A0	IL6A1	IL6A2
LYMA0	Pearson Correlation	1	.762(**)	.388(*)	.095	.220	-.017
	Sig. (2-tailed)	.	.000	.045	.638	.271	.934
	N	27	27	27	27	27	27
LYMA1	Pearson Correlation	.762(**)	1	.189	.043	.194	-.025
	Sig. (2-tailed)	.000	.	.346	.833	.332	.901
	N	27	27	27	27	27	27
LYMA2	Pearson Correlation	.388(*)	.189	1	-.135	-.051	.143
	Sig. (2-tailed)	.045	.346	.	.504	.801	.478
	N	27	27	27	27	27	27
IL6A0	Pearson Correlation	.095	.043	-.135	1	.942(**)	.257
	Sig. (2-tailed)	.638	.833	.504	.	.000	.195
	N	27	27	27	27	27	27
IL6A1	Pearson Correlation	.220	.194	-.051	.942(**)	1	.353
	Sig. (2-tailed)	.271	.332	.801	.000	.	.071
	N	27	27	27	27	27	27
IL6A2	Pearson Correlation	-.017	-.025	.143	.257	.353	1
	Sig. (2-tailed)	.934	.901	.478	.195	.071	.
	N	27	27	27	27	27	27

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

Correlations – IL-6 to Lymphocytes – Active

		IL6B0	IL6B1	IL6B2	LYMB0	LYMB1	LYMB2
IL6B0	Pearson Correlation	1	.577	.413	-.204	-.101	-.142
	Sig. (2-tailed)	.	.104	.269	.598	.796	.716
	N	9	9	9	9	9	9
IL6B1	Pearson Correlation	.577	1	.634	.443	.564	-.014
	Sig. (2-tailed)	.104	.	.067	.233	.114	.971
	N	9	9	9	9	9	9
IL6B2	Pearson Correlation	.413	.634	1	.490	.304	.348
	Sig. (2-tailed)	.269	.067	.	.181	.427	.359
	N	9	9	9	9	9	9
LYMB0	Pearson Correlation	-.204	.443	.490	1	.862(**)	.016
	Sig. (2-tailed)	.598	.233	.181	.	.003	.967
	N	9	9	9	9	9	9
LYMB1	Pearson Correlation	-.101	.564	.304	.862(**)	1	.118
	Sig. (2-tailed)	.796	.114	.427	.003	.	.762
	N	9	9	9	9	9	9
LYMB2	Pearson Correlation	-.142	-.014	.348	.016	.118	1
	Sig. (2-tailed)	.716	.971	.359	.967	.762	.
	N	9	9	9	9	9	9

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

Correlations – IL-6 to Lymphocytes - Passive

		IL6C0	IL6C1	IL6C2	LYMC0	LYMC1	LYMC2
IL6C0	Pearson Correlation	1	.993(**)	.811(**)	.052	.008	-.320
	Sig. (2-tailed)	.	.000	.008	.895	.983	.401
	N	9	9	9	9	9	9
IL6C1	Pearson Correlation	.993(**)	1	.856(**)	.082	.005	-.260
	Sig. (2-tailed)	.000	.	.003	.833	.989	.500
	N	9	9	9	9	9	9
IL6C2	Pearson Correlation	.811(**)	.856(**)	1	.144	.006	-.050
	Sig. (2-tailed)	.008	.003	.	.713	.987	.898
	N	9	9	9	9	9	9
LYMC0	Pearson Correlation	.052	.082	.144	1	.824(**)	.837(**)
	Sig. (2-tailed)	.895	.833	.713	.	.006	.005
	N	9	9	9	9	9	9
LYMC1	Pearson Correlation	.008	.005	.006	.824(**)	1	.619
	Sig. (2-tailed)	.983	.989	.987	.006	.	.076
	N	9	9	9	9	9	9
LYMC2	Pearson Correlation	-.320	-.260	-.050	.837(**)	.619	1
	Sig. (2-tailed)	.401	.500	.898	.005	.076	.
	N	9	9	9	9	9	9

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

Correlations – IL-6 to Neutrophils - Cryotherapy

		NPA0	NPA1	NPA2	IL6A0	IL6A1	IL6A2
NPA0	Pearson Correlation	1	.882(**)	.397(*)	.156	.130	.016
	Sig. (2-tailed)	.	.000	.041	.436	.516	.939
	N	27	27	27	27	27	27
NPA1	Pearson Correlation	.882(**)	1	.525(**)	.215	.195	-.063
	Sig. (2-tailed)	.000	.	.005	.283	.331	.753
	N	27	27	27	27	27	27
NPA2	Pearson Correlation	.397(*)	.525(**)	1	-.235	-.182	-.169
	Sig. (2-tailed)	.041	.005	.	.237	.364	.400
	N	27	27	27	27	27	27
IL6A0	Pearson Correlation	.156	.215	-.235	1	.942(**)	.257
	Sig. (2-tailed)	.436	.283	.237	.	.000	.195
	N	27	27	27	27	27	27
IL6A1	Pearson Correlation	.130	.195	-.182	.942(**)	1	.353
	Sig. (2-tailed)	.516	.331	.364	.000	.	.071
	N	27	27	27	27	27	27
IL6A2	Pearson Correlation	.016	-.063	-.169	.257	.353	1
	Sig. (2-tailed)	.939	.753	.400	.195	.071	.
	N	27	27	27	27	27	27

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

Correlations – IL-6 to Neutrophils – Active Recovery

		NPB0	NPB1	NPB2	IL6B0	IL6B1	IL6B2
NPB0	Pearson Correlation	1	.844(**)	.673(*)	-.463	-.300	-.443
	Sig. (2-tailed)	.	.004	.047	.209	.434	.233
	N	9	9	9	9	9	9
NPB1	Pearson Correlation	.844(**)	1	.582	-.139	.073	-.370
	Sig. (2-tailed)	.004	.	.100	.721	.852	.328
	N	9	9	9	9	9	9
NPB2	Pearson Correlation	.673(*)	.582	1	-.606	-.257	-.175
	Sig. (2-tailed)	.047	.100	.	.084	.504	.652
	N	9	9	9	9	9	9
IL6B0	Pearson Correlation	-.463	-.139	-.606	1	.577	.413
	Sig. (2-tailed)	.209	.721	.084	.	.104	.269
	N	9	9	9	9	9	9
IL6B1	Pearson Correlation	-.300	.073	-.257	.577	1	.634
	Sig. (2-tailed)	.434	.852	.504	.104	.	.067
	N	9	9	9	9	9	9
IL6B2	Pearson Correlation	-.443	-.370	-.175	.413	.634	1
	Sig. (2-tailed)	.233	.328	.652	.269	.067	.
	N	9	9	9	9	9	9

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

Correlations – IL-6 to Neutrophils – Passive Recovery

		NPC0	NPC1	NPC2	IL6C0	IL6C1	IL6C2
NPC0	Pearson Correlation	1	.921(**)	.644	.264	.218	.033
	Sig. (2-tailed)	.	.000	.061	.492	.573	.933
	N	9	9	9	9	9	9
NPC1	Pearson Correlation	.921(**)	1	.694(*)	.282	.224	-.034
	Sig. (2-tailed)	.000	.	.038	.462	.562	.931
	N	9	9	9	9	9	9
NPC2	Pearson Correlation	.644	.694(*)	1	-.300	-.366	-.665
	Sig. (2-tailed)	.061	.038	.	.434	.333	.051
	N	9	9	9	9	9	9
IL6C0	Pearson Correlation	.264	.282	-.300	1	.993(**)	.811(**)
	Sig. (2-tailed)	.492	.462	.434	.	.000	.008
	N	9	9	9	9	9	9
IL6C1	Pearson Correlation	.218	.224	-.366	.993(**)	1	.856(**)
	Sig. (2-tailed)	.573	.562	.333	.000	.	.003
	N	9	9	9	9	9	9
IL6C2	Pearson Correlation	.033	-.034	-.665	.811(**)	.856(**)	1
	Sig. (2-tailed)	.933	.931	.051	.008	.003	.
	N	9	9	9	9	9	9

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

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

APPENDIX J

Psychological Tests

PREPAREDNESS FOR EXERCISE

VAS

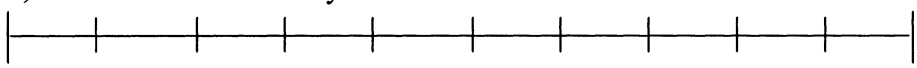
RPE
(Borg scale for ratings of perceived exertion)

REST, LIGHT EXERCISE OR CRYOTHERAPY: WHAT IS THE MOST EFFECTIVE WAY TO RECOVER BETWEEN REPEATED BOUTS OF INTENSE EXERCISE?

Date:_____ Subject ID:_____

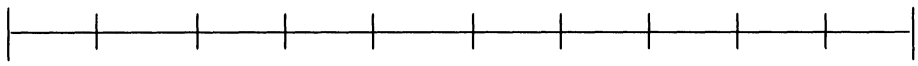
Preparedness for Exercise Scale
Hydrotherapy study

Please answer the following questions by placing a mark on the scale, following the question, that best describes how you feel **RIGHT NOW**.



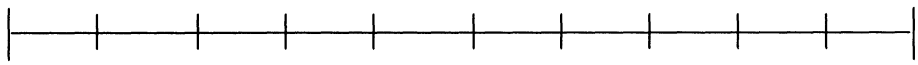
0 1 2 3 4 5 6 7 8 9 10
Not at all — a little — moderately — a lot — Extremely

1. Do you feel physically ABLE to exercise?



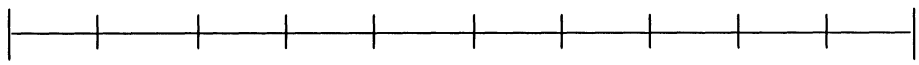
1 1 2 3 4 5 6 7 8 9 10
Not at all — a little — moderately — a lot — Extremely

2. How would you describe your legs?



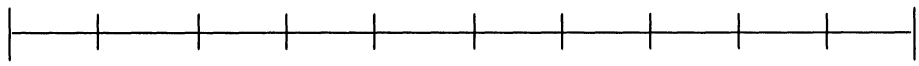
2 1 2 3 4 5 6 7 8 9 10
Energized — a little tired — Tired — Very Tired — "Dead"

3. Are you experiencing any PAIN in your legs related to exercise?



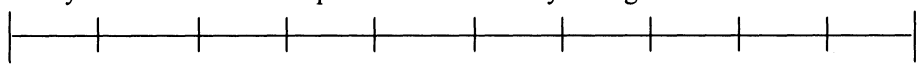
3 1 2 3 4 5 6 7 8 9 10
Not at all — a little — moderately — a lot — Extremely

4. Do you find it difficult to engage in your usual activities of daily living at this time?



4 1 2 3 4 5 6 7 8 9 10
Not at all — a little — moderately — a lot — Extremely

5. Do you find the treatment post-exercise makes your legs feel Better?



5 1 2 3 4 5 6 7 8 9 10
Not at all — a little — moderately — a lot — Extremely

VAS

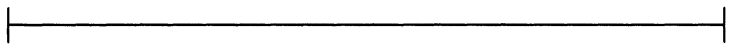
Date:_____ **Subject ID:**_____

Session #: _____
Bout #: _____

Visual Analogue Scale (VAS)
Pain

Please mark your perceived level of general pain on the line below.

NO Pain **Extreme Pain**



BORG – scale for ratings of perceived exertion

Date:_____ **Subject ID:**_____

Session #: _____

Bout #: _____

Borg - Rating of Perceived Exertion (RPE)

We want you to pay close attention to how hard you feel you're working during the exercise, and we will show this scale to you from time to time. This feeling should reflect your total amount of exertion and fatigue, combining all sensations and feelings of physical stress, effort and fatigue. Do not concern yourself with any one factor such as leg pain, shortness of breath or exercise intensity, but try to concentrate on your total feeling of exertion. . There are no right or wrong answers so be as accurate as you can.

.

6	
7	**Very, very light**
8	
9	**Very light**
10	
11	**Fairly light**
12	
13	**Somewhat hard**
14	
15	**Hard**
16	
17	**Very hard**
18	
19	**Very, very hard**
20	**Very, Very, Hard**