The effects of water immersion on the recovery and performance of competitive cyclists

by
Christa Magrieta Koekemoer

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Supervisor: Prof. Elmarie Terblanche
Department of Sport Science

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Declaration

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SUMMARY

Post-exercise recovery has become an important area in research due to the high demands placed on competitive athletes. Different recovery strategies are used by athletes during competition and training. For the competitive athlete it is important to maintain performances during competition and also to enhance performances during training. However, if the athlete fails to recovery from daily exhaustive training and competition, inadequate recovery may lead to poor performances, burn-out, sickness and even injuries. There is very little evidence available on the possible performance recovery effects of the use of water immersion during multi days of intensive endurance training. Theoretically, water immersion should aid the overall recovery process without any additional energy cost involved as with active recovery.

The objective of this investigation was to determine whether water immersion (cold water vs. neutral) has any effects on the post-exercise recovery rate of competitive cyclists during 3 days of intensive endurance training and whether recovery with water immersion is more effective than active recovery.

Seventeen competitive cyclists (mean ± SD age: 27.6 ± 5.94 years, weight: 78.8 ± 6.67 kg, height: 180.5 ± 4.42 cm VO_{2max}: 49.8 ± 4.13 $L.min^{-1}.kg^{-1}$, and PPO: 352.6 ± 35.94 Watts) completed 3 days of intensive endurance cycling sessions. Cyclists were randomly assigned to either a 20 minute ice bath (IB) (n = 6, 11 ± 0.9°C), neutral bath (NB) (n = 6, 30 ± 0.6°C), or active recovery (AR) (n = 5; 81 ± 1.74% of HR_{LT}) which were performed directly after the training sessions on Day 1 and 2. Dependent variables such as anaerobic performance, creatine kinase concentrations (CK), c-reactive protein concentrations (CRP), blood lactate concentrations, muscle soreness (VAS) and perceived fatigue (POMS), and limb circumferences were measured prior to the training sessions at Day 1, 2 and 3. In addition, changes in exercise performances over the last 2 days were also assessed.

There were significant increases over the three days in plasma [CK] ($P < 0.05$) and [CRP] ($P < 0.001$) demonstrating that muscle damage and inflammation occurred during and after the training sessions. However, there were no treatment or interaction effects observed for any of the dependent variables for any of the recovery interventions ($P > 0.05$). Blood [La] was significantly reduced on Day 2 for the IB group in comparison to the NB group ($P < 0.05$). A strong tendency was observed for [CK] when the IB and NB groups were combined (WG), indicating that AR had a strong tendency to enhance the recovery of [CK] in comparison to the WG ($P = 0.05$). Also, there were no significant time or interaction effects observed in % changes in performances for the last two 100km TTs between Day 2 and 3 for any of the recovery interventions ($P > 0.05$).

These findings suggest that neither cold water, nor neutral water therapy, have more beneficial effects on post-exercise recovery rates compared to active recovery. Importantly, however, is that the cyclists' were able to maintain their performances over the three consecutive days, indicating that water therapy *per se* is not detrimental to endurance performance.

OPSOMMING

Na-oefening herstel het ‘n belangrike area van navorsing geword, aangesien die eise wat aan elite atlete gestel word buitengewoon hoog is. Vir die kompeterende fietsryer is dit baie belangrik om prestasie tydens kompetitisie asook tydens inoefening te handhaaf. Inteendeel, as die atleet nie daarin slaag om effektief te herstel na daaglikse oefening en kompetisie nie, mag dit lei tot swak prestasie, uitbranding, siekte en beserings. Tot hede is daar geen baie min bewyse beskikbaar oor die potensiële voordele van waterterapie vir die herstel van atlete, veral tydens meervoudige dae van intensiewe uithouvermoë inoefening. Teoreties behoort waterterapie die algehele herstelproses bevorder sonder dat enige addisionele energiekostes betrokke is, soos in die geval van aktiewe herstel.

Die doel van die ondersoek was om vas te stel of waterterapie (koud teenoor neutraal) enige effekte het op die na-oefening hersteltempo van kompeterende fietsryers tydens 3 dae van intensiewe uithouvermoë oefening en om te bepaal of waterterapie meer effektief is as aktiewe herstel.

Sewentien kompeterende fietsryers (gemiddeld ± SD; ouderdom: 27.6 ± 5.94 jaar, gewig: 78.8 ± 6.67 kg, lengte: 180.5 ± 4.42 cm, VO_{2maks}: 49.8 ± 4.13 $L.min^{-1}.kg^{-1}$, en Piek krag uitset: 352.6 ± 35.94 Watts) het 3 dae van intensiewe uithouvermoë inoefeing voltooi. Die fietryers was lukraak ingedeel in ‘n 20 minute Ysbadgroep (IB) (n = 6, 11 ± 0.9^{o}C), neutrale bad groep (NB) (n = 6, 30 ± 0.6^{o}C) en ‘n aktiewe herstelgroep (AR) (n = 5; 81 ± 1.74% van HR_{LT}), Herstelsessies het op Dag 1 en 2 direk na die inoefeningsessies plaasgevind. Afhanklike veranderlikes soos funksionele kapasiteit, kreatienkinase konsentrasies (CK), c-reaktiewe proteïen konsentrasies (CRP), bloedlaktaat konsentrasie ([La]), spierseerheid en persepsie van vermoeienis (STEMS), en beenomtrekke was gemeet voor die inoefeningsessies op Dag 1, 2 en 3. Veranderinge in oefeningprestasie oor die laaste 2 dae was ook geassesseer.

Daar was ‘n statistiese betekenisvolle toename in plasma [CK] (P < 0.05) en [CRP] (P < 0.001) oor die drie dae, wat daarop wys dat spierskade en inflammasie wel plaasgevind het. Daar was geen behandeling of interaksie effekte waarneembaar vir enige van die intervensies nie (P > 0.05). Bloed [La] was beduidend verlaag op Dag 2 vir die IB groep in vergelyking met die NB groep (P = 0.05). Die verlaging in plasma [CK] na AR het gegrens aan statisties betekenisvolle resultate (P = 0.05) in vergelyking met die waterterapie (IB en NB gekombineer). Daar was geen statisites beduidende tyd of interaksie effekte waargeneem in die % veranderinge in oefeningprestasie vir die laaste twee 100km tydtoetse tussen Dag 2 en 3 vir enige van die herstelstrategieë nie (P < 0.05).

Die resultate wys dat waterterapie nie enige voordelige effekte op die na-inoefening herstel tempo het in vergelyking met aktiewe herstel nie. Dit is egter belangrik om daarop te let dat die fietsryers in staat was om hul oefeningprestasies te handhaaf oor die drie opeenvolgende dae, wat aandui dat waterterapie nie nadelig inwerk op uithouvermoë prestasie nie.

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LIST OF ABBREVIATIONS

A	:	area of water immersion
AR	:	active recovery
ATP	:	adenosine triphosphate
ANOVA	:	analysis of variance
AP:WT	:	average power relative to body weight
Bpm	:	beats per minute
Ca^{2+}	:	calcium
CK	:	creatine kinase
Cm	:	centimetre
CO	:	cardiac output
CRP	;	c-reactive protein
CVP	:	central venous pressure
CWT	:	contrast water therapy
CWI	:	cold water immersion
^{o}C	:	degree Celsius
DOMS	:	delayed onset of muscle soreness
EIMD	:	exercise-induced muscle damage
EMG	:	electromyography
EPOC	:	excess post-exercise oxygen consumption
ESF	;	electrostimulation
ES	:	effect sizes
g	:	gram
g	:	gravity

$g.kg^{-1}$	:	gram per kilogram
$g.kg^{-1}.h^{-1}$	:	gram per kilogram per hour
GLUT4	:	glucose transporter protein (4)
H^+	:	hydrogen ion
h	:	height of immersion
HWI	:	hot water immersion
hr:min:sec	:	hour(s):minute(s):second(s)
HR	:	heart rate
HR_{LT}	:	heart rate at lactate threshold
IB	:	ice bath
ISAK	:	international standards for advancement of kinanthopometry
$IU.L^{-1}$ or $u.L^{-1}$	:	units per liter
$J.kg^{-1}$	:	joule(s) per kilogram
kg	:	kilogram(s)
km	:	kilometre(s)
K_c	;	capillary coefficient
LT	:	lactate threshold
[La]	:	lactate concentration
La	:	lactate
LFF	:	low frequency fatigue
$Log_e[CK]$	:	log transformed creatine kinase concentration
m	:	mass immersed
Mb	:	myoglobin

MAP	:	mean arterial pressure
min	:	minute(s)
min:sec	:	minute(s) and second(s)
MVCF	:	maximum voluntary contraction force
MVC	:	maximum voluntary contraction
n	:	number of subjects
NB	:	neutral bath
PAS	:	passive recovery
PC	:	creatine phosphate
pH	:	hydrogen ion concentration
PPO	:	peak power output (W)
PO	:	power output
P_c	:	hydrostatic pressure in the capillary
Pi	:	hydrostatic pressure in the interstitial fluid
ρ	:	water density
r	:	correlation coefficient
Q	:	cardiac output
$rad.s^{-1}$	:	radius per second
RBE	:	repeated bout effect
RM	:	repetition maximum
ROM	:	range of motion
RPE	:	ratings of perceived exertion
SD	:	standard deviation
sec	:	seconds

SEM	:	standard error of meausurement
SR	:	sarcoplasmic reticulum
STEMS	:	Stellenbosch mood scale
SV	:	stroke volume
TPR	:	total peripheral resistance
TT(s)	:	time-trial(s)
TW:WT	:	total work performed relative to body weight
VAS	:	visual analog scale (mm)
VO_{2max}	:	maximum oxygen consumption ($L.min^{-1}.kg^{-1}$)
% VO_{2max}	:	fractional utilization of oxygen (%)
vs.	:	versus
V	:	immersed volume
VE	:	minute ventilation ($L.min^{-1}$)
W	:	watt(s)
~	:	about
π_c	:	osmotic pressure
π_i	:	osmotic pressure in interstitial fluid
+	:	increase
-	:	decrease

CONVERSIONS

1 mile	:	1.16 kilometers
0 ^oC	:	32 Fahrenheit

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CHAPTER ONE

INTRODUCTION

Daily intensive training and competition increases the physical and physiological stressors on athletes. Failure to recover from these daily stressors may lead to poor performances, ill health and psychological staleness (Budgett, 1998; Fry *et al.,* 1991). Optimal recovery during training and competition will help the athlete to maintain subsequent performances during competition, as well as to performance improvements from training. Also, the athlete who can recover faster will also have a competitive advantage during actual competitions, specifically if these competitions stretch over multiple days. Today, athletes use a variety of recovery strategies with the aim to enhance the overall recovery process following exercise. The most popular strategies include; massage, active recovery, compression garments, water immersion (cold and alternating hot and cold), stretching, ultra-sound, hyperbaric oxygen therapy, and treatment with pharmacological agents such as nonsteriodal anti-inflammatory drugs (Hing *et al.,* 2008; Barnett, 2006; Wilcock *et al.,* 2006[a]; Cochrane, 2004; Cheung *et al.,* 2003). The strategies that are of interest in this study are water immersion (cold and thermo-neutral temperatures) and active recovery (AR).

The enhanced lactate and metabolite clearance effect of active recovery is thought to be elicited by the increased blood flow to the working muscles (Barnett, 2006; Thiriet *et al.,* 1993). The durations of active recovery usually ranges from 5 – 20 minutes at intensities lower than the lactate threshold (Monedero & Donne, 2000; Thiriet *et al.,* 1993). Most of the research using active recovery has focussed on the effects of blood lactate clearance as well as subsequent performances linked to lactate clearance (Wilcock *et al.,* 2006[a]; Coffey *et al.,* 2004; Hamlin, 2007). Although it has been shown that active recovery results in reduced lactate concentrations, the effects on subsequent performance is unknown (Wilcock *et al.,* 2006[ab]; Coffey *et al.,* 2004; Thiriet *et al.,* 1993) and needs further investigation.

Water immersion has been extensively studied in numerous investigations ranging from physiological responses during immersion (Krasney & Pendergast, 2008; Yun *et al.,* 2004; Gabrielsen *et al.,* 2002; Poyhonen & Avela, 2002; Pump *et al.,* 2001; Sramek *et al.,* 2000; Gabrielsen *et al.,* 2000; Park *et al.,* 1999; Johansen *et al.,* 1997; Johansen *et al.,* 1992; Bonde-Petersen *et al.,* 1992; Lollgen *et al.,* 1981; Craig & Dvorak, 1966) to the effects on

delayed onset muscle soreness resulting from exercise induced muscle damage (Ingram *et al.,* 2009; French *et al.,* 2008; Goodall & Howatson 2008; Vaile *et al.,* 2008;

Vaile *et al.,* 2007; Bailey *et al.,* 2007; Sellwood *et al.,* 2007; Skurvydas *et al.,* 2006; Eston & Peters, 1999; Kuligowski *et al.,* 1998; Isabell *et al.,* 1992) and on post-exercise recovery following high intensity exercises (Morton, 2007; Hamlin, 2007; Wilcock *et al.,* 2006[a]; Coffey *et al.,* 2004; Nakamura *et al.,* 1996). Water immersion can be divided into four basic strategies, namely (i) cold water immersion (CWI; ≤ 15°C), (ii) hot water immersion or thermotherapy (HWI; > 36°C), (iii) thermo-neutral water immersion (~35°C), and (iv) contrast water immersion (alternating hot and cold temperatures).

The possibility that water immersion *per se* may enhance the overall recovery process is thought to be largely due to the hydrostatic effects exerted upon the immersed body (Wilcock *et al.,* 2006[a]). The hydrostatic effect of water immersion is related to the depth of the immersed body, and the greatest hydrostatic pressure is achieved by the immersion up to the level of the neck (Wilcock *et al.,* 2006[a]; Bove, 2002; Johansen *et al.,* 1997; Arborelius *et al.,* 1972). The resultant enhanced fluid shifts from the periphery to the central cavity increases stroke volume and cardiac output, which increases blood flow (Sramek *et al.,* 2000; Kwon *et al.,* 1999; Johansen *et al.,* 1997; Epstein 1992). The increase in total blood flow is accompanied by a decrease in the total peripheral resistance (Yun *et al.,* 2004; Park *et al.,* 1999; Echt *et al.,* 1974; Arborelius *et al.,* 1972). Therefore, the clearance of metabolic waste products, i.e. lactate, could be enhanced without any extra energy cost involved (Wilcock *et al.,* 2006[a]; Nakamura *et al.,* 1996). The increased recycling of metabolites will enable the body to recover faster and the body would also be able to replenish energy stores effectively.

Most of the studies using water immersion, have focussed on the physiological manifestations and effects from a single bout of high intensity exercise (Vaile *et al.,* 2008; French *et al.,* 2008;Goodall & Howatson, 2008; Sellwood *et al.,* 2007; Bailey *et al.,* 2007; Vaile *et al.,* 2007; Morton, 2007; Skurvydas *et al.,* 2006; Howatson *et al.,* 2005; Eston & Peters, 1999; Kuligowski *et al.,* 1998; Nakamura *et al.,*1996), while only a few studies have focussed on exercise performances (Ingram *et al.,* 2009; Montgomery *et al.,* 2008; Hamlin, 2007; Coffey *et al.,* 2004; Lane & Wegner, 2004). However, the findings of these studies are conflicting and it is therefore unclear whether the effects of water immersion on performance, if any, is related to the temperature effects of the water, or rather due to the hydrostatic pressure resulting from the water. Therefore, the aim of the current study is to eliminate the possible physiological effects of hydrostatic pressure during water immersion and rather focus on the temperature effects on performance recovery.

One of the first studies to examine the effects of recovery strategies on the cumulative fatigue resulting from more than two exercise bouts were done by Montgomery *et al*. (2008). They investigated the physical performance and cumulative fatigue during a 3 day basketball competition and concluded that CWI was better than carbohydrate replenishment and stretching routines. A more recent investigation by Rowsell *et al.* (2009) studied the efficacy of water immersion of two different temperatures (cold vs. thermo-neutral) on the physical as well as on recovery markers of junior soccer players over the course of four days of simulated competition. They concluded that cold water immersion does not affect physical performance or inflammation and muscle damage; however, it seemed that CWI managed to reduce the perception of soreness and fatigue between the matches. It was concluded that further research is needed to confirm their findings. More importantly, there are no other investigations done so far on the effects of recovery strategies during simulated cycling competition over three days. Cycling is a popular sport and competition usually comprises of single day stage races (i.e. Argus Cycle Tour), multi-day stage races (i.e. Cape Epic MTB race), and 3 week tour races (i.e Tour de France). The variation in terrain and distances (i.e. hilly routes, flat routes, time-trials, short stages, long stages etc.) require different performance attributes, but in all cases the cyclists' muscular, neuromuscular, metabolic, and nervous systems are pushed to exhaustion during both training and competition (Abbiss & Laursen, 2005).

The aim of the study is therefore to determine the effects of water immersion and active recovery strategies on the physical, physiological and psychological recovery of competitive cyclists' during three days of high intensity cycling. It is important to establish whether water immersion recovery strategies are more effective than active recovery (Wilcock *et al*. 2006[ab]) and also whether cold water immersion causes faster recovery rates than thermo-neutral water immersions.

In chapter two, the physiology of water immersion and the possible influence on recovery are discussed. In chapter three, the physiological responses during post-exercise recovery are outlined and in chapter four the literature regarding the effects of water immersion on recovery and performance will be reviewed.

It is hypothesised that water immersion would have more beneficial post-exercise recovery effects than AR, enhancing post-exercise recovery without any additional energy cost as in the case of AR. Water immersion will therefore enhance or maintain subsequent cycling performances over the course of the three days. This study will help us to better understand the effects of water immersion of different temperatures on the physiological, psychological and functional aspects of recovery and performance in the competitive cyclist. The fact that

recovery modalities can be used during competitions or as a daily recovery routine to maintain performance and enhance recovery may be further explored by this study, to determine if these strategies are worth the time and effort. In addition, this study could provide a framework for further investigations on the efficacy of water immersion and active recovery on performance recovery.

CHAPTER TWO

THE PHYSIOLOGY OF WATER IMMERSION

A. INTRODUCTION

The physiological effects of water immersion have been extensively studied in human subjects (Wilcock *et al.,* 2006[a]). Studies using thermo-neutral water immersion focussed specifically on hemodynamic, cardiovascular, neuroendocrine, renal and neuromuscular responses, as well as thermal regulation (Krasney & Pendergast, 2008; Yun *et al.,* 2004; Gabrielsen *et al.,* 2002; Poyhonen & Avela, 2002; Pump *et al.,* 2001; Sramek *et al.,* 2000; Gabrielsen *et al.,* 2000; Park *et al.,* 1999; Johansen *et al.,* 1997; Johansen *et al.,* 1992; Bonde-Petersen *et al.,* 1992; Löllgen *et al.,* 1981; Craig & Dvorak, 1966). Studies on cold water immersion (cryotherapy) focussed on its role in the treatment of inflammation in musculotendinious injuries (Deal *et al.,* 2002; Coté *et al.,* 1988; Barnes, 1979). Recently water immersion became more popular as a recovery modality after exercise (Ingram *et al.,* 2009; Montgomery *et al.,* 2008; French *et al.,* 2008; Vaile *et al.,* 2008; Howatson & van Someren *et al.,* 2008; Bailey *et al.,* 2007; Skurvydas *et al.,* 2006; Yanagisawa *et al.,* 2003; Howatson & van Someren, 2003; Eston & Peters, 1999). These recovery strategies include hot water immersion, cold water immersion, as well as contrast water immersion (combinations of hot and cold water immersions). The two mechanisms which mainly contribute to the physiological responses mediated by water immersion *per se* are (i) the hydrostatic pressure and (ii) the temperature of the water. In this chapter the different water immersion modalities will be discussed together with the physiology and the possible mechanisms that may contribute to the recovery of athletes.

B. THERMOTHERAPY

Thermotherapy has been practiced for many years. The Greeks used thermotherapy and other forms of spa therapy as part of their cleansing rituals and also before sporting events as part of their preparation (Tubergen & van der Linden 2002).

Thermotherapy involves the immersion of the body, or parts of the body, into hot water (> 36 ºC) with the aim to raise the core body temperature (Bonde-Petersen *et al.,* 1992). Immersion time usually ranges from 10-20 minutes. Other forms of thermotherapy or heat therapy include the use of heatwraps, spa and whirlpool baths. Thermotherapy is widely used in the rehabilitation of muscle and soft tissue injuries and in the treatment of

musculoskeletal disorders (Henricson *et al.,* 1984; Magness *et al.,* 1970) such as ankle injuries, muscle injuries and lower back pain (Nadler *et al.,* 2003; Coté *et al.,* 1998).

Studies on the effects of thermotherapy have focussed on pain relief (Nadler *et al.,* 2003), flexibility and muscle length (Funk *et al.,* 2001; Burke at al., 2001; Taylor *et al.,* 1995; Henrichson *et al.,* 1984), hemodynamic changes (Katoaka & Yoshida, 2005; Chaukraoun and Varene 1990; Weston *et al.,* 1987; Craig & Duarak 1966), swelling (Coté *et al.,* 1998), blood flow (Bonde-Petersen *et al.,* 1992; Knight and Londeree, 1980), the recovery from intense physical exercises (Viitasalo *et al.,* 1995; Clarke *et al.,* 1963), muscle strength (Burke *et al.,* 2000), and signs and symptoms of delayed-onset muscle soreness (Vaile at al., 2007; Kuligowski *et al.,* 1998).

1. Blood flow

Hot water immersion results in a rise in superficial temperature and an increase in cutaneous blood flow. The vasoconstrictor tone is lowered via the increase in skin temperature and the decrease in core temperature. This causes cutaneous vasodilation of the blood capillaries and enhanced blood flow to the skin (Bonde-Petersen *et al.,* 1992). However, increases in skin temperatures appear to be only present in the cutaneous and subcutaneous tissue layers, while tissue such as skeletal muscle (at depths greater than 2cm) are unaffected (Myrer *et al.,* 1997; Bonde-Petersen *et al.,* 1992; Wyper *et al.,* 1976). However, those tissues affected by the rise in temperature and blood flow may have enhanced cellular metabolism, waste removal and nutrient delivery, and this may aid the recovery process of injured cells (Wilcock *et al.,* 2006[a]; Cote *et al.,* 1998; Halvorson, 1990; Kalenak *et al.,* 1975).

2. Cardiac responses

It has been shown that heart rate increases rapidly during hot water immersion (Bonde-Petersen *et al.,* 1992; Weston *et al.,* 1987). Bonde-Petersen *et al.* (1992) found that heart rate increased by 32% compared to no water immersion, during 15 – 20 minutes of hot water immersion up to the level of the chest at a temperature of 43.8℃ ($P < 0.05$). In comparison with thermo-neutral water immersion, hot water raised heart rate by 47% ($P < 0.05$). Additionally, hot water immersion causes a rise in cardiac output, whereas stroke volume rises slightly or may be slightly reduced due to the decrease in cardiac filling time (Bonde-Petersen *et al.,* 1992; Weston *et al.,* 1987).

3. Muscle elasticity and range of motion

According to Taylor *et al*. (1995), the proposed effects of hot water therapy could include increased muscle elasticity, joint extensibility and a reduction in muscle spasms and these adaptations may improve muscle flexibility. However, Kubo *et al*. (2005) found that 30 minutes of superficial hot pack therapy application (42°C) did not change the mechanical properties of human muscle and tendon and therefore would have no effect on flexibility and range of motion. Some authors suggested that flexibility will only be enhanced when stretching is combined with hot water immersion (Taylor *et al.,* 1995; Henricson *et al.,* 1984). Burke *et al*. (2001) investigated this statement by comparing three different interventions, namely (1) stretching alone with no immersion, (2) cold treatment and stretching and (3) hot treatment and stretching. They found that hamstring length was significantly improved after all three interventions ($P < 0.05$), and that the changes in flexibility with hot water immersion (44 ± 1°C for 10 minutes) were not significantly more than with cold water immersion (8 ± 1°C for 10 minutes).

4. Pain

It has also been suggested that pain may be relieved by the application of heat therapy (Nadler, 2004). Mechano-receptor sensitivity (i.e. afferent nociceptors) rise when the skin temperature (thermal stimulation) is increased and this results in enhanced myelinated afferent fiber (*A beta*) activity (Nadler *et al.,* 2003; Fields & Levine, 1984; Melzack & Wall, 1965). Because the large diameter myelinated afferent fibers are activated, the pain stimuli from the afferent nociceptors are inhibited resulting in an analgesic effect. This mechanism is called the gate control theory (Fields & Levine, 1984), which was originally proposed by Melzak & Wall (1965).

Nadler *et al.* (2003[b]) studied the efficacy of continuous low-level heatwrap therapy (heating to 40°C within 30 minutes) for the relief of lower back pain. They concluded that the continuous application of an overnight heatwrap (8 hours for 3 nights), effectively relieved pain the following day, improved trunk flexibility and reduced muscle stiffness. These effects lasted more than 48 hours after the completion of the treatments. In spite of this, it remains unclear whether short-term application of heat has significant effects on acute muscular pain.

5. Side effects

Hot water immersion or thermotherapy, apart from the proposed positive physiological effects, has side effects. The possibility of burn injuries are the first most common concern and therefore temperatures should not exceed 43°C, as protein denaturation occurs at 45-50°C (Sherwood, 2004). Hot water immersion may also contribute to inflammation and

swelling (Cote *et al.,* 1988; Magness *et al.,* 1970). When the goal is to minimize swelling and inflammation, hot water immersion may prolong the recovery process. It has been shown that heat increased swelling in ankle sprains (Feibel & Fast, 1976). Cote *et al.* (1988) also found a significant increase in oedema in 10 subjects with first- and second- degree ankle sprains after 20 minutes of hot water immersion (38.9 – 41.1°C). The average volumetric increase over the 3 treatment days was 25.5% with hot water immersion compared to 3.3% in subjects receiving the cold water treatment ($P < 0.05$). They concluded that the increase in blood flow and cellular permeability may enhance and contribute to the increased inflammatory response and therefore increase oedema in the injured area.

6. Conclusion

It should be carefully considered whether hot water therapy is recommended as a recovery strategy after exercise. Athletes with swelling, infections, wounds, acute injuries or vascular disease (Wilcock *et al.,* 2006[a]), should be particularly cautious in using hot water immersion as a recovery strategy. Heat application may aggravate these conditions and would therefore be detrimental for recovery and performance.

B. COLD WATER IMMERSION

Cryotherapy involves immersion of the whole body, or parts of the body in cold water. Most studies use temperatures of ≤ 15°C (Sramek *et al.,* 2000; Lane & Wegner, 2004). In the research setting the immersion duration varies from 15 to 20 minutes (Lane & Wegner, 2004), however, in practice it could be anything longer than 1 minute.

Cryotherapy is generally used in the treatment of acute inflammation as part of the rehabilitation process of soft tissue injuries (Deal *et al.,* 2002; Cote *et al.,* 1988; Barnes, 1979). The application of cold decreases the skin temperature which leads to cutaneous vasoconstriction as a result of an increase in sympathetic nerve activity. Cold water immersion also reduces the permeability of lymphatic, cellular and capillary vessels, which will prevent fluid movement to the interstitium resulting in a reduction in acute inflammation and therefore, muscle oedema (Sendowski *et al.,* 1997; Cote *et al.,* 1988). Local cell damage may also be decreased because the formation of hematoma may be diminished through the application of cold (Meeusen & Lievens, 1986). The inflammatory process is linked to certain effects such as increased pain sensation, a loss of force generation and increased swelling of the injured area (Smith, 1990). The application of cryotherapy may therefore minimize these responses and potentially could enhance recovery after exercise.

1. Muscle oedema

It is known that high intensity exercises which involve exhausting muscle activity, results in an increased intracellular H^+ concentration causing the intracellular pH to drop and resulting in metabolic acidosis (Vanderborne *et al.,* 2000; Cheng *et al.,* 1995). This may lead to delayed post exercise recovery of the skeletal muscles and may therefore impair athletic performance. Interestingly, there appears to be a relationship between intracellular pH and muscle temperature, namely that intracellular pH increases with decreasing muscle temperature at resting conditions (Yoshioka *et al.,* 2002). This lead Yanagisawa *et al*. (2003) to postulate that if cooling decreased the intracellular pH post exercise, muscle oedema, resulting from secondary muscle damage, may be prevented. They tested this hypothesis using high intensity eccentric exercise and 15 minutes of cold water immersion (5°C) directly after exercise. They found that the intracellular pH of the cooling group was significantly elevated at 60 minutes post exercise ($p < 0.05$), with no significant changes in intramuscular water content and muscle damage. At 48 hours post exercise, the control group had a 9.2% ($p < 0.05$) increase in intramuscular water content, which indicates significant muscle oedema.

2. Cardiac responses

The increased localized vasoconstriction caused by cold therapy results in increased peripheral and arterial resistance leading to higher a heart rate and cardiac output (Sramek *et al.,* 2000; Park *et al.,* 1999; Bond-Peterson & Schultz-Pederson, 1992). Cardiac preload is therefore elevated and may enhance the substrate transport throughout the body. Recovery will therefore be enhanced when the body gets rid of waste products at a faster rate (Bailey *et al.,* 2007; Cheung *et al.,* 2003; Kwon *et al.,* 1999; Lori *et al.,* 1998). Cold water immersion also evokes reductions in intracellular fluid similar to thermo-neutral water immersion (Krasney & Pendergast, 2008), even though the plasma volume is reduced with cold water immersion (Stocks *et al.,* 2004). Sramek *et al.* (2000) found reductions in heart rate, systolic and diastolic blood pressure in subjects during 1 hour of head out water immersion in 14°C (5%, 7%, and 8%, respectively; $P < 0.05$). They concluded that the cardiovascular responses were mainly caused by the increased activity of the sympathetic nervous system. This was reflected in the increased noradrenaline (530%; $P = 0.003$) and dopamine production (250%). Thus, cold water immersion mainly stimulates the thermoreceptors, activating the sympathetic nervous system. Oxygen consumption as well as metabolism is also elevated to maintain core temperatures (Sramek *et al.,* 2000).

3. Pain

It is proposed that cryotherapy may cause a reduction in pain through several possible mechanisms, namely the inhibition of nociceptors, reduction in metabolic enzyme activity, reduction in muscle spasms or an altered nerve conduction velocity (Airaksinen *et al.,* 2003; Algafly & George, 2007).

In 1966 Abramson *et al*. showed that cold application to tissue results in a decreased neural transmission along the nerve fibres. This reduction in neural transmission can bring about two effects, namely the increase in the level of pain tolerance or threshold, and a reduction in muscle spasms (Wilcock *et al.,* 2006). Algafly & George (2007) studied the influence of cryotherapy on nerve conduction velocity and the associated effects on pain threshold and pain tolerance. They found that nerve conduction velocity at the treated ankle decreased significantly by 32.8% ($P < 0.05$) as ankle skin temperature was reduced to 10^{o}C. Changes in pain tolerance (76%) and pain threshold (89%) for the treated ankles were significantly higher than the control ankles receiving no treatment (56% and 71% for the control, respectively). However, these differences were temporary and were no longer significantly different at higher skin temperatures (15^{o}C).

4. Side effects

Although there are beneficial physiological effects resulting from the use of cold water immersion, there are some side effects, depending on the amount of body immersed and the actual temperature of the water. Exposure to extreme cold water and a drop in core temperature below 32^{o}C may cause hyperventilation. Hyperventilation may lead to blood acidosis because of the decreased arterial carbon dioxide concentration (Lloyd, 1994). Other side effects include acute peripheral vasoconstriction contributing to increased swelling if there is any swelling present, convulsions, sudden loss of consciousness, ventricular ectopy, cardiac arrest and even death (Lloyd, 1994; Wittmers & Savage, 1994).

5. Conclusion

The effects of cold water immersion appear to be mostly analgesic and short-term. It is also unclear whether the physiological effects are temperature related, or rather due to the hydrostatic pressure of water immersion itself.

C. CONTRAST WATER THERAPY

Contrast water therapy involves the alteration of cold and hot immersions. The duration varies from 30 to 300 seconds of one temperature, followed by 30 to 300 seconds of the

contrasting temperature. This can be repeated a number of times and may last anything from 4 to 30 minutes (Wilcock *et al.,* 2006[a]). The exact physiological mechanism behind contrast water therapy has not been clarified, but it is said that the vascular pumping caused by the variation in temperature may be involved in the overall post exercise recovery process (Vaile *et al.,* 2007; Hamlin, 2007 Wilcock *et al.,* 2006[a]; Cochrane, 2004; Coffey *et al.,* 2004; Stanton *et al.,* 2003; Vaile *et al.,* 2003).

The vaso-pumping action is similar to the alternating muscular contractions involved in low intensity exercise or active recovery. It is suggested that active recovery enhances the movement and removal of lactate and also reduces the intracellular fluid volume, thereby improving recovery (Wilcock *et al.,* 2006; Signorile *et al.,* 1993; Thiriet *et al.,* 1993; Hildebrandt *et al.,* 1992). With contrast water therapy, the alternating vasoconstriction and vasodilation is said to enhance muscle blood flow and metabolite removal. Recovery is therefore enhanced without the extra energy cost that would be involved in low intensity exercise. However, it is still questionable whether the vaso-pumping action is effective enough to bring about any meaningful physiological effects to enhance the recovery process. Two problems arise; the first is, for vasodilation to occur the alternating temperatures must be able to change the intramuscular temperature, and secondly, the vaso-pumping action must be strong enough to cause a physiological effect, i.e. enhanced metabolite waste removal. Higgins & Kaminski (1998) found that 31 minutes of contrast therapy was not sufficient enough to cause significant variations in muscle tissue temperatures at 4cm below the skin surface. They also confirmed that the typical 1 minute cold water immersion protocol was not long enough to significantly decrease tissue temperature after the exposure to hot water immersion. In an effort to achieve deeper temperature penetration, Myrer *et al*. (1997) used ice instead of cold water in their study.

However, they also failed to cause any significant intramuscular temperature variations. This suggests that vaso-pumping does not reach the intramuscular level, and is rather restricted to the subcutaneous level (Myrer at al., 1994). Furthermore, Wilcock *et al*. (2006a) argued that if vaso-pumping did occur because of the contrasting temperatures, it would seem doubtful to have any significant effects at such a slow frequency pumping action, bearing in mind that muscular pumping involved in active recovery would occur at a higher rate of about 2 Hz.

Although it seems that the proposed theory of vaso-pumping is unlikely to cause any significant physiological effects, a few studies actually found that contrast water therapy improved lactate and metabolite clearance (Hamlin *et al.,* 2007; Coffey *et al*. 2004; Vaile *et al.,* 2003). This may be suggestive of the involvement of another mechanism, namely the

hydrostatic pressure effect on metabolite clearance. Johansen *et al*. (1997) suggested that greater physiological responses can be expected with higher hydrostatic pressures. This issue will be discussed further in the next section.

D. THERMO-NEUTRAL WATER IMMERSION

Thermo-neutral water immersion (water immersion *per se*) is the most widely researched method. The temperature usually ranges from cool to thermo-neutral (16 - 35^{o}C), while the immersion duration time can ranges anything from 5 minutes to 5 hours (Wilcock *et al.,* 2006[a]).

It is postulated that the main effects of water immersion can be attributed to the hydrostatic pressure exerted upon the immersed body, rather than the actual temperature of the water. Wilcock *et al*. (2006[a]) suggested that buoyancy may perhaps play a role in reducing the perception of fatigue and thus aid in energy conservation. Hydrostatic pressure is the force that is exerted on the immersed body (Wilcock *et al.,* 2006[a]). Water is denser than air at any given depth, therefore water immersion results in greater pressures exerted on the body. These pressures are also related to the depth of the immersion – the deeper the immersion, the greater the pressure (Bove, 2002).

Little research has been done on the recovery and performance effects of neutral water immersion. Research have mainly focussed on hemodynamic changes, fluid shifts, thermal responses, and cardiovascular responses using mostly thermo-neutral water temperatures (Gabrielsen *et al.,* 2002; Pump *et al.,* 2001; Kwon *et al.,* 1999; Johansen *et al.,* 1997; Hinghofer-Szakay *et al.,* 1987; Hakumaki, 1987; McArdle *et al.,* 1984; Farhi & Linnarsson 1977; Arborelius *et al.,* 1972; Abramson *et al.,* 1966).

1. Weightlessness and perceived fatigue

An important factor to consider when studying the effect of hydrostatic pressure on the body is buoyancy, i.e. the upward force exerted by water or fluid on an object. The upward force exerted by the water helps to support a part or all of the weight of the immersed body. Thus, the immersed body will weigh less because the water exerts a net upward force or hydrostatic upthrust. According to the *Archimedes Principle* this force is equal to the weight of the fluid displaced by the body. The degree of buoyancy also depends on the immersed body's density. A person with higher fat mass, which is less dense, will be more buoyant than a person that has more lean body mass. The upward force that is created can be calculated as follow:

$F = h \times \rho \times g \times A$.....(2.1)

Where $F = V \times \rho \times g$

Thus, $F = m/g$ .....(2.2)

(h = height of immersion; ρ = water density; A = base area; m = mass; g = gravity; V = immersed volume) (Wilcock *et al.,* 2006[a]).

The effect that buoyancy may have on recovery relates to the reduction in the gravitational forces that act upon the musculoskeletal system, which leads to greater relaxation of the muscles, particularly those that support body posture. It may also contribute to the conservation of energy. Furthermore, the weightlessness experienced by the immersed body appears to decrease the perception of fatigue or pain (Wilcock *et al.,* 2006; Coffey *et al.,* 2004; Kuligowski *et al.,* 1998; Nakamura *et al.,* 1996; Sanders, 1996; Viitasalo *et al.,* 1995). According to Smith (1991), prostaglandins that are synthesized by macrophages (which are involved in the inflammatory process) may sensitize the nociceptors which may increase the perception of pain after muscle damaging exercises. If inflammation is reduced by water immersion, the sensitization of the nociceptors may be lowered and this will result in a decreased perception of pain. Another proposed theory is that the weightlessness in the water causes a reduction in neural transmissions, and therefore a decreased perception of pain. However, it is unclear at this stage whether the reduced perception of soreness or pain after water immersion is a result of the hydrostatic pressure exerted by the water, the temperature of the water or a combination of these factors.

2. Fluid Shifts

2.1 Fluid homeostasis

The human body comprise of between 60 - 70% water between the extracellular and intracellular fluid compartments (Wilcock *et al.,* 2006[a]). The extracellular fluid compartment can be further subdivided in an interstitial (between cells) and intravascular (plasma) fluid compartment. Fluid located in these compartments acts as a vehicle for the transport, as well as exchange of substances between the cells and the external environment, with the aim to maintain homeostasis (Sherwood, 2004).

The movement of substances and fluid occurs in the vascular capillaries between the intravascular and extravascular space. Movement across the vascular capillaries can occur via different processes such as vesicular transport, diffusion and filtration-reabsorption. Vesicular transport is the active transport of substances across the vascular membrane, requiring adenosine triphosphate as an energy source. Diffusion is the movement of fluid and/or substances from a high concentration of solutes to a low concentration of solutes

across the capillary membrane. Diffusion accounts for the largest exchange of fluids and substances in the human body (Sherwood, 2004).

Filtration-reabsorption is the net movement of fluid due to hydrostatic and osmotic pressures. According to the traditional view filtration occurs at the arteriolar ends and absorption at the venular ends of the capillaries. Therefore, the net effect is a small degree of filtration with the return of substances via the lymph. The modern view emphasizes the interstitial forces and that filtration occurs mainly across the entire length of the capillary. Most of the fluid that is not reabsorbed by the capillaries via filtration-reabsorption moves through the lymphatic vessels. The fluid and substances that are continuously being exchanged between the interstitial space and vascular space returns to the vascular space via the lymphatic vessels (Waterhouse *et al.,* 2006).

The movement of fluid and substances across the capillaries are directed by four forces. Collectively they are called Starling forces (Sherwood, 2004). They can be further sub-divided into two categories, namely hydrostatic (hydraulic) pressures and osmotic (oncotic) pressures.

Fluid movement across the capillaries can be expressed by the following equation:

Flow per unit area: Kc [(Pc – Pi) – ($\pi c - \pi i$)].....(2.3)

Where; Kc = capillary coefficient; Pc = hydrostatic pressure in the capillary; Pi = hydrostatic pressure in the interstitial fluid; πc = osmotic pressure in the capillary; πi = osmotic pressure in the interstitial fluid.

The net movement of water moving out of the capillary is the difference between the hydrostatic pressure gradient across the capillary wall (moving fluid out) and the osmotic pressure gradient (drawing fluid in). The rate of fluid movement is also determined by the permeability of the capillary wall to water, and this is expressed by the capillary filtration coefficient (Waterhouse *et al.,* 2006).

An increase in capillary hydrostatic pressure can be the result of reduced arteriolar constriction, or increased venous pressure. The latter may also be caused by water immersion. The increase in hydrostatic pressure causes an increase in net filtration. However, the increase in filtration is buffered by the resulting movement of water to the interstitial space which decreases the interstitial osmotic pressure and a new steady state is re-established (Waterhouse *et al.,* 2006). At resting conditions, there is a net movement of fluid and substances into the interstitial space from the capillaries. Together with the plasma proteins that may have leaked out of the capillaries, the fluid must be removed by the

lymphatic system to prevent the build-up of interstitial fluid and thus the development of oedema or inflammation (Waterhouse *et al.,* 2006).

2.2 Fluid shifts during and after exercise

High intensity exercise increases the volume of intramuscular water content (Yanagisawa *et al.,* 2003; Nosaka & Clarkson, 1996). During exercise, the increased hydrostatic capillary pressure caused by the elevation in the mean arterial pressure, generates fluid movement from the vascular space to the interstitial fluid compartments (Convertino, 1987). The increased permeability and capillary pressure that occurs during and after exercises, as well as the increased production of metabolic by-products such as lactic acid, increases the osmotic gradients between the extravascular space and capillaries (Yanagisawa *et al.,* 2004; Yabagisawa *et al.,* 2003). Fluid movement to the active muscles is enhanced thereby increasing the intramuscular water content (Yanagisawa *et al.,* 2004). Fluid shifts during exercise are related to the intensity of the exercise (Gillen *et al.,* 1991; Hildebrandt *et al.,* 1992; Green *et al.,* 1984), i.e. the higher the intensity, the greater the fluid shifts.

The body has mechanisms which protect the circulating plasma volume at given levels of exercise. The plasma volume stabilizes after the initial efflux of vascular fluids, in such a way that after 30 – 60 minutes of exercise the percentage loss of plasma is similar to that of 10 minutes of exercise (Senay, 1986). The plasma oncotic pressure in the capillaries is therefore increased, and the body secretes increased concentrations of vasopressin and renin-angiotensin. Vasopressin and renin-angiotensin are powerful vasoconstrictors and its concentrations during exercise are also related to the intensity of exercise. Vasoconstriction in inactive tissues decreases the mean capillary hydrostatic pressure and a greater net absorption of fluid into the vascular space from interstitial fluids is achieved (Sherwood, 2004; Convetino *et al.,* 1981). The continuous fluid shifts into the circulation from inactive tissue and out of the circulation into the active muscles allows for optimal blood volume and cardiovascular stability (Conventino *et al.,* 1981). This counterbalancing mechanism is especially important during prolonged exercise. Studies have shown that cycling at intensities of 30 - 120% of maximal oxygen uptake decreased blood plasma by 5 – 17%. Most of the fluid shifts were directed to the intramuscular compartment involving the active muscle (Hildebrandt *et al.,* 1992; Gillen *et al.,* 1991; Knowlton *et al.,* 1987; Mohesenin *et al.,* 1984; Green *et al.,* 1984).

2.3 Fluid shifts during water immersion

It is well known that water immersion causes an increase in central blood volume and that the degree of fluid shifts contributing to the central blood volume depends on the depth of immersion (Johansen *et al.,* 1997; Lollgen *et al.,* 1981; Echt *et al.,* 1974; Arborelius *et al.,* 1972). The increase in central blood volume is due to the increased diffusion and absorption (hemodilution) and displacement of blood from the peripheral tissues to the intrathoracic circulation. Hemodilution is mainly caused by a negative transcapillary pressure which results in the transfer of fluid from the interstitial space to the intravascular space of the legs (Johansen *et al.,* 1997). The end result being that capillary filtration is decreased and end-capillary or venular reabsorption of intracellular and interstitial fluid is increased (Khosla & DuBois, 1979).

Immersion to the level of the neck causes a more pronounced increase in central blood volume (Johansen *et al.,* 1997). This may be the result of the increased hydrostatic pressure during neck immersion compared to that of hip immersion. The increase in hydrostatic pressure may facilitate the displacement of fluid and blood from the legs to the lower pressure areas such as the thoracic cavity in combination with the displacement of blood from the abdomen (Wilcock *et al.,* 2006[a]; Johansen *et al.,* 1997; Löllgen *et al.,* 1981). The central blood volume is therefore elevated via an increased transvascular pressure gradient and this reduces the peripheral volume. It is also believed that the interstitial-intravascular gradients that are caused by the higher hydrostatic pressure may improve the reabsorption of interstitial fluids, thereby reducing oedema (Friden & Lieber, 2001).

Hinghofer-Szalkay *et al.* (1987) studied the fluid shifts in 6 men during 30 minutes of immersion (up to the neck) in thermo-neutral water (35 ± 0.2^{o}C). They found an 11 ± 3% increase in plasma volume after 30 minutes of immersion, with a decreased haematocrit (-1.0%). Intravascular fluid shifts were also accompanied by plasma protein shifts (i.e. albumin), suggesting that it may contribute to the increased intravascular fluid shift via an increased oncotic pressure in the extravascular compartment. This suggests that intracellular components (metabolic wastes) may leave the cells and interstitial space to be able to sustain an osmotic balance (Wilcock *et al.,* 2006[a]).

Thus, fluid shifts accompanying water immersion may result in the increased clearance of metabolic wastes from the cells and interstitial space. Together with the translocation of metabolites and by-products the ability of the athlete to recover from high intensity exercise could be enhanced (Wilcock *et al.,* 2006[a]; Stocks *et al.,* 2004; Hinghofer-Szalky *et al.,* 1987).

2. Oedema

Any disruption of the filtration-reabsorption transport process may result in an abnormal increase in interstitial fluid in the localized areas. This condition is known as swelling or oedema and is usually caused by changes in capillary pressure gradients, lymph blockages, reduced plasma concentrations or physical trauma (Waterhouse *et al.,* 2006; Wilcock *et al.,* 2006[a]).

3.1 Causes of oedema

3.1.1 Increased capillary pressure

Venous blood and lymph flow can be affected by gravitational forces (Waterhouse *et al.,* 2006). When an individual stands upright for prolonged periods, lymph and blood will pool in the lymphatic vessels and veins. However, rhythmic muscular contractions together with the working of the venous valves cause blood and lymph to be pumped away from the lower extremities towards the heart. In the absence of these mechanisms capillary pressure will increase leading to an elevated net capillary filtration (Waterhouse *et al.,* 2006). Because the outward capillary pressure is elevated, fluid movement will be enhanced towards the interstitial fluid compartment and will ultimately lead to regional oedema of the dependent tissues (Sherwood, 2004). Any localized restriction of venous return can also result in oedema. Examples are venous insufficiency, swelling during pregnancy and swelling during long flights (Waterhouse *et al.,* 2006; Sherwood, 2004; Kozlova *et al.,* 2000).

3.1.2 Lymph blockages

Lymph blockages cause oedema, because the excess fluid in the lymph vessels cannot be returned to the blood circulation. Lymph blockages can be caused by filariasis, a condition where small filarial worms infect the lymph vessels and thus prevent lymph drainage, or when major lymph nodes are removed during surgery (Sherwood, 2004).

3.1.3 Low concentration of plasma proteins

Low concentrations of plasma proteins cause a decrease in the plasma osmotic pressure and more fluid will be allowed to filter out than the amount of fluid that would then be reabsorbed by the capillaries (Sherwood, 2004; Kozlova *et al.,* 2000). Therefore, too much interstitial fluid will accumulate leading to oedema. There are a number of ways in which this could occur; for instance, when the liver is unable to synthesize plasma proteins, excessive loss of proteins in nephrotic syndrome caused by kidney disease, a loss of plasma proteins from skin burns or a diet deficient in protein (Waterhouse *et al.,* 2006; Sherwood, 2004; Kozlova *et al.,* 2000).

3.1.3 Muscular fatigue and damage

Muscle damage is the main cause of muscle oedema in athletes. Muscular fatigue resulting from overexertion can lead to acute breakdown of skeletal muscle fibers (Tiidus 1998), which is the result of the post exercise muscle acute inflammatory response. Neutrophils and macrophages filtrate into the muscle fibers and are responsible for the removal of damaged muscle tissue (degeneration). The neutrophils and marcophages also produce other reactive oxygen species which promote post exercise inflammation and help with muscle fiber repair (Evans & Cannon 1991). The acute inflammatory period is further accompanied by muscle swelling, and the amount of swelling depends on the degree of muscular damage. It usually results from the increased leakage of proteins from the capillaries as a result of the increased permeability (Waterhouse *et al.,* 2006; Deal *et al.,* 2002). The increased protein leakage causes an increased filtration of fluid out of the capillary into the interstitium, thereby causing muscle oedema (Waterhouse *et al.,* 2006).

Muscle oedema as a result of muscle damage or exercise may lead to the compression of blood capillaries and therefore impairs oxygen delivery to the localised cells. Because the capillaries are compressed (increased interstitial fluid volume), the rate at which metabolic wastes are cleared is decreased and this may result in secondary damage to the tissue (Wilcock *et al.,* 2006[a]; Friden & Lieber 2001; Tidus 1998; Shepard & Shek 1998).

It is suggested that the hydrostatic pressure exerted by water immersion causes an increase in the pressure gradient between the intravascular and interstitial compartment of the legs, thereby improving the reabsorption of interstitial fluids. The hydrostatic pressure may therefore reduce muscle oedema in a similar way to that of compression stockings (Jonkera *et al.,* 2001; Partsch *et al.,* 2004; Wilcock *et al*, 2006[a]).

However, water immersion may also result in increased transcapillary pressure. The resultant increase in plasma filtration may cause a delay in cellular infiltration by monocytes, leukocytes and neutrophils into the intersitium. By delaying the inflammatory process of cellular infiltration, further tissue degeneration may be attenuated (Vaile *et al.,* 2004; Vaile *et al* 2007; Wilcock *et al.,* 2006[a]; Lecomte *et al.,* 1998). Intramuscular pressure will also be decreased (Lecomte *et al.,* 1998) and the contractile function of the muscle fibres as well as strength may also be enhanced (Wilcock *et al.,* 2006[a]; Cesari *et al.,* 2004). Thus, secondary muscle damage may be decreased and the athlete's potential to recover from high intensity exercise could be enhanced from high intensity exercises (Wilcock *et al.,* 2006[a]).

3. Cardiac responses

The mechanical loading of the cardiovascular stretch receptors are responsible for the cardiovascular and circulatory responses to water immersion (Krasney & Pendergast, 2008). Water immersion causes an increase in the central blood volume (Johansen *et al.,* 1997) and the increase in the cardiac pre-load therefore elevates the stroke volume and thus the cardiac output (Wilcock *et al.,* 2006[a]; Sramek *et al.,* 2000; Kwon *et al.,* 1999; Epstein 1992). However, the magnitude of the cardiovascular effects is dependent on the water temperature and the immersion depth (Wilcock *et al.,* 2006[a]).

Thermo-neutral water immersion studies have reported that immersion up to the level of the hips increased stroke volume by 12 - 37% (Farhi & Linnarsson, 1977; Löllgen *et al.,* 1977). At the level of the xiphoid process stroke volume has been reported to increase to 38 - 67% (Gabrielsen *et al.,* 2002; Bonde-Petersen *et al.,* 1992; Löllgen *et al.,* 1981; Weston *et al.,* 1987; Farhi & Linnarsson, 1977), with more pronounced increases of 28 - 95% up to the level of the neck (Shiraishi *et al.,* 2002; Park *et al.,* 1999; Löllgen *et al.,* 1981; Farhi & Linnarsson, 1977; Arborelius *et al.,* 1972). Johansen et al. (1997) postulated that the increased hydrostatic pressure during neck immersion in comparison to hip level immersion may assist in the transfer of blood from the abdominal region together with the transfer of blood from the legs. As a result central blood volume is elevated which increases the cardiac pre-load and therefore stroke volume (Christie *et al.,* 1990).

Thermo-neutral water immersion generally causes a decrease in heart rate, with decreases of 4 - 6% at the level of the hips (Löllgen *et al.,* 1981; Farhi & Linnarsson, 1977), 11 - 18% at the level of the xiphoid process (Wilcock et al, 2006[a]; Gabrielsen *et al.,* 2002; Watenpaugh *et al.,* 2000; Bonde-Petersen *et al.,* 1992; Löllgen *et al.,* 1981; Fahri & Linnarsson 1977; Weston *et al.,* 1987;) and 3 - 15 % during immersion up to the level of the neck (Wilcock *et al.,* 2006[a]; Yun *et al.,* 2004; Shiraishi *et al.,* 2002; Gabrielsen *et al.,* 2000 Johansen *et al.,* 1997; Park *et al.,* 1999; Lollgen *et al.,* 1981; Farhi & Linnarsson, 1977; Arborelius *et al.,* 1972).

The increase in central blood volume achieved during water immersion elevates the atrial pressure (Gabrielsen *et al.,* 2002) and stimulates the baroreceptors. When the aortic baroreceptor activity is increased, heart rate will decrease and this response is known as bradycardia (Hakumaki, 1987). However, the atrial receptors are also stimulated, opposing the sympathetic response. The reflex causes an increase in the heart rate and this is called the Brainbridge reflex. Therefore, the effect of the baroreceptors must have overridden the influence of the atrial receptors, to be able to trigger a decrease in heart rate during water immersion (Hakumaki, 1987). Only a few studies have found non-significant decreases in

heart rate during thermo-neutral water immersion (Yun *et al.,* 2004; Arborelius *et al.,* 1972; Johansen *et al.,* 1997). The reason for this could be that the duration of immersions (shorter than 30 minutes) was not sufficient to elicit a significant response (Wilcock *et al.,* 2006[a]).

Cardiac output increases as a result of the increase in stroke volume and is also related to immersion depth (Krasney & Pendergast 2008; Wilcock *et al.,* 2006a). Cardiac output would increase via an increase in the stretching of the cardiac muscle fibers as a result of the elevated central blood volume. This cardiac length tension response can be explained by the Frank-Starling mechanism (Krasney & Pendergast, 2008). Cardiac output has been documented to increase between 14 - 29% at hip level immersion (Löllgen *et al.,* 1981; Farhi & Linnarsson, 1977), between 19 - 48% at the level of the Xiphoid process (Gabrielsen *et al.,* 2002; Bonde-Petersen *et al.,* 1992; Weston *et al.,* 1987; Löllgen *et al.,* 1981; Farhi & Linnarsson, 1977) and between 29 - 66% at head out water immersion (Shiraishi *et al.,* 2002; Park *et al.,* 1999; Lollgen *et al.,* 1981; Farhi & Linnarsson, 1977; Arborelius *et al.,* 1972).

5. Blood flow and peripheral resistance

Blood flow is usually autoregulated to meet the metabolic and oxygen demands of the peripheral tissues. Water immersion adjusts the autoregulation of blood flow in such a way that an increased systemic blood flow is achieved. Plasma volume is also increased as a result of the transcapillary fluid shift from the extravascular compartments (Krasney & Pendergast, 2008). The increase in plasma volume is mainly derived from the blood capillaries of the legs in upright immersion (Johansen *et al.,* 1995). The plasma osmotic pressure is also therefore decreased (Johansen *et al.,* 1995). Gabrielsen et al. (2000) found that forearm skeletal muscle blood flow is significantly increased after 30 minutes of thermo-neutral water immersion. Blood flow increased during immersion up to the level of the xiphoid process, and increased significantly by 49 ± 16 % ($P < 0.05$) at the level up to the neck. Muscular vascular resistance did not significantly change during the immersion up to the level of the xiphoid process, but decreased by 15 % up to the level of the neck ($P < 0.05$). Balldin et al. (1971) also found an increase in the lower leg skeletal muscle blood flow during thermo-neutral water immersion. They reported an average increase of 130% and concluded that a combination of sympathetically and locally mediated factors were responsible for the changes in vascular resistance.

Constriction of blood capillaries in most vascular beds is influenced by both local and humoral factors and the balance between these factors determines the resultant vascular tone (Hainsworth, 2004). It is suggested that during water immersion, peripheral venous tone decreases and that this loss is accompanied by a reduction in the general sympathetic tone (Echt *et al.,* 1974). The reduction in the sympathetic tone is the main cause for the reduced

total peripheral resistance achieved during thermo-neutral water immersion (Echt *et al.,* 1974; Arborelius *et al.,* 1972).

Total peripheral resistance can be expressed in several ways as:

TPR = (MAP – CVP)/Q (Yun *et al.,* 2004; Park *et al.,* 1999).....(2.4)

TPR = (MAP – right arterial pressure)/Q (Arborelius *et al.,* 1972).....(2.5)

TPR = MAP/Q (Bonde-Petersen *et al.,* 1992; Weston *et al.,* 1987).....(2.6)

Where; MAP = mean arterial pressure; CVP = central venous pressure and Q = cardiac output (Wilcock *et al.,* 2006[a]).

Therefore, the increase in cardiac output (caused by the hydrostatic pressure exerted by water immersion) is accompanied by a decrease in peripheral resistance. Echt et al. (1974) measured the peripheral venous tone through occlusion plethysmography and determined the venous volume elasticity coefficient during 3 hours of thermo-neutral water immersion from 5 subjects. They found a decrease in the venous tone of 8.2 % (baseline: 16.6 $mmHg.ml^{-1}.100\ g^{-1}$ tissue; 5 minutes of immersion: 13.3 $mmHg.ml^{-1}.100\ g^{-1}$ tissue) within the first 5 minutes of immersion up to the level of the neck. Venous tone significantly decreased by 30% post immersion ($P < 0.0025$) and remained reduced ($P < 0.025$) for 1 hour post-immersion. Most studies have shown that the reduction in peripheral resistance only occurs during immersions up to the level of the neck (Yun *et al.,* 2004; Park *et al.,* 1999; Echt *et al.,* 1974; Arborelius *et al.,* 1972), however, when the results are compared with non-immersion control groups, the findings are non-significant (Wilcock *et al.,* 2006[a]; Bonde-Petersen *et al.,* 1992; Weston *et al.,* 1987).

The resultant effects of water immersion, namely the increased cardiac output, reduction in peripheral resistance and vasodilation will cause an increase in blood flow throughout the body. Thus, blood flow may be enhanced to the muscles and organs' thereby increasing the body's potential to recycle metabolites and by-products to enable the body to recovery faster. The body would also be able to replenish energy stores more effectively. An indication that blood flow to the muscle capillary beds increases, is that blood lactate clearance is in fact enhanced after subjects were partially immersed into water (Coffey *et al.,* 2004; Morton, 2007; Nakamura *et al.,* 1996; Sanders, 1996).

E. CONCLUSION

Water immersion seems like an excellent modality for the enhancement of recovery in athletes involved daily in hard exhaustive training. The hydrostatic pressure together with the different temperature effects are the main mechanisms responsible for possible recovery effects. However, it still needs to be established whether the physiological effects are primarily because of the hydrostatic pressure, or mostly due to the temperature, or possibly a combination of both factors. Most studies are inconclusive whether the physiological responses mediated by temperature effects are effective in enhancing the overall recovery process of athletes.

CHAPTER THREE

PHYSIOLOGICAL CHANGES DURING POST EXERCISE RECOVERY

A. INTRODUCTION

Cycling is a popular sport and a competitive season can be anything between 3 – 4 months in duration. A competition usually comprises of single day stage races, multi-day stage races, and 3 week tour races (i.e. Tour de France). The variation in terrain and distance (i.e. hilly routes, flat routes, time-trials, short stages, long stages etc.) require different performance attributes, but in all cases the cyclists' muscular, neuromuscular, metabolic, and nervous systems are pushed to exhaustion during both training and competition (Abbiss & Laursen, 2005). These demands on the physiological systems may lead to temporary impairments in performance, which may last for a short duration of time (minutes – hours) or a prolonged period (up to a number of days) (Barnett, 2006). Metabolic disturbances following high intensity exercises are usually restored within minutes to hours (Westerblad *et al.* 2006) and the restoration of glycogen usually occurs within 24 hours together with adequate rehydration (Barnett, 2006). Impairments that last longer than 24 hours may be related to muscle injury, DOMS, inflammation and neuromuscular fatigue (Lepers *et al,* 2000). The latter may lead to overtraining, illness and injury.

The more competitive the athlete, the more important is performance outcomes. At elite level even the smallest difference in competitive advantage may have a tremendous impact on the end result. The performance of a cyclist is limited by the ability of the cyclist to resist fatigue, and the ability of the cyclist to enhance or maintain performance is influenced by the post-exercise recovery rate (Abbiss & Laursen, 2005). Since it can be assumed that all elite athletes train as hard and as smart as they can, a competitive advantage can only be achieved if they also optimize their recovery. To enhance the post-exercise recovery process athletes perform structuralized recovery sessions with the aim to recover from the stresses caused by high intensity training, and also enhancing the effect of the training stimulus (Barnett, 2006, Bompa, 1999). The post-exercise recovery process is a multi-dynamic process and is made up of different mechanisms, i.e. physiological and psychological. This discussion will focus on the most important physiological processes.

B. POST EXERCISE RECOVERY RATE

Recovery is a multidimensional complex process and the rate of recovery is influenced by factors such as, age, experience, gender, environmental factors (i.e. high altitude, extreme cold or hot temperatures), type of muscle fiber used, psychological factors (negative feelings etc.), as well as nutritional intake (availability and replenishment of macronutrients) (Bompa, 1999). More experienced athletes will generally recover faster following exhaustive exercise, because they are physiologically more adapted to their sport (Barnett, 2006; Bompa, 1999).

The recovery curve is not linear but curved, and the magnitude of the curve depends on the level of exhaustion or fatigue achieved (Bompa, 1999). The recovery curve can be divided into three phases. During the initial phase muscle glycogen is replenished, and heart rate, blood pressure, and blood lactate concentration return to resting levels (~ 30 minutes - 6 hours). During the second phase full replenishment of fuels of the entire system is achieved which may take up to ~24 hours. The last phase represents neural recovery which may take more than 24 hours (Bompa, 1999).

C. EXCESS POST-EXERCISE OXYGEN CONSUMPTION

Following exercise, oxygen uptake remains elevated above resting conditions for a period of time (Børsheim & Bahr, 2003). This elevated metabolism is known as excess post-exercise oxygen consumption (EPOC) (Børsheim & Bahr, 2003). EPOC can be divided into two phases, namely the rapid phase immediately after exercise (up to 2 – 3 minutes following exercise) and the slow phase, which lasts for a longer period of time (> 30 minutes and up to 24 hours following exercise) (Cochrane, 2004). Exercise intensity influences both the duration and magnitude of the elevated post-exercise metabolism (Powers & Howly, 2001) and is thus much greater and lasts longer following high intensity exercise compared to moderate exercise. The differences are due to the total amount of ATP and creatine phosphate (PC) depleted through exercise, the amount of body heat gained and the amount of circulating levels of epinephrine and norepinephrine (Powers & Howly, 2001).

The rapid segment of the EPOC curve depicts the time to restore and replenish PC and ATP in the muscle (deVries & Housh, 1994), replace oxygen stores in both blood and tissues (haemoglobin and myoglobin) and for removal of metabolic waste products, i.e. lactate (Cochrane 2004; Børshein & Bahr, 2003; Powers & Howly, 2001). The increased body temperature, ventilation and circulation, as well as the degree of muscle damage (Børshein & Bahr, 2003) also contribute to the rapid EPOC observed following exercise.

There are several factors that contribute to the slower segment of EPOC. Firstly, breathing rate, heart rate, circulation and body temperature remain elevated for several minutes following exercise, which requires additional oxygen above resting conditions, however, the cost of this is very low (Børshein & Bahr, 2003; Powers & Howly, 2001). Also, elevations in circulating hormones, such as epinephrine and norepinephrine may also contribute to the increased post-exercise metabolic rate. Then again, no sustained increase in the secretion of these hormones has been found following exercise (Børshein & Bahr, 2003). Other factors that may contribute to the prolonged segment following exhaustive prolonged endurance exercise includes an increased rate of fatty acid cycling and a shift from carbohydrate to fat utilization as substrate source (Børshein & Bahr, 2003; deVries & Housh, 1994).

D. METABOLIC RECOVERY

1. Post-exercise lactate kinetics

Lactate accumulation in the blood during exercise is a reflection of the balance between lactate production by the working muscles and lactate clearance by the liver, heart and muscle tissue (Gladden, 2004; Powers & Howly, 2001). The ability for trained athletes to remove accumulated lactate from the blood following exhaustive exercise is faster and more effective than for untrained individuals (Stallknecht et al., 1998). The reason for this is that trained individuals are physiologically more adapted to improve lactate clearance (Powers & Howly, 2001; Stallknecht et al., 1998).

The negative effects of lactate on muscle performance are due to the hydrogen ions (H+) that dissociates from the lactic acid (Gladden, 2004; Bompa, 1999). Elevated concentrations of H+ ions in the muscle are associated with negative effects on muscle function through (i) inhibition of myofibrillar ATPase for contraction, (ii) inhibition of the cross-bridge from the low to high force state, (iii) inhibition of the rate of glycolysis, (iv) inhibition of maximal shortening velocity, (v) reduction in cross-bridge activation by inhibiting the binding of calcium (Ca2+) to troponin-C, and (vi) inhibition of sarcoplasmatic ATPase activity and a slower Ca2+ re-uptake (Gladden, 2004; Tomlin & Wegner, 2001).

Blood lactate levels return relatively quickly (within 60 – 90 minutes) to resting values following exhaustive exercise, even with passive recovery (Barnett, 2006; Cochrane, 2004). The main tissues responsible for post-exercise lactate clearance are the heart, liver and muscle tissue (Powers & Howly, 2001; Stallknecht et al., 1998). Most of the lactate is directly oxidized by these tissues. In the liver, lactate is converted to glycogen via gluconeogenesis (Powers & Howly, 2001; Stallknecht et al., 1998). Lactate is also taken up by type I skeletal muscle fibers (muscle tissue) and converted to glycogen (Stallknecht et al., 1998). Although it

has been shown that blood lactate levels usually recovers within an hour following exercise, higher than normal resting blood lactate may be observed in athletes when muscle damage is evident. Elevated plasma creatine kinase has been previously associated with elevated resting blood lactate levels (Gleeson *et al.,* 2002; Gleeson *et al.,* 1995).

2. Glycogen replenishment

Muscle glycogen is the primary energy source during prolonged exercise at moderate-to-high intensities (Jentjens & Jeukendrup, 2003). When muscle glycogen becomes depleted during prolonged exercise, fatigue will set in and performance will decline. Therefore, optimal pre-exercise muscle glycogen stores are vital for best performances (Barnett, 2006; Rauch *et al.,* 2005; Cochrane, 2004; Ivy, 1991). Restoration of muscle glycogen levels following exhaustive prolonged exercise also influences the post-exercise recovery rate. Complete restoration of muscle glycogen can occur within 24 hours (Barnett, 2006; Jentjens & Jeukendrup, 2003), provided that enough carbohydrates are consumed. The recommended amount of carbohydrate intake for the highest muscle glycogen synthesis rate, immediately following exercise is around 1.0 – 1.85 $g.kg^{-1}.h^{-1}$ (Barnett, 2006; Jentjens & Jeukendrup, 2003; Bompa, 1999). If carbohydrate intake is delayed for several hours following exercise, the rate of muscle glycogen synthesis may be delayed by ~50%, resulting in a prolonged post-exercise recovery rate. The pattern of muscle glycogen synthesis occurs in two phases (Jentjens & Jeukendrup, 2003). The initial phase consists of a period of rapid synthesis of muscle glycogen that does not require any insulin action and lasts about 30 – 60 minutes. The rapid phase is characterized by the exercise-induced translocation of the glucose transporter carrier protein (GLUT4) to the cell surfaces, which increases the permeability of the muscle membrane and enhances glucose uptake (Jentjens & Jeukendrup, 2003). Following this rapid phase, the slower phase consists of a much slower rate of muscle glycogen synthesis and may last for several hours post-exercise (Jentjens & Jeukendrup, 2003). Insulin secretion becomes more important during this phase, thereby up-regulating the activity of glycogen synthase for the increased synthesis of muscle glycogen. This increased muscle insulin sensitivity following exercise can persist for > 48 hours depending on the amount and type of carbohydrate intake (Jentjens & Jeukendrup, 2003). Factors that may influence the rate of post-exercise glycogen synthesis are (i) training status or experience, (ii) the magnitude of muscle glycogen depletion, (iii) the muscle fiber types used during exercise, and (iv) the mode of exercise (Jentjens & Jeukendrup, 2003). In addition, muscle glycogen synthesis is also impaired following muscle fiber damage, particularly seen with eccentric exercise (Jentjens & Jeukendrup, 2003; Widrick *et al.,* 1993). However, this impairment is only noticeable at 24 – 72 hours post-exercise (Widrick *et al.,* 1993) and is possibly related to the inflammatory process associated with muscle damage.

E. NEUROMUSCULAR RECOVERY

The neuromuscular fatigue model is based on two theories, namely (i) the central activation failure theory (central fatigue), and (ii) the neuromuscular propagation failure theory (peripheral fatigue) (Abbis & Laursen, 2005; Millet & Lepers, 2004).

1. Central fatigue

It is suggested that following prolonged exercise, there is a reduced neural input to the muscles which leads to a decreased maximum contraction capacity of the muscle fibers. This alteration in neural input may be the result of a feed-forward control mechanism, called a central governor, which may possibly be located in the brain (Abbis & Laursen, 2005**;** Millet & Lepers, 2004; Millet *et al.,* 2002). Therefore, as fatigue develops during prolonged exercise, there is an increase in intracortical inhibition which inhibits the central neural drive and therefore the excitement and recruitment of skeletal muscle (Abbis & Laursen, 2005; Davis, 1995). Muscle power output therefore declines.

Central fatigue is much greater following prolonged running compared with cycling of the same intensity and duration (Abbis & Laursen, 2005; Millet & Lepers, 2004). The reason for this might relate to the greater degree of muscle damage caused by running, which will result in greater afferent sensory feedback and thus greater spinal modulation (Abbis & Laursen, 2005).

2. Peripheral fatigue

Neuromuscular fatigue may also be explained by peripheral mechanisms, for example, a reduced ability of the muscle to respond to an electrical stimulus at the level of the sarcolemma or alpha-motor neuron (Abbis & Laursen, 2005). Factors such as a reduction in conduction velocity of action potentials, alterations in the M-wave amplitude (due to a reduction in ionic concentrations of Na^+ and K^+ across the muscle membrane), and/or a decrease in pH may explain the reduced EMG activity during prolonged exercise (Green, 1997). Also, the increased intracellular lactate and extracellular K^+ concentrations will decrease the membrane excitability and ultimately reduce central activation (Green, 1997). Thus, any alteration or interference of the excitation-contraction coupling mechanism between the motor neuron and the muscle fiber can result in reduced force production and therefore muscle fatigue. This explanation for muscle fatigue is also referred to as the muscle power/peripheral failure theory (Abbis & Laursen, 2005).

Neuromuscular fatigue is therefore due to different interrelated mechanisms (central and peripheral factors). Prolonged exercise that will lead to neuromuscular fatigue may require 24 – 28 hours to recover (Miller *et al.,* 2004).

F. MUSCLE RECOVERY

Muscular overexertion beyond its breakpoint may lead to an acute breakdown of skeletal muscle (Tiidus, 1998). Damage to the muscle fibers and structures will influence the power-producing capacity of the muscles and therefore influence performance (Abbis & Laursen, 2005). The post-exercise muscle inflammatory response is usually associated with ultrastrucural muscle damage, muscle membrane disruption, disrupted Ca^{2+} homeostasis, increased free radical concentrations (reactive oxygen species), disruption in ionic gradients and membrane structures, loss of muscle functioning, swelling, and an increased sensation of pain or soreness (Abbis & Laursen, 2005; Smith, 1991). The aim of the inflammatory process is therefore to remove the damaged tissue and repair the damaged muscle tissue. However, these alterations may in turn reduce the neuromuscular activation and/or reduce the force and power production of the muscles, therefore, impairing cycling performance.

There are two mechanisms by which muscles are actually damaged, namely increased oxidative stress or increased mechanical stress. Exercise that elevates mitochondrial oxygen consumption results in the increased production of reactive oxygen species (free radicals) (Tiidus, 1998). Oxygen reactive species are believed to result in oxidative stress, where the production of oxygen radicals exceeds the ability of the tissue antioxidants to detoxify them, and may cause muscular damage due to the disruption of SR function and calcium homeostasis (Tiidus, 1998). Another form of muscular overtraining and damage is caused by exercise which predominantly involves eccentric muscular contractions (Howatson & van Someren, 2008; Tiidus, 1998). The increased mechanical stress that is loaded on the muscle fibers cause microscopic ruptures (Ebbeling & Clarkson, 1989).

Depending on the extent and degree of damage, skeletal muscle will need between 2 - 7 days to recover optimally (Cheung *et al.,* 2003). Any muscle damage, irrespective of the direct cause, will therefore attenuate the post-exercise recovery process.

G. CONCLUSION

Optimal recovery following exhaustive exercise is a multidimensional process, with many interrelated causes and mechanisms. The purpose of post-exercise recovery is to accelerate the reversal of all the physiological changes that have taken place during exercise, which may be related to fatigue and/or muscle damage. Therefore, the primary aim of recovery strategies is to aid in the regeneration, repair and adaptation of muscles, as well as to

replenish muscle and liver glycogen stores. It is therefore imperative to study the various recovery strategies that are employed by athletes in order to establish which strategies can efficiently minimize muscle fatigue and muscle damage, yet enhance training adaptations.

CHAPTER FOUR

EFFECTS OF WATER IMMERSION ON RECOVERY AND PERFORMANCE

A. INTRODUCTION

The modern athlete is increasingly looking for ways to recover faster between training sessions and competitions. Faster recovery times between training sessions may have an advantage in the competitive arena. Recovery is also very important when high intensity performance must be maintained and repeated on consecutive days of competition. It is known that when athletes train intensively hard without giving their bodies enough time to recover, it will lead to poor performances, burnout and a suppressed immune system (Budgett, 1998).

For the past number of years attention has focused on the role of water therapy, especially cold water immersion (cryotherapy) and contrast water therapy on post-exercise recovery (Ingram *et al.,* 2009; Hing *et al.,* 2008; Howatson & van Someren, 2008; Montgomery *et al.,* 2008; Vaile *et al.,* 2008; Bailey *et al.,* 2007; Willcock *et al.,* 2006[a,b]; Yanagisawa *et al.,* 2003; Howatson & van Someren, 2003; Eston & Peters, 1999). Despite a fair number of research studies on this topic some questions remain unanswered. For instance, it is not known what the most effective protocol is for water therapy, and whether temperature or hydrostatic pressure is responsible for the positive effects on recovery, or what exact physiological mechanisms are behind water therapy. Also there is very little evidence that water therapy may enhance the overall process, especially on performance and accumulated fatigue during multi-days of competition and training. The following chapter discusses the effects of water immersion on aspects of performance and post-exercise recovery as well as the long term effects on the adaptive and regenerative processes involved with training.

B. EXERCISE-INDUCED MUSCLE DAMAGE & DELAYED ONSET MUSCLE SORENESS

Most studies investigating the effects of water immersion on performance and post-exercise recovery have focussed on exercise induced muscle damage (delayed onset muscle soreness (DOMS)) (Ingram *et al.,* 2009; French *et al.,* 2008; Goodall & Howatson 2008; Vaile *et al.,* 2008; Vaile *et al.,* 2007; Bailey *et al.,* 2007; Sellwood *et al.,* 2007; Skurvydas *et al.,* 2006; Eston & Peters, 1999; Kuligowski *et al.,* 1998; Isabell *et al.,* 1992). DOMS is usually

caused by muscle damaging exercise, specifically unfamiliar or unaccustomed exercise (Howatson & van Someren, 2008; Clarkson & Hubal, 2002; Friden & Lieber, 2001), or eccentric exercise (Howatson & van Someren, 2008; Conolly *et al.,* 2003; McNeil & Khakee, 1992). During eccentric actions muscle are forced to lengthen while simultaneously produce tension and if the external load exceeds the muscle's ability to actively resist the load, will lead to microscopic damage to the myotendinous junctions (Howatson & van Someren, 2008; Cheung *et al.,* 2003; Friden & Lieber, 2001). Visible damage to the internal membrane system, Z-discs, contractile proteins and intermediate filaments provide evidence for the inflammation and damage theory explaining the mechanism of DOMS (Nosaka, 2007). On the other hand, alterations to the Z-disc structures also occur to some degree after concentric exercise (Gibala *et al.,* 1995) which may also lead to DOMS. Furthermore, Jones *et al.* (1986) documented that mononuclear cell filtration do not correspond with muscle pain and therefore the extent of ultrastructural alterations in muscle may not automatically determine the extent of muscle damage (NuretNBerg *et al.,* 1992). Symptoms of DOMS can range from muscular discomfort or tenderness to pain during active muscular contractions (Howatson & van Someren, 2008; Cheung *et al.,* 2003; Friden & Lieber, 2001). These symptoms usually reach a peak between 24 - 48 hours post exercise (Friden & Lieber, 2001).

Researchers investigating the mechanisms of DOMS have successfully induced muscle soreness using exercise protocols such as downhill running (Eston *et al.,* 2000; Donnelly *et al.,* 1990), isokinetic dynamometry (RodetNBurg *et al.,* 1994), stepping (Hasson *et al.,* 1993), eccentric resistance training (French *et al.,* 2008; Vaile *et al.,* 2007; Sellwood *et al.,* 2007; Eston & Peters, 1999; Isabell *et al.,* 1992) and plyometric exercise training (Goodall & Howatson, 2008; Skurvydas *et al.,* 2006). DOMS is categorized as a type 1 muscle strain injury (Cheung *et al.,* 2003). The associated symptoms include a reduced range of motion, elevated levels of muscle injury markers such as creatine kinase and myoglobin and prolonged strength and power loss (Howatson & van Someren, 2008; Cheung *et al.,* 2003; Connolly *et al.,* 2003; Friden & Lieber, 2001). Up to know, six hypothesised theories have been proposed to explain the mechanism of DOMS (Howatson & van Someren, 2008; Cheung *et al.,* 2003). These theories relate to lactic acid, connective tissue damage, inflammation, muscle damage and enzyme efflux (Howatson & van Someren, 2008; Cheung *et al.,* 2003). However, a combination of these mechanisms is more likely to be the cause of the muscular pain and tenderness associated with DOMS, rather than a single cause.

1. Indirect markers of muscle damage and soreness

1.1 Creatine kinase

Many researchers consider creatine kinase concentration as a reliable marker of skeletal muscle damage (Clarkson *et al.,* 1992; Hartmann & Mester, 2000; Stauber & Smith, 1998; Komulainen *et al.,* 1994; Cheung *et al.,* 2003). Even though the large inter-subject variability sometimes makes it difficult to interpret the data (Nosaka & Clarkson, 1996), the variability within a subject is more stable (Clarkson & Sayers, 2007). Subjects can be therefore be classified as either low responders or high responders (Chen, 2006; Totsuka *et al.,* 2002).

High intensity exercise that causes damage to the skeletal muscle cell structures at the level of the Z-discs and sarcolemma causes an alteration in membrane permeability and the release of enzymes, such as CK (Noakes, 1987; Hornemann *et al.,* 2000). Enzymes leak into the interstitial fluid and is then taken up by the lymphatic system and returned to the blood circulation (Bijsterbosch *et al.,* 1985). Downhill running which mainly involves eccentric muscular contractions causes the greatest increases in serum creatine kinase concentration in the blood. Similarly, high post-exercise serum creatine kinase levels is also observed after prolonged strenuous exercise such as triathlon events (Neubauer *et al.,* 2008) and ultra-distance marathon running (Nuviala *et al.,* 1992).

Factors that may influence the CK response in subjects are physical fitness and distinctive muscular properties such as individual muscle strength (Totsuka *et al.,* 2002; Brancaccio *et al.,* 2007). Peak serum CK activity is observed 24 – 48 h after isometric muscle contraction exercise (Totsuka *et al.,* 2002; Graves *et al.,* 1987) and 3 – 6 days after eccentric exercise (Howatson & van Someren, 2008; Brancaccio *et al.,* 2007; Chen, 2006; Nosaka & Clarkson, 1996). The CK response after endurance exercise (such as cycling) appears to be much lower in magnitude compared to an acute bout of eccentric exercise. Totsuka *et al.* (2002) observed a significant elevation in CK 3 hours post exercise ($P < 0.05$) and peak serum CK activity (331 ± 78 $IU.L^{-1}$) was only observed after the third day of endurance training. The subjects exercised at the same absolute workload for 90 minutes over 3 consecutive days. The high and low responders have demonstrated that there is a break point of CK release at 300 – 500 $IU.L^{-1}$ in serum after endurance exercise. The breakpoint is related to the individual's muscle strength, for instance when an individual has to increase muscle tension to be able to complete the same amount of absolute work , membrane permeability will be altered leading to an increased leakage of CK. The threshold would then be determined by muscle properties such as strength. Threshold values can differ from one individual to another; therefore relative loadings should rather be used to determine threshold values that would better explore Totsuka's (2002) findings. Köning *et al.* (2003) found significant

increases in CK concentrations (+ 26.6%; $P < 0.001$) after the fourth stage of a 5 day cycling race (length 156 km; mean duration 5 hours 13 minutes) compared to baseline values. The lower responses in CK resulting from exercise-induced skeletal muscle damage caused by intensive endurance exercises are different as proposed to the mechanisms involved with DOMS.

1.2 C-Reactive protein, inflammation & swelling

C-reactive protein is part of the non-specific acute phase response (Pepys & Hircshfield, 2003). It is secreted from the hepatocytes, under the control of the cytokine Interleuken-6, in response to forms of infection, tissue damage, trauma and inflammation. C-reactive protein is a sensitive marker of inflammation and tissue damage and for this reason studies have used CRP (Neubauer *et al.,* 2008; Pepys & Hircshfield, 2003; Köning *et al.,* 2001).

Exhaustive endurance exercise causes stress upon the different systems (hormonal, metabolic, thermal and oxidative systems) which results in the release of cytokines and other acute phase proteins (CRP) in addition to the upregulation of different immune cells (Nebauer *et al.,* 2008; Köning *et al.,* 2001).

Exercise induced muscle damage is also associated with post-exercise inflammation (Tiidus, 1998), although it is unclear whether the acute inflammatory process is the primary mechanism underlying DOMS (Smith, 1990). The inflammatory process involves muscular infiltration by neutrophils and marcophages. Reactive oxygen species and cytokines are produced and this enhances post-exercise inflammation with the aim to remove the damaged muscle tissue and repair the injured tissue (Tiidus, 1998; Tidball, 1995). The increased protein and enzyme leakage disrupts the oncotic osmotic pressure in such a way that fluid movement to the interstitial space is enhanced, therefore resulting in swelling (Cheung *et al.,* 2003; Smith, 1990). Peak oedema levels (measured by limb circumferences and volumes) appear to correspond with peak muscle soreness (Vaile *et al.,* 2008; Vaile *et al.,* 2007; Eston & Peters, 1999).

1.3 Muscular strength and power

Several researchers have observed significant reductions in strength and power during DOMS (Goodall & Howatson 2008; Vaile *et al.* 2008; Vaile *et al.,* 2007; Bailey *et al.,* 2007; Skurvydas *et al.,* 2006; Eston & Peters, 1999; Kuligowski *et al.,* 1998; Viitasalo *et al.,* 1995; Donnelly *et al.,* 1990) and these reductions in strength are usually observed 24 – 48 hours post-exercise (Lavender & Nosaka, 2006; Howatson & van Someren, 2008; Clarkson & Hubal, 2002). The duration, as well as the magnitude of strength loss, is much greater after

eccentric activity compared to concentric activity and needs 8 – 10 days to return to baseline values (Howatson & van Someren, 2008; Cheung *et al.,* 2003).

Both strength and power are important functional factors of athletic performance. For performance to be maintained the recovery of strength and power must be optimal. It is therefore, generally accepted that muscle strength and power is the deciding factor when a decision must be made on an athlete's readiness for action when experiencing DOMS (Kuligowski *et al.,* 1998).

Researchers use different ways of assessing the decline in muscular strength and power observed following EIMD or exhaustive exercise. Most studies used isometric testing tools such as isokinetic dynameters or force transducers (Howatson & van Someren, 2009; Goodall & Howatson, 2008; Vaile *et al.,* 2008; Sellwood *et al.,* 2007; Bailet *et al.,* 2007; Wilcock *et al.,* 2006[b] Howatson & van Someren, 2005; Eston & Peters, 1999; Kuligowski *et al.,* 1998) whereas the specificity of the assessments should be strongly considered in order to be a reliable tool in sports exercise contexts (Wilcock *et al.,* 2006[b]). However, very weak correlations have been shown previously between isokinetic dynometer and other sporting movements such as sprints and jump performances (Wilcock *et al.,* 2006[b]; Murphy *et al.,* 1994). Measurements that are more reliable in measuring muscular power and strength and are also more specific to sporting movements include countermovement jumps, vertical jumps, and squat jump performance (Rowsell *et al.,* 2009; French *et al.,* 2008; Montgomery *et al.,* 2008).

1.4 Muscle soreness & pain

The inflammation resulting from the disruption in the muscle fibers, may lead to subsequent muscular soreness or discomfort. The feeling of discomfort or muscular pain can be subjectively assessed by using Visual Analog Scales (Rowsell *et al.,* 2009; Montgomery *et al.,* 2008; Vaile *et al.,* 2008; Bailey *et al.,* 2007; Vaile *et al.,* 2007; Thompson *et al.,* 1999), although other subjective methods also exist (Eston & Peters, 1999; Kuligowski *et al.,* 1998). It has been shown that VAS scales are reliable in measuring acute pain (Bijur *et al.,* 2001).

Furthermore, muscle soreness usually peaks with peak oedema levels or swelling. This may be related to the increase in the chemical substances within the muscles which irritates the muscle nerve endings in the affected area where swelling (inflammation) is persistent (Smith, 1991, Clarkson & Hubal, 2002).

2. Effects of cold water immersion on exercise-induced muscle damage

Numerous recovery strategies (Howatson & van Someren, 2008; Cheung *et al.,* 2003), including water immersion (Goodall & Howatson 2008; Vaile *et al.* 2008; Vaile *et al.,* 2007; Bailey *et al.,* 2007; Skurvydas *et al.,* 2006; Howatson & van Someren, 2005; Eston & Peters, 1999; Kuligowski *et al.,* 1998; Viitasalo *et al.,* 1995), have been studied in the treatment of DOMS. Most studies involving water therapy have focused on the use of contrast water and cold water therapy in the treatment of DOMS associated with exercise-induced muscle damage.

Eston & Peters (1999) concluded that cold water immersion was not effective in reducing the signs and symptoms associated with exercise-induced muscle damage. Fifteen healthy women subjects performed a bout of muscle damaging eccentric exercises of the elbow flexors on an isokinetic dynamometer, consisting of 8 sets of 5 maximal reciprocal contractions at a velocity of 0.58 $rad.s^{-1}$ with 60 seconds of rest between each set. Thereafter the subjects were either allocated to a control group (n = 7) or treatment group (n = 8). The cryotherapy group immersed their arms in cold water (15^{o}C) for 15 minutes immediately after exercises and every 12 hours for a total of 7 sessions. Relaxed elbow angle was significantly greater and creatine kinase activity was lower for the cryotherapy group on days 2 and 3 in comparison with the control group ($P < 0.05$). Creatine kinase activity was still elevated (+ 28.8% from baseline) 72 hours post-exercise in the control group, whereas creatine kinase activity was closer to pre-exercise levels in the cryotherapy group (+ 3.3% from baseline). The authors postulated that the difference in creatine kinase response between the two recovery conditions may be associated with the effect of cryotherapy on the permeability of lymph and blood vessels, altering the efflux of creatine kinase. Another possible explanation may be that the cryotherapy had reduced the amount of post exercise muscle damage to the muscle tissue; therefore less CK would then be released from the muscle membrane which may have resulted in a reduced creatine kinase efflux.

Bailey *et al.* (2007) studied the effects of a single administration of cold water immersion on indices of muscle damage following a bout of prolonged intermittent shuttle running. Twenty active men completed a 90 minute intermittent shuttle run. Thereafter they were randomly assigned to either 10 minutes of cold-water immersion (average temperature 10^{o}C) or a non-immersion control group (passive recovery). Cryotherapy significantly reduced myoglobin 1 hour after exercises ($P < 0.05$), and muscle soreness at 1, 24 and 48 hours post exercise ($P < 0.05$) compared to the control group. In addition, smaller decrements in maximum voluntary contraction for the knee flexors was observed for the cryotherapy group at 24 and 48 hours (~ 12% and ~5% decrease from baseline, respectively) compared to the control group (~21%

and ~14% decrease from baseline). The reductions in DOMS observed at 24 and 48 hours post exercise support the assumption that the cryotherapy treatment was effective in reducing muscle injury rather than assisting in the removal of exercise-induced by-products. Bailey *et al*. (2007) further postulated that cryotherapy may attenuate the inflammatory response and reduce post-exercise muscle damage via a decreased permeability of blood and lymph vessels to myoglobin, attenuating the myoglobin efflux. The cooling effects of the CWI are responsible for the vasoconstriction of the blood vessels and lymphatic vessels thereby decreasing the permeability of the blood vessels (Hing, 2008; Meeusen & Lievens, 1986).

It appears that cold water immersion, performed immediately after muscle damaging exercise may in fact be more effective in reducing some indices associated with exercise-induced muscle damage than no treatment (passive recovery) at all.

On the other hand, Goodall & Howatson (2008) studied a total of eighteen physically active male subjects who each completed a bout of 100 drop jumps and was then randomly assigned to either a 12 minute cold water immersion group (15 ± 1°C) or a passive rest control group. Subjects received treatment immediately post-exercise and every 24 hours for the following 3 days thereafter. They concluded that the CWI did not attenuate any of the dependent variables and that repeated CWI does not enhance recovery from a bout of muscle damaging exercises. Therefore, CWI was not any better than performing no treatment. Although significant time effects were seen for all dependent variables ($P \leq 0.01$), there was no group or interaction effect. Gooddall & Howatson suggested that the reason for their non-significant results may be due to the fact that there was no significant cooling of the muscles. Intra-muscular temperature changes are related to the amount of adipose tissue (Myrer *et al.,* 2001); the greater the amount of adipose thickness, the greater the amount of time for maximum cooling to be achieved. Therefore, a 12 minute immersion protocol may not be enough to affect significant muscle cooling.

Skurvydas *et al.* (2006) investigated the effects of CWI on the time-course of indirect indicators of DOMS. The purpose of their study was to establish which of the indirect markers of muscle damage is the most sensitive to the effect of muscle cooling, when does the effects of cooling manifests itself, and lastly does the disappearance of low frequency fatigue depend on the effect of muscle cooling after exercise. Twenty untrained subjects underwent cold water immersions of 15 ± 1°C after completing 100 drop jumps. Subjects immersed their legs for 15 minutes, followed by a 10 minute rest period and another immersion of 15 minutes. All subjects underwent the same protocol twice (9 – 10 months apart), i.e. the control condition (passive rest) and CWI. CWI was performed immediately

after exercise, as well as 4, 8, and 24 hours post exercise. Maximal voluntary contraction force, electrostimulation (ESF) and jumping height was significantly decreased for both groups after exercise ($P < 0.001$), however, the jumping height and MVCF returned quicker to baseline values in the CWI group. There was no significant recovery in the control group. Although the CK activity was significantly lower in the CWI group at 24 and 48 hours compared to the passive rest group, the CK levels were still elevated at 48 hours post-exercise compared to baseline values in both groups. Furthermore, the CWI group reported significantly lower perception of muscle soreness from 24 to 72 hours post exercise ($P < 0.001$) compared to the passive rest group. In addition, cooling did not produce any changes in the dynamics of low frequency fatigue (LFF) disappearance. Skurvydas *et al.* (2006) suggested that the main mechanisms of the cold water immersion are aimed at reducing secondary muscle damage and may be supported by their results that were noticeable at 24 – 72 hours post exercise (during the time when symptoms associated with secondary muscle damage manifests). In conclusion, CWI appeared to be more effective than no treatment in accelerating the disappearance of some indirect markers of DOMS.

Sellwood *et al.* (2007) studied the efficacy of a more practical and commonly used protocol for cold water immersion for the prevention of delayed onset muscle soreness. The CWI protocol consisted of immersion in cold water of 5 ± 1^{o}C for 1 minute and then 1 minute out of the bath. The cycle was repeated three times. Forty untrained volunteers (women and men) were randomly allocated to either the ice-water or neutral water (24^{o}C) group. DOMS were induced in the quadriceps using an eccentric exercise protocol on a seated leg extension machine. No significant differences were found between the control and intervention groups at any time point with regard to all pain measures, serum creatine kinase, thigh circumferences, one-legged hop-for-distance or isometric strength. They concluded that the lack of a treatment effect may be due to low levels of muscle damage and that the muscle damaging protocol was insufficient.

Isabell *et al.* (1992) studied the effects of cold temperature application, in an attempt to exclude the effect of the hydrostatic pressure exerted by water itself. Healthy subjects (N = 22; 11 women and 11 men) were randomly assigned to either one of the four groups (e.g. ice massage, ice-massage combined with exercise, exercise and no treatment). Treatment groups underwent eight 15 minute treatments administered at 0, 2, 4, 6, 24, 48, 72, and 96 hours post-exercise. Muscle soreness was induced in the non-dominant arm using eccentric and concentric dumbbell curl exercises. Exercises consisted of elbow flexion and extension for 20 seconds, with 40 seconds rest. The cycle was repeated for 15 minutes. Ice massage and exercise was combined, so that 20 seconds of exercise treatment was followed by 40 seconds of ice massage treatment. The ice treatment failed to protect the muscle against

injury and Isabell *et al.* (1992) concluded that the ice application may even be contraindicated in the treatment and relief of delayed onset muscle soreness. In a more recent similar study (Howatson & van Someren, 2005) it was found that ice massage was ineffective in reducing the indices associated with exercise-induced muscle damage to the elbow flexors. A possible reason for these findings could be that the effects of cold was limited to the temperature effects only, eliminating the possible effects of hydrostatic pressure on the recovery process, namely fluid shifts and a reduction in the inflammatory process, oedema and secondary muscle damage.

3. Effects of contrast water therapy on exercise-induced muscle damage

The effectiveness of contrast water therapy (CWT) has also been extensively studied. Vaile *et al.* (2007) studied the effects of CWT in comparison to passive recovery (PAS) on the indices of exercise-induced muscle damage. Thirteen recreational athletes performed two treatment trials separated by 6 weeks in a randomized crossover design. Subjects performed a DOMS-inducing leg press protocol. After the exercise protocol subjects underwent a 15 minute recovery period consisting of either CWT or PAS. Immersion was up to the level of the anterior superior iliac spine alternating between a cold bath (8 - 10^{o}C) for 1 minute and hot bath (40 - 42^{o}C) for 2 minutes. Peak force production was significantly higher after CWT than PAS at 24 and 48 hours post exercise. Power developed after CWT was not significantly lowered at 24 and 48 hours post recovery, whereas after PAS it was still significantly lower at 24 (18.0 ± 11.6%) and 48 hours (22.7 ± 11.6%) post recovery (P < 0.006). No significant differences in the changes of CK concentrations as well as in perceived pain (P > 0.01) between the two recovery strategies were found. Furthermore, thigh volume was significantly less following CWT than for PAS recovery (P < 0.01). The authors concluded that CWT was associated with a faster recovery of power production during the jump squat.

In contrast, French *et al.* (2008) found that contrast water therapy was not more effective than passive recovery on improving recovery as well as performance from exercise induced muscle damage. Subjects included 26 active healthy men who performed an exercise-induced muscle damage protocol involving resistance exercises with an eccentric component The recovery interventions included contrast water therapy (CB) (n = 10) alternating between cold (8 - 10^{o}C) and hot (37 – 40^{o}C) water baths, a control group (n = 6) who did not receive any treatment, and the compression garment group (n = 10) where the subjects had to wear full-length compression tights for 12 hours over night. There were large within-group effect sizes for $\log_e$[CK] for contrast bathing at 24 hours (ES = 0.80) and 48 hours (ES = 0.84) post exercise. Significant within group differences from baseline were also found in all groups for

power produced during countermovement-jumps (CG, - 5.1%; CB, - 4.4%; CON, - 8.5%) as well as for muscle soreness scores (+ 213%, + 284%, + 284%). Swelling was apparent only in the CB (+ 1.4%) and CON (+ 1.6%) groups. Sprint performances over 30 meters were not influenced by any of the recovery interventions. French *et al.* (2008) concluded that the interventions did not demonstrate an obvious pattern of performance enhancement and/or recovery effects. A possible explanation for their findings, in contrast to the results of Vaile *et al.* (2008) and Vaile *et al.* (2007), may be that the duration of immersion was much shorter as well as the level of immersion being less in comparison to the other mentioned studies, therefore reducing the effect of the water temperature and the hydrostatic pressure. Also, the authors included active subjects (with resistance training experience of at least a year), and it is therefore possible that they could have maintained their performances as a result of their improved capacity to faster recovery.

For CWT to produce any effects, intramuscular muscle temperature must be altered to induce the vaso-pumping action, i.e. alternating vasoconstriction and vasodilation of the arteries. Though it is postulated that this mechanism is responsible for the reduced inflammation, secondary muscle damage, enhanced blood flow and clearance of substrates (Hing *et al.,* 2008) it has been shown that intramuscular temperature fluctuations (Higgins & Kamanski, 1998; Myrer *et al.,* 1997, Myer *et al.,* 1994) are not caused by alternating hot and cold water immersion. Therefore, other mechanisms such as hydrostatic pressure of water immersion must also be considered as well as the specific water temperature, rather than alternating temperatures. For any changes in intramuscular temperatures to take place during CWT, the immersion duration must be longer than 5 minutes (Myrer *et al.,* 1998). This method of recovery may therefore be more time consuming and any possible benefits may not be worthwhile.

4. The effects of water temperature

Kuligowski *et al.* (1998) compared the effects of warm whirlpool therapy (38.9^{o}C), cold whirlpool (12.8^{o}C) and contrast therapy (38.9^{o}C and 12.8^{o}C at a ratio of 3 to 1 minutes), and no treatment in 56 volunteers after a bout of eccentric contractions of the elbow flexors. They found that contrast water therapy and cold whirlpool therapy were more effective in restoring elbow flexion range of motion than warm whirlpool and no treatment. The authors postulated that the cold treatment probably minimized the acute inflammatory process and therefore reduced the amount of fluid build up in the surrounding tissue (oedema). This resulted in greater elbow joint mobility. Kuligowski *et al.* (1998) further suggested that the lower concentrations of prostaglandin, as a result of the reduced inflammatory response lead to a decrease in the sensitization of the nocireceptors therefore resulting in less perceived

soreness. Although contrast water therapy and cold water therapy relieved some of the symptoms associated with DOMS, none of the treatment interventions showed any significant effects on the maximal voluntary isometric strength of the biceps brachii.

The findings of Kuligowski *et al* (1998) are in contrast to those of Vaile *et al.* (2008) who reported significant recovery of power and isometric force after DOMS-inducing eccentric leg press exercises in response to cold water immersion (15^{o}C) and contrast water immersion (alternating between 15^{o}C and 38^{0}C), but not after HWI (38^{o}C). Furthermore, post-exercise swelling was significantly reduced following CWT and CWI, but not after HWI. The decreased levels of swelling may be due to an increase in the re-absorption of interstitial fluid caused by the hydrostatic pressure of the water, resulting in reduced oedema. This reduction in post-exercise oedema may not only improve the contractile functions within the muscle itself (evident by the jump squat and isometric strength improvement) but also decrease the secondary muscle damage. Their findings suggest that CWI and CWT appears to be more effective in reducing the functional and physiological deficits associated with DOMS compared to HWI.

It appears that HWI could have some functional advantages over passive recovery, but probably not any physiological advantages. Viitasalo *et al.* (1995) studied the effects of HWI (36.7 - 37.2^{o}C) on the recovery of well-trained athletes after 5 strength-power sessions performed in 3 days. Jumping power dropped by only 1.9% in the HWI group, compared to 8.3% in the control group, however, there were no significant differences in isometric strength between the two interventions. Importantly, both serum myoglobin and serum creatine kinase were significantly higher for HWI treatment compared to passive recovery. Viitasalo *et al.* (1995) suggested that the warm water caused increased leakage of muscle proteins which causes vasodilation, an increase in blood flow and an enhanced catalysation of metabolic reactions. This, however, would not be favourable in the recovery process, as it would enhance the inflammatory processes involved in exercise-induced muscle damage.

Ingram *et al* (2009) compared the post exercise effects of CWI and CWT to see whether the temperature of the water is more important than the effect of the hydrostatic pressure. Subjects performed 80 minutes of simulated team sports exercises (4 x 20 minute quarters of intermittent running) followed by a 20 meter shuttle run to exhaustion. Recovery, following exercise and 24 hours later, consisted of CWI (10^{o}C), CWT (10^{o}C and 40^{o}C), and passive recovery for 15 minutes. CWI resulted in significantly less muscle soreness 24 hours post exercise compared to CWT and passive recovery, however, no significant differences were found in CK or CRP at any time point between the three recovery conditions. Subjects in the CWI group performed better in the physical tests in the 48 hour post-exercise period, i.e.

there were smaller reductions in isometric strength (flexion and extension) and total sprint times (10 x 20 m sprints) after CWI compared to CWT and passive recovery. The authors concluded that CWI following exhaustive simulated team sport exercise offers greater recovery benefits than CWT and passive recovery. Furthermore, they suggested that these results can be attributed to the effect of temperature, rather than hydrostatic pressure. However, if the water temperature was the only factor involved in the recovery benefits, Isabell *et al.* (1992) and Howatson & van Someren (2005) would have found significant interactions for the ice massage groups in reducing the signs and symptoms of DOMS, which was not the case.

It seems that both CWI and CWT are more effective in reducing some indices of muscle damage associated with DOMS, than no treatment at all (PAS), but that HWI is not any better than receiving no treatment. However, it is not clear whether CWI is more effective than CWT in producing optimal results in indices of recovery and performance.

C. MUSCULAR STRENGTH, POWER AND ANAEROBIC PERFORMANCE

Water immersion may be beneficial in the recovery process after exercise induced muscle damage (Ingram *et al.,* 2009; Vaile *et al.* 2008; Bailey *et al.,* 2007; Vaile *et al.,* 2007; Skuvydas *et al.,* 2006). Then again, depending on the protocol and water temperature it may also be detrimental on subsequent performances. By understanding the acute effects and nature of water immersion upon subsequent performance, protocols of water immersion (temperature, duration, frequency, and timing between high intensity performances) can be better planned and scheduled so that it would not impair performance, but rather improve the recovery process between performance or training bouts.

1. Post-immersion effects and influence on functional performance

A number of studies have shown that functional strength performance is significantly impaired directly after cold water therapy. Howard *et al.* (1994) studied the post-immersion effects on isokinetic and isometric knee strength in 10 physically active male college students. All subjects performed three conditions in random order, namely CWI (12^{o}C), thermal neutral immersion (35.5^{o}C), or passive recovery for 45 minutes. They found that as joint movement velocity increases, average peak torque decreased significantly following CWI, compared to both neutral and room temperature treatments. Total work also decreased significantly after CWI for the faster joint movement velocities. Cross *et al* (1996) and Patterson *et al* (2008) found similar results with functional tests for speed, power, agility and range of motion, even though their subjects were only exposed to CWI for 20 minutes. After CWI, Patterson *et al* (2008) reported a significant reduction in vertical jump height (17.46%),

speed (10.55%), and agility (12.05%). The study by Crowe *et al.* (2007) confirmed that athletes participating in high intensity, short duration activities should be cautious in using CWI as a recovery strategy between events. Their subjects performed two 30 sec Wingate tests, separated by one hour, with either 15 minutes CWI (13°C – 14°C) or passive recovery in between. There were significant reductions in the peak power and total work of the second Wingate test after CWI.

Howard *et al.* (1994) postulated that the performance decrements may be attributed to cold-induced effects such as reduced nerve conduction velocity, cross-bridge deactivation, alterations of the motor unit recruitment order and increased tissue viscosity leading to added resistance to cross-bridge formation. Another proposed theory for the decreased maximal muscle activity is that the velocity at which adenosine triphosphate (ATP) splits to produce energy are highly dependable on the muscle tissue temperature. Therefore, the lower the intramuscular temperature, the slower energy will be produced for muscular force generation (Cross *et al.,* 1996). Patterson *et al* (2008) suggested that the cold water also alters the stretch reflex properties of the musculotendinious units and thus increases tissue stiffness, resulting in limitations in the range of movement of joints.

2. Effects of water immersion on performances separated by 24 hours and more

Previous studies have shown that there is a gradual increase in functional performance during the acute phase of the recovery period (~ 60 min post-CWI), which may be attributed to tissue rewarming through an increase in blood flow to the working muscles. This means that over a period of 24 hours after CWI, functional performance could be totally restored and possibly improved, in which case CWI would be shown to enhance the recovery process. Lane & Wegner (2004) investigated this possibility by having subjects perform two intermittent cycling trials (18 minutes of varying work intervals performed in succession at a resistance of 80 $g.kg^{-1}$ body weight) separated by 24 hours. The first trial was followed by one of 4 recovery conditions, namely active recovery (30% of VO_2 max), massage of the legs, CWI (15°C) and passive recovery for 15 minutes. During the second trial, total work was increased by 10% after CWI, although this change was not statistically significant. However, total work decreased by 16% following active recovery, 35% following massage, and 78% following passive recovery. This study suggests that CWI could facilitate the recovery process, provided that CWI is followed by at least 24 hours before the next bout of exercise.

Most studies on the effect of water immersion therapy involve laboratory tests or isolated field tests, i.e. vertical jump tests or 30 second cycle sprints. Thus there is a lack of evidence on the role of recovery strategies during actual exercise training or competition. Montgomery

et al. (2008) addressed this particular issue by comparing the effects of CWI and full length leg compression garments on the physical and functional performance and cumulative fatigue during a 3-day basketball tournament. CWI was limited to 5 x 1 minute intervals, while the compression garments were worn for ~18 hours after the game. Baseline measurements of physical performance (20-m acceleration, basketball line drill, Yo-Yo Level 1 intermittent recovery test, and sit and reach flexibility test) were measured prior to the start of the tournament as well as a day after the last game of the tournament. Subjects in the CWI group only had a 0.5% (SD = 1.4) reduction in performance time in the 20-m acceleration test after 3 days of basketball competition, compared to a 3.2% (SD = 1.6) reduction in the group wearing the compression garments. On all other tests, players performed equally well, however, their performances were significantly better than the control group, whose intervention involved stretching exercises and a carbohydrate snack. It thus appears that CWI may be an effective recovery strategy during multi-day competitions, provided that the competition bouts are more than 1 - 2 hours apart (Coffey *et al.,* 2004), however, CWI was not better than compression garments.

D. AEROBIC PEFORMANCE

Very limited studies have focussed on the effects of water immersion on aerobic performance (Coffey *et al.,* 2004; Nakamura *et al.,* 1996). Although the latter two studies used endurance type exercises (running and cycling), the exercise time was relatively short. It must be kept in mind that the cumulative fatigue caused by prolonged or intermittent aerobic exercise may be related to metabolic and neural factors that are different from fatigue that are caused by eccentric exercise inducing muscle damage. Therefore, the results of studies on the effect of water therapy on EIMD and DOMS can not necessarily be extended to aerobic exercise where the likelihood of serious muscle damage and soreness is much less, but where overall fatigue levels are significant.

Coffey *et al.* (2004) had subjects run to exhaustion on a treadmill at 120% and 90% of peak running speed over a 4 hour period. Subjects then followed one of three interventions, namely CWT (10^{o}C and 42^{o}C), active recovery (AR, running at 40% of peak treadmill running speed) or passive recovery for 15 minutes. The researchers found no significant differences in the time to exhaustion between the recovery conditions, although RPE values tended to be lower with CWT compared to both AR and PAS. Blood lactate concentrations were significantly lower for active recovery compared to passive recovery, but no significant differences were observed between active recovery and CWT.

Hamlin (2007) also compared the effectiveness of 6 min CWT (8 - 10^{o}C and 38^{o}C) and 6 min active recovery after a repeated sprint test. Subjects performed the second bout of sprinting

one hour after the recovery intervention. In contrast to the findings of Coffey *et al* (2004), Hamlin (2007) found significantly lower blood lactate concentrations after CWT compared to AR. However, similar to the findings of Coffey *et al* (2004), neither of the recovery interventions had a significant effect on sprinting performance.

Nakamura *et al.,* (1996) compared the effects of HWI (38^{o}C), thermo-neutral water immersion (30^{o}C) and passive recovery on subjects after 10 min cycling exercises at 80% VO2max. They found no significant differences in heart rate and blood pressure between the three recovery interventions, but lactate concentration was significantly lower after the thermo-neutral water treatment compared to passive recovery ($P < 0.05$). Furthermore, subjective feelings were lower after the thermo-neutral water treatment than after the control treatment.

Coffey at al. (2004) and Hamlin (2007) suggested that the lower post exercise blood lactate concentration after AR and CWT could be attributed to faster lactate clearance rates. They further proposed that the alternating vasoconstriction and vasodilation together with the effects of hydrostatic pressure caused by CWT will increase muscle perfusion and thus lactate clearance. Hamlin (2007) also proposed that the immersion into water immediately after high intensity exercises may actually, because of the increase in central blood volume, blunt the increased plasma catecholamine concentration, reducing adrenaline and noradrenaline release therefore resulting in a decreased heart rate and blood lactate response (Connely *et al.,* 1990). The improved lactate clearance after both CWT and neutral water immersion may thus be related to hydrostatic pressure exerted by the water, rather than temperature.

E. THE EFFECT OF WATER THERAPY ON THE RESPONSE TO TRAINING

Another area that has received more attention lately is the effects of water immersion (especially CWI) on the physiological and performance adaptations in response to endurance and resistance training.

Burke *et al* (2000) compared the changes in force production resulting from isometric strength training in combination with CWI (8 ± 1^{o}C), HWI (43 ± 1^{o}C) and passive recovery. Subjects exercised their right lower limb using an isometric strength training device for 5 consecutive days and the recovery interventions preceded each daily training session.

Although all three groups showed significant improvements in hip extensor isometric force production, the CWI group showed the greatest change (37%). Burke *et al.* (2000) explained this finding in light of the postulated decrease in nerve conduction velocity, increased subcutaneous sensory receptor thresholds, inhibition of sympathetic vascular mechanisms

and anaesthetic effects of CWI. They reasoned that these effects could interfere or depress the neural activity, which may impair the perception of the intensity of muscular contractions. Subsequently, the cold water group trained at a greater workload each day in comparison to the other groups which resulted in the greatest strength improvements. This assumption is supported by the fact that the daily 100% MIC produced by the cold water subjects was in fact greater than that of the other groups.

Yamane *et al.* (2006), however, concluded that cooling may actually interfere with the adaptation processes involved with endurance and resistance training. They had subjects who performed leg endurance training (25 min at 70% VO_{2max}) for four weeks and forearm flexor resistance training for six weeks in separate interventions. After each training session the volunteers were subjected to CWI, but only one leg and one arm was immersed in the cold water. The other leg and arm was therefore a control. The researchers found significantly greater improvements in performance time and one-leg VO_{2max} in the control leg compared to the cooled leg (15.9 ± 11.6% vs. 8.6 ± 7.9% and 8.2 ± 10.7% vs. - 2.2 ± 8.8%, for performance time and VO_{2max}, respectively). Similar results were observed for the resistance training interventions, i.e. maximal muscle strength and endurance.

Yamane *et al.* (2006) concluded that cooling affects the training-induced molecular and humoral adjustments involved in myofiber regeneration, muscle hypertrophy and improved blood supply, which are vital for training effects to occur. Cooling may attenuate these temperature dependent processes as well as heat shock protein formation and may therefore be disadvantageous when used in combination with training.

On the other hand, Howatson & van Someren (2009) came to a different conclusion after their study, although their experimental protocol specifically aimed to cause muscle damage, which is different to the training study of Yamane *et al* (2006). Howatson & van Someren (2009) postulated that if CWI did in fact inhibit the adaptation and regeneration processes involved with training, the repeated bout effect (RBE) observed after resistance training would be altered and greatly affected. Repeated bout effect is the phenomenon where a second bout of eccentric exercises following the initial exercise, reduces the amount of muscle damage. This adaptive effect demonstrates the ability of the neuromuscular system to adapt and some regard this as the most effective strategy by which to reduce muscle damage.

The aim of their study was therefore to investigate the effects of CWI following muscle damaging exercises on the repeated bout effect. Sixteen physically active subjects performed two bouts of drop jump exercises separated by 14 – 21 days. Participants were randomly divided to either a CWI (12 minutes at 15^{o}C) or a control group (12 minute seated

rest). Treatments were administered immediately after the first exercise bout, as well as 24, 48 and 72 hours post-exercise.

Following the second bout of exercise there was an equal and significant reduction in MIVC for both the control and CWI groups compared to the first bout. There was also a reduction in DOMS for both groups following the second bout compared to the first bout of exercise. These results show that the RBE did in fact take place under both conditions and that the RBE was not negatively affected by CWI.

From the above studies it is clear that there is no consensus on the effects of CWI on the adaptations to training. Further research is therefore needed to establish whether CWI alters the inflammatory response in muscles, or whether it interferes with the adaptation and regeneration processes (i.e. satellite proliferation and heat shock proteins) involved in the muscle tissue repair process.

F. CONCLUSION

Thus far, studies have not considered the effects of hydrostatic pressure and the effects of water temperatures separately on post-exercise recovery after prolonged exercise. It is therefore unclear whether the actual temperature of the water (cold, contrast or hot) plays a significant role in the proposed beneficial effects of water immersion, or whether it is the hydrostatic pressure *per se,* or a combination, that aids recovery. Thus, studies are needed to manipulate one of the two variables to determine which factor makes the actual difference during recovery. It is also important to establish whether water immersion is in fact more effective than active recovery, therefore aiding recovery without extra energy expenditure performance.

CHAPTER FIVE

PROBLEM STATEMENT

A. SUMMARY OF LITERATURE

Recovery after exercise has become an important area of research, and different recovery modalities have been studied to varying extents (Howatson & van Someren, 2008; Barnett, 2006; Wilcock *et al.,* 2006[a]). Among others, attention has specifically focussed on water immersion as a recovery modality (Ingram *et al.,* 2009; Montgomery *et al.,* 2008; French *et al.,* 2008; Howatson & van Someren, 2008; Hing *et al.,* 2008; Vaile *et al.,* 2008; Vaile *et al.,* 2007; Skurvydas *et al.,* 2006; Barnett, 2006; Wilcock *et al.,* 2006[a] Coffey *et al.,* 2004; Viitasalo *et al.,* 1995). The possibility for enhanced recovery without expending extra energy, as well as the possible advantageous physiological effects of the hydrostatic pressure and water temperature (i.e. hot and/or cold) makes water immersion a promising post-exercise recovery strategy. Most of the water immersion studies have focussed on EIMD and the associated DOMS (Ingram *et al.,* 2009; French *et al.,* 2008; Goodall & Howatson 2008; Vaile *et al.,* 2008; Vaile *et al.,* 2007; Bailey *et al.,* 2007; Sellwood *et al.,* 2007; Skurvydas *et al.,* 2006; Eston & Peters, 1999; Kuligowski *et al.,* 1998; Isabell *et al.,* 1992). The findings of most of these studies remain equivocal, and researchers believe that this may be attributed to the fact that the exact protocol (in terms of frequency, duration, temperature and immersion depth) has not yet been established.

In addition, not many studies have focussed on the effects of water immersion on prolonged aerobic performance, especially during multi-days of competition or intensive training. The accumulative fatigue caused by prolonged or intermittent aerobic exercise may be related to metabolic and neural-muscular factors that are different from fatigue that are caused by eccentric exercise induced muscle damage. There is only one study, to the researcher's knowledge, that investigated the effects of water therapy on post-exercise performance and recovery following high intensity treadmill runs (Coffey *et al.* 2004). They concluded that both active recovery and contrast water therapy reduced lactate accumulation after the first high intensity running session and that there were no significant differences in the subsequent performances of the athletes.

It has also been reported that the use of water immersion (especially cold water immersion) as a recovery strategy may be detrimental to subsequent performances where high velocity movements and power outputs are of importance (Howard, 1994). Some authors have studied the long-term use of cold water therapy and the effects on the adaptive and regenerative processes involved with endurance and resistance training (Howatson & van Someren, 2009; Yamane *et al.* 2006; Burke *et al.* 2000). Yamane *et al.* (2006) concluded that cooling negatively affects the training-induced molecular and humoral adjustments involved in myofiber regeneration, muscle hypertrophy and improved blood supply, which are vital for training effects to occur. Cooling may also attenuate heat shock protein formation and may therefore be disadvantageous when used in combination with training. However, this issue still needs to be investigated further, as two studies suggested that by allowing enough time between the cold water therapy and subsequent training session or competition (≥ 4 hours), performance may be maintained or even improved (Coffey *et al.* 2004; Lane & Wegner, 2004).

Another popular recovery modality that is used by athletes is active recovery (Barnett, 2006; Wilcock *et al.,* 2006[a]) or exercise of a light intensity. Active recovery is easy to perform and no special equipment or facilities are needed. However, active recovery results in extra energy cost and may not be as relaxing as water therapy (Wilcock *et al.,* 2006[a]). Most investigations on active recovery have focussed on its effect on the rate of post-exercise lactate clearance (Barnett, 2006), however, it is recognized that lactate is not the sole indicator of the overall recovery process (Barnett, 2006; Monedero & Donne, 2000). Only a few studies have compared active recovery with water therapy and the effects on post-exercise recovery and performance (Hamlin, 2007; Coffey *et al.,* 2004; Lane & Wegner, 2004; Isabell *et al.,* 1992). The results of these studies do not clearly show whether water immersion is more advantageous for recovery and performance than active recovery (Wilcock *et al.,* 2006[a,b]).

B. LIMITATIONS OF PREVIOUS STUDIES

The therapeutic effects of water therapy can be attributed to either the hydrostatic pressure exerted by the water, or the actual temperature of the water. Thus far, studies have not considered these two effects separately (Ingram *et al.,* 2009; Vaile *et al.,* 2008; Vaile *et al.,* 2007; Bailey *et al.,* 2007; Wilcock *et al.,* 2006[a]; Lane & Wegner, 2004; Coffey *et al.,* 2004; Burke *et al.,* 2000; Viitasalo *et al.,* 1995). Therefore, studies are needed to manipulate one of the two variables to determine which factor makes the actual difference during recovery.

In addition, there is limited evidence to support the assumption that water immersion improves recovery and performance (Wilcock *et al.,* 2006[a,b]). Moreover, there is no

considerable evidence to support the use of specific recovery modalities to improve the recovery between training sessions of more competitive athletes (athletes with more training experience in their particular sport) (Barnett, 2006), as most studies are limited to untrained subjects or recreationally active individuals (Ingram *et al.* 2009; Morton, 2007; Sellwood *et al.,* 2007; Skurvydas *et al.,* 2006; Howatson & van Someren, 2005; Lane & Wenger, 2004; Eston & Peters, 1999). Those studies that have included high performance athletes, focussed on sport specific context settings, where very little is known regarding sports such as cycling (Rowsell *et al.* 2009; Montgomery *et al.* 2008; Hamlin, 2007; Coffey *et al.* 2004).

It has been shown that water immersion *per se* has more beneficial post-exercise recovery effects than performing PAS (Vaile *et al.,* 2008; Vaile *et al.,* 2007; Coffey *et al.,* 2004; Lane & Wenger, 2004; Kuligowski *et al.,* 1998). It is, however, less clear whether water immersion strategies are more advantageous in the overall recovery process compared to active recovery (Wilcock *et al.,* 2006[a]; Cochrane, 2004).

The current study is thus the first to investigate the effects of different water immersion temperatures on the recovery of competitive cyclists in comparison with an active recovery modality during multi-day simulated training. This study will help us to better understand the effects of water immersion of different temperatures on the physiological, psychological and functional aspects of recovery and performance in the competitive cyclist. The fact that recovery modalities can be used during competitions or as a daily recovery routine to maintain performance and enhance recovery may be further explored by this study, to determine if these strategies are worth the time and effort.

C. OBJECTIVE OF THE STUDY

Aims of the study:

1. To determine if there is a difference between cold versus neutral water immersion in a supine position on the recovery and performance of competitive cyclists during 3 days of intensive endurance training.

2. To determine if there is a difference between water immersion and active recovery (moderate intensity exercise) on the recovery and performance of competitive cyclists during 3 days of intensive endurance training.

Specific questions of the study:

(i) Does water immersion have any recovery effects on muscular damage and associated inflammation? Specifically, will water immersion result in a decreased

response in muscle damage makers such as creatine kinase, and inflammatory markers such as C-reactive protein?

(ii) Is water immersion an effective metabolic recovery strategy following prolonged intensive cycling and would it therefore reduce the post-exercise recovery time for the cyclist to be able to maintain performance for consecutive days. In other words, will water immersion enhance metabolite clearance such as lactate?

(iii) Is water immersion effective in maintaining performance for consecutive days of intensive endurance cycling? Moreover, would water immersion maintain functional (Wingate) and endurance (100km time-trial) performances?

(iv) Does water immersion reduce the subjective perception of muscle soreness and fatigue as assessed by the Visual analog scale (VAS) and Profile of moods (STEMS)?

CHAPTER SIX

METHODOLOGY

A. STUDY DESIGN

This study was of an experimental nature. Exercise and recovery interventions were used to compare the effects of water immersion and moderate exercise on the recovery and performance of competitive cyclists. Subjects were randomly assigned to the different recovery intervention groups. A sampling by convenience method was used to select subjects.

Parameters of recovery that were assessed were as follow:

(i) Markers of muscle damage and inflammation: Creatine kinase and C-reactive Protein

(ii) Indirect measurement of swelling: Mid-thigh and mid-calve circumference

(iii) Cardiovascular responses: Heart rate response during the recovery modalities

(iv) Subjective perception of muscle soreness or fatigue: Visual analog scale (VAS) and Profile of moods (STEMS).

(v) Anaerobic exercise performance: Wingate sprint test.

(vi) Markers of muscular fatigue: Blood lactate concentration.

B. SUBJECTS

Seventeen competitive cyclists between the ages of 20 - 42 years old, volunteered to participate in the study. All the subjects underwent the same 3 day intensive endurance training program. Subjects were randomly assigned to a cold water (n = 6), neutral water (n = 6) or active recovery group (n = 5).

Cyclists were included in the study, if:

1. They participated competitively in more than 4 cycling races per year;

2. They were healthy, injury-free and at least 20 years old;
3. They trained for cycling at least 4 times a week;
4. They had a minimum of 1 year competitive cycling experience.

Cyclists were excluded from the study, if:

1. They had any cardiovascular problems;
2. They developed any upper respiratory tract infection prior to or during the study;
3. They had a history of smoking;
4. They had any haematological complications;
5. They had any immune deficiencies.

C. EXPERIMENTAL DESIGN

1. Place of study

All laboratory tests were done in the Exercise Physiology Laboratory in the Department of Sport Science at Stellenbosch University. The ice-bath and pool facilities of the Department of Sport Science were used for the study. All testing was done at temperatures between 18 – 20^{o}C.

2. Procedures

All blood samples were taken by either a medical doctor or a trained phlebotomist. All the tests and measurements, including physical, psychological and physiological, were standardised tests and protocols for elite athletes as described by the Australian Sports Commission (2000).

All participants were asked to read and sign a consent form (Appendix A) before they were finally included in the study. All procedures were verbally communicated to all participants. Participants were instructed to refrain from any physical exercise 48 hours before the start of the testing intervention. In addition, they were also requested to not take any anti-inflammatory drugs or pain medication and to not use any other form of recovery during the intervention period. Subjects were asked to complete a personal information questionnaire (Appendix B) to assess their weekly training schedule and their current health status. Additional recordings of participants' moods, general health and fatigue were also recorded over the course of the 3 days. Each of the subjects fluid losses (hydration status) were monitored during the three days by calculating the percentage loss of body weight, measured by body weight prior to and directly after the time trial sessions.

3. Laboratory visits

All subjects visited the laboratory on 4 separate occasions, at more or less the same time of day (~ 2hrs). Visit 1 served as an introductory session, where subjects were familiarized with testing and measurement procedures. One to two weeks later, all subjects completed the 3-day experimental protocol and recovery sessions.

Visit 1: The study protocol was explained to each subject. Subjects were also asked to complete and sign the consent form as well a questionnaire regarding their weekly training schedule and their current health status. The subject's percentage body fat, height and weight were also recorded. Thereafter, subjects completed a 100km time trial (100 km TT) using their own bicycle on the King Cycle trainer. This time trial served as a familiarization session. Subjects were instructed to consume water during the time trial as they would normally do during a long ride.

Visit 2: (1 – 2 weeks later) Baseline blood samples, VAS & STEMS questionnaires, circumference measurements and Wingate performance (functional) were assessed prior to the start of the high intensity time trial sessions. After the Wingate sprint subjects rested passively for 10 minutes. Thereafter, maximal aerobic capacity (VO_{2max}) was assessed on an electronically-braked cycle ergometer (Lode, Netherlands), followed by a 15 minute rest period before completing the 40km time trial (40 km TT) on the King Cycle trainer. Subjects performed one of the three recovery interventions directly after the 40km TT.

Visit 3 and 4: (24 and 48 hours later) Baseline measurements were repeated prior to the 100km TT on the King Cycle trainer. Thereafter, subjects performed one of the three recovery interventions directly after the TT.

Table 1. A summarized presentation of lab testing and procedures for the study.

VISIT 1	VISIT 2	VISIT 3	VISIT 4
Pre-liminary visit	(1 – 2 weeks) Baseline	24 hours	24 hours
Informed Consent	Blood samples	Blood samples	Blood samples
Personal information	VAS & STEMS	VAS & STEMS	VAS & STEMS
% Body Fat	Girths	Girths	Girths
Height	Wingate	Wingate	Wingate
Body weight	Body weight	Body weight	Body weight
100km time trial	VO_2max & lactate threshold	100km time trial	100km time trial
	40km time trial	Body weight	Body weight
	Body weight	Recovery intervention	
	Recovery intervention		

D. TESTS AND MEASUREMENTS:

1. Maximal aerobic exercise capacity (VO_2max)

Subjects performed a maximal incremental exercise test on an electronically-braked cycle ergometer (Lode, Netherlands) prior to the time trial intervention program. Subjects were informed to abstain from eating and any caffeine intake 90 minutes before testing. They were instructed to warm-up for 8-10 minutes at 50% of their perceived maximum effort. The maximal incremental test consisted of 2 minute 30 second work intervals with a 30 Watt increase in workload after every stage. All subjects had to maintain a pedal rate between 80 and 100 rpm. If the subject failed to maintain 80 rpm for more than 10 seconds the test was stopped. All subjects were verbally encouraged during testing. A blood sample was obtained after each workload increment from a finger prick (Softclix ®, Boehringer Mannheim), from which blood lactate concentration was measured with the Lactate Pro meter (Akray (Pty) Ltd, Japan).

The Quark b^2 cardio-pulmonary metabolic system (Cosmed, Italy) was used to continuously measure physiological variables. These measurements included:

- minute ventilation (VE), carbon dioxide production ($VCO_{2)}$, oxygen consumption ($VO_{2)}$, and respiration coefficient (R).

Heart rate was also measured throughout the test (Polar ® Heart rate monitor, Finland). Calibration of the gas analysers was performed prior to each test with known gas concentrations (16% O_2, 4% CO_2, balance N_2). The turbine flow meter was calibrated with a 3-L calibration syringe.

The outcome variables:

1. Maximum Oxygen uptake ($L.min^{-1}$ & $ml.kg^{-1}.min^{-1}$), maximum power output (Watt), maximum minute ventilation ($L.min^{-1}$) and maximum heart rate (bpm).
2. Lactate threshold was determined as the exercise intensity (heart rate and power output) where the blood lactate was 4 $mmol.L^{-1}$.

2. Kinanthropometric measurements

Body fat percentage was measured using a multi frequency bio-electrical impedance device (Bodystat Quadscan 4000, U.K.). Subjects were asked to lie supine in a relaxed position for 15 minutes prior to the measurements. Lead electrodes were placed just behind the second and third phalangeal joints and on the back of the wrist between the styloid process of the radius and ulna. The central leads were placed just behind the first and second metatarsal joints and between the medial and lateral malleolae. The skin was wiped with alcohol swabs before the placement of the electrodes on the specific anatomical sites.

Height and weight was measured using a stadiometer and an electronic scale (UWE BW – 150 free-weight, 1997 model, Brisbane Australia). Additionally, weight was recorded each day before the start of the time trial sessions, as well as directly after the sessions to monitor fluid losses during the intervention. The additional weight recordings were not used for statistical data analysis purposes, but were only used to ensure that the cyclists did not dehydrate during the time-trial sessions. Subjects were weighed while wearing minimal clothes and without shoes. The weight was measured to the nearest 0.1 kg. The stretch stature method was used for measuring standing height with a stadiometer (Siber-Hegner GPM, Switzerland) to the nearest 0.1 cm with the head in the Frankfort position, taking the measurement with the subject inhaling.

Mid thigh and calve measurements were taken with a metal steel anthropometric tape (Rosscraft, Canada) to the nearest 0.1 cm. Mid calve measurements were taken at the level of the medial calve skinfold site, and mid thigh measurements were taken at the level of the mid-trochanterion-tibiale laterale site (according to ISAK standards).

3. Functional assessment (maximal anaerobic capacity)

The Wingate anaerobic test was used to determine each subject's functional anaerobic capacity. It consists of a 30 second maximal effort sprint test on a cycle ergometer, where the subject had to cycle against a resistance calculated from each subject's individual body weight. The Wingate test was performed on an electronically braked cycle ergometer (Lode, The Netherlands). All cyclists performed a 10 minute warm–up on the cycle ergometer prior to testing. Outcome variables were determined by the Quark b^2 Computer Software program interfaced with the cycle ergometer.

Outcome variables: Peak power output (W), mean power output (W), total work ($J.kg^{-1}$).

4. Haematological measurements

All measurements were taken according to ISO standardized procedures as specified by Pathcare laboratories. Venous blood samples (8mL) were taken from a vein in the antecubital fossa from each subject, for the determination of creatine kinase and C-reactive protein concentrations. Blood lactate was measured via finger pricks (Softclix ®, Boehringer Mannheim) with the Lactate Pro meter (Akray (Pty) Ltd, Japan).

Outcome variables: Creatine kinase ($U.l^{-1}$), C-reactive protein ($mg.L^{-1}$) and lactate ($mmol.L^{-1}$).

5. Assessment of mood states

Subjects were asked to complete an adapted version of the Profile of moods states questionnaire, namely the Stellenbosch Mood Scale (STEMS) (Terry *et al.,* 2003). The questionnaire consists of factors relating to positive feelings (alertness, energy levels, activity) and factors relating to negative feelings (tension, depression, vigour, fatigue and confusion). A scale of 0 - 4 were used on each mood factor (0, not at all – 4, extremely). The total mood disorder was calculated by adding all the negative factors and subtracting the positive factors. In addition to the total mood scores, the fatigue factor scores was calculated and converted to raw T-scores (minimum 35 – maximum 78).

6. Assessment of perceived muscle soreness

Subjects were asked to complete a visual analoge scale (VAS) from 1 to 10 indicating the area (gluteals, quads, calves and thighs) and perception of muscle soreness (*Figure* 1). VAS was determined for each muscle group during stretching, activity (walking up and down stairs) and pressure (applied pressure for 3 seconds to the muscle belly).

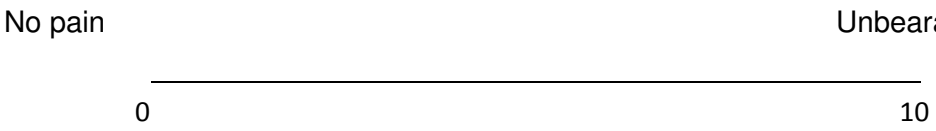


Figure 1. Schematic presentation of the VAS scale, indicating perception of pain or muscular discomfort.

7. Experimental interventions

1. Three-day intensive endurance training intervention program

The objective of the intensive endurance training program was to simulate hard cycling training or racing conditions where fatigue will be evoked. The time of each time trial session was standardised for each subject to ensure minimal day-to-day variation. Heart rate monitors (Polar ® Heart rate monitor, Finland) was used during each session to determine specific intensities. Subjects performed one of the three recovery interventions directly after each high intensity session.

Session 1: 40km TT

Subjects were asked to perform a 40km maximal effort TT on the King Cycle trainer (MK3 Interface, United Kingdom). The cyclists mounted their own bikes on the trainer. The coefficient of variation for this test on the King cycle trainer (time taken to complete the 40 km TT) is 1.0 ± 0.5%, which is highly reproducible (Palmer *et al.,* 1996). There is also a significant correlation between actual road race times and 40 km-laboratory times ($r = 0.98$, $P < 0.001$).

All subjects were instructed to warm-up at an intensity of 50% of their perceived maximal effort for 8-10 minutes. Cyclists were asked to complete the TT in the shortest possible time, using their own choice of pedal cadence and gear ratios. Time, distance, power output, pedal cadence and heart rate were measured with a PC Monitor System (King cycle physiological testing software MK3, Version 10.1, 2002, United Kingdom) that is interfaced with the King Cycle trainer. Polar watches (Polar ® Heart rate monitor, Finland) and an additional stopwatch were used as back-up for heart rate measurements as well as total time for the TT. A rating of perceived exertion (Borg scale: 1 -10) was obtained from every subject at 5 km intervals in the time trial.

Outcome variables: Performance time in minutes and seconds, average power output (watts) and pedal cadence (rpm), maximum and average heart rate (bpm) and rating of perceived exertion.

Session 2 and 3: 100km Time-trial

Subjects were asked to perform a 100km maximal effort time trial on the King Cycle trainer (MK3 Interface, United Kingdom). The 100km time-trial included 4 x 4km and 4 x 1km sprints at different distances (Schabort *et al.,* 1998). Subjects were instructed to complete the TT in the shortest possible time, but also to pace themselves in such a manner that they were able to do the sprints between 90 - 100% of maximal effort. All subjects were instructed to warm-up at an intensity of 50% of their perceived maximal effort for 8 - 10 minutes. Subjects were allowed to use their own choice of pedal cadence and gear ratios. Time, distance, power output, pedal cadence and heart rate was measured with a PC Monitor System that is interfaced with the King cycle trainer. Polar watches (Polar ® Heart rate monitor, Finland) and an additional stopwatch were used as back-up for heart rate measurements as well as total time for the time-trial. Perceived exertion rates (Borg scale: 1 -10) were noted at specific distances during the time trial.

Outcome variables: Time of test in minutes and seconds, average power output (watts) and pedal cadence (rpm), and Borg scale for perceived exertion (Scale from 1 - 10).

2. Recovery interventions

The study included three different recovery interventions, where each subject was randomly assigned to one of the three intervention groups. The recovery intervention was administered directly after each intensive endurance session on Day 1 (session 1) and Day 2 (session 2).

2.1 Ice bath group (cold water immersion; IB):

Subjects were immersed into an ice bath with a temperature of 10.1 ± 0.97^{o}C. All subjects were asked to lie in a supine position so that their body was immersed with water up to their necks; with the aim to eliminate the possible hydrostatic effects of water immersion *per se.* Subjects were asked to stay calm in a supine resting position for the duration of 20 minutes.

2.2 Neutral group (neutral water immersion; tNB):

Subjects were immersed in a swimming pool with a temperature range of 30 ± 0.62 °C. All subjects were asked to lie in a supine position so that their body was immersed up to their necks for a total duration of 20 minutes.

2.3 Active recovery group (AR):

Subjects were asked to perform an active recovery on the King cycle trainer. All subjects were asked to cycle continuously for 20 minutes at an intensity of 80 – 84 % of their individual lactate threshold.

8. Ethical aspects

The study protocol was approved by the Ethics Committee of Research Subcommittee A at the University of Stellenbosch (nr. 100/2008). All the subjects were given a consent form outlining the exact procedures of the study and the risks involved in the study. It was made clear that the subjects were voluntarily participating in this study and that they may withdraw from the study at any time. Medical doctors were on site at all times during testing.

E. STATISTICAL ANALYSIS

Outcome variables were analysed using a three-way ANOVA for repeated measures (Statistica version 8). Correlations were calculated using Microsoft Office Excel (Windows Vista 2007). Pairwise comparisons between recovery interventions were analyzed using the Fischer LSD test. Data presented in *Tables* were reported as mean and standard deviation (SD) and *Figures* were reported as mean and standard error of measurement (SEM). The level of significance was set at $P < 0.05$ for all analyses.

CHAPTER SEVEN

RESULTS

A. INTRODUCTION

A randomized controlled experimental study was done to examine the effects of three recovery strategies on the performance of competitive cyclists during three days of intensive training. The dependent variables included functional and aerobic performance, blood markers of muscle damage, namely plasma creatine kinase, C-reactive protein, blood lactate concentration and lower limb swelling, and subjective measures of perceived muscle soreness and overall fatigue.

B. DESCRIPTIVE CHARACTERISTICS

1. Subjects

The cyclists' physical characteristics are summarized in *Table 2.* Their experience at a competitive level varied between 1 to 12 years (mean ± SD; 3.7 ± 3.35 yrs).

Table 2: Subject characteristics (N = 17).

Characteristic	**Mean**	**SD**	**Range**
Age (*yrs*)	28.8	6.65	20 - 42
Weight (*kg*)	80	8.4	65.1 – 96
Height (*cm*)	180.9	5.91	168.8 – 190.2
% Body fat	13.0	3.1	6.9 – 18.3
VO_2max ($L.min^{-1}.kg^{-1}$)	49.7	5.82	36.1 – 60.9
PPO (*Watts*)	348.7	35.93	284 – 402
Peak Power:$Weight_{ratio}$	4.4	0.37	3.9 – 5
Power: $Weight_{LT}$	3.4	0.45	2.5 – 4.4

VO_{2max} ; Maximum aerobic capacity, PPO; Peak power output at maximum aerobic capacity, Power:$Weight_{LT}$; Power at lactate threshold.

At the time of the study the subjects trained four to five times a week (~10 hours) on their road bikes and they participated in more than 5 cycling races per year. All the subjects were in the pre-competition to competition transition phase of their yearly program. The cyclists' classification of maximal aerobic capacity ranged from average to superior, according to their age and gender. On average, their maximal aerobic capacity for the cyclists was classified as excellent (Brooke, 2005).

Subjects were randomly assigned to one of three recovery groups (Table 3). The IB group had a significantly higher average VO_{2max} compared to the tNB group ($P < 0.05$). However, there were no other significant differences found between the recovery groups for any of the other performance or physical variables that were measured prior to the intervention.

Table 3: Physical and performance characteristics of the cyclists in the different recovery groups. Values are expressed as means ± SD.

Characteristic	**Active recovery (AR)**	**Thermo-Neutral Bath (tNB)**	**Ice Bath (IB)**
	Mean ± SD	**Mean ± SD**	**Mean ± SD**
n	5	6	6
Age (*yrs*)	27.6 ± 5.94	31.3 ± 8.82	27.3 ± 4.89
Weight (*kg*)	78.8 ± 6.67	84.0 ± 9.12	76.9 ± 8.61
Height (*cm*)	180.5 ± 4.42	182.2 ± 4.31	180.1 ± 8.65
% Body fat	12.1 ± 3.51	13.7 ± 3.31	13.2 ± 2.93
VO_2max ($L.min^{-1}.kg^{-1}$)	49.8 ± 4.13	45.9 ± 6.30	53.3 ± 4.65*
PPO (*Watts*)	352.6 ± 35.94	359.3 ± 28.56	334.7 ± 43.52
Peak Power:$Weight_{ratio}$	4.5 ± 0.41	4.3 ± 0.41	4.4 ± 0.33
Power: $Weight_{LT}$	3.2 ± 0.54	3.5 ± 0.52	3.6 ± 0.26

VO_{2max} ; Maximum aerobic capacity, PPO; Peak power output, LT; Lactate threshold. * Significant difference between IB and tNB; $P < 0.05$.

C. PERFORMANCE OUTCOMES

All subjects performed a 40km time-trial (40km TT) on Day 1, and a 100km time-trial (100km TT) 24 hours (Day 2) and 48 hours later (Day 3). During the 40km TT the subjects cycled at an average heart rate intensity of 97% ± 6.43 (range; 80 - 107%) of their individual lactate threshold. During the 100km TT on Day 2 and Day 3, subjects cycled at an average heart rate intensity of 91% ± 6.44 (range; 83 - 98%) and 90.1 % ± 4.28 (range; 81 - 96%), respectively. There were no differences in the average heart rate intensities between the three recovery groups ($P > 0.05$). The heart rate intensity during the 40km TT on Day 1 was, however, statistically significantly higher compared to Day 2 ($P < 0.05$) and Day 3 ($P < 0.01$), however, this is expected as the distance is much shorter for the 40km TT compared to the 100km TT.

The performance outcome variables for the 3 days are summarized in *Table 4.* There were no statisticaly significant differences in performance times, average power output, as well as perceived exertion rating during the TTs between the recovery groups. For both 100km – TTs, the tNB group recorded the best performances, however, their times (hr:min:sec) were not significantly better than the AR and tNB groups ($P > 0.05$). The relative percentage changes in performance times, average power output and RPE scores for the 100km TTs during the last 2 days are illustrated in *Figure* 2 (a) – (c) for all recovery groups. The relative changes were compared to calculate the differences for each recovery group over the last two 100km TTs and to determine which recovery group showed improvements or decrements relative to their own times recorded.

Table 4: Performance times, average power and RPE scores for the different time trials performed on each day. Values are presented as means ± SD.

Recovery group		**Performance times [hr:min:sec]**	**Average power [W.kg^{-1}]**	**RPE scores**
Active recovery		01:02:36 ± 00:03:47	2.6 ± 0.44	6.8 ± 1.67
tNeutral bath	Day 1	01:02:57 ± 00:06:04	2.8 ± 1.06	6.5 ± 1.15
Ice bath		01:01:03 ± 00:04:52	2.9 ± 0.46	6.1 ± 0.66
Active recovery		02:45:40 ± 00:13:21	2.4 ± 0.44	6.5 ± 1.13
tNeutral bath	Day 2	02:39:12 ± 00:04:26	2.5 ± 0.33	6.7 ± 1.09
Ice bath		02:51:12 ± 00:17:11	2.3 ± 0.50	5.7 ± 0.93
Active recovery		02:50:22 ± 00:20:49	2.3 ± 0.43	6.4 ± 0.82
tNeutral bath	Day 3	02:41:43 ± 00:04:55	2.4 ± 0.36	7.0 ± 1.42
Ice bath		02:50:04 ± 00:15:54	2.4 ± 0.47	6.5 ± 1.35

RPE, Rate of Perceived Exertion. No significant difference; $P > 0.05$.

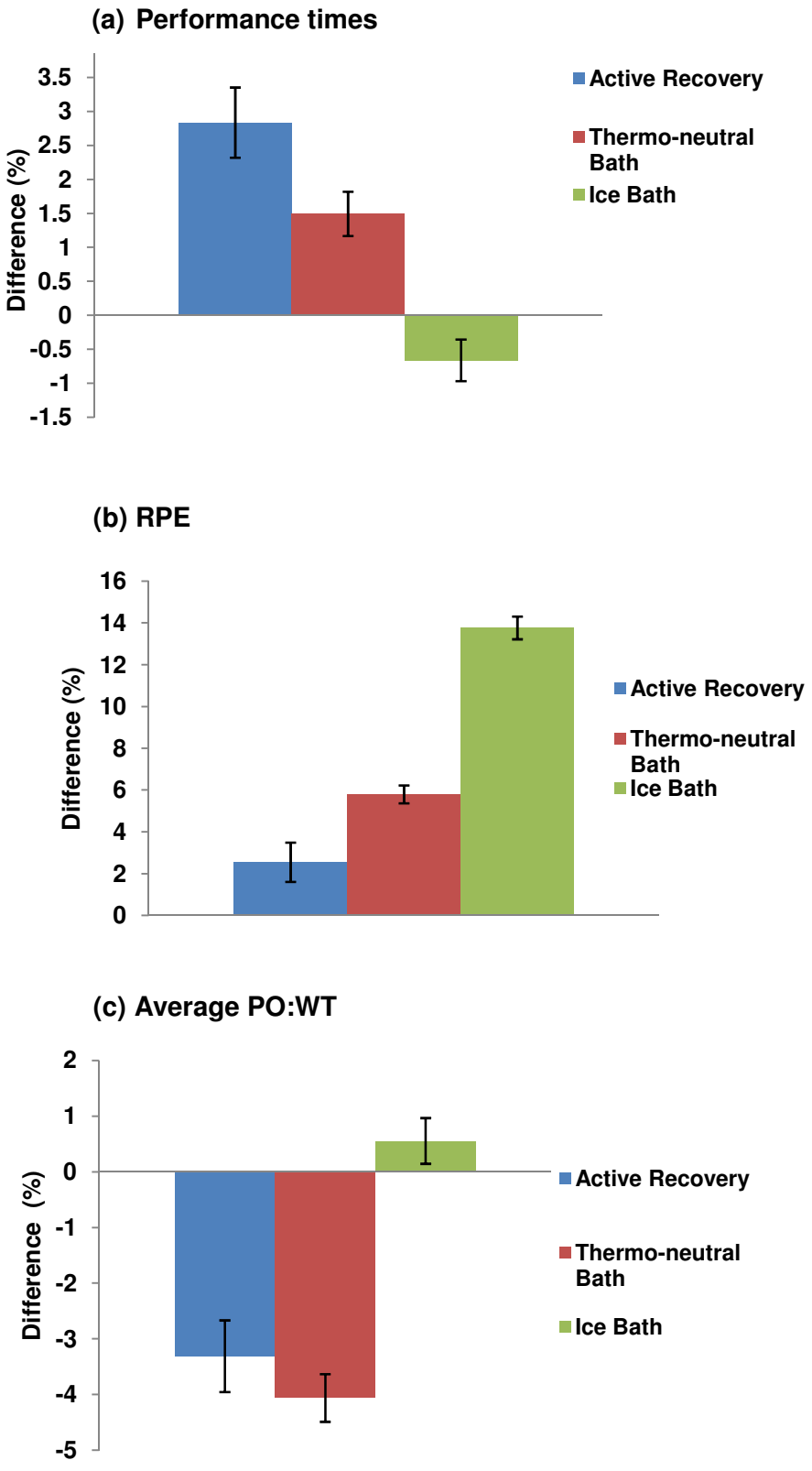


Figure 2: The relative change (%) in (a) performance times, (b) perceived exertion scores, and (c) average power (relative to bodyweight) for the 100km TTs between Day 2 and 3. A negative vaule for (a) indicates a better performance on Day 3 compared to Day 2. Values are expressed as means ± SEM.

Figure 2 (a) illustrates that the performance changes in average times for the IB group improved by 0.7% on the last day compared to the changes of the tNB and AR groups. The improved changes in times were achieved through a 0.6% increase in average power performed. However, in terms of the subjective feelings of exertion, the IB group reported a 13.8% higher change in RPE for the last 100km TT (*Figure* 2 (b)). However, the statistical analysis revealed that none of these differences were statistically significant, and there were no significant interaction effects between the different recovery groups. Overall, the changes in TT times over the last two 100km TTs for the IB group was 3.5% faster than for the changes recorded in the AR group, and 2.2% faster than the tNB group.

Figure 2 (c) illustrates that the changes in the average power performed (relative to bodyweight) between Day 2 and Day 3 was greater (0.6%) for the IB group compared to the - 5.3% decline in the AR group and - 3.8% decline in the tNB group. However, the differences in the these changes were not statistically significant ($P > 0.05$).

D. DETERMINANTS OF POST-EXERCISE RECOVERY

1. Functional capacity (*Table* 5, *Figure* 3 & 4)

Functional capacity was assessed through a Wingate 30 second sprint test, prior to the TTs on Day 1 to 3. The absolute changes in average power and total work performed are reported in *Table 5.* All performance outcome measurements are expressed relative to the subjects' bodyweight. The relative changes (%) in average power and total work performed from Day 1 are illustrated in *Figure* 3 and *Figure* 4.

Table 5: Wingate performance results for each day. Values are presented as means ± SD.

Recovery group		Average power: Body Weight [W.kg^{-1}]	Total Work: Body Weight [J.kg^{-1}]
Active recovery		9.3 ± 0.70	279.0 ± 19.60
tNeutral bath	Day 1	9.0 ± 1.08	269.9 ± 32.61
Ice bath		9.1 ± 0.39	271.6 ± 11.78
Active recovery		9.2 ± 0.49	274.6 ± 15.58
tNeutral bath	Day 2	8.6 ± 0.97	256.1 ± 29.18
Ice bath		8.7 ± 0.33	262.5 ± 9.84
Active recovery		9.1 ± 0.53	271.5 ± 15.36
tNeutral bath	Day 3	8.7 ± 1.25	262.6 ± 37.63
Ice bath		8.9 ± 0.44	266.4 ± 12.58

No significant differences; $P > 0.05$.

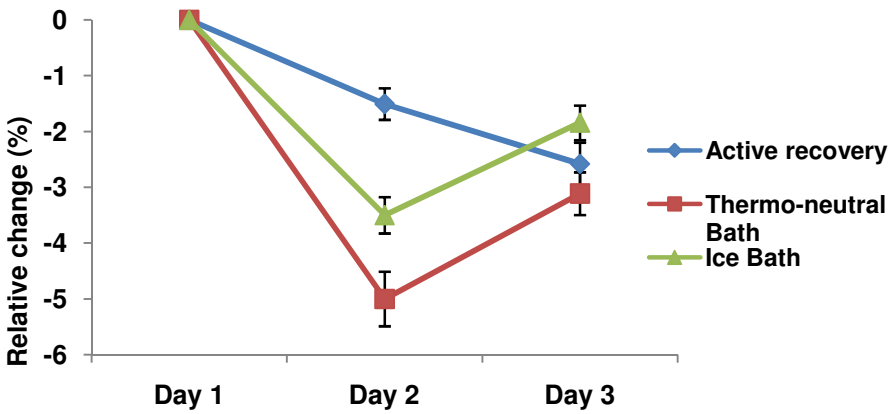


Figure 3: The relative changes (%) in average power during the Wingate sprint tests. Average power was calculated relative to bodyweight. Values are expressed as means ± SEM.

There were no statistically significant differences in AP:WT on Day 1 between the recovery groups. Figure 3 shows that the tNB and IB group reached their lowest AP:WT on Day 2 (- 5% and - 1.5%, respectively). Although these two groups performed slightly better on Day 3, they did not reach baseline values. The performance of the AR group was 1.5% lower on Day 2 and declined a further 2.6% on Day 3. There were, however, no statistically significant time or interaction effects observed for any of the recovery groups ($P > 0.05$).

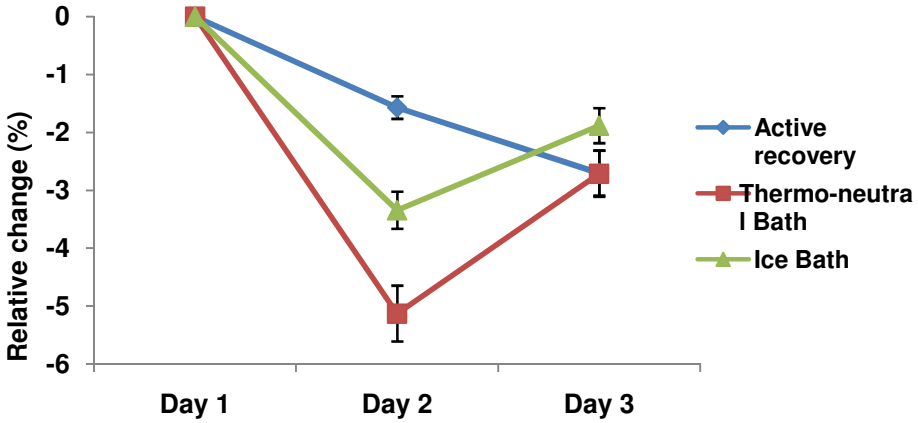


Figure 4: The relative changes (%) in total work performed during the Wingate sprint tests. Total work was calculated relative to bodyweight. Values are expressed as means ± SEM.

There were no statistically significant time effects for any of the recovery groups for total work performed and the pattern of change over the 3 days were comparable to the changes in average power. Both the water immersion groups completed the least amount of work on Day 2 (tNB; - 5.1% and IB; - 3.4%), but recovered slightly on Day 3 (tNB; - 2.7% and IB; - 1.9%). However, their performances were still worse compared to Day 1. On the other hand, the AR group showed a smaller decrement in performance on Day 2 (- 1.6%), but they failed to recover to any extent the following day. On Day 3, their performance was 2.7% lower than on Day 1. There were no significant interaction effects between any of the recovery groups ($P > 0.05$).

2. Blood markers

Blood samples were taken on each day prior to the exercise training sessions. Blood lactate concentration was meaured as an indicator of metabolic recovery, while plasma CK was measured to indicate the degree of muscle damage. CRP was determined as a measure of inflammation.

2.1 Blood lactate concentration (*Table* 6, *Figure* 5)

Peak blood lactate concentrations were observed on Day 2 for both the AR and tNB groups, and on Day 3 for the IB group. There was a significant treatment effect on Day 2, as the blood lactate concentration was significantly lower for the IB group compared to the tNB group ($P < 0.05$). For both the AR and tNB recovery groups the blood lactate concentrations were at or below baseline on Day 3. The differences in the relative changes in Day 3 between the recovery groups were not statistically significant.

Table 6: Resting blood lactate concentration on the three days prior to the training session. Values are presented as means ± SD.

Recovery group	**Day 1 [mm.L^{-1}]**	**Day 2 [mm.L^{-1}]**	**Day 3 [mm.L^{-1}]**
Active recovery	1.3 ± 0.59	1.54 ± 0.23	1.3 ± 0.29
tNeutral bath	1.78 ± 0.25	2.05 ± 0.98	1.62 ± 0.48
Ice bath	1.26 ± 0.45	1.43 ± 0.39*	1.52 ± 0.23

*Significant *treatment effect* for IB group; $P < 0.05$.

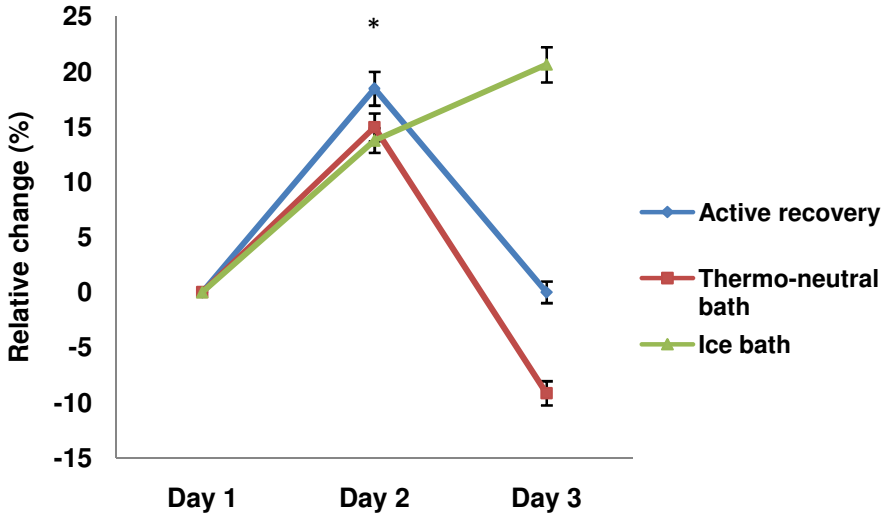


Figure 5: The relative change (%) in blood lactate concentration. Values are expressed as means ± SEM. (* Significant difference between tNB and IB; *treatment effect* $P < 0.05$).

2.2 Plasma CK (*Table* 7, *Figure* 6 & 7)

Plasma CK concentration changed statistically significantly over time for all of the recovery groups ($P < 0.05$), however, none of the interventions caused a significant treatment effect ($P > 0.05$). Peak plasma CK concentrations were observed on Day 2 for both the AR and tNB groups, whilst peak levels for the IB group were observed on Day 3. Plasma CK concentrations were still elevated on Day 3 for both the water recovery groups (IB; + 33.98% and tNB; + 14.96%).

Table 7: Total creatine kinase activity during the days prior to the training session. Values are presented as means ± SD.

Recovery group	**Day 1** [u.L^{-1}]	**Day 2** [u.L^{-1})	**Day 3** [u.L^{-1}]
Active recovery	156.4 ± 85.91	157 ± 78.48	148.4 ± 82.55*
tNeutral bath	157.7 ± 72.86	184.2 ± 72.16	181.3 ± 92.11*
Ice bath	144.5 ± 41.66	185.7 ± 59.06	193.6 ± 71.50*

*Significant change over time for all recovery groups; P < 0.05.

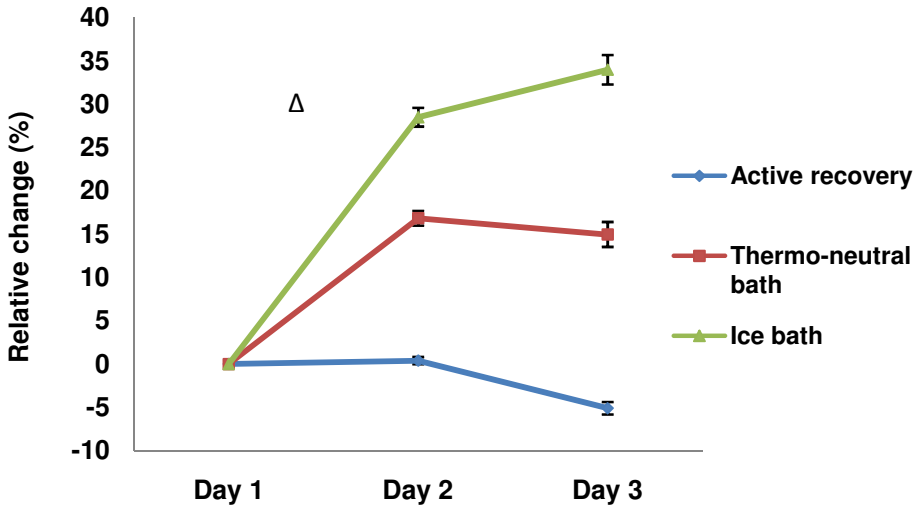


Figure 6: The relative change (%) in plasma creatine kinase concentrations. Values are expressed as means ± SEM. (Δ Significant change over time for all groups; $P < 0.05$).

For interest sake, the changes for the IB and tNB groups were pooled together (water group; WG) to see whether there was any significant differences in Plasma CK levels between

water immersion *per se* and AR. *Figure* 7 illustrates that the AR caused markedly lower plasma CK concentrations over the three days when compared to the WG, however, the differences were only borderline significant ($P = 0.05$).

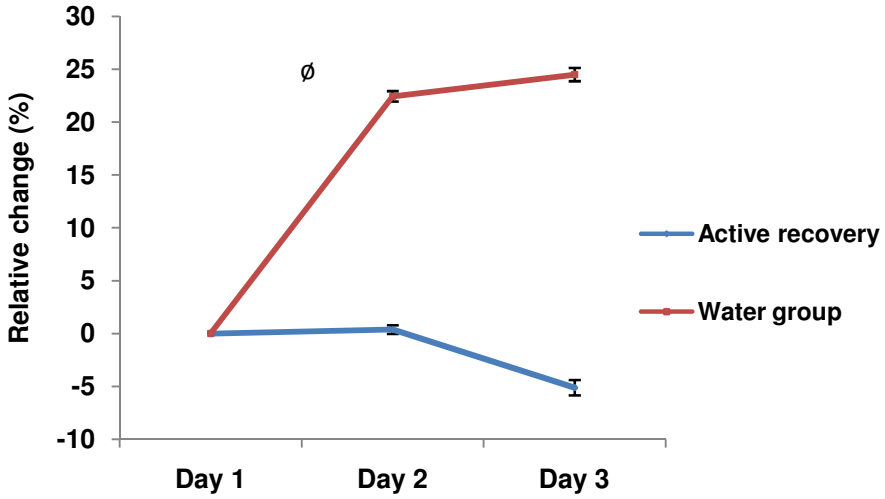


Figure 7: The relative change (%) in plasma creatine kinase for AR and the combined WG. Values are expressed as means ± SEM. (ø AR vs WG; $P = 0.05$).

2.3 CRP concentration (*Table* 8, *Figure* 8 & 9)

There was a statistically significant increase in [CRP] over the 3 days ($P < 0.001$) with peak levels for all three groups on Day 3. The AR group presented with the highest [CRP] on Day 3 (+ 470% increase). No interaction effects were observed for any of the recovery groups ($P > 0.05$).

Table 8: C-Reactive protein concentration measurements on the three days prior to the training session. Values are presented as means ± SD.

Recovery group	**Day 1 [mg.L^{-1}]**	**Day 2 [mg.L^{-1}]**	**Day 3 [mg.L^{-1}]**
Active recovery	0.6 ± 0.28	2.4 ± 3.01	3.4 ± 2.86*
tNeutral bath	1.0 ± 0.28	1.3 ± 0.71	2.3 ± 1.08*
Ice bath	0.7 ± 0.34	1.7 ± 1.25	3.3 ± 1.18*

* Significant changes over time for all groups; $P < 0.001$.

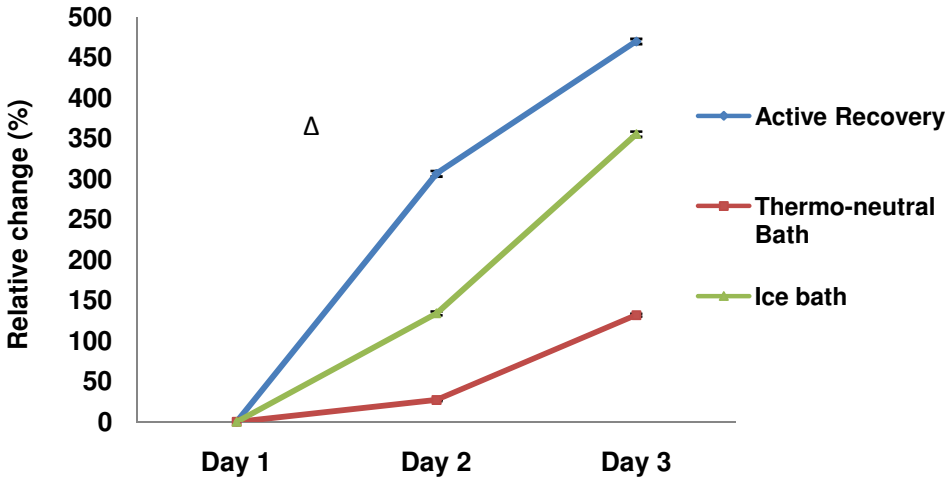


Figure 8: The relative change (%) in C-reactive protein concentration for the recovery groups. Values are expressed as means ± SEM. (Δ Significant change over time for all groups; $P < 0.001$).

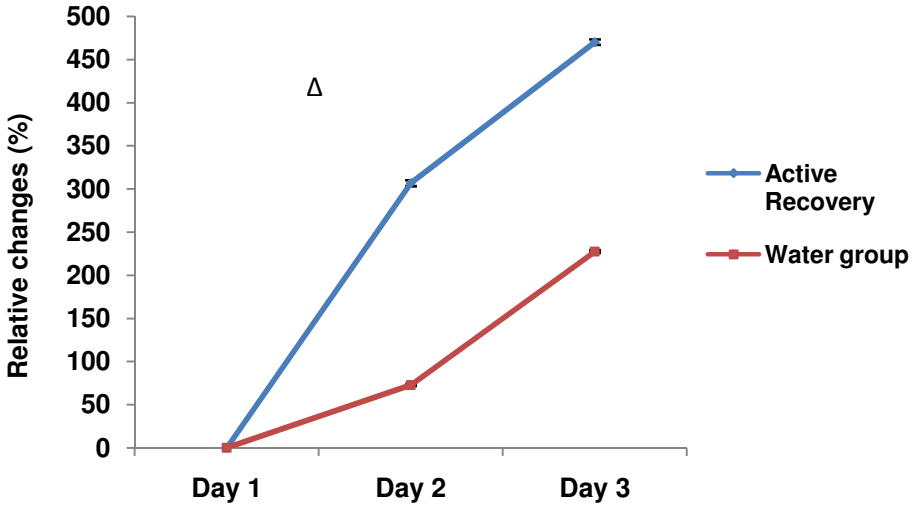


Figure 9: The relative change (%) in C-reactive protein concentration for AR and WG. Values are expressed as means ± SEM. (Δ Significant change over time for all groups; $P < 0.001$).

Figure 9 illustrates that the WG reponse in [CRP] over time was lower in magnitude compared to the AR group, however, the increases in [CRP] on Day 2 and Day 3 were not significantly less compared to the AR group. For both the water immersion groups, as well as the AR group, [CRP] was still significantly elevated on Day 3 compared to Day 1 (+ 470% and + 227.4%).

3. Lower limb swelling (*Table* 9, *Figure* 10 & 11)

Mid-thigh and mid-calve circumferences were measured prior to the TTs on each day. No significant time or interaction effects were observed for both mid-thigh and mid-calve circumferences for any of the recovery groups. Peak oedema levels of the thigh were observed on Day 2 for the IB group (+ 0.61%) and on Day 3 for both the tNB (+ 0.32%) and AR (+ 0.11%) groups. Although there was less swelling of the thigh muscle on Day 3 after the IB immersion, these values still did not reach baseline values.

Table 9: Absolute values for the circumferences of the mid thigh and mid calve over three days. Values are presented as means ± SD.

	Day 1		**Day 2**		**Day 3**	
Recovery group	**Thigh**	**Calve**	**Thigh**	**Calve**	**Thigh**	**Calve**
Active recovery	53.4 ± 1.61	37.8 ± 2.57	53.4 ± 1.73	37.9 ± 2.59	53.4 ± 1.70	37.8 ± 2.66
tNeutral Bath	55.9 ± 1.93	38.7 ± 2.36	56.0 ± 2.23	38.6 ± 2.47	56.1 ± 2.07	38.6 ± 2.49
Ice bath	53.6 ± 2.16	37.2 ± 1.89	53.9 ± 2.56	37.3 ± 1.99	53.7 ± 2.28	37.2 ± 1.97

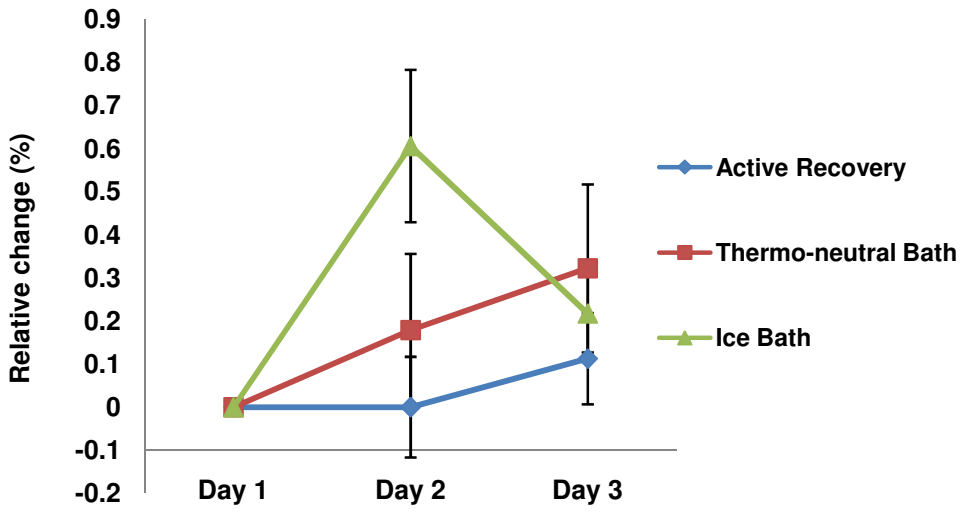


Figure 10: The relative change (%) in circumferences of the right thigh. Values are expressed as means ± SEM.

While the calf circumferences of the AR and IB groups peaked on Day 2 and then recovered below baseline on Day 3, there was no swelling observed in the tNB group on any of the days. The differences in swelling on Day 2 and Day 3, between the three groups, were however not statistically significant.

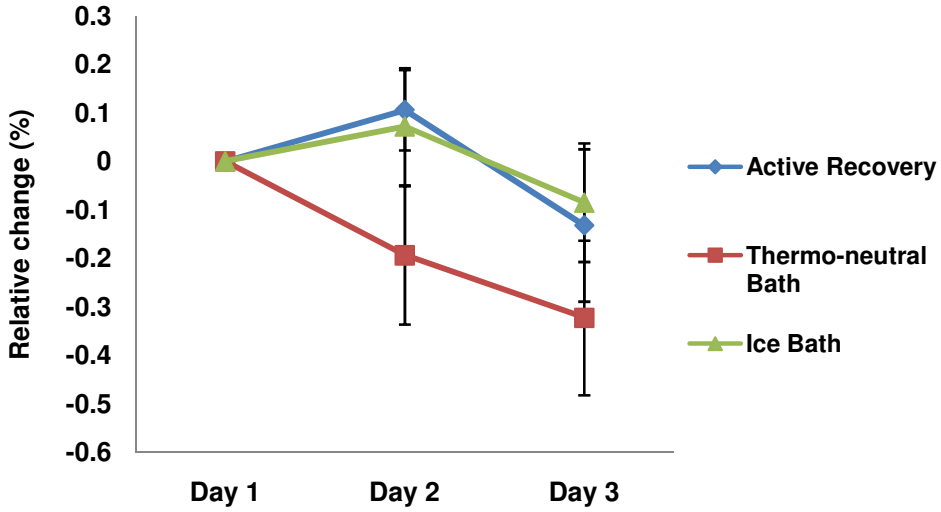


Figure 11: The relative change (%) in circumferences of the right calve for the recovery groups. Values are expressed as means ± SEM.

4. Profile of moods and perception of fatigue (*Figure* 12, 13 & 14)

STEMS were assessed prior to the TTs on each day as an indication of the cyclists mood state. There were no statistically significant time or interaction effects observed for total mood scores for any of the recovery groups.

The total mood disorder score was calculated by adding all the negative feelings (tension, depression, anger, vigor, fatigue and confusion) and substracting all the postive feelings (alertness, energy, activity). A Scale of 0 – 4 were used on each mood factor (0, not at all – 4, extremely). Therefore, a negative score shows that the subjects' overall moods were positive. Overall, the IB group showed positive moods over the course all three days, compared to the AR and tNB groups. On Day 2 both the AR and tNB showed the worst mood disorders, whereas total mood disorder was the worst on Day 3 for the IB group, however, it still indicated an overall positive mood.

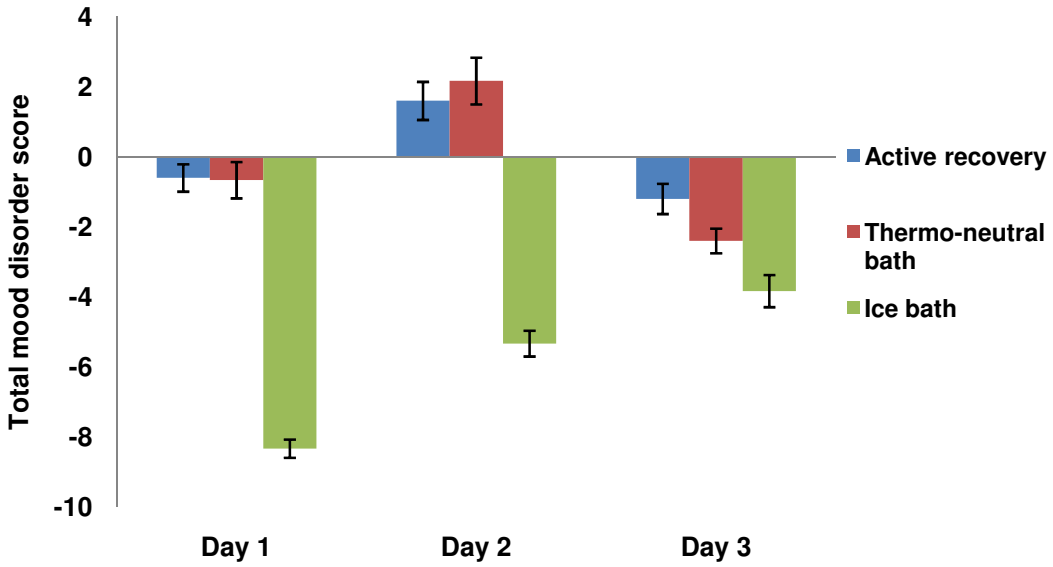


Figure 12: The total mood disorder score (STEMS) over 3 days. A negative value indicates an overall positive mood. Values are means ± SEM.

The fatigue T-scores were calculated from the raw scores in the STEMS. Fatigue were described as feelings of worn-out, exhausted, sleepy, and tired (scale; 0, not at all – 4, extremely). The raw scores were transformed to T-scores (minimum 35 – maximum 78). The highest fatigue T-scores were observed on Day 2 for the AR group and on Day 3 for both the tNB and IB recovery groups. The AR group recorded the lowest fatigue T-scores on Day 3.

There was a very strong tendency for fatigue T-score changes over time (*Figure* 13) (P = 0.05). There was also a strong tendency for lower fatigue T-scores in the AR group compared to the IB and tNB groups (P = 0.06).

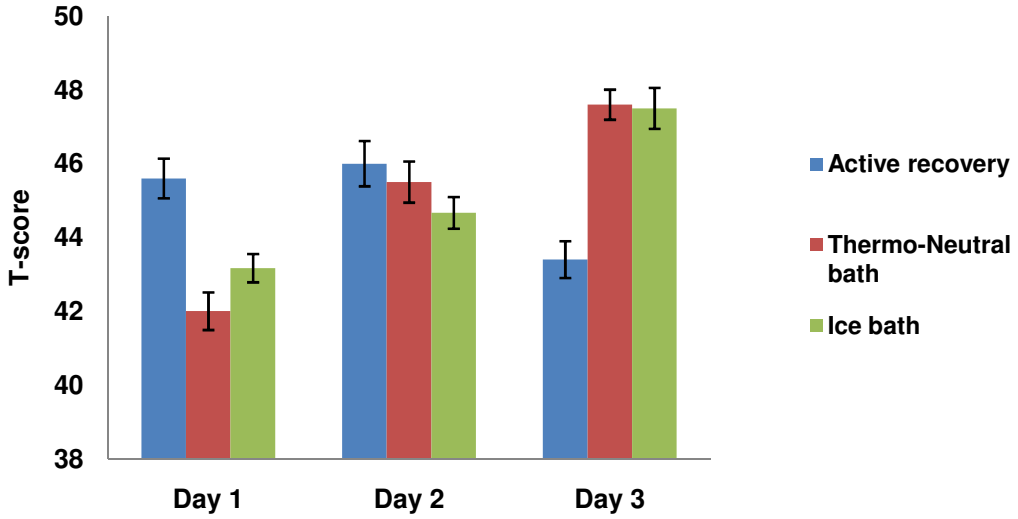


Figure 13: The fatigue factor T-scores over 3 days. Values are means ± SEM.

Statistically significant interaction effects were observed after combining the fatigue T-scores for the water groups (WG) (*Figure* 14), showing that the water immersion groups became progressively more fatigued over the three days. After similar scores on Days 1 and 2, the AR group had a statistically significant lower perception of fatigue on Day 3 ($P = 0.02$).

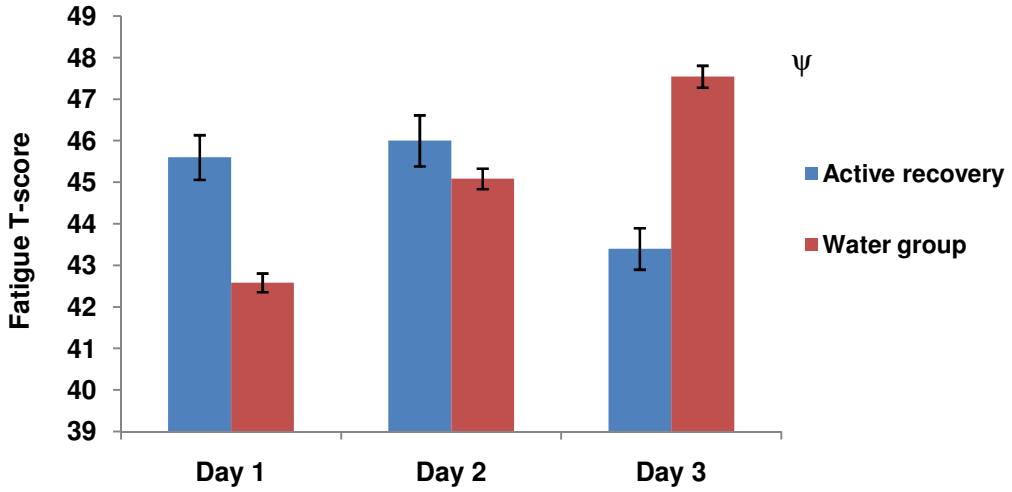
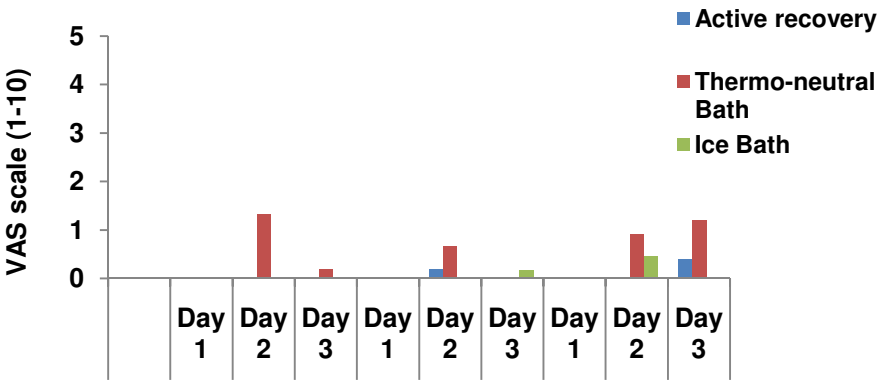


Figure 14: The fatigue factor T-scores over the three training days for AR and combined water group (WG). Values are expressed as means ± SEM.(Ψ Significant treatment effect for WG; $P < 0.05$).

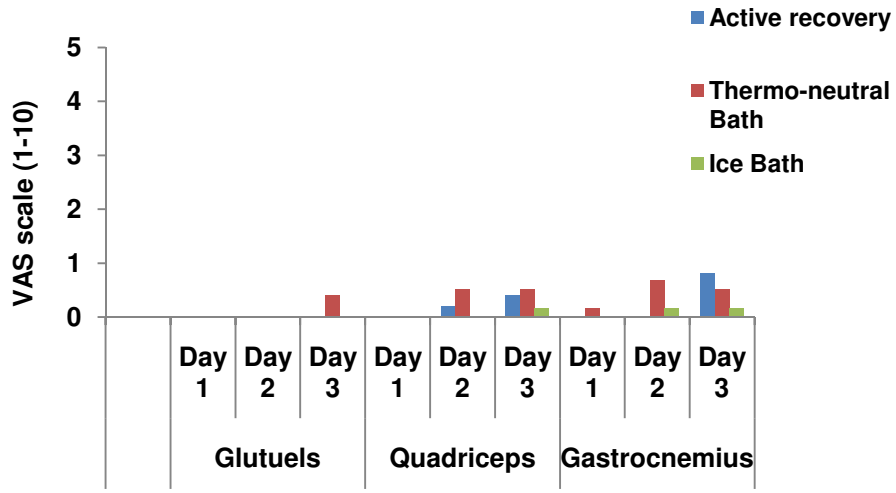
5. Perceived muscle soreness and discomfort (*Figure* 15 a- c)

Muscular pain was assessed using a VAS scale prior to the TTs on each day. No statistically significant results were found for any of the pain measurements during stretching, pressure and activity for any of the muscle groups (quadriceps, gastrocnemius and gluteuls).

(a) Stretching



(b) Applied pressure



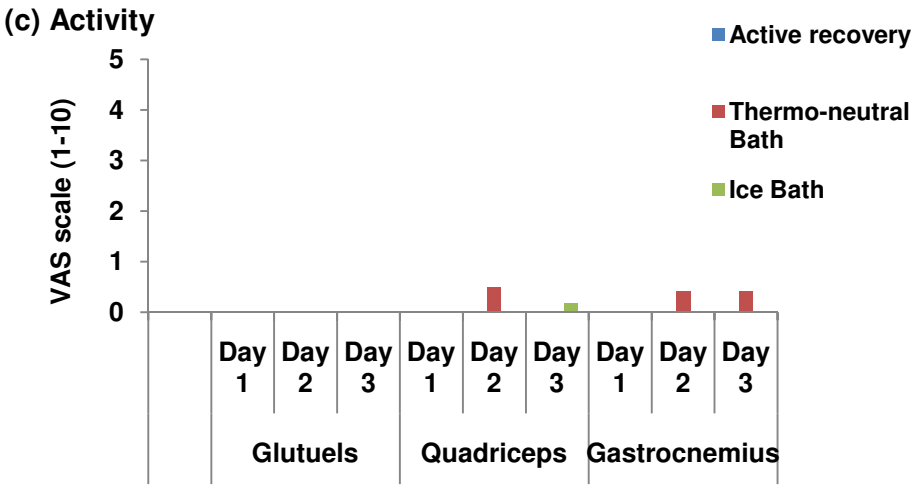


Figure 15: The VAS scores during (a) stretching, (b) applied pressure, and (c) activity for the muscle groups for each day. Values are expressed as means ± SEM.

The cyclists reported no soreness or discomfort after Day 1, while most cyclists in the tNB group reported soreness after Day 2 and 3. Cyclists in the AR group also reported soreness on Day 3, specifically with applied pressure to the muscle.

E. RESPONSES DURING RECOVERY INTERVENTIONS (Figure, 16 & 17)

Cyclists performed the recovery interventions directly after the TT sessions on Day 1 and Day 2. The average water temperatures for the IB and tNB recovery interventions were 11 ± 0.97^{o}C and 30 ± 0.62^{o}C, respectively. The average heart rate (% of LT) for the AR group during the recovery intervention was 81 ± 1.74%.

The perceived discomfort was rated during the recovery interventions on a scale from 0 (extremely comfortable) – 10 (extremely uncomfortable). Significant treatment effects were found for the perceived discomfort and % heart rate intensities during the recovery interventions ($P < 0.001$). The average heart rate (% of LT) was statistically significantly higher for AR compared to IB and tNB (*Figure* 15) ($P < 0.05$ and $P < 0.01$, respectively). There were no significant differences in average heart rate between the IB and tNB groups ($P > 0.05$). Perceived discomfort was statistically higher during the IB intervention compared to AR and tNB (*Figure* 16) ($P < 0.05$ and $P < 0.01$). However, there was no statistically significant difference in perceived discomfort between AR and tNB ($P > 0.05$).

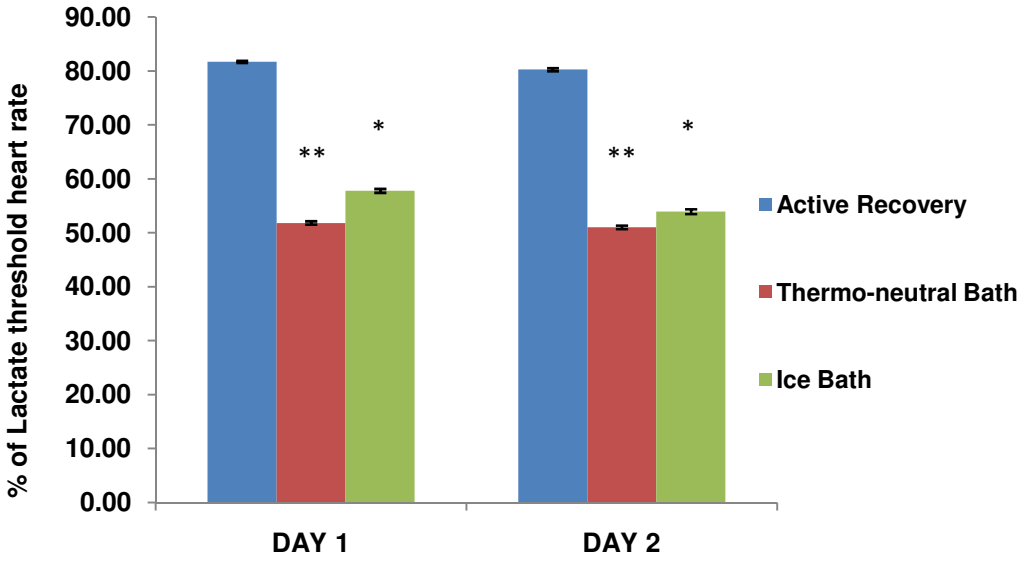


Figure 16: The average heart rate response (% of lactate threshold) during the recovery interventions. Values are expressed as means ± SEM. (* $P < 0.05$; IB vs. AR; ** $P < 0.01$; tNB vs. AR).

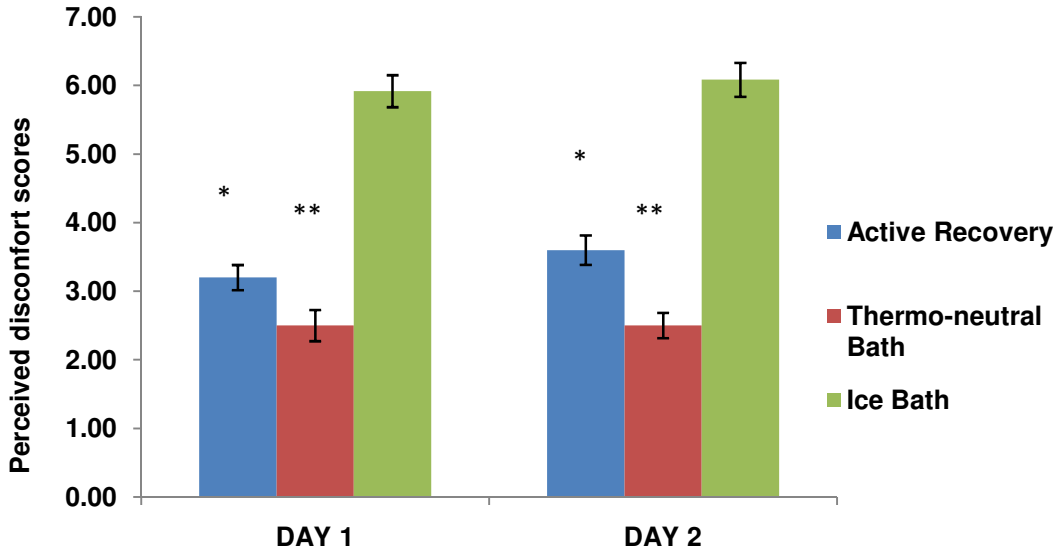


Figure 17: The perceived discomfort scores during the recovery interventions. Values are expressed as means ± SEM. (* $P < 0.05$; IB vs. AR; ** $P < 0.01$; IB vs tNB).

CHAPTER EIGHT

DISCUSSION

A. INTRODUCTION

The current investigation studied the effects of water immersion of different temperatures (cold and thermo-neutral) on the performances and post-exercise recovery during three days of intensive endurance training. To date, no evidence is available on the efficacy and practicality of water therapy on performance and recovery measures of competitive cyclists (Wilcock *et al.,* 2006[b]). Most studies thus far have focussed on the effect of water immersion on parameters of muscle damage and soreness and have therefore employed muscle damaging exercise protocols. This kind of exercise, however, does not simulate what really happens during real world exercise or competition and says little of what happens during actual training or competition. Furthermore, the few studies that have been done on cycling performance, used untrained and physically active men (Wilcock *et al*, 2006[b]; Lane & Wegner, 2004; Nakamura *et al.,* 1996) and the protocols used to induce fatigue was very short in duration. Therefore, the major limitations to their conclusions are that the results cannot necessarily be extrapolated to highly trained competitive athletes and it cannot be applied to real cycling events or prolonged training where fatigue, and not muscle damage, is the major limiting factor to performance. The effectiveness of the use of water immersion during multi-days of competition or training is still not been established and studies are limited to only team sport situations. Therefore, very little is known regarding other endurance sports such as cycling, where neuro-muscular fatigue and not necessarily EIMD is the major limitation to optimal performances. This is also the first study to compare different water temperatures to AR, rather than PAS. This is important, as PAS recovery is generally not used in cycling and it is already known that water immersion is more beneficial than performing PAS. This study also tries to determine whether the physiological effects of water immersion therapy are related to the temperature of the water, or the hydrostatic pressure exerted by the water. Therefore, the potential hydrostatic effects of water immersion were eliminated and this studied focussed mostly on the temperature effects of the water.

B. DESCRIPTIVE CHARACTERISITCS

The cyclists who volunteered for this study consisted of competitive road cyclists (N = 20). At the time of the study they were all well-trained and in the pre-competition to competition transition phase of their yearly program. Studies that have examined the effects of water immersion on post-exercise recovery between cycling bouts have used physically active men that were not competitive or specifically involved in intensive endurance cycling training (Wilcock *et al*, 2006[b]; Lane & Wegner, 2004; Nakamura *et al.,* 1996). The level of fitness directly influences an athlete's ability to recover after training sessions (Hug *et al.,* 2006; Børshein & Bahr, 2003; Jentjens & Jeukendrup, 2003; Bompa 1999). This is mostly due to the physiological adaptations (central and mostly peripheral) that are more apparent with training (Faria *et al.,* 2005; Bompa 1999). Trained athletes have better lactate clearance capacity rates than untrained individuals, and post-exercise lactate concentrations will reach resting conditions much faster (Brooks, 2001; Lucia *et al.,* 2000). Also, neuromuscular adaptations allows trained athletes to exert a greater power output due to the enhanced recruitment of motor units (Totuska *et al.,* 2002; Lucia *et al.,* 2000), and the increased strength capacity of the muscles will result in smaller elevations in plasma [CK] compared to untrained individuals (Totsuka *et al.,* 2002). More experienced athletes with higher fitness levels, specifically for their sport, will therefore recover faster, and the physiological effects of any additional recovery interventions may therefore be less pronounced compared to untrained individuals.

The physical and physiological comparison of the three recovery groups used in this study (ice bath, thermo-neutral bath, and active recovery), showed that the only significant difference was for VO_{2max} capacities between the IB and tNB groups ($P = 0.04$). This finding would not necessarily have affected the outcomes of the study, as research has confirmed that $VO2_{max}$ is not a very good indicator of cycling ability (Faria *et al.,* 2005; Bentley *et al.,* 2001; Lucia *et al.,* 1998). Instead, time trial performance is much stronger related to peak power output (PPO) achieved during a progressive incremental exercise test to exhaustion (maximal testing) (Bentley *et al.,* 2001; Bentley *et al.,* 1998). Bentley *et al.* (2001) only found a moderate correlation (r = 0.69) for VO_{2max} ($L.min^{-1}$) and average power output (W) during a short TT (20 minutes). During the longer TT (90 minutes), a very high correlation (r = 0.91) were found for PPO_{max} and average power output (W) during the TT. This is in agreement with the current study. A moderate correlation was found for PPO and 40km TT performance (r = 0.53) and a strong correlation (r = 0.84) for PPO and 100km TT performance. In contrast, the correlation between VO_{2max} and 40 km TT performance was only 0.44, and for the 100km TT performances it was 0.05. In accordance with these results, the tNB group with the highest PPO, also recorded the best 100km TT performance times (hr:min:sec), while the IB

group with the lowest PPO recorded the slowest 100 km TT performance times. These differences in PPO and 100 km TT performance times were, however, not statistically significant. Therefore it is unlikely that the difference in VO_{2max} would have a significant effect on the outcomes of the 100 km TT performances. On the other hand, the dietary intake, especially the amount of carbohydrate intake, by the cyclists may have influenced the TT performances. It has been previously shown by Wright *et al.* (1991) that carbohydrate feedings prior to exercise can improve time to exhaustion by 18% (at an intensity of 70% of VO_{2max}) in comparison to no carbohydrate feedings prior to exercise. It was clearly stated at the start of the study that carbohydrate intake during the three days was very important and a record of each cyclists' dietary intake was recorded. According to these records, all the participants' intake of carbohydrates was consistent over the three days and there were no significant differences in the diets of the three groups. Another point to consider that may have resulted in poorer performances (as in the case for the AR group) during the last day, is that the intensity of the AR may have impaired subsequent glycogen resynthesis after the recovery sessions, and this resulted in a delay in the replenishment of the muscle glycogen stores. Fairchild *et al.* (2003) showed that AR at an intensity of 40% of VO_{2max} for duration of 45 minutes did not influence glycogen resynthesis in Type II muscle fibers but resulted in glycogen mobilization in Type I muscle fibers.

C. PERFORMANCE OUTCOMES

This is the first study to investigate the effect of recovery strategies using an intensive endurance cycling training intervention. The idea was to simulate a multi-day stage cycling race, as well as challenge the cyclists with a variety of tests over three days to simulate the variable power demands of competitive road racing. The purpose of the long TT (100km) was to ensure that the cyclists exercised at high intensities for a prolonged duration (> 2hrs) so that fatigue was sufficiently induced. The total training impulse, which is an indication of the exercise load, is found to be much higher during longer TTs (> 40km) compared to shorter TTs (≤ 40km) (Padilla *et al.,* 1999). The training impulse is related to the duration of the TT and the HR achieved (maximum and average heart rate intensities).

The 100km TT protocol was the same laboratory test which was evaluated by Schabort *et al.* (1998). It included four 1km sprints at specific distance markers (10, 32, 52, and 72km) and four 4km sprints at distances 20, 40, 60 and 80km. This was done to create a more realistic competition situation. The between-test correlation was found to be 0.93 and the within-cyclist coefficient of variation was 1.7% (Schabort *et al.,* 1998).

Professional cyclists are able to maintain an average % HR intensity of 89 ± 5% of lactate threshold during longer TTs (49.2 ± 8.0 km) and an average % HR intensity of 95 ± 7%

during shorter TTs (28.0 ± 8.6 km) (Padilla *et al.,* 1999). The results of this study showed that the intensities at which the cyclists competed each day (40 km TT: 97% ± 6.43; 100 km TTs: 91% ± 6.44 and 90.1 % ± 4.28, respectively) were comparative to competition levels during cycling races. The laboratory tests were therefore realistic simulations of an actual competition and are a unique aspect of this study.

Table 4 indicates that there were no significant differences in the absolute performance times of the three recovery groups. However, from *Figure 1* (a) it is apparent that there were important differences in the relative changes between Day 2 and 3. The IB group improved their performance on the last day by 0.7%, which translates to a time of 1min 08 sec. Although this difference was not statistically significant, in practical terms, it could be a significantly better performance. During multi-stage racing, different stages are included (TTs, hill-climb TTs, short road races, long road races etc.) and the cyclist with the fastest time will take the winners jersey. The TTs are the most important stages where time advantages can be set. Considering that the other groups performed worse on Day 3 (AR: 2.8% and tNB: 1.5%), the subjects in the IB group had a significant competitive advantage on the last day and would have gained more than a minute on their fellow competitors if this was an actual race.

The effort that the IB group put in on the last day is reflected in their RPE scores, which was a lot higher (13.8%) than the previous day and also higher than the AR and tNB groups. However, this is an expected finding which is directly related to their performance levels; it is therefore unlikely that the higher RPE levels could be attributed to their specific recovery intervention.

D. POST-EXERCISE RECOVERY

1. Functional capacity

The Wingate sprint test is a valuable exercise test to assess the anaerobic power of cyclists (Faria *et al.,* 2005), however, for this study the aim of the Wingate sprint was to assess the functional capabilities of the cyclists over the three days. This test is not only specific to the study population that were used for the study but also offers an easy method of determining functional capacity. The duration of the Wingate sprints are very short (30 seconds) and requires an all-out effort. This test will therefore assess a cyclist's functional ability without having detrimental effects on subsequent tests or performances. At the same time, the Wingate sprint test is a cycle specific test and will be more functional to assess muscle power and strength than an isokinetic test on a Biodex.

When assessing recovery after exercise, one can study many parameters, as is also the case in this study. However, one of the most important indicators of recovery, whether after heavy exertion or after injury, is functional muscle strength and power (Kuligowski *et al.,* 1998). Muscular overexertion beyond its breakpoint may lead to an acute breakdown of skeletal muscle (Tiidus, 1998). Damage to the muscle fibres and structures will influence the power-producing capacity of the muscles, resulting in decrements of muscular strength (Howatson & van Someren, 2008; Cheung *et al.,* 2003) and therefore negatively influencing performance (Abbis & Laursen, 2005). The magnitude of strength loss generally depends on the muscular contractions involved (Howatson & van Someren, 2008; Cheung *et al.,* 2003). Exercise-induced muscle damage causes reductions in maximum muscle strength or power (Goodall & Howatson 2008; Vaile *et al.*2008; Vaile *et al.,* 2007; Bailey *et al.,* 2007; Skurvydas *et al.,* 2006; Eston & Peters, 1999; Kuligowski *et al.,* 1998; Viitasalo *et al.,* 1995; Donnelly *et al.,* 1990) and these reductions are usually evident at 24 – 48 hours post-exercise (Howatson & van Someren, 2008; Lavender & Nosaka, 2006; Clarkson & Hubal, 2002). Moreover, any alterations of neuromuscular functioning (related to central and mostly to peripheral factors) will affect the maximum force production capacity of the affected muscles. Neuromuscular fatigue has been shown to occur after prolonged cycling (Millet & Lepers, 2004; Lepers *et al.,* 2001; Lepers *et al.,* 2000). Therefore, alterations in maximal strength will influence power output, as power output is dependent on the force producing capacity of the muscles. In this study, changes in Wingate performance were expected mainly due to the fatiguing nature of the exercise protocol, but also due to some degree of muscle damage. Both Neubauer *et al.* (2008) and Nuviala *et al.* (1992) reported significant muscle damage during triathlon and an ultra-distance running event, indicating that it is not only strenuous eccentric contractions that may cause muscle damage.

The average power output (relative to bodyweight) during the Wingate sprint was reduced on Day 2 for all recovery groups, and the greatest decrements was observed for the tNB (-5.0%) and IB (-3.5%) group. On Day 3 AP: WT of both the tNB and IB group recovered to -2.7% and -1.9%, respectively, and was even more reduced in the AR group (-2.7%). What was noticeable is that none of the recovery groups recovered back to baseline values and their functional capacity was still reduced on Day 3. Total work (TW: WT) followed the same patterns as for AP:WT for all of the recovery groups.

These findings are in accordance with previous studies where it has been shown that prolonged cycling reduces the maximum strength producing capacity of muscles (Abbiss & Laursen, 2005; Millet & Lepers, 2004). The factors responsible for the altered neuromuscular functioning are related to changes in contractile and neural properties of the leg extensor muscle groups (Faria *et al.,* 2005; Millet & Lepers, 2005; Lepers *et al.,* 2004; Lepers *et al.,*

2000). Cycling for 2 hours at an intensity of 65 - 70% of VO_{2max} resulted in significant decrements in isometric muscle strength (13%), concentric muscle strength (12 %) and eccentric muscular contractions (12%) of the knee extensors in well-trained cyclists (Lepers *et al.,* 2000). The decrements were mostly due to lower neural input to the muscles and impaired excitation-contraction coupling.

The changes in the Wingate sprint performances over the three days were much smaller in comparison to the reductions in strength observed by other researchers (Vaile *et al.,* 2008; Montgomery *et al.,* 2008; Goodall & Howatson, 2008; Bailey *et al.,* 2007; Skurdyvas *et al.,* 2006; Lepers *et al.,* 2000; Eston & Peters, 1999; Kuligowski *et al.,*1998;). Although one cannot directly compare the outcomes of different functional capacity tests, there may still be specific reasons for the small decreases in Wingate performances in this study. It may be due to the specificity of the assessment used (Wingate sprint test), in other words, the cyclists were more familiar with the cycling movements performed during the Wingate sprint compared to isolated contractions performed on a Biodex dynamometer. The results may also be attributed to the training levels of the cyclists or perhaps lower levels of muscle damage. Whether the small changes in functional capacity could be ascribed to the recovery strategy, is unclear. However, it is clear that the water immersion therapy (both IB and tNB) did not cause any faster recovery rates compared to AR.

2. Blood markers

2.1 Blood lactate concentration

Blood lactate concentration was measured as an indication of metabolic recovery. Metabolic disturbances following high intensity exercises are usually restored back to resting conditions within ~ 30 minutes to 6 hours (Westerblad *et al.* 2006; Barnett, 2006; Cochrane, 2004; Bompa, 1999). Most of the lactate produced during exercise is directly oxidized by muscle tissue, heart and liver (Powers & Howly, 2001; Stallknecht *et al.,* 1998). In the liver, lactate is converted to glycogen via the process gluconeogensis (Powers & Howly, 2001; Stallknecht *et al.,* 1998). Lactate is also taken up by type I skeletal muscle fibres (muscle tissue) and converted to glycogen (Stallknecht *et al.,* 1998).

Peak blood [La] was observed on Day 2 for the AR and tNB groups, while there was a delayed response in the IB group, with peak levels only observed on Day 3. In addition, blood [La] was significantly lower ($P < 0.05$) in the IB group compared to the tNB group on Day 2. On Day 3, the blood [La] of the tNB group was lower than baseline values (-9.2%) and also returned to baseline in the AR group. However, there were no statistically

significant treatment effect for blood [La] and it must therefore be concluded that water immersion therapy did not result in faster lactate clearance rates than AR.

The effects of tNB on a lower [La] on Day 3, in comparison to IB recovery, may be related to temperature. Park *et al* (1998) found that 10 minutes of NWI (temperature of 30°C) resulted in significantly lower blood [La] in comparison with PAS after 10 minutes of high intensity (80% of VO_{2max}) cycling ($P < 0.05$). Park *et al* (1998) explained that the increased lactate clearance may be due to the increased stroke volume resulting from a greater cardiac preload. SV was significantly increased by 68.7% and CO by 44.7% after NWI, while total peripheral resistance was decreased by 31.6%. These changes in cardiovascular responses may assist in the increased clearance of metabolites such as lactate (Wilcock *et al.,* 2006[a]).

However, the changes in cardiovascular responses appears to be dependent on the immersion depth, moreover the magnitude of CBV expansion caused by the hydrostatic effects of water immersion (Krasney & Pendergast, 2008; Wilcock *et al.,* 2006[a]; Johansen *et al.,* 1997). The aim of this study, however, was to eliminate the hydrostatic pressure effects of water, by immersing the subjects in a supine position during the water therapy sessions. Therefore, it is unlikely that the subjects in this study would have experienced marked increases in SV and CO. However, the temperature of the tNB may have resulted in vasodilation of the capillaries which may have increased blood flow to the muscles and enhance the clearance of metabolites ([La]). The delayed response observed in the IB group may be due to the increased vasoconstriction which reduced blood flow to the muscles.

Previous research on the effect of water immersion on acute blood lactate recovery mostly reported positive results, however, in these studies changes in blood [La] was compared to passive recovery (PAS). Hamlin (2007) and Morton (2007) reported that CWT resulted in decreased blood [La] post-exercise in comparison to PAS, which they attributed to the vaso-pumping action caused by the fluctuations in temperatures. Coffey *et al.* (2004) studied the effects of CWT in comparison to AR, and concluded that although CWT reduced [La] following high intensity treadmill runs, the reductions in blood [La] was not significantly different from the changes after 15 minutes of AR, and CWT was not any better than active recovery in blood [La] clearance. There is no evidence that supports the assumption of vaso-pumping (Hing *et al.,* 2008; Higgins & Kaminski, 1998) caused by CWT, and the observed effects of CWT on blood [La] may be more related to the hydrostatic effects.

The enhanced blood [La] clearance during AR is mainly due to the increased blood flow to the working muscles as a result of the active skeletal and respiratory muscle pumps (Wilcock *et al.,* 2006[a]; Cochrane, 2003). Although not statistically significant, it seems that the intensity of AR for a duration of 20 minutes effectively reduced blood [La] back to baseline, however,

the lack of an overall treatment effect means that AR was not significantly better than water therapy.

2.2 Plasma CK

Plasma [CK] changed significantly over time for all recovery groups ($P < 0.05$), indicating that muscle damage was induced by the intensive endurance training sessions. The least changes were observed in the AR group, where there was only a 0.4% increase on Day 2 and a -5.1% decrease on Day 3. Previously it has been suggested that AR may improve lymphatic transport and subsequent clearance of CK (Sayers & Clarkson, 2003; Cochrane, 2003). This study therefore shows that water immersion therapy is not as effective as AR to clear CK from the circulation. However, the AR group did not manage to perform better in the Wingate test and the 100km TT on Day 3 as a result of this lower [CK]. In fact, their performances were the worst of the three groups on Day 3.

Plasma [CK] was elevated for both the tNB and IB groups on Day 2, but the IB group only reached their maximal values on Day 3. This increase of 34% on the last day was, however, not significantly different from the relative changes of the other two groups on Day 3. The magnitude of this change in plasma [CK] was similar to the increase of ~26.6% that was observed by Köning *et al.* (2003) in highly trained professional road cyclists after the finish of the fourth stage of a 5 day race (distance: 156 km, mean duration: 5 h 13 min). The elevated plasma [CK] in the IB group on Day 3 suggests that the cold water caused a somewhat delayed response compared to the tNB and AR groups. This is in agreement with the findings of Howatson *et al.* (2005) who observed that [CK] was still elevated at 48 hours post-exercise in the elbow-flexors of untrained men after 15 minutes of ice massage directly after exercise, with repeated treatments after 24 and 48 hours. Similar results were reported by Ingram *et al* (2009) who also found significantly elevated [CK] at 48 hours following intermittent high intensity running and CWI (10^{o}C) of 15 minutes. They also found that the high [CK] did not affect sprint performances negatively. In contrast, Eston and Peters (1999) and Vaile *et al.*(2008) found that 15 minutes of CWI (15^{o}C) significantly reduced [CK] at 24 and 48 hours post-exercise. It is postulated that the cooling effects of CWI leads to vasoconstriction which reduces the permeability of capillary, lymphatic and cellular vessels to proteins, attenuating the efflux of CK (Wilcock *et al.,* 2006[a]). The reduced inflammatory response may in turn reduce subsequent muscle pain, loss of power generating capacity and swelling (Wilcock *et al.,* 2006[a]). However, there is uncertainty whether cold water immersion leads to significant reductions in intramuscular temperature, which would then cause vasoconstriction and a resultant lower [CK] in the blood.

2.3 CRP concentration

CRP is mostly involved in the acute phase of the inflammatory response. The acute inflammatory response is also associated with oedema and inflammatory cell infiltration (Cheung *et al.,* 2003). High [CRP] is found when the immune system is stressed, thereby enhancing the release of cytokines which stimulates the release of acute phase proteins such as CRP, usually apparent after prolonged intensive training or strenuous racing (Neubauer *et al.,* 2008; Scharhag *et al.,* 2005; Fallon, 2001; Jeukendrup *et al.,* 2000).

In this study, [CRP] was significantly elevated over time for all groups, with the largest increases in the AR group (+470%; $P < 0.001$) and the smallest increases in the tNB group (+131.9%). These findings are in agreement with those of Ingram *et al.* (2009) and Scharhag *et al.* (2005) who both found significant elevations in [CRP] at 24 and 48 hours following high intensity intermittent running and 4 hours of cycling at moderate to high intensity, respectively.

The magnitude of the CRP response was much lower in the water therapy groups compared to AR ($P = 0.05$), although the differences were not statistically significant. The higher [CRP] response elicited in the AR group may be related to the additional muscular contractions exerted during the recovery strategy after the specific TTs on Day 1 and 2. The additional duration of exercise, as well as the intensity during AR may have increased the production and release of Interleuken-6 from the contracting muscles (Neubauer *et al.,* 2008; Pedersen *et al.,* 2007; Ronsen *et al.,* 2002), thereby enhancing the release of CRP from the liver (Febbraio & Pedersen, 2002; King *et al.,* 2003; Suzuki *et al.,* 1999). It is suggested that the elevated Interleuken-6 levels usually observed following prolonged exercise is a reflection of the combination of both low glycogen levels and inflammation (Neubauer *et al.,* 2008; Pedersen *et al.,* 2007). Then again, a study done by Wigernaes *et al.* (2000) found that 15 minutes of AR at an intensity of 50% VO_{2max} strongly counteracts the post-exercise drop in white blood cell count (WBCC), 5 minutes following intensive endurance exercise, and does not appear to affect the immune system. However, the researchers did not show whether this was a longer lasting response which actually influenced the immune response after exercise.

Very poor correlations were found between [CRP] and girth changes over time ($r = 0.26$). This finding suggests that [CRP] as an inflammatory marker is not specific enough to measure the amount of inflammation or oedema in muscles specifically. Therefore, the rise in [CRP] could rather be due to lower glycogen levels experienced by the cyclists in the AR group.

3. Lower limb swelling

No significant swelling was observed in any of the recovery groups ($P < 0.05$). Swelling measured in the thigh peaked on Day 2 for the IB group and on Day 3 for the tNB and AR groups. These patterns of swelling are consistent with previous findings (Vaile *et al.,* 2008; Vaile *et al.,* 2007; Eston & Peters, 1999). While swelling in the calve of the AR and IB groups peaked on Day 2 and then recovered below baseline on Day 3, there were no swelling observed in the tNB group on any of the days.

Exercise causes an increase in the amount of intramuscular water (Yanagisawa *et al.,* 2003; Nosaka & Clarkson, 1996) and this volume of fluid movement is directly related to the mode, duration and intensity of exercise (Gillen *et al.,* 1991; Hildebrandt *et al.,* 1992; Green *et al.,* 1984; Miles et a*.,* 1983). Most of the fluid shifts are directed to the intramuscular compartment involving the active muscle (Hildebrandt *et al.,* 1992; Gillen *et al.,* 1991; Knowlton *et al.,* 1987; Green *et al.,* 1984). The reduced calve circumferences over time could be due to the fact that most of the fluids shifted to the muscles of the thigh. It is therefore postulated that the cyclists may have predominantly used the thigh muscles during the TT performances, explaining the larger % changes observed in the thigh compared to the changes in the calve.

Disruptions of the filtration-reabsorption transport process due to the increased leakage of muscle proteins and metabolic by-products such as lactate, results in an abnormal increase in interstitial fluid in the localized areas (Waterhouse *et al.,* 2006; Wilcock *et al.,* 2006[a]). The higher elevations in blood [La] and plasma [CK] in the IB group may explain the slightly greater degree of swelling in this group. However, the amount of swelling was very marginal and may be related to the lower magnitude of muscle damage that occurred in this exercise protocol.

4. Profile of moods and perception of fatigue

Although there were no statistically significant differences in total mood scores of the three groups, there were some distinct patters over the three days. Overall, the AR group showed the worst total mood disorder scores on Day 1 and Day 3, while the IB group showed the best total mood disorder scores on all three of the days. The latter indicates that of the three groups, the IB group felt more ready and confident for each of the three days of intensive endurance training. One can therefore speculate whether the cold water treatment actually contributed to these feelings of readiness and alertness and possibly also contributed to the improved performances on the last day. The mood scores were consistent with the relative changes in performances over the last two days in the different groups. Therefore, not only

did the IB group exhibit a more positive mood, but they also performed better in the 100 km TT on the last day. In contrast, the AR group reported more negative feelings over the three days and consequently they also performed the worst on Day 3.

It is possible that the AR group actually felt more tired because they also had to perform additional exercise as part of their recovery after the long training sessions. However, the results for the fatigue scores are not consistent with this conclusion, as the AR group actually reported significantly lower fatigue scores over the three days ($P < 0.05$) compared to the water immersion groups. Furthermore, it was interesting to note that although the IB group reported the highest perceived discomfort scores (indicating that the ice baths were uncomfortable), it would seem that this intervention actually contributed to their more positive mood state over the three days compared to the negative mood states of the AR and tNB groups. Considering the fact that the IB group also had superior performances on Day 3, it would seem that the cold baths could have given them a psychological advantage, or that they at least felt more motivated and invigorated and that these subjective feelings contributed to their better performances.

5. Perceived muscle soreness and discomfort

The VAS scale is a valuable tool used mostly during studies where DOMS is deliberately induced (Vaile *et al.,* 2008; Goodall & Howatson, 2008; Vaile *et al.,* 2007). During the 3 days of intensive endurance training the cyclists reported no soreness or discomfort after Day 1, while most cyclists in the tNB group reported soreness after Day 2 and 3. Cyclists in the AR group also reported soreness on Day 3, specifically with applied pressure to the muscle.

The low VAS scores could most likely be due to the low levels of muscle damage that was caused by the cycling exercise. Subjects reported that they did not feel any serious pain; however, they did experience a feeling of numbness and heaviness especially during activities.

E. RESPONSES DURING RECOVERY

The average HR intensity during the water recovery session on Day and 2 was 51.4 ± 3.4% during tNB, and 55.8 ± 6.12% during the IB ($P > 0.05$). The higher average heart rate during IB is related to exposure to the cooler temperatures (11 ± 0.97^{o}C) in comparison to the higher temperatures (30 ± 0.62^{o}C) of the thermo-neutral bath. The increased vasoconstriction caused by the cold temperature results in increased peripheral and arterial resistance leading to increased heart rate and cardiac output (Sramek *et al.,* 2000; Park *et al.,* 1999; Bond-Peterson & Schultz-Pederson, 1992). The average HR intensity for AR for

both days was 81 ± 1.7%, which was significantly higher than the IB ($P < 0.05$) and tNB ($P < 0.01$) groups.

In this study, the control group followed an active recovery protocol. It has been shown that AR involving exercise activities of light intensities (40-50% of VO_{2max}) results in improved blood [La] clearance compared to PAS recovery (Wilcock *et al.,* 2006[a]; Barnett, 2006; Cochrane, 2004; Monedero & Donne, 2000). The increase in cardiovascular responses such as SV and CO, as well as the enhanced blood flow to the working muscles, enables the body to enhance metabolite clearance and oxidation (Wilcock *et al.,* 2006[a]). In this study intensity equal to 81 ± 1.74% (80 – 84%) of the cyclists individual lactate threshold (~ 55 – 65% of VO_{2max}) was used. This higher intensity was selected on the basis that the subjects in this study were well trained.

In terms of perceived comfort during the recovery interventions, cyclists considered AR and tNB as the most comfortable recovery sessions, while RPE ratings during the IB recovery was significantly higher for both days, indicating that the cold water baths were quite uncomfortable for the cyclists.

F. MAIN FINDINGS

This is the first study, to our knowledge, to investigate the effects of water immersion on the post-exercise recovery and performance of competitive cyclists. More importantly, the hydrostatic pressure effects of water immersion were excluded to determine the extent of temperature effects (cold vs. neutral) on recovery performance. Over the course of the three days, cyclists exercised at high intensities, similar to that of professional road cyclists during an actual competition (Padilla *et al.,* 1999). No significant differences were found for any of the recovery conditions for any of the dependent variables over the course of the three days.

The effects of water therapy on performance are inconclusive and most studies failed to observe any significant differences in improvements. Coffey *et al.* (2004) found that there was no influence of CWT on subsequent running performances separated by 4 hours and that high intensity running performance returned to baseline regardless of the recovery that was used. Morton (2007) and Hamlin (2007) also failed to find any effects on sprint cycling performance and repetitive sprinting performances, respectively. In contrast to these findings, Montgomery *et al.* (2008) found that CWI was substantially better than carbohydrate intake and stretching in maintaining line-drill performance during a 3 day basketball tournament. It is possible that the cold temperature effects may have limited the amount of damage at the initial stages thereby reducing the amount of secondary muscle damage, leading to improved performance.

No significant performance changes in times, and average power was observed for any of the water therapy groups (IB and tNB) over the last 2 days ($P > 0.05$). Neither IB, nor tNB had any effects on the performances of the cyclists during the three days of intensive training. In addition, water therapy was also not any better than AR on the performances during the last day. Although not significant, the greatest % performance decrement during Day 3 for the 100km TT was observed in the AR group and the best % performances was observed for the IB group. Muscle damage is associated with subsequent swelling and an increase leakage of proteins ([CK]) (Howatson & Van Someren, 2008; Cheung *et al.,* 2003; Smith, 1991). The possibility of CWI to reduce the amount of secondary damage would mean that swelling and plasma [CK] levels would be reduced. However, the temperature during the IB recovery appeared to not reduce any of these markers, as the highest elevations was observed for the IB group in swelling on Day 2 (+0.22%) and plasma [CK] on Day 3 (+34%), even though the IB showed the best performance improvements for the last 100km TT. We found that plasma [CK] and performance times was poorly correlated (r = 0.03), and it appears that performances are not related to the levels of [CK]. On the other hand, functional performances during the Wingate sprint was affected in all of the recovery groups.

Active recovery is well known to reduce blood [La] following exercise (Wilcock *et al.,* 2006[a]; Cochrane, 2004; Thiriet *et al.,* 1993). The intensity and the duration of the recovery used in this investigation, was optimal enough to result in reductions of blood [La] and plasma [CK] (strong tendency compared to WG; $P = 0.05$). However, the [CRP] response was affected in a way that the intensity of the AR intervention resulted in a higher acute phase [CRP] response in comparison to the WGs ($P > 0.05$). Moreover, WG recovery elicited a smaller [CRP] response compared to AR ($P > 0.05$), even though IB recovery was perceived as the most uncomfortable of all three recovery conditions.

NWI has been previously shown to result in reduce blood [La] following intensive short duration cycling exercises (Nakamura *et al.,* 1996). It is unclear, whether the enhanced blood [La] clearance is related to the temperature or hydrostatic pressure. The tNB group showed non-significant reductions below baseline values in blood [La] on Day 3 (-9.2%) compared to the IB and AR group. The subjects were immersed into a supine position with the aim in eliminating the possible hydrostatic effects of water immersion. Therefore, it could be concluded that the reduced blood [La] could be related to the vasodilation, increasing the blood flow to the muscles thereby enhancing metabolite clearance. However, it could be expected that any additional hydrostatic pressure effects together with the temperature effects may have resulted in an enhanced blood [La] clearance effect. The vasoconstriction caused by the cold temperatures of the IB may be responsible for the higher blood [La] levels observed on Day 3 (+20.6%), and may have been different in the presence of hydrostatic

pressure. It can be concluded that the tNB may be better than IB in the clearance of physiological markers of recovery, however, the possible effects may even be more pronounced with increasing level of immersion (hydrostatic pressure). However, there is no difference between these two temperatures on aerobic and functional performances, and water therapy *per se* was not any better than AR.

Interestingly, is that although the IB recovery was experienced as the most uncomfortable, the overall perceived moods of the IB group were much more positive for all 3 days in comparison to the tNB and AR groups. These findings suggests that the IB effects were more subjective, rather than physiological, which possibly resulted in better performances on the last day.

G. STUDY LIMITATIONS AND FUTURE STUDIES

The main limitation of this study was the low statistical power, due to the small sample size. The decision to include well-trained cyclists in this study not only meant that the initial pool of potential subjects were smaller, but high level athletes are always more apprehensive to participate in research studies in fear of the possible interference it would have on their training and the increased risk for injury. Furthermore, as the cyclists were all committed to a strenuous training program, many withdraw due to illness or injury or the lack of time to complete all the training sessions which formed part of this study.

In this study, subjects were randomly assigned to one of three intervention groups. Although the groups were comparable with regards to baseline physical and physiological characteristics, it is known that the inter-individual variations (Brancaccio *et al.,* 2007) in the blood markers of muscle damage and inflammation, blood lactate and even subjective feelings of fatigue and soreness are large. Therefore, together with the small sample size, these large inter-individual differences would make it very difficult to obtain statistically significant results. A solution to this problem is to follow a randomised cross-over design, where each subject serves as their own control. However, this type of design would prolong the study period significantly and increase the risk of drop-out bias and research mortality.

Physiological (blood [La], [CK], [CRP], and swelling) and functional assessments (Wingate sprint) were only measured up to 48 hours from baseline (Day 3). It is possible that measurements thereafter might have shown an additional response that may be important for the interpretation of the effects of water immersion (cold and neutral) and AR on performance recovery. In practice, on the other hand, cyclists participating in multi-day events need to recover within 24 hours and if one therefore do not see significant

improvements in recovery within this time period with any specific recovery strategy, it is questionable whether it is worth the effort.

The intensity of the AR period may have been too high (~55 – 60% of VO_{2max}), which resulted in an increased acute phase immune response ([CRP] and decreased aerobic performances. During the TTs on all three days, cyclists were not allowed to consume any carbohydrate beverages and were only allowed to consume water. The intensity and the duration of the TTs possibly resulted in muscle glycogen depletion and may have influenced subsequent recovery effects of the interventions. It is therefore advised to allow the cyclists to consume carbohydrate beverages during the TTs; however the concentration mixtures should then be standardized.

Another shortcoming was the possibility of a placebo effect that may have occurred in the IB group. Most athletes perform IB recovery on a daily basis and truely believe that it really works for them. The fact that the perceived mood scores were the best during all three days in the IB group and the improvement in performances during the last 100km TT showed that the cyclists in the IB group were more confident.

The popularity of CWI amongst athlete are increasing, however the long term effects on physiological adaptations during training requires further investigation. Future studies should also determine whether water immersion is however beneficial in the recovery process as well as the temperature range whereby the greatest effects may be induced. Also, the hydrostatic pressure should also be studied in isolation to determine to what extent the hydrostatic pressure of water immersion may play in the performance recovery. Field studies during competition would be the ideal study design whereby the findings can be directly applied to the real world setting during competition cycling.

H. CONCLUSION

Very few studies have investigated the effects of water therapy on post-exercise recovery and performance during multi-days of competition and training especially where accumulated fatigue is apparent (Rowsell *et al.,* 2009; Mongomery *et al.,* 2008). To date most studies have focussed on the effects of water immersion on post-exercise recovery from a single bout of exercises that mainly involved eccentric muscular actions (Ingram *et al.,* 2009; French *et al.,* 2008; Goodall & Howatson 2008; Vaile *et al.,* 2008; Vaile *et al.,* 2007; Bailey *et al.,* 2007; Sellwood *et al.,* 2007; Skurvydas *et al.,* 2006; Eston & Peters, 1999; Kuligowski *et al.,* 1998; Isabell *et al.,* 1992). Other studies examined the effects of water therapy on blood [La] clearance and relating lactate to subsequent performance (Morton, 2007; Hamlin, 2007; Coffey *et al.,* 2004). Limitations to these studies are that none of them have considered the

possible temperature and hydrostatic effects of water immersion separately from each other. The current investigation approached this problem by excluding the possible hydrostatic pressure effects.

The results of the study showed that the cyclists did experience muscle damage and fatigue during the 3 days of intensive endurance training. However, we have failed to show any statically significant differences on markers of post-exercise recovery and performance during 3 days of intensive endurance cycling, and there were no differences between the IB temperature and the tNB temperature. Future research is needed to determine the exact role of hydrostatic pressure in aiding post-exercise recovery during water immersion, as well as the optimal temperature range of the water.

In conclusion, neither cold water, nor neutral water therapy, had more beneficial effects on post-exercise recovery rates compared to active recovery. Importantly, however, is that the cyclists' were able to maintain their performances over the three consecutive days, indicating that water therapy per se is not detrimental to endurance performance. In addition, cold water immersion may be safely used by competitive athletes during multi-days of competition. However, the use of CWI by athletes as part of their daily routines during training, especially where the aim is to adapt from training stress, should be thoroughly investigated.

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APPENDIX A

INFORMED CONSENT:

Title of research project:

"The effects of cold and thermo- neutral water immersion on the recovery of competitive cyclists after high intensity training."

Reference number: _____

Consent of Subject:

I, the undersigned

_____[ID:_____],

from (address)

_____

_____

confirm that:

1. I was invited to participate in the above-mentioned project conducted by the Department of Sport Science at the University of Stellenbosch.

2. It was explained to me that:

2.1 the aim of this project is to determine if there is a difference in the effects of water immersion of different temperatures (cold versus neutral) on the recovery after exercise.

2.2 I will participate in the following tests:

2.2.1 One VO2 max test (blood lactate included).

2.2.2 One 40 km time trial and three 100km time trials.

2.3 Prior to and after exercise tests the following would be done:

2.3.1 Blood samples (10-20ml) will be taken from a vein in my arm.

2.3.2 I will complete the questionnaires of the degree of muscle soreness and how I feel in general.

2.3.3 I will undergo one of the following recovery interventions, e.g. an ice-bath, a neutral temperature bath, or active recovery on a bike.

3. If I am selected for the study, I agree that I will complete all the tests and measurements for all 4 days.

4. I was informed that invasive procedures (e.g. the draw of blood) will be administered by a medical doctor (Drs. Williams & Scwabbe, contact number: 021 808 3392) or a qualified phlebotomist (Pathcare).

5. I am warned that I might develop one or more symptoms during the exercise tests. This includes pain, nausea, dizziness, high or low blood pressure, heart beat disorders (too slow, too rapid, or irregular), muscular fatigue and soreness. I understand that I can stop the exercise tests at any time when i experience one of these symptoms. I was also informed that a medical doctor (Dr. PL. Viviers, Dr. A. Williams) will be present at all times at the Sports Medicine Practice to take care of medical emergencies.

6. I was informed that the information obtained during this study will be held confidential, but the findings may be published in a scientific journal.

7. I understand that the research/test observers and/or Stellenbosch University cannot be kept responsible for any injuries that might occur during any of the tests included in this project.

8. The above mentioned information was explained to me by Christa Koekemoer in English/ Afrikaans. I was also given the opportunity to ask questions and all my

questions were answered satisfactory. If I have any questions, I can contact: Miss. C. Koekemoer (Tel. 021 808 2818 or 083 566 5577; e-mail: 14051656@sun.ac.za) or Prof. E. Terblanche (tel. 021 808 2742 or 082 707 6501; e-mail: et2@sun.ac.za).

9. It was explained to me that my participation is voluntarily and that I can withdraw from the project at any time. I also understand that the researcher or medical doctor may withdraw m e from the study if deemed necessary for medical purposes.

10. I was informed that I can contact Me. Hunter-Husselman (Tel. 021 808 4623; e-mail: mh3@sun.ac.za) if I have any questions regarding my rights as a subject in a research project.

11. I was informed that there are no costs linked to my participation.

With this I volunteer to participate in the above-mentioned project. **I take responsibility** to endeavour to complete all tests (4 visits to the laboratory).

Signed at _____on _____20_____

_____ _____

Subject Witness

Statement of the Researcher

I, _____ declare that I:

1. Explained the information contained in this document to _____

2. Requested the subject to ask questions if anything was unclear.

3. Performed this conversation in English/Afrikaans.

Signed at _____ on _____ 20 _____

_____ _____

Researcher Witness

APPENDIX B

INGELIGTE TOESTEMMING:

Titel van navorsingsprojek:

"Die effek van koue en termo-neutrale water terapie op die herstel van kompeterende fietsryers na hoë intensiteit oefening."

Verwysingsnommer:_____

Verklaring deur toetspersoon:

Ek, die ondergetekende ,

_____[ID:_____],van (adres)

_____

_____

bevestig dat:

1. Ek uitgenooi is om deel te neem aan bogenoemde navorsingsprojek wat deur die Departement Sportwetenskap van die Universiteit van StelletNBosch onderneem word.

2. Daar aan my verduidelik is dat:

2.1 die doel van die projek is om te toets of daar 'n verskil is in die effekte van water terapievan verskillende temperature (koue en neutrale) op die herstel na oefening.

2.2 ek die volgende toetse sal aflê:

2.2.1 Een VO2 maks toets (bloedlaktaat ingesluit).

2.2.2 Een 40km tydtoets en drie 100km tydtoetse.

2.3 Voor en na die oefentoetse sal die volgende gedoen word:

2.3.1. Bloedmonsters (10-20 ml) sal geneem word van ‘n vene in my arm.

2.3.2 Ek sal vraellyste voltooi oor die mate van spierseerheid wat ek ervaar en hoe ek oor die algemeen voel.

2.3.3 Ek sal een van die volgende herstel intervensies ondergaan, nl. ’n Ysbad, ’n neutrale temperatuur bad, of aktiewe herstel op ’n fiets.

3. indien ek vir die studie gekies word, onderneem ek om al 4 dae se toetsing en metings te voltooi.

4. Ek ingelig is dat indringende prosedures (bv. bloedtrek) uitgevoer sal word deur ‘n mediese dokter (Drs. Williams & Dr. Scwabbe; kontaknommer: 021 808 3392) of ‘n gekwalifiseerde persoon (Pathcare).

5. Ek gewaarsku is dat daar ‘n moontlikheid bestaan dat ek een of meer simptome tydens die oefentoetse mag ondervind. Dit sluit in pyn, duiseligheid, naarheid, abnormale hoë of lae bloeddruk, abnormale hartklop (te stadig, te vinning of ongereeld), spiermoegheid en seerheid. Ek verstaan dat ek enige tyd die oefentoetse mag staak wanneer ek enige van hierdie simptome ondervind. Ek is ook ingelig dat ’n mediese dokter (Dr. PL. Viviers of Dr. A. Williams) aanwesig sal wees te alle tye by die Sport Medisyne Praktyk om na mediese noodgevalle om te sien.

6. Ek meegedeel is dat die inligting wat ingewin word as vertroulik behandel sal word, maar dat die bevindinge wel in wetenskaplike vaktydskrifte gepubliseer kan word.

7. Daar is aan my verduidelik dat nie die narvorsers/toetsafnemers en/of StelletNBosch Universiteit verantwoordelik gehou kan word vir enige besering wat ek moontlik kan opdoen gedurende enige van die toetse ingesluit in die projek nie.

8. Die inligting wat hierbo weergegee is, deur Mej. C Koekemoer aan my in Afrikaans/Engels verduidelik is. Ek ook ‘n geleentheid gegee is om vrae te vra en dat al my vrae bevredigend beantwoord is. Indien ek enige verdere navrae het, kan ek die volgende persone kontak: Mej. C. Koekemoer (Tel. 021 808 2818 of 083 566 5577; e-pos: 14051656@sun.ac.za) of Prof. E. Terbalnche (Tel. 021 808 2742 of 082 707 6501; e-pos: et2@sun.ac.za).

9. Daar is aan my verduidelik dat my deelname vrywillig is en dat ek enige tyd aan die projek mag onttrek. Ek verstaan ook dat die navorser of ’n mediese dokter my van die projek mag onttrek indien dit in my belang geag word a.g.v. medies-verwante redes.

10. Ek meegedeel is dat ek Me. Hunter-Husselman (Tel. 021 808 4623; e-pos: mh3@sun.ac.za) kan kontak indien ek enige vrae het oor my regte as ’n proefpersoon in ’n navorsingstudie.

11. Ek meegedeel is dat daar geen kostes aan my deelname verbonde is nie.

Ek stem hiermee vrywillig in om aan bogemelde projek deel te neem. **Ek neem die verantwoordelikheid** om ’n uiterste poging aan te wend om al die toetse te voltooi (4 besoeke aan die laboratorium).

Geteken te _____ op _____20_____

_____ _____

Toetspersoon Getuie

VERKLARING DEUR NAVORSER

Ek, _____, verklaar dat ek:

1. die inligting vervat in hierdie dokument aan _____

   verduidelik het;

ii. Sy versoek het om vrae aan my te stel indien daar enigiets onduidelik was;

iii. Dat hierdie gesprek in Afrikaans/Engels plaasgevind het.

Geteken te _____ op _____ 20_____

_____ _____

Navorser Getuie

APPENDIX B

PERSONAL INFORMATION/ PERSOONLIKE INLIGTING

Name/ Naam:_____ **Surname/ Van:**_____

Date of birth/ Geboortedatum: _____ **Age/ Ouderdom:** _____

ID:_____

Address/ Adres: _____

_____

_____

_____

Phone/ Telefoon (H): _____ **(W):**_____

(C): _____

E-mail address:_____

Occupation/ Beroep: _____

1. **Do you smoke/ Rook u?** **Yes/ Ja** **No/ Nee**

   If Yes/ Indien Ja;

   How many times a day/ Hoeveel keer 'n dag?_____

How long have you been smoking/ Hoe lank rook u al?_____

2. **Do you have any cardiovascular complications or disease/ Het u enige kardiovaskulere komplikasies of siektes? Yes/ Ja No/ Nee**

   If Yes, please provide detail/ Indien Ja, voorsien asseblief besonderhede:

   _____

   _____

3. **Do you have any immune deficiencies or disease/ Het u enige immuunsiektes of tekorte? Yes/ Ja No/ Nee**

   If Yes, please provide detail/ Indien Ja, voorsien asseblief besonderhede:

   _____

   _____

4. **Do you have any haematological problems/ Het u enige haematologiese problem? Yes/ Ja No/ Nee**

   If Yes, please provide detail/ Indien Ja, voorsien asseblief besonderhede:

   _____

   _____

5. **Do you use any type of medication/ Gebruik u enige tipe medikasie?**

   Yes/ Ja No/ Nee

   If yes, please state which type and for what reason/ Indien ja noteer die tipe en rede vir gebruik.

   _____

   _____

Training background and information/ Inoefenings agtergrond en inligting:

1. **How many times per week do you train/ Hoeveel maal per week oefen u?**

   _____

2. **What is your main discipline, road cycling or mountain biking or both?/ Wat is u se hoof dissipliene, padfiets of bergfiets of beide?**

   _____

3. **What is your level of participance/ Wat is u se vlak van deelname?**

   Recreational/ Rekrease_____

   Club/ Klub_____

   Provincial/ Provinsiaal_____

   National/ Nasionaal_____

4. **How many kilometers (or hours) do you cycle per week/ Hoeveel kilometer (of ure) ry u fiets per week?**

   _____

5. **How often do you participate in cycling races per year/ Aan hoeveel fietswedrenne neem u deel per jaar?_____**

6. **What is your personal best times for the following races and when did you achieve this time (year)/ Wat is u se persoonlike beste tye vir die volgende wedrenne en wanneer het u die tyd behaal (jaar):?**

   Cape Argus:_____

   Cape- Epic:_____

   Other/
   Ander:_____

   _____

7. **How long have you been competing as a competitive cyclist/ Hoe lank neem u kompeterend deel aan fietsry?_____**

8. **Do you participate in any other sports disciplines? (If so, please state how many times a week)/ Neem u deel aan enige ander sport disciplines? (As wel, die aantal kere beoefen per week).**

   _____

   _____