

**The effect of water immersion, active
recovery and passive recovery on repeated
bouts of explosive exercise and blood
plasma fraction**

Ian Wilcock

A thesis submitted to the
Auckland University of Technology
in partial fulfilment of the degree of
Masters of Health Science
November 2005

CONTENTS

TITLE PAGE	I
CONTENTS.....	II
LIST OF TABLES.....	V
LIST OF FIGURES.....	VI
LIST OF APPENDICES.....	VII
LIST OF PUBLICATIONS / PRESENTATIONS FROM THIS THESIS	VIII
ATTESTATION OF AUTHORSHIP.....	IX
ACKNOWLEDGEMENTS.....	X
DEDICATION.....	XII
INTELLECTUAL PROPERTY RIGHTS	XIII
ABBREVIATIONS.....	XIV
ABSTRACT	XVI
CHAPTER 1: INTRODUCTION	1
<i>Purpose statement</i>	4
<i>Aims and hypothesis</i>	4
<i>Significance of thesis</i>	4
<i>Limitations</i>	5
<i>Note to reader</i>	5
CHAPTER 2: PHYSIOLOGICAL RESPONSE TO WATER IMMERSION: A METHOD FOR SPORT RECOVERY?	6
<i>Introduction</i>	6
<i>Water immersion as a recovery strategy</i>	6
Cryotherapy	7
Thermotherapy	7

Contrast therapy	7
Water immersion per se	8
<i>Hydrostatic pressure</i>	9
Water pressure	9
Fluid shifts	11
Exercise-induced muscle edema	13
Cardiac response	14
Peripheral resistance and blood flow	17
Weightlessness and perceived fatigue	19
<i>Temperature</i>	21
Cold temperature effects	21
Hot water temperatures	24
Contrasting temperature	26
<i>Summary</i>	29
 CHAPTER 3: THE EFFECT OF PASSIVE RECOVERY, ACTIVE RECOVERY AND WATER IMMERSION ON PERFORMANCE FOR TOURNAMENT-STYLE EVENTS	 31
<i>Introduction</i>	31
<i>Definition of recovery techniques</i>	32
Passive recovery	32
Active recovery	32
Water immersion	33
<i>Recovery and performance</i>	34
Strength	35
Active recovery	35
Water immersion	39
Cycling	43
Active recovery	43
Water immersion	47
Running	49
Jumping	50
<i>Summary</i>	53
 CHAPTER 4: WATER IMMERSION, ACTIVE RECOVERY AND PASSIVE RECOVERY EFFECTS ON REPEATED BOUTS OF EXPLOSIVE EXERCISE AND BLOOD PLASMA FRACTION	 54
<i>Prelude</i>	54
<i>Introduction</i>	55

<i>Methods</i>	57
Subjects.....	57
Study design	58
Repetitive jumping protocol.....	59
Recovery protocols	60
<i>Measurements</i>	61
Leg Fatigue.....	61
Blood sampling	62
Jump kinetics.....	63
<i>Statistics</i>	63
<i>Results</i>	65
Reliability of jump protocol	65
Baseline measures.....	65
Total peak power.....	65
Total work	68
Blood plasma.....	70
Perceived leg fatigue	71
<i>Discussion</i>	72
CHAPTER 5: SUMMARY	76
<i>Practical Applications</i>	77
<i>Future research</i>	77
REFERENCES	78
APPENDICES	90

LIST OF TABLES

Table 1: Examples of contrast therapy protocols.....	8
Table 2: Cardiac response of thermo-neutral immersion compared to non-immersion.	16
Table 3: Water immersion and perception of fatigue.....	20
Table 4: Effect of active and passive recovery on maximal strength	36
Table 5: Effect of water immersion and passive recovery effect on maximal strength	39
Table 6: Effect of active recovery and water immersion on repeated bouts of cycling.....	44
Table 7: Effect of active recovery and water immersion on repeated bouts of running.....	49
Table 8: Effect of water immersion on jump performance	51
Table 9: Subject descriptive characteristics.....	58
Table 10: Scale used for perception of leg pain and leg fatigue.....	62
Table 11: Mean percent (90%CL) change of total peak power from jump session 1 for the different recovery modes.	67
Table 12: Mean percent (90%CL) change total work when compared to jump session 1 for the different recovery modes.	69
Table 13: Descriptive statistics of raw and calculated displacement data.	101
Table 14: Peak power and work variables calculated manually and determined by the linear displacement analysis program.....	102

LIST OF FIGURES

Figure 1: Schematic diagram of intracellular-intravascular fluid movement.....	11
Figure 2: Schematic diagram of pressures acting on an immersed body.....	19
Figure 3: Change in blood flow between contrast and cold water immersion.	27
Figure 4: Isoinertial supine squat machine.....	59
Figure 5: Total peak power (mean \pm SD) produced during each Jump Session for each recovery mode	66
Figure 6: Total work (mean \pm SD) produced during each Jump Session for each recovery mode.....	68
Figure 7: Change in blood plasma fraction (mean \pm SD) throughout the day with different recovery modes	70
Figure 8: Change in perceived leg fatigue throughout the day with different recovery modes	71

LIST OF APPENDICES

Appendix A: Subject consent form.....	91
Appendix B: Participant information sheet	92
Appendix C: Ethical approval.....	94
Appendix D: Labview data collection program (front panel)	96
Appendix E: Labview data collection program (back panel)	97
Appendix F: Validity of the data from Labview analysis program	99

LIST OF PUBLICATIONS / PRESENTATIONS FROM THIS THESIS

Wilcock, I. M., Cronin, J. B., & Hing, W. A. (2006). Physiological response to water immersion: a method for sport recovery? (Accepted by Sports Medicine on 10.02.06).

Wilcock, I. M., Cronin, J. B., & Hing, W. A. (2006). Water immersion: Does it enhance recovery from exercise? (Submitted to International Journal of Sports Physiology and Performance on 15.02.06).

Wilcock, I. M., Cronin, J. B., & Hing, W. A. (2006). Water immersion, active recovery and passive recovery effects on explosive exercise and blood plasma fraction. (Submitted to Journal of Sports Sciences on 19.03.06).

ATTESTATION OF AUTHORSHIP

I hereby declare that this submission is my own work and that to the best of my knowledge and belief, it contains no material previously published or written by another person nor material which to a substantial extent has been accepted for the qualification of any degree or diploma of a university or other institution of higher learning, except where due acknowledgement is made in the acknowledgements.

Signed.....

Ian Wilcock

Date.....

ACKNOWLEDGEMENTS

I would like to express my gratitude to the following individuals who have contributed to the completion of this thesis.

To Associated Professor John Cronin, my supervisor. Thank you for your support and mentorship over these last few years. Your enthusiasm and dedication to sport science students pursuing higher learning and work in the industry is invaluable. You have become a friend rather than a mentor and I look forward to working and gaining more knowledge and experience with you in the future.

To Dr Wayne Hing. Thank you for your input and time. It was great to get a different perspective on my writing and structure. I am looking forward to future collaborations in the field of recovery interventions.

To Belinda. Thank you for putting up with my one track mind whilst I have been a student. I look forward to actually spending time and having some occasional holidays together now.

To my old boss Harry Price, and to Peter Hall. I could not have asked for a more supportive and symbiotic relationship over the last five years. Being able to pay the rent and eat when times got tough made things so much easier.

To my other post-grad roomies, Katrina, Marcus, Chief, Nick, Nigel, Claire, and Grace. Thank you for all of the fun times, debates, support, Friday feeds and friendship.

To those who were subjects in my study. Thank you for your commitment to the time and pain involved in this research. I am sure I will be repaying some of these debts in the future.

To Professor Will Hopkins. Thank you for your statistical expertise (yes I did manage to understand most of it). As with Jamie, I apologize for the inclusion of P-values in this thesis.

I would also like to acknowledge the Auckland University of Technology and the Alumni Association for providing the fee-scholarship that allowed me to continue my education.

And finally, I would like to thank Kiri. Your questions were what aroused my interest into the practical use of recovery interventions.

DEDICATION

This thesis is dedicated to the grumpy café lady. Thank you for making me realise that life is good if you enjoy your work.

INTELLECTUAL PROPERTY RIGHTS

This thesis may be utilised for the purpose of research or private study provided that due acknowledgement is made where appropriate and that the author's written permission is obtained before any material from the thesis is published.

I agree that the Auckland University of Technology Library may copy this thesis, in electronic or photocopied form, to supply the collection of other libraries on request from those libraries. I also agree that this thesis may be photocopied for supply to any person in accordance with the provision of Section 56 of the Copyright Act 1994.

ABBREVIATIONS

The following abbreviations are used throughout this thesis.

90%CI	90 percent confidence interval
%BV	Percentage of blood volume
%MAP	Percentage of mean aerobic power attainable during exercise
%MAV	Percentage of maximal aerobic running velocity attainable during exercise
%RM	Percentage of maximal load at which a subject can perform one repetition
% $\dot{V}O_2$ max	Percentage of maximal rate of oxygen uptake attainable during exercise
A	Active recovery
AI	Active recovery followed by water immersion
BLa	Blood lactate
CV	Coefficient of variation
CVP	Central venous pressure
ES	Cohen's effect size
I	Water immersion

ICC	Intraclass correlation coefficient
MAP	Mean arterial pressure
MVIC	Maximal voluntary isometric contraction
P	Passive recovery
RPE	Rate of perceived exertion
Rpm	Revolutions per minute
SD	Standard deviation
SE	Standard error
TPR	Total peripheral resistance

ABSTRACT

Optimising recovery post-game or post-training could provide a competitive advantage to an athlete, especially if more than one bout of exercise is performed in a day. Active recovery is one common method that is thought to enhance the recovery process. Another recovery method that is gaining popularity is water immersion. The objective of this thesis was to analyse whether these two recovery methods provided greater recovery from explosive exercise than passive recovery. A physiological rationale that may explain the possibility of enhanced recovery with water immersion was initially investigated. The literature surrounding active recovery, water immersion and passive recovery on strength, cycling, running and jumping was then examined. Following these reviews an experimental study was conducted investigating the effects of water immersion, active recovery and passive recovery conducted after repeated bouts of explosive exercise.

The rationale for active recovery post-exercise is that during intense exercise, fluid from the blood is forced into the working muscles due to the increase in mean arterial pressure, which increases muscle volume and decreases blood plasma fraction. Active recovery reduces this exercise induced edema and, with an associated increase in blood flow throughout the body, may increase the metabolism of waste substrates produced during exercise. Researchers have observed this increased substrate metabolism with reductions in post-exercise blood lactate accumulation following active recovery. Water immersion would appear to cause a similar physiological response to active recovery without the need to expend extra energy. When a large portion of the body is immersed, hydrostatic pressure acts on the body's fluids within the immersed region. Fluids from the extravascular space move into the vascular system reducing exercise-induced increases in muscular volume and reducing soft tissue inflammation. Additionally, blood volume increases and is redistributed towards the central cavity, which in turn increases cardiac preload, stroke volume, cardiac output, and blood flow throughout the body. Cardiac output increases in relation to the depth of immersion and have been observed to increase by as much as 102% during head-out immersions. These cardiovascular responses occur without any increase in energy expenditure. If extra-intravascular fluid movement is enhanced,

then the movement and metabolism of waste substrates could increase. Observations of increased post-exercise blood lactate clearance with water immersion would support this theory

Most methodologies studying the performance benefits of active recovery and water immersion suffer many limitations. These limitations often consist of the experimental time schedule not replicating what is likely to occur in a practical situation, no isolation of water temperature and hydrostatic pressure effects, and lack of a sport-like exercise consisting of repeated expressions of explosive power. Light-intensity active recovery and water immersion do not appear to be detrimental to performance, but neither does there appear to be enough evidence to claim they are beneficial. Effects of active recovery and water immersion would seem to be trivial to small, with any benefits more likely following multiple bouts of high-intensity exercise and recovery or following muscle damaging exercise. There may be a link between blood plasma fraction and performance, however, evidence is inconclusive.

Given these issues and limitations the aim of this research was to investigate whether combinations of active recovery, water immersion and passive recovery could maintain peak power and work during subsequent bouts of explosive exercise. We also investigated whether there was any difference in subjects' blood plasma fraction and perceived fatigue between the recovery modes. A cross-over experiment was conducted on seven subjects over four weeks. On the same day of each week subjects performed three sessions of maximal jumping, each two hours apart, followed by a different recovery method. Each jump session consisted of three sets of 20 maximal jumps repeated every three seconds, with a minute's rest in-between. Immediately following the jumping subjects performed 10 minutes of either (A) active recovery on a cycle ergometer followed by seated rest, (I) immersion to the gluteal fold in 19°C water followed by seated rest, (AI) active recovery followed by immersion, or (P) seated passive rest. Jumping was conducted on an instrumented supine squat machine that allowed the measurement of total peak power and total work. Pre-jump, post-jump and post-recovery blood was taken and the percentage of blood plasma fraction calculated. Perceived leg fatigue was also measured at these times.

Observed differences in total peak power and total work between the recovery modes were non-significant. No differences were observed in the change of blood plasma fraction between the recovery modes or perceived fatigue. One reason for any lack of difference between the recovery modes may have been the brevity of the recovery time. Research that has observed significant benefits of active recovery and water immersion compared to passive recovery have used recovery times greater of 15 minutes or more. Additionally, changes in blood plasma fraction between active recovery, water immersion and passive recovery have not been apparent until at least 10 minutes post-recovery in previous research. Alternatively, rather than brevity, it may be that active recovery or water immersion simply does not provide any benefit to performance recovery. Overall there is a meagre amount of research into active recovery, water immersion and passive recovery. Further research that incorporates a variety of exercise and recovery protocols is required.

CHAPTER 1:

INTRODUCTION

Recovery from exercise involves the return of the body from a fatigued state towards its normal physiological and performance baseline. The recovery process occurs naturally over time after a fatiguing event, under normal circumstances. However, during tournaments athletes can perform numerous games or heats each day over a small number of days. In tournament situations the recovery process becomes even more critical as there can be inadequate time available to fully recover from a fatigued state before the next bout of exercise (Hamlin, 2001). Furthermore, implicit in maximising training adaptation is the restoration of the body to its normal biological status as soon as possible after the training stimulus. Quicker recovery from fatigue has potential benefits in that it allows athletes to train or compete at a higher intensity and could provide a competitive edge (Cochrane, 2004; Hamlin, 2001). Given this information, many sports attempt to hasten the recovery process between sessions so that athletes are ready to train or compete optimally. Methods used to achieve this range from nutritional supplementation to various physical modalities (e.g. massage, water baths, stretching, active recovery, whirlpools, water-jet massage and muscle stimulation). Three of these recovery methods that are of particular interest to this thesis are passive recovery, active recovery and water immersion.

Passive recovery refers to an athlete performing nothing out of the ordinary after strenuous activity (Sanders, 1996). In research, passive recovery generally consists of a subject sitting idle for the duration of the recovery time (Lattier *et al.*, 2004; Monedero & Donne, 2000; Sanders, 1996; Thiriet *et al.*, 1993; Vaile *et al.*, 2004; Weltman *et al.*, 1979; Weltman *et al.*, 1977), or if over a day maintaining a normal routine (Kuligowski *et al.*, 1998; Viitasalo *et al.*, 1995). Passive recovery is generally used as the control in recovery research, since no attempt is made at influencing the physiological responses of the body.

In terms of active recovery, light to moderate exercise has been generally accepted as a method to enhance recovery after strenuous physical performance (Calder, 2003). Mode, intensity and duration of the exercise employed in the course of active

recovery vary but normally consist of exercising at intensities less than the onset of blood lactate accumulation for a duration of 5 – 20 minutes (Lattier et al., 2004; Monedero & Donne, 2000; Netball New Zealand, 2004; Sanders, 1996; Thiriet et al., 1993; Vaile et al., 2004; Weltman et al., 1979; Weltman et al., 1977). Even though active recovery may play an important role in sports performance it is not well researched in regards to linking mechanisms and performance. It has been postulated that active recovery increases the ability to metabolise muscle lactate and maintain power outputs (Ahmaidi et al., 1996) and has a beneficial effect on psychological recovery by enhancing relaxation (Suzuki et al., 2004). Active recovery is thought to result in an increased blood flow through the muscles, which may improve nutrient delivery and waste metabolism. Removal of exercise-induced waste may be linked to intra- and extravascular fluid shifts as they have been observed to closely follow blood lactate kinetics (Thiriet et al., 1993). Research to date on active recovery has varied in regards to protocols and the variables measured, and have generally focused on linking blood lactate to performance (Bonen & Belcasro, 1977; Coffey *et al.*, 2004; Rontoyannis, 1988; Sanders, 1996). However, while active recovery reduces blood lactate accumulation (Bond *et al.*, 1991; Bonen & Belcasro, 1977; Sanders, 1996; Thiriet et al., 1993; Weltman et al., 1979; Weltman et al., 1977), the results regarding performance are not conclusive and whether active recovery reduces fatigue from strenuous physical activity and improves performance is debatable and a topic of research interest.

Another method of post-game or post-training recovery that is gaining popularity is water immersion (Calder, 2000; Cochrane, 2004). Throughout history immersion in hot and/or cold water has been used as a therapeutic treatment for restoring physical and mental health (Bender et al., 2004; Calder, 2003). There are four modalities of water immersion that can be used as a recovery strategy: cold water (cryotherapy), thermo-neutral water, hot water (thermotherapy) or contrasting water (alternating hot and cold water) immersion. After training or games some athletes may spend up to 20 minutes using one of these immersion modalities to enhance recovery (Calder, 2003; Cochrane, 2004). During immersion the pressure of water on the body causes a redistribution of body fluid (Arborelius *et al.*, 1972; Farhi & Linnarsson, 1977; Watenpaugh *et al.*, 2000), with increasing levels of immersion increasing the hydrostatic pressure on the body (Farhi & Linnarsson, 1977). Fluid shifts due to

immersion appear to increase cardiac output and muscle blood flow, and reduce peripheral resistance (Bonde-Petersen *et al.*, 1992; Gabrielsen *et al.*, 2000; Yun *et al.*, 2004) increasing blood lactate recovery (Hamlin & Magson, 2002; Nakamura *et al.*, 1996; Sanders, 1996) without a subject expending the energy required during active recovery. These changes could aid the recovery process.

Rather than analysing the actual effect on performance most water immersion studies have concentrated on the physiological effects of immersion. Research on the effects of cryotherapy, thermotherapy and contrast therapy on performance has produced conflicting results (Burke *et al.*, 2003; Clarke, 1963; Coffey *et al.*, 2004; Howard *et al.*, 1994; Kuligowski *et al.*, 1998; Lane & Wenger, 2004; Vaile *et al.*, 2004). Limited research has been conducted on water immersion per se and performance recovery. Water temperature may be the major factor in any benefit to performance recovery rather than water immersion itself, or vice versa (Nakamura *et al.*, 1996). For example Howard *et al.* (1994) observed significant reduction in higher-velocity ($180 - 400^{\circ}\text{s}^{-1}$) isokinetic torque after cold water immersion (12°C) but not following thermo-neutral immersion. To understand whether the hydrostatic pressure or temperature of water is causing possible performance effects there is a need to study each in isolation. More research is required that utilises thermo-neutral water temperatures to isolate hydrostatic pressure effects. There is also a need to use methods that could be practically applied 'in the field'. Facilities or resources to heat and cool water may not be available to sports teams. Due to the lack of research on water immersion per se and ease of use 'in the field' for teams, tap water has been used in this study. While tap water is generally cool ($15 - 20^{\circ}\text{C}$), it is water that is readily and naturally available.

Another problem with research into water immersion as a means of recovery is that researchers have not considered combinations of passive recovery, active recovery and water immersion. Such methodologies seem to lack face validity in terms of what actually happens in the sporting environment. That is, post-exercise athletes / teams often perform water immersion after a bout of active recovery.

This thesis will attempt to address these issues. Of particular interest are the potential performance benefits of water immersion and active recovery after exercise compared to passive recovery, and whether fluid shifts may be a factor in any benefit.

Purpose statement

The purpose of this thesis is to investigate the effect of water immersion, and active and passive recovery strategies on performance. First, the literature concerning water immersion and its possible physiological influences on recovery will be reviewed and discussed. Second, literature concerning the effect of water immersion, active recovery and passive recovery effects on performance will be discussed. Third, the effect of combinations of these three different recovery strategies on total peak power, total work, blood plasma fraction and fatigue during repeated bouts of explosive exercise will be experimentally examined and discussed.

Aims and hypothesis

The aim of this thesis is to determine the effect that combinations of active recovery, water immersion and passive recovery have on peak power and work during repeated bouts of explosive exercise. A secondary aim of this study is to progress the understanding of the effects of different recovery strategies on short-term performance and fluid shifts. It is hypothesised that post-exercise water immersion and active recovery have a similar effect, reducing performance decrement and increasing the return to homeostasis of blood plasma levels compared to passive recovery.

Significance of thesis

This thesis will improve the understanding of physical recovery modalities and performance by exploring possible performance benefits of recovery methods similar to those used in the field. The findings of this research will allow athletes, coaches, trainers and medical staff to make informed decisions regarding the recovery

strategies they adopt. It may also provide the framework for further research into the effectiveness of passive and active recovery and water immersion.

Limitations

1. The main limitation was the low subject number used in the experimental study. Due to time constraints and withdrawals a lower number of subjects participated than was sought, reducing the statistical power of the study.
2. All testing and training sessions were performed in a laboratory setting using a supine squat machine. The supine squat machine was chosen for safety, instrumentation, accessibility and reliability. Therefore, any results indicating recovery benefits in the laboratory setting may not transfer to the field.
3. Sports consist of a variety of intensities and movements. This research investigated only repeated bouts of maximal explosive jumping. Therefore, the findings of this study may be limited to repeated bouts of jumping.

Note to reader

This thesis is presented as five chapters, this introduction, three papers and a summary. Chapter two is a literature review summarising the physiological effects of water immersion and its possible benefit to performance recovery. The third chapter is another literature review that summarises the effect on subsequent performance that water immersion, active and passive recovery may have. The fourth chapter is an experimental paper that analyses the effect that water immersion, active recovery and passive recovery have on explosive exercise and blood plasma. Each of these sections has been written specifically for publication. Therefore, some of the information in this thesis may appear repetitive due to this format. Regardless, this thesis fulfils the AUT Master of Health Science guidelines for thesis submission.

CHAPTER 2:

PHYSIOLOGICAL RESPONSE TO WATER IMMERSION: A METHOD FOR SPORT RECOVERY?

Introduction

Water immersion has been used in some cultures for centuries as a means of health restoration (Bender et al., 2004; Calder, 2003). Recently water immersion has gained popularity as a means to improve recovery from exercise, though much of its use is based on anecdotal information (Calder, 2003). There is some basis for the use of water immersion (non-exercising) to enhance recovery from exercise as it can produce beneficial physiological changes within the body. These physiological changes have been attributed principally to effects of hydrostatic pressure and temperature. Exercise in water is also used for recovery by some sport teams. However, the concern of this review is the physiological response of the body during non-exercise immersion. This review will briefly describe the water immersion recovery strategies used in the laboratory and in the field. Thereafter, the effect that hydrostatic pressure may have on the body in thermo-neutral water, and the effect that cold and hot water temperature has on the body, will be examined.

Water immersion as a recovery strategy

There are four different methods of using water immersion in recovery; cryotherapy, thermotherapy, contrast therapy, and water immersion per se. Cryotherapy, thermotherapy and water immersion per se are immersion in water at a constant temperature, whereas contrast therapy is immersion in alternating extremes of temperature.

Cryotherapy

Cryotherapy is immersion in cold water. No specific water temperature range has been determined for cryotherapy. Low and Reed (1994) stated that the sensation of cold pain begins at 15°C, and some researchers have used temperatures of 15°C or less to study the effect of cold-water immersion on physiological (Bonde-Petersen et al., 1992; Sramek *et al.*, 2000) or performance changes (Burke et al., 2001; Burke et al., 2003; Lane & Wenger, 2004). Therefore, for the purpose of this review cryotherapy was considered to be immersion in water of 15°C or less. In the field cryotherapy normally consists of putting bags of ice in a container (such as a plastic drum) full of water, in which an athlete stands immersing their legs. For a large number of athletes, or team sports, this method can be impractical due to the time or resources required to immerse all of the athletes. In performance research the duration of immersion time varies from 15 – 20 min (Burke et al., 2003; Clarke, 1963; Lane & Wenger, 2004). However, in the field immersion time may be as little as 30 s due to the ability of the individual athlete's to withstand cold discomfort.

Thermotherapy

Thermotherapy refers to immersion in water that raises the core body temperature. This increase in core temperature occurs in water temperatures above 36°C (Bonde-Petersen et al., 1992; Greenleaf & Kaciuba-Uscilko, 1989; Weston *et al.*, 1987). Facilities that provide a heated pool are required to perform thermotherapy. Anecdotally, there are numerous teams that are based in facilities that provide heated baths/spas and who perform thermotherapy after training. However, compared to thermoneutral immersion, little research has been conducted on the physiological or performance effect of hot water immersion. An immersion duration of 10 – 20 min has been suggested by Brukner and Khan (2001) to aide athletic recovery and rehabilitation, though this time period does not appear to be based on research.

Contrast therapy

Contrast therapy is a post-exercise recovery method that has recently gained popularity (Cochrane, 2004). Contrast therapy necessitates alternating temperature immersion, from a hot to cold bath and vice versa. Protocols vary (see Table 1) but

generally consist of 30 to 300 seconds of one temperature extreme, immediately followed by 30 to 300 seconds of the contrasting temperature. This is repeated a number of times for a duration of four to 30 minutes in total. Vascular “pumping” caused by the variation in temperature has been proposed as the mechanism that could improve recovery (Cochrane, 2004; Myrer *et al.*, 1994; Sanders, 1996). Whether such a contention is supported by the literature will be discussed later in this review.

Table 1: Examples of contrast therapy protocols

Author/ reference	Temperature (°C)		Application time		Repeats	Order	
	Cold	Hot	Cold	Hot		Start	End
Research							
Coffey (2004)	10	42	1 min	2 min	5	Cold	Hot
Cote (1988)	10 – 15	39 – 41	1 min	3 min	4	Hot	Hot
Hamlin (2002)	8 – 10	38	1 min	1 min	3	Cold	Hot
Hamlin (2004)	8 – 10	38	1 min	1 min	3	Cold	Hot
Higgins (1998)	15	40	1 min	Initially 10 min, then 4 min	5	Hot	Cold
Kuligowski (1998)	13	39	1 min	3 min	6	Hot	Cold
Sanders (1996)	15	38	0.5 min	3.5 min	3	Hot	Cold
Vaile (2004)	8 – 10	40 – 42	1 min	2 min	5	Cold	Hot
Text							
Briggs (2001)	10 – 15	-	1 min	3 min	5 times/h	Hot	Cold
Brukner (2001)	15	40	1 min	4 min	3 – 7	Hot	Cold
Clover (2001)	13 – 18	38 – 43	1 min	4 min	3 - 4	-	-
Walsh (1996)	10 – 18	38 – 44	1 min	Initially 10 min, then 4 min	30 min total	Hot	Hot.
Zuluaga (1995)	-	45	1 min	3 min	NA	Cold	Cold

Water immersion per se

Water immersion per se is both the easiest method of application in the field and, compared to the other water immersion modes, widely researched (physiologically). No resources are required to heat or cool the water, only a container, bath or pool in

which to immerse athletes. The temperature widely used in this mode ranges from cool to thermoneutral, which for this review is considered to range from 16 to 35°C. Research into the physiological effect of water immersion generally has concentrated on the use of thermoneutral immersion, and has ranged in immersion time from 5 minutes to 6 hours. This review only considered the effect of immersion over a maximum of 30 minutes to replicate a time similar to post-exercise recovery sessions. Unlike cryotherapy, thermotherapy or contrast therapy, the main effect of water immersion per se comes from the effect of hydrostatic pressure and perhaps to a lesser degree buoyancy, rather than temperature. The few studies into water immersion as a performance recovery method have concentrated on cryotherapy, thermotherapy or contrast therapy, combining both the temperature and hydrostatic pressure effects (Burke et al., 2003; Coffey et al., 2004; Lane & Wenger, 2004; Sanders, 1996; Vaile, 2003; Viitasalo et al., 1995). To gain a greater understanding of water immersion the effects of hydrostatic pressure and temperature should be studied in isolation and therefore provide the focus for the remainder of this review.

Hydrostatic pressure

When a body is immersed, water exerts a compressive force on the body called hydrostatic pressure. This pressure can cause the displacement of fluids within the body from the extremities towards the central cavity. This displacement of fluid may increase the translocation of substrates from the muscles, increase cardiac output, reduce peripheral resistance, and increase the ability of the body to transport substrates. Additionally, the antigravity effect caused by buoyancy may reduce perception of fatigue and aid energy conservation. The following section will explore the physiological effects of hydrostatic pressure during thermoneutral immersion.

Water pressure

Air exerts pressure equally on all sides of the body. At sea level the pressure exerted around the body equates to approximately 1013 Pa. Water is more than 800 times denser than air, as a consequence at an equal depth water will create a greater pressure

than air (Bove, 2002). Due to this greater density water produces the same pressure at a depth of only 10 m as the entire atmosphere of air at sea level (Bove, 2002).

When immersed in water hydrostatic pressure acts on the body in relation to the depth of immersion. The amount of pressure that acts on a body is equals to $P = P_{atm} \cdot g \cdot \rho \cdot h$. Where P = water pressure; P_{atm} = atmospheric pressure (standard sea level 1013 hPa); g = gravity ($9.81 \text{ m}\cdot\text{s}^{-2}$); ρ = water density ($1000 \text{ kg}\cdot\text{m}^{-3}$), and; h = height of the water.

Water pressure does not correlate to the total weight of the water in a vessel, only to the depth. If the wall of a container is solid then the walls of the container exert a pressure on the water equal to the pressure of the water at that depth. This means that water pressure is a force per unit area and is transmitted equally throughout the water at a given level. On a body immersed in water the pressure varies relative to depth. A body part such as a foot immersed at a depth of 1 m would have 981 Pa of extra pressure acting on it, whereas at hip level (if at a 0.1 m depth) only an extra 98.1 Pa. To relate this external pressure to blood pressure measurement, for every 1 cm depth of immersion the pressure increases by 0.74 mmHg. The proportional change in pressure with depth causes an upward squeezing action on the body, which at 1 m depth (74 mmHg) is almost equal to normal diastolic blood pressure (80 mmHg).

The human body is mostly water with the addition of some oil (fats) and proteins. Because water is essentially non-compressible it occupies the same volume regardless of pressure (Chaplin, 2005). When external pressure on the body increases, gas and fluid substances are displaced to lower pressure areas (Bove, 2002; Farhi & Linnarsson, 1977; Lollgen *et al.*, 1981). Therefore, a person standing in water experiences compression on the body acting inwards and upwards. During hip-level immersion this “squeezing” causes the displacement of fluid from the lower extremities of a person into the thoracic region. During head-out immersion hydrostatic pressure on the central cavity reduces the residual air volume of the lungs increasing the displacement of fluids into the thorax (Farhi & Linnarsson, 1977). It is the movement of these fluids that may enhance the ability of an athlete to recover from exercise.

Fluid shifts

Under normal conditions the body is comprised of 50 – 60% of fluid, which is located either in the intracellular, interstitial (between cells), or intravascular (blood plasma) space (see Figure 1). Typical intracellular, interstitial and plasma volumes are 35 - 40%, 11 – 15% and 4 - 5% respectively of body weight (Lassiter & Gottachalk, 1980). Fluid within these compartments acts as a vehicle for the transport and exchange of materials, such as metabolic wastes and nutrients, between the body and the external environment.

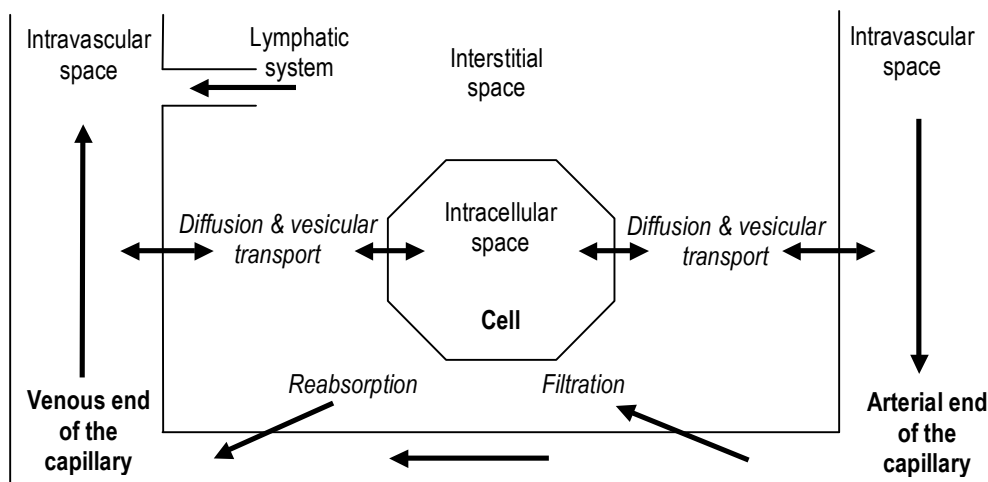


Figure 1: Schematic diagram of intracellular-intravascular fluid movement.

Movement of fluid and materials between the intravascular and extravascular space occur in the vascular capillaries. Fluid and substance movement across the capillaries occur via three processes; (1) diffusion, (2) vesicular transport, and (3) filtration-reabsorption. Diffusion is the movement of fluid/substances from a high concentration to a low concentration, whereas vesicular transport is active transport (requiring ATP) of substances across the vascular membrane. Diffusion occurs along all of the capillary membrane and accounts for the largest exchange of fluids and substances (Pappenheimer, 1953). Filtration-reabsorption is the net movement of fluid due to the capillary-interstitial pressure gradient. Filtration is the movement of fluid into the interstitial space at arteriolar ends of the capillaries, which is then reabsorbed at the

venular ends of the capillaries. Approximately two to four litres of this fluid per day is not reabsorbed by the capillaries through filtration-reabsorption, but moves through the lymphatic vessels and drains into the subclavian veins (Milnor, 1980). Disruption in the balance of filtration-reabsorption, through effects such as physical trauma, lymph blockage or changes in pressure gradients, can cause an abnormal increase in interstitial fluid in localised areas, a condition called edema, swelling, or inflammation.

It is well documented that water immersion causes a rise in central blood volume, which increases with the depth of immersion (Arborelius *et al.*, 1972; Echt *et al.*, 1974; Johansen *et al.*, 1997; Lollgen *et al.*, 1981). The increase in central blood volume is due to two effects, haemodilution (increased fluid reabsorption) and blood displacement. During immersion at hip level hemodilution occurs due to negative transcapillary pressure in the legs. This pressure gradient causes a fluid shift from the interstitial to intravascular space in the legs (Gabrielsen *et al.*, 2002; Johansen *et al.*, 1997; Stocks *et al.*, 2004).

With immersion above hip level additional increases in central blood expansion results as blood from the abdomen, which acts as a blood reservoir, is displaced (Johansen *et al.*, 1997). Norsk, Bonde-Petersen, and Warberg (1985) studied changes in plasma concentration during head-out immersion and observed significant decreases in haematocrit and haemoglobin concentration with an associated $6.5 \pm 1.9\%$ (mean \pm SE) increase in plasma volume. Hinghofer-Szalkay, Harrison and Greenleaf (1987) observed similar results when using plasma densitometry to measure transvascular fluid shifts during immersion to the neck. After 30 minutes of thermo-neutral immersion the six men in the study had plasma volume increases of $11 \pm 3\%$, with decreased blood and haematocrit densities (-1.5% and -1.0% respectively). While plasma dilution occurred in these studies, the intravascular fluid shift was also accompanied by a plasma protein shift of albumin.

Stocks *et al.* (2004) suggested that the increase in the blood volume ultimately comes at the expense of intracellular fluid, though further investigation and verification of this hypothesis is required. Such fluid shifts would increase the intracellular-

intravascular osmotic gradients and some intracellular constituents, such as metabolic wastes, may leave the cells and interstitial space to maintain an osmotic balance (Hinghofer-Szalkay et al., 1987; Stocks et al., 2004). It is possible then that immersion may cause improvements in the translocation of substrates, reducing transportation time and increasing the clearance of waste substrates (Coffey et al., 2004; Nakamura et al., 1996; Sanders, 1996; Tomasik, 1983). Hydrostatic pressure during water immersion aides the return of fluid from the muscles into the blood (Vaile, 2003) diluting the blood and improving diffusion gradients (Lassiter & Gottachalk, 1980). Such an increase in diffusion may be a factor in the increased clearance of blood lactate in subjects that have been immersed in water (Coffey et al., 2004; Hamlin & Magson, 2002; Hamlin & Sheen, 2004; Nakamura et al., 1996; Sanders, 1996). If plasma volume increases due to a fluid shift from the interstitial space, the translocation of substrates and the ability to metabolise waste products may enhance athletic recovery.

Exercise-induced muscle edema

Apart from assisting the possible removal of substances a positive gradient between internal tissue hydrostatic pressure and capillary filtration pressure may also improve the reabsorption of interstitial fluids, reducing edema (Friden & Lieber, 2001; Vaile, 2003). An increase in the pressure gradient between the interstitial compartment of the legs and the intravascular space caused by hydrostatic pressure should reduce edema in a similar fashion to compression stockings (Jonkera *et al.*, 2001; Partsch *et al.*, 2004). Exercise-induced edema may then be reduced during water immersion.

Exercise causes a shift of plasma from the blood into the muscles, with this fluid movement being relative to the intensity of the exercise (Collins *et al.*, 1989; Gillen *et al.*, 1991; Green *et al.*, 1984; Hildebrandt *et al.*, 1992; Knowlton *et al.*, 1987; Miles *et al.*, 1983). The mode of exercise does not appear to be a factor but rather the respondent increase in mean arterial pressure (Knowlton et al., 1987). Researchers have observed that cycling at intensities of 30 - 120% $\dot{V}O_2$ max decreased blood plasma by 5 – 17% as fluid shifted intramuscularly (Gillen et al., 1991; Green et al., 1984; Hildebrandt et al., 1992; Miles et al., 1983; Mohsenin & Gonzalez, 1984). Whereas during resistance training plasma decreases of 8 – 14% have been observed in relation

to an intensity range of 40 - 70%RM (Knowlton et al., 1987). The decrease in plasma volume observed by Knowlton et al. (1987) during the resistance training correlated highly with an increasing mean arterial pressure ($r = -0.98$). However, fluid shifts from the vascular space reflected movement into active but not inactive muscle during exercise (Ploutz-Snyder *et al.*, 1995). Using magnetic resonance imaging pre- and post-resistance exercise (six sets of 10RM squats) Ploutz-Snyder et al. observed that an increase in the cross-sectional area of the vasti and adductor muscle groups coincided with a 22% decrease in plasma volume (measured by Evans blue dye). The coefficient of determination between the plasma decrease and volume increase in the adductor and vasti muscle groups was strong ($r^2 = 0.75$, $p = 0.0157$). Muscles that were less active during the squat (rectus femoris and the hamstring muscle groups) had smaller non-significant increases in their cross-sectional area.

Edema in response to exercise or muscle damage may increase both the transport route and compression of localised capillaries, reducing oxygen delivery to localised cells. With excessive muscle edema such an increase in transportation time can cause an increase in cellular damage or death (Friden & Lieber, 2001; Northoff *et al.*, 1998; Shephard & Shek, 1998; Tiidus, 1998). A positively increased pressure gradient can reduce cellular infiltration by leucocytes and monocytes decreasing further tissue degeneration (Lecomte *et al.*, 1998; Mishra *et al.*, 1995; Vaile et al., 2004). Such reduction in muscular inflammation may improve contractile function as well as lowering the levels of inflammatory cells and muscle enzymes circulating in the blood (Cesari et al., 2004; Vaile et al., 2004). Reducing edema may therefore decrease secondary damage to tissue which in turn may increase the ability of an athlete to recover from muscle damaging exercise (Sayers *et al.*, 2000; Vaile et al., 2004).

Cardiac response

The predominant effect of water immersion and the associated increase in central blood volume expansion is an increase in cardiac pre-load. Central blood volume expansion increases atrial pre-load and stroke volume. Increasing the depth of immersion increases the stroke volume. Compared to non-immersion, at the level of the hips stroke volume has been reported to increase by 12 - 37% (Farhi &

Linnarsson, 1977; Lollgen et al., 1981), this increases to 38 – 67% at the level of the xiphoid process (Bonde-Petersen et al., 1992; Farhi & Linnarsson, 1977; Gabrielsen et al., 2002; Lollgen et al., 1981; Weston et al., 1987) and 28 - 95% during head-out immersion (Arborelius et al., 1972; Farhi & Linnarsson, 1977; Lollgen et al., 1981; Park *et al.*, 1999; Shiraishi *et al.*, 2002; Yun et al., 2004). The effect size between each level of immersion from these studies ranged from moderate to very large (ES = 0.75 – 3.95).

When immersed in thermoneutral water to the level of the hips heart rate has a tendency to decrease by approximately 4 – 6% (Farhi & Linnarsson, 1977; Lollgen et al., 1981). Increasing depth of immersion to the xiphoid process has decreased heart rates by 11 – 18% compared to non-immersion (Bonde-Petersen et al., 1992; Farhi & Linnarsson, 1977; Gabrielsen et al., 2002; Gabrielsen et al., 2000; Lollgen et al., 1981; Watenpaugh et al., 2000; Weston et al., 1987). However, rather than a linear decrease in heart rate with increasing immersion depth, the decrease in heart rate during head-out immersion (3 - 15%) (Arborelius et al., 1972; Farhi & Linnarsson, 1977; Gabrielsen et al., 2000; Johansen et al., 1997; Lollgen et al., 1981; Park et al., 1999; Shiraishi et al., 2002; Sramek et al., 2000; Yun et al., 2004) is less than that observed during immersion to the xiphoid process.

Individual heart rate response to immersion varies and decreases in heart rates have been non-significant in some studies (Arborelius et al., 1972; Bonde-Petersen et al., 1992; Johansen et al., 1997; Yun et al., 2004). While non-significant, a negative effect on heart rate was still apparent in the immersed subjects of these studies (see Table 2). The explanation for heart rate variance may be due to conflicting physiological feedback systems. Increasing mean arterial pressure causes arterial baroreceptors to bring about a reflex to slow the heart, most likely to prevent abnormally high blood pressure levels. Opposing this sympathetic response, an increased atrial stretch caused by the greater central blood volume (most notably when the water level rises above the hips) stimulates atrial stretch receptors and increases heart rate through a neural response called the Bainbridge reflex (Hakumaki, 1987). Individual physiological variables (such as heart size) would then determine the dominant reflex. Generally

speaking however, the mean heart rate of subjects appears to decrease during short-term thermoneutral immersion.

Table 2: Cardiac response of thermo-neutral immersion compared to non-immersion.

Author	% change in stroke volume (\pm SD)	% change in heart rate (\pm SD)	% change in cardiac output (\pm SD)
Hip level immersion			
Farhi (1977)	12 \pm 8	-4 \pm 9	14 \pm 12
Lillgen (1981)	37 \pm 19	-6 \pm 6	29 \pm 19
Xiphoid process immersion			
Farhi (1977)	64 \pm 16	-11 \pm 8	48 \pm 12
Bonde-Petersen (1992)	39 \pm 19	-14 \pm 14 ns	19 \pm 16
Gabrielsen (2002)	51 \pm 17	-13 \pm 8	33 \pm 12
Gabrielsen (2000)	-	-14 \pm 13	-
Lillgen (1981)	67 \pm 20	-11 \pm 6	48 \pm 20
Watenpaugh (2000) (male)	-	14 \pm 14	-
(female)	-	-18 \pm 20 ns	-
Weston (1987)	50 \pm 24	-11 \pm 15	31 \pm 23
Chin-out immersion			
Arborelius (1972)	28 \pm 22	-3 \pm 12 ns	29 \pm 18
Farhi (1977)	79 \pm 17	-7 \pm 11	66 \pm 13
Gabrielsen (2000)	-	-15 \pm 15	-
Johansen (1997)	-	-9 \pm 17 ns	-
Lillgen (1981)	79 \pm 20	-11 \pm 6	59 \pm 21
Park (1999)	55 \pm 30	-1 \pm 11 ns	53 \pm 28
Shiraishi (2002)	62 \pm 22	-9 \pm 9	52 \pm 23
Sramek (2000)	-	-8 \pm 3	-
Yun (2004)*1	52 \pm 11	-2 \pm 8 ns	49 \pm 21
*2	56 \pm 28	-6 \pm 9 ns	49 \pm 23
*3	95 \pm 33	-2 \pm 8 ns	102 \pm 37

ns = non-significant, * = different subject groups, 1 = breath hold divers (mean age 55), 2 = housewives (mean age 55), 3 = housewives (mean age 22). $p < 0.05$ unless otherwise stated.

Regardless of an individual's heart rate response the increase in stroke volume ultimately causes an increase in cardiac output. Observed cardiac outputs (see Table 2) vary but have been reported to be approximately 14 – 29% at hip level (Farhi & Linnarsson, 1977; Lollgen et al., 1981), 19 – 48% at the height of the xiphoid process (Bonde-Petersen et al., 1992; Farhi & Linnarsson, 1977; Gabrielsen et al., 2002; Lollgen et al., 1981; Weston et al., 1987) and 29 – 66% at head-out immersion (Arborelius et al., 1972; Farhi & Linnarsson, 1977; Lollgen et al., 1981; Park et al., 1999; Shiraishi et al., 2002). Yun et al. (2004) observed larger increases in cardiac output (102%) during head-out thermoneutral immersion, but was the only study in

which the subjects were both female and Korean. Subjects used in immersion research have for the most part been male and European.

Peripheral resistance and blood flow

Accompanying the increased cardiac output during immersion is a decrease in peripheral resistance implying that peripheral vasodilation occurs (Arborelius et al., 1972; Bonde-Petersen et al., 1992; Park et al., 1999; Weston et al., 1987; Yun et al., 2004). Total peripheral resistance (TPR) has been measured indirectly using the following calculations,

$$\text{TPR} = (\text{MAP} - \text{CVP}) / \dot{Q} \quad (\text{Park et al., 1999; Yun et al., 2004}),$$

$$\text{TPR} = (\text{MAP} - \text{Right atrial pressure}) / \dot{Q} \quad (\text{Arborelius et al., 1972}),$$

$$\text{TPR} = \text{MAP} / \dot{Q} \quad (\text{Bonde-Petersen et al., 1992; Weston et al., 1987}).$$

Where, MAP = mean arterial pressure; CVP = central venous pressure; \dot{Q} = cardiac output.

Decreases in peripheral resistance of 27 - 51% have been reported (Arborelius et al., 1972; Park et al., 1999; Yun et al., 2004) during head-out water immersion. Immersions at lower depths do not seem to reduce peripheral resistance. Gabrielsen et al. (2000) determined intramuscular blood flow using a counting signal between two cadmium-telluride detectors to measure the washout of injected ^{113}Xe -saline (k). From the determined blood flow, peripheral resistance was estimated as $\text{TRP} = \text{MAP} / k$. Muscular vascular resistance did not change significantly with immersion to the xiphoid process but decreased by approximately 15% with immersion to the neck. Similarly, Gabrielsen et al. (2000) observed that during immersion to the xiphoid process blood flow did not significantly increase but increased by $49 \pm 16\%$ (mean \pm SE) during head-out immersion. Bonde-Petersen et al. (1992) and Weston et al. (1987) also observed no significant decrease in total peripheral resistance during immersion to the xiphoid process.

A direct method of measuring venous tone during immersion is occlusion plethysmography. Echt et al. (1974) used this method to determine venous elasticity during a three-hour immersion to the neck of subjects. In the first 15 minutes of immersion the venous volume elasticity coefficient reduced from 16.6 to 13.5 mmHg·ml⁻¹·100g·tissue⁻¹, a decrease of 19%. However, reduction in peripheral resistance would seem to occur during head-out immersion only (Arborelius et al., 1972; Echt et al., 1974; Park et al., 1999; Yun et al., 2004). While studies of immersion at a depth under chin level have shown a decrease in peripheral resistance and increased blood flow (with small to very large effect sizes; ES = -0.59 - -2.62) the findings of these studies as compared to non-immersion have been non-significant (Bonde-Petersen et al., 1992; Weston et al., 1987).

With the increase in cardiac output, some vasodilation and reduction in peripheral resistance, increased blood flow may result throughout the body. During erect head-out immersion dogs have responded with large increases (>50%) in blood flow through the liver, intestinal tract, pancreas, spleen, renal cortex and skeletal muscle (Khosla & DuBois, 1979). If the responses displayed by dogs can be extrapolated to humans, greater organ and muscle blood flow may allow improved removal of metabolites and an increased ability to replenish energy stores. However, such extrapolation may not be possible. Blyden et al. (1989) observed that the clearance of lidocaine in man was unaltered by immersion to the neck, indicating no change in splanchnic blood flow. Epstein, Levinson, and Loutzenhiser (1976) using the clearance of p-aminohippuric acid and inulin to determine changes in renal plasma flow and glomerular filtration rate supported these findings of Blyden and colleagues. With greater cardiac output, lower peripheral resistance and vasodilation (at chin-level immersion at least), it could be logically assumed that an increase in blood flow through the muscles and perhaps some organs would occur. Results from Epstein et al. (1976) and Blyden et al. (1989) imply that renal blood flow is unaffected, though their research only analysed the clearance of certain chemicals not actual blood flows. Other metabolites, notably blood lactate, have an increased clearance rate when subjects have been partially immersed in water (Coffey et al., 2004; Hamlin & Magson, 2002; Nakamura et al., 1996; Sanders, 1996), indicating that blood flow through the muscle beds increase (Blyden et al., 1989). Whether blood flow increases to organs other than the kidneys is unknown and requires further investigation.

Weightlessness and perceived fatigue

One significant consequence of water pressure being proportional to the immersion depth is that the body will weigh less when immersed in a liquid i.e. it is easier to lift a rock in water than it is on dry land. This is because water exerts a net upward force on the body immersed in it. This upward force helps to support all or part of the weight of the body immersed in it. The upward force exerted by a fluid on any object placed in it is called buoyancy or hydrostatic upthrust.

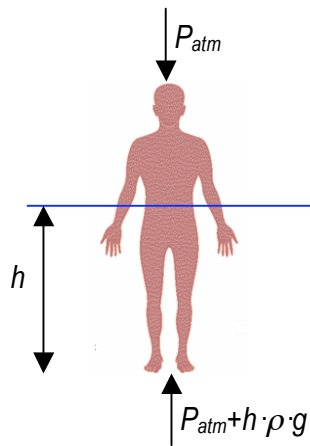


Figure 2: Schematic diagram of pressures acting on an immersed body

The force created by upthrust is calculated as,

$$F = h \cdot \rho \cdot g \cdot A$$

Taking the calculation further,

$$F = V \cdot \rho \cdot g$$

$$F = m/g$$

Where h = height; ρ = water density; g = gravity; A = base area; V = immersed volume, and m = mass.

Hence there is a net upward pressure, giving rise to an upward force equal to upward pressure times horizontal base area (as shown in Figure 2). In other words any body partly or wholly immersed in a liquid, experiences an upthrust that is equal to the weight of the liquid displaced (Archimede's Principle). The greater the body density

the less buoyancy a person has, which is why people with higher fat mass (less density) are more buoyant than those who are lean.

Table 3: Water immersion and perception of fatigue

Author	Scale	Exercise	Recovery	Measurement timing	Main findings
Coffey (2004)	20 point RPE recovery scale	Maximal sprints	15 min contrast therapy, active or passive.	4-, 8-, 12-, 16-, 20-min post sprint. Pre and post 2 nd set of sprints.	No sig. difference between recoveries
Kuligowski et al (1998)	12-cm Graphic pain scale	DOMS inducing arm curl	24 min warm water, cold water, contrast therapy or control	0-, 24-, 48-, 72-, 96-hrs post exercise.	↓ pain perception with cold water and contrast therapy
Nakamura et al (1996)	5 point fatigue scale	10 min of Submaximal cycling	10 min in 30°C or 38°C water bath, or control	Not specified	↓ fatigue for 30°C bathing
Sanders (1996)	10 point CR scale	Repeated Wingate test	12 min contrast therapy, active or passive.	3- and 7-min during recovery. Post recovery. Post 2 nd exercise bout	↓ fatigue at all times with contrast therapy
Vaile et al (2004)	10 cm VAS-score	DOMS inducing leg press	15 min contrast therapy or control	a) 0 b) 24 hrs c) 48 hrs d) 72 hrs	Both groups had increased pain. No sig. difference.
Viitasalo et al. (1995)	10 cm VAS-score	Strength, plyometric and sprint training over 3 days	20 min warm-water immersion or control	Over 2 days post-training	↓ DOMS during immersion week

DOMS = delayed onset of muscle soreness; VAS = visual analogue scale; CR = Borg category ratio scale; ↑ = significantly increased; ↓ = significantly decreased; No sig. = no significant difference.

The effect of buoyancy is a reduction in the gravitational forces that act on the musculoskeletal system, allowing for a greater relaxation of gravitational muscles and conservation of energy. Such greater relaxation would appear to reduce perceived fatigue. A number of studies (see Table 3) have observed lower perception of fatigue after exercise during and after water immersion. While two authors (Coffey et al., 2004; Vaile et al., 2004) did not observe any significant difference in perceived fatigue, both did observe a moderate effect of lower sense of fatigue with water immersion compared to other recovery modes (ES = 0.50 - 0.89).

The decrease in the perception of fatigue may also be due to reduced neuromuscular responses during water immersion (Koryak, 2002; Pöyhönen & Avela, 2002; Pöyhönen *et al.*, 1999). During water immersion EMG activity produced during maximal contractions have been observed to reduce by 11 – 35% (Pöyhönen & Avela, 2002; Pöyhönen *et al.*, 1999). In their 2002 study Pöyhönen and Avela also observed a 13% decrease in maximal voluntary contraction force during immersion. Immersion may modify the peripheral processes associated with contraction and change central and/or neural command contractions (Koryak, 2002), or trigger inhibitory mechanisms (Pöyhönen & Avela, 2002). The reduced perception of fatigue may result not only from a reduction in the neuromuscular activation required to maintain posture but also from an overall reduction in neural transmissions. However, more research is required on whether water immersion does reduce neural efficiency, whether such reductions are due to weightlessness or hydrostatic pressure (Pöyhönen & Avela, 2002), and if there are any post-immersion effects.

Temperature

The previous sections have considered the physiological response that thermoneutral immersion in water can have on a person. This literature review has concentrated on the immersion of subjects in thermoneutral water due to the abundance of research in this area. Colder, warmer or a variation in water temperature may alter these physiological responses, providing either additional benefits or detriment to any possible recovery enhancement.

Cold temperature effects

Thermo-neutrality is considered to occur in a small range (35°C) in which subjects can maintain their core temperature for at least one hour (Craig & Dvorak, 1968). Critical cold temperatures at which an individual cannot maintain core temperature for an hour range from 30 – 34°C depending on the cutaneous fat of the individual (Toner *et al.*, 1986). However, core temperatures can be maintained during chin-out

immersion at temperatures as low as 18°C for up to 30 minutes (Tikuisis *et al.*, 2000; Toner *et al.*, 1986).

Cooler temperatures do have some effect on the physiological responses of the body. As water temperature decreases so does heart rate (Sramek *et al.*, 2000; Weston *et al.*, 1987), which results in decreased cardiac output (Bonde-Petersen *et al.*, 1992; Park *et al.*, 1999; Sramek *et al.*, 2000). Additionally, arterial blood pressure and peripheral resistance increase (Bonde-Petersen *et al.*, 1992; Park *et al.*, 1999; Sramek *et al.*, 2000). The increase in peripheral resistance is due to blood being redirected from the periphery to maintain core temperature (Bonde-Petersen *et al.*, 1992; Knight & Londeree, 1980). Oxygen consumption and metabolism also increase to maintain core temperatures (Lee *et al.*, 1997; Park *et al.*, 1999; Sramek *et al.*, 2000).

Reduced permeability of cellular, lymphatic and capillary vessels due to localised vasoconstriction reduces fluid diffusion into the interstitial space (Enwemeka *et al.*, 2001; Eston & Peters, 1999). This reduced fluid diffusion can assist in reducing acute inflammation from muscle damage (Cote *et al.*, 1988). This in turn can reduce pain, swelling and the loss of force generation that is also associated with inflammation (Smith, 1990). Hence, cold is often used in the treatment of inflammation to improve the rehabilitation process (Cote *et al.*, 1988).

One metabolite that is used as a marker of muscle damage is the level of creatine kinase in the blood (Rawson *et al.*, 2001). Exercise induced injury is thought to increase the permeability of cells increasing the diffusion of myoproteins such as creatine kinase into the extracellular space (Enwemeka *et al.*, 2001; Eston & Peters, 1999; Warren *et al.*, 1999). Cold water immersion decreases the level of creatine kinase in the blood after exercise induced muscle damage (Eston & Peters, 1999; Howatson & Van Someren, 2003). Lower creatine levels are attributed to a decrease in cellular, lymphatic, and capillary permeability caused by vasoconstriction induced by the cooler temperature (Enwemeka *et al.*, 2001; Eston & Peters, 1999). However, caution is warranted when applying the presence of creatine kinase in the blood as an indication of muscle damage. Levels of creatine kinase in the blood reflect not only creatine kinase release rate but also the removal rate. Exercise-induced haemoconcentration or haemodilution and alterations of tissue clearance due to blood-

flow or function will affect creatine concentration in the blood. Creatine kinase may not then accurately indicate muscle damage or fatigue (Warren et al., 1999).

Neural components are also affected by the cold. Cooling of tissue decreases the rate of transmission along neurons by decreasing the production of acetylcholine (Abramson et al., 1966) and possibly stimulates superficial inhibitory cells that regulate the impulse of pain perception to the central nervous system (Sauls, 1999). Reduction of nerve impulse transmission by cold has two effects, reduced level of pain perception (analgesia), and reduction in muscle spasm (Sauls, 1999; Washington *et al.*, 2000). While reduction in pain may be of benefit, a reduced neural transmission may decrease muscular contractile speed (Abramson et al., 1966; Howard et al., 1994; Rutkove, 2001) and force generating ability of an athlete post-application (Johnson & Leider, 1977; Rutkove, 2001; Yona, 1997). Performance may then be initially inhibited if exercise is performed shortly after cold immersion.

There are risks to athletes whom may be immersed in cold water, dependent on the temperature extreme and amount of the body immersed. Generally, sudden *severe* cold immersion of a large portion of the body can produce hyperventilation, which may cause ventilation to increase up to five times the resting rate (Wittmers & Savage, 2001). The decrease in arterial carbon dioxide caused by hyperventilation may lead to blood acidosis and impaired consciousness, even in fit young people (Lloyd, 1994; Wittmers & Savage, 2001). Additionally, sudden cold immersion can cause tachycardia, and acute peripheral vasoconstriction producing sudden loss of consciousness, convulsions, ventricular ectopy, cardiac arrest and death (Lloyd, 1994; Wittmers & Savage, 2001).

While rare some people also have cold hypersensitivity and can be at risk if body parts are suddenly immersed in cold water. Conditions consist of allergic and possible anaphylactic reactions, Raynauld's phenomenon, and paroxysmal cold hemoglobinuria (Lloyd, 1994; Terrell *et al.*, 1996). Allergic reactions can consist of rashes, and wheals, which may advance into anaphylaxis. The signs and symptoms of full-blown anaphylaxis include hypotension, syncope, and vascular collapse and can lead to death (Lloyd, 1994; Terrell et al., 1996). Raynauld's phenomenon is peripheral vasoconstriction that leads to numbness, tingling, and burning pain (Paz & West,

2002), while paroxysmal cold hemoglobinuria is a rare and potentially life-threatening affliction that causes the release of haemoglobin from red blood cell into the urinary system causing acute transient anemia (Lippman *et al.*, 1987).

While cold hypersensitivity is rare, care should be taken when using cold immersion on athletes. Very cold water temperatures may be best only in a localised manner to treat acute injuries and reduce inflammation, rather than being used on a large portion of the body during recovery strategies.

Hot water temperatures

Considering the use of thermotherapies such as hot baths in physiotherapy there is a lack of research-based literature on the effect that superficial heat application has on a person. Apart from basic physiological responses much of the literature comes from texts which cite other texts or is based on anecdotal information.

Superficial application of heat increases subcutaneous and cutaneous tissue temperature while tissue temperature at depths over two centimetres remains unchanged (Myrer *et al.*, 1997). An increase in superficial tissue temperature causes an increase in the cutaneous blood flow, over short durations, due to peripheral vasodilation (Bonde-Petersen *et al.*, 1992; Knight & Londeree, 1980).

In response to hot water immersion, heart rate increases (Bonde-Petersen *et al.*, 1992; Weston *et al.*, 1987). This increase in heart rate may reduce stroke volume due to lack of cardiac filling time, but overall cardiac output increases compared to thermo-neutral immersion (Weston *et al.*, 1987). The increase in cardiac output and a lower peripheral resistance allows an increase in subcutaneous and cutaneous blood flow (Bonde-Petersen *et al.*, 1992; Weston *et al.*, 1987; Whitney & Wickline, 2003). An increase in subcutaneous and cutaneous blood flow increases the permeability of cellular, lymphatic and capillary vessels (V. J. Robertson & Duck, 2001). Increased permeability increases metabolism, nutrient delivery and waste removal from the cells which can increase healing (Cote *et al.*, 1988; Michlovitz, 1996; Starkie *et al.*, 1999). The increase in metabolism due to heat application also erodes muscle glycogen

stores quicker (Starkie et al., 1999). With short-duration superficial heat application any physiological changes are not likely to occur within the muscle but rather within the skin (Bonde-Petersen et al., 1992; Myrer et al., 1997; Wyper & McNiven, 1976). Bonde-Petersen et al. (1992) observed that while subcutaneous and cutaneous blood flow increased, blood flow through the muscle may decrease with hot water immersion compared to thermoneutral immersion. Thermo-neutral water temperatures may then have greater benefits in substrate transportation within a muscle.

Superficial heat may also increase neural transmission (Cotts *et al.*, 2004), proprioception and improve reaction time (Burke et al., 2001). Other proposed benefits of thermotherapy include increased muscle elasticity, joint extensibility, analgesia and reduction of muscle spasm (Brukner & Khan, 2001; Coffey et al., 2004; Kaul & Herring, 1994; Michlovitz, 1996; Tonnessen, 1999; Wilk *et al.*, 2004). While a large amount of anecdotal support is available little research-based evidence has been found to support these claims. Of studies that have analysed the effect of the superficial application of heat, flexibility was not enhanced unless accompanied with stretching (Henricson *et al.*, 1984; Prentice, 1982; Sawyer *et al.*, 2003; Taylor *et al.*, 1995). A report by Bigos et al. (1994) on back pain and thermal applications concluded that not enough data existed to recommend the use of heat in pain reduction. More recent research (Nadler et al., 2003a; Nadler et al., 2002; Nadler et al., 2003b) had observed that pain may be reduced if heat is applied continuously (8 hours a day) over a long-term (2 – 5 days). However, research is lacking regarding the short-term application of heat and subsequent effects on pain.

There are contraindications to immersion in hot water, the most obvious being the possibility of burns due to high water temperatures. At 45 - 50°C protein denaturation occurs and immersion water temperatures should be below this range (Michlovitz, 1996). Superficial heat application also causes an inflammatory response and swelling (Barnes, 1979; Cote et al., 1988; Magness *et al.*, 1970; Wallace *et al.*, 1979), which may prolong recovery time (Cote et al., 1988; Rutkove, 2001). Research by Cote et al. (1988) observed increased edema in patients (n = 30) with first and second degree ankle sprains when 20 min of hot water immersion was applied each day over three days. Volumetric increase in ankle size was 25% with hot water immersion of the foot compared to a 3% in patients receiving cold water immersion. The effect size of this

difference was large ($ES = 1.95$). If heat increases edema in sprains, it can be speculated that it may also increase muscle inflammation (Feibel & Fast, 1976).

Hot water immersion of a large portion of the body can produce a potentially dangerous strain on the cardiovascular system causing ectopic beats, hypotension, heat syncope, excessive tachycardia and even death (Nagasawa *et al.*, 2001; Turner *et al.*, 1980). Heat syncope is fainting and giddiness due to the collapse of vasomotor control and a decrease in blood pressure owing to rapid vasodilation (Greenleaf & Kaciuba-Uscilko, 1989). Care is warranted with athletes that have acute injuries, edema, vascular disease, wounds or infections as these can be exacerbated with heat application and increase potential risks (Michlovitz, 1996).

Contrasting temperature

Contrast therapy has been considered to enhance athletic recovery through stimulating area specific blood flow, increasing blood lactate removal, reducing inflammation and edema, stimulating circulation, relieving stiffness and pain, increasing range of motion, and reducing delayed onset of muscle soreness (Cochrane, 2004; Coffey *et al.*, 2004; Myrer *et al.*, 1994; Sanders, 1996). One reason behind such possible benefits to recovery is that contrast therapy may mimic one of the mechanisms attributed to active recovery without the same energy demands (Calder, 2003; Cochrane, 2004; Stanton *et al.*, 2003; Vaile, 2003). That is, recovery using active low-intensity exercise is considered to enhance recovery compared to passive modalities (Connolly *et al.*, 2003; Signorile *et al.*, 1993; Thiriet *et al.*, 1993) by the alternating muscular contractions acting in a pumping/squeezing action. Low intensity repetitive mechanical “squeezing” by the muscles during contraction-relaxation may increase the translocation and removal of metabolites such as lactate and reduce intracellular fluid volume (Signorile *et al.*, 1993). Much of the research and literature regarding contrast therapy perpetrate the theory of contrast therapy causing a similar action with vaso-pumping. Alternating vasoconstriction and vasodilation, is thought to act in a comparable way to muscle pumping, increasing blood flow and metabolite removal, enhancing recovery (Cochrane, 2004; Stanton *et al.*, 2003; Vaile, 2003). However,

vaso-pumping would seem unlikely to occur at a level that could act effectively in this manner.

During contrast therapy each alternation of temperature generally lasts for 30 to 120 seconds and is repeated two to five times. Vaso-pumping would then occur at a slow rate and only with two to five ‘pumps’ over a period of around two to 10 minutes (at a low frequency such as 0.03 - 0.008 Hz). Under active recovery, such as light-running, muscular pumping would occur at a rate of around 2Hz. If vaso-pumping does occur during contrast therapy, it would seem unlikely to cause a great effect at such a slow frequency.

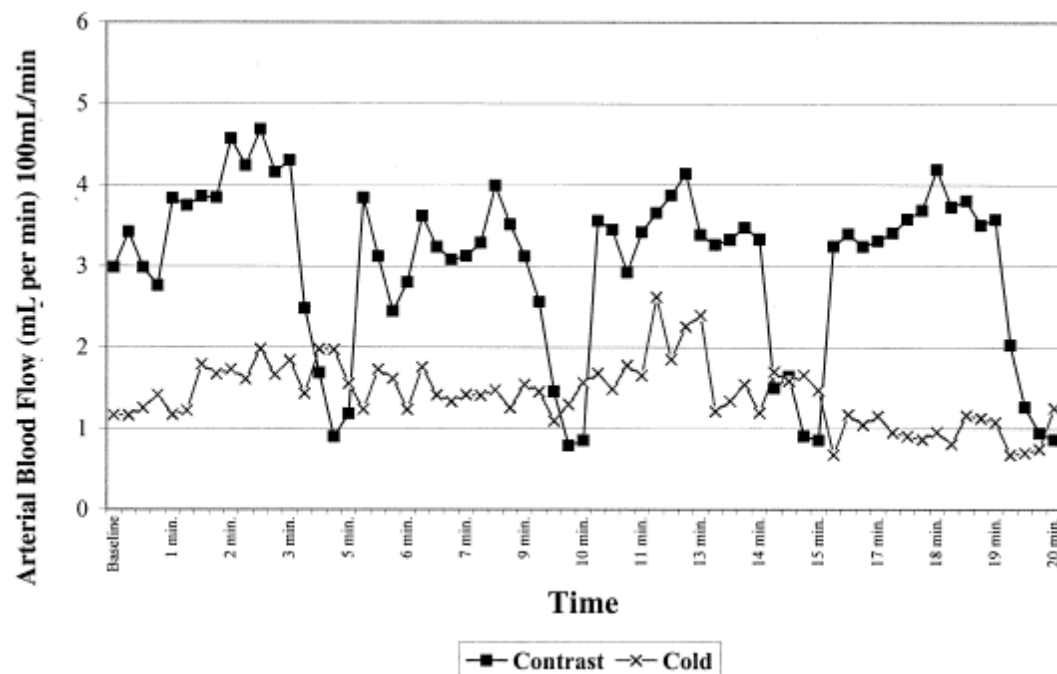


Figure 3: Change in blood flow between contrast and cold water immersion.

From “Changes in lower-leg blood flow during warm-, cold-, and contrast-water therapy” by Fiscus, Kaminski, and Powers, 2005, *Archives of Physical Medical Rehabilitation*, 86, p. 1407.

A recent paper by Fiscus, Kaminski, and Powers (2005) analysed blood flow in the lower leg during warm-, cold- and contrast-immersion over 20 minutes in 24 subjects. By means of occlusion plethysmography on the left thigh (3 min venous occlusion: 1 min rest) the volume change of the calf was measured using a mercury-in-rubber strain gauge. Cold immersion did not produce a difference to blood flow compared to the control, warm immersion (40°C) produced a $304 \pm 31\%$ increase in blood flow,

and contrast immersion produced an overall $209 \pm 116\%$ increase in blood flow. During contrast immersion the alternating (4 min hot: 1 min cold) change in immersion temperature was reflected by a change in blood flow (see Figure 3). Results from Fiscus et al. lend support to the theory that contrast therapy may cause alternating vasodilation-constriction. However, Fiscus et al. measured total blood flow to the lower leg and did not isolate subcutaneous and intramuscular blood flow.

A point to consider with vaso-pumping is that intramuscular temperature has not been observed to change with alternating contrasts, only subcutaneous temperature (Myrer et al., 1994; Myrer et al., 1997). A study by Higgins and Kaminski (1998) observed that at a four cm depth intramuscular temperature did not fluctuate with repeated one minute immersions into cold water (after four minutes of warm whirlpool therapy), but gradually increased by $0.85 \pm 0.60^{\circ}\text{C}$ over 31 minutes. If temperature does not change at deep tissue levels with alternating immersion any vaso-pumping would then be likely to occur at a subcutaneous level only. To aide recovery and intramuscular waste removal by vaso-pumping temperature changes would surely be required at a deeper tissue level. Additional to the unlikely vaso-pumping within deep tissue the sudden immersion into an icy bath from heat may not cause vasoconstriction. During high body temperatures, which may occur after intense athletic exertion and hot water immersion, the sudden immersion into cold may cause cutaneous vasodilation rather than vasoconstriction in a shock response (Bonde-Petersen et al., 1992; Kauppinen, 1989).

If vaso-pumping does not occur another explanation for elevated blood lactate removal during contrast therapy must exist. One area that is little discussed in contrast therapy literature that may explain increased removal of wastes from the body is simply the hydrostatic pressure caused by immersion in water.

Similar to heat application, contrast therapy may be harmful to athletes by causing inflammation. Compared to cold water immersion Cote et al. (1988) observed a 26.5% increase in edema using contrast therapy on patients with first and second degree ankle sprains. The effect size of this difference was large ($ES = 2.05$). However, Vaile, Blazeovich and Gill (2004) observed that 15 minutes of contrast

therapy after muscle damaging eccentric leg presses reduced thigh volume edema significantly over 72 hours compared to passive recovery. Mean (\pm SD) thigh measurements increased by $2.3 \pm 0.8\%$ with passive recovery compared to $0.6 \pm 0.6\%$ in the contrast therapy group. The reason for the benefit observed in Vaile et al.'s study may be due to the large portion of the body that was immersed (immersion to the gluteal fold), rather than the effect of temperature. A greater physiological response would have occurred due to the higher hydrostatic pressures compared to Cote's study where only the foot was immersed. Alternatively, in Cote's study subjects had acute injuries rather than induced muscle damage which may account for the possible effect difference of the contrast therapy. More research is required before any conclusion can be drawn on whether contrast therapy increases or decreases edema. Other contraindications of contrast therapy are likely to include those of both the hot and cold temperatures discussed previously (Michlovitz, 1996; Turner et al., 1980). Care is warranted when using extremes of temperature in contrast immersion.

Summary

Hydrostatic pressure from water immersion causes an inward and upward displacement of body fluid. This action reduces edema, increases extracellular fluid transfer into the vascular system and increases cardiac output. Greater cardiac output increases blood flow through the body and in response to increased arterial pressure vasodilation may occur. Increased blood flow through the body may assist in the metabolism of waste products that accumulate during exercise by reducing transport time. Additionally, reductions in edema due to fluid shifts may assist short-term in maintaining muscle function and assist muscular repair. Weightlessness when immersed in water decreases the perception of fatigue and may be due to reduced neuromuscular signal magnitudes.

Decreasing water temperature may reduce some of the physiological responses associated with hydrostatic changes. The body responds to cold by reducing heart rate and cardiac output, and inducing vasoconstriction. This response reduces peripheral blood flow and conserves body core temperatures. Additionally, central metabolism increases to maintain the core temperature, which increases the production of waste

products and erodes energy stores. However, cold temperature also assists the reduction of edema by increasing vasoconstriction and lowering peripheral metabolism, which may reduce secondary cellular death due to muscular damage. The analgesic effect of cold is likely due to reduced neural transmission magnitude and speed. Heat and contrast therapy are also unlikely to provide any additional benefits to the recovery process compared to thermoneutral immersion. Hot immersion increases edema, and like cold immersion causes an increase in the metabolic rate, eroding energy stores. Immersion in cool to thermoneutral water may provide the best option for recovery unless muscle sprains or strains have occurred, in which case a localised immersion in cold water may provide greater benefit. Extremes of water temperature have contraindications and cool to thermoneutral immersion may provide both a safer and more beneficial immersion strategy.

Physiologically speaking, hydrostatic pressure would seem the mechanism which could benefit exercise recovery. Ultimately, the aim of the recovery process is to enhance future performance. To date a small amount of research has been conducted into the use of water immersion as an exercise recovery strategy. More research incorporating performance measures and water immersion needs to be conducted to determine if water immersion post-exercise causes any worth-while performance benefit.

CHAPTER 3:

THE EFFECT OF PASSIVE RECOVERY, ACTIVE RECOVERY AND WATER IMMERSION ON PERFORMANCE FOR TOURNAMENT-STYLE EVENTS

Introduction

In a tournament-style event an athlete may play a number of games over two or three days, with more than one game each day. Over the course of such an event there may be inadequate time for an athlete to recover to their optimal physiological status before the next bout of exercise. Enhancing the recovery process in such situations could provide a competitive advantage to an athlete. Athletes, trainers and coaches use many different methods to improve recovery and subsequent performance following exercise. These range from rest and sleep, to nutritional strategies such as supplementation, to physical modalities such as massage, active recovery, and stretching (Calder, 2003). Another method that is gaining popularity as a means to improve recovery, is immersion in water (Calder, 2000; Cochrane, 2004). Numerous books on sport training also advocate the use of active recovery, and to a lesser extent, water immersion, as a method to enhance recovery. There appears to be limited evidence to substantiate the claim that active recovery or water immersion improves performance recovery. It is surprising that considering the importance of recovery, a relatively small amount of research has been conducted into the effect of active recovery and water immersion on performance. Of the studies that have been conducted the protocols used have varied considerably or have concentrated on the ability of different recovery methods to reduce blood lactate accumulation rather than maintaining performance. This review will evaluate the effect of three different recovery methods (passive, active and water immersion) on different types of performance and relate this evaluation to a tournament-style situation.

For study comparisons data has been converted into percentage differences between pre- and post-recovery performance measures and recovery modes. While most papers report recovery effects in relation to statistical significance many sport science articles are underpowered in terms of subject numbers and may report non-significance incorrectly as no effect (Hopkins, 1999). Effects are therefore additionally reported as Cohen effect sizes (ES) using Hopkins's (2002b) scale of magnitude (trivial, 0 – 0.2; small, 0.2 – 0.6; moderate, 0.6 – 1.2; large, 1.2 – 2.0; very large 2.0 – 4.0; near perfect, >4.0).

Definition of recovery techniques

Passive recovery

Passive recovery refers to inactivity post-exercise and the intrinsic return of the body to homeostasis (Sanders, 1996). The most basic form of such inactivity is sleep. However, for the purpose of this review and to replicate what is performed in the field, rather than sleep, passive recovery refers to an athlete doing nothing out of the ordinary after a game or training. Therefore, passive recovery is often seated inactivity (Lattier et al., 2004; Monedero & Donne, 2000; Sanders, 1996; Thiriet et al., 1993; Vaile et al., 2004; Weltman et al., 1979; Weltman et al., 1977), or if over a longer period of time (days), natural daily activity (Kuligowski et al., 1998; Viitasalo et al., 1995). Studies that have compared the effects of different recovery modes generally have used passive recovery (seated) as a control group. Because research into both active recovery and water immersion effects are compared to passive recovery, passive recovery is not discussed separately in the following sections.

Active recovery

In the field it is common for coaches, trainers and athletes to use active recovery as a method of improving recovery and maintain/improve performance (Best & Garret, 1993; Calder, 2003). Active recovery is a post-game or post-training exercise at an intensity that is lower than the game or training. Active cool-down is another name for active recovery. The intensity and mode of active recovery varies, but in research the

intensity is normally less than the anaerobic threshold ($< 65\% \dot{V}O_{2\max}$) and using a similar mode to the exercise that was performed (Lattier et al., 2004; Monedero & Donne, 2000; Sanders, 1996; Thiriet et al., 1993; Vaile et al., 2004; Weltman et al., 1979; Weltman et al., 1977). The duration of active recovery in research is normally between four and 20 minutes (Lattier et al., 2004; Monedero & Donne, 2000; Sanders, 1996; Thiriet et al., 1993; Vaile et al., 2004; Weltman et al., 1979; Weltman et al., 1977), which is approximately the duration recommended to athletes in the field.

Increased blood flow during light-intensity exercise has been postulated as the mechanism that improves recovery. Signorile et al. (1993) suggested that the pumping (contraction-relaxation) action of active muscles may increase the clearance of metabolic waste. By increasing blood flow the removal of metabolites, such as lactate, and the replenishment of substrates within the muscles could be enhanced (Bonen & Belcasro, 1977). Over the short-term active recovery may increase the contractile ability of the muscles and over the longer-term aide healing (McEniery *et al.*, 1997; Sayers et al., 2000). However, while low intensity activity may improve recovery higher-intensity active recovery may be contraindicative to recovery by increasing metabolic waste and depleting nutrient stores (McEniery et al., 1997).

Water immersion

Also gaining popularity is the use of water immersion (in a pool or water bath) as a method of recovery (Calder, 2000; Cochrane, 2004). Four basic modes of water immersion can be performed, a) cold immersion, b) hot immersion, c) alternating temperature immersion (contrast therapy) or, d) water immersion per se in a pool or bath. The difference between these four types of application is the immersion temperature. This variation in temperature poses a major problem in the current research of immersion and performance recovery. In most performance-immersion research papers it is not know whether any possible benefit to recovery result from hydrostatic pressure or water temperature. Due to the scarcity of water immersion and performance research, these different modes of water immersion will be discussed collectively in this review. Therefore the temperature of water used in the immersion studies could have some influence on findings.

The duration time of water immersion appears to vary among sporting texts (books) and teams that practice immersion recovery. Netball New Zealand (2004) have proposed an immersion time of five minutes to aide recovery, though the protocols used by elite netball teams are unstandardised and ad hoc (Bonham et al., n.d). Water immersion and exercise recovery research have used immersion times that range from six to 20 minutes (Coffey et al., 2004; Hamlin & Magson, 2002; Hamlin & Sheen, 2004; Sanders, 1996; Vaile et al., 2004).

To date, water immersion is thought to provide benefits similar to active recovery, such as increased blood flow and lactate removal, without the need to expend the extra energy associated with active recovery (Blyden et al., 1989; Hamlin & Magson, 2002; Nakamura et al., 1996; Sanders, 1996). Whether lactate is a component of fatigue is debateable (Cairns, 2005), but the increase in blood flow throughout the body could reduce the delivery and removal transport time of substrates within the body, aiding recovery.

Recovery and performance

Strength and power are considered to be a critical component of many athletic tasks (Abernethy *et al.*, 1995). Additionally, in most sports it is more important to produce repeated expressions of power rather than a single expression of maximum force or power (Newton *et al.*, 1996). For example, the ability to jump repeatedly in sports such as netball or volleyball, or repeatedly produce explosive bouts of running in games such as rugby or hockey. Hence, cyclic expressions of power can be critical to successful performance. Performance research using passive and active recovery and water immersion have varied in protocol and measurements. For the purpose of this review, the effect of recovery on strength and three cyclic expressions of power (cycling, running and jumping) provide the focus for discussion.

Strength

In strength recovery research the use of isometric and to a lesser degree isokinetic testing is common (Abernethy et al., 1995). However, these forms of testing bear little resemblance to sporting movements and have been observed to have low correlations to other performance measures such as sprints and jumps (Considine & Sullivan, 1973; Guy *et al.*, 1996; Mero *et al.*, 1981; Murphy *et al.*, 1994; Piatt *et al.*, 1999). The reader needs to be aware of this limitation when reading the following section.

Active recovery

The effect of active recovery on strength decrement due to fatigue has not been well researched. Four studies were found that have compared the effect of active and passive recovery on changes in maximal strength (see Table 4). Two of these studies (Lattier et al., 2004; Sayers et al., 2000) compared changes in isometric strength, while the other two studies compared changes in dynamic strength (Bond *et al.*, 1991; McEniery et al., 1997).

Lattier et al. (2004) investigated the effect of active recovery after exercise on the force produced during maximal voluntary isometric (MVIC) leg extensions. To induce fatigue subjects repeatedly ran on a treadmill for one minute at 120% of their maximal aerobic velocity (MAV) at an 18% gradient. Ten repetitions of the treadmill run were performed with a two-minute rest period between each run. This fatiguing protocol was generally maximal with subjects only able to complete on average (\pm SD) 7.0 ± 2.5 repetitions at 120%MAV with the remainder completed at a speed $0.5 \text{ km}\cdot\text{h}^{-1}$ slower. After the ten uphill runs subjects performed 20 minutes of either seated passive recovery or jogged at 50%MAV at a 3% gradient.

Pre-exercise, 45- and 65-minutes post-exercise the subjects' isometric leg extension strength was assessed using a force transducer. Maximal force production was significantly lower post-exercise but did not significantly differ between active and passive recovery modes. Post-recovery performance differences between the recovery modes were less than 2% with small magnitudes of effect ($ES < 0.33$). Lattier and colleagues concluded that active recovery did not enhance recovery, however, one

factor that may have influenced the results of this study was the intensity of the active recovery protocol. Active recovery was performed at 50%MAV_{3%}, which equated to a relatively fast mean velocity of $8.9 \pm 0.7 \text{ km}\cdot\text{h}^{-1}$. Lighter-intensity active recovery could possibly provide greater benefit to recovery performance (Corder *et al.*, 2000; McEniery *et al.*, 1997).

Table 4: Effect of active and passive recovery on maximal strength

Author	Subjects	Study design	Exercise	Recovery modes	Outcome measures	Main findings
Lattier <i>et al.</i> (2004)	8 males regional - national level athletes	X-over	10 maximal treadmill sprints	a) Passive b) Active jogging (both for 20 min)	Leg MVIC	MVIC ↓ for both recovery modes, no sig. difference between modes (ES < 0.33).
McEniery <i>et al.</i> (1997)	4 active males, 1 active female	X-over	4 x 30 s cycle sprints	a) Passive b) 30%VO _{2max} cycle c) 60%VO _{2max} cycle (all for 15 min)	Dynamometer peak leg torque	↑ recovery of peak torque following 30%VO _{2max} cycle (ES > 2.02).
Bond <i>et al.</i> (1991)	5 active males	X-over	Maximal 60 s cycling	a) Control (no cycle) b) Passive c) Active cycling (both for 20 min)	a) Peak torque b) Total work (Dynamometer) c) BLA	No sig. difference in kinetics between recovery modes. Conflicting moderate effect sizes between passive and active recovery, and peak power (0.71) and work (-0.75). Quicker ↓ BLA with active recovery.
Sayer <i>et al.</i> (2000)	27 non-weight trained men	RCT	50 Supra-maximal eccentric bicep curls	a) Passive control b) Immobilisation c) Daily, low intensity biceps curls	a) MVIC b) Soreness c) Range of motion	Strength recovery ↑ with active recovery (ES = 0.22) and immobilisation (ES = 0.34)

X-over = randomised crossover trial; RCT = random control trial; MVIC = maximal voluntary isometric contraction; BLA = blood lactate; ↑ = significant increase ($p < 0.05$); ↓ = significant decrease ($p < 0.05$); no sig = non significant; ES = Cohen's effect size.

McEniery *et al.* (1997) observed that the recovery of post-exercise leg extension peak torque was dependent on the intensity of the active recovery protocol. Strength recovery benefits were found only with low-intensity active recovery ($p < 0.01$). Subjects in McEniery *et al.*'s study performed four 30-second maximal cycle sprints with 240 seconds of passive rest between. For 15 minutes following the sprints subjects either sat passively, cycled at 30% $\dot{V}\text{O}_2\text{max}$ or at 60% $\dot{V}\text{O}_2\text{max}$. Peak torque was assessed pre, 1-, 6-, and 11-minutes during recovery and immediately post-recovery using a dynamometer (at an angular velocity of $60^\circ\cdot\text{s}^{-1}$). Peak torques after

16 minutes of either passive recovery or cycling at 60% $\dot{V}O_2$ max reduced to $85.2 \pm 3.6\%$ and $85.7 \pm 0.8\%$ respectively of pre-exercise peak torques. After light active recovery (30% $\dot{V}O_2$ max) a significant recovery of peak torque ($92.1 \pm 3.1\%$ of baseline) occurred. This smaller strength decrement amounted to a very large positive effect (ES = 2.02) when compared to passive recovery and a near perfect positive effect (ES = 7.00) when compared to higher-intensity active recovery. However, one major limitation of McEniery et al.'s study was that only five subjects took part, which would have reduced the statistical power of the study.

A study by Bond et al. (1991) used similar methods to McEniery et al. (1997) but observed no significant benefit compared to passive recovery to peak torque or work with a 30% $\dot{V}O_2$ max cycle active recovery. Like McEniery et al. (1997) the study had a low number of subjects ($n = 5$), which would again affect statistical power. From the data available active recovery produced a non-significant moderate detrimental effect to peak torque recovery (ES = -0.71), but a moderate benefit to total work (ES = 0.75) when compared to passive recovery.

There were some protocol differences between the studies by Bond et al. (1991) and McEniery et al. (1997), which may account for the difference in the results. When McEniery et al. assessed maximal isokinetic strength the peak torque of three leg extensions was used. In Bond et al. (1991), subjects performed 60 leg extensions over 1.5 minutes, with peak torque being the highest value over the first five repetitions and work the total of all 60 repetitions. With such a large number of contractions subjects may have initially regulated their force outputs to limit exhaustion. Additionally, McEniery et al. used an angular velocity of 60°s^{-1} , whereas Bond et al. used an angular velocity two and a half times as fast (150°s^{-1}). The higher speed used by Bond et al. may have reduced the subjects' ability to develop similar force outputs during each weekly testing session, decreasing peak torques and increasing mean variation (Bardis *et al.*, 2004). A major limitation of Bond et al.'s study was that no pre-exercise isokinetic assessment of the leg extensor torque was taken during each testing session. Even though a control group was included in the study (no exercise performed) the values presented may not reflect a true effect as peak torques and work values prior to exercise may have varied each week. The amount of variation and pre-

post change in torque cannot therefore be determined from the data provided by this research.

Sayers, Clarkson and Lee (2000) approached the study of active recovery and strength differently. In this study the long-term recovery of subjects' ($n = 26$) MVIC from supra-maximal eccentric arm flexion training was monitored. Recovery strategies included daily active recovery (50 curls with a 2.27 kg dumbbell), passive recovery, or arm immobilisation in a cast for four days post-exercise. MVIC of the arm was assessed daily over the following 12 days. Over this time subjects who used a daily routine of low intensity arm curls had significantly improved strength recovery (88% of their baseline by day 12). This amounted to a small beneficial effect ($ES = 0.22$) compared to passive recovery (82% strength recovery compared to baseline by day 12). Total immobilisation of the arm also significantly improved MVIC recovery (90% of their baseline by day 12) to a greater extent than active recovery ($ES = 0.34$). However, no significant difference in MVIC between the three recovery modes was observed until eight days post-exercise.

The benefit that both total immobilisation and low-intensity exercise may provide to maximal strength recovery could be greater than what Sayer et al. observed. Recovery of MVIC of the control group in Sayer et al.'s study was greater than what has been reported in other studies that have used a similar protocol to induce eccentric exercise muscle damage (Chleboun *et al.*, 1998; Nosaka & Clarkson, 1994). The mechanism that enhanced recovery during total immobilisation is unknown. Sayer et al. speculated that over four days of inactivity slow twitch muscle fibres may have reverted to a faster twitch myosin heavy chain isoform allowing greater force production during MVIC assessment. Or total immobilisation could allow greater healing of the muscle fibres since any remobilisation may cause damage to a healing fibre. From a practical perspective, it would be highly unlikely that total immobilisation of an athlete could be accomplished over a long period of time. No within-day effects were assessed by Sayers et al., so whether the long-term benefit of active recovery or total immobilisation of an athlete can be applied to a tournament style situation is unknown.

Water immersion

Little research has been conducted on water immersion as a means of strength recovery. Similar to active recovery, researchers studying post-exercise water immersion have observed conflicting effects of immersion on strength recovery (see Table 5).

Table 5: Effect of water immersion and passive recovery effect on maximal strength

Author	Subjects	Study design	Exercise	Recovery modes	Outcome measures	Main findings
Clarke (1963)	30 male subjects	X-over	2 x 2 min maximal hand grip	a) Passive b) Hot water immersion c) Cold water immersion (all for 10 min)	Hand MVIC	No sig. performance change.
Viitasalo et al. (1995)	6 female and 8 male national track and field athletes	X-over	Five strength-power sessions in 3 days	a) None b) Warm-water immersion (after evening trainings) (20 min)	a) Leg extension MVIC b) VAS-score	No sig. MVIC benefit using water immersion (ES = -0.12 to 0.67)
Burke et al. (2003)	21 female 24 male	RCT	5 days of isometric strength training	a) control b) Hot water c) Cold water (all for 10 min)	Hip extension MVIC	All groups ↑ strength. Cold group had a MVIC ↑ that was twice that of the control (ES = 0.7) and hot group (ES = 0.7).
Vaile et al. (2004)	9 female and 4 male recreational athletes	X-over	DOMS inducing leg press	a) Passive b) Contrast therapy (both for 15 min)	a) Squat MVIC b) Thigh volume	Greater reduction in MVIC with passive recovery (ES > 0.33) compared to contrast therapy. Immersion thigh volume increased less with contrast therapy

X-over = randomised crossover trial; RCT = random control trial; MVIC = maximal voluntary isometric contraction; ↑ = significant increase ($p < 0.05$); ↓ = significant decrease ($p < 0.05$); no sig = non significant.

In 1963 Clarke conducted a study on the effect of water immersion and temperature on hand-grip strength recovery. In this study 30 male students maximally gripped a hand dynamometer for two minutes. For 10 minutes following the exercise the subjects recovered by immersing their hand in either hot (46°C) or cold (10°C) water, or were a control (no immersion). Every 60 seconds during the recovery period subjects removed their hand from the water (if immersed) and were tested for

maximal grip strength. Strength, which had initially decreased by 75%, returned in a similar pattern (no significant differences) for the three recovery modes.

While Clarke (1963) may have observed no improvements with immersion, two mechanisms that may improve recovery would have been minimised. Fluid shifts and increased cardiac output may enhance the ability to transport and metabolise waste products. During Clarke's (1963) experiment, only the hand of the subjects was immersed, which would cause small physiological effects. It might be expected that if larger segments of body mass were submerged the effects of fluid shifts and cardiac output would be greater.

Larger segmental immersion has been used in other studies (Burke et al., 2003; Vaile et al., 2004; Viitasalo et al., 1995). Viitasalo et al. (1995) conducted a randomised crossover study on 14 national track and field athletes. Over three days subjects had five training sessions consisting of strength training, plyometric drills and sprinting drills. All sessions were standardised for volume and intensity. The subjects were randomly put into two groups. After the evening training sessions one group was immersed in warm-water (37°C) for 20 minutes, while the other group were a control. Two weeks later subjects underwent the same training routine followed by the opposite recovery mode. During the exercise weeks the social and recreational structure of the subjects was made as similar as possible, and in-between the experimental week's normal training was conducted. Leg extension MVIC was assessed pre-training and on three occasions over the two days following the training days. No significant differences between the immersion recovery strategy and the non-immersion treatment were reported in terms of isometric strength. While non-significant a moderate positive effect size ($ES = 0.65$) during the water immersion week occurred in the first post-exercise testing session. However, other measures taken 3 hours later on the same day and on the following day showed trivial detrimental effects ($ES = -0.12$ and -0.08).

Viitasalo et al.'s study used a subject group of elite athletes and they trained in their normal environment with event-related drills and exercises. However, large standard deviations (23 - 25% of the mean) were reported in the MVIC measurements, which

may have produced the non-significant results and may account for the varied effect sizes. Additionally, the testing protocol consisted of MVIC of the leg extensors using a dynamometer, rather than a compound movement involving the hamstrings, quadriceps and gluteal muscle groups. This isolation of the leg extensors may reduce the study's ability to detect fatigue if the fatigued state of each muscle group varies dependent on the individual athlete and the event-related training drills that they performed.

A similar study to Viitasalo et al. (1995) was conducted by Burke et al. (2000) in respect to multiple exercise sessions and water immersions over a number of days. Forty-five male and female subjects were randomly allocated to a control, hot immersion, or cold immersion group. Over five days subjects performed four, 8-second, repetitions of isometric contractions of the hip extensors at 60-, 70-, 80- and 100% MVIC. For 10 minutes following the exercise subjects were immersed in 8°C or 43°C water up to the gluteal fold, or rested. All groups significantly increased force production from the first day to the fifth day. Increased force production (58%) by the cold immersion group was more than twice ($p < 0.05$) the increase that occurred in the hot immersion (26%) and control group (27%). However, strength gains could be related to the experimental design of the study rather than due to the recovery benefits that may have resulted from immersion in cold water. Rather than have an absolute training load based on initial strength, intensity of muscular effort was based on the force produced during the 100%MVIC of the previous day's training. Apart from the initial strength assessment the daily MVIC produced by the cold group was higher than that of the hot or control group. This meant that while initially the lowest in strength, the cold group trained with a higher relative workload throughout the study than the hot or cold group.

Another problem with the Burke et al. (2000) study was that they investigated the daily training effects of immersion rather than within-day immersion recovery effects. Cold-water immersion has been observed to decrease muscle contraction velocity and hence power due to reduced neural activation (Abramson et al., 1966; Howard et al., 1994; Johnson & Leider, 1977; Rutkove, 2001) immediately post-immersion. The post-immersion time course of this effect has not been established. If cold water is

used as a recovery strategy during tournament-like competitions in normal conditions then the temperature effects may hinder performance, though it may enhance long-term training performance.

Another study that induced muscle fatigue in subjects was Vaile et al., (2004). This study attempted to induce muscle damage into the legs of subjects with supra-maximal eccentric leg presses then observe the effect passive recovery or contrast therapy had on the recovery of strength. Recovery was performed immediately post-exercise for 15 minutes, and then strength was assessed using a squat MVIC post-recovery and every 24 hours over a 72 hour period. The eccentric exercise induced both pain (DOMS) and edema in the legs for 72 hours post-exercise. A significant decrease ($15.0 \pm 11.9\%$) in peak isometric force was observed immediately following passive recovery. This decrease was $22.5 \pm 12.3\%$ at 48 hours returning at 72 hours to baseline. Peak isometric strength following water immersion was never significantly different to baseline levels. The effect size of the difference in strength recovery in the water immersion group compared to the passive group was moderate ($ES = 0.76 - 0.83$) over 48 hours and small ($ES = 0.33$) at 72 hours post-exercise. Unlike Viitasalo et al. (1995) Vaile used a compound isometric assessment (isometric squat) to determine strength, providing a more valid measure of leg fatigue. However, rather than use pure immersion Vaile's intervention consisted of contrast therapy. The effect that alternating temperature alone may have on the recovery of strength is not known. The exposure to 5 x 60 seconds of cold ($8 - 10^{\circ}\text{C}$) may have reduced the edema associated with DOMS and increased strength recovery (Cote et al., 1988). More research separating temperature variation from water immersion is required to determine which, if either, variable has some effect on post-exercise strength recovery.

Neither Vaile (2004), Viitasalo et al. (1995), Burke et al. (2003), or Clarke (1963) used active recovery in their water immersion studies for effect comparisons. From the studies that have been conducted it appears that light-intensity active recovery may improve maximal isometric strength recovery within a day. There is also a possibility that water immersion and active recovery may improve the recovery of maximal strength over a longer time period (days) if muscle damage has been

induced. However, a paucity research into active recovery and water immersion as a means to improve strength recovery provides little evidence to determine their validity or worth as a recovery strategy within a day or during a tournament-style event.

Cycling

Cycling offers one of the easiest methods to assess power with the Wingate cycle ergometer test being used a great deal in research. However, cycling consists of a concentric only application of force, whereas many sports consist of concentric-eccentric actions. The reader needs to be cognizant of this limitation when applying the findings of cycling research to other activities. One benefit of the research that has been conducted into active recovery following cycling is that some researchers have also used water immersion as a recovery intervention (see Table 6). This allows a comparison of the two recovery protocols.

Active recovery

Weltman et al. (1977) studied the effect of active and passive recovery after a one-minute supramaximal bout on a cycle ergometer (maximum intensity $33.0 \text{ kgm}\cdot\text{rev}^{-1}$). After 10 or 20 minutes of seated passive recovery or active cycling (at 60 W) another supramaximal bout of cycling was performed. Weltman et al. observed greater cumulative revolution scores (3.9%, $p < 0.01$) after 10 minutes of active recovery compared to 10 minutes of passive recovery. Following 20 minutes of active recovery no performance change from baseline was observed, whereas a 5.6 % ($p < 0.01$) decrease was observed after a passive 20-minute rest. While, Weltman et al. reported that the differences between active and passive recovery was significant, the effect sizes of these benefits were small for both 10 and 20 minute recoveries (ES = 0.25 and 0.25).

Monedero and Donne (2000) also observed some benefit from active recovery. Eighteen trained male cyclists were used to determine the possible effects of passive, active, massage and combined active-massage interventions on recovery. Subjects performed two maximal 5 km cycles on their own bikes on a Kingcycle with 20

Table 6: Effect of active recovery and water immersion on repeated bouts of cycling

Author	Subjects	Study design	Exercise	Recovery modes	Outcome measures	Main findings
Weltman et al. (1977)	11 male.	X-over	2 x Supra-maximal cycle	a) Passive (10 min) b) Passive (20 min) c) Active cycle (10 min) d) Active cycle (20 min)	a) Cycle rpm b) BLa	↑ post-recovery cycle revolutions with active recovery (ES = 0.25) and ↑ post recovery cycle revolutions for 20 min than 10 min recovery time. BLa ↓ with active recovery.
Monedero and Donne (2000)	18 male cyclists	X-over	2 x 5 km maximal cycle	a) Passive b) Active cycle c) Massage d) Active-massage (all 20 min)	a) Cycle time b) BLa	↑ recovery with active-massage (ES = 0.34) but no sig. for active alone (ES = 0.22). Quicker BLa recovery with active recoveries.
Weltman et al. (1979)	9 males	X-over	2 x Maximal cycle	a) Passive b) Active cycle (>AT) c) Active cycle (>AT+ O ₂) d) Active (<AT) (all for 20 min)	a) Cycle rpm b) BLa	No performance differences between recovery modes (ES = -0.02 – 0.07). Active recovery ↓ BLa. Active (>AT + O ₂) had quickest BLa clearance.
Thiriet et al (1993)	16 male	X-over	4 x 130% maximal cycling till exhaustion.	a) Passive b) Active cycle (legs) c) Active cranking (arms) (all for 20 min following each cycle)	a) Work b) Cycle time c) Power d) BLa	↓ work and time decrement with active leg (ES = 0.19 - 0.79; 0.22 - 0.57) and arm recovery (ES = 0.19 - 0.64; 0.35 - 0.74). No power decrement with active leg recovery (ES = 0.34 - 0.70). BLa ↓ with active recovery
Sanders (1996)	14 male state level hockey players	X-over	2 x Cycle Wingate test	a) Passive b) Active cycling c) Contrast therapy (all for 12 min)	a) Total work b) Average power c) Power decline d) Peak power e) Blood pressure f) BLa g) RPE h) HR	No performance difference between recovery modes. HR ↑ for active and contrast therapy. RPE lower at all times for immersion. BLa lower during recovery and post test for active and contrast therapy.
Lane and Wenger (2004)	10 males	X-over	2 x intermittent cycle sprints	a) Passive recovery b) Active cycling c) Cold water immersion d) Massage (all for 15 min)	a) Total work	↓ total work after passive recovery. No change with active recovery (ES = -0.02) or immersion (ES = 0.02).

X-over = randomised crossover trial; BLa = blood lactate; AT = anaerobic threshold; RPE = rate of perceived exertion; HR = heart rate; ↑ = significant increase; ↓ = significant decrease; no sig = non significant; ES = Cohen's effect size.

minutes of recovery in-between. Active recovery consisted of cycling at a load equivalent to $50\%\dot{V}O_{2\max}$. During testing the cycling time averaged approximately 380 seconds with a time increase of 2.9 – 9.9 seconds dependent on the recovery mode. A non-significant small beneficial effect ($ES = 0.22$) occurred in cycling time using active rather than the passive recovery intervention. However, a mixture of active recovery and massage provided a greater improvement to performance recovery compared to passive recovery ($ES = 0.34$).

The mechanism that caused the active-massage recovery improvement is unknown. It may be reasoned that it is unlikely to have been massage due to the trivial benefit ($ES = 0.17$) of massage alone and other research that has found no recovery benefit from massage (Hemmings *et al.*, 2000; Tiidus, 1997; Tiidus *et al.*, 2004; Weerapong, 2005). The shorter duration (7.5 minute compared to 15 minutes) of active recovery when combined with massage may have allowed greater glycogen sparing as well as improved metabolite clearance (Fairchild *et al.*, 2003), nevertheless this is speculation. While no other studies were found that provided supporting evidence it could be postulated that shorter active recovery times may be more beneficial for performance recovery. Experimental protocols using smaller recovery durations (30 seconds to 4 minutes) between short duration repeated maximal cycling (6 – 30 seconds) have observed small to moderate beneficial recovery improvements of 2% ($ES = 0.82$; Bogdanis, Nevill, Lakomy, Graham, and Louis, 1996) and 5% ($ES = 0.40$; Signorile, Ingalls, and Tremblay, 1993) using active recovery. However, these short recovery periods have been studied for intra-repetition recovery effects rather than for post-exercise/competition recovery effect.

Intensity of the active recovery protocol may also be an important factor for recovery, with low intensity activity allowing the body to recovery without producing additional metabolites or inhibiting substrate replenishment. In 1979 Weltman *et al.* conducted a study comparing 20 minutes of passive rest to 20 minutes of active recovery at two different intensities. A higher-intensity active recovery ($65\%\dot{V}O_{2\max}$) was included to analyse the recovery rate at the subjects' anaerobic threshold. No significant benefit was observed using either of the active recovery intensities ($40\%\dot{V}O_{2\max}$ or $65\%\dot{V}O_{2\max}$) compared to passive rest. This non-significance benefit with active recovery was in contrast to the earlier study by Weltman *et al.* (1977). Some differences existed

between the two studies by Weltman et al., notably that the 1979 study had two maximal cycle bouts of 5 minutes separated by a 20-minute recovery period, whereas the earlier study consisted of one-minute bouts of cycling. Data in the two studies were analysed using different methods. The 1977 study compared cumulative revolutions whereas the 1979 study used an ANOVA comparing rpm at the end of each minute between each recovery mode. However, if cumulative revolutions in the 1979 study are calculated and compared only trivial effects ($ES = -0.02 - 0.07$) were observed between passive recovery, low intensity cycling and high intensity cycle recovery.

Previous research studies only considered the effect of active recovery on one repeated bout of cycling. Thiriet et al. (1993) studied the effect of active recovery with repeated bouts of maximal exercise. Four maximal cycle bouts were interspersed with 20 min of recovery. The subjects used in the study were all gymnasts who were also engaged in other rigorous sports training. None were cyclists, but all subjects had six familiarisation sessions that equated to 1 - 1.5 hours of practice. During familiarisation individual $\dot{V}O_{2max}$ was determined and a test load consisting of approximately 130% of maximal aerobic power (MAP) was calculated. The fatiguing exercise involved cycling at this load on an ergometer at 70 rpm till failure. When pedalling frequency, monitored by electronic micro-switch, dropped below 40 rpm the test was stopped and recovery began. Average exercise duration was less than two minutes (92.4 – 105.3 s).

Recovery consisted of 20 minutes of either 30%MAP at 70 rpm of leg cycling, 30%MAP at 40 rpm of arm cranking, or seated rest. By the third exercise repetition duration of cycling had reduced by approximately 14% ($p < 0.03$) in the control and by less than 9% ($p > 0.05$) with either active recovery mode. Consequently work during cycling decreased by 20% with passive recovery and by less than 14% with the active recoveries. Differences in work and duration of cycling between active leg and active arm recovery for each exercise session were mainly trivial ($ES = 0.01 - 0.26$), with greater benefit using active leg recovery.

Thiriet et al. (1993) concluded that active recovery was beneficial in the preservation of performance. Since low-intensity active cycling with the arms helped in the ability

to maintain leg cycling performance the mechanism of this improved recovery is likely to be the increase in cardiac output and blood flow throughout the body. Leg cycling provided greater cardiac output and increased blood flow allowing for greater transportation of metabolites and nutrients within the body. Restitution of performance could also be linked to the return of blood plasma fraction as hemoconcentrations were higher throughout the testing with passive recovery ($p < 0.01$) despite significantly lower total fluid losses in this group. With multiple exercise sessions beneficial effects of active recovery may be cumulative. Effect sizes for the difference in the change in mean power between each cycle session and active leg and passive recovery progressed in Thiriet et al.'s (1993) study from trivial to small ($ES = 0.06, 0.23, 0.34$). However, both work ($ES = 0.19, 0.79, 0.52$) and duration ($ES = 0.35, 0.57, 0.22$) did not follow such compounding effects. More research into multiple exercise sessions and recovery is required to determine possible cumulative effects of active recovery.

Water immersion

Two studies were found that examined the effect of both water immersion and active recovery between bouts of cycling (see Table 6). Sanders (1996) examined the performance recovery effect of active, passive and water immersion on a repeated Wingate cycle test. After performing a 30-second bout of maximal cycling the 14 subjects recovered for 12 minutes either sitting on the cycle, performing low-intensity cycling, or contrast bathing (3 x 3.5 min hot: 30 s cold). Sanders observed no significant difference in power or work, though the performance decline of passive recovery (1.8%) was greater than active recovery (0.3%) and water immersion (0.5%). A major problem with Sander's study, like two other immersion studies (Coffey et al., 2004; Vaile et al., 2004), is that contrast therapy rather than water immersion per se was used. The effect that short-duration immersion in alternating temperatures may have on performance has not been established and while alternating temperature is unlikely to provide any benefit (Myrer et al., 1994; Myrer et al., 1997) the benefits of this therapy cannot be dismissed.

One other cross-over study (Lane & Wenger, 2004) has compared active, passive and water immersion as a cycling recovery strategy. A strong aspect of Lane and

Wenger's study was that it incorporated intermittent exercise, similar to the intermittent nature of team sports. The exercise consisted of an 18-minute cycling protocol of 22 intermittent maximal effort sprints of 5 – 15 s duration with a work to rest ratio of 1:5. Recovery post-exercise consisted of 15 minutes of passive sitting, cycling at 30% $\dot{V}O_2$ max, or immersion in cold water (15°C). Twenty-four hours later the subjects repeated the intermittent cycle protocol.

Change in performance was taken as the differences in work between the cycling sessions relative to the average work performed in the 5-second bouts. After 24 hours work decreased by $78 \pm 17\%$ with passive recovery, by $13 \pm 24\%$ with active recovery and increased with immersion by $11 \pm 19\%$. The beneficial effect sizes were large compared to passive recovery when active recovery ($ES = 1.20$) and water immersion ($ES = 1.65$) were used. How Lane and Wenger calculated these numbers is unclear. Comparison of the total work performed over each session and each recovery mode produced effects that were much smaller. When recovery was passive there was a significant decrement ($1.9 \pm 4.6\%$, $p < 0.05$) in total work in the second cycling bout, with no significant decrement in total work when active recovery or water immersion was the recovery method performed. While significantly different from passive recovery, the beneficial effect of active recovery or water immersion on total work was trivial ($ES = 0.11$ and 0.16 respectively). Small beneficial effect sizes could be due to the length of time (24 hours) between the exercise sessions. A within-day performance measure may have provided a better understanding of possible effects of the different recovery modes.

When considering the cycling studies there seems some support for the use of light-intensity cycling and water immersion for benefiting repeated cycle performance. While none of the studies found that light intensity cycling or water immersion were detrimental to performance, the benefits were likely to be trivial. With repeated bouts of exercise and active recovery, such trivial benefits may equate to a significant worthwhile benefit to performance.

Running

Most sports consist of muscles acting in eccentric-concentric cycles of movement. During eccentric loading higher forces can act on a muscle than during concentric loading and greater muscle damage can occur (Clarkson & Sayers, 1999). If mechanical damage to the muscle contributes to fatigue, then actions that replicate eccentric-concentric muscle actions, such as running or jumping, may induce greater fatigue.

Two studies have utilised treadmill running to assess active recovery (see Table 7). Lattier et al. (2004) induced fatigue in subjects using 10 one-minute repetitions of 120%MAV treadmill sprints at an 18% gradient, with two-minute rests between each sprint. After the uphill running subjects performed 20 minutes of either seated passive or active (at 50%MAV) recovery. Eighty minutes after the exercise subjects were assessed in an all-out run test at 90%MAV at an 18% gradient. No significant differences were observed in running performance between the two recovery groups and effect size was trivial (ES = -0.06).

Table 7: Effect of active recovery and water immersion on repeated bouts of running

Author	Subjects	Study design	Exercise	Recovery modes	Outcome measures	Main findings
Lattier et al. (2004)	8 males regional - national level athletes	X-over	10 maximal treadmill sprints	a) Passive b) Active jogging (both for 20 min)	a) Sprint time b) Leg MVIC	MVIC ↓ for all recovery modes, no sig. difference between modes. No sig. difference in all-out run times.
Coffey et al. (2004)	14 highly active males	X-over	2 sets of treadmill sprints (120% and 90% PRS)	a) Passive b) Active jogging c) Contrast therapy (both for 15 min)	a) Sprint times b) Critical power c) RPE _{rec} d) BL _a	No differences in time or power between recovery modes (ES = 0 – 0.17). ↓ RPE _{rec} with contrast therapy. ↓ BL _a recovery with passive recovery.

X-over = randomised crossover trial; RCT = random control trial; PRS = peak running speed; MVIC = maximal voluntary isometric contraction; BL_a = blood lactate; RPE_{rec} = rate of perceived recovery exertion; ↑ = significant increase ($p < 0.05$); ↓ = significant decrease ($p < 0.05$); no sig = non significant.

Coffey et al. (2004) also observed no benefit with active recovery, or water immersion compared to passive recovery on running performance. Fourteen highly trained athletes conducted two running sessions on a treadmill. Each running session consisted of a 120%MAV run to failure, 10 min rest, and 90%MAV run to failure.

Following these runs subjects rested passively, jogged at 40%MAV, or performed contrast therapy, for 15 min. Four hours later the two runs were repeated. While lower on the second bouts of running, no significant differences in run times or critical powers occurred between the recovery modes ($ES = 0 - 0.17$). A limitation with Coffey et al. (2004) was that the second bout of exercise was performed four hours post-recovery during which perceived fatigue, blood pH and lactate had returned to baseline levels. Such duration between one repeat of short exercise bouts may have reduced any effect that the different recovery modes had on performance. Time to exhaustion following active recovery or contrast therapy was 8.4 – 17.9 s longer than passive recovery, but these times were not significantly different to each other and amounted to trivial beneficial effect sizes of 0.08 and 0.16 respectively. Again, the use of contrast therapy rather than immersion at a constant temperature was used and could have influenced any benefit.

Two other studies have compared the effect of water immersion (contrast therapy) to other recovery modalities on repeated sprinting performance (Hamlin & Magson, 2002; Hamlin & Sheen, 2004). These studies were abstracts from conference presentations that supplied little information for critical analysis. If the data is used as anecdotal evidence, these two studies observed no significant benefit to sprints repeated one or two hours after water immersion. Overall, neither water immersion nor active recovery would appear to provide any benefit to recovery from short-duration maximal running.

Jumping

Fatigue from eccentric loading exercises such as plyometrics and eccentric presses appear to cause a reduction in jumping power (Friden & Lieber, 2001; Vaile et al., 2004; Viitasalo et al., 1995), which may be attributed to muscle damage (Friden & Lieber, 2001; Vaile et al., 2004). Two articles have assessed the effect of water immersion post-exercise with regard to jumping ability (see Table 8).

Viitasalo et al. (1995), who conducted the study on 14 national track and field athletes over five days, analysed changes in drop jump and rebound jumping kinematics and

kinetics. Subjects performed five strenuous sessions of strength, plyometric and sprint training over the course of three days. During the evening following training subjects were either immersed in warm water or not immersed. Drop jumps, and rebound jumping performance were measured pre-exercise and at three occasions post-intervention (the following morning and afternoon, and the next morning). A significant decrease ($8 \pm 8\%$; $p < 0.001$) in jumping power and increase ($6 \pm 8\%$; $p < 0.001$) in ground contact time was recorded during the week with non-immersion compared to the immersion week. The effect size of the difference was small to moderate ($ES = 0.37 - 0.68$).

Table 8: Effect of water immersion on jump performance

Author	Subjects	Study design	Exercise	Recovery modes	Outcome measures	Main findings
Vaile et al. (2004)	9 female and 4 male recreational athletes	X-over	DOMS inducing leg press	a) Passive b) Contrast therapy (both for 15 min)	a) Weighted jump squat b) MVIC c) VAS-Score d) Thigh volume	Immersion thigh volume increased less with contrast therapy Greater reduction in MVIC and power with passive recovery.
Viitasalo et al. (1995)	6 female and 8 male national track and field athletes	X-over	Five strength-power sessions in 3 days	a) Passive b) Warm-water immersion (after evening trainings) (20 min)	a) Drop jumps b) MIVC c) VAS-score d) Rebound jumps	↓ power output and ↑ contact time in rebound jumps when no water recovery was used.

X-over = randomised crossover trial; RCT = random control trial; MVIC = maximal voluntary isometric contraction; VAS = visual analogue scale; ↑ = significant increase ($p < 0.05$); ↓ = significant decrease ($p < 0.05$).

No other jump data was found to be significantly different by Viitasalo et al. (1995). However, if pre-post effect sizes are compared for the morning following the evening training small positive effects are also apparent with immersion recovery for drop jump height ($ES = 0.30$), drop jump contact time ($ES = 0.31$) and rebound jump height ($ES = 0.44$). Average effects of the three post-intervention measures resulted in small to moderate benefits for all performance measures except rebound jump height ($ES = -0.05$).

Vaile et al. (2004) investigated whether there was a relation between muscle damage, edema and muscle function and whether water immersion (contrast therapy)

influenced these variables. Vaile et al. (2004) induced damage into leg muscles and tracked weighted squat jump performance over the next three days. After the induction of muscle damage subjects rested passively or immersed themselves in water for 15 min. The contrast protocol alternated between one minute of cold (9°C) and three minutes of hot (41°C) immersion up to the subjects' gluteal fold. Each day squat jumps were performed on a Smith machine with a weight consisting of 30% of the subjects' MVIC squat force. The squat jumps were initiated from a knee angle of 90° with a two-second pause, thus the influence of the stretch shortening cycle would have been minimised. Over 72 hours the return in squat jump peak power towards baseline occurred quicker with water immersion than with passive recovery (24-versus 72-hrs respectively). This significantly quicker recovery of peak power had a moderate to large effect size (ES = 0.67 to 1.46).

Accompanying the changes in peak power was a lesser increase and faster reduction in thigh circumference (reduced edema). Whether this reduction in fluid build-up in the thighs was due to the cold temperature application (alternated with hot temperature) or due to the hydrostatic pressure of the water is again unknown. The trend of performance recovery and edema that Vaile et al. (2004) observed in the legs was similar to those observed by Thiriet et al. (1993). Perhaps performance recovery is linked to the restitution of fluid from the muscles into the blood and requires further investigation. However, both light active recovery (Sayers et al., 2000) and water immersion appear to be effective in the restoration of performance when muscle damage has been induced in the muscles.

From the two studies that measured jump performance and water immersion there appears to be some benefit to performance when using water immersion. Whether water immersion has any influence in repeated jump performance within a day has not been researched. Unfortunately no studies were found that compared active and passive recovery with jumping protocols and is another area requiring research.

Summary

Given the scarcity of quality research into recovery, and the varied methodological approaches taken in what little research there is, arriving at any consensus in terms of practical applications is difficult. However, the use of active recovery or water immersion does not appear to be detrimental in terms of recovery for performance. In some studies the use of active recovery or water immersion post-exercise sustained strength, cycling and jump ability, but not running performance. While low-intensity recovery may be beneficial, higher intensity (approaching the anaerobic threshold) active recovery is unlikely to provide any benefit to performance in the short-term. The beneficial effects of low-intensity active recovery or water immersion from a single-repeat short-duration exercise were found to be trivial to small or about the same as the mean variance. This explanation could account for inconsistent findings and the generally trivial-small effects in the literature. Compounding small recovery benefits through multiple bouts of exercise and recovery or highly fatiguing (muscle damaging) protocols may also explain the performance benefit observed in some studies. Reduced edema due to either cold water or hydrostatic pressure during water immersion, or muscular pumping during active recovery, may provide greater performance recovery.

In a tournament environment the use of post-game light-intensity active recovery or water immersion for a short duration (five to 10 minutes) may provide some small benefit to performance in upcoming games. Both modes of recovery are unlikely to cause any decrement to performance. Both active recovery and water immersion may also increase the ability of athletes to recover from training. Whether one is more advantageous in maintaining performance is unclear. More research into the use of active recovery and water immersion is required and the possible effect of water temperature(s) should be isolated in such studies.

CHAPTER 4:

WATER IMMERSION, ACTIVE RECOVERY AND PASSIVE RECOVERY EFFECTS ON REPEATED BOUNTS OF EXPLOSIVE EXERCISE AND BLOOD PLASMA FRACTION

Prelude

Despite a paucity of supporting evidence a considerable amount of literature recommends that athletes should perform active recovery and, to a lesser extent, water immersion after exercise to aide recovery and enhance future performance. Of the limited research conducted into post-exercise recovery there are numerous gaps in the literature, such as; (1) the separate performance effects of water temperature and hydrostatic pressure have not been determined; (2) water immersion is often conducted in the field after active recovery, but not so in research; (3) research exercise / assessment is often performed immediately post-recovery, whereas normally there would be a passage of time after a recovery period between one competitive event and the next event; (4) multiple bouts of exercise and recovery are seldom investigated, whereas athletes may compete a number of times within a day; and, (5) most sports (especially team sports) consist of an athlete performing repeated bouts of explosive power (such as jumping and short sprints), but there is a lack of research into the effect of active recovery or water immersion on repetitive expressions of explosive power.

When considering what mechanisms that may assist recovery, the ability to return fluid from the muscles to the blood post-exercise may be important. Some research has observed both a greater return of fluid into the blood and greater performance recovery utilising active recovery and water immersion compared to passive recovery after exercise. However, research that has incorporated both blood plasma and performance changes is scarce. Therefore, given these limitations the purpose of this study was to investigate what effect combinations of water immersion, active and

passive recovery had on total peak power and total work during repeated bouts of explosive exercise. What effect these recovery modes had on the change in blood plasma fraction throughout a day was also investigated.

Introduction

During tournament-type events athletes may compete a number of times a day over two or three days. Playing a number of high-intensity games within a short period of time may inhibit the ability of an athlete to fully recover and return to their physiological and performance baseline. The ability to recover quicker may provide a competitive advantage to an athlete. As such, numerous texts and sources recommend performing light activity after exercise to enhance recovery. Much of this advice is based on the premise that light-exercise post-game increases the metabolism of blood lactate (Bonen & Belcasro, 1977). Water immersions (such as contrast baths) are also gaining popularity as a method of enhancing recovery (Calder, 2003; Cochrane, 2004) as these methods are also thought to reduce post-exercise blood lactate accumulation (Hamlin & Magson, 2002; Sanders, 1996). However, it is questionable whether blood lactate is a determinant of performance fatigue as a number of studies have observed that elevated levels of blood lactate have no detrimental effect on maximal performance (Thiriet et al., 1993; Weltman et al., 1979; Weltman et al., 1977).

Rather than blood lactate, fluid kinetics may be a determinant to performance recovery from acute bouts of high-intensity exercise. During high-intensity exercise fluid is forced from the blood into the interstitial space within the acting muscles due to a peripheral increase in mean arterial pressure (Collins et al., 1989). Both active recovery and the immersion of large body segments have been observed to return blood plasma and blood constituent levels towards baseline faster than passive recovery (Thiriet et al., 1993; Tomasik, 1983). Some researchers (Thiriet et al., 1993; Vaile et al., 2004) have observed similar restitutions of performance and blood plasma after intense exercise using active recovery and water immersion. In a study by Thiriet et al. (1993) the recovery of blood plasma was measured over a day in which four bouts of maximal cycling was performed. Following the bouts of exercise either active recovery or passive recovery was performed. Active recovery increased the

return of fluid to the vascular system. Percentage difference in the change in blood hematocrit fraction between passive recovery and active leg recovery was on average $5.3 \pm 3.1\%$. The associated percentage difference in the change in work, cycle duration and mean power was $7.8 \pm 4.7\%$, $6.2 \pm 2.8\%$, and $3.1 \pm 2.1\%$ greater with active recovery. In a study by Vaile et al. (2004), after a muscle-damaging exercise bout the differences in the change in means of thigh volume over 72 hours was on average $1.7 \pm 1\%$ less when subjects recovered with water immersion. The associated percentage improvement in maximal isometric squat force and squat jump peak power recovery was on average (over 72 hours) $9.8 \pm 5.0\%$ and $12.6 \pm 5.0\%$. If fluid movement into the vascular system can be increased by active recovery or water immersion, then perhaps performance recovery may also increase.

Many sports, such as volleyball, netball and rugby, require a player to perform multiple expressions of explosive power (e.g. repeated jumping or sprinting) during competition rather than a single (e.g. shot-put) expression of power (Howell *et al.*, 2001). Multiple sessions of explosive jumping within a day, such as in a tournament-style competition, can cause large decreases in jump performance over the day (Skurvydas *et al.*, 2000). If water immersion or active recovery can increase an athlete's ability to recover from jumping and maintain jump performance, such recovery methods could be beneficial to sports that require repeated bursts of maximal explosive power. Water immersion has been observed to increase an athlete's ability to recover from fatigue and maintain explosive leg power over a 24 – 72 h period (Vaile et al., 2004; Viitasalo et al., 1995). Whether water immersion has any influence on repeated jump performance within a day has not been researched, though there is a possibility water immersion could aide within-day recovery from exercise (Lane & Wenger, 2004). Considering that active recovery is espoused as a method to reduce fatigue little research has been conducted on the effect of active recovery following repeated bouts of exercise within a day. Research by Thiriet et al. (1993) and McEniery, Jenkins and Barnett (1997) observed moderate to very large beneficial effects ($ES = 0.52$ and 7.0) of active recovery following repeated bouts of cycling. However, no studies were found that have compared active and passive recovery effects on repeated bouts of explosive movements (e.g. jumping), an activity typical of most athletic settings.

With regards to research into water immersion as a recovery strategy, there appear some fundamental limitations in the research to date that hinders our understanding of the true effect of such a recovery strategy. The majority of research in this area has focussed on contrast therapy (alternating immersion in hot and cold water) or, hot or cold water rather than water immersion per se. The limitations of such an approach are; (1) the separate effects of temperature and hydrostatic pressure have not been determined, and (2) in an applied setting facilities would be required to heat and cool water. Teams and individuals may not have access to such facilities in a practical situation. In the field many teams conduct water immersion in pools or containers filled with water or iced water. To replicate what happens in a practical situation and to remove the effect that temperature variation may have on performance, research needs to isolate the effects of water immersion alone. Furthermore, in the applied setting water immersions is usually conducted after a bout of active warm-down. However, no water immersion research to-date has replicated active-immersion recovery strategies. Given these limitations, the aim of this study is to analyse whether water immersion, active recovery or a combination of both may enhance performance recovery from repeated explosive bouts of jumps compared to passive recovery. Additionally, the changes in blood plasma fraction will be analysed to observe if there is any relationship with possible performance benefits from the different recovery modes.

Methods

Subjects

Seven healthy male university students who were currently involved with, and had a minimum of twelve months experience in high-intensity exercise/sport participated in this study. Each subject was pre-screened for current or previous injuries and medical conditions that would influence participation. All subjects signed an informed consent document before testing commenced. Ethics for this study was approved by the Auckland University of Technology Ethics Committee. The characteristics of the subjects are shown in Table 9.

Table 9: Subject descriptive characteristics

	Age (yr)	Height (cm)	Body mass (kg)	Total skinfold ^a (mm)
Mean \pm SD	27 \pm 6	1.75 \pm 0.05	74.6 \pm 6.9	24.5 \pm 3.0

^aCalf plus thigh skinfolds as measured by the techniques of the International Society for the Advancement of Kinanthropometry

Study design

This study was designed to compare the effects of four different recovery modes on total peak power and work during repetitive jumping and change in blood plasma fraction over a day. To investigate the acute responses a randomized cross-over experimental design was utilized. To negate any possible order effects, protocol orders were assigned through block randomization using a Latin square design. Subjects had a familiarization session and four days of testing, each a week apart. During preliminary testing, subjects' height, weight and skinfolds were measured, starting position of a squat machine sled was determined, and water bath height ascertained. Subjects were also familiarised with the protocol for repetitive jumping on the supine squat machine. The initial familiarisation of the jumping protocol was to reduce any acute learning effects. Subjects underwent testing on the same week day over four weeks using a different recovery protocol. All tests were performed at the same time of day to reduce the effect of any diurnal variations. Subjects were asked to wear the same clothing (swimming trunks) during each of the four test sessions and to refrain from any exercise 48 hours prior to testing. Over the day a subject performed three jump sessions each two hours apart (i.e. testing started at 7am, 9am, and 11am). At the start of a jumping session subjects rested (seated) for 5 minutes and some baseline measures were taken (heart rate, blood glucose, blood plasma fraction and leg fatigue). A standardised warm-up was then performed, consisting of five minutes cycling on a Monark 818e ergometer cycle (Monark Exercise AB, Vansbro, Sweden) at a workload of 60 W (60 rpm x 1 kp). Following the cycling subjects performed five sub-maximal jumps (approximately 70% of a subject's subjective maximal effort) on the supine squat machine for pre-test familiarisation, and then had another one minute of rest. One jump session was then performed, immediately followed by 10 minutes of recovery. Each test day consisted of a different recovery method. A heart rate monitor remained attached to the subject post-recovery and the subject was free until

the next jumping session. Subjects were instructed not to perform any strenuous activity between jump sessions and the heart rate monitor was used to assess the activity (mean heart rate) of the subject between testing bouts.

Repetitive jumping protocol

An isoinertial supine squat machine (Fitness Works, Auckland, NZ) was used for all jump sessions (see Figure 4). The sled of the squat machine allowed participants to perform explosive jumps with the back rigidly supported. The back support minimized the risk associated with similar explosive exercises, such as squats, performed in an upright position. The supine squat machine had a 300 kg pin-loaded weight stack to provide resistance. At maximum displacement the sled contacts springs that are attached to the machine frame. Resting position of the sled was adjustable and permitted standardisation of the knee joint angle at a starting position of 90° (taken as the angle between the lateral malleolus, lateral epicondyle of the knee and greater trochanter of the left leg).



Figure 4: Isoinertial supine squat machine

Attached to the supine squat machine was a linear position transducer (PA80, Unimeasure, Oregon, USA – mean sensitivity 0.499 mV/V/mm, linearity 0.05% full scale), which measured the vertical displacement of the weight stack with an accuracy of 0.01cm. The analogue signal produced by the transducer was converted to a digital signal using a BNC block (BNC-2110, National Instruments, Austin, Texas). Displacement data was sampled at 200 Hz by a computer running a custom built data acquisition program (LabView 6.1, National Instruments, Austin, Texas). Each jump session consisted of subjects performing three sets of 20 weighted jumps on the supine squat machine, using a resistance equivalent to bodyweight (60 jumps per jump session). Each jump began with a standardised vocal signal every three seconds. A one minute rest period separated each set. Subjects were instructed to control the load during the landing/eccentric phase of each repetition and to perform the concentric phase as explosively as possible to attain maximum displacement (Hoffman & Kang, 2002; Young *et al.*, 1995). At the start position of each jump the weight plates rested on the weight stack.

Recovery protocols

Following the three jump sets subjects performed one of four recovery protocols. The four recovery protocols for these testing sessions included:

- (A) Active recovery consisting of five minutes cycling on a Monark cycle ergometer at 60 W (60 rpm at 1 kp) followed by five minutes of seated rest.
- (I) Five minutes of water immersion up to the gluteal fold at a mean (\pm SD) water temperature of $19 \pm 1^\circ\text{C}$, followed by five minutes of rest.
- (AI) Five minute of active recovery followed by five minutes of water immersion.
- (P) Passive seated recovery for 10 minutes.

One minute was allowed after the last set of jumps for measurements and recovery preparation (e.g. removal of shoes before immersion). One minute was also allocated between the first 5-minutes of recovery and the second 5-minutes of recovery to allow for towel drying after immersion. Active recovery and water immersion duration were based on those recommended to netball players competing in a tournament setting (Netball New Zealand, 2004). A load of 60 W was used for active recovery as

previous research into active recovery (Weltman et al., 1977) observed significant benefits at this wattage. Immersion was conducted in a wheeled container made from high-density polyethylene (Stowers, Auckland, New Zealand) in which the subjects stood. Total volume of the immersion bath was 240 L. The immersion bath was filled with fresh mains water before any testing. No supportive studies are provided by Netball New Zealand for the duration and depth of water immersion, and the protocols used by elite netball teams are unstandardised and ad hoc (Bonham et al., n.d). However, research by Vaile et al. (2004) observed significant performance recovery with a depth of immersion to the gluteal fold and anecdotally this depth appears to be used in practice by many New Zealand sports teams.

Measurements

On test days subjects were fitted with a heart rate monitor (Polar T810, Polar Electro Oy, Kempele, Finland), and rested (seated) for five minutes prior to exercise. Baseline levels of blood plasma fraction, blood glucose, perception of leg fatigue, and resting heart rate were taken. Measures of heart rate and leg fatigue were taken pre-warm-up in the first jump session to gain a perception of possible pre-test fatigue. Post-jump the subjects' rate of perceived exertion (RPE) was measured using the 14-point Borg scale. Post-jump RPE was measured to determine whether the subjects were exercising maximally. Between each jump session the heart rate monitor remained attached to the subject and voluntary fluid consumption (using a measured drink container) was measured. Mean heart-rate (sample rate 0.2 Hz) and fluid consumption between the jump sessions was recorded to determine if differences in activity or hydration may effect blood plasma or performance.

Leg Fatigue

Perceived leg fatigue was measured pre-warm-up, post-jump and post recovery using a scale (see Table 10) that was based on the 10-point OMNI Perceived Exertion Scale (R. J. Robertson et al., 2003). Similar scales have had high reliability and construct validity in measuring perceived pain (Cook *et al.*, 1997) and exertion (R. J. Robertson et al., 2004; R. J. Robertson et al., 2003). Other research (Sanders, 1996; Vaile et al.,

2004) investigating the effects of different recovery modes have used such 10-point scales to measure changes in muscular fatigue.

Table 10: Scale used for perception of leg fatigue

Perceived leg fatigue	
0	No fatigue
1	Very slight fatigue
2	
3	Mild fatigue
4	
5	
6	Very fatigued
7	
8	Very, very fatigued
9	
10	Extremely intense fatigue

Modified from Cook et al. (1997)

Blood sampling

Before the first set of jumps in each session the level of glucose in the blood was measured using a MediSense Optium analyser (Abbot Laboratories, Bedford, MA, USA). Prior to sampling the MediSense Optium was calibrated as per the operating instructions provided by the manufacturer. After calibration a blood droplet was obtained from the right finger of the subject and sampled using sterile techniques (Watenpaugh et al., 2000). Within 20 seconds the analyser displayed the level of blood glucose. A study by Hawkins (2004) observed that over the glucose range of 4 – 8 mmolL⁻¹ the MediSense Optium has a measurement error of 1.0 – 2.9% with a coefficient of variation (CV) of 2.4 – 2.9% when compared to laboratory plasma glucose measurements. Blood glucose was measured to record the effect that low glucose levels may have had on jump performance. This measure was used due to a lack of breakfast affecting one subject's performance during pilot-testing. Subjects were asked to maintain a similar nutritional intake on test days and if a subject's blood glucose level was less than 4 mmolL⁻¹ then the test session was cancelled. No testing was cancelled due to low blood glucose during the actual study.

Blood plasma fraction was measured pre-jumping, post-jumping and post recovery. To measure plasma fraction 75 μ L of blood was milked from the finger of the subjects' to fill a haematocrit capillary tube (Heinz Herenz, Medizinalbedarf, Germany). Tubes were then sealed at one end with a vitrex plasticene and centrifuged at 11,800 rpm (Hawksley Micro-Haematocrit Centrifuge MkIV, Hawksley & Sons, Sussex, England) for 5 min. The centrifugal force causes the separation of the blood haematocrit and plasma. Plasma and haematocrit fraction was measured to the nearest 0.5% using a circular perspex reader (Hawksley Haematocrit Reader, Hawksley & Sons, Sussex, England). Previous research has used this method to determine the concentrations of plasma and haematocrit in the blood (Kiew *et al.*, 2003). To reduce error blood sampling and plasma level determination was conducted by the same investigator throughout the study.

Jump kinetics

The total concentric peak power and total concentric work of each jump set was calculated from displacement data derived from the linear transducer. Peak power was reported rather than mean power due to peak power having a greater correlation to vertical jump performance (Dugan *et al.*, 2004). Peak power and work of each set of jumps was determined using a Labview program (National Instruments Corp., Austin, Texas, USA) designed for analysing concentric and eccentric data during multiple repetitions (Denton, 2005). Reliability of displacement, mean force, peak force, and mean power values (ICC = 0.92 - 0.98, $p < 0.001$) have been reported previously for single jumps on a supine squat machine (Cronin *et al.*, 2004; Cronin *et al.*, 2001b, 2003). Previous research using the same instrumented supine squat machine has verified a comparison of data simultaneously collected by an accelerometer (Cronin *et al.*, 2003).

Statistics

A one-way analysis of variance was performed to determine any significant difference in baseline values of blood plasma fraction, leg fatigue, RPE, and heart rate, and in pre-jump blood plasma levels, between experimental days. A one-way analysis of

variance was also used to determine if any differences in inter-jump session fluid consumption or activity occurred between recovery modes.

Descriptive statistics are presented as means \pm standard deviation (SD). Total peak power and work data were log transformed to reduce non-uniformity of the data and to express reliability and effects as percentage changes. To determine reliability of the jumping protocol test-retest intraclass correlations (ICC) and coefficients of variation (CV) were calculated for power and work between the first jump session of week one and two. Ninety percent confidence limits (90%CL) for ICC and CV are also presented. The ICC and CV were calculated using a spreadsheet available from <http://www.sportsci.org/resource/stats/xrely.xls> (Hopkins, 2000).

Two methods were used to determine effects of the different recovery modes on total peak power and total work (1) hypothesis testing using a two-way repeated measures analysis of variance (4 x recovery mode, 3 x jump session), with Holm-Sidak post-hoc comparisons, to determine any significance between jump sessions and/or recovery modes; and (2) inferential statistics based on the precision of the estimate. Inferential statistics interpret the practical importance of confidence limits for each effect, and the likelihood that an effect is beneficial, detrimental or unclear (Hopkins, 2002a; Stuart *et al.*, 2005). Quantitative and qualitative statistic effects were calculated using a modified spreadsheet (Hopkins, 2005) that combined the standard deviation of the groups being analyzed for each jump session comparison (a fully controlled cross-over trial). Passive recovery was used as the comparison (control) for the three other recovery modes. Due to the uncertainty of a clinical threshold level (percent change in performance), the thresholds of uncertainty were based on an effect difference of 0.2 Cohen's (ES). Using the spreadsheet the qualitative description of "unclear" was based on a >5% chance of benefit and harm (a 90% confidence interval overlap). Otherwise the qualitative magnitude of the observed value of benefit/harm provided by the spreadsheet was reported (25–75%, possible; 75–95%, likely; 95–99%, very likely; 99%, almost certain).

To determine significant changes in blood plasma fraction and perceived leg fatigue a two-way repeated measures analysis of variance (4 x recovery, 3 x sample time) with the Holm-Sidak post-hoc comparisons was used. Hypothesis testing was calculated

using SigmaStat 3.1 (Systat Software Inc, Richmond, California, USA) using an alpha level of 0.05.

Results

Reliability of jump protocol

Test-retest reliability of total peak power was high with an ICC of 0.95 (90%CL = 0.69 – 0.99) and a CV of 3.1% (90%CL = 2.1 – 6.7%) between the first jumping session of week one and week two. Test-retest results for total work were also high with an ICC of 0.82 (90%CL = 0.22 – 0.97) and CV = 4.7% (90%CL = 3.2 – 10.2%).

Baseline measures

No significant differences existed in baseline levels of blood plasma fraction (55.5 ± 2.7 %BV; $p = 0.74$), leg fatigue (0.9 ± 1.1 RPE; $p = 0.34$), heart rate (67 ± 8 bpm; $p = 0.76$), or the initial session total peak power (100482 ± 13557 W) and total work (23219 ± 2842 J) between the different recovery modes. Differences in pre-jump blood glucose levels were non-significant (5.7 ± 0.8 mmolL⁻¹; $p = 0.83$). RPE following the jumps did not significantly differ between recovery modes, but increased significantly from Session 1 to Session 2 (RPE = 16.4 ± 2.0 and 16.8 ± 1.8 respectively; $p = 0.011$), and Session 2 to Session 3 (RPE = 16.8 ± 1.8 and 17.4 ± 1.9 respectively; $p = 0.0017$). Between jumping sessions the intensity of subject activity as measured by average heart rate did not differ between the recovery modes (76 ± 1 bpm; $p = 0.84$), nor did water consumption between jump sessions (mean 260 ± 170 mL; $p = 0.57$).

Total peak power

The mean total peak power (\pm SD) for each recovery mode and each jump set for each jump session throughout the day can be observed in Figure 5. There were no significant difference in total peak power between jump sessions ($p = 0.69$) or recovery mode and jump session ($p = 0.081$). However, there was a significant difference in total peak power between jump sets. That is, the total power output for

the last jump set of each session (jump sets 3, 6 and 9) was lower than the first in jump session 1 and 3 (jump set 1 and 7).

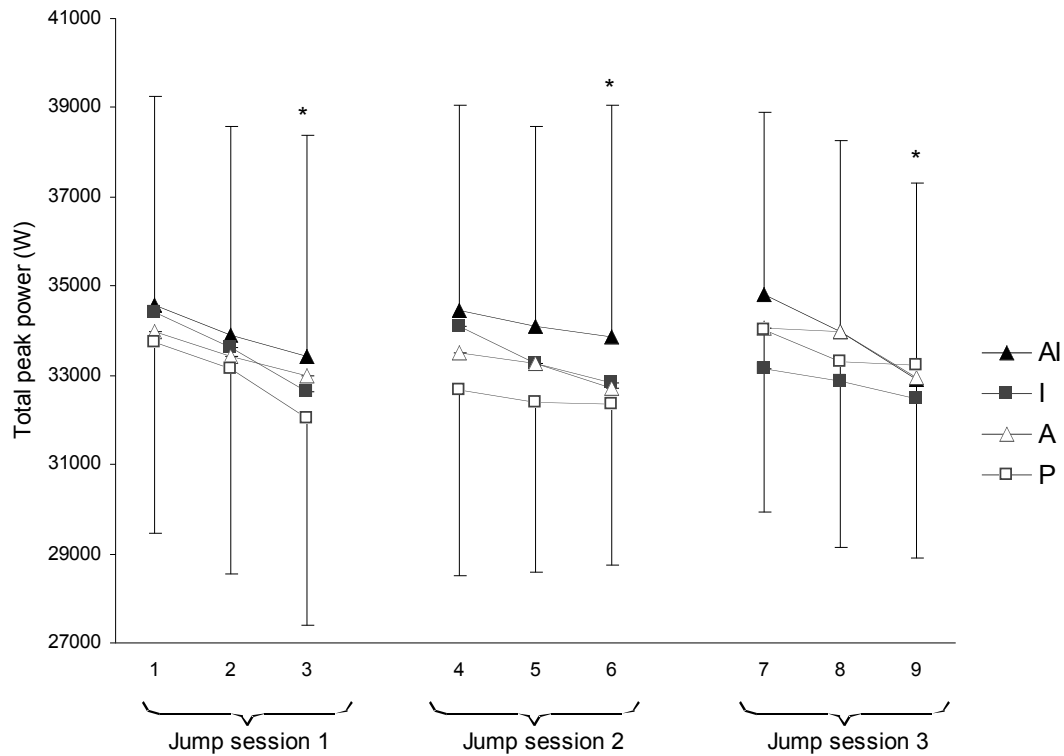


Figure 5: Total peak power (mean \pm SD) produced during each Jump Session for each recovery mode

Note. AI = active recovery and water immersion; I = water immersion; A = active recovery; P = passive recovery; *significantly different from set 1 and 7 ($p \leq 0.0011$)

The difference in log transformed total peak power of *A*, *I* and *AI* compared to *P* between jump session 1 and, 2 and 3 are shown in Table 11. Immersion in water post-intervention is likely to cause a decrement in total peak power (90%CL = -7.9 to -0.1%) by the third jump session compared to passive recovery. There is also a possibility that active-immersion could benefit (90%CL = -0.8 to 4.6%) jump performance after one session but be detrimental (90%CL = -5.1 to 1.5%) to a third jump session.

Table 11: Mean percent (90%CL) change of total peak power from Jump Session 1 for the different recovery modes.

	Change in performance (%)			
	Passive recovery	Active recovery	Water immersion	Active recovery & water immersion
Jump session 2-1				
mean \pm SD	-1.4 \pm 3.6	-0.8 \pm 2.7	-0.8 \pm 4.7	0.5 \pm 2.2
Difference compared to passive recovery; \pm 90%CL		0.6; \pm 2.9	0.6; \pm 4.2	1.9; \pm 2.7
Practical inference ^a		Unclear	Unclear	Possible benefit
Jump session 3-1				
mean \pm SD	1.7 \pm 4.9	0.8 \pm 3.8	-2.3 \pm 2.9	0.0 \pm 3.1
Difference compared to passive recovery; \pm 90%CL		-1.0; \pm 3.3	-4.0; \pm 3.9	-1.8; \pm 3.3
Practical inference ^a		Unclear	Likely detriment	Possible detriment

90%CL: add and subtract this number to the difference to obtain the 90% confidence limits for the true difference in effects.

^aBased on a smallest beneficial or detrimental change in performance of 0.2 Cohen's ES.

Total work

There was no significant difference in total work between jump sessions ($p = 0.77$) or recovery mode and jump session ($p = 0.72$) or jump sets. Total work produced during the passive recovery day was significantly ($p = 0.41$) less than the active-immersion recovery day (see Figure 6). There were no other significant differences in total work performed. The difference in log transformed total work of the four recovery modes between the Session 1, and Sessions 2 and 3 is shown in Table 12. There is a possibility that immersion in water post-intervention could be detrimental (90%CL = - 7.7 to 0.9%) to work compared to passive recovery. There is also a possibility that active-immersion could benefit (90%CL = -0.8 to 5.0%) the second jump session.

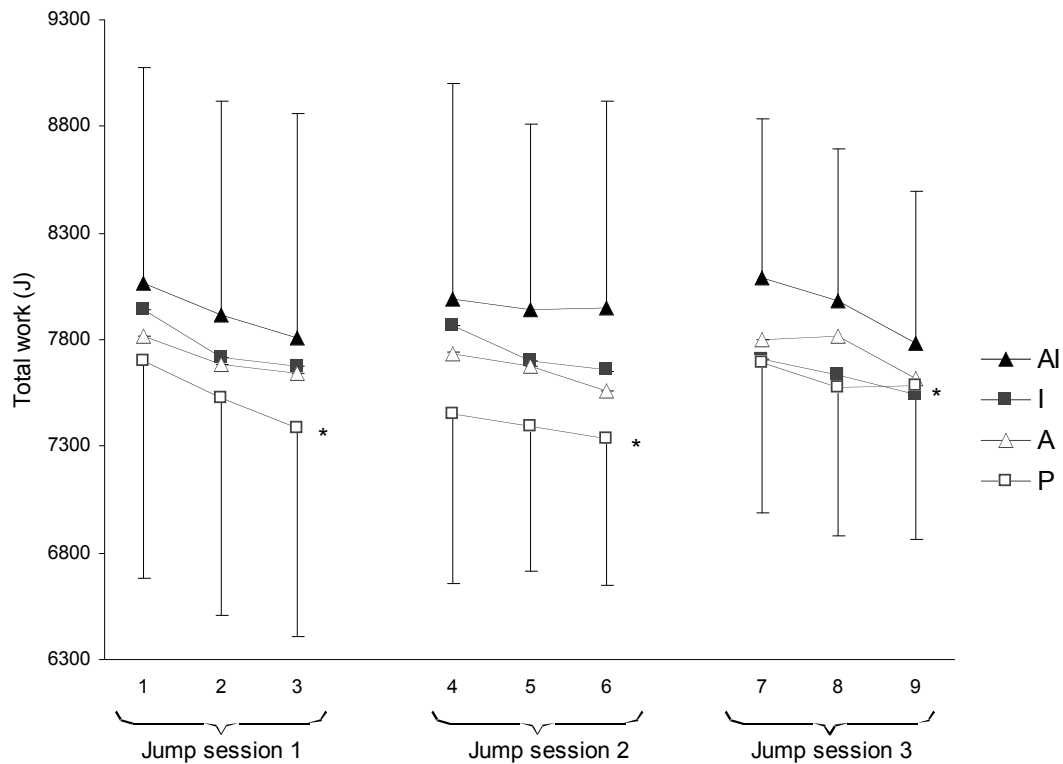


Figure 6: Total work (mean \pm SD) produced during each Jump Session for each recovery mode

Note. AI = active recovery and water immersion; I = water immersion; A = active recovery; P = passive recovery; *Passive recovery significantly different from active-immersion ($p = 0.047$)

Table 12: Mean percent (90%CL) change total work when compared to Jump Session 1 for the different recovery modes.

	Change in performance (%)			
	Passive recovery	Active recovery	Water immersion	Active recovery & water immersion
Jump session 2-1				
mean \pm SD	-1.6 \pm 4.3	-0.4 \pm 3.4	-0.6 \pm 3.6	0.5 \pm 3.2
Difference compared to passive recovery; \pm 90%CL		1.1; \pm 2.3	1.0; \pm 4.0	2.1; \pm 2.9
Practical inference ^a		Unclear	Unclear	Possible benefit
Jump session 3-1				
mean \pm SD	1.5 \pm 5.4	0.8 \pm 5.1	-2.0 \pm 4.4	0.6 \pm 4.3
Difference compared to passive recovery; \pm 90%CL		-0.7; \pm 3.7	-3.4; \pm 4.3	-0.9; \pm 3.0
Practical inference ^a		Unclear	Possible detriment	Unclear

90%CL: add and subtract this number to the difference to obtain the 90% confidence limits for the true difference in effects.

^aBased on a smallest beneficial or detrimental change in performance of 0.2 Cohen's ES.

Blood plasma

Change in blood plasma fraction over a day for each of the recovery modes is illustrated in Figure 7. There was no significant difference in blood plasma fraction between recovery modes. Blood plasma fraction post-intervention was $-2.5 \pm 3.0\%$ (mean \pm SD) lower than pre-jump values and $-2.0 \pm 2.8\%$ lower than post-recovery values.

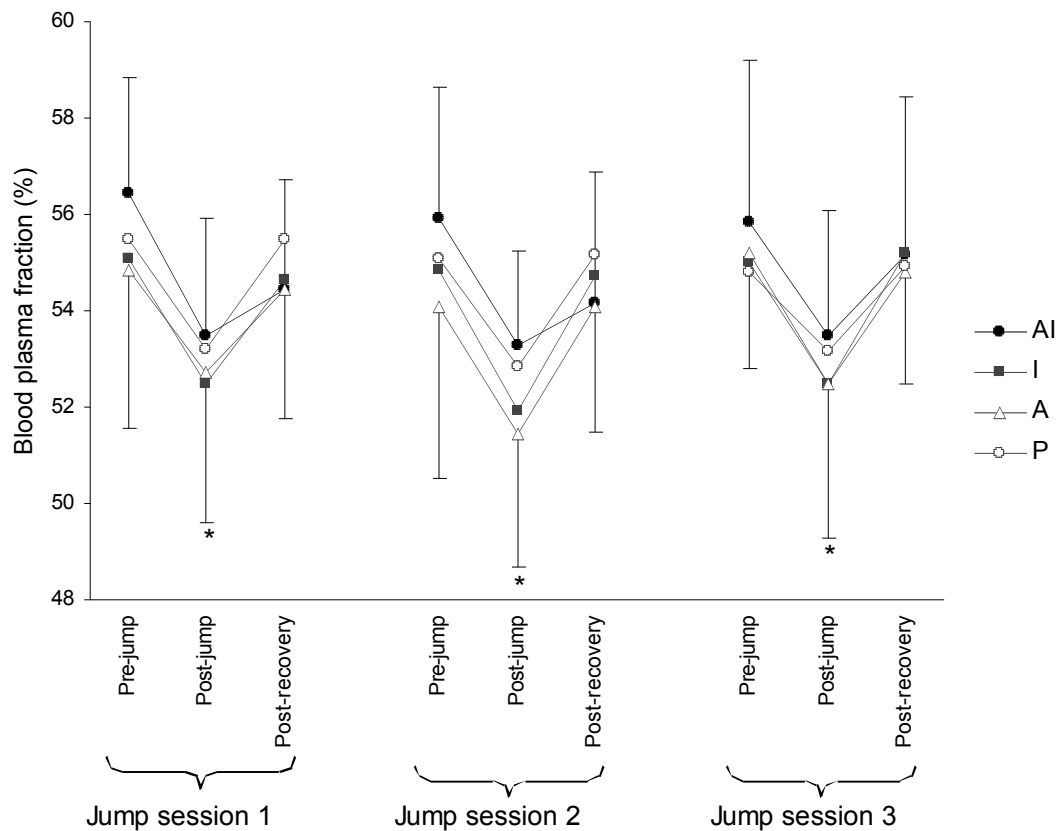


Figure 7: Change in blood plasma fraction (mean \pm SD) throughout the day with different recovery modes

Note. AI = active recovery and water immersion; I = water immersion; A = active recovery; P = passive recovery; *Post-jump significantly different from Pre-jump ($p = 0.00000052$) and Post-recovery ($p = 0.000051$).

Perceived leg fatigue

Change in perceived leg fatigue throughout the jumping sessions is illustrated in Figure 8. No significant differences existed between the different recovery modes. Post-jump fatigue (index = 6.3 ± 1.9) was greater than pre-jump (index = 1.5 ± 1.3) and post-recovery fatigue (index = 2.4 ± 1.4). Post-recovery fatigue for each Session was greater than baseline levels (index = 0.89 ± 1.1) of fatigue. Pre-jump fatigue in Session 3 (index = 2.1 ± 1.2) was also greater than baseline fatigue. Sensation of fatigued increased with each subsequent Jump Sessions increasing from a total mean index of 3.0 ± 2.6 , to 3.4 ± 2.6 , to 3.8 ± 2.5 .

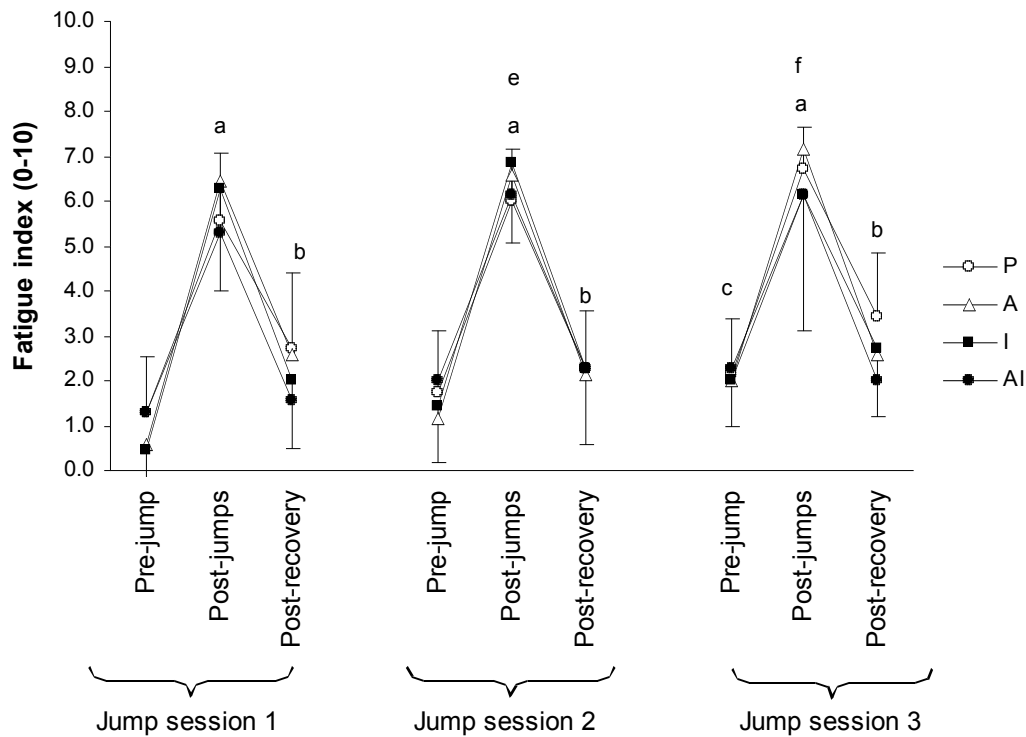


Figure 8: Change in perceived leg fatigue throughout the day with different recovery modes

Note. AI = active recovery and water immersion; I = water immersion; A = active recovery; P = passive recovery; a = significantly greater than Pre-jump and Post-recovery values ($p < 1.7E^{-11}$); b = significantly greater than Pre-jump values ($p < 0.00078$); c = significantly greater than baseline fatigue ($p = 0.0018$); e = significantly greater than Jump session 1 ($p = 0.0093$); f = significantly greater than jump session 2 ($p = 0.0018$).

Discussion

The reliability of displacement, mean force, peak force, and mean power values ($ICC = 0.92 - 0.98, p < 0.001$) from jumping on an instrumented supine squat machine have been reported in previous studies (Cronin *et al.*, 2001a, 2001b, 2003). However, these studies did not consider the reliability of total peak power or total work produced from repetitively jumping on a supine squat machine. Other studies have observed a high reliability (range of values) of repetitive jumping on force plates to assess power (Bosco *et al.*, 1983a; Bosco *et al.*, 1983b; Hoffman & Kang, 2002) and power fatigue (Bosco *et al.*, 1983a). Similar to these studies, repetitive maximal jumping on a supine squat machine was found to be highly reliable in terms of total peak power ($ICC = 0.95$; $CV = 3.1\%$) and total work ($ICC = 0.82$; $CV = 4.7\%$) in this study. Therefore the instrumented supine squat machine was a reliable tool to use in the assessment of jumping kinetics.

Successive sessions of repetitive jumping on the supine squat machine induced increased levels of perceived exertion during exercise and leg muscle fatigue throughout the day, but did not cause any significant performance decrement. Decrement in peak power did occur within each jump session. From the results, it would appear that there were small and non-significant differences between the recovery modes' effect on performance, perceived exertion and fatigue in subsequent bouts of explosive exercise. No significant difference in blood plasma fraction was apparent between the recovery modes indicating that there was no associated improvement or decrement in intravascular fluid return. From the inferential statistics there is a possibility that water immersion could cause a detrimental rather than a beneficial effect on subsequent explosive power, though caution is warranted with this statement. While there were likely differences in kinetics between water immersion and passive recovery test days, these effects were small (Cohen's effect sizes for total peak power and total work in the last jump session was -0.35 and 0.30 respectively). Active recovery and active-immersion effects compared to passive recovery were trivial (Cohen's effect sizes of -0.14 to 0.17).

Based on other research (Thiriet et al., 1993; Vaile et al., 2004), it was expected that immersion in water or active recovery following the jumping protocol would increase the return of fluid into the blood. Along with this fluid return it was postulated that jumping performance would be maintained to a greater degree using active recovery and immersion compared to passive recovery. Additionally, it was expected that fatigue would cause a decrement in performance in each successive jump session, with the degree of the decrement dependent on the recovery mode. However, on the passive recovery day the total peak power and work produced in the last jump session increased compared to the initial session of the day. Performance decreased or increased to a lesser extent with the other recovery modes. These non-significant changes in power and work may be due to natural variation, or the typical error, of the measure. That is, the variability the subjects had in replicating their performance during different test days. Compared to Session 1, change in total peak power and total work in Session 2 and Session 3 for all of the recovery modes ranged from -1.7 to 2.2% and -1.1 to 1.9% respectively whereas the coefficient of variations observed during reliability testing were 3.1% and 4.7% respectively. The coefficient of variation is the typical error of the measure expressed as a percentage change in performance. This means we would expect a typical variation from test to test of $\pm 3.1\%$ and $\pm 4.7\%$ for the power and work produced by the subjects using this protocol. The effect each recovery mode had on performance fell well within this error range. The ‘likely’ detrimental effect of water immersion compared to passive recovery as determined by the inferential statistics may be no more than natural variance of the measurements, which moved in opposite directions (+ or -). The possible inferential effects from active-immersion recovery may also be false artefacts due to the same reasoning.

The major limitation in our study was the small subject number ($n = 7$), though this was minimised with the randomised crossover design. Using Hopkin’s (2001) formula, $n = (1 - r)(32/ES^2)/2$, where r = test-retest correlation and ES = the smallest worthwhile effect (0.2 Cohens), the number of subjects that was required to detect an effect was 20 for total peak power and 72 for total work. The low subject number would reduce the statistical power of the study and our ability to detect significantly small effects. It may be that active recovery, water immersion or a combination of

both provide no greater benefit or detriment to maintaining performance than passive recovery, as the non-significant differences observed in this study was similar to other research (Bond et al., 1991; Coffey et al., 2004; Lattier et al., 2004; Sanders, 1996; Weltman et al., 1979). Other limitations were that the free time between jump sessions was not controlled, neither was the nutritional intake of the subjects. Activity and nutritional intake relied on the subjects' self-standardisation and apart from heart rate and water consumption, was not monitored.

Blood plasma fraction following the explosive jumping was lower than pre-jump values. This blood plasma change is consistent with other research into intense short duration exercise (Collins et al., 1989; Gillen et al., 1991; Knowlton et al., 1987). Unlike Vaile et al. (2004) and Thiriet et al. (1993) no similar return of fluid to the blood (or associated benefit to performance) was observed in our study. One reason for the lack of difference in intravascular fluid return between the different recovery modes may be the duration of recovery time. Research into the physiological response of subjects to head-out immersion has observed no significant increase in blood plasma fraction compared to controls until a period of 10 minutes has passed (Hinghofer-Szalkay et al., 1987; Johansen et al., 1997). Vaile et al. (2004) used 15 minutes of post-exercise immersion and considered daily rather than within-day change in fluid shifts, which may account for the difference when compared to the results of our study. During the study by Thiriet et al. (1993) active recovery between the four bouts of intense exercise was performed for 20 minutes. Blood measures were taken 5-minutes into the recovery period as well as after 20-minutes of recovery. No difference was observed in the blood plasma fraction between recovery modes in the 5-minute measures for the first three repetitions, only in the 20-minute measures. Longer bouts (15 - 20 minutes) of active recovery or water immersion may be required to cause significant fluid shifts in the body and effect subsequent performance.

In addition to longer recovery duration, an increased immersion depth may be a factor in fluid shifts and possibly performance. Our immersion depth was based on those used by Vaile et al. (2004) who observed significant benefits to performance compared to passive recovery and and this depth appears to be used in practice by

many New Zealand sports teams. Physiological studies on water immersion has observed significantly greater rises in central blood volume, increased cardiac output, and lower peripheral resistance with increased depth of immersion (Farhi & Linnarsson, 1977; Gabrielsen et al., 2000; Lollgen et al., 1981), indicative of increased intra-extravascular fluid shifts (Gabrielsen et al., 2002; Hinghofer-Szalkay et al., 1987; Norsk et al., 1985). Chin-out immersion may increase any possible beneficial exercise recovery effects and requires further investigation.

The experimental conditions in this study and in other studies (Bond et al., 1991; Coffey et al., 2004; Lattier et al., 2004; Sanders, 1996; Weltman et al., 1979) that observed non-significant effects are not the same as those conditions encountered in many sports. As an example, past research has used high intensity exercise (normally maximal effort) in their experimental protocols but the exercise duration is generally short, lasting normally less than 5 minutes and rarely over 15 minutes. However, many sports consist of durations of an hour or more. Numerous factors, such as impact during contact sports, duration and intensity of movement, the amount of induced systemic fatigue, etc, may influence whether active recovery or water immersions provide any benefit to exercise recovery. Additionally, it may be that only certain sports, due to their nature, could benefit from active recovery or water immersion. To aid our understanding of recovery, more research incorporating a variety of sport specific exercises along with different recovery protocols is required.

In conclusion, there would appear to be no benefit in performing five minutes of active recovery, water immersion, or a combination of both, after explosive bouts of jumping that is repeated throughout a day. Whether movement of fluid between the intra-extravascular spaces is relevant to performance is unclear. Longer active and immersion recovery times may differ in their influence on performance and fluid shifts and requires further investigation. Research into water immersion and active recovery is in its infancy and our knowledge would benefit from more studies incorporating a variety of exercise and recovery protocols.

CHAPTER 5:

SUMMARY

Active recovery and water immersion may cause similar physiological responses by increasing extra-intravascular fluid shifts and blood flow through the body increasing the metabolism of waste substrates. Therefore compared to passive recovery, active recovery and water immersion post-exercise may enhance the ability of an athlete to recover and increase subsequent performance. An additional benefit of immersion in thermo-neutral water is that no extra energy is expended during recovery conserving glycogen stores, unlike active recovery.

The literature concerning water immersion, active recovery and passive recovery and their ability to maintain exercise performance is limited and inconclusive. Critical examination of studies to date would indicate that there is no detrimental effect from light-intensity ($< 65\% \dot{V}O_{2\max}$) active recovery or water immersion (apart from cryotherapy) on subsequent exercise. Positive benefits, however, are likely to be trivial to small with greater benefit to multiple bouts of exercise or following muscle damaging protocols. Both active recovery and water immersion appear to reduce exercise induce muscle edema. There is a possibility that there is a relationship between the return of post-exercise intramuscular fluid to the blood and performance recovery, though this theory requires verification.

It was hypothesised that post-exercise water immersion and active recovery would have a similar effect, reducing performance decrement and increasing the return to homeostasis of blood plasma levels compared to passive recovery. Our experimental study did not support this hypothesis. Blood plasma, total peak power, total work and perceived muscle fatigue were not significantly different between the recovery modes following repeated bouts of explosive exercise. However, a major limitation was the low subject numbers, which reduced the study's statistical power and the possibility of detecting small effects.

Practical Applications

From our study there would appear to be no benefit to performing five minutes of light active recovery, water immersion, or 10 minutes of both recovery modes following explosive exercise, compared to passive recovery. During a day consisting of repeated bouts of explosive exercise we would recommend that the post-exercise recovery mode reflect the personal preference of the individual athlete.

Future research

From the literature review and the experimental paper, a number of areas require further investigation.

Difference in blood plasma fractions have not been apparent in previous studies until at least 10 minutes of active recovery or water immersion has been performed. Research should consider the effect that recovery duration may have on both blood plasma and subsequent exercise performance.

There is a lack of research into water immersion at constant temperatures and subsequent exercise performance. Possible water temperature effects on exercise recovery needs further study.

Most studies have investigated the acute effects of active recovery or water immersion on exercise performance. The benefits of active recovery or water immersion may occur over a longer cumulative period. Longitudinal studies into overtraining and overreaching and recovery should be considered.

Some researchers have indicated that active recovery and water immersion may be beneficial following exercise that induces muscle damage. These studies require further verification.

Overall, research on the effect of active recovery and water immersion is limited. Research incorporating a variety of post-exercise recovery protocols on different performance parameters would increase the understanding of their practical worth.

REFERENCES

- Abernethy, P., Wilson, G. J., & Logan, P. (1995). Strength and power assessment: Issues, controversies and challenges. *Sports Medicine*, 19(5), 402-417.
- Abramson, D. I., Chu, L. S. W., Tuck, S., Lee, S. W., Richardson, G., & Levin, M. F. (1966). Effect of tissue temperatures and blood flow on motor nerve conduction velocity. *Journal of the American Medical Association*, 198(10), 156-162.
- Ahmaidi, S., Granier, P., Taoutaou, Z., Mercier, J., Dubouchaud, H., & Prefaut, C. (1996). Effects of active recovery on plasma lactate and anaerobic power following repeated intensive exercise. *Medicine and Science in Sports and Exercise*, 28(4), 450-456.
- Arborelius, M., Baildin, U. L., Lilja, B., & Jacobs, D. (1972). Hemodynamic changes in man during immersion with the head above water. *Aerospace Medicine*, 43, 592-598.
- Bardis, C., Kalamara, E., Loucaides, G., Michaelides, M., & Tsaklis, P. (2004). Intramachine and intermachine reproducibility of concentric performance: A study of the con-trex mj and the cybex norm dynamometers. *Isokinetics and Exercise Science*, 12(2), 91-97.
- Barnes, L. (1979). Cryotherapy: Putting injury on ice. *The Physician and Sportsmedicine*, 7(6), 130-136.
- Bender, T., Karagülle, Z., Bálint, G. P., Gutenbrunner, C., Bálint, P. V., & Sukenik, S. (2004). Hydrotherapy, balneotherapy, and spa treatment in pain management. *Rheumatology International*(Accepted 3 May 2004; Published online 15th July 2004), 10 pages. Retrieved 15th August, 2004, from <http://springerlink.metapress.com>.
- Best, T. M., & Garret, W. E. (1993). Warming up and cooling down. In P. A. Renstrom (Ed.), *Sports injuries: Basic principles of prevention and care* (Vol. IV). London: Blackwell Scientific.
- Bigos, S. J., Bowyer, O. R., Braen, G. R., Brown, K., Deyo, R., Haldeman, S., et al. (1994). *Acute low back problems in adults* (Clinical Practice Guideline 14, Agency for Health Care Policy and Research Publication No. 95-0643). Washington, DC: U.S Department of Health and Human Services.
- Blyden, G., Silverstein, F., Epstein, M., Norsk, P., Brenner, B., Nwadike, N., et al. (1989). Lidocaine pharmacokinetics during water immersion in normal humans. *Journal of Applied Physiology*, 66(1), 57-60.
- Bogdanis, G. C., Nevill, M. E., Lakomy, H. K. A., Graham, C. M., & Louis, G. (1996). Effects of active recovery on power output during repeated maximal sprint cycling. *European Journal of Applied Physiology*, 74(5), 461-469.
- Bond, V., Adams, R. G., Tearney, R. J., Gresham, K., & Ruff, W. (1991). Effects of active and passive recovery on lactate removal and subsequent isokinetic muscle function. *Journal of Sports Medicine and Physical Fitness*, 31(3), 357-361.
- Bonde-Petersen, F., Schultz-Pedersen, L., & Dragsted, N. (1992). Peripheral and central blood flow in man during cold, thermoneutral, and hot water immersion. *Aviation, Space and Environmental Medicine*, 63, 346-350.
- Bonen, A., & Belcasro, A. N. (1977). A physiological rational for active recovery exercise. *Canadian Journal of Sport Sciences*, 2(2), 63-65.

- Bonham, S., Talbot, L., Gall, M., Werder, Y., Walters, M., & Petersen, C. (n.d). The effect of a contrast hydrotherapy recovery technique on the post one hour performance of netball players. *Unpublished paper*, School of Physiotherapy, Human Performance Centre, University of Otago, NZ. Accessed 23rd August 2005 from http://www.peakcentreireland.com/docs/research_hotcold.doc.
- Bosco, C., Komi, P. V., Tihanyi, J., Fekete, G., & Apor, P. (1983a). Mechanical power test and fibre composition of human leg extensor muscles. *European Journal of Applied Physiology*, 51, 129-135.
- Bosco, C., Luhtanen, P., & Komi, P. V. (1983b). A simple method for measurement of mechanical power in jumping. *European Journal of Applied Physiology*, 50, 273-282.
- Bove, A. A. (2002). Medical disorders related to diving. *Journal of Intensive Care Medicine*, 17, 75-86.
- Briggs, J. (2001). *Sports therapy: Theoretical and practical thoughts and considerations*. Champaign, IL: Human Kinetics.
- Brukner, P., & Khan, K. (2001). *Clinical sports medicine* (2nd ed.). Sydney: McGraw-Hill.
- Burke, D. G., Holt, L. E., Rasmussen, R. L., MacKinnon, N. C., Vossen, J. F., & Pelham, T. W. (2001). Effects of hot or cold water immersion and modified proprioceptive neuromuscular facilitation flexibility exercise on hamstring length. *Journal of Athletic Training*, 36(1), 16-19.
- Burke, D. G., MacNeil, S. A., Holt, L. E., MacKinnon, N. C., & Rasmussen, R. L. (2003). The effect of hot or cold water immersion on isometric strength training. *Journal of Strength and Conditioning Research*, 14(1), 23-25.
- Cairns, S. P. (2005). Does lactic acid really impair exercise performance? *Unpublished, submitted review article*, Auckland University of Technology.
- Calder, A. (2000). Update on recovery techniques. *Sports Coach*, 23, 19.
- Calder, A. (2003). Recovery strategies for sports performance. *USOC Olympic Coach E-Magazine*, August 1- October 31, 2003, Retrieved 17th August, 2004 from <http://coaching.usolympicteam.com>.
- Cesari, M., Penninx, B. W., Pahor, M., Lauretani, F., Corsi, A. M., Williams, G. R., et al. (2004). Inflammatory markers and physical performance in older persons: The inchiatti study. *The Journals of Gerontology*, 59A(3), 242-248.
- Chaplin, M. (2005, 1 April). Water structure and behaviour. Retrieved 14th March, 2005, from <http://www.lsbu.ac.uk/water/index.html>
- Chleboun, G. S., Howell, J. N., Conatser, R. R., & Giesey, J. J. (1998). Relationship between muscle swelling and stiffness after eccentric exercise. *Medicine and Science in Sports and Exercise*, 30(4), 529-535.
- Clarke, D. H. (1963). Effect of immersion in hot and cold water upon recovery of muscular strength following fatiguing isometric exercises. *Archives of Physical Medical Rehabilitation*, 44, 565-568.
- Clarkson, P. M., & Sayers, S. P. (1999). Etiology of exercise-induced muscle damage. *Canadian Journal of Applied Physiology*, 24(3), 234-248.
- Clover, J. (Ed.). (2001). *Sports medicine essentials: Core concepts in athletic training and fitness instruction*. Orange, CA: Carrer Publishing.
- Cochrane, R. (2004). Alternating hot and cold water immersion for athlete recovery: A review. *Physical Therapy in Sport*, 5, 26-32.
- Coffey, V., Leveritt, M., & Gill, N. (2004). Effect of recovery modality on 4-hour repeated treadmill running performance and changes in physiological variables. *Journal of Science and Medicine in Sport*, 7(1), 1-10.

- Collins, M. A., Cureton, K. J., Hill, D. W., & Ray, C. A. (1989). Relation of plasma volume change to intensity of weight lifting. *Medicine and Science in Sports and Exercise*, 21(2), 178-185.
- Connolly, D. A. J., Brennan, K. M., & Lauzon, C. D. (2003). Effects of active versus passive recovery on power output during repeated bouts of short term, high intensity exercise. *Journal of Sports Science and Medicine*, 2(2), 47-51.
- Considine, W., & Sullivan, W. (1973). Relationship of selected tests of leg strength and leg power on college men. *Research Quarterly for Exercise and Sport*, 44, 404-415.
- Cook, D. B., O'Connor, P. J., Eubanks, S. A., Smith, J. C., & Lee, M. (1997). Naturally occurring muscle pain during exercise: Assessment and experimental evidence. *Medicine and Science in Sports and Exercise*, 29(8), 999-1012.
- Corder, K. P., Potteiger, J., Nau, K. L., Figoni, S. F., & Hershberger, S. L. (2000). Effects of active and passive recovery conditions on blood lactate, rating of perceived exertion, and performance during resistance exercise. *Journal of Strength and Conditioning Research*, 14(2), 151-156.
- Cote, D. J., Prentice, W. E., Hooker, D. N., & Shields, E. W. (1988). Comparison of three treatment procedures for minimizing ankle sprain swelling. *Physical Therapy*, 68(7), 1072-1076.
- Cotts, B. E., Knight, K. L., Myrer, J. W., & Schulthies, S. S. (2004). Contrast-bath therapy and sensation over the anterior talofibular ligament. *Journal of Sport Rehabilitation*, 13, 114-121.
- Craig, A. B., & Dvorak, M. (1968). Thermal regulation of man exercising during water immersion. *Journal of Applied Physiology*, 25(1), 28-35.
- Cronin, J. B., Hing, R. E., & McNair, P. J. (2004). Reliability and validity of a linear position transducer for measuring jump performance. *Journal of Strength and Conditioning Research*, 18(3), 590-593.
- Cronin, J. B., McNair, P. J., & Marshall, R. N. (2001a). Developing explosive power: A comparison of technique and training loads. *Journal of Science and Medicine in Sport*, 4, 59-70.
- Cronin, J. B., McNair, P. J., & Marshall, R. N. (2001b). Relationship between strength qualities and motor skills associated with court performance. *Journal of Human Movement Studies*, 40, 207-224.
- Cronin, J. B., McNair, P. J., & Marshall, R. N. (2003). Lunge performance and its determinants. *Journal of Sports Sciences*, 21(1), 49-57.
- Denton, J. (2005). *The kinematic, kinetic and blood lactate profiles of continuous and intra-set rest loading schemes*. Unpublished master thesis, Auckland University of Technology, Auckland.
- Dugan, E. L., Doyle, T. L. A., Humpries, B. D., Hasson, C. J., & Newton, R. U. (2004). Determining the optimal load for jump squats: A review of methods and calculations. *Journal of Strength and Conditioning Research*, 18(3), 668-674.
- Echt, M., Lange, L., Gauer, O. H., & Dannenburg, H. (1974). Changes of peripheral venous tone and central transmural venous pressure during immersion in a thermo-neutral bath. *Pflugers Archives*, 352, 211-217.
- Enwemeka, C. S., Allen, C., Avila, P., Jason, B., Konrade, J., & Munns, S. (2001). Soft tissue thermodynamics before, during, and after cold pack therapy. *Medicine and Science in Sports and Exercise*, 34(1), 45-50.

- Epstein, M., Levinson, R., & Loutzenhiser, R. (1976). Effects of water immersion on renal hemodynamics in normal man. *Journal of Applied Physiology*, 41(2), 230-233.
- Eston, R., & Peters, D. (1999). Effects of cold water immersion on the symptoms of exercise-induced muscle damage. *Journal of Sports Sciences*, 17, 231-238.
- Fairchild, T. J., Armstrong, A. A., Rao, A., Liu, H., Lawrence, S., & Fournier, P. A. (2003). Glycogen synthesis in muscle fibres during active recovery from intense exercise. *Medicine and Science in Sports and Exercise*, 35(4), 595-602.
- Farhi, L. E., & Linnarsson, D. (1977). Cardiopulmonary readjustment during graded submersion in water at 35°C. *Respiration Physiology*, 30, 35-50.
- Feibel, A., & Fast, A. (1976). Deep heating of joints: A reconsideration. *Archives of Physical Medical Rehabilitation*, 57(11), 513-514.
- Fiscus, K. A., Kaminski, T. W., & Powers, M. E. (2005). Changes in lower-leg blood flow during warm-, cold-, and contrast-water therapy. *Archives of Physical Medical Rehabilitation*, 86(7), 1404-1410.
- Friden, J., & Lieber, R. L. (2001). Eccentric exercise-induced injuries to contractile and cytoskeletal muscle fibre components. *Acta Physiologica Scandinavica*, 171, 321-326.
- Gabrielsen, A., Pump, B., Bie, P., Christensen, N. J., Warberg, J., & Norsk, P. (2002). Atrial distention, haemodilution, and acute control of renin release during water immersion in humans. *Acta Physiologica Scandinavica*, 174, 91-99.
- Gabrielsen, A., Videbæk, R., Johansen, L. B., Warberg, J., Christensen, N. J., Pump, B., et al. (2000). Forearm vascular and endocrine responses to graded water immersion in humans. *Acta Physiologica Scandinavica*, 169, 87-94.
- Gillen, C. M., Lee, R., Mack, G. W., Tomaselli, C. M., Nishiyasu, T., & Nadle, E. R. (1991). Plasma volume expansion in humans after a single intense exercise protocol. *Journal of Applied Physiology*, 71(5), 1914-1920.
- Green, H. J., Thomson, J. A., Ball, M. E., Hughson, R. L., Houston, M. E., & Sharratt, M. T. (1984). Alterations in blood volume following short-term supramaximal exercise. *Journal of Applied Physiology*, 56(1), 145-149.
- Greenleaf, J. E., & Kaciuba-Uscilko, H. (1989). *Acclimatization to heat in humans*. Moffett Field, CA: National Aeronautics and Space Administration, Ames Research Centre.
- Guy, M., Piatt, C., Himmelberg, L., Ballmann, K., & Mayhew, J. L. (1996). Isometric strength measurements as predictors of physical performance in college men. *Iowa Association For Health, Physical Education, Recreation And Dance Journal*, 30(1), Retrieved May 5th, 2005 from <http://www.iowaahperd.org/journal/>.
- Hakumaki, M. O. (1987). Seventy years of the bainbridge reflex. *Acta Physiologica Scandinavica*, 130(2), 177-185.
- Hamlin, M. J. (2001). Are your athletes overtraining? *The New Zealand Coach*, 9(3), 18-20.
- Hamlin, M. J., & Magson, P. (2002, 30th October). *The effects of post-exercise hydrotherapy on blood lactate and performance recovery in netball players*. Paper presented at the 2002 Conferences of the Australasian College of Sports Physicians, Sports Medicine New Zealand, and Sport Science New Zealand, Christchurch, New Zealand.
- Hamlin, M. J., & Sheen, A. M. (2004, August 5th - 11th). *The effect of contrast-temperature water therapy on performance recovery of rugby players*. Paper

- presented at the Pre-Olympic Congress: sport science thru the ages: challenges in the new millenium, Thessaloniki, Greece.
- Hawkins, R. C. (2004). Evaluation of roche accu-chek go and medisense optium blood glucose meters. *Clinica Chimica Acta*, 353(1-2), 127-131.
- Hemmings, B., Smith, M., Graydon, J., & Dyson, R. (2000). Effects of massage on physiological restoration, perceived recovery, and repeated sports performance. *British Journal of Sports Medicine*, 34, 109-114.
- Henricson, A. S., Fredriksson, K., Persson, I., Pereira, R., Rostedt, Y., & Westlin, N. E. (1984). The effect of heat and stretching on the range of hip motion. *Journal of Orthopaedic and Sports Physical Therapy*, 6, 110-115.
- Higgins, D., & Kaminski, T. W. (1998). Contrast therapy does not cause fluctuations in human gastronemius intramuscular temperature. *Journal of Athletic Training*, 33(4), 336-340.
- Hildebrandt, W., Schutze, H., & Stegemann, J. (1992). Cardiovascular limitations of active recovery from strenuous exercise. *European Journal of Applied Physiology*, 64, 250-257.
- Hinghofer-Szalkay, H., Harrison, M. H., & Greenleaf, J. E. (1987). Early fluid and protein shifts in men during water immersion. *European journal of applied physiology and occupational physiology*, 56(6), 673-678.
- Hoffman, J., & Kang, J. (2002). Evaluation of a new anaerobic power testing system. *Journal of Strength and Conditioning Research*, 16(1), 142-148.
- Hopkins, W. G. (1999). How to write a literature review. *Sportscience*, 3(1), Accessed 11th October 2005 from <http://sportsci.org/jour/9901/wghreview.html>.
- Hopkins, W. G. (2000). Reliability from consecutive pairs of trials (excel spreadsheet). In *A new view of statistics*: Sportscience.org, Internet Society for Sportscience. www.sportsci.org/resource/stats/xrely.xls.
- Hopkins, W. G. (2001). Generalizing to a population: Estimating sample size. *A new view of statistics* Retrieved April 20th, 2005, from <http://www.sportsci.org/resource/stats/index.html>
- Hopkins, W. G. (2002a). Probabilities of clinical or practical significance. *Sportscience*, 6, Retrieved 11th October, 2005 from <http://www.sportsci.org/jour/0201/wghprob.htm>.
- Hopkins, W. G. (2002b). Summarizing data: Simple statistics and effect statistics. In W. G. Hopkins (Ed.), *A new view of statistics*: Retrieved 11th October, 2005 from www.sportsci.org/resource/stats/index.html.
- Hopkins, W. G. (2005). A spreadsheet for fully controlled crossovers. *Sportscience*, 9, Retrieved 12th December 2005, from <http://www.sportsci.org/index.html>.
- Howard, R. L., Kraemer, W. J., Stanley, D. C., Armstrong, L. E., & Maresh, C. M. (1994). The effects of cold water immersion on muscle strength. *Journal of Strength and Conditioning Research*, 8(3), 129-133.
- Howatson, G., & Van Someren, K. A. (2003). Ice massage. *Journal of Sports Medicine and Physical Fitness*, 43(4), 500-505.
- Howell, A. K., Gaughan, J. P., Cairns, M. A., Faigenbaum, A. D., & Libonati, J. R. (2001). The effect of muscle hypoperfusion-hyperemia on repetitive vertical jump performance. *Journal of Strength and Conditioning Research*, 15(4), 446-449.
- Johansen, L. B., Jensen, T. U. S., Pump, B., & Norsk, P. (1997). Contribution of abdomen and legs to central blood volume expansion in humans during immersion. *Journal of Applied Physiology*, 83(3), 695-699.

- Johnson, D. J., & Leider, F. E. (1977). Influence of cold bath on maximum handgrip strength. *Perception Motor Skills*, 44(1), 323-326.
- Jonkera, M. J., de Boera, E. M., Adèrb, H. J., & Bezemer, P. D. (2001). The oedema-protective effect of lycra support stockings. *Dermatology*, 203(4), 294-298.
- Kaul, M. P., & Herring, S. A. (1994). Superficial heat and cold: How to maximise the benefits. *The Physician and Sportsmedicine*, 22(12), 65-74.
- Kauppinen, K. (1989). Sauna, shower, and ice water immersion: Physiological responses to brief exposures to heat, cool and cold: Part ii. Circulation. *Arctic Medical Research*, 48(2), 64-74.
- Khosla, S. S., & DuBois, A. B. (1979). Fluid shifts during initial phase of immersion diuresis in man. *Journal of Applied Physiology*, 46(6), 703-708.
- Kiew, O. F., Singh, R., Sirisinghe, R. G., Suen, A. B., & Jamalullail, S. M. S. (2003). Effect of a herbal drink on cycling endurance performance. *Malaysian Journal of Medical Sciences*, 26(1), 78-85.
- Knight, K. L., & Londeree, B. R. (1980). Comparison of blood flow in the ankle of uninjured subjects during therapeutic applications of heat, cold, and exercise. *Medicine and Science in Sports and Exercise*, 12(3), 76-80.
- Knowlton, R. G., Hetzler, R. K., Kaminsky, L. A., & Morrison, J. J. (1987). Plasma volume changes and cardiovascular responses associated with weight lifting. *Medicine and Science in Sports and Exercise*, 19(5), 464-468.
- Koryak, Y. (2002). "dry" immersion induces neural and contractile adaptations in the human triceps surae muscle. *Environmental Medicine*, 46(1-2), 17-27.
- Kuligowski, L. A., Lephart, S. M., Giannantonio, F. P., & Blanc, R. O. (1998). Effects of whirlpool therapy on the signs and symptoms of delayed-onset muscle soreness. *Journal of Athletic Training*, 33(3), 222-228.
- Lane, K. N., & Wenger, H. A. (2004). Effect of selected recovery conditions on performance of repeated bouts of intermittent cycling separated by 24 hours. *Journal of Strength and Conditioning Research*, 18(4), 855-860.
- Lassiter, W. E., & Gottachalk, C. W. (1980). Volume and components of the body fluids. In V. B. Mountcastle (Ed.), *Medical physiology* (Vol. 2). St Louis: C. V. Moby.
- Lattier, G., Millet, G. Y., Martin, A., & Martin, V. (2004). Fatigue and recovery after high-intensity exercise: Part ii: Recovery interventions. *International Journal of Sports Medicine*, 25, 509-515.
- Lecomte, J. M., Lacroix, V. J., & Montgomery, D. L. (1998). A randomized controlled trial of the effect of naproxen on delayed onset muscle soreness and muscle strength. *Clinical Journal of Sport Medicine*, 8(2), 82-87.
- Lee, D. T., Toner, M. M., McArdle, W. D., Vrabas, I. S., & Pandolf, K. B. (1997). Thermal and metabolic responses to cold-water immersion at knee, hip and shoulder levels. *Journal of Applied Physiology*, 82(5), 1523-1530.
- Lippman, S. M., Winn, L., Grumet, F. C., & Levitt, L. J. (1987). Evans' syndrome as a presenting manifestation of atypical paroxysmal cold hemoglobinuria. *American Journal of Medicine*, 82(5), 1065-1072.
- Lloyd, E. L. (1994). Abc of sports medicine: Temperature and performance i: Cold. *British Medical Journal*, 309, 531-534.
- Lollgen, H., Nieding, G. V., Koppenhagen, K., Kersting, F., & Just, H. (1981). Hemodynamic response to graded water immersion. *Klin Wochenschr*, 59, 623-628.
- Low, J., & Reed, A. (1994). *Electrotherapy explained: Principles and practice* (2nd ed.). Oxford: Butterworth and Heinemann.

- Magness, J., Garrett, T. R., & Erickson, D. I. (1970). Swelling of the upper extremity during whirlpool baths. *Archives of Physical Medical Rehabilitation*, 51(5), 297-299.
- McEniery, C. M., Jenkins, D. G., & Barnett, C. (1997). The relationship between plasma potassium concentration and muscle torque during recovery following intense exercise. *European Journal of Applied Physiology*, 75, 462-466.
- Mero, A., Luhtanen, P., Viitasalo, T., & Komi, P. V. (1981). Relationship between maximal running velocity, muscle fibre characteristics, force production and force relaxation of sprinters. *Scandinavian Journal of Sports Science*, 3(1), 16-22.
- Michlovitz, S. L. (Ed.). (1996). *Thermal agents in rehabilitation* (3rd ed.). Philadelphia: F. A. Davis company.
- Miles, D. S., Sawka, M. N., Glaser, R. M., & Petrofsky, J. S. (1983). Plasma volume shift during progressive arm and leg exercise. *Journal of Applied Physiology*, 54, 491-495.
- Milnor, W. R. (1980). Capillaries and lymphatic vessels. In V. B. Mountcastle (Ed.), *Medical physiology* (Vol. 2). St Louis: C. V. Moby.
- Mishra, D. K., Friden, J., Schmitz, M. C., & Lieber, R. L. (1995). Anti-inflammatory medication after muscle injury. A treatment resulting in short-term improvement but subsequent loss of muscle function. *Journal of Bone and Joint Surgery (America)*, 77(10), 1510-1519.
- Mohsenin, V., & Gonzalez, R. R. (1984). Tissue pressure and plasma oncotic pressure during exercise. *Journal of Applied Physiology, Respiration, Environment and Exercise physiology*, 56(1), 102-108.
- Monedero, J., & Donne, B. (2000). Effects of recovery interventions on lactate removal and subsequent performance. *International Journal of Sports Medicine*, 21, 593-597.
- Murphy, A. J., Wilson, D. G., & Pryor, J. F. (1994). The use of isoinertial force mass relationship in the prediction of dynamic human performance. *European Journal of Applied Physiology*, 69(3), 250-257.
- Myrer, J. W., Draper, D. O., & Durrant, E. (1994). Contrast therapy and intramuscular temperature in the human leg. *Journal of Athletic Training*, 29(4), 318-324.
- Myrer, J. W., Measom, G., Durrant, E., & Fellingham, G. W. (1997). Cold- and hot-pack contrast therapy: Subcutaneous and intramuscular temperature change. *Journal of Athletic Training*, 32(3), 238-241.
- Nadler, S. F., Steiner, D. J., Erasala, G. N., Hengehold, D. A., Abeln, S. B., & Weingand, K. W. (2003a). Continuous low-level heatwrap therapy for treating acute non-specific low back pain. *Archives of Physical Medical Rehabilitation*, 84, 329-334.
- Nadler, S. F., Steiner, D. J., Erasala, G. N., Hengehold, D. A., Hinkle, R. T., Goodale, M. B., et al. (2002). Continuous low-level heat wrap therapy provides more efficacy than ibuprofen and acetaminophen for acute low back pain. *Spine*, 27(10), 1012-1017.
- Nadler, S. F., Steiner, D. J., Petty, S. R., Erasala, G. N., Hengehold, D. A., & Weingand, K. W. (2003b). Overnight use of continuous low-level heatwrap therapy for relief of low back pain. *Archives of Physical Medical Rehabilitation*, 84, 333-342.
- Nagasawa, Y., Komori, S., Sato, M., Tsuboi, Y., Umetani, K., Watanabe, Y., et al. (2001). Effects of hot bath immersion on autonomic activity and

- hemodynamics: Comparison of the elderly patient and the healthy young. *Japanese Circulation Journal*, 65(7), 587-592.
- Nakamura, K., Takahashi, H., Shimai, S., & Tanaka, M. (1996). Effects of immersion in tepid bath water on recovery from fatigue after submaximal exercise in man. *Ergonomics*, 39(2), 257-266.
- Netball New Zealand. (2004). Recovery strategies for a tournament environment. Retrieved 1st May, 2005, from http://images.tvnz.co.nz/netballnz/thegame/tournament_recovery.pdf
- Newton, R. U., Kraemer, W. J., Hakkinen, K., Humpries, B. J., & Murphy, A. J. (1996). Kinematics, kinetics, and muscle activation during explosive upper body movements. *Journal of Applied Biomechanics*, 12, 31-43.
- Norsk, P., Bonde-Petersen, F., & Warberg, J. (1985). Central venous pressure and plasma arginine vasopressin during water immersion in man. *European Journal of Applied Physiology*, 54, 71-78.
- Northoff, H., Berg, A., & Weinstock, C. (1998). Similarities and differences of the immune response to exercise and trauma: The ifn- γ concept. *Canadian Journal of Physiology and Pharmacology*, 76(5), 497-504.
- Nosaka, K., & Clarkson, P. M. (1994). Effect of eccentric exercise on plasma activities previously elevated by eccentric exercise. *European Journal of Applied Physiology*, 69, 492-497.
- Pappenheimer, J. R. (1953). Passage of molecules through capillary walls. *Physiological Review*, 33(3), 387-423.
- Park, K. S., Choi, J. K., & Park, Y. S. (1999). Cardiovascular regulation during water immersion. *Applied Human Science*, 18(6), 233-241.
- Partsch, H., Winiger, J., & Lun, B. (2004). Compression stockings reduce occupational leg swelling. *Dermatologic Surgery*, 30(5), 737-743.
- Paz, J. C., & West, M. P. (2002). *Acute care handbook for physical therapists* (2 ed.). Boston: Butterworth Heinemann.
- Piatt, C., Ballmann, K., Scanlan, J., Guy, M., & Mayhew, J. L. (1999). Relationship between isometric strength measurements and physical performance in college women. *Iowa Association For Health, Physical Education, Recreation And Dance Journal*(Spring 1999), Retrieved May 5th, 2005 from <http://www.iowaahperd.org/journal/>.
- Ploutz-Snyder, L. L., Convertino, V. A., & Dudley, G. A. (1995). Resistance exercise-induced fluid shifts: Change in active muscle size and plasma volume. *American Journal of Physiology*, 269, R536-R543.
- Prentice, W. E. (1982). An electromyographic analysis of the effectiveness of heat and cold and stretching for inducing relaxation in injured muscle. *Journal of Orthopaedic and Sports Physical Therapy*, 3(3), 133-140.
- Pöyhönen, T., & Avela, J. (2002). Effect of head-out water immersion on neuromuscular function of the plantarflexor muscles. *Aviation, Space and Environmental Medicine*, 73(12), 1215-1218.
- Pöyhönen, T., Keskinen, K. L., Hautala, A., Savolainen, J., & Mäkiä, E. (1999). Human isometric force production and electromyogram activity of knee extensor muscles in water and on dry land. *European Journal of Applied Physiology and Occupational Physiology*, 80(1), 52-56.
- Rawson, E. S., Gunn, B., & Clarkson, P. M. (2001). The effects of creatine supplementation on exercise-induced muscle damage. *Journal of Strength and Conditioning Research*, 15(2), 178-184.

- Robertson, R. J., Goss, F. L., Dube, J., Rutkowski, J., Dupain, M., Brennan, C., et al. (2004). Validation of the adult omni scale of perceived exertion for cycle ergometer exercise. *Medicine and Science in Sports and Exercise*, 36(1), 102-108.
- Robertson, R. J., Goss, F. L., Rutkowski, J., Lenz, B., Dixon, C., Timmer, J., et al. (2003). Concurrent validation of the omni perceived exertion scale for resistance exercise. *Medicine and Science in Sports and Exercise*, 35(2), 333-341.
- Robertson, V. J., & Duck, F. A. (2001). A review of therapeutic ultrasound: Biophysical effects. *Physical Therapy*, 81, 1351-1358.
- Rontoyannis, G. P. (1988). Lactate elimination from the blood during active recovery. *Journal of Sports Medicine*, 28, 115-123.
- Rutkove, S. B. (2001). Effects of temperature on neuromuscular electrophysiology. *Muscle & Nerve*, 24, 867-882.
- Sanders, J. (1996). *Effect of contrast-temperature immersion on recovery from short-duration intense exercise*. Unpublished thesis, University of Canberra, Canberra.
- Sauls, J. (1999). Efficacy of cold for pain: Fact or fallacy? *The Online Journal of Knowledge Synthesis for Nursing*, 6(8), Retrieved October 15th, 2002, from <http://www.stti.iupui.edu/library/ojksn/articles/060008.pdf>.
- Sawyer, P. C., Unl, T. L., Mattacola, C. G., Johnson, D. L., & Yates, J. W. (2003). Effects of moist heat on hamstring flexibility and muscle temperature. *Journal of Strength and Conditioning Research*, 17(2), 285-290.
- Sayers, S. P., Clarkson, P. M., & Lee, J. (2000). Activity and immobilization after eccentric exercise: I. Recovery of muscle function. *Medicine and Science in Sports and Exercise*, 32(9), 1587-1592.
- Shephard, R. J., & Shek, P. N. (1998). Immune responses to inflammation and trauma: A physical training model. *Canadian Journal of Physiology and Pharmacology*, 76(5), 469-472.
- Shiraishi, M., Schou, M., Gybel, M., Christensen, N. J., & Norsk, P. (2002). Comparisons of acute cardiovascular responses to water immersion and head-down tilt in humans. *Journal of Applied Physiology*, 92, 264-268.
- Signorile, J. F., Ingalls, C., & Tremblay, L. M. (1993). The effects of active and passive recovery on short-term, high intensity power output. *Canadian Journal of Applied Physiology*, 18(1), 31-42.
- Skurvydas, A., Jascaninas, J., & Zachovajevs, P. (2000). Changes in height of jump, maximal voluntary contraction force and low-frequency fatigue after 100 intermittent or continuous jumps with maximal intensity. *Acta Physiologica Scandinavica*, 169, 55-62.
- Smith, L. L. (1990). Acute inflammation: The underlying mechanism in delayed onset muscle soreness. *Medicine and Science in Sports and Exercise*, 23(5), 542-551.
- Sramek, P., Simeckova, M., Jansky, L., Savlikova, J., & Vybiral, S. (2000). Human physiological responses to immersion into water of different temperatures. *European Journal of Applied Physiology*, 81, 436-442.
- Stanton, D. B., Bear-Lehman, J., Graziano, M., & Ryan, C. (2003). Contrast baths: What do we know about their use? *Journal of Hand Therapy*, 16(4), 343-346.
- Starkie, R. L., Hargreaves, M., Lambert, D. L., Proietto, J., & Febbraio, M. A. (1999). Effect of temperature on muscle metabolism during submaximal exercise in humans. *Experimental Physiology*, 84(4), 775-784.

- Stocks, J. M., Patterson, M. J., Hyde, D. E., Jenkins, A. B., Mittleman, K. D., & Taylor, N. A. S. (2004). Effects of immersion water temperature on whole-body fluid distribution in humans. *Acta Physiologica Scandinavica*, 182, 3-10.
- Stuart, G. R., Hopkins, W. G., Cook, C., & Cairns, S. P. (2005). Multiple effects of caffeine on simulated high-intensity team-spot performance. *Medicine and Science in Sports and Exercise*, 37(11), In print.
- Suzuki, M., Umeda, T., Nakaji, S., Shimoyama, T., Mashiko, T., & Sugawara, K. (2004). Effect of incorporating low intensity exercise into the recovery period after a rugby match. *Journal of Sports Medicine*, 38, 436-440.
- Taylor, B. F., Waring, C. A., & Brashear, T. A. (1995). The effects of therapeutic application of heat or cold followed by static stretch on hamstring muscle length. *Journal of Orthopaedic and Sports Physical Therapy*, 21(5), 283-286.
- Terrell, T., Hough, D. O., & Alexander, R. (1996). Identifying exercise allergies: Exercise-induced anaphylaxis and cholinergic urticaria. *The Physician and Sportsmedicine*, 24(11), Accessed 17th July 2005 from www.physsportsmed.com/issues/1996/2011_2096/terrell.htm.
- Thiriet, P., Gozal, D., Wouassi, D., Oumarou, T., Gelas, H., & Lacour, J. R. (1993). The effects of various recovery modalities on subsequent performance, in consecutive supramaximal exercise. *Journal of Sports Medicine and Physical Fitness*, 33(2), 118-129.
- Tiidus, P. M. (1997). Manual massage and recovery of muscle function following exercise: A literature review. *Journal of Orthopaedic and Sports Physical Therapy*, 25(2), 107-112.
- Tiidus, P. M. (1998). Radical species in inflammation and overtraining. *Canadian Journal of Physiology and Pharmacology*, 76(5), 533-538.
- Tiidus, P. M., Dawson, K. A., Dawson, L., Roefs, A., & Bombardier, E. (2004). Massage does not influence muscle soreness or strength recovery following a half-marathon. *Medicine and Science in Sports and Exercise*, 36(5 suppl.), S15-S16.
- Tikuisis, P., Jacobs, I., Moroz, D., Vallerand, A. L., & Martineau, L. (2000). Comparison of thermoregulatory responses between men and women immersed in cold water. *Journal of Applied Physiology*, 89, 1410-1411.
- Tomasik, M. (1983). Effect of hydromassage on changes in blood electrolyte and lactic acid levels and haematocrit value after maximal effort. *Acta Physiologica Polonica*, 34(2), 257-261.
- Toner, M. M., Sawka, M. M., Holden, W. L., & Pandolf, K. B. (1986). Effects of body mass and morphology on thermal response in water. *Journal of Applied Physiology*, 60(2), 521-525.
- Tonnessen, D. (1999). Strains and sprains: Hot and cold therapy. In H. E. Aldred (Ed.), *Sports injuries sourcebook: Basic consumer health information about common sports injuries*. Detroit, MI: Omnigraphics.
- Turner, B., Pennefather, J., & Edmonds, C. (1980). Cardiovascular effects of hot water immersion (suicide soup). *Medical Journal of Australia*, 2(1), 39-40.
- Vaile, J. (2003). *The effect of recovery strategy on symptoms of delayed onset of muscle soreness (doms)*. Unpublished Report completed in partial fulfilment of the Bachelor of Sport and Exercise Science Degree, Waikato Institute of Technology, Hamilton, New Zealand.
- Vaile, J., Blazeovich, A. J., & Gill, N. (2004). *The effect of contrast therapy on symptoms of delayed onset muscle soreness*. Unpublished paper, Waikato Institute of Technology, Hamilton, New Zealand.

- Viitasalo, J. T., Niemela, K., Kaappola, R., Korjus, T., Levola, M., Mononen, H. V., et al. (1995). Warm underwater water-jet massage improves recovery from intense physical exercise. *European Journal of Applied Physiology*, 71, 431-438.
- Wallace, L., Knortz, K., & Esterson, P. (1979). Immediate care of ankle injuries. *Journal of Orthopaedic and Sports Physical Therapy*, 1, 46-50.
- Walsh, M. T. (1996). Hydrotherapy: The use of water as a therapeutic agent. In S. L. Michlovitz (Ed.), *Thermal agents in rehabilitation* (3rd ed.). Philadelphia: F. A. Davis company.
- Warren, G. L., Lowe, D. A., & Armstrong, R. B. (1999). Measurement tools used in the study of eccentric contraction-induced injury. *Sports Medicine*, 27(1), 43-59.
- Washington, L. L., Gibson, S. J., & Helme, R. D. (2000). Age-related differences in the endogenous analgesic response to repeated cold water immersion in human volunteers. *Pain*, 89, 89-96.
- Watenpaugh, D. E., Pump, B., Bie, P., & Norsk, P. (2000). Does gender influence human cardiovascular and renal responses to water immersion? *Journal of Applied Physiology*, 89, 621-628.
- Weerapong, P. (2005). *Preexercise strategies: The effects of warm-up, stretching, and massage on symptoms of eccentric exercise-induced muscle damage and performance*. Unpublished PhD, Auckland University of Technology, Auckland.
- Weltman, A., Stamford, B. A., & Fulco, C. (1979). Recovery from maximal effort exercise: Lactate disappearance and subsequent performance. *Journal of Applied Physiology*, 47(4), 677-682.
- Weltman, A., Stamford, B. A., Moffatt, R. J., & Katch, V. L. (1977). Exercise recovery, lactate removal, and subsequent high intensity exercise performance. *Research Quarterly*, 48(4), 786-796.
- Weston, C. E. M., O'Hare, J. P., Evans, J. M., & Corral, R. J. M. (1987). Haemodynamic changes in man during immersion in water at different temperatures. *Clinical Science*, 73, 613-616.
- Whitney, J. D., & Wickline, M. M. (2003). Treating chronic and acute wounds with warming: Review of the science and practice implications. *Journal of Wound, Ostomy, and Continence Nursing*, 30(4), 199-209.
- Wilk, K. E., Macrina, L. C., Reinold, M. M., & Schram, J. (2004). Team physician's corner: Common modalities in sports medicine. *SportsMedicine Update*, July-August, Accessed 17th July 2005 from www.sportsmed.org.
- Wittmers, L., & Savage, M. V. (2001). Cold water immersion. In C. B. Wenger & R. S. Pozos (Eds.), *Medical aspects of harsh environments* (Vol. 1): Office of the Surgeon General, Department of the Army, United States of America.
- Wyper, D. J., & McNiven, D. R. (1976). Effects of some physiotherapeutic agents on skeletal muscle blood flow. *Physiotherapy*, 62, 83-85.
- Yona, M. (1997). Effects of cold stimulation of human skin on motor unit activity. *Japanese Journal of Physiology*, 47(4), 341-348.
- Young, W. B., Pryor, J. F., & Wilson, G. J. (1995). Effects of instruction on characteristics of countermovement and drop jump performance. *Journal of Strength and Conditioning Research*, 9(4), 232-236.
- Yun, S. H., Choi, J. K., & Park, Y. S. (2004). Cardiovascular responses to head-out water immersion in Korean women breath-hold divers. *European Journal of Applied Physiology*, 91, 708-711.

Zuluaga, M., Briggs, C., Carlisle, J., McDonald, V., McMeeken, J., Nickson, W., et al. (Eds.). (1995). *Sports physiotherapy: Applied science and practice*. South Melbourne, Australia: Churchill Livingstone.

APPENDICES

Consent to Participation in Research



Title of Project: Water immersion, active and passive recovery and their effects on repeated performance and blood plasma

Project Supervisor: John Cronin

Researcher: Ian Wilcock

-
- I have read and understood the information provided about this research project (Information Sheet dated 7th February 2005.)
 - I have had an opportunity to ask questions and to have them answered.
 - I understand that I may withdraw myself or any information that I have provided for this project at any time prior to completion of data collection, without being disadvantaged in any way.
 - I agree to take part in this research.
 - I wish to receive a copy of the report from the research:

tick one: Yes ☐ No ☐

Participant signature:

Participant name:

Participant Contact Details:

.....
.....
.....
.....

Date:

Approved by the Auckland University of Technology Ethics Committee on the 14th March 2005. AUTEK Reference number 05/31A

Participant Information Sheet



Date Information Sheet Produced: 07 February 2005

Project Title

Water immersion, active and passive recovery and their effects on repeated performance and blood plasma.

Invitation

You are invited to participate in this study. Your participation in this study is completely voluntary and can be declined at any time without you giving a reason or being disadvantaged in any manner. You may also withdraw any information you have provided at any time.

What is the purpose of the study?

Water immersion has been gaining popularity as a method of recovery, though there is scarce amount of research into its ability as a recovery agent. The purpose of this study is to determine the effectiveness of different recovery strategies on power and blood plasma. This information ultimately is to help find greater post-game/training restorative practices for coaches and athletes.

How are people chosen to be asked to be part of the study?

Any male who engages regularly in sport or exercise, is uninjured and over 20yrs of age, is invited to be part of this study.

What happens in the study?

Pre-testing, height, weight and leg skinfold measurements will be taken during a familiarisation session within a week of the first testing session. The exercise protocol consists of subjects performing three sets of twenty jumps for maximum displacement on a supine squat machine. Following the power protocol subjects would perform either; (a) active recovery (5 min cycling on a Monark cycle ergometer) followed by passive rest (5 min); (b) active recovery followed by water immersion (5 min) of the lower body; (c) water immersion followed by passive rest (d) or passive rest. Two hours later the subject would repeat the three sets of jumps, and the same recovery protocol. A further two hours later another three sets of jumps is to be performed. During testing blood glucose and plasma samples are taken. Blood glucose involves a pin prick of the finger and a drop of blood analysed with a hand held meter. Blood plasma requires the milking of 75 μ L of blood and would be measured pre-test, after the final set of jumps and immediately post-recovery. Subjects would repeat the test a week later using a different recovery protocol. In total one familiarisation and four test sessions (a week apart) are required.

What are the discomforts and risks?

As the power test is repeated jumping, subjects may feel exhaustion and discomfort from the test, and there is chance of soft tissue injury. Subjects may also feel pain from the pin pricks during blood sampling.

How will these discomforts and risks be alleviated?

The principal investigator is trained in first aid. Physiotherapists are also located 30-m from the testing venue.

What are the benefits?

The results of this study may have benefits to you as an individual and to the sport in general. The benefits to you may include:

- Determining your leg power and power decrement from repeated jumping.

The results of this study may also have many benefits to sport. These may include the following:

- Determining the effectiveness of water immersion on power and blood plasma shifts.
- Determining more effective recovery strategies for coaches and athletes.

Oral feedback will be presented to participants about their leg power and blood plasma response during the study. Written feedback about the research will be available in the thesis.

What compensation is available for injury or negligence?

If an injury occurs compensation is available through the Accident Compensation Corporation within its normal limitations.

How will my privacy be protected?

After collection of the data, your data will be allocated an identification code which all your data will then be analysed under. Data sheets will be locked in an office filing cabinet. The researcher and project supervisor will be the only people to have access to the data. No subjects will be identified unless prior permission from the subject is gained.

What are the costs of participating in the project? (including time)

There are no costs involved in the participation in this study, except your time commitment. Total time is expected to be approximately 8 hours over 4 weeks (a 20 min familiarisation session and 12 x 30-40 min test sessions)

Opportunity to consider invitation

You will have some time (14 days) to consider your participation in this study. A set date for data collection to begin will be discussed and individuals invited will be notified. If you participate in this study you may withdraw at any time with giving reason.

Opportunity to receive feedback on results of research

Immediate oral feedback will be available on your leg power and blood plasma profile.

Participant Concerns

Any concerns regarding the nature of this project should be notified in the first instance to the Project Supervisor.

Project Supervisor Contact Details:

Dr John Cronin
Auckland University of Technology
Private Bag 92006
Auckland 1020
Ph 917 9999 Ext. 7353
john.cronin@aut.ac.nz

Researcher Contact Details:

Ian Wilcock
Auckland University of Technology
Private Bag 92006
Auckland 1020
Ph 917 9999 Ext. 7119
021 359979
iwilcock@aut.ac.nz

Concerns regarding the conduct of the research should be notified to the Executive Secretary, AUTEK, Madeline Banda, madeline.banda@aut.ac.nz, 917 9999 ext 8044.

Approved by the Auckland University of Technology Ethics Committee on 22 March 2005
AUTEK Reference number 05/31

Appendix C: Ethical approval



MEMORANDUM

Academic Services

To: John Cronin
From: **Madeline Banda**
Date: 22 March 2005
Subject: Ethics Application Number 05/31 **Hydrotherapy, active and passive recovery effects on repeated power performance and blood lactate clearance**

Dear John

Thank you for providing written evidence as requested. I am pleased to advise that it satisfies the points raised by the Auckland University of Technology Ethics Committee (AUTEC) at their meeting on 14 March 2005. Your ethics application is now approved for a period of three years until 22 March 2008.

I advise that as part of the ethics approval process, you are required to submit to AUTEC the following:

- A brief annual progress report indicating compliance with the ethical approval given using form EA2 which is available online at http://www.aut.ac.nz/research_showcase/pdf/appendix_g.doc, including a request for extension of the approval if the project will not be completed by the above expiry date;
- A brief report on the status of the project using form EA3 which is available online at http://www.aut.ac.nz/research_showcase/pdf/appendix_h.doc. This report is to be submitted either when the approval expires on 22 March 2008 or on completion of the project, whichever comes sooner;

You are reminded that, as applicant, you are responsible for ensuring that any research undertaken under this approval is carried out within the parameters approved for your application. Any change to the research outside the parameters of this approval must be submitted to AUTEC for approval before that change is implemented.

Please note that AUTEC grants ethical approval only. If you require management approval from an institution or organisation for your research, then you will need to make the arrangements necessary to obtain this.

To enable us to provide you with efficient service, we ask that you use the application number and study title in all written and verbal correspondence with us. Should you have any further enquiries regarding this matter, you are welcome to contact Charles Grinter, Ethics Coordinator, by email at charles.grinter@aut.ac.nz or by telephone on 917 9999 at extension 8860.

On behalf of the Committee and myself, I wish you success with your research and look forward to reading about it in your reports.

Yours sincerely



Madeline Banda

Executive Secretary

Auckland University of Technology Ethics Committee

Cc: Ian Wilcock iwilcock@aut.ac.nz «Copy_Correspondence_to»

From the desk of ...
Madeline Banda
Academic Services
Student Services

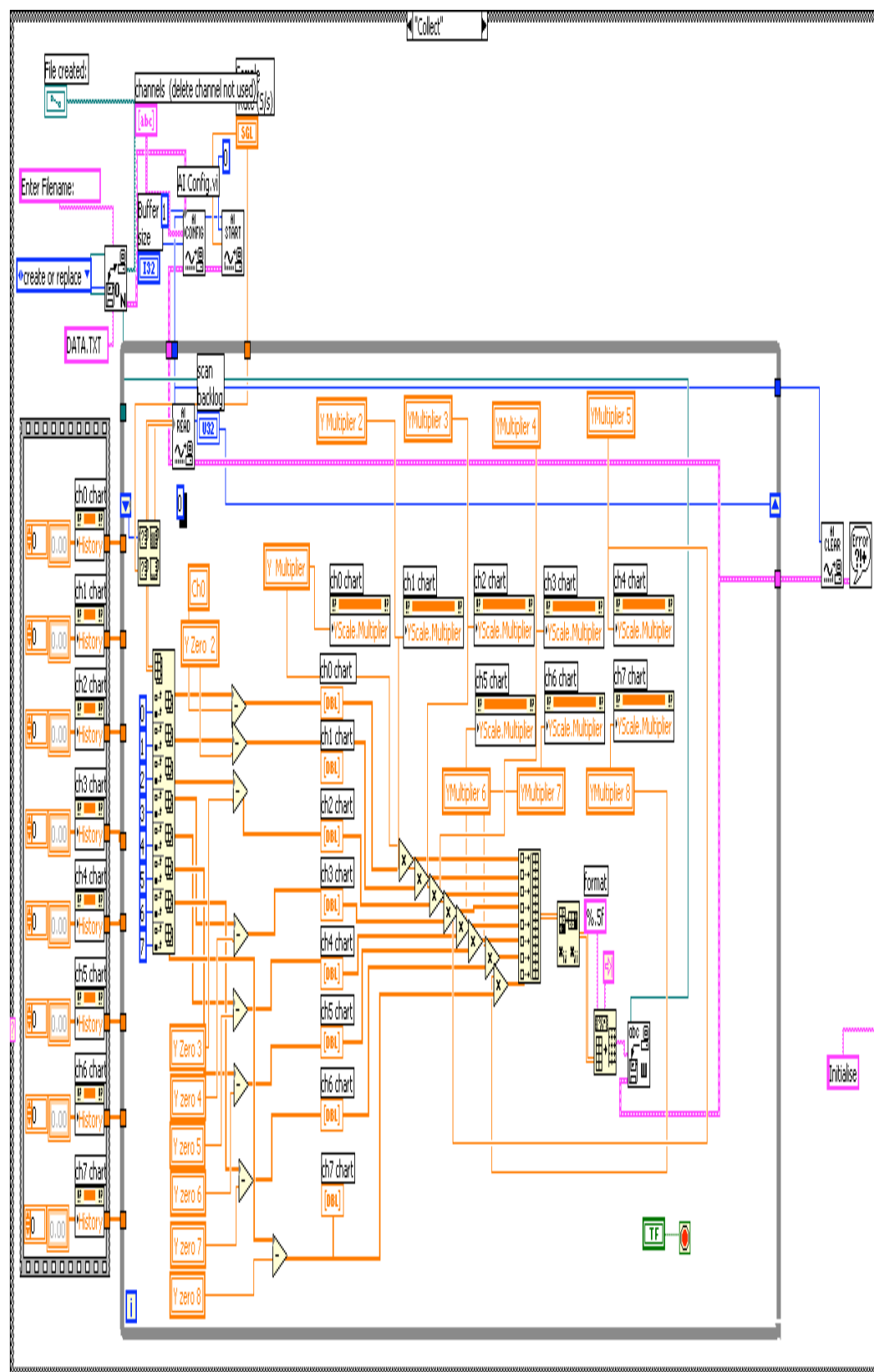
Private Bag 92006, Auckland 1020
New Zealand
E-mail: madeline.banda@aut.ac.nz

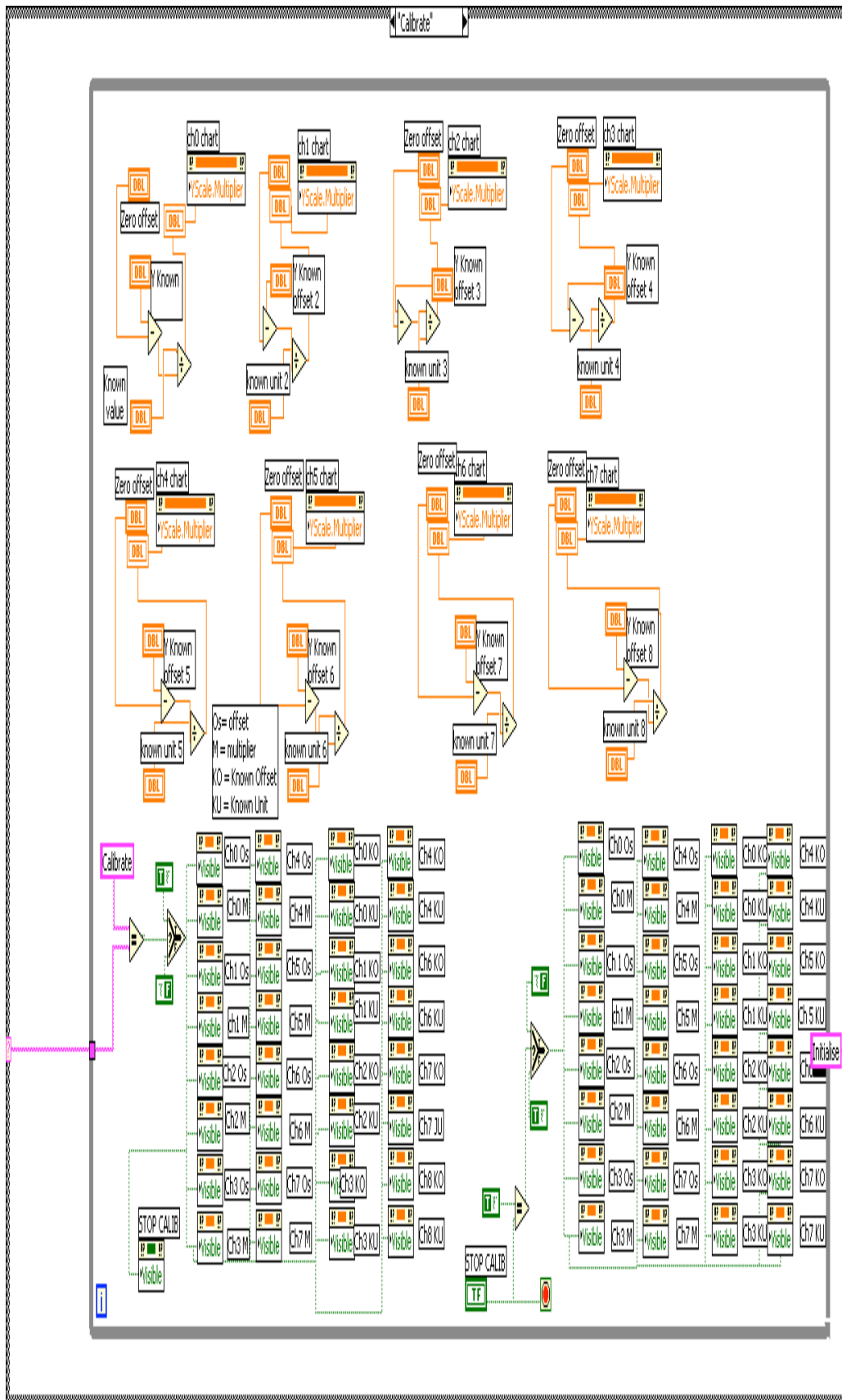
Tel: 64 9 917 9999
ext 8044
Fax: 64 9 917 9812

Appendix D: Labview data collection program (front panel)



Appendix E: Labview data collection program (back panel)





Appendix F: Validity of the data from Labview analysis program

Validity is the degree to which a test measures what it is intended to measure. One type of validity is construct validity. Construct validity seeks agreement between a theoretical concept and a specific measuring device or procedure. In the case of this thesis, whether the kinetic data provided by the Labview analysis program was the data that was required.

The data analysis program used in this thesis converted collected displacement data from a text file and provided kinetic and kinematic data for both the concentric and eccentric phases of movement. This analysis program was used as it greatly reduced the time taken to calculate kinetic data compared to manually analysing the raw data. The analysis program used data provided by a linear transducer and converted the displacement text file into two additional summary files and a complete data file of kinetic and kinematic values. The summary files provided maximum force, mean force, impulse, maximum power, mean power, maximum velocity, mean velocity, work, peak work, maximum acceleration and mean acceleration values during the concentric or eccentric phase of each repetition of movement, i.e. with 10 repetitions the summary files will display 10 values of each kinetic and kinematic variable. The complete data file stored the values of displacement (filtered), velocity, acceleration, force, power and work at each point in time (dependent on the sampling rate), i.e. over 3 s at a sampling rate of 1000 Hz the file would contain 3000 data points for each kinetic and kinematic variable. However, to remove any starting spikes the analysis program defaulted to remove the first 0.5 s of displacement data. The program used a low-pass Butterworth filter on the displacement data with a defaulted set at 5 Hz. Additionally the program differentiated point-to-point values of kinetic and kinematic data. To determine if the analysis program provided valid concentric peak power and work values, these values were determined manually from the raw data and compared to those provided by the program.

Data files from one jump set of 20 supine squat jumps performed during pilot testing was analysed using an Excel spreadsheet. A text file of raw displacement data from a linear transducer operating at 1000 Hz was compared to the filtered displacement

provided by the analysis program. A total of 65535 data points (equating to a time of 65.535 s) of raw displacement data and displacement filtered by the program were loaded onto an Excel spreadsheet. The phasing of the raw data was then adjusted to the filtered data phasing by removing 500 data points. The raw data and filtered displacement data was then statistically compared to ensure that the filtering did not significantly change the values at each point in time. To determine if the values for peak power and work obtained from the analysis program were valid kinematic and kinetic variables were calculated from the filtered displacement data using an Excel spreadsheet. Filtered displacement data was used to reduce the noise effect associated with the cumulative calculations required to determine power and work.

The calculations used were,

$$v_n = \frac{\left(\frac{d_{n+1} + d_n}{2}\right) - \left(\frac{d_n + d_{n-1}}{2}\right)}{\Delta t}$$

Where v = velocity, d = displacement, n = a data point in time, and Δt (change in time) = 0.001

$$a_{(body)n} = \frac{\left(\frac{v_{n+1} + v_n}{2}\right) - \left(\frac{v_n + v_{n-1}}{2}\right)}{\Delta t}$$

Where $a_{(body)}$ = acceleration of the body

$$a_{(total)n} = g + a_{(body)n}$$

Where $a_{(total)}$ = total acceleration, and g (gravity) = 9.81 m.s⁻²

$$F_n = m \cdot a_n$$

Where F = force, and m = mass (73 kg)

$$P_n = F_n \cdot V_n$$

Where P = power

$$W_n = F_{mean} \cdot d_{max}$$

Where W = work, F_{mean} = mean concentric force, d_{max} = maximum displacement

Each level of the kinematic calculations were differentiated by averaging consecutive data points to any reduce noise (as shown in the calculations). The data was then graphed and the peak power and work data points for the concentric phase of the first ten jumps determined. Statistical comparisons of the values determined manually and those provided by the analysis program were then performed.

Comparison of displacement before and after filtering was analysed using a 2-way paired sample t-test and Pearson correlation to determine if there was a significant difference in the means, or if a correlated difference existed. Paired sample t-tests and Pearson correlations were also used to compare differences in the means of calculated peak power and work values and those from the analysis program. For all calculations SPSS 11.5.1 statistical software (SPSS Inc, Chicago, IL, USA) was used. An alpha level of 0.05 was set for all statistical procedures.

Table 13: Descriptive statistics of raw and calculated displacement data.

	Minimum	Maximum	Mean	Standard deviation
Raw displacement (m)	0.38763	1.03770	0.5548806	0.21774178
Filtered displacement (m)	0.38538	1.03608	0.5548975	0.21776330

The mean difference presented in Table 13 between the raw and filtered displacement was 0.000017 m. This difference in the displacement means was insignificant ($t = 0.17$, $p = 0.987$) with a perfect correlation ($r = 1$, $p < 0.000$). Mean difference (see Table 14) was trivially higher for calculated values compared to the program derived values for power (0.15%) and work (0.32%). Paired sample t-test of differences in the means were non-significant and correlations high for both peak power ($t = 1.69$, $p = 0.125$; $r = 0.999$, $p < 0.0000$) and work ($t = 1.958$, $p = 0.082$; $r = 0.996$, $p < 0.0000$).

Table 14: Peak power and work variables calculated manually and determined by the linear displacement analysis program

Jump	Calculated peak power (W)	Program peak power (W)	Calculated work (J)	Program work (J)
1	1450	1448	662	658
2	1758	1748	711	706
3	1839	1834	751	748
4	1640	1632	728	731
5	1646	1635	711	709
6	1601	1602	702	696
7	1542	1552	697	702
8	1493	1492	696	694
9	1490	1482	678	673
10	1341	1341	665	662
Mean \pm SD	1580.0 \pm 148.4	1577.6 \pm 146.9	700.1 \pm 27.5	697.9 \pm 28.5

Initial displacement filtering by the analysis program does not significantly change the displacement data and can be reliably used to calculate other kinetic variables. Higher kinetic values obtained during calculations may be due to the analysis program's filtering process reducing peak values or the method the program uses to determine peak values. Regardless the difference between the peak power and work values calculated and those provided by the analysis program are trivial and insignificant. The conclusion is that the analysis program was a valid construct and provided accurate concentric peak power and work values. Therefore the program was a valid tool to use in this thesis.