



Swansea University E-Theses

Investigating factors which may influence recovery and preparation in professional rugby union.

Jones, Marc Rhys

How to cite:

Jones, Marc Rhys (2014) Investigating factors which may influence recovery and preparation in professional rugby union.. thesis, Swansea University. http://cronfa.swan.ac.uk/Record/cronfa42486

Use policy:

This item is brought to you by Swansea University. Any person downloading material is agreeing to abide by the terms of the repository licence: copies of full text items may be used or reproduced in any format or medium, without prior permission for personal research or study, educational or non-commercial purposes only. The copyright for any work remains with the original author unless otherwise specified. The full-text must not be sold in any format or medium without the formal permission of the copyright holder. Permission for multiple reproductions should be obtained from the original author.

Authors are personally responsible for adhering to copyright and publisher restrictions when uploading content to the repository.

Please link to the metadata record in the Swansea University repository, Cronfa (link given in the citation reference above.)

http://www.swansea.ac.uk/library/researchsupport/ris-support/

Investigating factors which may influence recovery and preparation in professional rugby union

By

Marc Rhys Jones BSc MSc

Submitted to Swansea University in fulfilment of the requirements for the Degree of Doctor of Philosophy

Swansea University

2014

This work is part-funded by the European Social Fund (ESF) through the European Union's Convergence programme administered by the Welsh Government



Ysgoloriaethau Sgiliau Economi Gwybodaeth Knowledge Economy Skills Scholarships





ProQuest Number: 10801716

All rights reserved

INFORMATION TO ALL USERS The quality of this reproduction is dependent upon the quality of the copy submitted.

In the unlikely event that the author did not send a complete manuscript and there are missing pages, these will be noted. Also, if material had to be removed, a note will indicate the deletion.



ProQuest 10801716

Published by ProQuest LLC (2018). Copyright of the Dissertation is held by the Author.

All rights reserved. This work is protected against unauthorized copying under Title 17, United States Code Microform Edition © ProQuest LLC.

> ProQuest LLC. 789 East Eisenhower Parkway P.O. Box 1346 Ann Arbor, MI 48106 – 1346

ABSTRACT

To enhance understanding of recovery and preparation in rugby union, the aim of this thesis was to examine the impact of competition on key parameters and investigate factors which may influence the recovery process from competition and training.

The findings of study one demonstrate that movement patterns and thus the physiological demands of match-play vary considerably between different positional groups. Additionally, study two demonstrates that the movement characteristics which determine the extent of muscle damage post-match are position specific, and that movement characteristics may be used to prospectively tailor individual recovery and manage subsequent training.

Recovery patterns may also be influenced by factors not associated with match-play such as sleep, which has important physiological and psychological restorative effects. The findings of study three suggest that sleep patterns may vary considerably within a squad with many players presenting evidence of sleep disruption, particularly post-match which may be detrimental to recovery.

Recovery following exercise may also be modulated by the application of post-exercise recovery strategies such as cold water immersion. However, study four demonstrates that cold water immersion may impede adaptation to strength training in rugby union players. When no recovery intervention was administered during a five week pre-season period, isometric mid-thigh pull peak force and relative peak force significantly increased by 5.4 ± 4.7 and $5.8 \pm 5.4\%$ respectively. However when individuals were immersed in cold water post-training there were no significant changes in strength during the training period. These findings may have great implications for strength training, particularly during periods of physical development.

The findings of the thesis have furthered understanding of the characteristics of performance and identified several factors which influence recovery from training and competition. This in turn may be used to inform best practice procedures in attempt to 'optimise' preparation and recovery in rugby union. This work has not previously been accepted in substance for any degree and is not being concurrently submitted in candidature for any degree.

Signed

Date _____7/10/2014

This thesis is the result of my own investigations, except where otherwise stated. Where correction services have been used, the extent and nature of the correction is clearly marked in a footnote(s).

Other sources are acknowledged by footnotes giving explicit references. A bibliography is appended.

.....

Signed

Date _____7/10/2014

I hereby give consent for my thesis, if accepted, to be available for photocopying and for inter-library loan, and for the title and summary to be made available to outside organisations.

.....

Signed

Date $-\frac{10}{2014}$

TABLE OF CONTENTS

Abstract	I	
Declaration and statements	II	
Table of contents	III	
Acknowledgements	IX	
Publications/communications	X	
List of tables	XII	
List of figures	XIV	
List of abbreviations	XV	
General introduction	1	
1. Introduction	2	
1.1. Specific aims of experimental chapters		
Review of literature	7	
2. Analysis of movement characteristics in rugby union	8	
2.1. Why assess movement characteristics in rugby union?	8	
2.2. Methods of assessing movement in rugby union	8	
2.2.1. Time motion analysis (TMA)	8	
2.2.2. Global positioning systems (GPS)	12	
2.2.3. Summary	26	
2.3. Analysis of movement in rugby union	26	
2.3.1. Distances covered	26	

2.3.2. Physical contacts	30
2.3.3. Work to rest patterns	31
2.3.4. Temporal patterns in performance	33
2.3.5. Variance and trends in movement associated with performance	36
2.3.6. Summary	38
2.4. Application of movement analysis to enhance performance and recovery in rugby union	39
2.4.1. Physical preparation and rehabilitation	39
2.4.2. 'Real-time' monitoring	39
2.4.3. Recovery and player management	40
2.4.4. Summary	44
3. Sleep, performance and recovery	46
3. Sleep, performance and recovery 3.1. Stages of sleep cycle	46 46
 3. Sleep, performance and recovery 3.1. Stages of sleep cycle 3.2. Effect of sleep disruption on physiological and psychological function 	46 46 47
 3. Sleep, performance and recovery 3.1. Stages of sleep cycle 3.2. Effect of sleep disruption on physiological and psychological function 3.2.1. Autonomic nervous system (ANS) 	46 46 47 47
 3. Sleep, performance and recovery 3.1. Stages of sleep cycle 3.2. Effect of sleep disruption on physiological and psychological function 3.2.1. Autonomic nervous system (ANS) 3.2.2. Endocrine system 	46 46 47 47 47
 3. Sleep, performance and recovery 3.1. Stages of sleep cycle 3.2. Effect of sleep disruption on physiological and psychological function 3.2.1. Autonomic nervous system (ANS) 3.2.2. Endocrine system 3.2.3. Biochemistry, genetics and immune function 	46 46 47 47 47 49 54
 3. Sleep, performance and recovery 3.1. Stages of sleep cycle 3.2. Effect of sleep disruption on physiological and psychological function 3.2.1. Autonomic nervous system (ANS) 3.2.2. Endocrine system 3.2.3. Biochemistry, genetics and immune function 3.2.4. Learning and motor skill retention 	46 47 47 47 49 54 55
 3. Sleep, performance and recovery 3.1. Stages of sleep cycle 3.2. Effect of sleep disruption on physiological and psychological function 3.2.1. Autonomic nervous system (ANS) 3.2.2. Endocrine system 3.2.3. Biochemistry, genetics and immune function 3.2.4. Learning and motor skill retention 3.2.5. Physical performance and recovery 	46 46 47 47 49 54 55 55
 3. Sleep, performance and recovery 3.1. Stages of sleep cycle 3.2. Effect of sleep disruption on physiological and psychological function 3.2.1. Autonomic nervous system (ANS) 3.2.2. Endocrine system 3.2.3. Biochemistry, genetics and immune function 3.2.4. Learning and motor skill retention 3.2.5. Physical performance and recovery 3.2.6. Psychological performance and recovery 	46 47 47 49 54 55 55 61
 3. Sleep, performance and recovery 3.1. Stages of sleep cycle 3.2. Effect of sleep disruption on physiological and psychological function 3.2.1. Autonomic nervous system (ANS) 3.2.2. Endocrine system 3.2.3. Biochemistry, genetics and immune function 3.2.4. Learning and motor skill retention 3.2.5. Physical performance and recovery 3.2.6. Psychological performance and recovery 3.3. Sleep research in elite athletes 	46 47 47 49 54 55 55 61 68
 3. Sleep, performance and recovery 3.1. Stages of sleep cycle 3.2. Effect of sleep disruption on physiological and psychological function 3.2.1. Autonomic nervous system (ANS) 3.2.2. Endocrine system 3.2.3. Biochemistry, genetics and immune function 3.2.4. Learning and motor skill retention 3.2.5. Physical performance and recovery 3.2.6. Psychological performance and recovery 3.3. Sleep research in elite athletes 3.3.1. How is it measured? 	46 47 47 49 54 55 55 61 68 68

3.3.2. Observational studies in elite athletes69

3.3.3. Methods to enhance sleep quality	75
3.3.4. Methods to enhance performance following sleep disruption	75
3.3.5. Summary	78

4. Recovery and adaptation following exercise	79
4. 1. Physiological characteristics of fatigue and recovery	80
4.1.1. Exercise induced muscle damage (EIMD): process of events leading to further damage, functional impairment and adaptation	80
4.1.1.1. Initial damage	80
4.1.1.2. Inflammation and secondary muscle damage	81
4.1.1.3. Effect of EIMD and inflammation on muscle function and recovery	82
4.1.1.4. Repair and adaptation	84
4.1.2. Autonomic nervous system - parasympathetic reactivation	84
4.1.3. Disruption to hormonal matrix	86
4.1.4. Suppression of immune system	86
4.2. Strategies to facilitate the physiological recovery process immediately following training/competition	87
4.2.1. Cold water immersion (CWI)	87
4.2.2. Contrast water therapy (CWT)	93
4.2.3. Compression garments	93
4.2.4. Whole body cryotherapy (WBC)	94
4.2.5. Active recovery	95
4.2.6. Other strategies	96
4.2.6.1. Electrostimulation	96

4.2.6.2. Occlusion	97
4.2.6.3. Nutrition and hydration	97
4.3. Evidence that post-exercise recovery strategies may attenuate adaptation to exercise	98
4.3.1. Cold water immersion (CWI)	99
4.3.2. Dietary supplementation	103
4.3.4. Summary	105
4.4. Other considerations and contraindications for the use of post-exercise recovery strategies	105
4.4.1. Relationship between psychological and physiological recovery	105
4.4.2. Inter-individual variation	106
4.5. Summary	107
Experimental study 1	108
5. Quantifying positional and temporal movement patterns in professional rugby union using GPS	109
5.1. Introduction	109
5.2. Methods	111
5.3. Results	113
5.4. Discussion	124

5.5. Practical applications

128

Experimental study 2	129
6. Biochemical responses to physical contacts and high-speed running in professional rugby union players	130
6.1. Introduction	130
6.2. Methods	132
6.3. Results	135
6.4. Discussion	139
6.5. Practical applications	143
Experimental study 3	144
7. Does cold water immersion impair adaptation to strength training in professional rugby union players?	145
7.1. Introduction	145
7.2. Methods	147
7.3. Results	154
7.4. Discussion	158
7.5. Practical applications	163
Experimental study 4	164
8. The effect of competition on the sleep patterns of elite rugby union	165
8.1. Introduction	165
8.2. Methods	167
8.3. Results	169
8.4. Discussion	171

8.5. Practical applications	174
General discussion	175
9. Discussion	176
9.1. Practical applications	181
9.2. Recommendations for future research	182

Appendices

Appendix A: Applications for Ethical Committee approval of a research project	185
Appendix B: Subject information sheets	201
Appendix C: Raw data (see attached CD)	208

References

210

ACKNOWLEDGEMENTS

I would like to acknowledge a number of people for their help, encouragement and/or guidance, without which this thesis would not be possible:

First and foremost my supervisor, Dr. Liam Kilduff, I will be eternally grateful for the amount of time you have invested in me and for providing me with the opportunities to develop within academia and professional sport. Your consistent support and guidance has been invaluable throughout my studies at Swansea University.

To Charlie Finn, Dr. Blair Crewther, Dr. Tom Love, Dr. David Shearer, Dr. Daniel West, Dr. Christian Cook, Nick Owen, Wendy Clark and Rebecca Dietzig, I am extremely grateful for your support and/or advice throughout my studies. Furthermore I would like to thank Chris Jones, Tom Taylor, Natalie Williams, Ben Gray, Becky Gosney, Dan Cunningham, and Jamie George for your assistance during data collection.

Sincere thanks to the Scarlets, in particular Bradley Harrington, for allowing me to carry out my studies. I consider myself extremely fortunate to have had the opportunity to conduct research within a high performance environment, and I am grateful for the cooperation and respect afforded to me by players and staff. Additionally I'd like to thank the S&C staff, in particular Ryan Campbell, for all the stimulating conversations, thoughts and advice exchanged.

To Adam Beard and Ryan Chambers from the Welsh Rugby Union, and Perform Better Limited, thank you for kindly providing GPS units for studies one and two.

Special thank you to the Knowledge Economy Skills Scholarships (KESS) programme for providing the unique opportunity to conduct research while working at the Scarlets. I consider myself fortunate to have had the opportunities made possible by KESS such as presenting in Melbourne, and would like to thank the support and guidance from the KESS office in particular Cassy Froment, Jane Kelly, Penny Dowdney and Jane Phipps.

Finally I would like to thank my family and friends for their support throughout my studies. In particular I would to thank my parents and grandparents for their unwavering love and support. Your pride and confidence in me has helped me to achieve goals that I did not believe were attainable. Last but certainly not least, thank you to my fiancée Frances for putting up with me throughout my studies. How you tolerated all those weekend nights in, the occasional meltdown and the continual moaning I will never know. Without your love and encouragement this thesis would not have been possible.

PUBLICATIONS/COMMUNICATIONS

Publications arising from the material presented in this thesis thus far.

Journal

Jones MR, Shearer DA, Harrington DJ, Kilduff LP. The effect of competition on the sleep patterns of elite rugby union players. *J Aus Str Cond* 21(S2), 2013.

Book chapter

Cook CC, Kilduff LP and Jones MR. Recovering effectively in high performance sport. In *High-performance training for sports*, eds. Joyce D and Lewindon D. Oxford, Routledge, to be published June 2014.

Peer reviewed publications arising during candidature.

Journals

Jones RM, Cook CC, Kilduff LP, Milanovic Z, James N, Sporis G, Fiorentini B, Fiorentini F, Turner A and Vuckovic G. Relationship between repeated sprint ability and aerobic capacity in professional soccer players. *Scientific World Journal* Oct 1;2013:952350, 2013.

West DJ, Finn C, Cunningham DJ, Shearer DA, Jones MR, Harrington B, Crewther BT, Cook CJ and Kilduff LP. The neuromuscular function, hormonal, and mood responses to a professional rugby union match. *J Strength Cond Res* 28: 194-200, 2014.

West DJ, Owen NJ, Jones MR, Bracken RM, Cook CJ, Cunningham DJ, Shearer DA, Finn CV, Newton RU, Crewther BT and Kilduff LP. Relationships between force-time characteristics of the isometric strength pull and dynamic performance in professional rugby league players. *J Strength Cond Res* 25: 3070-5, 2011.

Communications arising from the material presented in this thesis thus far.

Poster presentation

Jones MR, Shearer DA, Harrington DJ, Kilduff LP. *The effect of competition on the sleep patterns of elite rugby union players*. Presented at 2013 ASCA International Conference on Applied Strength and Conditioning, November 2013, Melbourne, Australia.

Lecture/conference presentations

Jones MR. *Neuromuscular, endocrine and mood responses to an elite rugby union match.* The 3rd Annual Strength and Conditioning Conference. Presentation at Middlesex University, March 2012, London, UK.

MR Jones. *Sleep assessment in sport*. Recovery Symposium. Invited seminar presentation at Sport Wales, June 2013, Cardiff, UK.

LIST OF TABLES

Table 2.1. GPS sampling frequency validation and reliability studies.	15
Table 2.2. Impact zone classification using Team AMS software (McLellan et al., 2011b).	25
Table 2.3. Weighted between-subject correlation coefficient (r) for game statistics with respect to the change in CK from pre-game to post-game (mean games per player = 3.4) for 23 elite rugby union players (Smart et al., 2008).	42
Table 3.1. Effect of sleep restriction on physiological performance and recovery.	57
Table 3.2. Summary of cognitive performance effects of sleep deprivation(Goel et al., 2009).	62
Table 3.3. Effect of sleep restriction on cognitive performance and recovery.	64
Table 3.4. Definitions of each sleep variable measured using wristwatch actigraphy (Leeder et al., 2012b).	74
Table 3.5. Potential non-pharmacological means for promoting sleep quality and/or quantity (adapted from Duffield et al., 2013; Halson, 2008; Howatson et al., 2012).	77
Table 4.1. Examples of studies which have assessed the efficacy of post-exercise recovery strategies compared to a control condition where no recovery intervention was implemented.	90
Table 5.1a. Mean (±SD) distance covered (m) within each speed zone.	115
Table 5.1b. Mean (\pm SD) meterage (m.min ⁻¹) within each speed zone.	116
Table 5.2a. Mean (±SD) forwards sprinting characteristics from competition.	117
Table 5.2b. Mean (±SD) backs sprinting characteristics from competition.	119
Table 5.3a. Mean (±SD) description of forwards repeated high intensity exercise (RHIE) bout characteristics from competition.	121

Table 5.3b. Mean (±SD) description of backs repeated high intensity exercise122

(RHIE) bout characteristics from competition.

Table 5.4. Temporal changes (mean \pm SD) in movement patterns throughout123match-play (n=71).

Table 6.1. Summary of performance markers derived from global positioning136system (GPS) and performance analysis data. Data presented as mean ±SD.

Table 6.2. Correlation between changes in creatine kinase (CK) and performance 137 analysis characteristics.

Table 6.3. Correlation between changes in creatine kinase (CK) and movement138and performance markers derived from global positioning system (GPS) data.

Table 7.1. Weekly training schedule during five week pre-season period.148

Table 7.2. Total number of training sessions and number of sessions150performed by players within each group (expressed as mean ± SD).

Table 7.3. Baseline and post-training body mass, body fat measure (sum of 8)155and estimated fat free mass (FFM) in the cold water immersion (CWI, n=11)and control (CON, n=9) recovery groups.

Table 7.4. Baseline and post-training values for isometric mid-thigh pull156(IMTP) peak force (PF) and relative force (Rel. PF), and bench press and156chin-up predicted 1RMs in the cold water immersion (CWI) and control(CON) recovery groups.

Table 8.1. Sleep actigraphy data of professional rugby union players prior and170post-match (n=28).

LIST OF FIGURES

Figure 2.1. Camera locations around playing area for automated vision	11
based tracking system (Roberts et al., 2008).	
Figure 2.2. Calculation of instantaneous player load (Aughey et al., 2011a).	20
Figure 2.3. The relationship between two accelerometers placed on the same	22
player during Australian rules football matches. The solid line is a line of	
best fit; hatched lines represent 95% confidence limits (Boyd et al., 2011).	
Figure 2.4. Mean (SD) m.min ⁻¹ per 10min of match-play. *significantly	35
(p<0.05) different from forwards, ^significantly (p<0.05) different from	
0-10min, #significantly (p<0.05) different from 10-20min, ~significantly	
(p<0.05) different from 40-50min (Austin and Kelly, 2013).	
Figure 3.1. The human sleep cycle (adapted from Walker and Stickgold, 2004).	48
Figure 3.2. Schematic representation of the effects of sleep debt on	53
skeletal muscle metabolism (adapted from Dattilo et al., 2011).	
Figure 3.3. Mean number of lapses on Psychomotor Vigilance Task (and	63
Standard error) across days as a function of time in bed group	
(Belenky et al., 2003).	
Figure 4.1. Summary of the adaptation process initiated by muscle damage.	85
Figure 4.2. Peak power (W) during the 10min time trial during the HIIT.	102
Clear and dark circles represent the CON and CWI groups respectively.	
B = Baseline, ITP = Intensified training (Halson et al., 2014).	
Figure 7.2. Changes in isometric mid-thigh pull (IMTP) peak force (PF)	157
values from baseline to post-training in the cold water immersion (CWI)	
and control (CON) recovery groups.	

XIV

LIST OF ABBREVIATIONS

α	alpha
μΙ	microliter
Δ	change
η^2	eta squared
±	plus or minus
>	greater than
<	less than
~	approximately
%dec	percentage decrement; fatigue index
%∆CK	percentage change in creatine kinase
ΔCK	change in creatine kinase
°C	degrees centigrade
2-D	two dimensional
3-D	three dimensional
acc	acceleration
AFL	Australian Football League
AKt	V-akt murine thymoma viral oncogene homolog 1
ANOVA	analysis of variance
ANS	autonomic nervous system
ARE	antioxidant response element
AST	aspirate transaminase
ATP	adenosine triphosphate
CA	constant attention
Ca ²⁺	calcium
CG	compression garments
CI	confidence interval
CK	creatine kinase
COD	change of direction
CON	control
COX	cyclooxygenase
CMJ	countermovement jump
CNS	central nervous system
CRP	C-reactive protein
CV	coefficient of variation
CWI	cold water immersion

ł

CWT	contrast water therapy
CWT6	contrast water therapy (6mins)
CWT12	contrast water therapy (12mins)
CWT18	contrast water therapy (18mins)
dec	deceleration
DOMS	delayed onset muscle soreness
ECG	electrocardiography
EEG	electroencephalography
E:R	exercise to rest ratio
EI	exertion index
EIMD	exercise induced muscle damage
EOG	electrooculography
FFM	fat free mass
FRA	fragmentation
G	gravitational force(s)
GAS	general adaptation syndrome
GH	growth hormone
GPS	global positioning systems
GRF	ground reaction force
h	hour(s)
H^{+}	hydrogen ion
HI	high-intensity
HIIS	high-intensity interval exercise sessions
HIIT	high-intensity interval test
HIR	high-intensity running
HPA	hypothalamo-pituitary-adrenal
HRV	heart rate variation
HWI	hot water immersion
Hz	hertz
ICC	inter class correlation
IGF	insulin growth factors
IGFBP	insulin-like growth factor-binding agent
IL	interleukin
impacts.min ⁻¹	impacts per minute
IMTP	isometric mid-thigh pull
ISAK	International Society for the Advancement of Kinanthropometry (ISAK)
ISE	intermittent sprint exercise

IU	international unit
IU.day ⁻¹	international units per day
km	kilometre(s)
km.h⁻¹	kilometres per hour
kN	kilonewton(s)
LB	lower body
LFF	low frequency fatigue
lipoic acid.kg ⁻¹	lipoic acid per kg body weight
LIST	Loughborough intermittent shuttle test
load.min ⁻¹	player load per minute
m	metre(s)
m.min ⁻¹	metres per minute
m.s ⁻¹	metres per second
m.s ⁻²	metres per second squared
min	minute(s)
mg	milligram
mg.day ⁻¹	milligrams per day
mg.kg ⁻¹	milligrams per kilogram
ml	millilitre
mm	millimetre(s)
mm.Hg ⁻¹	millimetre of mercury
mmol.kg ⁻¹	millimoles per kilogram
MP	mean power
$\mathrm{MMP}_{\mathrm{4min}}$	maximal time trial (Halson et al., 2014)
mRNA	messenger ribonucleic acid
MS	muscle soreness
mTOR	mammalian target of rapamycin
MVC	maximal voluntary contraction
NPC	National Provincial Championship
NREM	non-rapid eye movement
NRL	National Rugby League
NSAIDs	non-steroidal anti-inflammatory drugs
NSW	New South Wales
p	statistical significance
P13K	$phosphatidy linositol \hbox{-}4, 5 \hbox{-} bisphosphate 3 \hbox{-} kin ase$
Pa	pascal
PE	physical education

•

PF	peak force
PGC-1a	peroxisome proliferator-activated receptor-gamma coactivator-1alpha
PNS	parasympathetic nervous system
PP	peak power
PSD	partial sleep deprivation
PSDB	partial sleep deprivation at beginning of night
PSBE	partial sleep deprivation at end of night
PSG	polysomonography
PVT	psychomotor vigilance test
r	Pearsons product momentum correlation coefficient
r ²	coefficient of determination
rad.s ⁻¹	radians per second
RBE	repeated bout effect
Rel. PF	relative peak force
REM	rapid eye movement
RESTQ-Sport	Recovery-Stress Questionnaire for Athletes
RF	resting fatigue
RFD	rate of force development
RHIE	repeated high intensity bout
RM	repetition maximum
ROS	reactive oxidative species
rpm	revolutions per minute
RSA	repeated sprint ability
RT	reaction time
RV-39	rhinovirus 39
S	second(s)
SA	selective attention
SD	standard deviation
SSC	stretch shortening cycle
SEM	standard error of measurement
SJ	squat jump
SNS	sympathetic nervous system
SWA	slow wave activity
SWC	smallest worthwhile change
SWS	slow wave sleep
Т	time point
TD	total distance

XVIII

TEM	technical error of measurement
TMA	time motion analysis
TNF-a	tumour necrosis factor-α
TQRP	total quality recovery perception
TSD	total sleep deprivation
TW	total work
URTI	upper respiratory tract infection
VHIR	very high-intensity running
vitamin E.kg ⁻¹	vitamin E per kg body weight
VJ	vertical jump
VLa4	velocity corresponding to blood lactate concentration of 4 mmol.L ⁻¹
V _{max}	maximum running velocity
VO_{2max}	maximal aerobic uptake
vVO_{2peak}	velocity at peak oxygen uptake
W	watt
WB	whole body
WBC	whole body cryotherapy
yrs	years
YYIRT1	Yo-Yo intermittent recovery test level 1
YYIRT2	Yo-Yo intermittent recovery test level 2

GENERAL INTRODUCTION

1. Introduction

Rugby union is a physically demanding game characterised by repeated high-intensity bouts of relatively short duration exercise, with varying and often incomplete recovery periods (e.g. Cahill et al., 2013; Roberts et al., 2008). The high-intensity bout characteristics are largely positional dependent (Cahill et al., 2013); however all players are exposed to a high magnitude of physical contacts and collisions (Cunniffe et al., 2010; Quarrie et al., 2013), and high intensity stretch-shortening cycle (SSC) movements. As a consequence, rugby union players may take several days to fully recover following competition (e.g. West et al., 2014).

Research demonstrates large elevations in blood markers of muscle damage (e.g. creatine kinase; CK) (Cunniffe et al., 2010; Smart et al., 2008; Takarada, 2003), and disruptions to neuromuscular (West et al., 2014), hormonal (Cunniffe et al., 2010; Elloumi et al., 2003; West et al., 2014), immune (Cunniffe et al., 2010) functions and mood (West et al., 2014) for several days following competition. With the accumulation of subsequent training and performance, insufficient recovery prior to the start of the training week may compromise players' ability to train, and with the added stress from training, players' preparation for subsequent games may be compromised (McLean et al., 2010). Insufficient recovery may also predispose an individual to a greater risk of injury (Lazarim et al., 2009) and the development of overtraining syndrome (McLellan et al., 2012). Thus the topic of recovery has become an important focus of player management and research in rugby union within recent years.

Previous research has also highlighted the individual nature of recovery in professional rugby union (West et al., 2014). For example, West et al. (2014) found countermovement peak power output of 50% of players had not returned to pre-match values 60h post-match. Knowledge of individual recovery could therefore be used to benefit player management through individual modification of subsequent training and recovery strategies (West et al., 2014). However, assessing recovery following each game is often associated with invasive collection procedures and analysis which may take several hours or days to conduct (e.g. hormone analysis from saliva sample, (Thorpe and Sunderland, 2012); muscle damage from blood sample; (Cunniffe et al., 2010)); thus information attained may not be used for individual recovery and subsequent weekly training modulation.

Under the premise that a greater understanding of movement characteristics will lead to a better understanding of the physiological demands of the sport, researchers have investigated the movement characteristics of players during match-play in rugby union (Austin et al.,

2011a; Austin et al., 2011b; Cahill et al., 2013; Coughlan et al., 2011; Cunniffe et al., 2009; Deutsch et al., 1998; Deutsch et al., 2007; Eaton and George, 2006; Quarrie et al., 2013; Roberts et al., 2008). Furthermore, research suggests that performance characteristics could be used to prospectively predict individual recovery demands in rugby union (Cunniffe et al., 2010; Takarada, 2003) and other team sports (McLellan et al., 2011a; McLellan et al., 2012; Thorpe and Sunderland, 2012). For example, physical contacts have been correlated to CK level in rugby union (Cunniffe et al., 2010; Smart et al., 2008; Takarada, 2003) and neuromuscular recovery in rugby league (McLellan et al., 2012), suggesting recovery may be determined by the extent of mechanical damage induced through contact during performance in rugby codes. With the metabolic, mechanical and neural elements associated with fatigue following SSC activity (Nicol and Komi, 2003), it is also likely that muscle damage and neuromuscular fatigue following rugby union may partly be determined by the extent of high-intensity movement characteristics. Indeed, recent research has shown correlations between high-speed running characteristics and changes in CK (Thorpe and Sunderland, 2012; Young et al., 2012) and neuromuscular function (Duffield et al., 2012) during the recovery period in team sports (soccer, rugby league and Australian rules football). However no research has been conducted to assess the correlation between high-speed running and markers of recovery in rugby union.

To date, the majority of studies that have investigated the movement characteristics of rugby union have done so through time motion analysis (TMA) systems (e.g. Austin et al., 2011a; Austin et al., 2011b; Deutsch et al., 1998; Deutsch et al., 2007; Eaton and George, 2006; Roberts et al., 2008). Despite advancements in movement analysis through automated analysis (Lacome et al., 2013; Quarrie et al., 2013), methodological issues still remain when assessing rugby union using TMA; with considerable manual input and potential measurement inaccuracies. More recently, advancements in global positioning system (GPS) technology have allowed for the accurate, detailed and automated analysis of movement in rugby union which may be assessed in 'real-time' improving the effectiveness of movement analysis for impacting and analysing performance. In particular, units sampling at frequencies of 10Hz have been shown to have acceptable reliability and validity to analyse movement in team sports (Castellano et al., 2011; Johnston et al., 2014b; Varley et al., 2011). However, to date research in rugby union have assessed movement at 1Hz (Cunniffe et al., 2009) and 5Hz (Coughlan et al., 2011; Cahill et al., 2013), with research suggesting the reliability and validity of sampling at these frequencies may be acceptable for measuring long straight line running efforts (Barbero-Alvarez et al., 2010) but for the assessment of brief, high-speed straight-line running (Barbero-Alvarez et al., 2010; Jennings et al., 2010; Johnston et al., 2012; Petersen et al., 2010), accelerations or efforts involving a change in

direction (Duffield et al., 2010; Jennings et al., 2010; Portas et al., 2010), large error may be present. Using valid and reliable units, research is therefore required in rugby union to understand the movement characteristics of performance and how they may correlate to markers of recovery post-match. This in turn may be used to help understand the physiological demands and consequences of performance which may be used to aid player preparation and recovery.

In attempt to accelerate recovery following training and competition, it is common practise to employ one or more post-exercise recovery strategies, such as cold water immersion (CWI); with this being reported to reduce the extent of muscle damage, inflammation and contractile function impairment following exercise (Wilcock et al., 2006). During periods of physical development such as pre-season, CWI may be used post-training in attempt to optimise training adaptation; by potentially allowing greater training loads (increased frequency, intensity or duration) to be performed in subsequent sessions, thus increasing the stimulus for adaptation (Versey et al., 2013). However, despite enhancing physiological recovery for subsequent sessions, the use of strategies such as CWI to enhance adaptation during these periods may be counterintuitive. For example, it has been suggested that muscle damage and the subsequent inflammatory response is a vital precursor for the signalling mechanisms which initiate the repair and growth of cells (Carlson and Faulkner, 1983), therefore incorporating strategies which attempt to blunt the effects of exercise induced muscle damage may in fact reduce the potential for training adaptation to occur. Indeed research has demonstrated that several strategies which aim to reduce the extent of the inflammatory process; CWI (Yamane et al., 2006), local cold pack application (Nemet et al., 2009) and antioxidant supplementation (Fischer et al., 2004; Gomez-Cabrera et al, 2008; Strobel et al., 2010), may inhibit the stimulus for adaptive physiology. Therefore although recovery strategies appear to be important components of athlete recovery in periods of competition, it has been questioned whether they should be applied during periods of training which target physical development (Cook et al., in press; Leeder et al., 2012a; Versey et al., 2013).

Currently there is lack of literature examining the effect of CWI on adaptation in elite athletes; therefore best practice recovery protocols during periods of adaptation are unclear. In attempt to understand the role CWI has on adaptive physiology in elite athletes, further research is required using elite athletes during training periods where adaptation is of principal importance.

Much research has investigated the efficacy and optimal application of post-exercise recovery strategies (e.g. Leeder et al., 2012a). However, recent research suggests sleep may

be the single most efficacious recovery strategy (Halson, 2008), while athletes perceive sleep to be the most important post-exercise recovery modality when compared to other modalities including CWI, active recovery and massage (Venter, 2012). Sleep is a basic requirement for human health and is recognised as an important component of athlete recovery due to its physiological (Datillo et al., 2011) and psychological (Meerlo et al., 2008) restorative effects, however the importance of sleep is most evident when disrupted (i.e. total or partially deprived and/or fragmented) with studies demonstrating the negative effects of sleep disruption on autonomic nervous system (e.g. Spiegel et al., 1999), endocrine system (e.g. Spath-Schwalbe et al., 1991), biochemical (e.g. Abedelmalek et al., 2013), genetic (e.g. Moller-Levet et al., 2013), cognitive (e.g. Cook et al., 2011) and neuromuscular (e.g. Bambaeichi et al., 2005) functions and mood states (e.g Sinnerton and Reilly, 1992).

Despite its proposed importance, it has been suggested that frequently disrupted and restricted sleep is a prevalent problem in modern society (Meerlo et al., 2008) which may also be common within elite athletes (e.g. Leeder et al., 2012b). Research demonstrates that several factors associated with training and competition (e.g. exercise intensity; Trinder et al., 1985, training volume; Jurimae et al., 2002; Jurimae et al., 2004; Taylor et al., 1997, and competition stress; Lastella et al., 2012) may have negative consequences to athlete sleep quality. Furthermore, research has shown that subsequent (Lastella et al., 2012) and preceding (Richmond et al., 2004) performance may also disrupt sleep which would have great implications for the preparation and recovery of rugby players. To date, no research has been performed to characterise sleep patterns in rugby union, therefore research is required to examine the sleep patterns of professional rugby union players.

1.1. Specific aims of experimental chapters

1. Study one will assess the movement patterns of professional rugby union players using GPS units sampling at 10Hz.

2. Study two will examine the relationships between muscle damage and performance characteristics associated with physical contacts and high-speed movement in professional rugby union.

3. The aim of study three is to assess the effect of CWI on adaptation to strength training during pre-season in professional rugby union.

4. The purpose of study four is to examine the sleep patterns of professional rugby union players', with data collected prior and post-match-play to assess the potential influence of competition on sleep patterns.

REVIEW OF LITERATURE

.

7

2. Analysis of movement characteristics in rugby union

2.1. Why assess movement characteristics in rugby union?

Researchers have investigated the movement characteristics of players during match-play in rugby union (Austin et al., 2011a; Austin et al., 2011b; Cahill et al., 2013; Coughlan et al., 2011; Cunniffe et al., 2009; Deutsch et al., 1998; Deutsch et al., 2007; Eaton and George, 2006; Roberts et al., 2008; Quarrie et al., 2013) under the premise that a greater understanding of these characteristics will lead to a better understanding of the physiological demands of the sport. This in turn may facilitate the planning and implementation of training programmes that enhance physical preparation for performance (Austin et al., 2011a). Furthermore, knowledge of expected trends in performance may aid assessment of individual and team performance (Quarrie et al., 2013), and may be used to aid tactical decisions during performance (Kempton et al., 2013b).

Assessment of movement may also have important implications for player management following competition. Research in team sports have shown that certain movement characteristics may correlate to physiological markers of recovery (Cunniffe et al., 2009; Nedelec et al., 2014; Smart et al., 2008; Takarada et al., 2003; Thorpe and Sunderland, 2011; Young et al., 2012) and soft tissue injury risk (Gabbett and Ulah, 2012), therefore an appreciation of how markers relate to recovery may aid in modulating subsequent recovery and training sessions post-competition.

At present there are two means of assessing movement in team sports: time motion analysis (TMA) and global positioning systems (GPS). The following section will review the methods and limitations of TMA and GPS; before reviewing the literature that has assessed movement characteristics in rugby union. Finally the application of movement analysis to enhance the preparation and management of athletes will be discussed.

2.2. Methods of assessing movement in rugby union

2.2.1. Time motion analysis (TMA)

To date, the majority of studies that have investigated the movement characteristics of rugby union have done so through TMA systems (e.g. Austin et al., 2011a; Austin et al., 2011b; Deutsch et al., 1998; Deutsch et al., 2007; Eaton and George, 2006; Roberts et al., 2008) which involve the analysis of performance characteristics from video recordings. The most commonly used method of TMA is notational analysis, which is the subjective quantification

of individual player(s) activity by an investigator, such as the frequency and duration of a particular movement. Methods of notational analysis in team sport have ranged from manually recording positional information (e.g. transferring video footage onto transparent foil to determine distances according to a reference grid (Erdmann, 1992)) to using shorthand notational analysis (e.g. Ali and Farrally, 1990) which may be performed live or retrospectively. More recently commercial available notational analysis software has become popular in rugby to assess performance. Software such as Sportscode 9 Elite (Sportstec; Warriewood, NSW) allow analysts to 'code' player incidents (e.g. tackle, pass, kick, carry) both in 'real-time' and retrospectively with an on-screen dashboard. This allows the total number of 'positive' and 'negative' incidents to be assessed, with user friendly tabs which allow for the analysis of individual and team incidents. However, such software has limitations around its inability to distinguish and quantify movement characteristics such as jogging and sprinting (Barris and Button, 2008).

In order to assess locomotion characteristics by notational analysis a video camera must follow the movement of an individual player for an entire match. Retrospectively or subjectively, the analyst must decide the type of movement from a given category. Distances may be estimated using field markings for guides, from which total distance covered, distance and time spent in different time zones may be reported (Deutsch et al., 1998; Duthie et al., 2005; Eaton and George, 2006). Earlier research provided information on the number of efforts, time spent and distances covered at different speeds, and work to rest patterns (e.g. Deutsch et al., 1998; Duthie et al., 2005), however, the reliability of this subjective data entry procedure and the ability to reproduce the same results when analysis is repeated, is a major limitation of notational analysis (Duthie et al., 2003b). For example, previous research assessed the test-rest reliability of an experienced analyst (10 games, ~20h) from the footage of 10 elite rugby union players during 'Super 12' matches which were analysed twice separated by one month (Duthie et al., 2003b), and found that the total time spent in the individual movements of walking, jogging, striding, sprinting, static exertion and being stationary had moderate to poor reliability (5.8-11.1% TEM). Furthermore, the frequency of individual movements had good to poor reliability (4.3-13.6% TEM), while the mean duration of individual movements had moderate reliability (7.1-9.3% TEM). For comparisons to be made between matches and studies, inter-observer consistency is considered crucial in establishing the reliability of TMA systems and data collected (Barris and Button, 2008). As one camera is required to film one individual's movement, another limitation of this method is that either many cameras and therefore analysts may be required, or limited players may be analysed.

Despite the limitations of previous methods, player movement may now be tracked by an automated vision based TMA system, which use multiple cameras mounted around a pitch to assess player movement according to a constructed co-ordinate system. For example, Roberts et al. (2008) assessed the movements of 29 players from an English Premiership team recorded over five games. Five video cameras were positioned around a rugby pitch at predetermined locations ensuring the total area of the playing surface was visible, from which a global 2-D Cartesian co-ordinate system was constructed with the origin located in one corner of the playing area. Cameras were calibrated by recording sequences of four calibration poles (height = 1.0m) positioned on the playing surface to known locations (Figure 2.1.).

Using the co-ordinate system constructed, displacement data of each player was used to categorise player movements into discrete activity classifications, removing subjectivity of movement (Roberts et al., 2008). The system used in the study by Roberts et al. (2008) improved reliability of data collected compared to previous methods used (e.g. Deutsch et al., 1998; Duthie et al., 2005), with previous research demonstrating the methods used had good and moderate correlations of inter and intra-operator reliability for distances travelled (0.9 and 0.5%) and speeds obtained (6.0 and 3.4%) respectively (Roberts et al., 2006). Furthermore, compared to measured routes and speeds, the method used returned CVs of 2.1 and 8.3% respectively (Roberts et al., 2006). However the system used in this study still has its limitations. In situations where multiple players cluster in restricted playing areas, the system may lose track of player movement; which is a major issue in team sports like rugby union (Barris and Button, 2008). Furthermore, with movement captured at a rate of 1Hz, high-speed movement may be under-estimated (Roberts et al., 2008). Another limitation of the method used by Roberts et al. (2008) was that data was extrapolated from 40min (from 20-60min) to give full match analysis (80min). Thus, variation in values for total distance travelled at high intensity and sprinting speeds, and total time spent in work activities were 15.3% and 12.7% respectively between predicted and actual values.



Figure 2.1. Camera locations around playing area for automated vision based tracking system (Roberts et al., 2008).

Several semi-automatic TMA systems with reasonably high levels of accuracy and reliability have recently become commercially available (Barris and Button, 2008). In a comprehensive analysis of international rugby by Quarrie et al. (2013), 763 player data files from 90 international matches played by the New Zealand national team from 2004 to 2010 were analysed using Verusco Technologies Inc. (Palmerston North, New Zealand; Quarrie et al., 2013). Although the method of analysis used by Quarrie et al. (2013) is a progression in TMA in terms of numbers of players analysed and the time taken to analyse, validity for the software used is not presented in literature and methodological issues still remain. Despite reporting accuracies of player position between 140-200mm, with position captured at 25 frames per second and sampled at 2Hz, approximately 10% of player movement was undetected and required manual correction. Furthermore, to estimate the distance travelled by players between plays, the straight-line displacement from the position of the player at the end of one play to the beginning of the next was calculated thus underestimating movement. Another comprehensive analysis of international rugby used Amisco Pro® (Sport Universal Process, Nice, France) software which also captured data at 25Hz, but processed the raw data at 10Hz and has previously been demonstrated to have high levels of accuracy and reliability when compared to known distances (Zubillaga et al., 2006). However, despite advancements in movement analysis (Lacome et al., 2013), these systems are unable to detect and quantify static activities such as scrums, rucks, mauls and tackles, therefore these activities still require manual input.

2.2.2. Global positioning systems (GPS)

More recently, the emergence of portable GPS tracking units in sport has potentially provided an alternative to overcome some of the limitations associated with TMA systems (McLellan et al., 2011b). GPS allows the tracking of a change in position (displacement) of an object (e.g. an athlete) in real-time by calculating the displacement between the signal (satellite) and the receiver (GPS unit). This calculation utilises a doppler frequency calculation, whereby the phase shift difference between the satellite and an oscillator-produced signal within the receiver is measured. When 4 or more satellite signals can be received simultaneously, a 3-D location can be ascertained (Dobson and Keogh, 2007). GPS units are typically worn and held within a harness or vest so that it is positioned in the area of the upper thoracic spine, between the left and right scapulae. Player movement may be assessed in 'real-time' or retrospectively.

To date research in rugby union have assessed movement at 1Hz (Cunniffe et al., 2009) and 5Hz (Cahill et al., 2013; Coughlan et al., 2011), however studies assessing the reliability and construct validity of units sampling at these frequencies suggest that they lack the sensitivity to accurately quantify changes in movement patterns in team sport (Table 2.1.). Research suggests the reliability and validity of these units may be acceptable for measuring long straight line running efforts (Barbero-Alvarez et al., 2010) but for the assessment of brief, high-speed straight-line running (Barbero-Alvarez et al., 2010; Jennings et al., 2010; Johnston et al., 2012; Petersen et al., 2010), accelerations or efforts involving a change in direction (Duffield et al., 2010; Jennings et al., 2010; Portas et al., 2010) large error may be present. For example, Jennings et al. (2010) assessed the reliability (CV) and construct validity (SEM) of running over a range of distances at different speeds using 1 and 5Hz units (MinimaxX, Team 2.5, Catapult Innovations, Scoresby, Australia). The authors found poor reliability and validity for 1 and 5Hz units when performing a 10m sprint (39.5% and 32.4 \pm 6.9%, 77.2% and $30.9 \pm 5.8\%$ respectively), however units had greater reliability and validity when distance increased and speed decreased (e.g. 5Hz 40m walk; CV 9.2% SEM $9.8 \pm 2.0\%$). Furthermore, 1Hz (SPI Elite, GPSports, Australia) and 5Hz (MinimaxX, Team Sport Model, Catapult, Australia) units have been shown to have poor reliability for detecting distance covered when performing court based activity representative of team sport (CV 26.7% and 35.3% respectively; Duffield et al., 2010). Indeed, research suggests that subjective notational analysis may be a more valid and reliable method of tracking player movement for field sports or sports where short distances and changes in direction are observed, when compared to 1Hz GPS analysis (Dogramaci et al., 2011).

From the research presented it may be questioned whether 1 and 5Hz units are sensitive enough to accurately assess the movement characteristics in rugby union. However, recent advancements in GPS technology have made 10Hz units commercially available which appear acceptable for quantifying movement patterns in rugby union (Castellano et al., 2011; Varley et al., 2011). Using the MinimaxX v4.0 (Catapult Innovations, Melbourne, Australia) operating at a sampling frequency of 10Hz, Castellano et al. (2011) found inter-device reliability to be 1.3% and 0.7% over 15m and 30m sprinting distances. Compared to units sampling at 5Hz (MinimaxX v2.0; Catapult Innovations, Melbourne, Australia), Varley et al. (2011) also found 10Hz units to be 2-3 times more accurate for instantaneous velocity during tasks completed at a range of velocities compared to a criterion measure, 6 times more reliable for measuring maximum instantaneous velocity and importantly were able to detect the smallest worthwhile change during all phases of acceleration/deceleration. Therefore from these studies it appears that GPS units sampling at 10Hz produce sufficient accuracy to quantify the acceleration, deceleration and constant velocity running phases in rugby union and other team sports (Varley et al., 2011).

More recently, the validity and inter-unit reliability of 10Hz (MinimaxX S4; Catapult Innovations, Scoresby, Australia) units when performing team sport based movements were assessed (Johnston et al., 2014b) using a circuit which has been used in previous research to assess the validity and reliability of 1 and 5Hz units (Coutts et al., 2010; Jennings et al., 2010; Johnston et al., 2012). The authors found that 10Hz GPS units were a valid and reliable measure of total distance (TEM 1.3%), and when examining peak speed the interunit reliability of 10Hz GPS units (1.64%) were a clear improvement on previous research examining both 1 and 5Hz units (2.3-7.2%; Barbero-Alvarez et al., 2010, Coutts et al., 2010; Jennings et al., 2010). However as the speed of movement increased, the level of error also increased. A poor level of inter-unit reliability was evident for the distance covered, time spent and the number of efforts performed at very high running speed (20km. h^{-1} ; >10%). Nevertheless, the inter-unit reliability for the distance covered, time spent and number of efforts performed at low and high speed (0.00-13.99 and 14.00-19.99km.h⁻¹) demonstrated that the level of error for the 10Hz units ranged from moderate to good (<10%) which is a substantial improvement on what has been reported for 1Hz (<32.5%; Coutts et al., 2010) and 5Hz units (<17%; Jennings et al., 2010) using the same speed zones. Therefore literature suggests that 10Hz units are a great advancement in GPS technologies and may be suitable for detecting movement in rugby union, however caution should be given when interpreting data >20km.h⁻¹ (>5.5m.s⁻¹) (Johnston et al., 2014b). Despite GPS units being commercially available to sample movement at 15Hz (SPI-ProX; GPSports, Canberra, Australia), Johnston et al. (2014b) found that 10Hz units are capable of detecting movement demands with greater validity and reliability (Johnston et al., 2014b). However Johnston et al. (2014b) found 15Hz units exhibited greater validity and reliability in measuring movement demands compared to 1 and 5Hz units in previous studies (Coutts et al., 2010; Jennings et al., 2010).
1) Significant correlations between mean sprint tim GPS peak speed for 15m (r²=0.87, p<0.0001) and 2) The CVs and ICCs for summated maximal spee peak speed were low and high respectively. Poor C the fatigue index (%dec) obtained from GPS (36.2% Greater intra-device reliability over 30m compar 15m. Inter-devise reliability CV 1.3% and 0.7% fo All devices measured within 5m of actual lap distan CVs for total distance (3.6-7.1%) and peak speed Poor CV for HIR (11.2-32.4%) and VHIR (11.5-3 for all devices. Greater error in older SPI-10 units. had a good level of reliability (CV<5%). Findings (r²=0.94, p<0.001) sprints. 5.8%) good to moderate. and 30m respectively. high-intensity running distance 2) Assess test-re-test reliability of distance covered for each bout with sprint time from timing gates 6 laps around a 128.5m course and lap 2) high-intensity running (>14.4km.h⁻¹; HIR) and very I) Correlate peak speed from GPS peak speed, summated speed and fatigue index from 7x30m RSA Sprints performed with timing gates at 15 and 30m. Timing gates used for criterion sprinting time. Used to distinguish start/end Performance measures: 1) total (>20km.h⁻¹; VHIR) during each Filmed by video camera 25Hz. involving intermittent exercise. times for GPS exporting. Methods (15 and 30m) bout. test. 2) 14 young moderately Subjects 1) 21 PE 9 trained students players athletes trained soccer 2 Frequency 10Hz lHz 1Hz WiSPI, 2 SPI-Manufacturer and product GPSports (2 (MinimaxX SPI Elite, 2 (SPI Elite) GPSports Catapult v4.0) 10) and GPS g sampling frequency of GPS for high-1) Assess convergent device for measuring Assess validity and intensity intermittent measuring running speed in team sport reliability of a GPS running speed and RSA in team sport intra-model reliability validity of GPS for 2) Assess test re-test exercise performance. using of of different Aim(s) quantifying Reliability accuracy devices athletes. athletes devices 10Hz. Coutts and Duffield ы al. Barbero-Alvarez et Reference Castellano al. (2010) (2010) (2011)

Table 2.1. GPS sampling frequency validation and reliability studies.

Both devices under reported distance covered as w mean and peak speed compared to VICON ($p^{<}$ Faster the speed of movement and the more repet over a similar location the greater the error. CVs greater for MinimaxX devices compared to SP for all drills.	No significant differences were detected in the numl mild, moderate and heavy collisions detected vi MinimaxX units and those coded from video recor of the events. A strong correlation ($r=0.96$, $p<0.001$ observed between collisions recorded via the Miniu units and those coded from video recordings of the e	Measurement accuracy decreased as speed of locon increased in both straight line and COD courses. Difference between criterion and GPS measured di ranged from 9 to 32.4%. A higher sampling rate improved validity regardle distance and locomotion in the straight line, COI simulated running circuit trials. Reliability improved as distance travelled increase decreased as speed increased. Total distance simulated running circuit exhibited lowest variation 3.6%) while sprinting over 10m demonstrated highe 77.2% (1Hz).
 Repetitions of 4 drills replicating court-based movements. A 22 camera motion analysis system (100Hz; VICON) tracked position of 18mm reflective marker affixed to one of the GPS devices. Two of each device was worn in same harness. 	Number and intensity of collisions and the incidence of collision injuries were monitored. Total 237 events (tackles, hit-ups, decoy runs, and support runs) coded from 21 training appearances and 1 trial match. Mild, moderate and heavy contacts were compared between GPS and analyst recordings.	 Straight line movements (walk, jog, stride, sprint) at various speeds (10, 20, 40m). COD courses of 2 different frequencies (gradual and tight). A team sport running simulation circuit.
1 male	NRL	21 elite Aussie rules players wearing 2 units in same harness
1Hz and 5Hz	SHz	1Hz and 5Hz
Catapult (5Hz MinimaxX), GPSports (SPIElite, 1Hz)	Catapult (MinimaxX x2.0)	Catapult (MinimaxX), GPSports (SPI-10, SPI- Pro) Pro)
Accuracy and reliability of GPS measures of distance and speed compared to high resolution motion analysis when measuring confined movement patterns in court-based sports.	Establish validity of tackle intensity setting.	Assess validity and reliability of distance data measured by GPS units sampling at 1 and 5Hz during movement patterns common to team sport.
Duffield et al. (2010)	Gabbett et al. (2010)	Jennings et al. (2010)

GPS provides acceptable measure of TD and peak for flying sprints. GPS also a reliable measure of total distance cover the simulation circuit ($p>0.05$, %TEM < 5%). Player load, time spend and number of efforts perfort at certain velocity zones provided reliable me (%TEM <5% to >10%). Level of GPS error found to increase with veloc exercise.	Mean distance recorded was $6821 \pm 7m$ and mean was 7.0 ± 1.9 km.h ⁻¹ , compared to actual distance 6 and mean speed 7.0 ± 1.9 km.h ⁻¹ .	Reliability of estimating distance travelled by walk striding ranged from 0.3 (0.2 to 0.4%) to 2.9% (7 4.0%). Mean reliability of estimating different sprinting dist over 20 to 40m ranged from 2.0 (1.6 to 2.8%) to 3 (23.2 to 43.3%).
Team sport simulation circuit as used in Coutts and Duffield (2010) and flying 50m sprints. Two units were worn together.	 14 laps of a measured circuit incorporating intermittent running and changes of direction, representative of field hockey. Compared statistically with timing gates and distances measured using calibrated trundle wheel. Validation of speed of movement of each participant separately was also made, using data from each timing gate over a range of speeds. 	 20 trials of cricket specific movement patterns. 2) Sprinting 20-40m intervals and 'run-a-three' drill.
9 well trained	9 games players (5 male, 4 female)	1 male
5Hz	lHz	5Hz, IHz (SPI-10)
Catapult (MinimaxX)	GPSports (SPI Elite)	Catapult (MinimaxX), GPSports (SPI-10, SPI- Pro)
Investigate validity and reliability of 5Hz units	Construct validity of a non-differential GPS system for assessing player movement patterns	 Determine reliability and validity of GPS monitoring for quantifying movement patterns of cricket. Assess the validity and reliability of 3 commercial GPS units.
Johnston et al. (2012)	MacLoed et al. (2009)	Petersen et al. (2010)

Both underestimated criterion velocity of acceleration. Lower errors were associated with co velocity running. Instantaneous velocity overesti during deceleration phase; however magnitude dec with sampling frequency across all phases. Criterion and GPS velocities correlated stronger sampling at a higher rate. Weaker correlations associated with higher constan starting velocities during all phases in 5Hz GPS unit: A higher sampling rate did demonstrate imp reliability during the constant velocity, acceleration and deceleration phase. 5Hz GPS units were incapa detecting SWC during all phases of test (i.e. CV> ³ however 10Hz were acceptable.
Criterion measure instantaneous velocity was detected using a tripod mounted laser. Subjects wore both units. Subjects were asked to produce accelerations within ranges 1-6m. ² . Subsequently 1-3, 3-5, 5-8m.s ² bands were analysed.
m
5Hz and 10Hz
Catapult (MinimaxX v2.0, 4.0)
Validity and reliability of 5 and 10Hz GPS units for measuring instantaneous velocity during acceleration, deceleration and constant velocity.
Varley et al. (2011)

Caution has also been given in literature when assessing high acceleration and deceleration $(>4m.s^{-2})$ movements between different units and models sampling at 15Hz (Buchheit et al., 2014). Positioned together on a sled towed during a standardised running routine, fifty 15Hz units (15 SPI-proX, chip version 2.3.4; and 35 SPI-proX2 (17 SPI-proX2a, chip version 2.6.1; and 18 SPI-proX2b, 2.6.4), 15Hz, GPSports, Canberra, Australia) displayed small to very large between model differences and very large between-unit variations (e.g. 56%, deceleration >4m.s⁻²). Buchheit et al. (2014) also assessed the effect of software updates on measures derived from GPS and found that one update led to large and small decreases in the occurrence of accelerations and decelerations respectively. Therefore the authors recommend that care is applied when comparing data collected with different models or units, or when updating software.

To enhance the accuracy of detecting short-high acceleration and deceleration movements, many units contain tri-axial accelerometers. These accelerometers incorporate the measurement and quantification of contacts which would not only remove subjective analysis, but would provide a much a greater understanding of the physical demands of rugby union, especially for forward players who are involved in greater contact and static exertion events. Tri-axial accelerometers are highly responsive motion sensors used to measure the frequency and magnitude of movement in three dimensions (anterior-posterior, mediolateral and longitudinal). Accelerometers have a higher sample rate compared to GPS; for example MinimaxX units (Catapult Innovations, Scoresby, Australia) contain a tri-axial piezoelectric linear accelerometer system which samples at 100Hz (Boyd et al., 2011). The reliability of accelerometers within and between MinimaxX 2.0 devices both in the laboratory and in the field during semi-professional Australian rules football was previously investigated by Boyd et al. (2011) using the measure 'player load'; a measure calculated from the instantaneous rate of change of acceleration and deceleration in the forward, upward and sideward directions (Figure 2.2; Aughey et al., 2011a). That is, it measures all acceleration and deceleration in all directions (Young et al., 2010).



where

 $a_y =$ Forward accelerometer

 $a_{\rm x}$ = Sideways accelerometer

 $a_1 = Vertical accelerometer$

Figure 2.2. Calculation of instantaneous player load (Aughey et al., 2011a).

Boyd et al. (2011) showed accelerometers have good within and between reliability for both static (CV 1.0% and 1.0% respectively) and dynamic laboratory trials (CV 0.91-1.05% and 1.02-1.04% respectively). Furthermore, when ten semi-professional Australian rules footballers wore two mimimaxX accelerometers taped together during match-play, results indicated that all differences in player load between units were $\leq 2.80\%$, and relationships between data from devices on the same individual ranged between r=0.996 and r=0.999 (Figure 2.3). Both the laboratory and field setting measurement error was less than the signal (SWC 5.88%), therefore the authors suggest accelerometers within may be suitable for detecting physical activity in team sports (Boyd et al., 2011).

The potential advantage of using player load is that it accumulates from non-running activities such as kicking and jumping, and impacts in tackles and collisions. Young et al. (2012) explain that because acceleration is proportional to force, player load may provide a useful measure of the total load applied to a player in a team sport. Although unpublished, observations have shown player load relates strongly to total distance measured by GPS units in Australian rules football (r=0.898; Aughey and Boyd, *unpublished observations in* Aughey et al., 2011a).



Figure 2.3. The relationship between two accelerometers placed on the same player during Australian rules football matches. The solid line is a line of best fit; hatched lines represent 95% confidence limits (Boyd et al., 2011).

Player load therefore provides a quantification of the physiological impact of performance but it does not give a measure of the frequency or extent of physical contact from collisions. However, research suggests that tackle detection; a feature of the MinimaxX unit (Catapult Innovations, Melbourne, Australia) which incorporates accelerometers, gyroscopes and magnetometers imbedded within the unit, provides a valid quantification of the contact load in rugby league (Gabbett et al., 2010). Gabbett et al. (2010) assessed the validity of the minimaxX tackle setting by comparing collisions automatically recorded from the GPS units of 30 professional rugby league players compared to those coded from video recordings during 21 training appearances and 1 trial match. For a collision to be detected, the unit was required to be in a non-vertical position; meaning the player was leaning forwards, backwards, or to the left or right, and a spike in the instantaneous player load was required shortly before the change in orientation of the unit. Collisions were defined as (i) mild contact made with player but able to continue forward progress/momentum out of tackle, (ii) moderate - contact made with player, forward progress/momentum continued until tackled and (iii) heavy - contact made with player, forward progress/momentum stopped, and forced backwards in tackle. In total 237 collisions were coded.

No significant differences were detected in the number of mild, moderate, and heavy collisions detected via the mimimaxX units and those coded from video recordings of the events, and a strong correlation (r=0.96, P<0.01) was observed between collisions recorded via the mimimaxX units and those coded from video recordings of the events (Gabbett et al., 2010). Therefore the authors concluded that minimaxX units (Catapult Sports, Australia) are valid in determining the locomotion characteristics and the number and extent of physical collisions in rugby league (Gabbett et al., 2010). However with contact situations likely to vary between rugby codes (e.g. scrums and rucks when body orientation does not necessarily change following impact), further research is required to validate 'tackle detection' in rugby union.

Another attempt of quantifying player collisions using accelerometers within GPS units has been made by McLellan et al. (2011b) (5Hz; GPSports, Canberra, Australia) in rugby league. Using a zone classification system as a basis for analysis (Table 2.2), collisions were quantified and compared to those from video analysis for validation. However, McLellan et al. (2011b) found the average number of 'impacts' performed per player (830 \pm 135) were very much different to the actual number of tackles (14.9 \pm 10.5) and hit ups (10.2 \pm 3.8) coded from video. Even when changes of direction while running, and minor collisions with the ground and opposing players were excluded from their data, an average of 464 'impacts' involving moderate to severe collisions with players were recorded per player. Therefore despite advances in understanding the physiological load associated with performance, further research is required to validate contact detection measures in rugby union.

Tri-axial accelerometers and gyroscopes within minimaxX units (Catapult Sports, Melbourne, Australia) have also been utilised to enhance the detection of repeated highintensity exercise (RHIE) bouts. With the tackle detection setting shown to be valid in rugby league (Gabbett et al., 2010), Gabbett et al. (2011) modified previous definitions of a RHIE bout (Austin et al., 2011a; Spencer et al., 2004) so that it may be automatically detected and defined as three or more high acceleration (>2.79m.s⁻²), high-speed (5m.s⁻¹) or contact efforts with less than 21s recovery between efforts. The automatic detection of acceleration efforts in addition to sprinting and contact thus allows the inclusion of many of the high-intensity intermittent bursts in rugby union that do not allow the attainment of high-speed.

Another custom setting which may account for short high intensity efforts is 'exertion index'. Exertion index was established to measure cumulative physical load based on the sum of a weighted instantaneous speed, a weighted accumulated speed over 10s and a weighted accumulated speed over 60s (Wisbey, 2010). This computation ensures that both short sharp efforts and long sustained efforts are accounted for. The weighting is based on a polynomial relationship in which higher speeds were given a higher exertion value (in arbitary units) than lower speeds (Wisbey, 2010). At present no literature has examined exertion index in rugby union, however in Australian rules football exertion index has previously been demonstrated to be a distinguishing measure between top four and bottom four teams (Wisbey, 2008; Wisbey, 2010).

ce Collision Classification	Very light impact, hard acceleration/deceleration/change of direction while running.	Light to moderate impact, minor collision with opposition player, contact with the ground.	Moderate to heavy impact, making tackle or being tackled at moderate velocity.	Heavy-impact, high-intensity collision with opposition player(s), making direct front on tackle on opponent traveling moderate velocity, being tackled by multiple opposition players when running at sub-maximum velocity.	Very heavy impact, high-intensity collision with opposition player(s), making direct front on tackle on opponent traveling high velocity, being tackled by multiple opposition players when running at near maximum velocity.	Severe impact, high-intensity collision with opposition players(s), making direct front on tackle on opponent traveling high velocity, being tackled by multiple opposition players when running at maximum velocity.
Gravitational force (G Force)	<5.0-6.0	6.1-6.5	6.5-7.0	7.1-8.0	8.1-10.0	>10.1
Zone	1	7	3	4	Ś	9

Table 2.2. Impact zone classification using Team AMS software (McLellan et al., 2011b).

2.2.3. Summary

Traditionally, the labour-intensive nature of retrospective video recording analysis makes such analysis prolonged with feedback often restricted to a few players. Also, due to the complex movement patterns and varied nature of game play considerable subjectivity may exist when interpreting data making comparison between coders and studies potentially problematic. Despite advances in TMA technologies through automated analysis, methodological issues still remain when assessing rugby union; with considerable manual input and potential measurement inaccuracies.

Advancements in GPS technology have allowed for the accurate, detailed and automated analysis of movement in rugby union which may be assessed in 'real-time' improving the effectiveness of movement analysis for impacting and analysing performance. In particular, units sampling at frequencies of 10Hz have been shown to have acceptable reliability and validity to analyse movement in team sports, however some caution should be given when analysing high-speed movements.

With tri-axial accelerometers imbedded within the units, research exists to suggest that the number and extent of collisions may be quantified during performance. Not only would this remove a great amount of manual coding that would need to be performed post-match, but knowledge of the physical demands of rugby union may be enhanced. However further research is required to validate contact measures within rugby union. Through the use of accelerometer data, automated details of RHIE efforts and short-sharp accelerations may be investigated. Custom parameters (e.g. player load and exertion index) may also provide global measures of physiological load however validation of these measures in rugby union is required.

2.3. Analysis of movement in rugby union

2.3.1. Distances covered

Time motion analysis studies have reported variances in total distance covered during matchplay in rugby union (Austin et al., 2011b; Deutsch et al., 1998; Lacome et al., 2013; Quarrie et al., 2013; Roberts et al., 2008). For example, outside backs have been reported to cover distances between 4744 \pm 1017m (Austin et al., 2011b) and 8079 \pm 539m (Lacome et al., 2013). The reason for varied findings may be related to several methodological factors including those described in section 2.2, the level of rugby assessed (e.g. Quarrie et al., 2013) vs. Deutsch et al., 1998), the time of year research was conducted (e.g. Roberts et al., 2008) and the number of games assessed within the study (e.g. Quarrie et al., 2013 vs. Deutsch et al., 1998). However commonalities have been found between studies, with greater distances reported for backs compared to forwards (e.g. Austin et al., 2011b), and loose forwards (e.g. back row) covering greater total distances than tight forwards (e.g. prop, second row) (Roberts et al., 2008). For example, Roberts et al. (2008) reported total distances of 5408 \pm 702m, 5812 \pm 666m, 6055 \pm 455m and 6190 \pm 929m for tight forwards, loose forwards, inside backs and outside backs respectively.

In addition to positional differences in total distances covered, research demonstrates differences in distances covered at different speeds (Quarrie et al., 2013). Following six years of data collection from International rugby by TMA, Quarrie et al. (2013) conducted a cluster analysis of movement analysis; placing players into positional groups which display similar activity and movement patterns. The authors found very little similarity between forwards and backs groups (1-8 and 9-15), and found positional differences within each group. Within the forwards, differences in running demands at higher speeds, in particular by flankers were notable. Props covered greater distance than flankers between 2-4m.s⁻¹; between 6-8m.s⁻¹ flankers moved further than hookers, props and locks, number 8s moved further than hookers and props, and hookers and locks moved further than props; and above 8m.s⁻¹ flankers and number 8s moved further than props and hookers (Quarrie et al., 2013). Within the backs, wingers and fullbacks travelled further than half backs under 2m.s⁻¹ however scrum halves covered greater distance at 2-4m.s⁻¹ and 4-6m.s⁻¹ than wingers and all other backs respectively. Furthermore, above 8m.s⁻¹ wingers travelled greater distances than half backs. The authors highlight the specialised role of the scrum half, who displayed different movement patterns to any other positional group. Furthermore, the distinctive movement patterns of outside backs, in particular wingers are highlighted by the findings of Quarrie et al. (2013). Compared to previous research these findings suggest that the grouping of players (e.g. forward or loose forward) when analysing the performance characteristics of rugby may not reflect specific positional demands. Furthermore they question the inclusion of hookers as 'tight forwards' due to their movement profiles. Despite similarities between groups, these findings not only highlight physical differences in performance, but suggest that the physiological characteristics, preparation and recovery of players may be specific to each position. Nevertheless, analysis of players when grouped showed that tight and loose forwards, and inside and outside backs covered greater distances above 5m.s⁻¹ than those reported by English Premiership players (Roberts et al., 2008). Although the later study was conducted using a much smaller sample size, Quarrie et al., (2013) suggest that international

rugby is characterised by greater distances at higher speeds, which may be a key distinguishing factor between elite club and international rugby.

The two earliest studies to use GPS in rugby union assessed the movement patterns of a forward and a back during a single match (Coughlan et al., 2011; Cunniffe et al., 2009). Both studies reported distances much greater than those reported in TMA (6427 and 7002m, Coughlan et al., 2011; 6680 and 7227m, Cunniffe et al., 2009). These reasons may be methodological; for example, both studies only assessed two players, also previous TMA studies only assessed the movements of players when the ball is in play (e.g. Quarrie et al, 2013).

A recent comprehensive GPS analysis of English Premiership rugby has been published using 440 data files from 120 players, collected from 44 matches using GPS units (SPI Pro, GPSports) which recorded movement profiles at 5Hz (Cahill et al., 2013). Similar to previous research (Cunniffe et al., 2009; Quarrie et al., 2013), backs covered greater distances than forwards (6545m vs. 5850m) at a greater rate (71.1m.min⁻¹ vs. 64.6m.min⁻¹). Backs moved predominantly in the lowest speed category (46.3%) whereas forwards covered most of their distance (46.2%) whilst jogging. Furthermore, backs moved more (36.9%) in the standing and walking category, but covered more of their total distances sprinting (35.4%) than the forwards (Cahill et al., 2013) Positional findings were similar to those reported by Quarrie et al. (2013). Scrum halves travelled furthest during matches (7098m, 78.5m.min⁻¹) and the front row the least (5158m, 62.3m.min⁻¹); a difference of nearly 2km, or 37.6% (Cahill et al., 2013). Outside backs were distinguished by their attainment of the highest peak speed (31.7km.h^{-1}) and their movement in the slowest speed category (51.9% of)total distance) being a significantly higher proportion than the other positions. These findings reiterate the distinctive movement patterns of outside backs as identified by Quarrie et al. (2013). Additionally, they covered twice as much distance in the sprinting category than inside backs. In the forwards, the front row covered almost 50% of their total distance 'jogging', which was significantly more than any other group. The open-side flanker was found to cover the most distance relative to playing time within the forwards (66.7m.min⁻¹) which was comparable to the left wing and full-back positions.

The findings of Cahill et al. (2013) re-iterate many of those by Quarrie et al. (2013) however it should be noted that comparison of values to other studies should be done with caution due to the criteria used to distinguish locomotion variables. Traditionally arbitrary predetermined absolute velocity thresholds have been used to distinguish movements. For example, high-speed running has been categorised as movement above 5m.s⁻¹ in many studies irrespective of the sport played or the sex, level or age of athlete (e.g. Gabbett et al.,

2010: Gabbett, 2013a: McLellan et al., 2011b). However, unique to the research by Cahill et al. (2013) was that player's time in speed zones were based on their maximum running speed (V_{max}) determined from any game played throughout the season analysed. The authors state that peak match speeds that can be attained by the backs can be as much as 37% greater than players in the forwards (Duthie et al., 2003a). With lower work to rest ratios, forwards recovery times may never allow the attainment of maximal speed, thus the authors argue that forwards intensity profile may be misrepresented. However, it may also be argued that if maximal speed is not attained by forwards during performance V_{max} may be somewhat submaximal, therefore distances covered at high-speed may be overestimated in some positions, in particular forwards. Indeed Cahill et al. (2013) found that collectively forwards averaged 369m sprinting (81-95% V_{max}) whereas backs averaged only 323m which is inconsistent to previous research. Dwyer and Gabbett (2012) also identify that given a proportion of field sports sprints involve maximal efforts of a short duration (\sim 1-2s), a limitation of setting a velocity threshold is that it fails to capture short-duration, effortful movements that start at low velocities but do not achieve the sprinting velocity threshold. Furthermore it has been demonstrated that the metabolic cost of running at a constant velocity is significantly lower than the cost of acceleration (di Prampero et al., 2005; Osgnach et al., 2010), therefore being able to detect these efforts are important for quantifying physiological load and performance.

Recently Lacome et al. (2013) reported acceleration values derived from displacement data captured from TMA. The authors found positional differences, with mean maximal acceleration duration significantly higher in the backs compared to the forwards (3.67 ± 0.35 vs. 3.24 ± 0.39 s, respectively; p<0.05). Back rows were shown to have the highest mean acceleration value, compared to front rows, inside backs and outside backs (2.50 ± 0.95 , 2.41 ± 0.89 , 2.38 ± 0.90 and 2.34 ± 0.98 m.s⁻², respectively; p<0.001). Inside backs and outside backs both had significantly lower mean acceleration values than front rows (p<0.05), while outside backs had significantly lower mean values than inside backs (p<0.05). Furthermore, back rows had a lower percentage of acceleration values ranging between 1 and 2m.s⁻² than the other sub-groups with a higher percentage of acceleration values >3m.s⁻² than inside backs and outside backs in the medium-intensity run, jogging and walking standing zones. Therefore these findings support previous suggestions that forwards perform a greater number of maximal efforts of short duration which may not be detected by traditional movement classifications.

In attempt to detect maximal efforts which do not allow attainment of maximal speed, Dwyer and Gabbett (2012) have established a new definition for determining sprints using acceleration data at each velocity range. Using 125 data sets (5 complete games from 5 individuals in 5 different team sports) the highest 5% of accelerations within each velocity range were used to determine a threshold, over which a movement may be considered as a sprint. Thus a sprint may be defined as when a) a movement reaches or exceeds the sport specific sprint threshold velocity for at least 1s and b) the acceleration of a movement occurs above a threshold which corresponds with the highest 5% of accelerations in the associated velocity range. When assessing the movement of a male soccer player during performance, the authors found the player performed 125 sprints using the new definition compared to 19 when using to using the traditional definition for sprinting (>5.6m.s⁻¹). Further research is required to establish thresholds in rugby union, however Dwyer and Gabbett (2012) have proposed a method of assessing sprint efforts which may be suitable to the short, sharp movement demands of rugby union; particularly within the forward players.

2.3.2. Physical contacts

In addition to the running demands of performance, players frequently engage in physical collisions during attack and defence, therefore for practitioners to gain further knowledge of the physiological demands of performance, contact demands need to be assessed (Johnston et al., 2014b).

Physical contacts during match-play have typically been assessed as 'static' activities in research, banding together movements such as rucks, mauls, tackling and scrums (Duthie et al., 2005; Roberts et al., 2008). The most comprehensive analysis of physical contacts during performance was performed by Quarrie et al. (2013) who found large differences between forwards and backs in International rugby using TMA. On average the number of tackles made per game was greater within the forwards compared to the backs (\sim 7-13 vs. \sim 4-9), with the most number of tackles made by starting flankers (13.0 ± 5.7) and the least amount made by starting full backs (3.8 ± 2.5) . Furthermore, when in possession the average number of rucks attended ranged from $\sim 18-24$ in the forwards (hookers to props) which was significantly greater than the number attended by backs (~3-10), with starting props and midfield backs attending the most amount of rucks in the forwards and backs respectively $(24 \pm 10 \text{ and } 9.7 \pm 5.5 \text{ respectively})$. When not in possession the number of rucks attended was less than when in possession, however similar trends were shown between forwards and backs, with starting flankers and midfield backs attending the most amount of rucks in the forwards and backs respectively $(8.7 \pm 5.0 \text{ and } 3.4 \pm 2.5)$. Quarrie et al. (2013) also found large differences between and within forwards and backs for the total time spent in scrums,

rucks and mauls. For example, number 8's were found to spend the longest combined time in scrums, rucks and mauls $(3:37 \pm 0.54 \text{min})$ while scrum halves spent the least time engaged in these activities $(0:23 \pm 0.16 \text{min})$. The greater number of static exertions may help explain why forwards, in particular tight forwards cover greater distances under 4m.s⁻¹ (Quarrie et al., 2013). Quarrie et al. (2013) also found that the average number of scrums per game in were 25 which is comparable to other TMA research in International rugby using similar methods, which found on average 22 scrums were performed per match (Lacome et al., 2013).

To date no studies have been conducted to detect the number of physical contacts from match-play in rugby union, however as described in section 2.2.2 (Gabbett et al., 2010; McLellan et al., 2011b) GPS technologies are advancing so that the potential for automated tackle detection and quantification is a possibility.

2.3.3. Work to rest patterns

To assist understanding of the physiological demands of performance, studies have assessed the duration and frequency of 'work' efforts. Research demonstrates that the time spent in high intensity effort is relatively brief during match-play (e.g. 15% of total time; Deutsch et al., 1998). Furthermore, including activities requiring static exertion (e.g. scrummaging, rucking, mauling) as high intensity exercise, studies using TMA have found positional differences in the nature and duration of high-intensity activity performed. Roberts et al. (2008) found that forwards spent 12% of their playing time in high-intensity activities, sprinting on 16 occasions, while backs spent 4% of their time in high intensity activity but sprint an average of 23 times (Roberts et al., 2008). These findings are similar to those of Duthie et al. (2005) who found that time spent 'working' was significantly different between forwards and backs (12:22 \pm 3:39min vs. 4:51 \pm 1:15min respectively; p<0.05), with forwards performing approximately 50 more 'work' events compared to the backs (122 ± 21 vs. 72 ± 23 respectively, p<0.05). These TMA studies help explain previous findings that tight forwards cover the least absolute and relative distances (Quarrie et al., 2013; Cahill et al., 2013), as their primary roles are to contest possession at set-pieces and break downs (Quarrie et al., 2013) and in doing so they engage in more static activity, involving pushing and pulling actions, than that of the backs (Roberts et al., 2008).

Using GPS sampling at 1Hz, Cunniffe et al. (2010) found mean work to rest ratios of 1:5.7 and 1:5.8 between backs and forwards respectively, with rest periods distinguished as any

'low-intensity' movement between 0-8km.h⁻¹. However, the distinction of work and rest periods in this study may greatly underestimate the extent of work done as activities such as rucks, scrums and mauls may register as 'low-intensity' movement. Therefore although the findings by Cunniffe et al. (2010) give an indication of the intermittent nature of elite rugby union, it may not provide a true reflection of work rates, in particular for forwards who perform a greater number of activities in 'static' exertion (Quarrie et al., 2013).

Using TMA, Lacome et al. (2013) present a novel way of assess work to rest patterns using motion data collected from 67 international player files by Amisco Pro® (Sport Universal Process, Nice, France). Using measures derived from an intermittent progressive running test, displacement at velocities higher than velocity corresponding to blood lactate concentration of 4mmol.L⁻¹ (VLa4) and static activity were considered as exercise, and activity lower than VLa4 as recovery. Using these criteria, exercise to rest ratio (E:R) was calculated by dividing exercise period duration by the following recovery duration. For each period of exercise corresponding to running, mean exercise velocity was calculated and subdivided into medium-intensity running velocity (medium-intensity run, ranging between VLa4 and maximal aerobic velocity) and supramaximal running velocity (higher than maximal aerobic velocity). Using this method, mean work to rest ratio was 1:6.5 and 1:8.5 in forwards and backs respectively (Lacome et al., 2013) which demonstrates longer rest but similar positional differences compared to previous TMA studies (Roberts et al., 2008; Austin et al., 2011b). The authors found that the total number of exercise bouts were significantly lower in backs than in forwards $(137.1 \pm 28.8 \text{ vs.} 178.7 \pm 25.7, \text{ respectively};$ p < 0.05). Furthermore, the number of exercise efforts were similar in front rows compared to back rows (185.9 \pm 29.3 and 172.2 \pm 29.3, respectively) and significantly higher in inside backs that in outside backs (155.4 \pm 13.9 and 118 \pm 16.1 respectively, p<0.05). Inside backs however performed a lower number of exercise efforts than front rows (p<0.05), while outside backs performed less than back rows (p<0.05). Lacome et al. (2013) also found that front row and back rows showed similar mean total exercise times (11.9 \pm 1.3 and 11.2 \pm 2.6min respectively). Inside backs showed a mean total exercise time of 10.0 ± 1.4 min, which was similar to back rows, shorter than front row (p<0.05) and longer than outside backs $(7.1 \pm 1.3 \text{min}, p < 0.05)$.

Although the total accumulated time that players engage in high-intensity exercise during a match may be brief, it has been suggested that the ability of players to perform repeated bouts of high intensity may be critical to the outcome of the game (Spencer et al., Roberts et al., 2008; Austin et al., 2011a). Austin et al. (2011a) assessed the RHIE characteristics of players during performance using a modification of previous work by Spencer et al (2004)

with a RHIE bout being defined as three or more sprints, scrums/rucks/mauls, and/or tackle efforts with less than 21s recovery between high-intensity efforts. Outside backs were involved in significantly fewer (p<0.05) total RHIE bouts in each game (7 \pm 3) compared with the front row forwards (15 \pm 3), the back row forwards (17 \pm 4) and the inside backs (16 \pm 2). Furthermore, average duration of RHIE bouts for the front row forwards and back row forwards were 45 \pm 9 and 52 \pm 7s respectively, each of which were significantly greater than the average durations for the inside backs (26 \pm 4s, p<0.05) and outside backs (28 \pm 4s, p<0.01).

Despite characterising work to rest patterns the studies presented have not included acceleration/decelerations as high-intensity exercise therefore work to rest patterns and RHIE characteristics may have been mis-represented. As described in section 2.2.2., advances in GPS technologies allow automatic detection of RHIE characteristics, including the detection of high acceleration movements and physical contacts, thus GPS may be used to enhance knowledge of RHIE characteristics. However no research has been conducted in rugby union to examine the RHIE demands of players using GPS software.

2.3.4. Temporal patterns in performance

Investigating how performance characteristics change throughout performance may further aid the preparation of players for specific periods of the match. Roberts et al. (2008) compared movement patterns between the first and second half and found no differences between halves for total distance covered $(3020 \pm 302 \text{ vs. } 2987 \pm 359 \text{ m})$, distance covered in high-intensity running and sprinting combined ('running work; 223 ± 132 vs. 208 ± 94 m) and time spent in high intensity activity $(3:11 \pm 2:06 \text{min vs.} 2:57 \pm 1:57 \text{min})$. Analysis of the distances travelled over successive 10min periods of match-play revealed some interesting findings, with greater total distance covered in the first 10min compared with the periods of 50-60 and 70-80min. However, no differences were found between 10min time periods for distances travelled in high-intensity running, sprinting or 'running work', and there were no differences between the total, average or maximum time spent in high-intensity activities or in static exertion over the 10min periods. Previous research in football has shown that when games are broken down into 5min periods, the most intense periods of high-intensity precede a significant deterioration in high-intensity activity (Mohr et al., 2003). The notion of 'temporary fatigue', whereby high-intensity activity is significantly reduced immediately following an intense bout but subsequently recovers later in performance was not found by Roberts et al. (2008), however due to limitations associated with TMA, the quality/speed of movement and quantity of static exertion could not be determined. The findings of Roberts et al. (2008) therefore suggest that fatigue during performance may be most evident in recovery from high intensity and RHIE recovery efforts which may be characterised by an inability to maintain defensive position or run supporting lines in attack. Therefore from the findings of Roberts et al. (2008) it is suggested that 'temporary fatigue' in rugby union may be manifested in the ability to maintain 'low' intensity activities.

To date no studies have been conducted to assess the temporal movement patterns in rugby union using GPS. However, a recent assessment of 185 professional rugby league players from 28 National Rugby League (NRL) games has used GPS (5Hz SPI Elite; GPSports Systems, Canberra, Australian Capital Territory, Australia) to assess temporal movement patterns throughout a game (Austin and Kelly, 2013). Assessed by m.min⁻¹, mean work rate for the complete game was 85 ± 4 and 86 ± 5 m.min⁻¹ for forwards and backs, however mean work rate ranged between 75 to 97m.min⁻¹ and 72 to 100m.min⁻¹ for forwards and backs respectively. Furthermore, the maximum work rate of a 10min block of match-play was 115 and 120m.min⁻¹ for forwards and backs respectively. The study demonstrated that professional rugby league players experienced decrements in most physical performance measures during the second-half of match-play compared to the first half. They noted an 18 and 15% decrement in m.min⁻¹ for forwards and backs for the last 10min of the 1st half compared to the 1st 10min (Figure 2.4). The second half of match-play also had a smaller percentage in performance decrement (13% forwards and 8% backs) from the first to the last 10min periods of play.



Figure 2.4. Mean (SD) m.min⁻¹ per 10min of match-play. *significantly (p<0.05) different from forwards, ^significantly (p<0.05) different from 0-10min, #significantly (p<0.05) different from 10-20min, ~significantly (p<0.05) different from 40-50min (Austin and Kelly, 2013).

Kempton et al. (2013a) divided rugby league performance into 5min sections and also found that distances travelled decreased throughout each half, with meterage greater in the 0-5 and 40-45min periods compared to the 30-35, 35-40, 70-75 and 75-80min periods (p<0.001). However Kempton et al. (2013a) found that reductions in movement were also associated with a reduction in technical performance. Similar to the findings of Mohr et al. (2003) the authors found that following the peak 5min of exercise intensity there were reductions in meterage (p<0.001), quality of skill movements (p<0.001), number of involvements (p<0.001) and collisions (p<0.001).

Coaches should also be aware of the influence physical preparedness may have on transient fatigue during performance. Mooney et al. (2013) separated Australian rules footballers into two groups following completion of the Yo-Yo intermittent recovery test level 2 (YYIRT2) to compare match-play movement characteristics and found that although reductions in m.min⁻¹, high-speed m.min⁻¹ and load.min⁻¹were observed in both groups from the 1st to the 4th quarter, a significant decline in low-speed m.min⁻¹ was observed from the 1st to the 4th quarter in the group which performed worse in the YYIRT2 only. The authors also found that YYIRT2 was significantly related to m.min⁻¹, high-speed m.min⁻¹ and load.min⁻¹ throughout the whole match. Mooney et al. (2013) suggest that reductions in low speed compared to high speed movements may be evidence of 'pacing' whereby players sacrifice distances covered at low speeds to compensate for the demands of high speed movement. Similar to the findings of Roberts et al. (2008) it may therefore be suggested that player load.min⁻¹ and low-speed m.min⁻¹ may be the most sensitive to detect fatigue during performance (Mooney et al., 2013).

2.3.5. Variance and trends in movement associated with performance

As discussed in section 2.3.4., changes in markers of movement have been reported throughout match-play in rugby union (Roberts et al., 2008). However the between match variation of movement has not been assessed. Longitudinal research across 23 NRL matches showed that despite total running distance showing little variation (CV 3.6%), between match variability for high-speed (>15km.h⁻¹; CV 14.6%) and very high-speed (>21km.h⁻¹; CV 37.0%) running was much greater, suggesting that there may be great between game variations in movement demands (Kempton et al., 2013b). Furthermore, when matches were analysed in halves and 10min periods (e.g. 0-10min) the extent of between-match variation was increased for total distance, high-speed running and very-high-speed running. Although no research has been performed in rugby union, variances in movement as observed by

Kempton et al. (2013b) would have great implications for understanding movement demands. However, to understand why variances in movement may be observed between and within matches, research is required (Kempton et al., 2013b).

Studies comparing the effect of the opposition, possession, territory, success and playing standard on movement characteristics in rugby league (Gabbett, 2013a; Gabbett, 2013b; Gabbett et al., 2013) and Australian rules (Aughey et al., 2011b; Sullivan et al., 2014) have demonstrated trends which may be applied to further understand movement characteristics, which may enhance physical preparation and rehabilitation (Kempton et al., 2013a). For example, research has shown physical demands determined by GPS vary according to team playing standard (Gabbett, 2013a), the opposition (Gabbett, 2013b) and team success (Gabbett, 2013c) in rugby league. Gabbett (2013c) monitored 22 NRL players across 16 games and found players covered significantly greater (p<0.05) absolute and relative distance at high speeds when playing against bottom 4 teams than when competing against top 4 teams. Furthermore, notable differences were observed when players were competing in teams winning versus losing. For example, a greater absolute and relative number of maximal accelerations, and repeated high-intensity effort bouts were performed, and total distance per minute of match-play and relative distance at low speeds were greater when matches were won. Mean and maximum number of efforts in a repeated high-intensity effort bout was also higher in winning teams, although the recovery between efforts was shorter in losing teams. Moderate (7-17 points) and large (>18 points) winning margins were associated with greater relative distances covered and distances covered at low speeds than small winning margins (Gabbett, 2013c).

Converse to the findings of Gabbett (2013c), research demonstrates that the physical demands of defending are significantly greater than those when attacking in rugby league (Gabbett et al., 2013) and Australian rules (Sullivan et al., 2014). Gabbett et al. (2013) found moderate to large significant differences between defence and attack for distance covered (109 ± 16 m.min⁻¹ vs. 82 ± 12 m.min⁻¹), low speed distance (104 ± 15 m.min⁻¹ vs. 78 ± 11 m.min⁻¹), frequency of collisions (1.9 ± 0.7 min⁻¹ vs. 0.8 ± 0.3 min⁻¹), and repeated high-intensity effort bouts (1 every 4.9 ± 5.1 min vs. 1 every 9.4 ± 6.1 min). Repeated high-intensity efforts were also identified to occur more frequently when defending the team's own try-line and when attacking the opposition's try-line (Gabbett et al., 2013). The authors therefore suggest that specific training drills could be designed to replicate the attacking and defensive demands of different field positional zones, which may be effective in preparing players for the most demanding activities that occur in professional rugby league match play (Gabbett et al., 2013). Research in team sports also suggest that reductions in technical

competency (Sullivan et al., 2014; Bradley et al., 2013) and losing (Sullivan et al., 2014) correlate with increases in high-intensity movement. Therefore the increased physical demands of players from less successful teams may increase the risk of fatigue-related decline in performance (Sullivan et al., 2014).

Variances in movement patterns have also been identified according to the level of competition. Research from Australian rules football identifies that running demands increase in AFL finals games compared to regular season games, with players nearly doubling the number of maximal accelerations in finals (Aughey et al., 2011b). Furthermore, total player load, total distance and high-intensity running distance increased during finals games (Aughey et al., 2011b).

The findings of the presented studies in team sports (Aughey et al., 2011b; Gabbett, 2013a; Gabbett, 2013b; Gabbett, 2013c; Gabbett et al., 2013; Sullivan et al., 2014) infer great implications for strength and conditioning coaches wishing to prepare individuals for 'worst case scenarios' during match-play in rugby union however research is required. In addition to assessing the effect of the opposition, possession, territory, success and playing standard on movement characteristics in rugby union, longitudinal research could assess other factors such as the effect of the playing conditions and substitutions on movement in rugby union.

2.3.6. Summary

Variances in motion analysis techniques have made comparisons between studies difficult, nevertheless common positional trends have been reported which may enhance understanding of the demands of performance.

With advancements in GPS technologies, several aspects of performance have been identified within the section for future research, such as RHIE. Furthermore, research in other team sports demonstrate large variances in movement which are related to several factors associated with performance, therefore to further understand the demands of performance in rugby union, research is required to establish between and within-match variations, and their potential causes.

2.4. Application of movement analysis to enhance performance and recovery in rugby union

2.4.1. Physical preparation and rehabilitation

A greater understanding of player movement characteristics such as distances covered, distances at different speeds, work to rest ratios, types of activity, types of high intensity activity, high intensity and repeated high intensity characteristics; together with anthropometric and physiological characteristics of players may give an indication of the positional requirements of performance. This in turn may facilitate the planning and implementation of training programmes that elicit appropriate and specific physiological adaptations in players (Austin et al., 2011a), and may be used to establish specific exercise protocols for rehabilitation (Coughlan et al., 2011).

Drills and tests may be developed which allow progression and ultimately provide coaches/rehabilitators a sense of preparedness of their players to meet the demands of play likely to be encountered during competition. Dobson and Keogh (2007) recommend that practitioners should take into account the variation of the reported values when planning. Furthermore trends in performance should be identified (Aughey et al., 2011b; Gabbett, 2013a; Gabbett, 2013b; Gabbett, 2013c; Gabbett et al., 2013; Sullivan et al., 2014) which may be used to establish situation specific training including 'worst-case' scenarios, which are likely to represent the maximum periods of activity coupled with the minimum periods of recovery.

As shown by Quarrie et al. (2013) and Cahill et al. (2013), individual positional analysis and repeated measures of players are likely to give more representative data of positional demands and may also provide individual 'norms' from which to establish individualised return to play protocols and/or preparedness standards.

2.4.2. 'Real-time' monitoring

Knowledge of position demands and expected movement patterns may enable coaches to monitor individual and team performance during the match (Quarrie et al., 2013). Indeed it is common for practitioners in sports such as rugby league to use real-time GPS data to inform interchange and tactical decisions (Kempton et al., 2013b). From the research described by Kempton et al. (2013a) in section 2.3.4., it is possible that analysts may be able to predict phases of reduced movement and performance quality following a period of repeated high intensity or high work rate patterns. Understanding expected typical and individual

decrements in performance (e.g. m.min⁻¹, load.min⁻¹) during 10min periods using GPS may therefore allow sport scientists to detect fatigue during performance from which tactical decisions may be made. Indeed with consideration to previous research in rugby union monitoring of work at lower intensities (e.g. cruising) may be most sensitive to detect fatigue (Roberts et al., 2008).

Despite the potential applications of real-time monitoring, consideration should be given to the findings of Kempton et al. (2013b) as discussed in section 2.3.5., which suggests temporal data may be misleading when used to inform tactical decisions due to likely between-match variation, particularly at high-intensities. Furthermore, research by Aughey and Falloon (2010) demonstrated that real time data collected by GPS units (MinimaxX, Team Sport 2.0, Catapult Innovations) during two elite Australian rules matches showed low concurrent validity against data analysed post-game, with the range of error increasing at higher-speeds. Although it was unclear as to the source of error, it is possible that interference in the real time signal may be a partial cause of error. Therefore despite its potential applications, the authors recommend caution when using real time data to monitor performance (Aughey and Falloon, 2010).

2.4.3. Recovery and player management

Analysis of movement demands in rugby union may not only provide information regarding the physiological demands of competition, but may give an indication of the mechanisms of fatigue and the subsequent recovery response. It has been proposed and demonstrated that increases in muscle damage from competing in rugby union are largely determined by the extent of mechanical damage induced through physical contacts (Takarada et al., 2003; Cunniffe et al., 2009). Following assessment by TMA, previous research has shown correlations between creatine kinase (CK) level and the number of tackles at 24h (r=0.922, p<0.01; Takarada et al., 2003), and number of contact events (r=0.65, p<0.05) and tackles (r=0.63, p<0.05) at 14h (Cunniffe et al., 2009). These findings may have great application prescribing when recovery strategies and planning training loads following competition. However, despite exhibiting high correlations, these studies have not accounted for the extent of running performed. Furthermore, Takarada et al. (2003) only assessed incidents when a player was tackled or when tackles were made in front of the player therefore a large number of contact events were not accounted for.

Using TMA, Smart et al. (2008) investigated whether changes in CK level were correlated to various contact and movement statistics. Twenty three players from a National Provincial Championship team (NPC) in New Zealand volunteered to take part in the study which observed CK levels from pre to post game in five league games. Transdermal exudate samples were taken pre-game (approximately 210min prior to the start of each game) and post-game (as soon as possible after the completion of the event, within a maximum time window of 30min). Game statistics and video analysis were drawn from a semi-automated vision based tracking system (AnalySports, Version AS10.0307, 2002, Palmerston North, NZ). The identified statistics included game time, time defending, tackles made, hit-ups, first three players on attack (being one of the first three players at the breakdown while their team is attacking), first three players on defence (being one of the first three players at the breakdown while their team is defending) and the number of scrums. In addition to these statistics, total impacts (sum of tackles made, hit-ups, first three on attack and first three on defence) and collisions per minute (impacts.min⁻¹) were also calculated. These specific game statistics were deemed to be important in determining the relationship between impact and CK and were calculated for each individual player across each game. An average of 3.4 data points were collected for each player. As Table 2.3 shows, both game time and time defending were significantly correlated with changes in CK for both the forwards and backs.

Table 2.3. Weighted between-subject correlation coefficient (r) for game statistics with respect to the change in CK from pre-game to post-game (mean games per player = 3.4) for 23 elite rugby union players (Smart et al., 2008).

	Forwards (n = 12)	Backs (n = 11)
Game time (min)	0.63*	0.82*
Time defending (min)	0.74*	0.72*
Tacties made	0.34	0.11
Hit ups	0.16	0.74*
First three (attack)	0.47	0.79*
First three (defence)	-0.06	0.50
Impact (I)	0.34	0.71*
Collisions per min (Vmin)	-0.03	0.47
Scrum number	0.73*	1
Scrum totat	0.29	I
*Statistically significant	(p<0.05).	

First three (attack), first three players to nuck/maul on attack; First three (defence), first three players to ruck/maul on defence.

With multiple factors therefore likely to contribute to the initiation of muscle damage, backwards random-effects maximum likelihood regression equations were developed by the authors for forwards and backs in attempt to estimate changes in CK levels following performance. The predicted CK values were moderate to highly significantly correlated with the mean CK level for forwards (r=0.69; p<0.01) and backs (r=0.74; p<0.01), however with research showing muscle damage and/or neuromuscular fatigue for several days following plyometric exercise (Duffield et al., 2010; Tofas et al., 2008), repeated sprint activity (Howatson and Milsk, 2009; Twist and Eston, 2005), non-contact team games (Ascensao et al., 2008; Fatouros et al., 2010) and long distance endurance events (Avela et al., 1998; Avela et al., 1999; Howatson et al., 2009), it is unlikely that the measure of game time or time defending is likely to account for specific positional and individual variances in movement patterns (Cahill et al., 2013; Ouarrie et al, 2013). Indeed, correlations have been shown between high-intensity running characteristics and changes in CK (Nedelec et al., 2014; Thorpe and Sunderland, 2012; Young et al., 2012), muscle soreness (Nedelec et al., 2014) and neuromuscular function during the recovery period in team sports (Duffield et al., 2012; Nedelec et al., 2014). For example, Thorpe and Sunderland (2012) found that changes in CK (Δ CK) from pre- to post-match correlated to the number of sprints (>5m.s⁻¹; r=0.86), sprint distance (r=0.89) and high-intensity distance covered (>4.167m.s⁻¹; r=0.92) by 7 players during in a semi-professional football match.

Movement associated with acceleration and deceleration have also been shown to be correlated to the extent of muscle damage (Young et al., 2012), muscle soreness (Nedelec et al., 2014) and neuromuscular fatigue (Nedelec et al., 2014) post-match in team-sport. Nedelec et al. (2014) assessed the movement of 10 professional football players across 4 matches, comprising 14 data files for analysis by TMA. Post-match, the authors found significant correlations between the number of sprints performed <5m during the match and increases in muscle soreness at 48h (r=0.74, p<0.01) and 72h post-match (r=0.57, p<0.05). Furthermore, a significant relationship was established between the decrement in countermovement jump performance at 24h and the number of hard changes in direction performed during the match (r=0.55, p<0.05).

Research in team sports suggests that recovery is to some extent dependent on high-intensity movement and physical contacts therefore monitoring the number and extent of both may have important implications for player management during recovery in rugby union. With advancements in GPS technology, quantification of movement and physical contacts by GPS should give great insight to physiological load during performance. Indeed, despite validation issues with accelerometer impact assessment as previously discussed, research

suggests that impacts recorded (SPI-Pro, GPSports, Canberra, ACT, Australia) during rugby league performance is correlated neuromuscular and biochemical changes post-match (McLellan et al., 2011a; McLellan et al., 2012). McLellan et al. (2011a) showed that the number of impacts recorded by fifteen players in zone 5 (8.1-10.0G) and zone 6 (>10.1G) during match-play was significantly correlated (p<0.05) to Δ CK at 24, 48 and 72h post an elite rugby league match. Furthermore, impacts in zones 5 and 6 were significantly (p<0.05) correlated to changes in countermovement jump peak rate of force development and peak power 24h post-match (McLellan et al., 2012).

Prior to utilising GPS technologies for enhancing player management in professional rugby union, research assessing the effect of contacts and movement from match-play on recovery needs to be conducted. However, some considerations need to be made when making predictions of recovery from match-play, for example physical preparedness may influence the extent of muscle damage and fatigue following match-play. Research has shown that runners who had the greatest reductions in running pace during a marathon had significantly elevated post-race myoglobin, lactate dehydrogenase and CK levels in comparison with marathon runners that preserved their running pace reasonably well throughout the race (Del Coso et al., 2013). Additionally, recent research demonstrates that post-match fatigue in rugby league is lower in players with well-developed high-intensity running ability and lower body strength, despite these players exhibiting greater internal and external match loads (Johnston et al., 2014a). Thus, for two players who perform an equal volume of work in rugby union, the player with the greater levels of physical preparedness may not display elevations in muscle damage or fatigue as great as one less prepared. Furthermore, as described above, Mooney et al. (2013) showed that players with lower levels of physical preparedness may exhibit greater reductions in movement throughout match-play; therefore increases in work done may not therefore be correlated to the extent of fatigue.

2.4.4. Summary

Research demonstrates that advancements in motion analysis, in particular GPS technologies, may be of great importance for player preparation and management. Knowledge of expected position and individual 'norms', and how they may vary according during performance, may be used to devise training drills to provide a sense of preparedness for performance. Research also suggests that GPS could be used to prospectively manage players post-match, as previous research demonstrates correlations between muscle damage and physical contacts; however no research has been performed to correlate movement

changes and markers of muscle damage or physical function following performance, therefore further research is required.

'Real-time' GPS analysis may also be used for tactical purposes during match-play; however practitioners should be consider the current limitations of real-time analysis.

3. Sleep, performance and recovery

Sleep is a basic requirement for human health and is recognised as an important component of athlete recovery and performance due to its physiological and psychological restorative effects (e.g. Leeder et al., 2012b). For example, sleep is associated with autonomic nervous system and hormonal alterations which facilitate the restoration and development of physiological processes (Datillo et al., 2010) and contributes to the consolidation and enhancement of memory and motor skill learning (Walker and Stickgold, 2004). However, the importance of sleep is most evident when restricted with studies reporting sleep disruption may have implications for autonomic nervous system (e.g. Spiegel et al., 1999), hormonal (e.g. Spath-Schwalbe et al., 1991), biochemical (e.g. Abedelmalek et al., 2013), genetic (e.g. Moller-Levet et al., 2013), cognitive (e.g. Cook et al., 2011) and neuromuscular (e.g. Bambaeichi et al., 2005) functions. Indeed research demonstrates the detrimental effect of total (e.g. Skein et al., 2013) and partial (e.g. Souissi et al., 2013) sleep restriction on athlete performance during periods of training and competition.

The importance of good sleep practice in elite athletes has been highlighted in the literature (e.g. Leger et al., 2008); with Halson (2008) suggesting sleep may be the single most efficacious recovery strategy. However, it is been suggested that frequently disrupted and restricted sleep is a common problem for many people (Meerlo et al., 2008), with recent literature suggesting this is also the case for elite athletes (e.g. Leeder et al., 2012b).

The following section will discuss the effect of sleep disruption on several physiological and psychological functions, before assessing the literature assessing sleep patterns in elite athletes. Firstly, the human sleep cycle will be described which will help enable understanding of how sleep facilitates physiological and psychological restoration.

3.1. Stages of sleep cycle

Sleep is a functional state that comprises a complex combination of physiological and behavioural processes. It has some characteristic manifestations, such as a cyclic pattern, relative immobility and an increase in the response threshold to external stimuli (Santos et al., 2007). Walker and Stickgold (2004) explain that sleep has been broadly classified into two distinct types: non-rapid eye movement (NREM) sleep and rapid eye movement (REM) sleep, with NREM sleep being further divided into four sub-stages (1 through 4; Figure 3.1.). Non-REM and REM sleep alternate or 'cycle' through the night, with the ratio of NREM to REM within each 90min cycle changing so that that early in the night stages 3 and 4 of

NREM dominate, while stage 2 NREM and REM sleep prevail in the latter half of the night (Figure 3.1.).

As NREM sleep progresses, electroencephalographic (EEG) activity begins to slow in frequency, therefore the deepest stages of NREM, stages 3 and 4 are thus often grouped together under the term 'slow wave sleep' (SWS). The different stages of sleep may have different functions in the restoration and development of physiological and psychological processes, which will be discussed where relevant in the following sections.

3.2. Effect of sleep disruption on physiological and psychological function

3.2.1. Autonomic nervous system (ANS)

The autonomic nervous system (ANS) is a component of the central nervous system (CNS) controlling whole-body homeostasis by coordinating different organs and tissues in response to external challenges. The ANS divides into two distinct systems: the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS).

The SNS is associated with the 'fight or flight' response, allowing individuals to deal with stressful situations. Physical exercise causes an increase in sympathetic activity, concomitant with parasympathetic withdrawal resulting in higher heart rates (Al Haddad et al., 2009). Activation of the SNS results in the release of noradrenaline (norepinephrine) from sympathetic nerve terminals throughout the body and the secretion of adrenaline (epinephrine) from the adrenal medulla. These catecholamines play an important role in the regulation of energy balance (mobilisation and use of energy) and cardiovascular function (transport of fuel and oxygen to tissues in need of energy; Meerlo et al., 2008).

As SNS activity decreases, parasympathetic reactivation occurs to facilitate recovery and restoration of physiological functions. The time needed to restore pre-exercise ANS level has been shown to depend on both exercise intensity and duration. High-intensity exercise reduces the autonomic activity for longer time than submaximal exercise (Seiler et al., 2007; Buchheit et al., 2007) and research suggests that exercise induced muscle damage (EIMD) and the subsequent inflammatory response may temporarily attenuate parasympathetic reactivation (Jae et al., 2010). Furthermore, research shows that greater parasympathetic activity is usually associated with a better recovery state (Chen et al., 2011; Pichot et al., 2000) and readiness to perform (Buchhiet et al., 2009; Garet et al., 2004; Kiviniemi et al., 2007) suggesting ANS regulation is important in facilitating recovery, and parasympathetic reactivation is a useful indicator when monitoring the recovery status of an elite athlete.





During sleep, the autonomic balance of cardiovascular regulation shifts to a parasympathetic dominance, with heart rate variation (HRV) analysis showing a decrease in SNS input during night-time sleep concomitant with an increase in PNS activity (Meerlo et al., 2008). The change in balance is further displayed by a rapid decline in circulating catecholamines at sleep onset, with lower levels of adrenaline and noradrenaline observed during sleep compared to wakefulness (Meerlo et al., 2008).

However sleep deprivation has been associated with an increase in sympathetic activity (Burgess et al., 1997; Spiegel et al., 1999) and catecholamine levels (Irwin et al., 1999) towards the levels seen during wakefulness. Importantly, increased sympathetic activation not only occurs with prolonged and continuous sleep deprivation but also with fragmented and interrupted sleep. A number of studies suggest that the extent of increased sympathetic activation of sleep deprivation is more related to the disruption and discontinuity of sleep than to the duration of sleep deprivation or the amount of sleep that is lost (Ekstedt et al., 2004; Irwin et al., 1999; Tiemeier et al., 2002). Furthermore, initiating and maintaining wakefulness in a state of sleep debt may require a relatively higher sympathetic activation (Meerlo et al., 2008). Thus sleep restriction may increase SNS activity both during sleep and wakefulness, which may impair athlete recovery and readiness to perform.

3.2.2. Endocrine system

Traditionally hormones have been viewed in their role in facilitating protein synthesis and degradation following exercise (Crewther and Cook, 2012a). Growth hormone (GH) plays a significant anabolic role in skeletal muscle growth via an increase in protein synthesis and reduction in protein breakdown. Growth hormones are polypeptides released from the pituitary gland which also stimulate the cell release of insulin growth factors (IGFs) (liver and muscle types; shown to stimulate myoblast proliferation and differentiation, inhibit proteolysis, increase glucose and amino acid uptake and increase protein synthesis) and stimulate the release of amino acids for protein synthesis (Kraemer and Mazzetti, 2003).

Peak plasma GH concentration generally occurs during early sleep, in temporal association with the first phase of deep SWS (Spiegel et al., 2000). The amount of GH secreted during the first SWS episode is correlated with the duration of SWS as well as with the intensity of SWS, as estimated by slow-wave activity (SWA). Thus sleep has been associated as an important process of the repair and growth of tissues (Datillo et al., 2011). However, the importance of sleep on repair and growth is perhaps most evident by the effect of sleep restriction on hormonal changes which impact skeletal muscle metabolism. For example,

individuals deprived of sleep for 72h showed higher urinary excretion of urea, suggesting greater muscle proteolysis (Kant et al., 1984). Nedeltcheva et al. (2010) also found evidence of muscle proteolysis following partial sleep deprivation. Individuals followed a restricted calorie diet for 14 days however they either slept for 5.5 or 8.5h each night. Although similar reductions were found in body mass, under the conditions of sleep restriction the decrease in fat mass was 55% lower and, interestingly, the loss of muscle mass was 60% higher. This suggests that alterations in hormone section following sleep deprivation may promote different effects in modulating repair and body composition, and that skeletal muscle mass can potentially be compromised.

The findings by Nedeltcheva et al. (2010) are likely due to a shift in the release of several hormones. For example, under situations of stress; including sleep restriction, a series of events initiated by the hypothalamo-pituitary-adrenal (HPA) axis results in the secretion of cortisol. Speigel et al. (1999) assessed the activity of the HPA axis of 11 men after time in bed has been restricted to 4h a night for 6 nights, compared to 12h sleep per night for 6 nights. The authors found evening cortisol concentration was raised in the afternoon and early evening following 6 nights of restricted sleep. Cortisol has been reported to fulfil many different functions related to coping with stress, but it also provides an important negative feedback signal to inhibit the primary stress systems and help restore the resting state after successfully dealing with a stressor. In that sense, one might consider the glucocorticoids as anti-stress hormones (Meerlo et al., 2008). However, cortisol also stimulates lipolysis in adipose cells, increases protein degradation and decreases protein synthesis in muscle cells resulting in greater release of lipids and amino acids into circulation (Kraemer and Mazzetti, 2003). Similar to the effects of sleep on the ANS, increased cortisol levels may not only occur with continuous sleep deprivation but may also occur as a result of arousal from sleep and sleep fragmentation (awakenings; Spath-Schwalbe et al., 1991).

Literature also shows that circulating anabolic hormones may be reduced following sleep deprivation (Everson and Crowley, 2004; Cote et al., 2013). Everson and Crowley (2004) showed that 15 days partially or totally sleep deprived rats exhibited lower levels of GH, IGF-1 and prolactin while Cote et al. (2013) found one night of total sleep deprivation lowered testosterone and aggression in men. Reductions in testosterone and IGF-1 concentrations may decrease the activity of pathways critical in the process of muscle cell repair and growth (e.g. IGF-1/PI3K/AKt and mTOR pathways) and may diminish the signal inhibition for myostatin expression, thereby promoting protein degradation (Datillo et al., 2011; Figure 3.2.). Additionally, research indicates that testosterone has more complex training roles outside of muscle repair and growth; in regulating adaptive physiology and physical performance itself (Crewther and Cook, 2012a). Reductions in testosterone may
impair calcium processes linked to muscle and contractile function (Curl et al., 2009; Estrada et al., 2003) and motor cortex output (Bonifazi et al., 2004). In individuals with relatively high strength levels, free testosterone is a strong individual predictor of the expression of strength and power qualities (Crewther et al., 2011; Crewther et al., 2012). Furthermore, Crewther and Cook (2012a) explain that testosterone is very important as a stress biomarker and the testosterone responses to a challenge can provide information on dominance. Indeed, winning in sports competition is often accompanied by elevated testosterone concentrations relative to losing (Elias, 1981; Fry et al., 2011; Mazur and Lamb, 1980).

A reduction in habitual sleep of as little as 1.5h over 3 weeks has also been shown to lead to changes in insulin sensitivity (Robertson et al., 2013). Reductions in insulin sensitivity following sleep restriction have been shown to reduce glucose tolerance (Skein et al., 2011; Spiegel et al., 1999) which may further explain the findings by Nedeltcheva et al. (2010) described earlier in this section. Skein et al. (2011) assessed the effect one night total sleep deprivation had on consecutive-day intermittent sleep performance (Table 3.1.) and muscle glycogen content. Ten male team sport athletes performed 2 consecutive day trials, one separated by no sleep and the other following normal sleep patterns (control), with all food and fluid during the consecutive day trials matched and standardised between the two conditions. On the second day, muscle glycogen concentration was lower before exercise following no sleep compared to the normal sleep $(209 \pm 60 \text{ mmol.kg}^{-1} \text{ dry weight vs. } 274 \pm$ 54mmol.kg⁻¹ dry weight, p=0.05) demonstrating the effect sleep restriction has on muscle glycogen synthesis. Previous research suggests that a state of reduced muscle glycogen may negatively affect exercise performance (Balsom et al., 1999); thus, Skein et al. (2011) suggest the reduction in muscle glycogen following sleep deprivation may have contributed to a decline in performance (Table 3.1.).

Sleep restriction therefore has potentially large implications for muscle repair and growth. However, the effects of sleep deprivation on HPA axis activation and increased levels of stress hormones rapidly disappear during subsequent recovery sleep (Meerlo et al., 2002). Furthermore, literature suggests that reductions in GH following restriction may be compensated by GH pulses occurring during waking periods (Brandenberger and Weibel, 2004), while evidence of GH secretion adaptation is evident from chronic sleep restriction (Spiegel et al., 2000). Nevertheless, with evidence of hormonal and glycogen synthesis disruption several days following competition (Cormack et al., 2008; Haneishi et al., 2007; Krustrup et al., 2011; McLellan et al., 2010) and increases in training volume (Argus et al., 2009), disrupted sleep may accentuate these disruptions and thus possibly prolong the stress/recovery response. Furthermore, with small reductions in sleep increasing insulin

sensitivity, chronic mild sleep deprivation may have great implications for the recovery and body composition of athletes.





3.2.3. Biochemistry, genetics and immune function

Sleep deprivation has been associated with alterations in cytokines which are responsible for maintaining immune function (Meerlo et al., 2008). Indeed, Cohen et al. (2009) demonstrated that poorer sleep efficiency and shorter sleep duration in the weeks preceding exposure to a rhinovirus (RV-39) were associated with lower resistance to illness. In this study, a total of 153 healthy men and women (21-55yrs) self-reported sleep duration and efficiency (sleep duration divided by time in bed) for 14 consecutive nights prior to being quarantined, administered nasal drops containing a rhinovirus, and monitored for the development of a clinical cold (infection in the presence of objective signs of illness) for 5 days after exposure. Participants with less than 7h sleep during the two weeks prior to exposure to a virus were 2.94 times more likely to develop a cold than those with 8h or more of sleep. Furthermore, participants with less than 92% sleep efficiency during the pre-exposure period were 5.5 times more likely to develop a cold than those with greater than 98% sleep efficiency.

Partial sleep deprivation has also been shown to alter pro-inflammatory response to an exercise stressor. For example, Abedelmalek et al. (2013) compared two sessions at 08:00h following a baseline night sleep (22:30-07:00h) or a night of partial sleep deprivation (22:30-03:00h). During each session 30 healthy footballers performed 4x250m runs on a treadmill at constant intensity of 80% of personal maximal speed with 3min recovery between each run. Results showed that plasma concentration of interleukin-6 (IL-6) and tumour necrosis factor- α (TNF- α) were higher (p<0.05) following partial sleep deprivation following the first and last run, and remained elevated 60min following exercise. Thus, sleep deprivation may exacerbate the stress response to exercise. Recently, Moller-Levet et al. (2013) demonstrated that reductions in immunity and stress response as a consequence of sleep restriction may be due to alterations in genetic homeostasis. The authors found that 1 week of sleep restriction $(5.70 \pm 0.03h)$ changed the regulation of 711 genes associated with immunity, inflammation and stress responses, when compared to 'sufficient sleep' $(8.50 \pm 0.11h)$. Genes effected also have implications for circadian rhythms, repair/growth, sleep homeostasis and metabolism. With circadian rhythms controlling endocrine and metabolic rhythms, circadian disruptions may manifest the consequences of sleep restriction (Moller-Levet et al., 2013). Furthermore, physiological alterations following sleep restriction (as discussed previously) may further predispose individuals to health problems. For example, increased sympathetic activity has been associated with cardiac illness and disease (Buchheit et al., 2007), while decreased tolerance increases risk factors for development of insulin resistance, obesity and hypertension (Spiegel et al., 2010).

3.2.4. Learning and motor skill retention

Sleep has also been shown to contribute to the consolidation or enhancement of memory and motor skill learning; however sleep deprivation may not only impair this process, but may result in a loss of previously formed experience-dependent memories (Walker and Stickgold, 2004). Previous research has demonstrated that memory consolidation (i.e. previous memory becomes more stable) is initiated by SWS-related processes, while learning is related to REM sleep towards the end of sleep (Gais et al., 2000). Indeed, several neuroimaging studies have explored the possibility that patterns of brain activity elicited during initial task training are 'replayed' during subsequent sleep (e.g. Walker and Stickgold, 2004). Macquet et al. (2000) showed that activation patterns elicited during practice of a serial reaction time motor skill task prior to sleep reappeared during subsequent REM sleep episodes. Furthermore, when re-tested the next morning, subject's performance had improved significantly relative to the evening training sessions. Subsequent research also shows that the extent of improvement during training has a direct relationship with the amount of subsequent reactivation during REM sleep (Peigneux et al., 2003). Thus sleep, or the lack of, has an important role in the skill and tactical development (e.g. motor skills, learning playing patterns and learning from playing experiences), with the consolidation and enhancement of memories associated with SWS and REM/stage 2 NREM sleep respectively.

3.2.5. Physical performance and recovery

Due to acute or chronic sleep restriction, one may assume that alterations to physiological function (as described in previous sections) may impair performance and recovery from exercise. Indeed, although the exact mechanisms are unclear, evidence exists (Table 3.1.) to show that one night of total sleep deprivation impairs anaerobic performance (Soussi et al., 2003) and recovery from anaerobic/alactic (Edge et al., 1999; Skein et al., 2013), aerobic (Oliver et al., 2009) and intermittent (Edge et al., 1999; Skein et al., 2011) performance. However, evidence to support the effect of partial sleep deprivation on physical performance and recovery is less conclusive. Mejri et al. (2013) found partial sleep deprivation at the beginning or end of the night had no effect on YoYo Intermittent Recovery Test Level 1 (YYIRT1) performance in 10 male Taekwondo players, while Abedelmalek et al. (2013) and Souissi et al. (2008) found no effect of partial sleep deprivation at 08:00 and 07:00h respectively the next day on repeated bike sprint performance. However, Abedelmalek et al. (2013) found that sprint peak power and mean power decreased at 18:00h following a night when sleep was deprived at the end (4h). Indeed research suggest that the greatest effects of sleep deprivation on physical performance are more pronounced in the afternoon/early

evening, and when sleep is restricted at the end of the night as opposed to the start of the night (Bambaeichi et al., 2005; Haj Salem et al., 2013; Soussi et al., 2008; Souissi et al., 2013; Table 3.1.). For example, Souissi et al. (2013) assessed the effects of partial sleep deprivation on short-term maximal performances of judokas in the morning and afternoon of the following day. Twelve male judokas performed tests to assess maximal voluntary contraction of the elbow flexors, maximal handgrip strength and power from a 30s Wingate test at 9:00 and 16:00h the day before and following a reference night sleep (23:00 to 06:00h), when sleep was deprived (4h) at the beginning of the night (PSDB) and when sleep was deprived at the end of the night (PSDE). Following the reference night sleep, muscle power and strength was significantly greater at 16:00 than 09:00h (p<0.05), however partial sleep deprivation blunted diurnal increases in strength and power. In addition, PSDE resulted in significant decreases of short-term maximal performance in the afternoon (p<0.01) compared to the reference night sleep.

Research therefore suggests; despite equivocal evidence, that partial sleep deprivation may impair subsequent performance, with detriments to performance greater when sleep is deprived at the end of the night. It is unclear why the effects of sleep deprivation are most evident in the afternoon/early evening, however one possible reason may be due to the effect of sleep restriction on circadian rhythm patterns (Moller-Levet et al., 2013), as time-of-day peaks in athletic performance have generally been reported to occur during this period (Drust et al., 2005).

With exception to Sinnerton and Reilly (1992) and Cook et al. (2011), studies have assessed the effect of only one night partial sleep deprivation. Cook et al. (2011) found basic skill performance was significantly impaired by 5 nights of partially deprived sleep (3-5h) compared to conditions of normal sleep (7-9h), however Sinnerton and Cook (1992) found that 2.5h sleep did not affect maximum strength or swimming performance over 4 consecutive days compared to conditions of normal sleep. As discussed in section 3.2.3., sleep restriction of 5.7h (N.B. longer duration than any of the studies listed in Table 3.1.) for 1 week resulted in up or down-regulation of 711 genes (Moller-Levet et al., 2013), thus future research over longer periods of partial sleep deprivation may give greater insight to the effects of partial sleep deprivation. Table 3.1. Effect of sleep restriction on physiological performance and recovery.

Key findings	Compared to CON, PP and MP were not affected by PSDE at 08:00h. However, PP and MP decreased after PSDE at 18:00h.	In both conditions a diurnal variation was observed in peak torque of knee flexors; values at 18:00h were 4.5 and 5.9% higher at 1.05 and 3.14rad.s ⁻¹ respectively than at 06:00h. No effect of partial sleep loss or interaction effect was observed for muscle strength measures. However, the performance rhythms were in phase with the circadian rhythm in rectal temperature.	PSD resulted in significant fall in skill performance accuracy on both the dominant and non-dominant passing sides (p<0.001).
Methods	Cross over study. Performance tests (30s Wingate Test) conducted at 08:00 and 18:00h following normal sleep (CON) or 4h PSD when players were woken early (PSDE).	Cross over study. Measurements of muscle strength (isokinetic and isometric peak torque of knee extensors and flexors) were carried out at 06:00 and 18:00h after the control night and following PSD (2.5h). Rectal temperature was measured 30min prior to strength testing.	Cross over study, players had between 7-9h sleep on 5 passing skill trial days and between 3-5h sleep on the other 5.
Subjects	12 football players	8 sedentary eumenorrheic females	10 elite rugby players
Extent of sleep restriction	PSDE	PSD	PSD
Aim	Investigate effect of PSD on short- term maximal performance.	Assess the interaction between the effects of partial sleep loss and time of day on muscle strength.	Investigate effect of caffeine and creatine supplementation on skill execution following PSD
Reference	Abedelmalek et al. (2013)	Bambaeichi et al. (2005)	Cook et al. (2011)

Sprint and bound performance significantly impaired during the exercise protocol. MVC pre-exercise was significantly lower in TSD condition compared to CON. Self-paced jogging was non-significantly less following TSD, suggesting TSD had a negative effect on pacing.	PP and MP from Wingate significantly decreased in both conditions however values were lower at both time points following PSDE.	PSDB or PSDE did not affect intermittent aerobic performance. Following TSD, participants ran 187m less	compared to CON, suggesting 15D had negative effect on pacing and/or aerobic performance. No decrements observed in strength (back and grip strength) or swimming time (4x50m trials, 1x400m trial).
Cross over study (TSD vs. CON). Athletes performed a graded vVO _{2peak} test on a treadmill followed by a 50min intermittent exercise that included 50x15m maximal sprints interspersed by fast running, jogging, walking and plyometric bounds in a gymnasium.	32h from previous sleep) or CON. Pre and post exercise 15 MVC of right knee extensors assessed. Cross over study. Performed in a session after PSDE and CON. During each session they	hand grip test before and after (1hr) judo match. Cross over study. YYIRT1 performed following PSDB, PSDE or CON. Cross over study. 30min	treadmin run periorned before and following TSD (approximately 30h) or CON (8.3h). Eight swimmers were tested on 4 consecutive days, morning (6:30h) and evening (17:30h), under conditions of normal sleep (CON) and under PSD (2.5h).
9 male athletes from Intermittent sports (New Zealand)	21 Judokas	10 male taekwondo players 11 male	athletes 8 swimmers
TSD	PSDE	PSDB, PSDE TSD	PSD
Investigate effect of TSD on maximal strength and intermittent high intensity exercise.	Examine effects of PSDE on anaerobic performances and strength in judokas	Study effects of PSDB and PSDE on taekwondo players' intermittent aerobic performance. Examine effect of TSD on aerobic	performance Investigate the effect of PSD on strength and swimming performance.
Edge et al. (1999)	Haj Salem et al. (2013)	Mejri et al. (2013) Oliver et al.	(2009) Sinnerton and Reilly (1992)

Mean sprint times were slower following TSD compared with previous day and CON trial.	reduced during initial 10min in TSD group compared to CON displaying a negative effect of	TSD on pacing.		Reduction in CMJ height greater from post to 16h	post following TSD compared to CON (p=0.10-0.16)					Performance unaffected following 24h sleep	deprivation, however TSD resulted in decrements	in peak, mean and max power at 36h.	Performance improved in all 3 conditions from	morning to afternoon in all tests.	No difference between CON and PSDB.		However, morning to afternoon improvement was	smaller following FSDE condition compared to CON and PSDB.
Cross over study. Two consecutive day trials separated by either normal (CON) or no sleen (TSD: annrox 30h)	Each session included a 30min	graded exercise run and 50min intermittent-sprint exercise protocol. including a 15s	maximal sprint every minute and self-paced exercise bouts of varving intensities.	Cross over study. Players were	tested morning of match, immediately nost-match 2h	post and the following morning	(16h post-match). Following the	match players either had normal	(CON; approx. 8h) or deprived sleep (TSD).	Cross over study (TSD vs.	CON). Performance tests (6 and	30s bike sprints) carried out at 06:00 and 18:00h.	Cross over study (PSDB, PSDE	vs. CON). Performance tests (6 and 30s hike sprints) carried out	at 07:00 and 18:00h.			
10 male sprint athletes				11 male amateur	rugby league nlavers					13 males			11 subjects	(4h				
TSD				TSD						TSD			PSDB,	PSDE sleen)				
Determine effects of TSD on intermittent sprint performance.				Effect of TSD on physiological	recovery following competitive					Determine effect of one night's	sleep deprivation on anaerobic	performance the following day.	Determine whether delaying	bedtime or advancing rising time by 4h affects anaerohic	performance of individuals the	following day in the morning and	afternoon.	
Skein et al. (2011)				Skein et al.	(2013)					Souissi et al.	(2003)		Souissi et al.	(2008)				

Muscle power and strength significantly higher at 16:00 than 9:00h (p <0.05). These diurnal variations disappeared after PSDB and PSDE and after combat. In addition, PSDE resulted in significant decreases of short-term maximal performance in the afternoon (p <0.01). PSDE might therefore blunt diurnal variations of short-term maximal exercise.	Thus, early rising is more detrimental than late bedtime to muscle strength and power for judo athletes when competitions are scheduled in the afternoon hours.	After TSD, exercise time to task failure was shorter (1137 \pm 253s vs. 1236 \pm 282s, p<0.05) than CON. Maximal peripheral voluntary activation decreased by 7% (p<0.01) and cortical voluntary activation tended to decrease by 5% (p=0.059) with exercise.
Cross over study (PSDB, PSDE vs. CON). Maximal voluntary contraction of the elbow flexors, maximal handgrip strength and 30s wingate tests before and after a judo combat performed at 9:00 and 16:00 the day before, and following either CON (23:00-06:00), PSDB or PSDE (4h deprived at beginning or end).		Cross over study. On day 1, subjects performed baseline neuromuscular testing. After one night of TSD or CON, subjects repeated day 1 testing and then performed 40min submaximal cycling and a cycling task to failure. Neuromuscular functions were evaluated during the cycling protocol and at task failure.
12 judokas		12 active males
PSDB, PSDE		TSD
effects of time of day and ep deprivation on short- mal performance of judo rs		e effects of sleep n on neuromuscular
Examine e partial sle term maxi competitor		Determine deprivation function.
Souissi et al. (2013)		Temesi et al. (2013)

Research to examine the long-term effect of sleep fragmentation on performance is also required due to the effects sleep fragmentation may have on ANS (Ekstedt et al., 2004) and hormonal (Spath-Schwalbe et al., 1991) function. Furthermore, many of the studies listed in Table 3.1. use 'normal' night's as control values, thus comparisons may actually be made to sleep deprived nights. Future research therefore needs to monitor the duration and quality of both deprived and control sleep.

3.2.6. Psychological performance and recovery

Research demonstrates that sleep deprivation may negatively impair cognitive function (Table 3.2.). For example, studies assessing the effect of total sleep deprivation on cognitive performance have shown impairments to reaction time (Doran et al., 2001; Skein et al., 2013; Van Dongen et al., 2003), memory tasks (Van Dongen et al., 2003) and subjective ratings of sleepiness (Van Dongen et al., 2003) (Table 3.3.). Furthermore, partial sleep deprivation has been shown to have similar effects (Table 3.3.) with the extent of impairment sleep-dose-dependent (Belenky et al., 2003; Van Dongen et al., 2003). Van Dongen et al. (2003) allocated individuals to either 4, 6 or 8h sleep over 14 days or total sleep deprivation over 3 days, and compared the findings to 3 days baseline sleep. The authors found the greatest cognitive effects were observed following 3 days total sleep deprivation, whereas 4 and 6h sleep per night resulted in significant cumulative, dose-dependent deficits in cognitive performance. Similarly, Belenky et al. (2003) found acute dose-dependent reductions in reaction time when subjects slept for 3, 5 and 7h sleep for a week, however decreases in reaction time stabilised at a sub-optimal level after several days in the 5 and 7h sleep conditions, suggesting some adaptation at a compromised cognitive state (Figure 3.3.).

Following all restriction conditions, subjects slept for 8h each night for 3 days; however baseline cognitive function was not restored. Therefore in contrast to research by Sallinen et al. (2012) (Table 3.3.), these findings suggest baseline cognitive function may take several days to recover following sleep restriction (Figure 3.3.; Belenky et al., 2003).

Table 3.2. Summary of cognitive performance effects of sleep deprivation (Goel et al., 2009).

- Involuntary micro-sleeps occur.
- Attention-intensive performance is unstable with increased errors of omission (lapses) and commission (wrong responses)
- Cognitive slowing occurs in subject-paced tasks, whereas time pressure increases cognitive errors.
- Psychomotor response time slows.
- Both short-term recall and working memory performances decline.
 - Reduced learning (acquisition) of cognitive tasks occurs.
 - Performance requiring divergent thinking deteriorates.
- Response suppression errors increase in tasks primarily sub-served by the prefrontal cortex.
 - Response perseveration on ineffective solutions is more likely to occur.
- Increased compensatory effort is required to remain behaviorally effective.
- Tasks may begin well, but performance deteriorates as task duration increases.
- Growing neglect of activities judged to be nonessential (loss of situational awareness)

occurs.



Figure 3.3. Mean number of lapses on Psychomotor Vigilance Task (and standard error) across days as a function of time in bed group (Belenky et al., 2003).

Table 3.3. Effect of sleep restriction on cognitive performance and recovery.

Ì

Findings	Seven days of sleep restriction degraded performance in a sleep-dose-dependent mau With mild to moderate sleep restriction (5 to performance initially declined and, after a days, appeared to stabilise at a lower than bass level for the remainder of the sleep restri- period. However levels did not recover di recovery period. Speed and lapses in PVT remained at baselii 9h group, while in 3h group continued to deci throughout period. During recovery, PVT li returned to those similar to 5 and 7h sleep in 3h group.	Subjective sleepiness and mood were imp following sleep-fragmentation nights. No performance deficits were apparent; how EEG and event-related potentials (electrodes) data illustrated impairment information-processing capabilities assoc with reduced arousal. Note total sleep time was not reduced follo fragmented night sleep.
Methods	Allocated to one of four conditions: 3, 5, 7 or 9h sleep for 7 days following 3 days of 8h sleep. Subjects were assessed on PVT and subjective alertness/ sleepiness throughout the study. Following 7 day period subjects had 8h recovery sleep for 3 days.	Participants spent 4 consecutive 24h periods in the laboratory. On nights 2 and 3, sleep was fragmented using auditory stimuli that were delivered with increasing intensity until an arousal was noted (EEG). All sleep periods were 8h in duration. During the day, participants performed a 40min computerised test battery at 2h intervals, which included measures of mood, sleepiness, reaction time, and serial addition or subtraction.
Subjects	66 volunteers	×
Extent of sleep restriction	PSD	PSD/FRA
Aim	Determine the effects of several levels of restricted and one level of augmented sleep over seven consecutive days on objective and subjective alertness and objective performance. Determine extent to which 3 days of subsequent recovery sleep restored performance and alertness.	Effect of sleep fragmentation on daytime sleepiness.
Reference	Belenky et al. (2003)	Cote et al. (2003)

PVT reaction time mean and SD increased ar subjects and within each individual subjects i TSD condition.	During TSD, variability in PVT perform reflected a combination of normal ti responses, errors of omission (i.e. lapses), errors of commission (i.e. responding whe stimulus was present).	Significant effect of PSD on parametranslating to an increased RT and reduced of attention.	RT more affected by PSDE than PSDB, how SA and CA more affected by PSDB than PSD	Multi-tasking performance, self-perceived performance, sleepiness and mental fatigue	baseline during the recovery phase.		Decline in incongruent word-colour rea times were increased following TSD compar CON.
13 subjects tested every 2h on 10min, sustained-attention, PVT throughout 88h of TSD.	CON groups (n=15) conduced the same tests but were permitted a 2h nap every 12h.	3 cognitive tests performed following PSDB, PSDE and CON.		Split into TSD (n=13) and CON (n=7) group.	On the first 2 nights, PSD group had an 8h sleep opportunity that was restricted to 4h for the next 5 nights, and then restored to 8h for last 2 nights.	Participants accomplished 50min multitask session and gave self- ratings.	Cross over study. Players were tested morning of match, immediately post- match, 2h post and the following morning (16h post-match). Following the match players either had normal (CON; approx. 8h) or deprived sleep (TSD).
28		, 12 males		20			11 male amateur rugby league players
TSD		PSDB PSDE		PSD			TSD
Investigate variability in performance as a function of sleep performance.		Effect of PSD on reaction time and attentional capacities in handball.		Investigate effect of sleep restriction on multi-tasking			Effect of TSD on perceptual recovery following competitive rugby league.
Doran et al. (2001)		Jarraya et al. (2013)		Sallinen et al. (2012)			Skein et al. (2013)

Temesi et al. (2013)	Determine effects c deprivation on cognition.	of sleep	TSD	12 active males	Cross over study. On day 1, subjects performed baseline cognitive tests. After one night of TSD or CON, subjects repeated day 1 testing and then performed 40min submaximal cycling and a cycling task to failure. Cognitive functions were evaluated during the cycling protocol and at task failure.	After TSD, RPE during 40min submay cycling was greater (p <0.01) than in CON. N reaction time was 8% longer (p <0.05) cognitive response omission rate before cy was higher (p <0.05) than in CON.
Van Dongen et al. (2003)	Investigate whether slee chronically reduced consequences.	ep can be without	TSD, PSD	48 healthy adults	Individuals randomised to one of three sleep doses (4, 6 or 8h per night) for 14 days, or TSD for 3 days. All data was compared to 3 baseline (CON) days.	No cognitive deficits occurred following 8h in bed. Greatest cognitive deficits observed followidays TSD.
					Cognitive tests (PVT, a working memory task, and cognitive 'throughput' task) were performed every 2h from 7:30am to 11:30pm.	Chronic restriction of sleep periods to 4 or 6 night over 14 consecutive days resulte significant cumulative, dose-dependent defic cognitive performance on all tasks.
						Subjective sleepiness ratings showed an a response to sleep restriction but only small furincreases on subsequent days, and did significantly differentiate the 4 and 6h conditional significantly differentiate the 4 and 6h conditional significantly differentiate the 4 and 6h conditional subsequent days are shown as the statement of the statement
						Cognitive deficits accumulated much rapidly when no sleep was allowed than whe same amount of sleep was lost more grad over days of PSD.
						Regardless of the mode of sleep deprive lapses in behavioural alertness were near-lin related to the cumulative duration of wakefu in excess of 15.84h.
						Sleepiness ratings suggest that subjects largely unaware of these increasing cogn deficits.

Research suggests that cognitive performance may be impaired due to increased perceived task difficulty (Sallinen et al. 2012), and performance instability which includes compensatory effort (Doran et al., 2001). Doran et al. (2001) observed variability in psychomotor vigilance test performance during sleep deprivation, which was reflected by a combination of normal timely responses, errors of omission (i.e. lapses), and errors of commission (i.e. responding when no stimulus was present). Furthermore, errors of omission and errors of commission were highly inter-correlated across deprivation in the total sleep deprivation condition (r=0.85, p=0.0001) suggesting that performance instability is more likely to include compensatory effort than a lack of motivation.

Sleep restriction may also have detrimental effects to well-being. Under conditions of restricted sleep, the first signs of alterations in the way people deal with challenges appear to be on the level of emotional perceptions (Meerlo et al., 2008). Sinnerton and Reilly (1992) assessed the physiological and psychological effects of partial sleep deprivation (2.5h a night) over 4 nights compared to 4 nights normal sleep for 8 swimmers and found no decrements in back or grip strength, lung function or 50 and 400m swimming trial times. However, the authors found sleep loss affected self-reported mood states, including increased depression, tension, confusion, fatigue and anger, while decreasing vigour significantly. Although no physiological effects were observed following sleep restriction by Sinnerton and Reilly (1992), more recent research has demonstrated that physiological function and recovery may be somewhat determined by the psychological response to a stressor (Cook and Crewther, 2012a; Cook and Crewther, 2012b). Thus increased mood disturbance as a result of sleep restriction may affect playing performance and subsequent recovery.

Despite the majority of research focussing on total sleep time as a measure of sleep quality, measures of fragmentation (e.g. awakenings) may provide important information when assessing sleep quality. Cote et al. (2003) found that when participants were woken at regular intervals throughout a sleep period, subjective sleepiness and mood, EEG activity and arousal (scalp electrodes) were impaired when performing computerised tasks, possibly due to a reduction in SWS and stage 2 NREM sleep. Lastella et al. (2012) therefore refers to 'disrupted sleep'; which represented both periods in which sleep has been partially restricted (e.g. sleep deprivation) and/or where sleep has been fragmented (e.g. awakenings during a sleep period).

The effect of sleep deprivation at the beginning or end of a night on psychological function has not received much attention relative to physiological function; nevertheless Jarraya et al. (2013) found that reaction time of 12 male handball players was more effected by sleep restricted at the end of the night. However, the authors found greater disruptions to selective and constant attention following an evening when sleep was deprived at the beginning of the evening (Jarraya etal., 2013); therefore it is unclear whether the effects of sleep deprivation on cognitive performance are greatest when restricted at the beginning or the end of the night.

3.3. Sleep research in elite athletes

3.3.1. How is it measured?

The gold-standard of sleep measurement is polysomnography (PSG). Polysomnography is a multi-parametric test which may monitor several body functions during sleep, including eye movement (electrooculography; EOG), muscle activity or skeletal muscle activation (electromyography; EMG) and heart rate rhythm (electrocardiography; ECG) (Penzel and Canisius, 2006). However an integral part of analysis by PSG is made by assessing brain activity (electroencephalography; EEG). As NREM sleep progresses, EEG activity begins to slow in frequency. The deepest stages of NREM; SWS, reflect the occurrence of lowfrequency waves (0.5-4Hz and <1Hz; Walker and Stickgold, 2004), thus EEG allows the researcher to not only determine the duration, but also the stage of sleep. Additional measures may then add information of interest to enhance knowledge and/or address a particular research question. For example, in attempt to detect sleep-related breathing disorders, it is necessary to record several cardiorespiratory measures (Penzel and Canisus, 2006). However, despite being considered the gold standard method of sleep measurement, PSG requires a somewhat intrusive and expensive assessment of EEG and other physiological variables (Leeder et al., 2012b). Assessment by PSG typically requires attendance at a sleep laboratory with specialist staff, which is not only inconvenient but does not reflect athletes habitual sleep patterns.

The most commonly used method with elite athletes is subjective sleep questionnaires; however, these have been shown to have a poor relationship with objective measures of sleep (Leeder et al., 2009; Richmond et al., 2004). An alternate, objective measure of sleep is actigraphy which has become a major assessment tool in sleep research and sleep medicine over the last two decades (Sadeh, 2011). Actigraphy is based on small wrist-watch like devices that monitor movements for extended periods of time. The raw activity scores (e.g. in 1min epochs) are translated to sleep-wake scores based on computerised scoring algorithms (Sadeh, 2011). Wristwatch actigraphy is a non-intrusive, cost-effective tool used to estimate sleep quantity and quality which is ideally suited to the monitoring of elite

athletes. However conflicting views have been presented as to whether actigraphy is a valid sleep-wake indicator to be used in the assessment of sleep disorders (Pollak et al., 2001). Nevertheless, validation studies comparing wrist actigraphy monitors with PSG report high correlations for sleep duration (i.e. r=0.84-0.89) and moderate to high correlations for wake time within sleep (i.e. r=0.65-0.76) (Jean-Louis et al., 1999; Sargent et al., 2014; Weiss et al., 2010). Actigraphy has also been shown to have increased sensitivity to awakenings when compared to self-reported sleep measures; which underestimate both duration and total number of nocturnal awakenings in comparison to measures derived from actigraphy (Kushida et al., 2011; Lockley et al., 1999: Wilson et al., 1998). However, actigraphy should not be held to the same expectations as PSG. Actigraphy is one dimensional, whereas PSG may comprise of several types of data (Ancoli-Israel et al., 2003). Furthermore, unlike PSG, actigraphy cannot determine the stage of sleep an individual is in. It should also be considered that individual monitors and the custom algorithms they provide will vary between units thus one cannot assume that all actigraphy watches are valid for sleep assessment (Sadeh, 2011). Therefore any conclusions of actigraph validity should be made specific to the devise that is used.

3.3.2. Observational studies in elite athletes

When asked which recovery modality was perceived to be most important, previous research has shown that athletes (n=507) perceive sleep to be the most important compared to common post-exercise modalities including cool down, fluid replacement, supplementation, cold water immersion, contrast water therapy and massage (Venter, 2012). However, considering its proposed importance (Halson, 2008; Meerlo et al., 2008), a limited amount sleep research has been conducted in elite sport compared to other recovery strategies (e.g. cold water immersion, Leeder et al., 2012a). Nevertheless, research suggests that athlete sleep, or the lack of, may be related to travel (Richmond et al., 2004), training volume (Jurimae et al., 2002; Jurimae et al., 2004; Taylor et al., 1997), mood (Lastella et al., 2012) and performance (Leger et al., 2008).

Longitudinal observations of female swimmers by PSG showed that SWS formed a very high percentage of total sleep during peak (31%) training periods of a season, but was significantly reduced following a pre-competition taper (16%), supporting the theory that the need for restorative SWS is associated with increased physical demands (Taylor et al., 1997). Indeed a meta-analysis of sleep research has shown physical exercise increases total sleep time, delays REM sleep, increases stage 4 NREM sleep and reduces REM sleep time when sedentary and physically active individuals are compared (Youngstedt et al., 1997).

However, the time at which the exercise is performed may be crucial in the modulation of sleep, as research suggests that exercise performed in the evening may disrupt subsequent sleep (Driver and Taylor, 2000; Opp et al., 1998; Youngstedt et al., 1997). This may partly be explained by a delay in parasympathetic reactivation (as discussed in section 3.2.1.), which is dependent on the intensity and duration of exercise. Furthermore pro-inflammatory cytokines (in particular IL-6) stimulated by exercise may directly mediate sleep regulation, or indirectly by their action on HPA activation to increase body temperature, increase cortisol secretion, decrease the amount of nREM sleep and increase wakefulness (Vgontzas and Chrousos, 2002). Thus high-intensity, stressful exercise and/or exercise which induces muscle damage may impair subsequent sleep, particularly when performed in the evening. Furthermore, consideration should be given to the effect of exercise early morning training on sleep patterns as recent research suggests exercise performed too early may in fact impair preceding night's sleep (Sargent et al., 2014). Seven swimmers from the Australian Institute of sport were monitored in preparation for the 2008 Olympic games for 14 days, 12 of which were training days beginning at 06:00. Analysis revealed that on nights prior to training days, bedtimes and get-up times were significantly earlier, time spent in bed was significantly shorter and the amount of sleep obtained was significantly less, when compared to nights prior to rest days. Therefore in some sports where early-morning starts are common practice (e.g. swimming, rowing), sleep restriction may negate the effectiveness of training (Sargent et al., 2014).

The effect of exercise on sleep may also be related to the nature of exercise performed (Trinder et al., 1985). Previous research has also compared sleep patterns of aerobically trained endurance runners; power trained weightlifters and bodybuilders; athletes with mixed anaerobic, aerobic, and power training; and an unfit, non-athletic sedentary, control group (Trinder et al., 1985). Comparisons showed no differences between the control group and the combined athletic group. However, the aerobic group had more SWS and NREM sleep, slept longer, and had shorter sleep onset latencies than the power group. Therefore the findings suggest aerobic exercise is associated with improved sleep quantity and quality in comparison to power training (Trinder et al., 1985).

Chronic increases in pro-inflammatory cytokines have also been associated with the development of pathologies responsible for sleep disturbances. Thus athletes subjected to high training or competition loads and/or are not permitted sufficient recovery may be at increased risk of disrupted sleep. Sleep disorders; specifically short sleep time and poor sleep quality, are common complaints among athletes in a state of overreaching or suffering from overtraining (Smith, 2000). For example, using the Recovery-Stress Questionnaire for Athletes (RESTQ-Sport) Jurimae et al. (2004) found increased somatic components of stress

(fatigue, somatic complaints, fitness/injury) and decreased recovery factors including sleep quality (success, social relaxation, sleep quality, being in shape, self-efficacy) following a six day training camp for competitive rowers (n=21), in which training volume increased by approximately 100% (Jurimae et al., 2004). Furthermore, relationships were observed between training volume, and fatigue (r=0.49), somatic complaints (r=0.50) and sleep quality (r=0.58) at the end of the training period (Jurimae et al., 2004). Following a taper or de-load period, research demonstrates that sleep quality may be restored to pre-overload values (Hausswirth et al., 2014). However, if sufficient recovery or reduction in load does not follow a period of heavy training, the effects of sleep disruption may manifest to further impair physiological and psychological restoration, mood and sleep quality, continuing in a vicious cycle and potentially spiralling to a state of overtraining, characterised by a chronic performance plateau, increased risk of illness, inability to recover, depressed mood, general apathy, decreased self-esteem and irritability that cannot be influenced positively by short amounts of rest and recovery (Kellmann et al., 2010).

Disrupted sleep; and subsequently mood (Lastella et al., 2012) in athletes may also be related to increased stress of competition or training (Mezick et al., 2009). For example, Lastella et al. (2012) assessed the self-reported pre-competitive sleep behaviour of 103 marathon runners and how it related to subsequent pre-competitive mood and performance. Results revealed that on the night before competition athletes slept well under the recommended target of eight hours of sleep for healthy adults, with almost 70% of athletes experiencing poorer sleep than usual. The authors reported anxiety, noise, need to use bathroom and early event times amongst most commonly reported causes of disrupted sleep in athletes on the night prior to competition. Furthermore, negative moods of fatigue and tension were both significantly negatively correlated with pre-competitive relative sleep quality and total sleep time, tension was positively correlated with the number of awakenings and vigour was seen to be significantly positively associated with relative sleep quality. However it is often unclear whether disrupted sleep precedes alterations to mood, or whether it is a symptom of increased psychological stress (Leeder et al., 2012b). Fietze et al. (2009) also observed increases in sleep disruption prior to an event; in professional ballet dancers leading up to a premiere. Using wrist actigraphy, the authors found significant reductions in sleep duration, from 418 ± 43 to 391 ± 42 min, and sleep efficiency, from 81 ± 4 to $79 \pm 5\%$ which although may have been as a consequence of accumulative training volume is likely a reflection of increased anxiety associated with performing (Fietze et al., 2009). Disrupted sleep did not demonstrate any significant relationship with relative performance in the Lastella et al. (2012) study; however the authors propose that disrupted sleep for one night may not be sufficient to overwhelm most athletes' self-regulatory mental skills. Nevertheless, with the evidence presented in section 3.2., it may be that long-term or repeated life stressors may have accumulative effects on physiological and psychological performance and recovery by repeatedly disrupting sleep.

In addition to assessing sleep patterns leading up to an event, previous research has also assessed sleep patterns pre and post team sport competition (Richmond et al., 2004). Assessing 10 Australian rules footballers using actigraphy (Micro Mini-Motion Logger, Ambulatory Monitoring Inc., Ardsley, NY) and subjective sleep quality questionnaires, results interestingly showed an increase in sleep the night before the game compared to the average of 5 non-game related night's sleep (baseline). However, following the game sleep duration was significantly decreased below baseline (p<0.05). The authors also found a significant difference between sleep duration between home and away games post match (p<0.05), which was suggested to be as a consequence of traveling following competition (Richmond et al., 2004). The reason for improved sleep the night before a game was proposed to likely reflect a belief that a good night's sleep will maximise game performance the next day (Richmond et al., 2004). Indeed, research has demonstrated that an extended period of sleep extension (5-7 weeks; minimum 10h) improves physical and cognitive performance, as well as well-being (Mah et al., 2011). However research also shows that short term sleep extension (3 days) does not return physiological (Pejovic et al., 2013) or cognitive (Belenky et al., 2003) performance levels to pre-sleep deprived levels following a period of restriction. Nevertheless, with recent research showing a relationship between individual perception of recovery and subsequent performance (Cook and Beaven, 2013), increasing pre-match sleep duration may be an important component of individual's preparation. In addition to the large physical and psychological demands associated with competition, athletes may have disrupted sleep post-match due to several other factors associated with performance (e.g. caffeine, Youngstedt et al., 2000) and post-match activity (e.g. alcohol consumption, Feige et al., 2006).

Due to the large physical and psychological demands associated with competition, travel and varied time of competition it may be difficult to control some of the factors which influence the extent of intra-individual variation in sleep patterns. However Mezick et al. (2009) suggest that irregularity in sleep from night to night may be related to increased physiological and self-reported psychosocial stress. Therefore, in addition to sleep duration and quality, intra-individual variation should be monitored in athletes; particularly with individuals who compete and travel regularly. It may also be important to try and identify when sleep is most disrupted and/or the potential underlying reasons why sleep is disrupted and provide potential interventions or advice to try and enhance sleep quality and/or reduce

the extent of variability in sleep patterns. Coaches should also be aware of the effect of travel or training schedules may have on sleep variation.

Coaches and sport scientists should also attempt to identify individuals who are most susceptible to disrupted sleep. Research by Leeder et al. (2012b) showed in general, athletes (n=46) appeared to get a comparable quantity of sleep compared to the control group (n=20) when 4 nights of non-competition night sleep were assessed; however, there were significant differences between controls and athletes for all other variables (Table 3.4.), suggesting quality of athlete sleep was inferior. Furthermore, Leeder et al. (2012b) highlight that the elite athlete group also had considerably larger measures of spread (SD) for every sleep variable compared with the control group, suggesting there was considerable individual variation. Indeed, when assessing individuals on a case by case basis, the authors report that many individuals had comparable sleep to the control group; however, only certain individuals displayed signs of sleep disruption. Thus within a group of athletes, sleep quality may vary considerably which may be due to several factors, including individual tolerance to physical load or stress associated with training/competition or other life stressors (Leeder et al., 2012b).

Table 3.4. Definitions of each sleep variable measured using wristwatch actigraphy (Leeder et al., 2012b).

Sleep latency (min) The difference between sleep onset time* and bedtime as defined by the Time in bed (h:min) The difference between bedtime and get-up time as defined by the participant. participant.

Time asleep (h:min) The actual time spent asleep determined from sleep start to sleep end, minus any wake time*.

Time awake (h:min) The actual time spent awake* determined from sleep start to sleep end*.

Sleep efficiency (%) The sleep duration expressed as a percentage of time asleep* from bedtime* to sleep end*.

Actual sleep percentage (%) The sleep duration expressed as a percentage of time asleep from sleep start* to sleep end*.

Moving minutes (min) The actual time spent moving* during time in bed.

Percentage moving time (%) The amount of time spent moving* as a percentage of time in bed

Fragmentation index A measure of restlessness during sleep, using the percentage of epochs where activity is >0

*parameters determined by the Actiwatch software algorithm.

3.3.3. Methods to enhance sleep quality

Several methods have been proposed to enhance sleep quality in literature (Table 3.5.). Indeed research shows sleep hygiene recommendations can improve ensuing post-exercise recovery in athletes (Duffield et al., 2013). Sleep hygiene may refer to behaviours that promote improved sleep quantity and quality by avoiding behaviours which interfere with sleep patterns (e.g. Table 3.5., Halson, 2008). For example, the consumption of caffeine prior to bedtime may be detrimental to sleep quality and quantity (Bonnet and Arand, 1992; Youngstedt et al., 2000). Bonnet and Arand (1992) found that caffeine (>100mg, e.g. 250ml coffee) within 2h of bedtime increased sleep latency, decreased SWS, and decreased total sleep times (Bonnet and Arand, 1992). These findings may have greatest implications for players who use large amounts of caffeine for performance enhancement and are therefore more likely to experience disrupted sleep post-match.

Sleep hygiene may also relate to the inclusion of behaviours and strategies which may enhance sleep quality and quantity (Table 3.5.). For example, research suggests tart cherry juice concentrate; which contains a high concentration of melatonin, may increase total sleep time and sleep efficiency when supplemented daily (Howatson et al., 2012). Furthermore, several practical suggestions are recommended in literature (Table 3.5.), including the gradual reduction of night-time light exposure prior to bedtime (Duffield et al., 2013) as light has been shown to suppress the production of melatonin; which is the major hormone that controls sleep and wake cycles (Figueiro et al., 2011). In particular, it is established that short-wavelength or 'blue' light is most melatonin-suppressive; which is the light typically emitted by devices such as televisions, computer screens, and smartphones (Wood et al., 2013). Therefore the use of these devices may reduce sleep quality and quantity and should be avoided immediately prior to bedtime (Duffield et al., 2013; Table 3.5.)

3.3.4. Methods to enhance performance following sleep disruption

Extending the duration of night time sleep may be a relatively simple means of enhancing athlete performance following short or long term sleep disruption (Mah et al., 2011). Mah et al. (2011) found that when 11 basketball players increased their habitual night sleep patterns (2-4 weeks) by sleeping a minimum 10h each night (5-7 weeks), subjects demonstrated faster sprint times, shooting accuracy improved and reaction time decreased. Furthermore, following a period of sleep extension, profile of mood state scores and overall ratings of physical and mental well-being during practice and games were improved. These findings therefore suggest that sleep patterns need to be optimised for athletic potential to be realised,

and suggest that sleep extension may enhance physiological and psychological performance (Mah et al., 2011). However many athletes training patterns may not allow such increases in sleep-wake cycles. Furthermore, if sleep is disrupted the night before an event (e.g. increased anxiety, Lastella et al., 2012) strategies need to be implemented prior to that event which may diminish the detrimental effects of sleep disruption and therefore positively impact performance.

Research suggests that short-duration sleep (naps) during the day may be an effective means of enhancing performance following sleep disruption (Postolache et al., 2005; Waterhouse et al., 2007). For example, Waterhouse et al. (2007) assessed the effect of a lunchtime nap (13:00-13:30h) on performance following partial sleep deprivation (4h less than normal) and found 20m performance improved ($3.88 \pm 0.05 \text{ vs.} 3.97 \pm 0.05\text{ s}$), alertness improved and sleepiness decreased compared to when individuals did not nap. Napping may also have a positive influence on cognitive performance (Postolache et al., 2005), as naps can be beneficial when learning skills, strategies or tactics in sleep-deprived individuals (Postolache et al., 2005). Short sleeps during the day may therefore be beneficial for athletes who have to routinely wake early for training or competition (Sargent et al., 2014) and for athletes who are experiencing sleep deprivation (Halson, 2013). However, athletes and coaches should compensate for individual responses to napping with respect to duration, timing and sleep inertia (i.e. short-term impairment in wakefulness when woken) when planning naps as part of recovery and preparation for performance (Bird, 2013).

Table 3.5. Potential non-pharmacological means for promoting sleep quality and/or quantity (adapted from Duffield et al., 2013; Halson, 2008; Howatson et al., 2012).

- Ensure appropriate recovery (physical, nutritional and psychological) from training and competition.
- Consume tryptophan containing foods such as milk, meat, fish, poultry, eggs, peanut, cheese and leafy green vegetables.
- Consume a high glycaemic index meal 4h before bedtime.
- Cherry juice supplementation (e.g. 2x30ml daily; 30min upon wakening and 30min prior to evening meal).
- Consume a balanced, healthy diet.
- Minimise alcohol intake prior to bedtime.
- Minimise caffeine intake prior to bedtime.
- Be cautious of fluid intake following completion of training/competition and bedtime. For athletes who are repeatedly waking at night to use the bathroom, hydration testing and fluid balance assessment may be useful to prescribe type and quantity of fluid both during the day and during the recovery period.
- Skin warming (in cool environmental conditions)- this can be achieved through prior warm baths/ spa baths, hot footbaths, warm blankets and wearing of socks.
- Skin cooling (in warm environmental conditions)- this can be achieved through cool showers, the appropriate use of air-conditioning.

Sleep hygiene – the following sleep hygiene strategies are commonly recommended:

- If you cannot sleep within 15min, get out of bed and try another strategy.
- Eliminate the bedroom clock.
- Avoid coffee, alcohol and nicotine.
- Regularise the bedtime.
- Be conscious of food and food intake.
- Eliminate the use of TV, smartphones etc. and excessive light 30min prior to bedtime.

Explore the use of muscle relaxation and cognitive relaxation.

Following sleep disruption, a time-efficient means of alleviating decrements in skill performance may be supplementation with a single dose of creatine (50-100mg.kg⁻¹) and/or caffeine prior to performance (1-5mg.kg⁻¹ (Cook et al., 2011). It is proposed that acute sleep deprivation reduces brain creatine, therefore creatine supplementation may enhance performance when sleep is acutely deprived prior to training and/or competition, with 100mg.kg⁻¹ appearing to elicit a trend towards greater effect in skill performance than 50mg.kg⁻¹ (Cook et al., 2011). Furthermore, creatine may be a convenient alternative for individuals who experience disrupted sleep following caffeine supplementation.

3.3.5. Summary

Research has shown that sleep disruption (total or partially deprived sleep, fragmented sleep or large intra-individual variation) may have implications to performance and recovery of athletes due to alterations in physiological and psychological processes, whether chronic or acute. Furthermore, chronic sleep disruption has been linked to several health and performance based issues, including depression and overtraining.

Large inter-individual variation has been demonstrated in athletes, with evidence suggesting elite athletes may be susceptible to sleep disruption. Although it may be difficult to control many factors which effect athletes sleep (e.g. time of competition), practitioners should be aware of the effect certain variables have on sleep (e.g. training time, travel arrangements) which should be attempted to be controlled to reduce the extent of intra-individual variation. Monitoring of athletes both out and in competition may give an indication of certain individuals susceptible to sleep disruption and the periods of competition when it is most likely to occur.

Practitioners and athletes should be aware of several strategies which have been proposed in literature to enhance athlete sleep patterns and performance following sleep disruption. However, individual responses to strategies should be understood prior to implementation during important periods of training and competition (e.g. napping, caffeine).

4. Recovery and adaptation following exercise

Rugby union players are in a cyclic state of adaptation and recovery from the stressors associated with training and competition (Smith, 2003); however, as players seek to optimise performance, sufficient recovery is frequently overlooked to allow for increases in overload, intensity and volume (Cochrane, 2004). In addition to large training volumes, research suggests that players may take several days to fully recover following match-play (e.g. West et al., 2014). For example, research demonstrates large elevations in blood markers of muscle damage (e.g. creatine kinase) (Cunniffe et al., 2010; Smart et al., 2008; Takarada et al., 2003), and disruptions to neuromuscular (West et al., 2014), hormonal (Cunniffe et al., 2010; Elloumi et al., 2003; West et al., 2014), immune functions (Cunniffe et al., 2010) and mood (West et al., 2014) for several days following competition. An imbalance between the exercise stressors and recovery can result in a fatigued state, which may be defined "as an acute impairment of performance that includes both an increase in the perceived effort to exert a desired force or power and/or any reduction in the ability to exert maximal muscle force or power" (Rampinini et al., 2011).

Optimising the recovery-stress state is critical. Indeed, Kellmann (2010) propose that effective recovery from intense loads often faced by elite athletes can often determine sporting success or failure. Physiological recovery is marked by the return of muscle function to a pre-exercise state (Pointon and Duffield, 2011) and in accordance with Seyle's General Adaptation Syndrome (GAS; Seyle, 1946), an athlete will return to resting function improvements in their physiological or possibly show capabilities through 'supercompensation' when sufficient recovery is provided between exercise bouts. However, an athlete may suffer from 'non-functional overreaching' when insufficient recovery is provided or they may even experience a state of 'overtraining', a longer lasting condition where performance continues to be suppressed despite reductions in training stress (Cormack et al., 2008).

The physiological recovery process is facilitated by the integration of several physiological systems and is influenced by multiple factors. For example, functional recovery is strongly correlated to the extent of muscle damage induced by exercise and the subsequent inflammatory response (Nosaka et al., 2006). Exercise induced muscle damage (EIMD), which is specific to the exercise stimulus and athlete training status, impairs contractile function and signals an inflammatory response that impairs contractile function and places stress on several systems (e.g. neuromuscular, endocrine, autonomic nervous system) which augment but ultimately facilitate the fatigue response.

In attempt to optimise recovery following training and competition, it is common practise to employ one or more post-exercise recovery strategies. Although the mechanisms of these strategies are not clearly understood, research exists to show that they may enhance physiological recovery following exercise. However, research exists to suggest that the use of recovery strategies may impede subsequent performance and the stimulus for adaptation. For example, it has also been suggested that muscle damage and the subsequent inflammatory response is a vital precursor to the signalling mechanisms which initiate the repair and growth of cells (Carlson and Faulkner, 1983); therefore, imposing strategies which attempt to reduce the effects of EIMD may in fact reduce the potential for training adaptation to occur (Fischer et al., 2004; Gomez-Cabrera et al, 2008; Nemet et al., 2009; Strobel et al., 2010; Yamane et al., 2006). The use of recovery strategies post-exercise may therefore be 'situation dependent'.

The following section will examine the rationale for employing the most common postexercise recovery strategies. To understand how certain strategies may enhance recovery the characteristics of fatigue and recovery will be explored. Subsequently, the rationale and evidence for using commonly used recovery strategies will be presented before exploring the possibility that post-exercise recovery strategies may be detrimental to training adaptation.

4. 1. Physiological characteristics of fatigue and recovery

4.1.1. Exercise induced muscle damage (EIMD): process of events leading to further damage, functional impairment and adaptation

4.1.1.1. Initial damage

Most hypotheses have highlighted mechanical loading as the main factor contributing to EIMD. In particular, high-force, eccentric work may exceed the muscles ability to actively resist the load, forcing the muscle to lengthen and generate greater active tension (Stauber, 2004). As a result, greater tension per active unit is developed increasing the risk of EIMD to the vulnerable myotendinous junction (Cheung et al., 2003), including broadening, smearing and/or total myofibrilar disruption of z-lines. The initial trauma may also produce swelling and disruption of the sarcolemma, sarcoplasmic reticulum and T-tubule system, mitochondria and extracellular matrix. Subsequent damage to the sarcolemma, sarcoplasmic reticulum and T-tubule system, can also diminish calcium (Ca²⁺) uptake and release, resulting in an increase in intracellular free Ca²⁺ (Howatson and van Someren, 2008; Nicol and Komi, 2003).

The above mechanisms may help explain the elicitation of EIMD following eccentric/quasiisometric and stretch shortening cycle (SSC) type movement patterns (e.g. sprinting, jumping, changes of direction); however, structural damage may also occur when a muscle is subjected to sudden, heavy compressive forces, such as a direct blow to the muscle (Jarvinen et al., 2005). From an exercising perspective, the extent of mechanical damage will be determined by several factors including the strain rate, the amount of external work done, the force level, the duration of stretch and number of stretching activities, and the extent to which an individual is accustomed to exercise (Hortia, 2000).

It is also possible that EIMD is due, in some part, to oxidative stress. During periods of oxidative stress pro-oxidants overwhelm the antioxidant defences in cells and damage cellular constituents (Powers and Jackson, 2008). Reactive Oxidative Species (ROS), predominantly sourced from the mitochondria relative to elevations in oxygen consumption, can also contribute to the disruption of cell structures (Powers and Jackson, 2008). Similarly, metabolic pathways relating to the induction of ischemia or hypoxia during physical activity offer another source of EIMD (Armstrong, 1984; Ebbeling and Clarkson, 1989) resulting in changes in metabolic waste accumulation, ion concentration, and adenosine triphosphate deficiency (Byrnes and Clarkson, 1986). Ultimately, muscle cell damage shifts the sarcomere length-tension relationship, thereby reducing force output (Horita, 2000).

4.1.1.2. Inflammation and secondary muscle damage

Increased permeability of cell structures as a consequence of initial damage to the muscle allows for an influx of protein-rich fluid (exudate) into the muscle, leading to oedema and the attraction of inflammatory cells. Within the injured muscle macrophages are activated, which produce additional chemotactic signals for circulating inflammatory cells (Jarvinen et al., 2005). Macrophages secrete and attract cytokines which further degrade the muscle tissue by removing necrotic debris by phagocytosis, and alter endothelial permeability leading to phagocyte infiltration and further oedema. Arterioles within the injured area may also dilate, increasing blood flow to the site of injury (Jarvinen et al., 2005; Smith et al., 2008).

The increased permeability to cells facilitates the influx of intracellular Ca^{2+} into the cytosol and this activates calcium dependent proteolytic enzymes (proteases and phospholipids), which further degrades and weakens the cell structures (Cheung et al., 2003) resulting in further influx of Ca^{2+} and thus greater proteolytic enzyme activation and cell destruction.

The ensuing disruption of z-lines and damage to the sarcolemma enhances the diffusion of soluble enzymes, such as creatine kinase (CK), into the interstitial fluid (Cheung et al., 2003). An increase in plasma CK, as an indirect marker of muscle damage, also attracts more immune cells to the injured site. In addition to CK, satellite cells within the cell structure and necrotised parts of cells release various chemoattractants (e.g. histamine) which amplify the influx of inflammatory phagocytic cells and proteolytic enzymes, via the process of extravasation (Jarvinen et al., 2005; Smith et al., 2008). This process includes the influx of neutrophils from the bone marrow to the injured site, thereby destroying dead tissue and releasing proteolytic enzymes and ROS that can further degrade tissue and increase membrane permeability (Smith et al., 2008) further exacerbating the inflammation response.

4.1.1.3. Effect of EIMD and inflammation on muscle function and recovery

The extent of EIMD and its effect on subsequent performance is more pronounced in individuals previously unaccustomed to a given exercise stimulus, who themselves will be more susceptible to experiencing delayed onset muscle soreness (DOMS) (Cheung et al., 2003). However, it has been assumed that muscle damage produced following exposure to a bout of eccentric exercise results in an adaptation such that there is more resistance to subsequent damage and soreness when the exercise bout is repeated, known as the repeated bout effect (RBE; Clarkson and Hubal, 2002). Furthermore, research has shown that adaptation can occur in the absence of significant muscle damage through low volume exposure to eccentric exercise (Paddon-Jones and Abernethy, 2001) suggesting the RBE may be due to neural adaptations to a task (Clarkson and Hubal, 2002; Paddon-Jones and Abernethy, 2001). However, despite the potential protective effect of the RBE evidence still exists in the literature to suggest the presence of EIMD and/or fatigue several days following competition in elite athletes (Avela et al., 1999; Cormack et al., 2008; Dawson et al., 2005; Gill et al., 2006).

The most pronounced effect of EIMD on muscle performance is the ensuing decrease in strength and power, resulting from a shift in the muscle length/sarcomere length-tension relationship (Horita, 2000). Oedema-induced increases in passive muscle stiffness may also increase following the induction of EIMD and this is characterised by reduced joint angles at rest and a decrease in the joint range of motion (Nicol and Komi, 2003; Nosaka, 2006). The disruption of Ca^{2+} homeostasis due to EIMD can lead to an inhibition of cellular respiration at the mitochondrial level, thereby hindering adenosine triphosphate (ATP) regeneration. These changes have important consequences for contractile function, due to the role of Ca^{2+}

in binding troponin C, instigating movement of tropomyosin and allowing the cycling of cross bridges (Allen et al., 2008). It has been proposed that an increase in intracellular Ca^{2+} is a primary cause of low frequency fatigue (LFF), which may influence SSC type activities (Allen et al., 2008).

Typically, EIMD is accompanied by varying levels of pain and tenderness at the muscle site, or DOMS. Subjectively, DOMS is most often reported 24-48h post exercise (Cheung et al., 2003; Clarkson and Hubal, 2002), which may be explained by the increased inflammation and secondary muscle damage. As inflammation increases, an osmotic pressure is exerted and pain is transmitted following stimulation of polymodal group III and IV afferents, a phenomenon termed 'hyperalgesia' (Nicol and Komi, 2003). Presynaptic inhibition following stimulation of these afferents may also contribute to an attenuated stretch reflex during SSC type activities (Avela et al., 1999; Nicol and Komi, 2003). It has been proposed that these afferents have negative feedback on the α -motorneuron via the inhibitory interneuron (presynaptic reflex inhibition; Bigland-Ritchie et al., 1986).

It has been widely demonstrated that the functional expression of power and strength are compromised for several days following exercise (Avela et al., 1999; Bailey et al., 2007; Cormack et al., 2008; Dawson et al., 2005; Fatouros et al., 2010; Highton et al., 2009; Horita, 2000; Ingram et al., 2009; Kraemer et al., 2010; West et al., 2014) and strong correlations between functional recovery and blood markers of muscle damage (e.g. creatine kinase) indicate that these patterns of recovery are partly dependent on the extent of EIMD (Avela et al., 1999; Horita, 2000).

A loss in muscle function may result in compensatory neuromuscular alterations (e.g. altered kinematics, increased ground contact times, reduced movement velocity) (Highton et al., 2009; Horita, 2000). Potentially, these changes may require an individual to work at a much higher intensity to maintain performance (Chen et al., 2009) and/or cause unaccustomed strain on the compensating muscles, joints, ligaments and tendons increasing the risk of injury (Cheung et al., 2003). EIMD has been also been shown to impair oxygen kinetics (Davies et al., 2008) and glycogen repletion (O'Reilly et al., 1987), both of which can impair performance, especially in endurance and intermittent sports that rely on these metabolic outputs. On an individual level, the perception of these functional impairments has further implications for subsequent skill acquisition and injury susceptibility (Saxton et al., 1995).

Although the induction of EIMD and the ensuing inflammatory response may be responsible for an acute reduction in muscle function, this process appears to be necessary for the subsequent repair and adaptive remodelling of muscle tissue to also improve muscle function (Figure 4.1).

Macrophages play an important role in repair despite initiating events that elicit further muscle damage (Butterfield et al., 2006; Jarvinen et al., 2005; Smith et al., 2008). It has been proposed that unless there is a proliferation of macrophages to the damaged muscle fibre, it remains in a stage of intrinsic degeneration and the activation of satellite cells and thus regeneration proceeds no further (Carlson and Faulkner, 1983). The process of macrophage phagocytosis removes necrotic tissue while preserving the cylinders of the basal laminas surrounding the necrotised parts of the injured myofibres and thus allowing a 'scaffold' structure to remain inside which the viable satellite cells begin the formation of new myofibres (Jarvinen et al., 2005). Macrophages also secrete pro-inflammatory cytokines, fibrinectin and proteoglycans which help to promote cell adhesion and stimulate fibroblast proliferation and collagen synthesis.

4.1.2. Autonomic nervous system - parasympathetic reactivation

Physical exercise causes an increase in sympathetic activity, concomitant with parasympathetic withdrawal resulting in higher heart rates (Al Haddad et al., 2010). The time needed to restore pre-exercise autonomic nervous system (ANS) level has been shown to depend on both exercise intensity and duration. High-intensity exercise reduces the autonomic activity for longer time than a submaximal exercise (Buchheit et al., 2007; Seiler et al., 2007) and research suggests that EIMD and the subsequent inflammatory response may temporarily attenuate parasympathetic reactivation (Jae et al., 2010). Furthermore, as discussed in section 3.2.1., research shows that greater parasympathetic activity is usually associated with a better recovery state (Chen et al., 2011; Pichot et al. 2000) and readiness to perform (Buchhiet et al., 2009; Garet et al., 2004; Kiviniemi et al., 2007) suggesting ANS regulation is important in facilitating recovery, and parasympathetic reactivation is a useful indicator of athlete recovery status.



Figure 4.1. Summary of the adaptation process initiated by muscle damage.

4.1.3. Disruption to hormonal matrix

Hormones are chemical messengers, designed to be released from specific cells where they are carried to their target tissue for binding to receptors (Winchester, 2008). Traditionally hormones have been viewed in their role in facilitating protein synthesis and degradation following exercise (Crewther and Cook, 2012a). Two hormones in particular have been researched; testosterone; a steroid hormone, is anabolic in nature, important in facilitating muscle hypertrophy and increasing muscle glycogen synthesis (Cormack, 2008), and cortisol; catabolic in nature, causing increases in protein degradation in muscle and connective tissue, and reductions in muscle protein synthesis.

Research has shown evidence of hormonal disruption several days following competition (West et al., 2014) and increases in training volume (Argus et al., 2009) in rugby union, with cortisol suggested to be most sensitive to change (Passerlegue and Lac, 1999). However, literature indicates that testosterone has more complex training roles outside of direct hypertrophy, in regulating adaptive physiology and physical performance itself (Crewther and Cook, 2012a). Testosterone has been linked to calcium processing in muscle and contractile function (Curl et al., 2009; Estrada et al., 2003) and motor cortex output (Bonifazi et al., 2004). In individuals with relatively high strength levels, free testosterone is a strong individual predictor of the expression of strength and power qualities (Crewther et al., 2011; Crewther et al., 2012). Furthermore, Crewther and Cook (2012a) explain that testosterone is very important as a stress biomarker and the testosterone responses to a challenge can provide information on dominance. Winning in sports competition is often accompanied by elevated testosterone concentrations relative to losing (Elias, 1981; Fry et al., 2011; Mazur and Lamb, 1980).

4.1.4. Suppression of immune system

In addition to the risk that high loads of training and competition may have on performance, athletes, coaches and medical personnel should be alert to the increased risk of immune function impairment. Immune function impairment is characterised by decreases in circulating numbers and functional capacity of cells associated with inflammation, likely related to increased levels of stress hormones and anti-inflammatory cytokines and free radicals during exercise.

A single bout of exercise may change neutrophil function, with the change linked to the intensity and duration of exercise (Takahashi et al., 2007). As described in section 4.1.1.,
following muscle damage neutrophils are attracted to the damaged site which engulf microorganisms and produce ROS. Reactive oxidative species production capability is therefore increased by most types of exercise loading (Pedersen and Bruunsgaard, 1995) but may be impaired when exercise is continuous, prolonged (greater than 90min), of moderate to high intensity $(55-75\%VO_{2max})$ and performed with minimal nutritional support or where multiple maximal bouts are performed on one or a series of days, in which case there may not be sufficient time for the immune system to recovery fully (Gleeson, 2007). When athletes are engaged in heavy training/competing over a season, this may lead to a chronically depressed immune function (Gleeson, 2007). Individuals engaged in heavy training/competing programmes, particularly those involved in endurance events appear to be more susceptible than normal to upper respiratory tract infection (URTI; Gleeson, 2007).

4.2. Strategies to facilitate the physiological recovery process immediately following training/competition.

4.2.1. Cold water immersion (CWI)

The main rationale of immersing body parts in cold water, or CWI, is based on the effects of hydrostatic pressure (Wilcock et al., 2006). When external pressure on the body is increased, gas, fluid and substances are displaced to lower pressure areas. Hydrostatic pressure increases the pressure gradient between the interstitial compartment and the intravascular space to aid in the removal of plasma/exudate, thus reducing the cellular infiltration by leukocytes and monocytes (Wilcock et al., 2006). Reducing the inflammation response may improve nutrient delivery to cells, enhance metabolic waste removal, improve contractile function and reduce secondary muscle damage. Water immersion can also facilitate greater relaxation of gravitational muscles and energy conservation through the buoyancy effect (Wilcock et al., 2006).

Cold water immersion can also reduce skin temperature and stimulate cutaneous receptors to excite sympathetic adrenergic fibres (Cheung et al., 2003). This causes the constriction of local arterioles and venules, which reduces the permeability of cellular lymphatic and capillary vessels, thus restricting fluid diffusion into the interstitial space. This reduction in swelling and a decreased rate of metabolism reduces the inflammatory response, vascular permeability and the formation of oedema (Cheung et al., 2003). The cooling of tissue may also help to reduce the perception of pain by decreasing the production of acetylcholine and superficial inhibitory cells that regulate the impulse of pain perception to the central nervous

system (CNS; Wilcock et al., 2006). Similarly, reductions in inflammation may reduce the sensation of pain by the stimulation of group III and IV afferent fibres.

The orientation of the body, or body parts, in water is an important consideration. Wilcock et al. (2006) explained that for a person immersed standing to the superior aspect of the iliac crest, the foot immersed at a depth of 1m would have 981Pa extra pressure acting on it, whereas at hip level (0.1m) only an extra 98.1Pa. Therefore, the lower limbs will be subjected to greater pressure when a person is standing immersed in cold water, as opposed to someone sitting or kneeling in water to the same landmark. Water temperature is another consideration. Sramek et al. (2000) reported similar changes in whole blood flow following 10 minutes immersion in either 8°C or 22°C water. This would seem to be counterintuitive to the efficacious use of CWI. However, more blood was distributed to the skin at the 8°C temperature, suggesting that CWI may be related to changes in blood distribution (Gregson et al., 2011).

Water immersion itself can also trigger parasympathetic heart control (Mourot et al., 2008; Perini et al., 1998). The rise in hydrostatic pressure on the body from the surrounding water leads to an increase in vagal-related heart rate variability indexes (Lemaitre et al., 2008); perhaps related to the rise in central blood volume, cardiac output, stroke volume and central venous pressure (Lemaitre et al., 2008). However, the use of cold water temperatures may be counterproductive to the beneficial parasympathetic effect of hydrostatic pressure due to an increased sympathetic activation as described above (Buchheit et al., 2009).

Overall the findings of literature examining CWI as a recovery tool are inconclusive (Table 4.1.); with some studies (Paddon-Jones and Quigley, 1997; Sellwood et al., 2007) demonstrating no additional benefit when compared to passive recovery on the outcomes of pain, swelling and performance 24, 48 and 72h following unaccustomed eccentric loading. However, Howatson and Van Someren (2008) stated that the temperatures used in these studies ($5 \pm 1^{\circ}$ C) were too cold, possibly eliciting the 'Hunting reaction'. This phenomenon occurs when tissue temperature falls below 18°C and there is periodic vasodilation and warming of the tissue, which may eliminate any potential benefits of CWI. A recent meta-analysis of 14 studies by Leeder et al. (2012a) also found that CWI had little effect on recovery of strength (isometric/isokinetic knee extension or elbow flexion), but this approach was effective in improving the recovery rate of muscle power post exercise during more 'functional' SSC activities. Furthermore, Halson (2011) found that CWI had a greater effect on weight bearing activity compared to a non-weight bearing activity, which is more representative of the actual performance movement undertaken by rugby players. Indeed, previous research has found that SSC type activities are more sensitive to contractile failure

following EIMD than concentric only activities (Horita, 2000). These data highlight some of the difficulties in determining the efficacy of recovery strategies due to variation in study methodologies (see Table 4.1).

Despite not showing evidence of enhanced function, data exists to show that 5min CWI may accelerate parasympathetic recovery 5min (Buchheit et al., 2009) and 12h post intervention (Al Haddad et al., 2012).

Research shows that repeated exercise performance in the heat may be improved when a short period of CWI is applied between exercise bouts (Peiffer et al., 2010). However, when multiple competitive bouts are performed on the same day, evidence exists to suggest that the use of cold water immersion following one bout may impair performance in the next (Crowe et al., 2007; Parouty et al., 2010). Cooling of tissue stimulates inhibitory cells that regulate the impulse of pain perception to the central nervous system; however it may also decrease the rate of transmission along neurons by decreasing the production of acetylcholine. Reduction in neural transmission would therefore decrease muscular contractile speed and force generating ability which may inhibit subsequent performance (Wilcock et al., 2006). A decrease in heart rate following CWI may also decrease cardiac output and arterial blood pressure thus decreasing blood flow to the prime mover muscles which may also impair performance. Furthermore, enhanced parasympathetic activity following CWI (Buchheit et al., 2009) may inhibit cardio-acceleration from exercise onset compromising the attainment of peak heart rate, possibly reducing cardiac output and oxygen delivery which may increase blood lactate accumulation and oxygen deficit (Parouty et al., 2010).

Table 4.1. Examples of studies which have assessed the efficacy of post-exercise recovery strategies compared to a control condition where no

recovery intervention was implemented.

Reference	Subjects	Mode of exercise	Post-exercise recovery protocol	Main between group significant findings
Cold water immersic	on (CWI)		•	
Bailey et al. (2007)	20 active males; 10 CWI, 10 control	Loughborough intermittent shuttle test (LIST)	10min (10 ± 0.5°C)	Reduced markers of muscle damage (myoglobin) after exercise following CWI.
				Reduced decrements in knee flexion MVC at 24 a 48h post LIST following CWI.
Brophy-Williams et al. (2011)	8 male athletes (cross-over study)	High-intensity interval exercise session (HIIS; 8x3min at 90% vVO _{2max})	15min (15 °C) immediately or 3h post-exercise	Improved YYIRT1 performance 24h post H following CWI immediately or 3h post exercise.
				Enhanced TQRP 24h post-HISS following C immediately post exercise.
Eston and Peters (1999)	15 females; 8 CWI, 7 control (activity level not stated)	8x5 maximum elbow flexions at 0.58rad.s ⁻¹ with 60s rest between sets on isokinetic dynamometer	15min (15°C) repeated every 12h for a total of seven sessions	Significant attenuation of increase in CK and gree relaxed elbow angle following CWI on days 2 and
Goodall and Howatson (2008)	18 active males; 9 CWI, 9 control	5x20 drop jumps (0.6m); 10s between jumps, 2min between sets	12min ($15 \pm 1^{\circ}$ C) repeated every 24h thereafter for 3 days	No significant differences between groups.
Howatson et al. (2009)	16 active males; 8 CWI, 8 control	5x20 drop jumps (0.6m); 2min between sets	12min (15 °C) repeated every 24h thereafter for 3 davs	No significant differences between groups.
Ingram et al., (2008)	11 male team sport athletes (cross-over study)	80min simulated team sport exercise followed by 20min shuttle run to exhaustion	2x5min (10°C); 2min 30s seated between each set	Reduced rating of MS 24 and 48h following CWI. Leg flexion. extension strength. speed recovered
Jakeman et al. (2009)	18 physically active females	10x10 CMJ	10min (10 ± 1°C)	48h post following CWI only. No significant differences between groups up to 9 post-exercise.
Lane and Wenger (2004)	10 active males (cross- over)	Intermittent cycling session; 18min varying work intervals at a resistance of 80g.kg ⁻¹	15min (15°C)	CWI prevented reduction in TW performed wl protocol repeated 24h following.

Paddon-Jones and 8 re Quigley (1997) Sellwood et al. 40 u (2007) cont	sistance trained males			
Sellwood et al. 40 u (2007) cont		of eccentric contractions of elbow flexors with each arm	One arm subjected to 5x20min immersions (5 ± 1°C), separated by 60min	No significant differences between groups.
	ıntrained; 20 CWI, 20 trol	5x10 eccentric contractions of knee extensors of non-dominant leg (120% 1RM)	3x1min (5 ± 1°C), separated by 1min	No significant differences between groups.
Contrast water therapy (CV	WT)			
Gill et al. (2006) 23 (distr rugh	(unknown group ribution) professional by players	Professional rugby union competition	3 x 1min CWI (8-10°C), 2min HWI (40-42°C)	Reduced interstitial CK elevations 36 and 84h p following CWT.
Ingram et al. 11 r (2008) (cro	male team sport athletes ss-over study)	80min simulated team sport exercise followed by 20min shuttle run to exhaustion	3x2min CWI (10°C), 2min HWI (40°C); 30s transition between sets	Reduced rating of MS at 24h post following CWI.
Vaile et al. (2007) 13 r mal stud	recreational athletes; 4 le, 9 female (cross-over ly)	5x10 eccentric leg press (140% IRM)	5x1min CWI (8-10°C), 2min HWI (40-42°C)	18kg SJ PP returned to baseline measures 24h p following CWI compared to 72h in the control grou
Versey et al. 11 t (2011) trial CW	rrained male cyclists (4 l cross-over study: 3 7T trials, 1 control)	75min cycling protocol; 6x(5x15s) sprints interspersed by 3x5min	1min HWI (38.4 ± 0.6°C), 1min CWI (14.6 ± 0.3°C) x3 (CWT6), x6 (CWT12) x9 (CWT18)	Reduced core temperature following CWT12 a CWT18 2h post-CWT. When protocol repeated 24h following, CV improved time trial and sprint performance a CW12 improved sprint TW and PP.
				All CWT conditions improved self-perceiv measures of thermal sensation, whole body fatig and muscle soreness within the 2h post intervention
Active recovery				
Dawson et al. 17 s (2005) Aus ove	semi-professional stralian rules (cross- rr)	Semi-professional Australian rules competition	15min multidirectional easy pool walking (28°C)	VJ, 6s cycle sprint PP and TW returned to basel 15h post following active recovery only.
Gill et al. (2006) 23 (dist rugl	(unknown group tribution) professional by players	Professional rugby union competition	7min cycling; 80-100rpm, ~150W	Reduced interstitial CK elevations 36 and 84h p following CWT.

Lane and Wenger (2004)	10 active males (cross- over)	Intermittent cycling session; 18min varying work intervals at a resistance of 80g.kg ⁻¹	15min cycling (30% VO _{2max})	Active Recovery prevented reduction in T performed when protocol repeated 24h following.
Compression garme	nts (CG)			
Duffield et al. (2009)	11 trained team sport athletes (cross-over)	10min exercise protocol; 20m sprint & 10 repeated double leg bounds repeated every min	24h LB CG	Reduced markers of muscle damage (AST) and M at 24h following CG.
Argus (unknown)	19 male recreational athletes; 10 CG, 9 control	12x20min sprints & 10x10 maximum concentric and eccentric contractions of knee flexors performed on days 1, 2, 3	12h LB CG following each exercise sessions	Pain values lower following CG at day 7. Average sprint time lower following CG on day 2, and 7.
				Fastest sprint time lower following CG at day 7.
				Mean concentric and eccentric work higher followin CG on days 2 and 7 respectively.
Jakeman et al., (2010)	17 active female; 8 CG, 9 control	10x10 drop jumps (0.6m)	12h LB CG	Reduced decrements in VJ performance followin CG up to 72h.
				Reduced MS following CG up to 72h.
				Reduced SJ and isokinetic knee extensor streng decrements throughout study period (96h).
Kraemer et al., (2010)	20 highly resistance trained; 11 men and 9 women (cross-over)	8 exercise WB heavy resistance protocol using barbells (3x8-10; 2min-2min 30s rest)	24h WB CG	Significant between group differences in vitality, R MS, ultrasound measure swelling, bench press thro and CK at 24h.

4.2.2. Contrast water therapy (CWT)

In contrast to CWI, hot water immersion (HWI) is thought to increase tissue temperature and local blood flow, and enhance muscle elasticity, leading to local vasodilation, an increase in metabolite production and a reduction in muscle spasms (Cochrane, 2004). In response to HWI, heart rate is also elevated to increase cardiac output and lower peripheral resistance, and this increases subcutaneous and cutaneous blood flow to improve the permeability of cellular, lymphatic and capillary vessels (Wilcock et al., 2006) The net effect is improvements in metabolism, nutrient delivery and waste removal from the cells (Wilcock et al., 2006). However, increasing the permeability of these vessels (through HWI) may increase the inflammatory response by facilitating the influx of exudate and inflammatory cells into the interstitial space (Wilcock et al., 2006).

Contrast water therapy (CWT) is also proposed to reduce the extent of oedema and enhance recovery. This method combines the properties of CWI and HWI to alternate the vasoconstrictor and vasodilator response of blood vessels to temperature changes (Gill et al., 2006). Vaile et al. (2007) further proposed that alternating between hot and cold water might produce rapid changes in muscle perfusion, which is commonly referred to as the 'pumping effect'. Vaile et al. (2007) demonstrated that CWT (8-10°C, 60s; 40-42°C, 120s; x5) enhanced recovery compared to passive recovery (15min seated, room °C) following an eccentric resistance exercise bout (5x10 eccentric leg press; Table 4.1.). Data collected immediately, 24, 48 and 72h after recovery showed that jump squat peak power returned to baseline levels 24h following exercise using CWT, compared to 72h using passive recovery. Furthermore, restoration of serum CK levels were observed at 48h following CWT, which was significantly elevated levels at 72h with the passive intervention. Research also exists to suggest that CWT may enhance parasympathetic reactivation following 60min high intensity cycling (Stanley et al., 2012).

4.2.3. Compression garments

Compression garments are becoming increasingly widespread within sport and exercise as a recovery aid (Jakeman et al., 2010) Typically, these garments are elastic, body-moulded suits with an engineered compression gradient that can be worn as an upper, lower or full body piece (Duffield et al., 2007). Compression garments are thought to enhance muscle recovery by exerting pressure on the covered limbs to improve blood flow and reduce

inflammation (Jakeman et al., 2010), possibly via similar mechanisms to hydrostatic pressure from water immersion.

In clinical settings, compression stockings have been worn to reduce oedema in individuals with chronic venous disorder, chronic venous insufficiency, and have been worn in attempt to prevent deep vein thrombosis in at risk individuals (e.g. long flights). A meta-analysis of randomised controlled trials was published that compared stockings exerting an ankle pressure of 10-20mmHg with placebo or no treatment, and with stockings exerting a pressure of more than 20mmHg (Amsler and Blattler, 2008). Overall, compression with 10-20mmHg had a clear effect on oedema compared with <10mmHg pressure, placebo stockings or no treatment (p<0.0001). Furthermore, the reviewed literature showed that there was no difference between compression stockings exerting 10-20mmHg and >20mmHg of pressure. Research investigating the effects of compression garments on oedema during a working day has also found little difference between 11.2 \pm 1.2, 18.1 \pm 2.7 and 21.8 \pm 1.8mmHg compression stockings (Partsch et al., 2004). Nevertheless, all garments tested in this study were found to be more effective than a light support stocking (5.9 \pm 2.4mmHg) (Partsch et al., 2004).

Kraemer et al. (2010) demonstrated that the custom-fitting of garments is one consideration in the mechanical blocking of oedema during recovery from EIMD. Although garments are commercially available in different sizes, it has been questioned whether these products can exert enough pressure to be effective for each individual, due to widespread differences in leg dimensions and tissue structure within a population (Davies et al., 2009). Nevertheless, some studies in this area have reported beneficial effects in terms of reducing EIMD and perceived muscle soreness, and restoring exercise performance (Table 4.1.). The use of compression garments (and other recovery strategies) may further create a positive placebo effect on individual's perception of muscle soreness and functional recovery, due to the positive contagion induced by peers or elite athletes advocating its beneficial properties (Lee et al., 2011).

4.2.4. Whole body cryotherapy (WBC)

Whole body cryotherapy (WBC) has been predominantly applied and researched within clinical settings for inflammatory conditions such as swelling and acute localised pain. However, WBC may also be a means of accelerating recovery post-exercise. Research has shown health benefits following exposure to very cold air (e.g. -110°C; Pournot et al., 2011).

A single exposure has been shown to alter hormonal profiles in men (Zagrobelny, 1993) and increase antioxidant defence system activity (Wozniak et al., 2007). Furthermore, data exists to suggest repeated exposures may enhance motor performance (Hagner et al., 2009, Klimek et al., 2011; Luczak and Michalik, 2006), anaerobic capacity (Chwalbinska-Moneta, 2003), increase levels of white blood cells (Lubkowska et al., 2010) and anti-inflammatory cytokines (e.g. IL-10) while decreasing pro-inflammatory cytokines (IL-2, IL-8; Banfi et al., 2009). However, limited research has been performed following exercise using WBC, largely due to the expense and limited availability of cryochamber facilities; which consequently explains why WBC is utilised by very few athletes at present. Mechanisms for enhanced recovery are likely to be related to vasoconstriction and a reduction in nervous activity as described in section 4.2.1.

Following a single simulated trial race, WBC (3min at -110°C) was shown to restrict the inflammatory response compared to a passive recovery group (Pournot et al., 2011). Levels of C-Reactive Protein (CRP) significantly increased in both groups 24h post-exercise, however levels in the WBC groups returned to pre-exercise 48h post while CRP levels in the passive group remained elevated at 96h. Using the same exercise and recovery protocols, Hausswirth et al. (2011) found WBC enhanced recovery of maximal isometric muscle strength and perceived pain up to 48h post-race. Research also suggests that a week of daily WBC sessions following training reduced markers of muscular damage (CK and lactate dehydrogenase) and pro-inflammatory cytokines compared to a passive recovery group within an International rugby union team (Banfi et al., 2009).

4.2.5. Active recovery

Performing low intensity exercise following exercise may enhance recovery by inducing a 'pumping effect'. Repetitive mechanical 'squeezing' by the muscles during contractionrelaxation may increase blood flow and improve range of motion which may increase the translocation and removal of markers of inflammation and metabolites, such as lactate from the muscle (Gill et al., 2006).

Active recovery is commonly used when several hours separate exercise bouts. Exercise which breaks down glucose without oxygen to produce energy for ATP resynthesis produces the biproducts lactate and hydrogen ions (H^+), with increases in the later proposed to cause muscular fatigue, demonstrated by a strong correlation between the decline in intramuscular pH and a reduction in force or power output (Cady et al., 1989; DeGroot et al., 1993).

Therefore the ability to remove lactate and H^+ from the muscle following exercise is important where time between bouts is limited. Approximately 65% of lactate is converted to pyruvate by lactate dehydrogenase which then undergoes aerobic degradation via the krebs cycle and electron transport system (Parkhouse and McKenzie, 1984), therefore an improved oxygen supply may enhance clearance.

Dodd et al. (1984) found that a recovery period of moderate continuous intensity facilitated lactate removal faster than passive recovery. Additionally, a combination of high intensity ($65\% \text{ VO}_{2\text{max}}$) and low intensity ($35\% \text{ VO}_{2\text{max}}$) was no more beneficial than a recovery of low intensity ($35\% \text{ VO}_{2\text{max}}$) for 40min. Active recovery may be performed in various ways however performing an active recovery session immersed in water may provide the added effects of buoyancy, hydrostatic pressure and parasympathetic reactivation which may enhance recovery. Furthermore, activities such as deep water running and water aerobics are convenient methods of recovery due to the non-weight bearing nature of activity thus avoiding exacerbation of soft-tissue injury while providing active recovery (Reilly and Ekblom, 2005).

4.2.6. Other strategies

4.2.6.1. Electrostimulation

Despite little research being performed, electrostimulation may be an effective treatment in enhancing athlete recovery following exercise. Electrodes are positioned on the posterior aspect of the knee to stimulate the common peroneal nerve and simultaneously activate the tibialis, peroneous longus, and lateral gastrocnemius muscles to enhance blood flow (Tucker et al., 2010).

When worn with compression garments, Beaven et al. (2012) showed that rugby players perceived improved recovery following training compared to compression garments alone. Furthermore, the combination of methods was shown to be more effective in attenuating increases in CK following competition compared to compression garments (Beaven et al., 2012).

Also when used in isolation, electrostimulation has recently been shown to enhance recovery of function 24h following a simulated team circuit compared to a passive recovery condition, with effects similar to a CWT treatment (Finberg et al., 2012).

4.2.6.2. Occlusion

Brief repeated periods of occlusion followed by reperfusion have previously been shown to mitigate the injurious effects of prolonged ischemia in cardiac muscles, as well as attenuate other cellular damage (Eisen et al., 2004; Iliodromitis et al., 2007).

Recent research suggests that occlusion may enhance recovery following a high intensity exercise protocol. Following 2x3min lower limb occlusion of each leg at 220mm.Hg⁻¹, various markers of power displayed improved recovery 24h following exercise when compared to a condition when 15mm.Hg⁻¹ compression was applied (Beaven et al., 2012). Although the mechanisms are unclear, the authors propose that the cycles of ischemia and reperfusion may enhance blood flow hence increasing muscle oxygenation, vasodilation, and oxygen delivery (Beaven et al., 2012).

4.2.6.3. Nutrition and hydration

Dietary antioxidants have been proposed to enhance recovery mainly by reducing ROS production during the phagocytic/secondary phase and through other means including sarcolemma stabilisation (vitamin E) and reducing pro-inflammatory mediators (omega-3 fatty acids) however the literature is equivocal with respect to their effects on EIMD (Bloomer, 2007).

The supplementation of polyphenols both in preparation and recovery from training and competition is becoming more prevalent in elite sport. Polyphenols are found in plant foods (e.g. vegetables, cereals, legumes, fruits, nuts) and beverages (e.g. wine, cider, beer, tea, cocoa) however the efficacy of polyphenols as antioxidant compounds greatly depends on their chemical structure which is beyond the scope of this chapter and covered in greater depth elsewhere (Bravo, 1998). High antioxidant and anti-inflammatory activity have been identified in tart cherries, which are considered good sources of phenolic compounds (Connolly et al., 2006). Consumption of approximately 45 cherries per day has been shown to reduce circulating concentrations of inflammatory markers in healthy men and women (Jacob et al., 2003; Kelley et al., 2006) however supplementation of tart cherry juice is a more efficient and economical alternative. Following a period of supplementation, tart cherry juice has been shown to reduce the extent of muscle damage, improve recovery of function and alleviate the sensation of pain following a bout of eccentric elbow flexion contractions (Connolly et al., 2006).

Non-steroidal anti-inflammatory drugs (NSAIDs; e.g. ibuprofen, aspirin) are common ergogenic means in sport proposed to reduce EIMD by inhibiting the synthesis of prostaglandin (Howatson and van Someren, 2008). Ibuprofen taken before and after eccentric arm exercise (2400mg) has been shown to reduce CK appearance in the blood indicating reduced muscle damage (Pizza et al., 1999), while other research has found no effect of ibuprofen on recovery of strength or muscle soreness following downhill running which is characterised by a large eccentric demand (Donnelly et al., 1990).

Various nutritional strategies are available to maintain immune function and avoid infection including colostrum, herbals (e.g. echinacea, ginseng, kaloba), glutamine and zinc lozenges however there is no strong evidence to suggest that any of these prevent exercise-induced immune depression (Gleeson, 2010). Supplementation with probiotics may enhance immune function and reduce URTI and gastrointestinal symptoms however further research is required (Gleeson, 2010).

Maintaining hydration status is also important for performance and recovery. The use of rehydration strategies following exercise should be considered in terms of providing a favourable hormonal environment for adaptation and facilitating preparation for the next session (Judelson et al., 2008). For example, based on research by Shirreffs et al. (1996) it is common for to calculate the required fluid volume to rehydrate by weighing the athlete pre and post exercise ([post exercise weight – pre exercise weight] x1.5). Consideration should also be given to electrolyte content of post-exercise beverages and/or nutrition, which may be prescribed according to individual needs (Holway and Spriet., 2011).

4.3. Evidence that post-exercise recovery strategies may attenuate adaptation to exercise

Reducing the extent of fatigue by using post-exercise recovery strategies may have great importance for enhancing athlete recovery and subsequent performance; however research suggests that the inflammation process is necessary for adaptation to an exercise stressor (Arnold et al., 2007).

Despite initiating a series of events which result in further muscle damage and functional impairment, it appears that inflammatory cells (including neutrophils) are also mitogenic activators for different cells involved in repair (Butterfield et al., 2006; Jarvinen et al., 2005; Smith et al., 2008). Research has shown that macrophages recruited by injured muscle of a phagocytic, pro-inflammatory phenotype may convert to an anti-inflammatory phenotype releasing growth factor (Arnold et al., 2007). Furthermore, increasing the cell volume of

tissue from lactating Wistar rats has been shown to improve protein synthesis and this suggests that cell volume (i.e. inflammation) may be an important signal for protein metabolism (Grant et al., 2000; Millar et al., 1997). Therefore, reducing the extent of EIMD and thus inflammation by means of post-exercise recovery strategies may have important implications for training adaptations.

4.3.1. Cold water immersion (CWI)

Two studies have assessed the effect of CWI on training adaptation by immersing one limb in CWI while the other acts as a control. In their study, Yamane et al. (2006) demonstrated that CWI of one leg following endurance training sessions (3 sessions per week for 6 weeks) compromised gains in maximal oxygen uptake, and ventilation threshold as well as gains in cycle time to fatigue and arterial growth when compared to the leg exposed to passive recovery. Furthermore, cryotherapy of one arm following forearm flexor strength training (3) sessions per week for 4 weeks) resulted in compromised gains in muscular endurance and brachial artery diameter compared to the arm receiving CWI. In both cases, the proportions of significant versus non-significant training effects in the control and CWI groups were different from what would have been expected from random occurrence (p<0.05), strongly suggesting that post-exercise cooling can attenuate training adaptations. Similar to the findings of Yamane et al. (2006), Frohlich et al. (2014) found that CWI attenuated adaptation to strength training. Seventeen male sport students completed 5 weeks of strength training which consisted of single leg curl training performed twice a week. Following each session individuals immersed one leg in cold water for 3x4min intervals ($12.0 \pm 1.5^{\circ}C$; 30s rest between), while the other was not cooled. Following 5 weeks training, there were increases in 1RM and 12RM leg curl test measures in both the cooled and non-cooled legs however increases tended to be greater in the leg which was not-cooled post-strength training. Furthermore, following a two week detraining period there was tendency for further increases in strength, which were significantly larger in the control leg for the 12RM test but not the 1RM test.

The results of Yamane et al. (2006) and Frohlich et al. (2014) therefore suggest that CWI used post-training may be counterproductive to training adaptation. Yamane et al. (2006) explained that the myofibre micro-damages, and cellular and humoral events induced by endurance and strength training are preconditions not only for repair processes, such as myofibre regeneration, but also for the adaptive processes leading to improved muscular performance. In particular, they highlight satellite cell proliferation and recruitment as the

basis of myofibre hypertrophy and increased capillary supply. However, by reducing the extent of muscle damage post exercise, the stimulation and proliferation of satellite cells are reduced and this interferes with the regenerative processes and "thus retard rather than support the desired improvement of muscular performance" (Yamane et al., 2006). Research shows that superficial cooling may also impair anabolic and inflammatory responses post exercise (Nemet et al., 2009). Twelve elite junior handball players completed an intervention involving 4x250m treadmill runs at 80% of each individuals maximum speed followed by a rest period with and without (resting supine in a quiet room for 1h, 21°C) local cold pack application to the hamstring muscle (applied using a compression wrap for 15min, followed by a 15min interval and an additional 15min application). Significant differences were found 1h post exercise with local cold pack application associated with significant decreases in IL-1β (pro-inflammatory), IL-1ra (anti-inflammatory), IGF-I and IGFBP-3 (anabolic hormones) and a greater increase of IGFBP-1 (catabolic agent). Thus, the authors concluded that the recovery strategy may attenuate the anabolic effects of preceding training (Nemet et al., 2009), which over a period of training may explain the findings of Yamane et al. (2006) and Frohlich et al. (2014).

Despite the evidence presented, recent research in trained athletes does not support the suggestion that CWI attenuates adaptation to training (Halson et al., 2014). During a precompetition training period, male endurance-trained cyclists competing at national level were randomised to a CWI group (n=10) or a control group (n=11) for 7 days of baseline training, 21 days intensified training and an 11 day taper. Throughout the training period two weekly tests were used to track changes in fatigue and performance. Test efforts were performed on a commercially available air braked ergometer (Watt Bike, UK) which allowed measurement of power and cadence throughout the efforts. The first test involved 2x4min maximal efforts completed 42min apart (2xMMP_{4min}), while the second was a high intensity interval test (HIIT). Cold water immersion was performed four times per week in the CWI group, in which subjects submerged their body (excluding head and neck) for 15min (15.3 \pm 0.3°C) within 30min of the cessation of training or testing.

Following the training period, both groups improved in the $2xMMP_{4min}$ test however the CWI group (~9.3%) did so to a slightly greater extent than the CON group (~6.5%). Furthermore, during the HIIT both groups increased self-selected power output in the timed pursuits, however increases were bigger in the CWI group for pursuit 1 (~2.4%) and pursuit 2 (~2.1%). Not only do these findings suggest CWI does not impair training adaptation within trained athletes, but they suggest that CWI may enhance adaptation when compared to a control group (Halson et al., 2014). However there are limitations to the study design.

Firstly, CWI was only administered 4 times a week; therefore CWI was not administered after training on 13 out of 30 training days. Furthermore, it should be noted that post-training performance was taken from days 4 and 5 of the taper rather than at the end, as not all subjects were willing to perform maximal efforts towards the end of the taper due to subsequent National cycling competition. It has been demonstrated in literature that CWI may enhance the short term recovery of performance compared to CON (e.g. Vaile et al., 2008), which may explain why 10min time trial performance in the HIIT appears to be reduced to a greater extent during the intensified period in the CON group compared to the CWI group (Figure 4.2.). With training planned so that performance was optimised at the end of the taper, improvements in physical preparation were unlikely to be realised after 4-5 days. Therefore post-training data used in the study may not reflect training adaptation from the training period, particularly when the extent of fatigue during the intensified training period appears to be greater in the CWI group (Figure 4.2).





Figure 4.2. Peak power (W) during the 10min time trial during the HIIT. Clear and dark circles represent the CON and CWI groups respectively. B = Baseline, ITP = Intensified training (Halson et al., 2014).

4.3.2. Dietary supplementation

Dietary antioxidants offer an alternative passive treatment for enhancing muscle recovery by reducing ROS production and through other means such as sarcolemma stabilisation (vitamin E) and reducing pro-inflammatory mediators (omega-3 fatty acids). Despite evidence of dietary antioxidants attenuating muscle damage (Connolly et al., 2006; Howatson et al., 2009), research suggests they may in fact impede the processes which lead to adaptation (Fischer et al., 2004; Gomez-Cabrera et al., 2008; Nemet et al., 2009; Strobel et al., 2010; Trappe et al., 2002; Yamane et al., 2006).

Using human and animal (rat) models, Gomez-Cabrera et al. (2008) studied the effect of vitamin C supplementation during 8 weeks training on VO_{2max} and endurance capacity. Vitamin C administration increased the plasma concentrations of the antioxidant in both men and rats compared to a control group, but only VO_{2max} improved in the control group in the rat study. Although the difference was not significant, the control group improved their VO_{2max} to a greater extent than the supplemented group (17.0 vs. 4.7% respectively) in humans. Skeletal muscle analysis of rats indicated that training increased antioxidant enzyme concentrations, whereas lower mRNA concentrations of two antioxidant enzymes were found in the supplemented group. This suggests that supplementation may hinder these adaptations possibly by preventing the activation of mitochondrial biogenesis, which is indicated by no changes in protein concentrations of cytochrome C. These authors concluded that antioxidant supplementation lowered training efficiency, such that their use during certain phases of the season should be questioned.

Previous research has shown that antioxidant supplementation (28 days) can reduce cortisol and C-reactive protein following repeated knee-extensions (Fischer et al., 2004), but this treatment also blunted the release of IL-6, demonstrating that antioxidant supplementation has the potential to enhance recovery by reducing inflammation. More recently, Strobel et al. (2010) investigated the long-term effects of antioxidant supplementation with vitamin E and α -lipoic acid on changes in markers of mitochondrial biogenesis. Male wistar rats were divided into four groups; sedentary control diet, sedentary antioxidant diet, exercise control diet and exercise antioxidant diet. The exercise groups performed 90min of exercise 4 days a week at 70% VO_{2max} for 14 weeks. Rats were fed powdered standard rat chow with or without 1000 IU vitamin E.kg⁻¹ diet and 1.6 α -lipoic acid.kg⁻¹ diet. Training elevated markers of mitochondrial biogenesis PGC-1 α mRNA and protein, COX IV and cytochrome C protein abundance, citrate synthase activity and

ARE transcription factor. However, supplementation reduced PGC-1 α mRNA, PGC-1 α and COX IV protein, and citrate synthase enzyme activity in both sedentary and exercise trained rats (Strobel et al., 2010). This study provides evidence that vitamin E and α -lipoic acid supplementation could impair the endogenous metabolic and redox status of skeletal muscle in sedentary people, and prevent some of the beneficial adaptations to exercise training (Strobel et al., 2010).

In addition to attenuating training induced increases in endogenous antioxidant defence capacity, Ristow et al. (2009) found supplementation of vitamin C (1000mg.day⁻¹) and vitamin E (400IU.day⁻¹) impaired increases in insulin sensitivity following 4 weeks of training in trained and untrained individuals. By blunting the exercise-induced formation of ROS, Ristow et al. (2009) proposed dietary antioxidants may prevent the activation of ROS-dependent transcriptional coactivators, transcription factors and their targets, which may be important factors in promoting insulin sensitivity. Not only does this study show that antioxidant supplementation may prevent some of the protective effect of exercise on health, but this study may have great implications for an athlete's dietary recovery/preparation and body composition.

Research also demonstrates that analgesic drugs, commonly consumed to reduce or prevent pain and soreness associated with unaccustomed exercise may blunt protein synthesis (Trappe et al., 2002). Twenty four males were allocated to either a maximal over-the-counter dose of ibuprofen (1200mg.day⁻¹), acetaminophen (4000mg.day⁻¹) or a placebo following 10-14 sets of 10 eccentric repetitions at 120% of concentric 1RM with the knee extensors. Compared to the placebo group, neither analgesic had an effect on whole body protein breakdown or perceived muscle soreness; however, they blunted the increase in muscle protein synthesis, as measured by protein fractional synthesis rate. Thus, the authors concluded that the long term use of these (or similar) drugs may inhibit the normal hypertrophic response to resistance training (Trappe et al., 2002). However, adaptations to resistance training have shown to be enhanced by acetaminophen and ibuprofen supplementation in older adults compared to consumption of a placebo (Trappe et al., 2011). Supplementation of acetaminophen (4000mg.day⁻¹) and ibuprofen (12000mg,day⁻¹) increased muscle volume (12.5 and 10.9% respectively) and knee extensor 1RM strength (19 \pm 2kg both groups) to a greater extent than the placebo (8.6% and 15 \pm 2kg increase in volume and strength respectively). Furthermore, muscle biopsies from the vastus lateralis before and after training showed muscle protein content, muscle water content, myosin heavy chain distribution and muscle content of the two known enzymes potentially targeted by the supplements taken (COX-1 and COX-2) were not influenced by supplementation of either acetaminophen or ibuprofen. Another study from this group reported that 12 weeks of knee extensor training increased tendon cross sectional area (3%, p<0.05) when older adults consumed acetaminophen (4000mg.day⁻¹), but no differences were observed when individuals consumed either ibuprofen (1200mg.day⁻¹) or a placebo during training (Carroll et al., 2011). Supplementation with acetaminophen was also associated with greater tendon mechanical adaptations compared to the ibuprofen and placebo groups (Carroll et al., 2011).

4.3.4. Summary

Although equivocal evidence has been presented, literature does exist to support the hypothesis that attenuating the inflammatory process associated with muscle damage (using different recovery strategies) may inhibit the stimulus for adaptive physiology (Fischer et al., 2004; Frohlich et al., 2014; Gomez-Cabrera et al., 2008; Nemet et al., 2009; Strobel et al., 2011; Trappe et al., 2002; Yamane et al., 2006).

To assist in the development of best practices for both recovery and training, further research is needed assessing the effect of strategies on adaptation with particular attention to elite athletes under normal training and in particular with measurement in competitive conditions.

4.4. Other considerations and contraindications for the use of post-exercise recovery strategies

4.4.1. Relationship between psychological and physiological recovery

Research exists to suggest that physiological functions may be somewhat determined by the psychological response to a stressor. For example, in non-athletic settings acute changes in testosterone concentration have been demonstrated following the intervention of video footage (Hellhammer et al., 1985; Pirke et al., 1974; Stoleru et al., 1993). Studies have also shown that a testosterone response elicited from watching different video clips may influence subsequent exercise performance in elite athletes (Cook and Crewther, 2012b). After the viewing of erotic, aggressive and training clips, athlete testosterone levels increased which correlated to an improvement in subsequent 3RM squat (r=0.85, p<0.001) when compared to a control condition when no video was shown (Cook and Crewther, 2012b).

Previous research has also shown that perceptions and emotions associated with stress, such as depression and anger (e.g. Gounin et al., 2008), negatively affected wound healing through psychoneuroimmunological pathways (e.g. Christian et al., 2006; Keicolt-Glaser et al., 1998). Data also suggests that the physiological responses of elite sportspeople may exhibit a degree of plasticity that can be influenced by prior exposure to stressful events or perceptions of those events (Crewther and Cook, 2012a). The post-game presentation of individual specific video footage appears to influence the free hormonal state of rugby players and game performance several days later (Crewther and Cook, 2012a). Research therefore suggests that the use of relatively simple 'psychological' recovery interventions, with specific relevance to that event, may modify the free hormonal state of the athlete which may influence subsequent recovery and performance. Indeed, a recent study by Cook and Beaven (2013) suggests that psychological perception and physiological change summate (or more importantly can act in opposition) in terms of recovery outcome. Their study using elite rugby players exhibited that the degree of body temperature normalisation after exercise following a cool immersion predicted recovery outcomes but was co-founded by the psychological perception of the treatment. Namely if temperature declined but players reported not enjoying the procedure recovery was less than predicted by the temperature change itself. This opens up an exciting avenue around psychological recovery and its influence on physical expression.

4.4.2. Inter-individual variation

Practitioners should be weary of inter-individual responses to a stressor. Research shows an individual nature of recovery following contact sport suggesting that recovery and subsequent training should be individually prescribed (West et al., 2014). Effective monitoring procedures should be established to determine expected recovery patterns, and how these may vary according to performance, and to identify athletes who may require altered recovery/training provision.

The inter-individual response to post-exercise strategies should also be considered. The interactions between psychological and physiological recovery as described previously would suggest a rationale for the application of recovery strategies aimed to enhance physiological function which are perceived to reduce pain, soreness and fatigue within competition periods. It

would also be pertinent for practitioners to monitor individual responses to various means which seek to enhance psychological recovery.

4.5. Summary

Despite the mechanisms underlying each method remaining unclear, evidence exists to suggest that various strategies used post-exercise may attenuate the extent of fatigue and enhance recovery. Such strategies could be employed by athletes between training and/or competitive bouts to reduce acute impairments in performance and the risk of injury induced by fatigue, whilst also reducing the risk of illness and overreaching in the longer term.

Recent research suggests that recovery strategies may enhance adaptation in trained athletes however literature also supports the alternative hypothesis that the induction of muscle damage and the subsequent inflammatory response may be a pre-requisite for the signalling mechanisms which initiate repair and growth of cells, leading to greater adaptive changes. For example, some recovery strategies (e.g. CWI, dietary antioxidants) have been shown to impair the acute inflammatory response to exercise and attenuate adaptations to training in non-athletic populations. Therefore, further research is required within athletic settings to assess the effect recovery strategies may have on adaptation.

Recent literature also suggests that physiological and psychological recovery may be intertwined; as research suggests that individual appraisal to a stressor may have great influence on subsequent physiological recovery and readiness to train/compete. These findings challenge previous approaches to the research and provision of athlete management, opening an exciting and previously untapped area of recovery science.

EXPERIMENTAL STUDY 1

5. Quantifying positional and temporal movement patterns in professional rugby union using GPS

5.1. Introduction

A greater understanding of player movement patterns in rugby union, together with the anthropometric and physiological characteristics of players, may give an indication of the positional requirements of performance. This in turn may facilitate the planning and implementation of training programmes that elicit physiological adaptations specific to individual playing demands. Furthermore, knowledge of movement characteristics may enable coaches to monitor individual and team performance during match-play (Quarrie et al., 2013), and may provide an indication of the physiological stress post-match (Takarada, 2003)

To date, the majority of studies have investigated the game demands in rugby union through time motion analysis systems (Austin et al., 2011a; Austin et al., 2011b; Roberts et al., 2008). For example, recent research from Super 14 rugby has reported front row forwards, back row forwards, inside backs and outside backs to cover ~4662, 5262, 6095 and 4774m respectively, with sprinting contributing to ~10, 10, 15 and 13% of the total distance, respectively (Austin et al., 2011a).

Although the total accumulated time players engage in high-intensity exercise during a match can be relatively brief and the distance sprinted by players short, it has been proposed that the ability of players to perform repeated high intensity efforts (RHIE) may be critical to the outcome of the game (Austin et al., 2011a; Roberts et al., 2008; Spencer et al., 2004). Using time motion analysis, previous research has been performed in an attempt to quantify the characteristics of rugby union, with specific attention to RHIE (Austin et al., 2011a). From understanding the movement, and more specifically the RHIE characteristics of performance, drills may then be developed to provide a position-specific training stimulus. However, as highlighted by Cunniffe et al. (2009), labour-intensive motion analysis methods are largely dependent on trained users. Due to the complex movement patterns and varied nature of game play considerable subjectivity may exist when interpreting data making comparison between coders and studies potentially problematic. More recently, the emergence of portable Global Positioning System (GPS) tracking units in sport have provided an alternative means of analysis (McLellan et al., 2008). To utilise the data collected from GPS it is important sport scientists and coaches have an understanding of expected movement patterns. However, currently only three studies have examined time motion analysis of elite rugby union using GPS units (Cahill et al., 2013; Coughlan et al., 2011; Cunniffe et al., 2009) with two of these studies using only a very small sample of players (n=2; Coughlan et al., 2011; Cunniffe et al., 2009). Furthermore, the reliability and construct validity of 1 and 5Hz devices used in these studies suggest they may lack the sensitivity to accurately quantify changes in movement patterns in team sport (Coutts and Duffield, 2010; Jennings et al., 2010).

Recent advancements in GPS technology have made 10Hz units commercially available which appear to be more acceptable for quantifying movement patterns in rugby union (Castellano et al., 2011; Varley et al., 2012). For example, Varley et al. (2012) reported that a 10Hz GPS unit was 2-3 times more accurate for instantaneous velocity during tasks completed at a range of velocities compared to criterion measure, 6 times more reliable for measuring maximum instantaneous velocity and importantly was able to detect the smallest worthwhile change during all phases of acceleration/deceleration. Therefore, to enhance knowledge of the movement patterns of elite rugby union players using GPS, further research is required sampling at 10Hz.

Previous research has also assessed how movement patterns and physical demands change throughout match-play in team sports using GPS (Austin and Kelly, 2013). Understanding how movement variables change may give an indication of the most demanding periods of play and may help understand the effects of fatigue and/or pacing throughout a game (Austin and Kelly, 2013). This may further facilitate preparation of position specific drills and may aid player evaluation during and following match-play.

The aim of this study was to examine the movement patterns of elite rugby union using GPS sampling at a frequency of 10Hz, with particular attention to the sprint and RHIE characteristics of performance and temporal changes in movement.

5.2. Methods

To examine the movement patterns of elite rugby union match play, 33 professional rugby players (mean \pm SD: 24.8 \pm 3.5yrs, 104.0 \pm 10.6kg) provided 141 GPS data files from 6 European Cup and 7 Celtic League matches' between November to February during the 2012-2013 season. Prior to providing informed consent, participants were given information outlining the rationale, potential applications and procedures associated with the study (Appendix B). Ethical approval was given by the Swansea University Ethics Committee (Appendix A).

GPS units (MinimaxX v.4.0, Catapult Innovations, Melbourne, Australia) were fitted into the back of a custom made vest so that the unit was positioned in the centre area of the upper back and slightly superior to the shoulder blades, with no restriction to the range of movement of the upper limbs and torso. To facilitate familiarisation to wearing the units, players were required to wear the vests and GPS units during outdoor training sessions throughout the season. Within the units a tri-axial piezoelectric linear accelerometer system, which samples at a frequency of 100Hz allowed for the detections of short high-intensity bursts in rugby union that do not allow the attainment of high speed. Using these accelerometers, previous research by Gabbett et al. (2011) reported that tackle detection; a feature of the MinimaxX unit (Catapult Innovations, Melbourne Australia) which also incorporates gyroscopes and magnetometers imbedded within the unit, was valid in quantifying the contact load of collision sport athletes. These findings also allowed Gabbett et al. (2012) to modify previous definitions of a repeated high intensity (RHIE) bout (Austin et al., 2011a; Spencer et al., 2004) so that it may be defined as 3 or more high acceleration (>2.79m.s⁻²), high speed (5m.s⁻¹) or contact efforts with less than 21s recovery between efforts. Thus in addition to quantifying distances and distances at different velocities, the study analysed the contact and RHIE characteristics of players. In addition, two custom parameters derived from GPS analysis software to express the instantaneous and accumulative demands of exercise; exertion index (Wisbey et al., 2010) and player load are reported (Young et al., 2012).

Data files were only included for the analysis of movement if a minimum of 60min match play was performed (McLellan et al., 2011b). Analysis was therefore conducted from 53 forward and 59 back data files. To analyse positional demands players were separated into positional groups; tight forwards (27 data files; prop, hooker, second rows), loose forwards (26 data files; open-side flanker, blind-side flanker, number 8), half-backs (14 data files; scrum-half, outside-half), inside backs (17 data files; inside centre, outside centre) and outside backs (28 data files; wingers, full-

bac:). Movement data was downloaded and analysed using Catapult Sprint software (Catapult Innvations, Melbourne, Australia) for analysis of distances covered, distances at different velocities, max velocity, accelerations/decelerations, contacts, exertion index, player load, sprint patterns and RHIE. A further analysis of sprinting and RHIE characteristics was also performed. Disances at different velocity zones were analysed according to the classification system described by McLellan et al. (2011b). Walking (0-1.6m.s⁻¹), jogging (1.6-2.7m.s⁻¹), cruising (2.7-3.8n.s⁻¹) and striding (3.8-5.0m.s⁻¹) were categorised as low-speed movement, while high-intensity running (5.0-5.5m.s⁻¹) and sprinting (>5.6m.s⁻¹) were regarded as high-speed movements (Gabbett et al., 2012).

In addition, a temporal analysis of movement patterns was investigated using data files from players who completed the full game (n=71). Matches were separated into 10min periods with time played >40min in each half excluded. For the analysis of temporal movement, distances covered at low (1-2m.s⁻²), moderate (2-3m.s⁻²) and high (>3m.s⁻²) accelerations and decelerations were categorised according to previous temporal research by Akenhead et al. (2013).

As game time varied between positional groups, a comparison of positional movement demands was made relative to game time (e.g. metres per minute; referred to as meterage, m.min⁻¹). Positional differences were examined using one-way repeated measures ANOVA with bon/erroni corrected post-hoc analysis. Time-course changes in movement patterns were examined using repeated measures ANOVA, with bonferroni corrected pairwise comparisons. Data were analysed using SPSS v20 for Windows (SPSS Inc., Chicago, IL), with the level of significance set at p<0.05, and represented as mean \pm SD.

5.3. Results

There were significant differences between positional groups for game-time (p<0.001), exertion index (p<0.001), player load (p<0.01), and total number of contacts (p<0.001). On average, tight forwards, loose forwards and half backs played 78 ± 12 , 87 ± 12 and 83 ± 12 min respectively. Post-hoc analysis shows on average inside and outside backs played significantly longer than tight forwards (91 ± 11 vs. 92 ± 10min respectively; p<0.05). Post-hoc analysis also showed half backs, inside backs and outside backs had significantly greater exertion index values (43.2 ± 11.3, 45.9 ± 7.8 and 44.1 ± 10.0 respectively) than tight forwards (32.8 ± 8.3; p<0.05), but not loose forwards (37.5 ± 9.9; p>0.05).

Player load values for loose forwards and half backs (625 ± 104 and 617 ± 81 respectively) were significantly greater than tight forwards (528 ± 97 ; p<0.05), however inside and outside backs were not significantly different from other positions (582 ± 63 and 590 ± 77 respectively; p>0.05). A greater number of total contacts were detected for tight forwards compared to outside backs (30 ± 15 vs. 16 ± 8 , p<0.05). Furthermore, there was a significantly greater number of contacts for loose forwards (38 ± 16) than half backs (19 ± 9), inside backs (21 ± 11) and outside backs (p<0.05).

Comparisons of absolute and relative distances covered are shown in Tables 5.1a and 5.1b respectively. There were significant differences between positional groups for total absolute difference covered and absolute distances covered walking, striding, high-intensity running, low-speed running and high-speed running (p<0.05; Table 5.1a). Furthermore, there were significant differences in relative total distance and relative distances covered between positional groups for walking, cruising, striding, high-intensity running, sprinting, low-speed running and high-speed running (p<0.05; Table 5.1a).

A summary of sprint characteristics displayed in Tables 5.2a and 5.2b also display significant positional group differences for the number of sprints performed, maximum speed and the mean and maximum sprint distance (p<0.05; Tables 5.2a and 5.2b).

A summary of RHIE characteristics are displayed in Tables 5.3a. and 5.3b. There were significant positional group differences for the number of RHIE bouts, the maximum number of efforts per bout, the mean and maximum effort recovery time within a RHIE bout, and the mean recovery time between RHIE bouts (p<0.05; Tables 5.3a and 5.3b).

Despite there being no difference in meterage between the two halves, Table 5.4 demonstrates significant time effect differences in player load, cruising $(m.min^{-1})$ and striding $(m.min^{-1})$ from the first to the second half. Table 5.4 also displays temporal changes in movement patterns in 10min periods, with significant time effect changes in player load, RHIE bout number, contacts, total meterage and meterage at several velocities (p<0.05, Table 5.4).

	Number	Total	Walking	Jogging	Cruising	Striding	HI running	Sprinting	Low-speed	High-spe
Tight forwards	27	4757 ± 885	2085 ± 435	1071 ± 228	1014 ± 283	441 ± 184	81 ± 43	65 ± 46	4610 ± 856	147 ± 80
Prop	4	3698 ± 574	1756 ± 309	917 ± 102	706 ± 352	218 ± 147	51 ± 34	51 ± 51	3596 ± 507	102 ± 8
Hooker	7	4746 ± 651	1832 ± 278	1151 ± 197	1094 ± 198	523 ± 187	88 ± 40	58 ± 26	4600 ± 625	147 ± 5′
Second row	16	5027 ± 864	2278 ± 424	1074 ± 251	1056 ± 261	460 ± 153	86 ± 45	72 ± 52	4869 ± 846	158 ± 8 (
Loose forwards	26	5244 ± 866	2225 ± 340	1092 ± 221	997 ± 262	620 ± 219^{a}	140 ± 63^{a}	166 ± 116^{a}	4935 ± 755	306 ± 17
Blind-side flanker	7	4868 ± 854	2024 ± 493	969 ± 221	1017 ± 236	611 ± 231	134 ± 28	113 ± 34	4621 ± 847	247 ± 29
Open-side flanker	6	5741 ± 627	2249 ± 167	1194 ± 139	1090 ± 212	748 ± 200	185 ± 72	268 ± 129	5281 ± 509	453 ± 19
Number 8	10	5059 ± 924	2344 ± 295	1087 ± 252	899 ± 306	512 ± 182	104 ± 48	112 ± 78	4843 ± 818	216 ± 11
Half backs	14	5693 ± 823^{a}	2436 ± 372	1123 ± 265	1041 ± 243	711 ± 236^{a}	155 ± 71^{a}	226 ± 112^{a}	5311 ± 705	381 ± 17
Scrum half	S	4987 ± 725	2225 ± 371	909 ± 238	852 ± 192	674 ± 281	144 ± 98	177 ± 107	4661 ± 557	322 ± 2(
Outside half	6	6086 ± 595	2254 ± 336	1241 ± 203	1146 ± 206	731 ± 222	160 ± 56	253 ± 111	5673 ± 491	413 ± 15
Incida hacke		5007 + 700 ⁸	7545 + 301 ^{8b}	1067 + 187	1008 ± 173	700 ± 176 ⁸	300 + κζ^{ab}	379 ± 1 40^{abc}	5371 ± 610	586 + 181
Inside centre	È C	5661 + 875	2357 + 320	1090 + 218	791 + 070	680 + 134	00 - 702	344 + 102	5114 + 733	548 + 13
Outside centre	27	6258 ± 276	2315 ± 332	1034 ± 142	1050 ± 98	717 ± 123	216 ± 47	425 ± 196	5616 ± 110	642 ± 23
Outside backs	28	6272 ± 1065^{ab}	2999 ± 590^{abcd}	1201 ± 310	908 ± 205	593 ± 132^{a}	174 ± 52^{a}	392 ± 135^{abc}	$5701 \pm 1007^{\mathrm{ab}}$	566 ± 17 1
Winger	18	6181 ± 1121	2980 ± 633	1140 ± 303	862 ± 186	604 ± 134	178 ± 53	409 ± 151	5586 ± 1066	587 ± 1 9
Full back	10	6436 ± 991	3032 ± 536	1314 ± 306	991 ± 222	572 ± 133	166 ± 53	361 ± 99	5909 ± 907	527 ± 13

 * Significant difference compared to tight forwards; ^bsignificant difference compared to loose forwards; ^csignificant difference compared to half backs; ^dsignificant difference compared to inside backs. p<0.05

	Number	Total	Walking	Jogging	Cruising	Striding	HI running	Sprinting	Low-speed	High-speed
Tight forwards	27	60.7 ± 6.0	26.5 ± 2.8	13.7 ± 2.1	12.9±3.2°	5.6 ± 2.3	1.1 ± 0.6	0.8 ± 0.6	58.8±5.3	1.9 ± 1.0
Prop	4	52.7 ± 4.1	25.1 ± 3.7	13.2 ± 1.9	9.9 ± 4.2	3.0 ± 1.8	0.7 ± 0.5	0.7 ± 0.7	51.3 ± 3.1	1.4 ± 1.0
Hooker	7	65.0 ± 2.9	25.2 ± 3.4	15.7 ± 1.5	15.0 ± 2.1	7.1 ± 2.4	1.2 ± 0.5	0.8 ± 0.4	63.0 ± 3.0	2.0 ± 0.7
Second row	16	60.8 ± 5.4	27.5 ± 2.1	12.9 ± 1.9	12.8 ± 2.8	5.6 ± 1.9	1.1 ± 0.6	0.9 ± 0.7	58.8 ± 4.5	2.0 ± 1.2
Loose forwards	26	60.8 ± 8.4	25.6 ± 2.3	12.6 ± 2.4	11.6 ± 3.3	7.2 ± 2.7	1.6 ± 0.6	1.9 ± 1.2^{a}	57.0 ± 7.4	3.5 ± 1.7 ^ª
Blind-side flanker	7	61.8 ± 8.6	25.3 ± 2.0	12.3 ± 2.7	13.1 ± 3.7	7.9 ± 3.6	1.7 ± 0.4	1.4 ± 0.5	58.7 ± 8.6	3.2 ± 0.5
Open-side flanker	6	63.2 ± 8.2	24.3 ± 2.0	12.9 ± 1.7	11.9 ± 2.8	8.1 ± 2.3	2.0 ± 0.7	2.9 ± 1.3	<i>5</i> 7.3 ± 7.1	4.9 ± 2.0
Number 8	10	57.9 ± 8.2	26.9 ± 2.1	12.5 ± 2.8	10.3 ± 3.1	5.8 ± 1.8	1.2 ± 0.5	1.3 ± 0.8	55.5 ± 7.2	2.4 ± 1.2
	;									
Hall Dacks	14	C./ ± 1.20	C.C ± 0.42	8.2 ± 0.01	17.0 ± 2./	0° / ± 0.2	1.9 ± 0.9	2. / ± 1.3	04.5 ± 0.1	4.0 ± 2.U
Scrum half	5	71.7 ± 7.1	32.0 ± 4.5	13.0 ± 2.8	12.3 ± 2.8	9.7 ± 3.9	2.0 ± 1.3	2.5 ± 1.4	67.1 ± 4.9	4.6 ± 2.7
Outside half	6	67.7 ± 7.7	28.2 ± 1.9	13.9 ± 2.9	12.8 ± 2.8	8.2 ± 2.8	1.8 ± 0.7	2.8 ± 1.3	63.1 ± 6.5	4.6 ± 1.8
Inside backs	17	65.6 ± 5.1	28.0 ± 2.2	11.8 ± 2.1	11.1 ± 1.3	7.7 ± 1.2^{a}	2.3 ± 0.6^{ab}	$4.2 \pm 1.7^{\rm abc}$	58.6 ± 3.3	6.5 ± 2.0^{abc}
Inside centre	10	65.4 ± 4.8	26.9 ± 1.3	12.4 ± 2.0	11.2 ± 1.3	7.8 ± 0.8	2.3 ± 0.6	3.9 ± 1.1	58.3 ± 3.5	6.2 ± 1.2
Outside centre	7	65.9 ± 5.8	29.5 ± 2.2	10.9 ± 2.0	11.1 ± 1.5	7.6 ± 1.7	2.3 ± 0.6	4.5 ± 2.3	59.1 ± 3.2	6.8 ± 2.8
Outside backs	28	68.8 ± 10.0	32.6 ± 4.7^{abd}	13.2 ± 3.2	10.0 ± 2.2^{ac}	6.5 ± 1.5	1.9 ± 0.6^{a}	$4.3 \pm 1.6^{\rm abc}$	62.2 ± 9.1	$6.3 \pm 2.0^{\mathrm{abc}}$
Winger	18	67.6 ± 10.3	32.5 ± 5.2	12.5 ± 3.1	9.5 ± 1.9	6.7 ± 1.5	2.0 ± 0.6	4.6 ± 1.9	61.1 ± 9.4	6.5 ± 2.3
Full back	10	71.0 ± 9.7	32.8 ± 4.0	14.3 ± 3.3	10.9 ± 2.5	6.3 ± 1.6	1.8 ± 0.5	3.9 ± 1.0	64.3 ± 8.6	5.7 ± 1.4

^aSignificant difference compared to tight forwards; ^bsignificant difference compared to loose forwards; ^csignificant difference compared to half backs; ^dsignificant difference compared to inside backs; ^csignificant difference compared to outside backs. p<0.05

	Tight forwards (n=27)	Prop (n=4)	Hooker (n=7)	Second row (n=16)	Loose forwards (n=26)	Blind-side flanker (n=7)	Open-side flanker (n=9)	Number 8 (n=10)
Number of sprints	4 ±3	4 ±3	4 ±2	5±3	10 ± 6^{a}	8 ± 2	15 ± 7	7±4
Max velocity (m.s ⁻¹)	6.2 ± 0.7	6.0 ± 0.6	6.2 ± 0.4	6.3 ± 0.8	6.9 ± 0.9^{a}	6.6 ± 0.6	7.2 ± 0.6	6.9 ± 1.2
Sprint distance (m)								
Mean	13.1 ± 5.4	13.1 ± 3.6	13.3 ± 5.3	13.0 ± 6.1	15.5 ± 3.9	14.7 ± 2.7	17.4 ± 3.9	14.4 ± 4.3
Мах	19.5 ± 10.1	16.0 ± 6.8	19.3 ± 9.2	20.5 ± 11.3	29.4 ± 11.7^{a}	26.8 ± 6.7	40.0 ± 11.4	21.7 ± 7.3
Number of sprints								
0-5m	$0 \neq 0$	$0 \neq 0$	$0 \neq 0$	0 ± 0	0 ± 1	0 ± 0	0 ± 1	0 ± 1
5-10m	1 ± 1	1 ± 1	1 ± 2	2 ± 1	3±3	2 ± 2	4 ± 3	2 ± 2
10-40m	3±2	3 ± 3	3 ± 2	3 ± 2	7 ± 5	5 ± 2	10 ± 7	4 ± 3
>40m	0 = 0	0 ∓ 0	0 ∓ 0	0 = 0	0 ± 1	$0 \neq 0$	1±1	$0 \neq 0$
Starting acceleration of	cominto							
$\frac{1}{2}$	evinde	- c	(- (Ċ		÷		
0-1m.s ²	2 ± 1	7 ± 1	7 ± 7	2 ± 1	3 ± 2	3 ± 1	5 ± 2	2 ± 2
$1-2m.s^{-2}$	2 ± 2	2 ± 2	1 ± 1	2±2	5±3	3 ± 1	7±4	3 ± 2
2-4m.s ⁻²	1 ± 1	0 ± 0	0 ± 1	1 ± 1	2 ± 2	2 ± 1	2 ± 2	1 ± 2
>4m.s ⁻²	0 ± 1	0 ± 0	0 ± 0	0 ± 1	0 ± 0	0 ± 0	0 ± 1	0 ± 0
Recovery time between	sprints							
0-0.5min	$0 \neq 0$	$0 \neq 0$	$0 \neq 0$	0 ± 0	1 ± 2	0 ± 0	2 ± 3	0 ± 1
0.5-1min	$0 \neq 0$	$0 \neq 0$	0 ± 0	0 ± 0	1 ± 1	0 ± 0	1 ± 1	$0 \neq 0$
1-2min	$0 \neq 0$	$0 \neq 0$	$0 \neq 0$	$0 \neq 0$	1 ± 1	1 ± 1	2 ± 1	0 ± 1
2-5min	1±1	1 ± 2	1 ± 1	1 ± 1	2 ± 2	1 ± 1	4 ± 3	1 ± 1
>5min	2 ± 2	2 ± 2	2 ± 1	3±2	5 ± 2^{a}	4 ± 2	6 ± 2	4 ± 2

117

Table 5.2a. Mean (±SD) forwards sprinting characteristics from competition.

.

*Significant difference compared to tight forwards. p<0.05

	Half back (n=14)	Scrum half (n=5)	Outside half (n=9)	Inside back (n=17)	Inside centre (n=10)	Outside centre (n=7)	Outside back (n=28)	Winger (n=18)	Full back (n=10)
Number of sprints Max velocity (m.s ⁻¹)	12 ± 5^{a} 7.5 ± 0.4 ^a	8 ± 4 7.3 ± 0.6	13 ± 5 7.6 ± 0.3	20 ± 7^{abc} 8.0 ± 0.6 ^{ab}	19 ± 4 8.0 ± 0.7	23 ± 10 7.9 ± 0.4	20 ± 6^{abc} 7.8 ± 0.5^{ab}	21 ± 7 7.9 ± 0.5	19 ± 5 7.7 ± 0.5
Distance (m) Mean Max	19.1 ± 5.7 ^ª 40.2 ± 13.9ª	19.3 ± 6.1 35.3 ± 12.0	19.1 ± 5.9 42.9 ± 14.8	17.8 ± 2.7^{a} 49.2 ± 16.1 ^{ab}	17.5 ± 3.0 47.5 ± 17.8	18.3 ± 2.3 51.7 ± 14.3	18.5 ± 3.1^{a} 42.3 ± 10.5 ^{ab}	18.4 ± 3.4 42.1 ± 8.9	18.7 ± 2.6 42.6 ± 13.5
Number of sprints 0-5m	0 ± 1	0 + 0	0 ± 1	0±2	1±2	0 ∓ 0	1±2	1 ± 2	1±2
5-10m	4 ±2	3 ± 1	4 ± 3	6 ± 2^{ab}	5 ± 2	7 ± 3	$6 \pm 4^{\rm abc}$	7 ± 4	6 ± 3
10-40m	7±5	5 ± 4	8 ± 5	13 ± 6^{abc}	12 ± 5	14 ± 7	13 ± 6^{abc}	13 ± 7	12 ± 5
>40m	1±1	1 ± 1	1±1	1 ± 1^{ab}	1 ± 1	1 ± 1	1 ± 1^{a}	1±1	0 ± 1
Starting acceleration c	of sprints								
0-1m.s ⁻²	4 ± 3^{a}	3 ± 3	4 ± 3	5 ± 2^{a}	5 ± 2	4 ± 2	5 ± 2^{a}	5 ± 2	6 ± 2
1-2m.s ⁻²	4 ± 2^{a}	3 ± 1	5±3	$8 \pm 3^{ m abc}$	8 ±3	8 ± 3	$8 \pm 3^{\rm abc}$	8 ± 3	8 ± 3
2-4m.s ⁻²	3 ± 2^{a}	2 ± 1	4 ± 2	7 ± 3^{abc}	6 ± 3	8 ± 4	$6 \pm 3^{\rm abc}$	7±3	5 ± 2
>4m.s ⁻²	0 ± 0	$0 \neq 0$	0 ± 0	2 ± 2^{abc}] ± 1	3 ± 2	1 ± 1^{abc}	2 ± 2	1±1
Recovery time betwee	en sprints								
0-0.5min	1 ± 1	1 ± 1	1±1	2 ± 2^{ab}	2 ± 1	3 ± 2	2 ± 2^{ab}	3 ± 2	2 ± 1
0.5-1min	0 ± 1	0 ± 0	1 ± 1	2 ± 1^{abc}	2 ± 1	2 ± 1	2 ± 2^{abc}	2 ± 2	2 ± 2
1-2min	1±1	1 ± 1	1 ± 2	4 ± 3^{abc}	3 ± 1	5 ± 3	$3 \pm 2^{\rm abc}$	3±2	3 ± 1
2-5min	3 ± 2	2 ± 2	3 ± 3	$6 \pm 4^{\rm abc}$	6 ± 3	6 ± 5	$6 \pm 3^{\rm abc}$	6 ± 3	5 ± 2
>5min	5±2ª	4 ± 1	6 ± 1	6 ± 1ª	6 ± 1	6 ± 2	6 ± 2^{a}	6 ± 2	6 ± 2

119

Table 5.2b. Mean (\pm SD) backs sprinting characteristics from competition.

*Significant difference compared to tight forwards; ^bsignificant difference compared to loose forwards; ^csignificant difference compared to half backs p<0.05

	Tight forwards (n=27)	Prop (n=4)	Hooker (n=7)	Second row (n=16)	Loose forwards (n=26)	Blind-side flanker (n=7)	Open-side flanker (n=9)	Number 8 (n=10)
RHIE bouts	11 ± 8	6 ±4	9 ± 10	13 ± 8	13 ± 7 ^{ce}	11 ± 7	18±6	8±5
Efforts per	bout							
Mean	4 ± 1	3 ± 0	4 ±]	4 ± 1	4 ± 1	4 ± 1	4 ± 0	4 ± 0
Max	7 ± 4	5±1	7 ± 4	7 ± 4	7 ± 3 ^d	8 ±3	7 ± 2	6 ± 3
Effort reco	verv time (s)							
Mean	8.1 ± 2.0^{-2}	9.0 ± 3.2	1.3 ± 2.5	8. 2 ± 1.6	7.3 ± 1.2^{-2}	1.7 ± 1.2	6.6 ± 0.8	7.8 ± 1.4
Мах	19.3 ± 1.9^{de}	19.5 ± 0.8	17.9 ± 3.1	20.0 ± 0.8	18.8 ± 2.7^e	19.8 ± 1.1	19.3 ± 1.5	17.7 ± 3.8
	-	,						
RHIE boun	t recovery time (s)						
Min	153 ± 201	184 ± 172	280 ± 347	99 ± 105	90 ± 122	170 ± 203	32 ± 15	85 ± 61
Mean	398 ± 219	422 ± 60	421 ± 353	384 ± 184	457 ± 376	674 ± 603	262 ± 66	484 ± 250
Max	886 ± 492	749 ± 175	822 ± 636	935 ± 490	1039 ± 579	1334 ± 967	738 ± 344	1110 ± 616

^aSignificant difference compared to tight forwards; ^bsignificant difference compared to loose forwards; ^csignificant difference compared to half backs; ^dsignificant difference compared to inside backs; ^csignificant difference compared to outside backs. p<0.05

Table 5.3a. Mean (±SD) description of forwards repeated high intensity exercise (RHIE) bout characteristics from competition.

side Winger Full back tck (n=18) (n=10) 28)	± 6 7 ± 7 5 ± 3		±0 3 ±0 4 ± 1	± 2 5 ± 3 5 ± 1		±1.9 4.6 ± 2.3 4.8 ± 1.3	± 5.7 14.1 ± 6.5 17.5 ± 3.7		± 460 325 ± 541 253 ± 158	\pm 405 455 \pm 239 667 \pm 536	
Out ba	6∃		4	S. ⊐		5 4.7 =	2 15.4		0 304=	7 551 =	
Outside centre (n=7)	5 ± 2		3 ± 0	4 ± 1		5.3 ± 1.€	16.5±4.		551 ± 71	958±56	
Inside centre (n=10)	8 ± 8		3 ± 1	4 ± 2		4.5 ± 1.6	14.4 ± 6.1		134 ± 98	591 ± 309	
Inside back (n=17)	7±7		3 ± 0	4 ±2		4.9 ± 1.6	15.3 ± 5.3		343 ± 533	751 ± 463^{a}	
Outside half (n=9)	5 ± 5		3 ± 1	5±2		5.9 ± 2.3	15.5 ± 5.5		503 ± 689	609 ± 186	
Scrum half (n=5)	5 ± 1		3 ± 0	4 ± 1		7.5 ± 2.1	20.0 ± 0.8	(s)	222 ± 101	616 ± 402	
Half Back (n=14)	5 ± 4	bout	3 ± 0	5±2	•	very time (s) $6.5 \pm 2.3^{\circ}$	17.3 ± 4.8	recovery time	375 ± 513	612 ± 286	
	RHIE bouts	Efforts per	Mean	Max	5 1	Effort reco Mean	Max	RHIE bout	Min	Mean	

Table 5.3b. Mean (±SD) description of backs repeated high intensity exercise (RHIE) bout characteristics from competition.

^aSignificant difference compared to tight forwards; ^bsignificant difference compared to loose forwards; ^csignificant difference compared to half backs; ^dsignificant difference compared to inside backs; ^csignificant difference compared to outside backs. p<0.05
	0-10min	10-20min	20-30min	30-40min	40-50min	50-60min	60-70min	70-80min	First half	Second h
Player load.min ⁻¹	7.8 ± 1.1	7.0 ± 1.2^{ad}	6.7 ± 1.4 ^{ad}	6.2 ± 1.5^{ad}	7.6 ± 1.2	6.3 ± 1.1 ^{abd}	5.8 ± 1.7^{abcd}	6.0 ± 1.5^{abcd}	6.9 ± 0.8	6.5 ± 0.
Meterage (m.min ⁻¹)	75.3 ± 13.6	68.4 ± 12.5^{a}	65.8 ± 11.1^{ad}	62.0 ± 14.0^{ad}	74.3 ± 12.9	62.5 ± 10.2^{ad}	58.2 ± 16.3^{abcd}	61.8 ± 18.1^{abcd}	67.6 ± 8.0	64.7 ± 1(
E.I.min ⁻¹	0.58 ± 0.19	0.49 ± 0.16^{ad}	0.46 ± 0.14^{ad}	0.41 ± 0.16^{ad}	0.56 ± 0.16	0.42 ± 0.12^{ad}	$0.39 \pm 0.16^{\text{abcd}}$	0.41 ± 0.17^{abd}	0.49 ± 0.12	0.45 ± 0.
Walking (m.min ⁻¹)	29.3 ± 7.1	30.1 ± 5.0	30.0 ± 5.5	30.6 ± 6.4	30.2 ± 6.6	29.2 ± 5.6	28.6 ± 6.7	30.6 ± 9.3	29.8 ± 4.3	29.8 ± 5
Jogging (m.min ⁻¹)	14.5 ± 4.6	13.7 ± 4.0	12.1 ± 3.7^{ad}	12.3 ± 5.0^{d}	14.8 ± 3.9	12.3 ± 3.3^{d}	11.6 ± 5.7^{ad}	11.8 ± 5.4^{d}	12.9 ± 2.6	12.8 ± 3
Cruising (m.min ⁻¹)	15.1 ± 4.5	12.0 ± 4.4^{a}	11.1 ± 4.0^{ad}	9.7 ± 3.7 ^{ad}	13.8 ± 5.0	10.6 ± 3.9^{ad}	9.1 ± 5.1 ^{abd}	9.0 ± 4.3^{abd}	11.8 ± 2.7	10.6 ± 2 .
Striding (m.min ⁻¹)	9.1 ± 3.9	7.3 ± 3.0^{a}	7.4 ± 3.4^{a}	5.7 ± 3.5 ^{ad}	8.8 ± 3.8	6.4 ± 3.4^{ad}	5.0 ± 3.0^{abcd}	5.7 ± 3.6 ^{ªd}	7.4 ± 2.0	6.5 ± 2.3
HI running (m.min ⁻¹)	2.2 ± 1.6	1.9 ± 1.6	2.1 ± 1.6	1.4 ± 1.3^{ad}	2.1 ± 1.5	1.7 ± 1.3	1.3 ± 1.0^{ad}	1.6 ± 1.4	1.9 ± 0.8	$1.7 \pm 0.$
Sprinting (m.min ⁻¹)	4.8 ± 4.0	3.3 ± 3.0	3.1 ± 2.8^{a}	2.1 ± 2.4^{ad}	4.6 ± 4.4	2.2 ± 2.7^{ad}	2.6 ± 3.0^{ad}	3.0 ± 3.1	3.4 ± 2.1	3.0 ± 2 .
High-speed (m.min ⁻¹)	7.0 ± 4.9	5.2 ± 4 .1	5.2 ± 3.8	3.4 ± 3.1 ^{ad}	6.8 ± 5.1	3.9 ± 3.6^{ad}	3.9 ± 3.5^{ad}	4.6 ± 4.0	5.3 ± 2.7	4 .7 ± 2.
Low-speed (m.min ⁻¹)	68.0 ± 12.7	63.1 ± 10.2	60.6 ± 9.2^{ad}	58.5 ± 12.1ªd	67.6 ± 10.4	58.5 ± 8.9^{ad}	54.2 ± 14.9^{abcd}	57.1 ± 16.7^{ad}	62.0 ± 6.6	59.7±9
High dec >-3m/s/s (m)	7.2 ± 3.6	5.6 ± 3.1^{ad}	5.9 ± 3.7	4.4 ± 3.3^{ad}	7.4 ± 4.0	4.3 ± 3.1^{acd}	4.2 ± 3.0^{acd}	4.0 ± 3.4^{acd}	28.3 ± 10.6	25.9±13
Mod dec -3 to -2 m.s ⁻² (m)	11.2 ± 5.0	9.5 ± 4.0^{ad}	8.7 ± 3.7 ^{ad}	7.7 ± 4.0ªd	11.7 ± 4.2	8.0 ± 3.6^{ad}	7.0 ± 4.2^{abd}	7.1 ± 4.2^{abd}	44.2 ± 11.7	42.3 ± 1 3
Low dec -2 to -1 m.s ⁻² (m)	34.2 ± 10.7	29.2 ± 9.5^{d}	27.8 ± 9.1^{ad}	24.4 ± 10.0^{ad}	34.6 ± 8.9	24.9 ± 8.3^{ad}	23.0 ± 10.6^{abd}	23.1 ± 9.8^{abcd}	133.8 ± 23.2	128.6 ± 2
Low acc 1 to 2m.s ⁻² (m)	43.5 ± 12.9	36.6 ± 10.4^{ad}	37.2 ± 11.0^{ad}	31.5 ± 11.3^{ad}	44.4 ± 10.8	32.5 ± 10.4^{ad}	29.9 ± 12.6^{abcd}	30.8 ± 12.4^{acd}	172.6 ± 29.0	165.8 ± 3
Mod acc 2 to 3m.s ⁻² (m)	16.2 ± 5.7	13.5 ± 4.8^{ad}	13.4 ± 5.1^{ad}	11.4 ± 5.2^{ad}	17.2 ± 6.2	11.8 ± 4.3^{ad}	10.7 ± 5.3^{abcd}	11.5 ± 6.0^{ad}	64.0 ± 14.6	62.9±18
High acc >3m.s ⁻² (m)	11.7 ± 5.3	9.9 ± 4.8	10.3 ± 5.9	9.1 ± 5.5	11.2 ± 4.9	8.5 ± 3.7^{ad}	8.6 ± 5.1^{ad}	9.1 ± 6.9	47.3 ± 15.2	46.5 ± 2 ₄
RHIE bouts	1.2 ± 1.2	1.2 ± 1.4	1.7 ± 2.2	1.3 ± 2.1	1.7 ± 1.6	1.0 ± 1.2^{d}	0.9 ± 1.2^{d}	0.9 ± 1.0^{d}	4.6 ± 4.1	5.3 ± 4.
Contacts	2.9 ± 2.5	3.1 ± 3.0	4.1 ± 4.6	3.7 ± 5.0	4.0 ± 3.8	2.5 ± 2.2^{d}	2.3 ± 2.1 ^d	2.5 ± 2.4^{d}	12.3 ± 9.5	12.6±9

Table 5.4. Temporal changes (mean \pm SD) in movement patterns throughout match-play (n=71).

*Significant difference from 0-10min; ^bsignificant difference from 10-20min; ^csignificant difference from 20-30min; ^dsignificant difference from 40-50min; *significant difference from 1st half. p<0.05

5.4. Discussion

The aim of the present study was to assess the movement patterns of professional rugby union players which may help coaches and practitioners understand the physiological demands of performance (Austin et al., 2011a). Sampling at a frequency of 10Hz, the present study is the first assessment of the movement patterns in rugby union using units valid and reliable for detecting high acceleration/deceleration and high-speed movements associated with rugby union (Castellano et al., 2011; Varley et al., 2012). The study is also the first to report exertion index, player load, and provide a detailed analysis of sprint and repeated high intensity characteristics using GPS technology. Furthermore, we are first to demonstrate temporal changes in movement characteristics throughout match-play in rugby union.

Total distances reported in the present study are smaller than those previously reported (Cahill et al., 2013; Coughlan et al., 2011; Cunniffe et al., 2009) using GPS to assess movement patterns. However values for all players, forwards and backs are similar to values obtained by time motion analysis (TMA) reported by previous research (Austin et al., 2011b; Quarrie et al., 2013; Roberts et al., 2008). With advancements in methodology one may assume that the greatest discrepancies in results would be with TMA methods. However methodological issues may explain differences with previous GPS research. For example research by Coughlan et al. (2011) and Cunniffe et al. (2009) only assessed the movement patterns of 1 forward and 1 back. However, this does not explain differences to the research by Cahill et al. (2013) who conducted a comprehensive analysis of English Premiership rugby which assessed 276 GPS data files. Furthermore, 1 and 5Hz units have been proposed to underestimate distance covered following a tortuous route (Duffield et al., 2010; Petersen et al., 2009), therefore variances in the total distances covered in the present study compared to previous research may not directly relate to measurement error. Other possible explanations for differences in movement characteristics may be due to differing playing standards, the team assessed, opponents' tactics, varied game characteristics and the period of the season from which games were assessed.

Our findings support previous research which show that movement demands and thus the physiological demands of performance vary between positional groups (Cahill et al., 2013; Quarrie et al., 2013; Roberts et al., 2008). For example, this study found outside backs covered a greater distance (~6272 vs. ~5244m), at a greater meterage (~68.8 vs. ~60.8m.min⁻¹), performed a greater number of sprints (~20 vs. ~10) and reached a greater maximum velocity (~7.8 vs. $6.9m.s^{-1}$) than loose forwards (p<0.05).

In addition to quantifitying movement patterns, the paper is the first to characterise EI and player load accumulated during performance. Exertion index values further demonstrate that forwards have smaller running demands compared to backs, however they have greater contact and repeated high intensity demands. Loose forwards performed a significantly greater number of contacts (\sim 38) compared to half, inside and outside backs (\sim 19, \sim 21, \sim 16), while tight forwards performed a significantly greater number than outside backs (~16). Furthermore on average loose forwards performed the greatest amount of RHIE bouts (~13) which was significantly greater than half backs (~5) and outside backs (~6). Previous TMA studies have included measures of static exertion (tackling, rucking, scrummaging etc.) as high-intensity activity and shown that despite performing less high-intensity running, forwards spent the greatest time in high-intensity activity due to their greater demands in static exertion (Austin et al., 2011b; Roberts et al., 2008). For example, Roberts et al. (2008) found forwards performed a significantly greater number of static exertion activities (~89 vs. \sim 24) and spent greater time performing high intensity activities (running and static exertion) than backs (~9:09 vs. ~3:04min). Coupled with locomotive demands, this may explain why although non-significant, loose forwards had greater player load values than half, inside and outside backs. Loose forwards did however have significantly greater player load values than tight forwards. Despite performing a similar number of total contacts to loose forwards, an important role of tight forwards is to scrummage where prolonged static exertion may not be detected by accelerometers. For example tight forwards exert and are subjected to greater forces during scrummaging (Quarrie et al., 2000) which may result in 'temporary fatigue'; whereby there is a reduction in high-intensity activity performed immediately following an intense bout, with a subsequent recovery later in performance (Mohr et al., 2003). This may be characterised by reduced locomotive patterns compared to loose forwards following a scrum. Thus, despite advances in inertial sensor technology which may help to characterise the movement patterns of rugby players, in particular loose forwards, further research is required to quantify the physiological demand of performance.

The present study has also assessed temporal changes in movement patterns from data files of players who completed full games. Similar to the findings of Austin and Kelly (2013) the present study found transient fatigue throughout each half in multiple measures of low and high intensity movement and low to high acceleration and deceleration movements. Furthermore, although the number of RHIE bouts and contacts did not significantly change during any 10min period during the first half, both measures were significantly reduced at 50-60, 60-70 and 70-80min compared to 40-50min. The study also shows that player load.min⁻¹, cruising.min⁻¹ and striding.min⁻¹ values were significantly reduced from the first to second half.

Despite high-speed meterage exhibiting the greatest percentage reduction from 0-10min between 30-40min, the greatest reductions in movement in the second half compared to the first 10min were found in low speed movements such as cruising and striding supporting previous research in rugby union by Roberts et al. (2008). Previous TMA research by Roberts et al. (2008) found no difference between halves for total distance covered (~3020 vs. 2987m), distance covered in high-intensity running and sprinting (~223 vs. 208m) and time spent in high intensity activity (~3:11 vs. 2:57min). However, further analysis of the distances travelled over successive 10min periods of match-play revealed that greater total distance was covered in the first 10min compared with the periods of 50-60 and 70-80min (Roberts et al., 2008). However, Roberts et al. (2008) found no differences between 10min time periods for distances travelled in high-intensity running, sprinting or 'running work', and there were no differences between the total, average or maximum time spent in highintensity activities or in static exertion over the 10min periods, suggesting that changes in distance covered may have been characterised by a reduction in low intensity activities, which may be characterised by an inability to maintain defensive position or run supporting lines in attack (Roberts et al., 2008).

The present study also demonstrates an increase in high-intensity, sprinting and high-speed meterage during the final 10min of the match to values non-significantly different to any other 10min period. Mooney et al. (2013) suggest that reductions in low speed compared to high speed movements may be evidence of 'pacing' whereby players sacrifice distances covered at low speeds to compensate for the demands of high speed movement. Coaches and sport scientists should therefore be aware of transient fatigue in rugby, with further research required to investigate differences between positions (Austin and Kelly, 2013) and temporary fatigue characteristics (Mohr et al., 2003). Following recent research (Cormack et al., 2013; Mooney et al., 2013), the effect of preparedness and fatigue on work rate and transient fatigue should also be investigated in rugby union. Furthermore, the effect of substitutes on team movement patterns requires investigation.

The data presented from the present study, together with anthropometric and physiological characteristics of players, may enhance knowledge of the positional requirements of performance. This in turn may help monitor preparedness and performance (Austin et al., 2011a), inform the planning of position specific programmes that elicit physiological adaptations (Austin et al., 2011a) and facilitate rehabilitation (Coughlin et al., 2011). Austin et al. (2011a) explain that by using maximum periods of activity coupled with minimum periods of recovery; the most demanding passages of play may be replicated providing coaches with a sense of their players' preparedness to meet the requirements of competition.

For example, using the present data, a protocol for an inside back which would represent the mean average RHIE characteristics of match-play would require players to perform four high intensity efforts interspersed by 4.9s with 12:31min between bouts. However, it is proposed that coaches should use minimum, mean, maximum and standard deviation values reported in the study as a reference to modulate RHIE protocols which may allow progressions in intensity during training and rehabilitation. Varying the high intensity activity periods may allow coaches to work on and integrate various high intensity activities patterns. Therefore activity periods may be shorter or may consist of multiple activities. To assist in the identification of activity patterns during RHIE bouts, synchronisation of GPS software with motion analysis software coaches may allow coaches to identify the characteristics of RHIE bouts which may allow great specificity and progression of protocols.

Consideration should also be given to the duration of 'recovery' between RHIE bouts. Using average and standard deviation values, recovery times may be adjusted to increase intensity of effort. Furthermore, temporal analyses from the study highlight the most intense periods of performance and the expected reductions in several measures of movement for all players. Knowledge of 'worst-case' meterage values may be used to modulate and manage intensity of effort, and may be a valuable monitoring tool when quantifying preparedness which would be an important process in assessing the progress of an injured player (Coughlan et al., 2011). Temporal analysis from the study also highlights the potential importance GPS monitoring may have during match-play by allowing sport scientists to observe individual or team movement patterns and potentially advise on tactical decisions (e.g. substitutions). Furthermore, knowledge of variations in movement patterns may aid the application of match-day strategies to enhance performance (e.g. half-time re-warm up; Mohr et al., 2004).

5.5. Practical applications

The findings of the present study may be used to facilitate the planning and implementation of training programmes that elicit appropriate and specific physiological adaptations. Knowledge of team, position and individual temporal movement patterns may also aid the preparation of conditioning and rehabilitation drills. Monitoring of temporal patterns, in particular low speed movements and player load may allow coaches and sport scientist to assess preparedness for performance and fatigue during match-play. Knowledge of transient fatigue may also have important implications for tactical decisions made during match-play and the use of match-day strategies to enhance performance.

This study has characterised the movement patterns of professional rugby players, demonstrating variances in positional demands which may be used to help understand the physiological demands of individuals. This in turn may be used to devise positional specific drills to enhance physiological preparation for performance and may be used for the monitoring and management of individuals. We also show evidence of transient fatigue in rugby union which may also have important implications for physiological preparation, assessment and monitoring of performance.

EXPERIMENTAL STUDY 2

6. Biochemical responses to physical contacts and high-speed running in professional rugby union players

6.1. Introduction

Rugby union is a physically demanding game characterised by repeated high-intensity bouts of relatively short duration exercise, with varying recovery periods (Cahill et al., 2013; Roberts et al., 2008). The high-intensity bout characteristics are largely positional dependent (Cahill et al., 2013); however, all players are exposed to a high magnitude of physical contacts and collisions (Cunniffe et al., 2010; Quarrie et al., 2013), and high intensity stretch-shortening cycle (SSC) movements. Research has demonstrated large elevations in blood markers of muscle damage (e.g. creatine kinase) (Cunniffe et al., 2010; Smart et al., 2008; Takarada, 2003), and disruptions to neuromuscular (West et al., 2014), hormonal (Cunniffe et al., 2010; Elloumi et al., 2003; West et al., 2014), immune functions (Cunniffe et al., 2010) and mood (West et al., 2014) for several days following competition and as a consequence, players may take several days to fully recover following competition (West et al., 2014).

Insufficient recovery following competition may compromise players' ability to train, and with the accumulative stress of subsequent training, may compromise players' preparation for subsequent games (McLean et al., 2010). Furthermore, acute and chronic insufficient recovery may predispose an individual to a greater risk of injury (Lazarim et al., 2009) and the development of overtraining syndrome (Kellmann, 2010) respectively, therefore insufficient recovery may have great implications for player preparation and performance.

Previous research has also highlighted the individual nature of recovery in professional rugby union (West et al., 2014). For example, West et al. (2014) found countermovement peak power output of 50% of players had not returned to pre-match values 60h post-match. Knowledge of individual recovery patterns could therefore be used to benefit player management through individual modification of subsequent training and recovery strategies (West et al., 2014). However, assessing recovery following each game is often associated with invasive collection procedures and analysis which may take several hours or days to conduct (e.g. hormone analysis from saliva sample, (Thorpe and Sunderland, 2012); muscle damage from blood sample; (Cunniffe et al., 2010)); thus information attained may not be used for individual recovery and subsequent weekly training modulation. It has been suggested that match play performance characteristics could be used to prospectively modulate individual recovery in rugby union (Cunniffe et al., 2010; Takarada 2003) and other team sports (McLellan et al., 2011a; McLellan et al., 2012; Thorpe and Sunderland,

2012). Physical contacts have been demonstrated to correlate with post-match creatine kinase (CK; an indirect blood marker of muscle damage) responses in rugby union (Cunniffe et al., 2010; Smart et al., 2008; Takarada, 2003) and neuromuscular recovery in rugby league (McLellan et al., 2012), suggesting recovery may be determined by the extent of mechanical damage induced through contact during performance in rugby codes.

With the metabolic, mechanical and neural elements associated with fatigue following SSC activity (Nicol and Komi, 2003), it is also likely that muscle damage and neuromuscular fatigue following rugby union may partly be determined by the extent of high-intensity movement characteristics. Indeed, through advances in match analysis technologies, global positioning system (GPS) tracking has shown relationships between high-speed running characteristics and changes in CK (Thorpe and Sunderland, 2013; Young et al., 2012) and neuromuscular function (Duffield et al., 2012) during the recovery period in team sports, such as soccer (Thorpe and Sunderland, 2012) and Australian rules football (Young et al., 2012), however at present this analysis has not been carried out in professional rugby union.

Therefore, the aim of this study was to examine whether performance characteristics associated with high-speed movements and physical contacts during match play are correlated to post-match changes in creatine kinase; a surrogate marker of muscle damage in professional rugby union players.

Performance characteristics and the subsequent recovery process were assessed following four group stage matches of the 2012-2013 European Cup, the elite competition in Northern Hemisphere club rugby. Players were required to report to testing 2h prior to kick off and approximately 16 and 40h following its completion. On each testing occasion a finger-prick blood sample was collected for the assessment of creatine kinase (CK); a blood marker of muscle damage. Players were separated into forwards and backs groups to assess the correlation between pre to post-match changes in CK and performance characteristics derived by use of global positioning system (GPS) and notational analysis.

Thirty-six male rugby union players volunteered for the study. Participant data files were only included in the analysis if they performed a minimum of 60min of match play (McLellan et al., 2011a; McLellan et al., 2012). Subsequently, only data from 28 players (age 25.1 ± 3.1 yrs) were analysed (15 forwards, 26.7 ± 2.8 yrs, 111.6 ± 5.7 kg; 13 backs, 23.4 ± 2.6 yrs, 94.2 ± 7.9 kg). Prior to providing informed consent, participants were given information outlining the rationale, potential applications of the study and procedures (Appendix B). Ethical approval was given by the Swansea University Ethics Committee (Appendix A).

Upon arrival on testing days, players placed their non-dominant hand in a basin of warm water (40-42°C) prior to capillary blood collection. Following 2min, a pinprick on the finger of the warmed hand was made using a spring-loaded single use disposable lancet (Accu-Chek Safe-T-Pro Plus; Roche, Basle, Switzerland) which allowed approximately 300μ l of capillary whole blood to be collected (Microcuvette® CB300, Sarstedt, Numbrecht, Germany). Samples were then put on ice until being centrifuged for 10min at 3000rpm (Heraeus Sepatech Labofuge 200; Kendro Laboratory Products, Germany), from which plasma was pipetted into an eppendorf tube and stored at -70°C until analysis. This process was repeated 16 and 40h post-match. Plasma was analysed for CK using an automated clinical chemistry analyser (Cobas Miras; ABX diagnostics, Beds., UK). The intra-assay coefficient of variance (CV) was $0.93 \pm 0.00\%$.

Global positioning system units (10Hz; MinimaxX v.4.0, Catapult Innovations, Melbourne, Australia) were fitted into the back of a custom-made vest so that the unit was positioned in the centre area of the upper back and slightly superior to the shoulder blades, with no restriction to the range of movement of the upper limbs and torso. GPS data was downloaded and analysed using Catapult Sprint software (Catapult Innovations, Melbourne, Australia) for analysis of distances covered. Distances covered at low and high-speed movements were analysed according to velocity thresholds previously reported (Gabbett et al., 2011). Highspeed measures analysed were high-speed running distance (>5m.s⁻¹), sprint distance (>5.6m.s⁻¹) and number. The units were also used to provide total contacts from performance. Tackle detection; a feature of the MinimaxX unit (Catapult Innovations, Melbourne, Australia) which incorporates a tri-axial piezoelectric linear accelerometer system, gyroscopes and magnetometers imbedded within the unit has been shown to provide a valid quantification of the contact load in rugby league (Gabbett et al., 2010).

Analyses of physical contacts were also provided by the teams' performance analysis department. Using Sportscode (Sportstec, NSW), the number of tackles, hit ups (Smart et al., 2008) and the number of times the player made contact at the breakdown area were noted, providing a measure of total impacts. Furthermore the number of scrums engaged by forwards were also analysed.

Following each game, players performed the same standardised recovery process. Immediately post-game, contrast water therapy was administered (2min warm (40-42°C), 2min cold (10-15°C) x3), with players finishing in cold water. Following next day testing (approx. 16h post-match), players then undertook an active recovery session which involved 3 sets of 4min bike or cross-trainer at $65 \pm 5\%$ age predicted max heart rate, followed by 4min light movement on the same equipment.

Analysis of movement and contact characteristics and their correlations with changes in CK were conducted within the forwards (props, hooker, second rows, flankers and number 8) and backs (scrum half, outside half, centres, wingers and full back) positional groups due to movement and contact variances between groups (Quarrie et al., 2013). To identify and remove outliers from the data, Z-scores for CK at each time points were analysed. The criterion for an outlier at a time point was defined as any value greater than two standard deviations from the position mean (Field, 2005).

Global positioning system data analysed using Catapult 'Sprint' software (Catapult Innovations, Melbourne, Australia) were exported to CSV files for further analysis in Microsoft Excel (Microsoft Office Excel 2010, Microsoft Corporation, Berkshire, UK). A mixed model ANOVA was used to assess changes in CK concentrations from pre-game to post-game and assess movement and contact statistics within each positional group. Significant differences in CK concentrations were located by a Bonferroni corrected pairwise comparisons. The criterion level for significance was set at p<0.05. Correlations between CK and performance characteristics within each group were analysed using the Pearson Product-Moment Correlation Coefficient. Magnitudes of effect of the correlations were determined as follows: trivial <0.10; small \leq 0.10-0.29; moderate 0.30-0.49; large 0.50-0.69; very large 0.70-0.89; and nearly perfect 0.90-0.99 (Hopkins et al., 2009). All statistical analyses were performed using the Statistical Package for the Social Sciences (SPSS for Windows, version 14.0; SPSS, Inc., Chicago, IL, USA).

6.3. Results

In total 57 data files (45 including GPS) were collected with each forward and back playing an average of 2.0 ± 0.6 and 2.1 ± 1.0 games each. When separated into position groups, 27 backs (22 including GPS) and 30 forwards (23 including GPS) data files were used for analysis.

Table 6.1 shows positional differences between forwards and backs. Results demonstrate that contact demands were significantly greater for forwards, while backs; who played significantly longer than forwards (p<0.05), covered greater total distances (p<0.01), covered greater distance per minute (m.min⁻¹; p<0.01), performed a greater number of sprints (p<0.001), and covered greater distances sprinting and at high-speed (p<0.001; Table 6.1).

There was a significant time effect (p<0.05) and a significant effect of player positional group (p<0.05) in the plasma CK responses. Baseline Plasma CK concentrations were significantly different between forwards and backs (274 ± 155 and 368 ± 127 IU.L⁻¹; p<0.05). CK concentrations were significantly increased at all post-match time points (p<0.05), with changes in CK greater in backs, when compared to forwards at 16h (forwards Δ 705 ± 483 vs. backs Δ 1237 ± 871 IU.L⁻¹; p<0.01) but not at 40h post-match (forwards Δ 383 ± 329 vs. backs Δ 540 ± 412 IU.L⁻¹, p=0.12).

Correlations between changes in CK and performance analysis and GPS measures from match play are displayed in Tables 6.2 and 6.3, respectively.

Analysis of forwards show significant moderate correlations between various contact measures derived from performance analysis and both ΔCK and % change in CK (Δ % CK) at 16 and 40h post-match (Table 6.2, p<0.05).

Significant moderate to large correlations were observed for the backs between contact measures derived from performance analysis and both Δ CK and Δ % CK at 16 and 40h post-match (p<0.05). Furthermore, non-significant, moderate correlations were observed between GPS measures associated with high-speed movement and both Δ CK and Δ % CK at 16 and 40h post-match (Table 6.3).

No significant correlations were found between total contacts detected by micro-sensors within the GPS units and changes in CK, however a moderate effect size correlation was observed with $\Delta\%$ CK at 40h post-match for the backs (Table 6.3).

	Forwards	Backs
Performance analysis	n=30	n=27
Game time (min)	80 ± 13	87 ± 11^{a}
Tackles	5 ± 3	4 ± 3
Hit-ups	5 ± 2	5 ± 3
Contacts hit	$15 \pm 6^{\circ}$	6 ± 4
Total impacts	25 ± 9^{c}	15 ± 7
Scrum number	13 ± 5^{c}	0 ± 0
GPS	n=23	n=22
Distance (m)	4906 ± 902	5959 ± 1013 ^b
m.min ⁻¹	60.4 ± 7.8	67.8 ± 8.2^{b}
Sprint number	7 ± 6	18 ± 6^{c}
Sprinting (m)	121 ± 112	$333 \pm 122^{\circ}$
High-speed running (m)	231 ± 167	$509 \pm 150^{\circ}$
Total contacts	31 ± 14^{c}	16 ± 7

Table 6.1. Summary of performance markers derived from global positioning system (GPS) and performance analysis data. Data presented as mean ±SD.

Value significantly greater than other positional group ^a<0.05 ^b<0.01 ^c<0.001

		Forwards	s (n=30)			Backs ((n=27)	
	ΔCI	К	Δ%	cK	ΔC	×	₩7	сK
	+16hrs	+40hrs	+16hrs	+40hrs	+16hrs	+40hrs	+16hrs	+40hrs
Tackles			0.331 Moderate	0.305 Moderate		0.579** Large		0.386 Moderate
Hit-ups	0.448* Moderate	0.406* Moderate	0.494** Moderate	0.387* Moderate	0.407* Moderate		0.309 Moderate	
Contacts hit					0.395 Moderate	0.518** Large	0.463 * Moderate	0.444* Moderate
Total impacts	0.330 Moderate		0.438* Moderate	0.343 Moderate	0.404 Moderate	0.638** Large	0.388 Moderate	0.454* Moderate
Scrum number								

Table 6.2. Correlation between changes in creatine kinase (CK) and performance analysis characteristics.

Level of significance *p<0.05; **p<0.01 Effect sizes 0.30-0.49 moderate, 0.50-0.69 large, 0.70-0.89 very large, 0.90-0.99 nearly perfect (trivial to small not displayed)

Table 6.3. Correlation between changes in creatine kinase (CK) and movement and performance markers derived from global positioning system (GPS) data.

$\begin{array}{c c c c c c c c c c c c c c c c c c c $			Forwards	: (n=23)			Backs	(n=22)	
$ \begin{array}{c c c c c c c c c c c c c c c c c c c $		ΔC	K	4%	CK	ΦC	Ķ	∇ %	CK
Sprint number0.3390.4190.3640.32Sprint numberModerateModerateModerateModerateSprinting (m)0.4090.4200.4470.359Sprinting (m)0.4090.4200.4470.359Sprinting (m)0.4090.4240.3460.437Sprinting (m)0.4340.3460.4370.434Sprinting (m)0.4340.3460.4370.434Sprinting (m)0.4340.3460.4370.434Sprinting (m)0.4340.3460.4370.434Sprinting (m)0.4340.4340.3460.437Sprinting (m)0.4340.4340.4340.436Sprinting (m)0.4340.4340.4360.437Sprinting (m)0.4340.4340.4360.436Sprinting (m)0.4340.4340.4360.436Sprinting (m)0.4340.4340.4360.436Sprinting (m)0.4340.4340.4460.436Sprinting (m)0.4340.4460.4460.436Sprinting (m)0.4460.4460.4460.447Sprinting (m)0.4460.4460.4460.447Sprinting (m)0.4460.4460.4460.446Sprinting (m)0.4460.4460.4460.446Sprinting (m)0.4460.4460.4460.446Sprinting (m)0.4460.4460.4460.446Sprinting (m)		+16hrs	+40hrs	+16hrs	+40hrs	+16hrs	+40hrs	+16hrs	+40hrs
Sprinting (m)0.4090.4200.4470.359ModerateModerateModerateModerateModerateIgh-speed (m)0.4340.3460.437Total contactsModerateModerateModerateTotal contactsModerateModerate0.363	Sprint number					0.339 Moderate	0.419 Moderate	0.364 Moderate	0.332 Moderate
ligh-speed (m) 0.434 0.346 0.437 Moderate Moderate Moderate Moderate 0.363 0.363	Sprinting (m)					0.409 Moderate	0.420 Moderate	0.447 Moderate	0.359 Moderate
0.363 Moderate	ligh-speed (m)						0.434 Moderate	0.346 Moderate	0.437 Moderate
	Total contacts								0.363 Moderate

Level of significance *p<0.05 Effect sizes 0.30-0.49 moderate, 0.50-0.69 large, 0.70-0.89 very large, 0.90-0.99 nearly perfect (trivial to small not displayed)

6.4. Discussion

The aim of this study was to investigate the relationships between performance markers associated with physical contacts and high-speed movement, and post-match changes in creatine kinase (CK) in professional rugby union players.

Here we demonstrate that muscle damage, as indicated by CK concentrations, is correlated with the number of physical contacts performed during rugby union match play. Furthermore, we show that the muscle damage experienced by backs is associated with highspeed running derived from GPS.

In accordance with previous research (Cunniffe et al., 2009), peak increases in CK levels were observed at 16h post-match (~417%), with values remaining significantly elevated from pre-game values at 40h post-match (267%). Furthermore, we demonstrated significant relationships between various contact measures derived from performance analysis (e.g. tackles made, hit-ups) and changes in CK concentrations at both 16 and 40h post-match, which is consistent with previous research demonstrating that increases in muscle damage from rugby union match play are largely determined by the extent of mechanical damage induced through physical contact (Cunniffe et al., 2010; Smart et al., 2008; Takarada, 2003), with body to body contacts, collisions and tackles being significantly related to changes in CK concentrations (Cunniffe et al., 2010). These findings may be explained by the blunt force trauma from physical contacts which disrupt skeletal tissue structure integrity; subsequently increasing cell permeability and the diffusion of soluble enzymes such as CK into the interstitial fluid (Cheung et al., 2003).

The present study is the first to demonstrate that muscle damage induced by match play is to some extent determined by high-speed movement characteristics in rugby union backs. Results from this study demonstrate moderate effect size correlations between sprint number, sprint distance, and high-speed running distance with Δ CK and Δ % CK at 16 and 40h postmatch for backs. These correlations may be explained by high-force, eccentric work when performing SSC activities such as high-speed running, which may exceed the muscles ability to actively resist load, forcing the muscle to lengthen and generate greater active tension (Stauber, 2004). Therefore, in addition to monitoring the extent of contact performed, monitoring the extent of high-speed running conducted may be important in attempting to assess the physiological impact of match-play in back players.

The relationships between movement characteristics and changes in CK concentrations found in the present study support previous research which show correlations between measures related to high intensity running distance (>4.167m.s⁻¹) and an increase in CK levels in semi-professional football (Thorpe and Sunderland, 2012), however, we are the first to demonstrate this in professional rugby union. Thorpe and Sunderland (2012) found that the change in Δ CK was related to the total number of sprints performed (>5 m.s⁻¹; r=0.80) and sprint distance (r=0.78), while percentage change in $\%\Delta$ CK correlated to sprint number (r=0.86), sprint distance (r=0.89) and high intensity distance covered (r=0.92). Furthermore in Australian rules football, reductions in neuromuscular function and changes in CK postmatch have been demonstrated to have stronger correlations with measures associated with high-speed running than total running distance (Young et al., 2012), suggesting that muscle damage and neuromuscular recovery following competition in rugby union is more likely determined by the extent of high-intensity SSC activity performed than total distance covered. Although we did not measure neuromuscular function this should not detract from the application of our data, as previous research has demonstrated strong correlations between functional recovery and blood markers of muscle damage (Avela et al., 1999; Horita, 2000).

Although correlation is by no means causation, a potential explanation for the relationships between high-speed movement and changes in CK only being observed in backs is likely due to their greater high-speed running demands when compared to the forwards as demonstrated in study one. Additionally, it may also be due to the velocity thresholds used in the present study. Dwyer and Gabbett (2012) identify that many sprints in team sport involve maximal efforts of a short duration (~1-2s) which do not allow the attainment of high-speed and are thus not reported as high-speed or sprinting efforts. Previous research has demonstrated that changes in CK concentration following Australian rules football were correlated to high acceleration metres covered (Young et al., 2012), thus correlations for forwards and backs may have been stronger in the present study if high-acceleration movements were included in analysis in addition to high-speed movement.

Previous research has demonstrated that forwards spend a greater duration in static exertion (Roberts et al., 2008) and are involved in a greater number of contacts (study one) than backs. However, with only moderate correlations found between contact statistics and muscle damage in the present study for forwards, quantifying the extent of contact as opposed to the number of contacts may be more important in determining the extent of work done. For example, in the present study small effect size correlations were found between scrum number and changes in CK. Smart et al. (2008) describe that at engagement, the large impact created from the momentum of both packs is likely to create substantial physical trauma and impact in the front row, however the impact and trauma experienced by back row

forwards; who play a 'supporting role', is likely to be less substantial. Therefore although contact statistics may give an indication of the extent of muscle damage experienced by players, it does not account for the extent of contact or exertion. Furthermore, in the present study, total contacts detected within the GPS units only showed a moderate correlation with $\%\Delta CK$ within the backs at 40h post-match. Thus, despite advances in GPS technology in quantifying physical contacts, further research is required.

We were limited in our ability to perform additional measures alongside the measurements of CK, such as neuromuscular function and hormonal responses due to limited time and facilities available for testing prior to each match. Indeed, it would be important to examine the extent performance characteristics correlate to changes in these post-match measures as there is potential for neuromuscular function reduction to remain, despite normalised CK (West et al., 2013), and steroid hormone profiles (West et al., 2014). Another potential limitation of the study was that arterialised blood was used to analyse CK levels. Previous studies in rugby union have obtained blood samples by venepuncture (Cunniffe et al., 2010) or cannulation (Takarada, 2003) which are considered the gold standard approach for indirectly determining skeletal muscle damage (Nunes et al., 2006). However similar to previous research in team sports (McLellan et al., 2011a; Thorpe and Sunderland, 2012) a capillary blood sample was collected in the present study via a fingertip puncture, which provides a simpler, less-invasive and safer means of blood collection (Knoblauch et al, 2010). Capillary blood sampling may be more convenient within a team-sport setting, especially prior to competition; however this method also requires arterialisation via hot water to enable a free flowing sample, which may influence collected measures (Maughan et al., 2006). Where sampling occurs over time, therefore, and where the degree of arterialisation will influence measures made, this may cause major problems (Maughan et al., 2006). However, research has shown strong correlations between capillary and venous samples when monitoring CK levels post eccentric exercise, suggesting capillary blood is a valid alternative to venous blood when evaluating changes in CK post-exercise (Knoblauch et al., 2010).

Despite these potential limitations, the results of this study add to the literature and our understanding of post-match changes in muscle following rugby union match play; we believe our data have implications for player management post-match. As an alternative to assessing individual recovery following each game, research suggests that certain performance characteristics could be used to prospectively predict individual recovery in rugby union (Cunniffe et al., 2010; Smart et al., 2008; Takarada, 2003) and other team sports (McLellan et al., 2011a; McLellan et al., 2012; Thorpe and Sunderland, 2012). From the

present study it has been demonstrated that muscle damage, as indicated by CK concentrations, following professional rugby union is to some extent dependent on the number of physical contacts for all players, and the extent of high-speed running for backs. Therefore, despite being involved in the same team, the extent of muscle damage and subsequent recovery for one player may vary greatly from another player based on their performance characteristics. Indeed, as found by West et al. (2014), the present study demonstrates a highly individual nature of recovery; with a large range in Δ CK at 40h postmatch (30 to 1849 IU.L⁻¹). Thus it is proposed knowledge of individual recovery could be used to benefit player management through individual modification of training and recovery strategies. Future research to establish which recovery modalities may be more appropriate for each mechanism of muscle damage may further enhance provision post-match.

Despite the exciting implications of the study findings, several considerations should be made. Firstly, correlations were found for forwards and backs groups and measures of performance however the strength of correlations and relative importance of different characteristics may vary within these groups. For example, study one found that outside backs covered significantly greater high-speed running distances than half backs during match-play, therefore high-speed running may be a greater determinant of muscle damage for outside backs compared to half-backs. Similar to previous research, future research to establish multiple regression equations which account for both contact and high-speed movements to predict recovery may be warranted (Smart et al., 2008).

When determining the physiological cost of match-play from performance markers, practitioners should also be aware of the influence physical preparedness may have on muscle damage and fatigue. Research has shown that runners who had the greatest reductions in running pace during a marathon had significantly elevated post-race myoglobin, lactate dehydrogenase and creatine kinase levels in comparison with marathon runners that preserved their running pace reasonably well throughout the race (Del Coso et al., 2013). Additionally, recent research demonstrates that post-match fatigue in rugby league is lower in players with well-developed high-intensity running ability, and lower body strength, despite exhibiting greater internal and external match loads (Johnston et al., 2014a). Thus, for two players who perform an equal volume of work in rugby union, the player with the greater levels of physical preparedness may not display elevations in muscle damage or fatigue as great as one less prepared. Furthermore, Mooney et al. (2013) showed that neuromuscular fatigue resulted in reductions in exercise intensity. Thus either neuromuscular fatigue prior to or during match-play may increase susceptibility to muscle damage.

6.5. Practical applications

This study is the first to assess the relationship between the biochemical responses to highspeed running and physical contacts using GPS technologies and performance analysis methods. The findings of the study suggest that the factors associated with muscle damage induced from match play are positional dependent. As demonstrated by previous performance analysis studies, the extent of muscle damage post-match is to some extent dependent on the total number of physical contacts induced during performance for all positions. Furthermore, the study is the first to show that for back players, muscle damage may also be due to the extent of high-speed running performed.

With previous studies demonstrating an individual nature of recovery, the findings of the study suggest that GPS and performance analysis may be used by coaches and practitioners to prospectively tailor individual recovery strategies and subsequent training following match play. Thus GPS and performance analysis may have great implications for player management in rugby union.

EXPERIMENTAL STUDY 3

7. Does cold water immersion impair adaptation to strength training in professional rugby union players?

7.1. Introduction

Rugby union is a physically demanding contact sport characterised by repeated highintensity bouts of short duration exercise, with varying and often incomplete recovery periods (Cahill et al., 2013; Coughlan et al., 2011; Cunniffe et al., 2010; Roberts et al., 2008). As a consequence of competition, players may take several days to fully recover (e.g. West et al., 2014). For example, research demonstrates large elevations in blood markers of muscle damage (e.g. creatine kinase) (Cunniffe et al., 2010; Smart et al., 2008; Takarada et al., 2003), and disruptions to neuromuscular (West et al., 2014), hormonal (Cunniffe et al., 2010; Elloumi et al., 2003; West et al., 2014), immune functions (Cunniffe et al., 2010) and mood (West et al., 2014) for several days following competition.

With the accumulation of subsequent training and performance, insufficient recovery prior to the start of the training week may compromise players' ability to train, and with the added stress from training, players' preparation for subsequent games may be compromised (McLean et al., 2010). Insufficient recovery may also predispose an individual to a greater risk of injury (Lazarim et al., 2009) and the development of overtraining syndrome (McLellan et al., 2012). Therefore, in an attempt to accelerate recovery following training and competition, it is common practise to employ one or more post-exercise recovery strategies, such as cold water immersion (CWI); with these being reported to reduce the extent of muscle damage, inflammation and contractile function impairment following exercise (Wilcock et al., 2006). Indeed a recent meta-analysis found that CWI is an effective strategy to enhance recovery of muscle power post-exercise (Leeder et al., 2012a). Furthermore, research demonstrates that CWI (e.g. 2x5min at 10°C; Ingram et al., 2008) is an effective strategy in reducing blood markers of muscle damage (e.g. Vaile et al., 2008) and delayed onset of muscle soreness (DOMS) post-exercise (e.g. Vaile et al., 2008), and may enhance recovery of strength (e.g. Bailey et al., 2007), speed (e.g. Ingram et al., 2008), and parasympathetic nervous system activity (e.g. Buchheit et al., 2009). Therefore, CWI appears to be an integral component of recovery and preparation during periods of competition.

In addition to teams using CWI to speed up recovery from matches, CWI may be used posttraining during periods of physical development such as pre-season in attempt to optimise training adaptation; by potentially allowing greater training loads (increased frequency, intensity or duration) to be performed in subsequent sessions, thus increasing the stimulus for adaptation (Versey et al., 2013). However, despite enhancing physiological recovery for subsequent sessions, the use of CWI to enhance adaptation may be counterintuitive. For example, it has been suggested that muscle damage and the subsequent inflammatory response is a vital precursor for the signalling mechanisms which initiate the repair and growth of cells (Carlson and Faulkner, 1983), therefore incorporating strategies which attempt to blunt the effects of exercise induced muscle damage may in fact reduce the potential for training adaptation to occur. Indeed research has demonstrated that several strategies which aim to reduce the extent of the inflammatory process; CWI (Frohlich et al., 2014; Yamane et al., 2006), local cold pack application (Nemet et al., 2009) and antioxidant supplementation (Fischer et al., 2004; Gomez-Cabrera et al, 2008; Strobel et al., 2010), may inhibit the stimulus for adaptive physiology. Therefore, during periods where physical development is of prime importance such as pre-season, the use of recovery strategies such as CWI may in fact impede adaptation.

Currently there is lack of literature examining the effect of CWI on adaptation in elite athletes; therefore best practice recovery protocols during periods of adaptation are unclear. In attempt to understand the role CWI has on adaptive physiology in elite athletes, the aim of this study was to assess the effect of CWI on adaptation to strength training during preseason in rugby union.

7.2. Methods

Subjects were members of a senior professional rugby union team, with the study conducted during pre-season prior to the 2013-2014 season. Players with existing injuries or modified training patterns were excluded from the study. Consequently, 22 male professional rugby union players volunteered for the study. All players had been engaged in a structured weight-training program for at least 2 years and were able to complete an isometric mid-thigh pull (IMTP) with correct technique as assessed by a qualified strength and conditioning coach. Prior to providing informed consent, participants were given information outlining the rationale, potential applications of the study and procedures (Appendix B). Ethical approval was given by the Swansea University Ethics Committee (Appendix A).

lday	ĮĮ,	
Sur	0	
Saturday	Off or extra conditioning	
Friday	Upper body strength (60min)	Conditioning & rugby (60-75min)
Thursday	Skills/speed mechanics (45- 60min)	Lower body strength (60min)
Wednesday	Off or extra conditioning	
Tuesday	Upper body strength (60min)	Conditioning & rugby (60-75min)
Monday	Skills/speed mechanics (45- 60min)	Lower body strength (60min)
	AM	Md

Table 7.1. Weekly training schedule during five week pre-season period.

A randomised control trial research design was used to investigate the effect of CWI on adaptation to strength training during a five week training block (Table 7.1). To determine baseline maximal strength, IMTP testing was conducted on the first day of pre-season for the measurement of peak force (PF) and relative peak force (Rel. PF). The following day players then completed bench press and chin-up 3RM tests as part of their upper body strength session. Body composition tests were also conducted prior to baseline testing by an International Society for the Advancement of Kinanthropometry (ISAK) accredited technician. Following testing, players were pair-matched into two post-training recovery groups; CWI (n=11, 24 \pm 3yrs) or a control group (CON; n=11, 24 \pm 4yrs), with consideration to strength results, playing position, training experience and key lift selection. Recovery sessions were implemented immediately following the final session of each day (within 10 and 20min of lower body strength training and conditioning/rugby sessions respectively) during the five week period. To establish the efficacy of strength training on adaptation, the IMTP, bench press and chin-up tests were repeated one week following the completion of the five week training period. All testing, training and recovery sessions were conducted and monitored at the team's training facilities. Prior to the five week training period, players were required to complete two weeks of prescribed self-directed preparatory training which was designed to re-introduce the exercises used in pre-season in attempt to reduce muscle soreness and injury. Pre-season training followed a weekly pattern as shown in Table 7.1; however IMTP strength testing preceded speed and skills training on the Monday of the first week. The number of training sessions completed by players is shown in Table 7.2. Key lifts for lower (squat or trap bar) and upper (bench press and chin-up) strength training during the five weeks were prescribed by the team's strength and conditioning coach (4-5 sets x 3-6 reps; 80% 1RM).

Players were not scheduled to train on Wednesday and Saturday, however extra conditioning was prescribed for all players on Saturday during weeks 2 and 5. Furthermore, certain players were required to perform extra conditioning sessions due to body composition issues. Players were provided with pre and post-training nutrition throughout the training period. Hydration status was assessed prior to the first session of the day on Monday and Thursdays to provide feedback to players on hydration status (Osmocheck; Cranlea. Birmingham, UK).

	Lower body strength	Upper body strength	Conditioning	Skills/speed mechanics	Extra conditioning
Number of sessions	6	10	10	10	2
CWI	9 ± 1	10 ± 1	9 ± 1	10 ± 1	4 ± 2
CON	8 ± 1	9±1	9 ± 1	9 ± 1	4 ±2

Table 7.2. Total number of training sessions and number of sessions performed by players within each group (expressed as mean \pm SD).

Following the final training session of the day players assigned to the CWI group were immersed in cold water to the level of the anterior superior iliac spine at 10-15°C (Comark PDT300; Comark Instruments, Norwich, UK) in a portable ice bath (White Gold Fitness, Bedford, UK). Immersion protocols were progressed to facilitate tolerance to CWI based on previous experiences with the subject group. During weeks 1-3 players were immersed for 2x5min (Ingram et al., 2008), with 2min between immersions standing out of the bath. For the final two weeks of the training block players were immersed for 1x10min (Bailey et al., 2007). Players were permitted a maximum of 2min to shower clean prior to CWI however they were not permitted to return to the shower between or following immersions as this may have been counterproductive to the vasoconstrictive effects of CWI (Cochrane, 2004). Players were requested to attend for CWI immediately following their final session of the day (with exception to any extra conditioning sessions on Wednesdays or Saturdays) however they were not restricted to nutritional supplements immediately post-exercise which were prescribed during this period. Players assigned to the CON group received no post-exercise recovery intervention during the training period.

The IMTP testing was carried out with players standing on a portable force platform (type 92866AA, Kistler Instruments Ltd., Farnborough, United Kingdom), which was positioned on the floor centred underneath the bar of a power rack.

Prior to pre-training IMTP testing, players were positioned so that they assumed a body position similar to that of athletes in the initiation of the second pull of a clean. This position allowed athletes to maintain a knee angle of approximately 120-140° as previously used (Haff et al., 2005; Stone et al., 2004). The pull-bar (Keiser UK, Tetbury, Gloucestershire, United Kingdom) could be fixed to various heights above the force platform, to accommodate different sized players. The height at which players pulled from was recorded so that players adopted the same position for post-training testing. The custom made pull-bar prevented any bending of the bar during the pull and the rack was anchored to the floor.

Prior to testing players commenced a standardised warm up which included 5min dynamic movement followed by sequential 60, 90 and 120kg mid-thigh clean pull or rack pulls (2-3 reps) depending on the players' preference. In preparation for maximal effort testing, one submaximal practice effort was then performed which also allowed investigators to ensure starting position could be maintained during testing.

When testing, the players stood on the force platform, and their hands were strapped to the bar using lifting straps. Minimal pre-tension was allowed to ensure that there was no slack in the subjects' body prior to the initiation of the pull (Beckham et al., 2013). The portable

force platform with built-in charge amplifier was used to measure the vertical component of the ground reaction force (GRF) of the subjects during performance of a maximal effort IMTP. A sample rate of 1,000 Hz and a vertical force range of 20 kN were used for all trials. The force-time data were recorded on a portable computer using a 16-bit analog to digital converter. A sample length of 10 seconds was used for all trials, consisting of a 5s quiet standing phase, and a 5s period when players performed the IMTP following the command to 'go'. The platform's calibration was checked before and after each testing session. During each trial, subjects were instructed to pull as hard and as fast as possible for a period of approximately 5s. These commands were based on previous research indicating that the use of these instructions produces optimal results for the attainment of maximal force (Bemben et al., 1990; Rahmani et al., 2001).

During upper body testing, players were asked to build up to a 3RM on the bench press and the chin-up. Prior to testing players completed a 5min dynamic warm up and 2-4 sets of 3-6 reps (~35-80% 1RM) of both bench press and chin-ups in preparation for maximal effort testing. When performing the bench press, players were requested to keep their hips on the bench and feet on the floor, and perform a full range of motion (i.e. to complete a repetition bar was lowered to chest and returned to a 'locked-out' arms position). Hand width on the bar performing the bench press was self-prescribed.

To add resistance to the chin-up, players added weights to a belt which was supported around the waist. Using a shoulder width supinated grip, players lowered themselves to hang so that arms were 'locked-out', and paused before being instructed to start the test. Each repetition was initiated from this position and was completed once the shoulders reached the line of the bar. Players were asked to work up to a 3RM however 2 and 4RM efforts were also accepted.

A period of familiarisation was not required for IMTP, bench press or chin-up testing prior to the initiation of the study, as the players were used to the tests and procedures as part of their normal testing battery.

Calculation of peak force from GRF-time history was determined using methods previously used in professional rugby league using similar testing procedures (West et al., 2011). A reliable start time or initiation of the IMTP was needed as a reference point for calculation of PF (Newtons; N). The force-time history was not suitable to define a start time because it lacked stability because of the subject holding onto the bar of the rack thus causing a, sometimes considerable, variation in the force being transmitted through the force platform during the pre-command quiet standing phase. However, the rate of change of force with respect to time, that is, rate of force development (RFD) did show stability during this period, and therefore, this variable was suitable to determine a quiet standing baseline value and threshold, beyond which the IMTP could be defined as having started. The instantaneous rate of change of force with respect to time was calculated from the first derivative of the vertical component of the GRF-time history. The GRF-time history was first filtered using a dual-pass Butterworth filter (low pass, 20-Hz cut-off) and then numerically differentiated using the central difference method. Filter settings were determined from a pilot study based on Fourier analysis and inspection. The first second of the first derivative-time history was then discarded to avoid the edge effects associated with digital filtering, and a mean and SD were calculated for the remaining 1 second of quiet standing immediately before the trigger point. The start time of the IMTP was then defined as the instant, after the trigger point, that the first derivative exceeded the mean value plus 5 SDs. The PF was determined from the vertical component of the GRF-time history and was defined as the peak produced during the IMTP minus the subject's body weight, which was measured prior to testing (Seca 876; Seca, Birmingham, U.K.). Subsequent to the determination of PF, Rel. PF was then calculated by dividing PF by the subject's body weight.

Calculation of bench Press and chin-up 1RMs from 3RM (± 1) testing were determined by use of prediction tables (Baechle and Earle, 2008). Body fat was measured as the sum of 8 calliper measurements (triceps, subscapular, bicep, iliac crest, supraspinale, abdominal, front thigh and calf; ISAK, 2007) and expressed as total millimetres (mm). Excluding iliac crest, the remaining 7 measures were then used to provide an estimate of fat mass (Wilmore and Behnke, 1970; Siri, 1961) from which an estimate of fat free mass was calculated.

Data are presented as mean \pm SD. Within-group changes and between-group differences in strength measures were analysed using a two-way (group*time) repeated measures analysis of variance (ANOVA) with paired t-tests carried out were relevant. The criterion level for significance was set at p<0.05. Statistical analyses were performed using the Statistical Package for the Social Sciences (SPSS for Windows, version 14.0; SPSS, Inc., Chicago, IL, USA).

7.3. Results

Following a 5 week pre-season training period, two players from the CON group were unable to have their body composition re-tested. Nevertheless, Table 7.3 demonstrates that were significant reductions in body fat (F=24.616, p<0.001) and increases in estimated fat free mass (F=43.047, p<0.001) in both groups, with no significant differences in the extent of change between the two groups.

Mean pre and post-training strength values from both training groups are shown in Table 7.4, which display that strength values were similar between the two groups at baseline. Predicted 1RMs for bench press and chin-up significantly improved in both groups (F=32.303, p<0.001; F=22.376, p<0.001; respectively), but there was no effect of recovery condition on either strength measure. However there were significant time*recovery group interactions observed for IMTP PF (F=6.138, p<0.05) and Rel. PF (F=7.146, p<0.05).

Significant improvements in IMTP PF (Figure 7.2) and Rel. PF were observed following training for participants in the CON group (PF 181 \pm 153N, p<0.05; 95% CI=78-284N; Rel. PF 1.8 \pm 1.6N.kg⁻¹, p<0.05; 95% CI=0.6-2.9N.kg⁻¹), however no significant changes were observed for those in the CWI group (PF -23 \pm 226N, p>0.05; 95% CI=-175-129N; Rel. PF - 0.6 \pm 2.4N.kg⁻¹, p>0.05; 95% CI=-2.2-1.0N.kg⁻¹).

Table 7.3. Baseline and post-training body mass, body fat measure (sum of 8) and estimated fat free mass (FFM) in the cold water immersion (CWI, n=11) and control (CON, n=9) recovery groups.

	Body m	iass (kg)	Sum of	8 (mm)	Estimated	FFM (kg)
	Baseline	Post-training	Baseline	Post-training	Baseline	Post-training
CWI	105.0 ± 13.1	105.3 ± 11.6	101.6 ± 40.3	91.4 ± 34.8 *	88.7 ± 7.3	91.4 ± 6.9*
CON	107.2 ± 14.4	107.4 ± 14.3	98.6 ± 28.4	88.2 ± 29.7 *	92.1 ± 9.7	93.7 ± 9.8 *

Data presented as mean \pm SD. *Significant difference from baseline (p<0.05).

Table 7.4. Baseline and post-training values for isometric mid-thigh pull (IMTP) peak force (PF) and relative force (Rel. PF), and bench press and chin-up predicted 1RMs in the cold water immersion (CWI) and control (CON) recovery groups.

p (kg)	Post-training	135.8 ± 7.2 *	$142.2 \pm 15.4^*$	
Chin-u	Baseline	131.6 ± 8.8	135.5 ± 14.6	
sss (kg)	Post-training	146.3 ± 11.5*	141.8 ± 19.7 *	
Bench pre	Baseline	139.5 ± 12.1	135.5 ± 17.7	
el. PF 5 ⁻¹)	Post-training	30.2 ± 5.3	$32.2 \pm 3.6*$	
IMTP Rel (N.kg ⁻¹	Baseline	30.8 ± 4.8	30.4 ± 3.2	
F (N)	Post-training	3161 ± 383	3357 ± 446 *	
IMTP P	Baseline	3184 ± 305	3176 ± 456	
		CWI	CON	

Data presented as mean \pm SD. *Significant difference from baseline (p<0.05).



Figure 7.2. Changes in isometric mid-thigh pull (IMTP) peak force (PF) values from baseline (Pre) to post-training (Post) in the cold water immersion (CWI) and control (CON) recovery groups.

Data presented as mean \pm SD. N= Newtons. *Significant difference from baseline (p<0.05).

7.4. Discussion

During periods of training such as pre-season in rugby union, a high frequency, intensity and volume of training may result in fatigue; which may characterised by the sensation of tiredness and/or decrements in muscular performance and function (Abbiss et al., 2005). Therefore post-exercise recovery strategies are utilised in attempt to accelerate recovery and thus maintain training quality, sustain greater training loads or enhance the effect of a given training load (Barnett, 2006). In particular, CWI is used widely in team and contact sports (e.g. Barnett, 2006) due to its demonstrated ability to enhance recovery of physical function and reduce the extent of DOMS post-exercise (Leeder et al., 2012a).

The mechanisms of CWI are proposed to be largely based on the effects of hydrostatic pressure (Wilcock et al., 2006). When external pressure on the body is increased, gas, fluid and substances are displaced to lower pressure areas. Hydrostatic pressure from CWI increases the pressure gradient between the interstitial compartment and the intravascular space to aid in the removal of plasma/exudate, thus reducing the cellular infiltration of inflammatory cells such as leukocytes and monocytes (Wilcock et al., 2006), which may in turn reduce the extent of secondary muscle damage and subsequent contractile function impairment (Cheung et al., 2003). Changes in skin temperature as a consequence of CWI may also reduce the extent of inflammation (Cote et al., 1988). A reduction in skin temperature stimulates cutaneous receptors to excite sympathetic adrenergic fibres (Cheung et al., 2003). This causes the constriction of local arterioles and venules, which reduces the permeability of cellular lymphatic and capillary vessels, thus restricts fluid diffusion into the interstitial space. The cooling of tissue may also help to reduce the perception of pain by decreasing the production of acetylcholine and superficial inhibitory cells that regulate the impulse of pain perception to the central nervous system (Wilcock et al., 2006). Reductions in inflammation may also reduce the sensation of pain by the stimulation of group III and IV afferent fibres. It is proposed that these afferents have negative feedback on the α motorneuron via the inhibitory interneuron (presynaptic reflex inhibition; Bigland-Ritchie et al., 1986); therefore reduced stimulation may enhance recovery of function during stretchshortening cycle type activities.

Reducing the extent of inflammation by means of CWI may have great importance for enhancing athlete recovery and subsequent performance; however research suggests that the inflammation process is necessary for adaptation to an exercise stressor (Arnold et al., 2007). For example, following exercise induced muscle damage it appears that inflammatory cells (including neutrophils) and growth factors play an important role in repair and adaptive remodelling of tissue despite initiating events that elicit further muscle damage (Yamane et
al., 2006). Research has shown that macrophages recruited by injured muscle of a phagocytic, pro-inflammatory phenotype facilitate a specific response to an exercise stimulus, removing necrotised parts of the injured myofibres (Jarvinen et al., 2005) prior to converting to an anti-inflammatory phenotype, releasing growth factors (Arnold et al., 2007) and secreting pro-inflammatory cytokines, fibrinectin and proteoglycans which facilitate the regeneration of myofibres (Jarvinen et al., 2005). Previous research also suggests that cell volume (i.e. inflammation) may be an important signal for protein metabolism (Grant et al., 2000; Millar et al., 1997). Therefore although CWI appears to be an important component of athlete recovery in periods of competition, it has been questioned whether it should be applied during periods of training which target physical development (Cook et al., *in press*; Leeder et al., 2012a; Versey et al., 2013). Indeed, previous research suggests that the application of post-exercise recovery strategies which restrict inflammation may actually attenuate adaptations to training (Fischer et al., 2004; Frohlich et al., 2014; Gomez-Cabrera et al., 2008; Nemet et al., 2009; Strobel et al., 2010; Yamane et al., 2006). In particular, Yamane et al. (2006) found that CWI attenuated adaptation to strength and endurance training in non-athletic individuals (Yamane et al., 2006). Strength training of the forearm flexors was conducted using a weight-loaded handgrip ergometer (3x8RM) for four weeks with one arm subjected to post-training CWI (20min, $10 \pm 1^{\circ}$ C), while the other received no immersion. Although no significant differences in maximal strength improvement were observed between the two arms, increases in strength endurance at 30% 1RM (determined prior to training) were significantly greater in the control arm compared to the arm which was immersed in cold water (91 \pm 81 vs. 53 \pm 40%; Yamane et al., 2006).

Despite the findings of Yamane et al. (2006) and Frohlich et al. (2014), recent research suggests that CWI may in fact enhance adaptation following an intensified training period in trained athletes. Therefore further research is required to establish the effect CWI may have on adaptation to strength training in trained athletes (Halson et al., 2014).

During pre-season we assessed whether CWI has an effect on adaptation to strength training in professional rugby union players by separating the training squad into two groups so that one group received CWI post-training while the other received no recovery strategy intervention. In both the CWI and CON group, bench press $(5.1 \pm 5.4\%; 4.6 \pm 1.6\%)$ and chin-up $(3.7 \pm 4.8\%; 5.7 \pm 3.8\%)$ strength significantly increased following 5 weeks of strength training during pre-season. A previous meta-analysis of 27 rugby union, rugby league and American football training studies found that on average, two strength sessions per muscle region (e.g. upper body) a week resulted in 0.9\% increases in strength per week (McMaster et al, 2013). Therefore the upper body strength gains observed were comparable to the values expected according to previous literature (McMaster et al., 2013).

When no recovery intervention was administered, IMTP PF and Rel. PF significantly increased during the pre-season phase by 5.4 ± 4.7 and $5.8 \pm 5.4\%$ respectively. However, there were no significant changes in IMTP PF or Rel. PF following training in individuals who were immersed in cold water post-training $(-1.2 \pm 7.4 \text{ and } -1.8 \pm 7.1\%)$. The results from the study may therefore demonstrate that CWI impedes adaptation to strength training in rugby union players. This finding supports the research presented by Yamane et al. (2006) who explain that by reducing the extent of muscle damage post exercise by administering CWI post-exercise, the stimulation and proliferation of satellite cells are reduced which interferes with the regenerative processes and "thus retard rather than support the desired improvement of muscular performance" (Yamane et al., 2006). Research also shows that superficial cooling may impair the anabolic and inflammatory responses post exercise (Nemet et al., 2009). Nemet et al. (2009) had twelve elite junior handball players complete an intervention involving 4x250m treadmill runs at 80% of each individuals maximum speed followed by a rest period with and without (resting supine in a quiet room for 1h, 21°C) local cold pack application to the hamstring muscle (applied using a compression wrap for 15min, followed by a 15min interval and an additional 15min application). Significant differences were found 1h post exercise with local cold pack application associated with significant decreases in IL-1ß (pro-inflammatory), IL-1ra (anti-inflammatory), IGF-I and IGFBP-3 (anabolic hormones) and a greater increase of IGFBP-1 (catabolic agent). Thus, the authors concluded that the application of cold compression may attenuate the anabolic effects of preceding training (Nemet et al., 2009), which over a period of training may explain the findings of Frohlich et al. (2014), Yamane et al. (2006) and the present study.

In contrast to the findings of the present and previous research (Frohlich et al., 2014; Yamane et al., 2006), recent research in trained athletes does not support the suggestion that CWI attenuates adaptation to training (Halson et al., 2014). During a pre-competition training period, male endurance-trained cyclists competing at national level were randomised to a CWI group (n=10) or a control group (n=11) for 7 days of baseline training, 21 days intensified training and an 11 day taper. Throughout the training period two weekly tests were used to track changes in fatigue and performance. Test efforts were performed on a commercially available air braked ergometer (Watt Bike, UK) which allowed measurement of power and cadence throughout the efforts. The first test involved 2x4min maximal efforts completed 42min apart ($2xMMP_{4min}$), while the second was a high intensity interval test (HIIT). Cold water immersion was performed four times per week in the CWI group, in

which subjects submerged their body (excluding head and neck) for $15\min(15.3 \pm 0.3^{\circ}C)$ within 30min of the cessation of training or testing. Following the training period, both groups improved in the 2xMMP_{4min} test however CWI (~9.3%) did so to a slightly greater extent than the CON group (~6.5%). Furthermore, during the HIIT both groups increased self-selected power output in the timed pursuits, however increases were bigger in the CWI group for pursuit 1 (\sim 2.4%) and pursuit 2 (\sim 2.1%). Not only do these findings suggest CWI does not impair training adaptation within trained athletes, but they suggest that CWI may enhance adaptation when compared to a control group (Halson et al., 2014). However there are a number of potential imitations to the study design. Firstly, CWI was only administered 4 times a week; therefore CWI was not administered after training on 13 out of 30 training days. Furthermore, it should be noted that post-training performance was taken from days 4 and 5 of the taper rather than the end, as not all subjects were willing to perform maximal efforts towards the end of the taper due to subsequent National cycling competition. With training planned so that performance was optimised at the end of the taper, improvements in physical preparation were unlikely to be realised after 4-5 days. Therefore post-training data used in the study may not reflect training adaptation from the training period.

Similar to research by Halson et al. (2014), it is also possible that fatigue may have influenced the results in the present study. Although non-significant, the CWI group completed more lower body weights sessions than the CON group $(9 \pm 1 \text{ vs. } 8 \pm 1 \text{ vs. } 1 \text{ vs. } 8 \pm 1$ respectively), therefore it is possible that residual fatigue from the training period may have masked training adaptation. Furthermore, a limitation of the present study is that the volume of load lifted, rugby/conditioning volume and extra conditioning session volume were not reported, therefore it is possible that results may have been influenced by differences in training volume. In retrospect, monitoring training loads and conducting a weekly marker of function; as conducted by Halson et al. (2014) would have provided greater substance to conclusions drawn from the present study. As the study was cross-sectional and not blinded, it is also possible that individual perception of the presence or absence of CWI may modulate recovery kinetics post-training and therefore may influence subsequent training (Cook and Beaven, 2013). However, as Halson et al. (2014) explain, the study was aimed to represent a real-world scenario, reflecting that athletes either do or do not engage in a form of recovery following training. Consequently, a lack of placebo may confound the elucidation of the mechanisms associated with the observed results (Halson et al., 2014).

Despite the findings of Halson et al. (2014) and the limitations discussed, the present study is the first to suggest that CWI may impair adaptation to strength training in professional athletes. Although it is unclear why differences in training effects between the two recovery groups were only observed in the IMTP, this may be explained by the fact CWI was only implemented following lower body and not upper body strength sessions. Furthermore, individuals were only immersed to the level of the anterior superior iliac spine; therefore with consideration to the proposed mechanisms of CWI it is unclear whether immersions would have had any effect on the recovery of upper body strength or training adaptation. Nevertheless, the findings of the present study have great implications for strength training in rugby union, particularly during periods when physical development is a key objective such as pre-season. Opportunities for physical development are limited in northern hemisphere rugby due to the duration of the competition phase, and with physical qualities shown to have great influence on individual and team performance in rugby codes (Gabbett et al., 2011; Gabbett and Seibold, 2013; Sedeaud et al., 2012), it is vital that periods of training such as pre-season are utilised to improve physical qualities such as strength.

7.5. Practical applications

For the first time we have demonstrated that CWI may impair the stimulus for adaptive physiology following strength training in elite athletes. It is therefore recommended that during periods of physical development that CWI and other post-exercise strategies which seek to reduce inflammation and muscle damage are avoided in attempt to optimise adaptation from training.

The results of this study therefore have great implications for the use of post-exercise recovery strategies for athletes however it should be considered that CWI was the only strategy investigated, which may vary in its mechanisms and efficacy in enhancing recovery compared to other strategies. Research is therefore required to assess the effect of other recovery strategies on adaptation to exercise. Furthermore, research is also required to investigate whether CWI impairs adaptation to other modalities of exercise.

EXPERIMENTAL STUDY 4

8. The effect of competition on the sleep patterns of elite rugby union players

8.1. Introduction

Rugby union is a physically demanding sport which results in large disruptions to both physiological and psychological (West et al., 2014) functions for several days following competition. With the accumulation of subsequent training, insufficient recovery prior to the start of the training week may compromise an individual's ability to train, reducing quality of training and the stimulus for adaptation and/or maintenance of physiological attributes. Insufficient recovery may also predispose an individual to greater risk of injury (Lazarim et al., 2009) and may compromise preparation for subsequent performances (McLean et al., 2010). Furthermore, an extended exposure to the demands of training and match play with insufficient recovery may lead to the development of overtraining syndrome (Kellmann, 2010).

Sleep is a basic requirement for human health and is recognised as an important component of athlete recovery due to its physiological (Dattilo et al., 2011) and psychological (Meerlo et al., 2008) restorative effects. For example, sleep is associated with an increase in anabolic hormones such as growth hormone (GH), which play a significant role in skeletal muscle growth and repair via an increase in protein synthesis and a reduction in protein breakdown (Kraemer and Mazzetti, 2003). Furthermore, learning and memory consolidation are sleepdependent processes (Walker and Stickgold, 2004), thus sleep has an important role in tactical and skill development and performance (Walker and Stickgold, 2004). However, the importance of sleep is most evident when disrupted (i.e. total or partially deprived and/or fragmented) with studies demonstrating the negative effects of sleep disruption on autonomic nervous system (e.g. Spiegel et al., 1999), endocrine system (e.g. Spiegel et al., 1999), biochemical (e.g. Spiegel et al., 1999), genetic (e.g. Moller-Levet et al., 2013), cognitive (e.g. Belenky et al., 2003) and neuromuscular (e.g. HajSalem et al., 2013) functions and mood states (e.g. Sinnerton and Reilly, 1992).

The importance of good sleep practice in elite athletes has been highlighted in literature; with Halson (2008) suggesting sleep may be the single most efficacious recovery strategy. Indeed, recent research suggests athletes perceive sleep to be the most important post-exercise recovery modality when compared to other modalities including cold water immersion, active recovery and massage (Venter, 2012). However, it has been suggested that frequently disrupted and restricted sleep is a prevalent problem in modern society (Meerlo et al., 2008) which may also be common within elite athletes (e.g. Leeder et al., 2012b). Research demonstrates that several factors associated with training and competition (e.g. training volume; Jurimae et al., 2004) may have negative consequences to athlete sleep.

Furthermore, research has shown that subsequent (Lastella et al., 2012) and preceding (Richmond et al., 2004) competition may disrupt sleep which would have great implications for the preparation and recovery of rugby players. To date no research has been published assessing sleep patterns of rugby union players with respect to match-play. Therefore, the aim of the present study was to examine the sleep patterns of professional rugby union players', with data collected prior and post-match-play to assess the potential influence of competition on sleep patterns.

8.2. Methods

Four matches were identified for the study due to their similarities in location (all home games), kick off times (6:30-7:30pm) and training schedules prior and post-match. Players conducted a strength (~45min) and rugby (~75min) session two days prior to the match, and a 'captains run' (~20min) session the day prior to the match. No training or recovery sessions were prescribed during the observed post-match period. Players were approached to volunteer for the study based on their selection for one of the matches. Consequently, twenty-eight male rugby union players volunteered for the study (24.4 ± 2.9yrs, 103.9 ± 12.2kg). Prior to providing informed consent, participants were given information outlining the rationale, potential applications of the study and procedures (Appendix B). Ethical approval was given by the Swansea University Ethics Committee (Appendix A). During the sleep period, activity outside of prescribed training was not restricted however players were asked to refrain from alcohol.

Players were monitored continuously (with the exception of any exercise involving physical contact) using an Actiwatch® worn on the non-dominant hand (Actiwatch 2; Phillips Respironics, UK) from two days prior to the match until 3 days post-match to observe sleep behaviour and whether it was altered with respect to competition. Actigraphy is a non-intrusive, cost-effective tool used to estimate sleep quantity and quality with a recent review suggesting it has reasonable validity and reliability in assessing sleep-wake patterns in normal individuals with average or good sleep quality (Sadeh, 2011). Actigraphy has also been shown to have increased sensitivity to awakenings when compared to self-reported sleep measures, which underestimated both duration and total number of nocturnal awakenings in comparison to measures derived from actigraphy (Kushida et al., 2001).

Each night players were requested to hold the marker button on the Actiwatch® for 3s when going to bed ('lights off') and immediately upon waking. This allowed manufacturers' software (Respironics Actiware 5; Phillips Respironics, UK) to calculate sleep behaviour based on manually determined time in bed, and time asleep; automatically detected from 1min epoch periods. Sleep behaviour was analysed for the following range of variables; time in bed, sleep latency (time to fall asleep from 'time to bed'), time asleep, time awake, percentage of time sleeping while in bed (sleep efficiency), actual sleep percentage, percentage of time moving and sleep restlessness (fragmentation index), as previously described in Table 3.4 (Leeder et al., 2012b).

To determine 'sleep quality' by actigraphy, measures of sleep efficiency and fragmentation index were used (Leeder et al., 2012b). All variables are presented as mean \pm SD. To assess changes in sleep patterns, sleep variables from each night were compared to two nights prior

to the match (reference night sleep) using a repeated measures analysis of variance (ANOVA). Significant differences were located by a Bonferroni corrected pair-wise comparisons. The criterion level for significance was set at p<0.05. Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS for Windows, version 14.0; SPSS, Inc., Chicago, IL, USA).

8.⁽. Results

Masures of sleep data for professional rugby players' prior and post competitive match-play are presented in Table 8.1.

Aralysis of variances show significant time-dependent differences for time to bed (F=26.425, η^2 =0.495, p<0.001), get up time (F=21.175, η^2 =0.440, p<0.001), time spent in bel (F=10.669, η^2 =0.283, p<0.001), time asleep (F=8.752, η^2 =0.245, p<0.001), and pecentage of time moving (F=4.602, η^2 =0.146, p<0.05).

Post-hoc tests revealed significant differences from the reference night sleep were largely due to changes in pre and post-match sleep behaviour. For example, compared to the reference night sleep, time in bed increased the night before the match (p<0.01; 95 % CI=0:10-1:28h; 9.7 \pm 13.5%), but returned to similar durations to the reference night post-match (p>0.05; -9.7 \pm 19.8%). Furthermore, time asleep decreased post-match (p<0.05; 95 % CI=0:03--1:59h; -19.5 \pm 19.8%) compared to two nights pre-match, but returned to similar durations to the reference night the following evening (p>0.05; -3.4 \pm 17.3%).

Despite results suggesting a tendency for sleep efficiency to reduce post-match compared to two nights pre-match, no significant changes in measures of sleep quality were observed (p>0.05, Table 8.1). Furthermore, no significant changes in sleep latency, time awake, actual sleep percentage or percentage of time moving were observed on any night compared to the reference night sleep (Table 8.1).

Tabl	e 8.1. Sleep acti _i	graphy data of f	professional rug	by union player	s prior and post	-match (n=28)	•			
Night of sleep	Time to bed	Get up time	Time in bed	Sleep latency	Time asleep	Time awake	Sleep efficiency (%)	Fragmentation index	Actual sleep (%)	Moving tir (%)
Pre-match+1	22:43 ± 0:42	7:40 ± 0:31	8:49 ± 0:49	$0:34 \pm 0:40$	7:04 ± 1:01	$1:05 \pm 0:39$	79.0 ± 9.2	32.2 ± 7.9	85.4 ± 8.7	18.6 ± 6 .
Pre-match	23:02 ± 0:45	8:48 ± 0:58*	9:38 ± 1:11*	$0:28 \pm 0:25$	7:37 ± 1:14	1:30 ± 1:02	78.3 ± 11.3	33.2 ± 9.6	83.7 ± 11.1	19.1 ± 5 .
Post-match	$0:49 \pm 1:11*$	8:56 ± 1:11 *	7:56 ± 1:48	$0:38 \pm 0:34$	6:02 ± 1:27 *	1:03 ± 1:03	74.7 ± 11.1	33.2 ± 9.9	87.0 ± 11.3	21.7 ± 5 .
Post-match+1	23:46 ± 1:27 *	8 :30 ± 1:28	8:39 ± 1:10	$0:29 \pm 0:30$	6:56 ± 1:09	1:07 ± 0:45	79.3 ± 8.0	30.0 ± 8.9	86.7 ± 8.4	18.5 ± 5 .
Post-match+2	22:45 ± 0:37	$7:02 \pm 0:37*$	8:10 ± 0:57*	$0:24 \pm 0:25$	6:40 ± 1:18	$0:59 \pm 0:52$	80.2 ± 11.8	28.3 ± 12.7	86.6 ± 11.9	16.5 ± 7 .

Presented as mean \pm SD; *significant difference from pre-match+1; p<0.05. All time measures in h:min.

170

The aim of this study was to examine the sleep patterns of professional rugby union players', and to assess the potential influence of competition on these patterns. The main finding of the study was that significant differences in several variables were observed pre and post competition when compared to a reference night sleep. Notably, the study demonstrates that players spent significantly less time asleep post-match which may have detrimental consequences for post-match recovery. However, as demonstrated by large standard deviation values, our results also suggest that large inter-individual differences in sleep patterns may exist within a squad.

Prior to the match, our results show that players spent on average 49 ± 68 min longer in bed compared to the previous night (p<0.05), which is similar to the findings of Richmond et al. (2004) who reported an increase in sleep duration the night before an Australian rules football match compared to the average of five night's non-match related sleep. The reason for increased sleep the night before a match was proposed to reflect a belief that a good night's sleep will maximise match performance the next day (Richmond et al., 2004). Indeed, research has demonstrated that an extended period of sleep extension (5-7 weeks; minimum 10h) improves physical and cognitive performance, as well as well-being (Mah et al., 2011). However research also shows that short term sleep extension (3 days) does not return physiological (Pejovic et al., 2013) or cognitive (Belenky et al., 2003) performance levels to pre-sleep deprived levels following a period of recovery and subsequent performance (Cook and Beaven, 2013), increasing pre-match sleep duration may be an important component of individual's preparation.

Similar to previous research by Richmond et al. (2004), the present study found a significant reduction in time spent asleep post-match compared to the reference night (Table 8.1). Acute or chronic sleep disruption may alter several physiological (e.g. autonomic nervous system; Spiegel et al., 1999) and cognitive functions (e.g. Belenky et al., 2003) which may be detrimental for recovery and preparation for exercise. Considering players have been shown to take up to 60h to recover post-match in rugby union (West et al., 2014), sleep deprivation post-match may therefore exacerbate physiological and psychological recovery times. Disrupted sleep post-match may be related to several factors related to match-play. For example, increases in pro-inflammatory cytokines from match-play (Cunniffe et al., 2010) may directly affect sleep regulation, or indirectly, by their action on Hypothalamic-pituitary-axis activation to increase body temperature, increase cortisol secretion, decrease the amount of nREM sleep and increase wakefulness (Vgontzas et al., 2002). Furthermore, sleep patterns

pot-match may be affected by disruptions in mood and other psychological factors (e.g. wried about consequences of performance). For example, Lastella et al. (2012) previously denonstrated significant negative correlations between tension, sleep quality and the number of awakenings in marathon runners pre-competition. In addition to the large physical and psychological demands associated with competition, athletes may also have disrupted sleep du to other factors associated with performance. For example, high-doses of caffeine (e.g. ~1200mg; Youngstedt et al., 1997); which were not controlled for in the present study, have bein demonstrated to impair sleep quality post-exercise (Youngstedt et al., 1997). Consideration should also be given to the time of kick-off (Youngstedt et al., 1997), the effects of travel (Richmond et al., 2004), and alcohol consumption during the post-match peiod (Feige et al., 2006) which was controlled for in the present study.

Research in non-athletic populations has also reported correlations between inter-night vaiability in actigraphy measures and measures of psychological and physiological stress (Mzick et al., 2009). However converse to the effect stress may have on sleep, it may be suggested from the findings by Mzick et al. (2009) that increases in inter-individual sleep paterns may increase markers of physiological and psychological stress. Consequently, with corsideration to the effects competition may have on sleep patterns, it may be suggested that efforts to establish consistency in time to bed and get up time should be encouraged for rugby union players. This may have implications for training and travel scheduling, and also challenges the belief that increasing the time in bed before match-play enhances performance (Richmond et al., 2004). However before recommendations are made on consistency of sleep paterns, future research is required to investigate the effect of a single night sleep extension on performance following sleep disruption or normal sleep, and the effect inter-night varability has on physiological and psychological stress in elite athletes.

Sleep patterns observed two days prior to match-play were similar to those reported by Leeder et al. (2012b) who assessed the average of 4 non-competitive night's sleep from 46 athletes. Leeder et al. (2012b) found that despite quantity of sleep being comparable between the athlete and non-sporting control group, significant differences between groups for variables including sleep latency, sleep efficiency and fragmentation index suggest quality of sleep was inferior for athletes. However Leeder et al. (2012b) found considerably larger variance for each sleep variable compared with the control group, suggesting there was considerable individual variation. Furthermore, when assessing individuals on a case by case basis, the authors report that many individuals had comparable sleep to the control group. However, only certain individuals displayed signs of sleep disruption. Standard deviation values from rugby players within this study were on average greater than those reported by Leeder et al. (2012b), with values suggesting greater inter-individual variation post-match

than on other nights for several variables. Thus although from the results it may be suggested that rugby players have inferior quality of sleep compared to non-sporting controls (Leeder et al., 2012b), sleep quality may vary considerably between individuals; particularly on match-day.

8.5. Practical applications

The results of the study demonstrate that sleep is deprived post-match which may have detrimental effects to the recovery process. Sport scientists and coaches should therefore attempt to address individual's post-match sleep in attempt to enhance recovery and preparations for subsequent training and/or performance. Certain supplements may be used to enhance sleep (e.g. cherry juice, Howatson et al., 2012) however recent research demonstrates that simply recommending changes to sleep preparation and environment (e.g. limiting use of television, mobiles phones and computers within 30mins of retiring to bed, Duffield et al., 2013) may be effective in enhancing time asleep and subsequent ratings of perceived soreness and fatigue the morning following exercise, when used in conjunction with other recovery modalities (e.g. cold water immersion and compression garments, Duffield et al., 2013). Following this study it would be pertinent to investigate the impact certain strategies may have on sleep patterns in rugby union players, however this progresses outside the main aim of the thesis and consequently requires further investigation.

Large standard deviation values observed for all variables pre and post-match suggest sleep quality, quantity and patterns may vary considerably between individuals. Individuals should therefore be assessed on a case-by-case basis with consideration to physiological and psychological monitoring/screening data. Consideration should also be given to administering subjective sleep questionnaires to athletes (e.g. Pittsburgh Sleep Quality Index; Buysse et al., 1989), as this may enhance understanding of individual sleep patterns from actigraphy (e.g. caffeine intake), or highlight issues which may not otherwise be detected (e.g. sleep inertia; Tassi and Muzet, 2000). Furthermore it is acknowledged that certain factors may have influenced sleep patterns which were not recorded or reported during the study. For example, napping, or factors associated with the 'sleep environment' such as alarm clocks or light may have influenced sleep quality and quantity.

With study two demonstrating that movement patterns may partly determine the extent of muscle damage post-match, future research is also required to investigate whether sleep patterns are to some extent determined by variances in movement patterns from performance.

174

GENERAL DISCUSSION

9. Discussion

Findings from studies one and two demonstrate that individual performance characteristics and the characteristics which determine the extent of muscle damage post-match are position specific. Previous research demonstrates that players make take several days to fully recover following competition in rugby union (West et al., 2014); however recovery patterns have been observed to vary greatly within a team (West et al., 2014). Therefore the findings in studies one and two may enhance understanding of individual performance demands and recovery patterns, which may have great implications for the physical preparation and management of players in rugby union.

The aim of study one was to assess the movement patterns of players during performance under the premise that a greater understanding of movement characteristics, together with the anthropometric and physiological characteristics of players, will lead to a greater understanding of the physiological demands of the sport. Sampling at a frequency of 10Hz, study one is the first assessment of the movement patterns in rugby union using GPS units which are valid and reliable for detecting high acceleration/deceleration and high-speed movements associated with rugby union (Castellano et al., 2011; Johnston et al., 2014b; Varley et al., 2011). Similar to previous research (Cahill et al., 2013; Quarrie et al., 2013; Roberts et al., 2008) the findings of study one demonstrate that movement patterns and thus physiological demands of match-play vary considerably between different positional groups. For example, the study found outside backs covered a greater distance (\sim 6272 vs. \sim 5244m), at a greater meterage (~ 68.8 vs. ~ 60.8 m.min⁻¹), performed a greater number of sprints (~ 20 vs. ~ 10) and reached a greater maximum velocity (~ 7.8 vs. 6.9m.s⁻¹) than loose forwards (p<0.05). However, loose forwards performed a significantly greater number of contacts (~38) compared to half, inside and outside backs (~19, ~21, ~16). Furthermore on average loose forwards performed the greatest amount of repeated high intensity effort (RHIE) bouts (~13) which was significantly greater than half backs (~5) and outside backs (~6).

Although the total accumulated time players engage in high-intensity exercise during a match can be relatively brief and the distance sprinted by players short, it has been proposed that the ability of players to perform RHIE may be critical to the outcome of the performance (Roberts et al., 2008; Austin et al., 2011a). Study one provides a detail analysis of positional RHIE demands. Using this data it has been suggested that by using maximum periods of activity coupled with minimum periods of recovery; the most demanding passages of play may be replicated providing coaches with a sense of their players' preparedness to meet the requirements of competition (Austin et al., 2011a). Furthermore, it is proposed that coaches should use minimum, mean, maximum and standard deviation values reported in the study as

a refrence to modulate RHIE protocols which may allow progressions in intensity during training and rehabilitation. Varying the high intensity activity periods may allow coaches to workon and integrate various high intensity activities patterns. Therefore activity periods may le shorter or may consist of multiple activities. Consideration should also be given to the dration and activity performed during 'recovery' between RHIE bouts. Using average and sandard deviation values, recovery times may be adjusted to increase intensity of effort.

In addition to the full match analysis, a temporal analysis of movement patterns was investgated using data files from players who completed a full game. Matches were separted into 10min periods with time played >40mins in each half excluded. Temporal analysis of all players displayed significant differences in player load.min⁻¹, cruising.min⁻¹ and sriding.min⁻¹ between halves, with measures of low and high intensity movement and acceleration/deceleration significantly declining throughout each half. Despite high-speed meterige exhibiting the greatest percentage reduction from 0-10mins between 30-40mins, the gratest reductions in movement in the second half compared to the first 10mins were foundin low speed movements such as cruising and striding supporting previous research in rugby union (Roberts et al., 2008). Research by Roberts et al. (2008) and Mooney et al. (2013 suggest that reductions in movement patterns throughout match-play in rugby codes may be caused by a reduction in low intensity activities, which may be characterised by an inability to maintain defensive position or run supporting lines in attack. Furthermore, reductons in low speed compared to high speed movements may be evidence of 'pacing' whereby players sacrifice distances covered at low speeds to compensate for the demands of high speed movement (Mooney et al., 2013). Knowledge of variances in movement patterns may turther inform practitioners with the demands likely to be encountered during compatition. Furthermore, understanding expected typical and individual decrements in performance (e.g. m.min⁻¹, load.min⁻¹) during 10min periods using GPS may allow sport scientists to detect fatigue from which tactical decisions may be made. Indeed with considuration to previous research in rugby union, monitoring of work at lower intensities (e.g. cuising) may be most sensitive to detect fatigue (Roberts et al., 2008). However, despite the potential applications of real-time monitoring, consideration should be given to the possible errors associated with real-time data collection (Aughey and Falloon, 2010).

Following study one, it is understood that movement characteristics may vary greatly within a team which may partly explain why recovery patterns also vary between players (West et al., 2014). Indeed, previous research suggests that performance characteristics could be used to prospectively predict individual recovery in rugby union (Cunniffe et al., 2010, Takarada, 2003) and other team sports (McLellan et al., 2011a; McLellan et al., 2012, Thorpe and

Sunderland, 2012). Physical contacts have been correlated to creatine kinase (CK; an indirect blood marker of muscle damage) level in rugby union (Cunniffe et al., 2010, Smart et al., 2008, Takarada, 2003) and neuromuscular recovery in rugby league (McLellan et al., 2012), suggesting recovery may be determined by the extent of mechanical damage induced through contact during performance in rugby codes. However no research had been conducted to assess the correlation between high-speed running and markers of recovery in rugby union.

Performed during the group stages of the 2012-2013 European Cup, study two assessed the correlation between changes in CK from 2hrs pre-game to 16 and 40hrs post-match, with performance characteristics associated with high-speed movement and physical contacts derived by use of GPS and notational analysis. In accordance with previous research (Cunniffe et al., 2009), match-play resulted in large increases in CK, with peak increases observed at 16hrs post-match (~417%), and values remaining significantly elevated from pre-game values at 40hrs post-match (~267%). Furthermore, as found by West et al. (2014), results from the study suggest a highly individual nature of recovery; with a large range in change in CK from pre-match (Δ CK) at 40h post-match (30 to 1849 IU.L⁻¹).

Similar to previous research the study demonstrates that muscle damage induced by professional rugby union match play is to some extent predicted by the number of physical contacts induced during performance (Cunniffe et al., 2010, Smart et al., 2008, Takarada, 2003). For example, moderate-large effect-size correlations were identified between contact statistics from performance analysis and changes in CK at 16 and 40hrs post-match in forwards and backs. These findings may be explained by the blunt force trauma from physical contacts which disrupt skeletal tissue structure integrity; subsequently increasing cell permeability and the diffusion of soluble enzymes such as CK into the interstitial fluid (Cheung et al., 2003). Furthermore, this study is first to show that muscle damage in backs players is predicted by high-speed running measures derived from GPS. For example, moderate effect-size correlations were found between measures of high-speed running and sprinting, and change in CK at 16 and 40hrs post-match within the backs. These correlations may be explained by high-force, eccentric work when performing SSC activities such as high-speed running, which may exceed the muscles ability to actively resist load, forcing the muscle to lengthen and generate greater active tension (Stauber, 2004). Although correlation is by no means causation, a potential explanation for the relationships between high-speed movement and changes in CK only being observed in backs is likely due to their greater high-speed running demands when compared to the forwards as observed in study one.

Nevertheless, the findings of study two support previous suggestions that performance characteristics may be used to manage player recovery and subsequent training post-match.

Recovery kinetics post-match may be modulated by the application of post-exercise recovery strategies such as cold water immersion (CWI), which has been demonstrated to enhance recovery of physical function and reduce the extent of muscle soreness post-exercise (Leeder et al., 2012a). In addition to teams using post-exercise recovery strategies to speed up recovery from matches, they may also be used post-training during periods of physical development such as pre-season in attempt to optimise training adaptation; by potentially allowing greater training loads (increased frequency, intensity or duration) to be performed in subsequent sessions, thus increasing the stimulus for adaptation (Versey et al., 2013). However, despite enhancing physiological recovery, the use of strategies such as CWI to enhance adaptation may be counterintuitive. For example, it has been suggested that muscle damage and the subsequent inflammatory response is a vital precursor for the signalling mechanisms which initiate the repair and growth of cells (Carlson and Faulkner, 1983), therefore incorporating strategies which attempt to blunt the effects of exercise induced muscle damage may in fact reduce the potential for training adaptation to occur. Indeed research has demonstrated that several strategies which aim to reduce the extent of the inflammatory process; CWI (Frohlich et al., 2014; Yamane et al., 2006), local cold pack application (Nemet et al., 2009) and antioxidant supplementation (Fischer et al., 2004; Gomez-Cabrera et al, 2008; Strobel et al., 2010), may inhibit the stimulus for adaptive physiology.

During a 5 week pre-season training period the effect of CWI on adaptation to strength training was assessed by separating the training squad into two groups so that one group received CWI post-training while the other received no recovery strategy intervention. When no recovery intervention was administered, isometric mid-thigh pull (IMTP) peak force (PF) and relative peak force (Rel. PF) significantly increased during the pre-season phase by 5.4 ± 4.7 and $5.8 \pm 5.4\%$ respectively. However, there were no significant changes in IMTP PF or Rel. PF following training when individuals were immersed in cold water post-training (-1.2 \pm 7.4 and -1.8 \pm 7.1%). The results from the study may therefore demonstrate that CWI impedes adaptation to strength training in rugby union players. This finding supports the research presented by Yamane et al. (2006) who explain that by reducing the extent of muscle damage post exercise by administering CWI post-exercise, the stimulation and proliferation of satellite cells are reduced which interferes with the regenerative processes and "thus retard rather than support the desired improvement of muscular performance" (Yamane et al., 2006).

In contrast to the findings of the present and previous research (Yamane et al., 2006; Frohlich et al., 2014), recent research in trained athletes does not support the suggestion that CWI attenuates adaptation to training (Halson et al., 2014). It is unclear why the findings of study three contrasts with those from Halson et al. (2014), nevertheless the present study is the first to suggest that assess the effect of CWI may impair on adaptation using to strength training in professional athletes. The findings of study three therefore have great implications for strength training, particularly during periods of physical development.

Understanding how performance characteristics may affect recovery kinetics has great implications for player management post-match. However recovery kinetics may also be influenced post-match by factors not associated with match-play. For example, sleep is suggested to be an important modulator of recovery due to its physiological (Datillo et al., 2011) and psychological (Meerlo et al., 2008) restorative effects. However, recent research suggests that frequently disrupted and restricted sleep may be common among elite athletes (Leeder et al., 2012b), which may have detrimental effects to athlete recovery and preparation. Study four therefore examined the sleep behaviour of professional rugby union players', with data collected prior and post-match-play to assess the potential influence of competition on sleep patterns.

Using actigrapy, sleep behaviour pre and post-match was compared against a reference night sleep (non-competition; 2 nights prior to match) which suggested alterations to pre and postmatch sleep behaviour. Prior to the match, results show that players spent ~49mins longer in bed compared to the previous night, which is similar to the findings of Richmond et al. (2004) who reported an increase in sleep duration the night before an Australian rules football match compared to the average of five night's non-match related sleep. The reason for increased sleep the night before a match was proposed to reflect a belief that a good night's sleep will maximise match performance the next day (Richmond et al., 2004). Indeed, research has demonstrated that an extended period of sleep extension (5-7 weeks; minimum 10hrs) improves physical and cognitive performance, as well as well-being (2011). However research also shows that short term sleep extension (3 days) does not return physiological (Pejovic et al., 2013) or cognitive (Belenky et al., 2003) performance levels to pre-sleep deprived levels following a period of restriction. Nevertheless, with recent research showing a relationship between individual perception of recovery and subsequent performance (Cook and Beaven, 2014), increasing pre-match sleep duration may be an important component of individual's preparation. Following match-play, the study also demonstrates that players spent significantly less time asleep compared to the reference night sleep (<~62mins; 6:02hrs), which may be detrimental to recovery as acute sleep disruption

may alter several physiological (e.g. autonomic nervous system; Spiegel et al., 1999) and cognitive functions (e.g. Belenky et al., 2003). Considering players have been shown to take up b 60hrs to recover post-match in rugby union (West et al., 2014), sleep deprivation post-math may therefore exacerbate physiological and psychological recovery times.

Slep patterns observed two days prior to match-play were similar to those reported by Leeler et al. (2012b) who assessed the average of 4 non-competitive night's sleep from 46 athlets compared to 20 non-sporting controls. Leeder et al. (2012b) found that despite quartity of sleep being comparable between the athlete and non-sporting control group, significant differences between groups for variables including sleep latency, sleep efficiency and fragmentation index suggest quality of sleep was inferior for athletes. However Leeder et al (2012b) found considerably larger variance for each sleep variable compared with the contol group, suggesting there was considerable individual variation. Furthermore, when assessing individuals on a case by case basis, the authors report that many individuals had comparable sleep to the control group. However, only certain individuals displayed signs of sleet disruption. Standard deviation values from rugby players within this study were on avenge greater than those reported by Leeder et al. (2012b), with values suggesting greater interindividual variation post-match than on other nights for several variables. Thus although from the results it may be suggested that rugby players have inferior quality of sleer compared to non-sporting controls (Leeder et al., 2012b), sleep quality may vary consderably between individuals; particularly on match-day.

9.1. Practical applications

- The data presented from study one may enhance knowledge of the positional requirements of performance. This in turn may help monitor performance and inform the planning of position specific programmes that elicit physiological adaptations.
- Monitoring of temporal patterns, in particular low speed movements and player load may allow coaches and sport scientist to assess preparedness for performance and fatigue during match-play. Knowledge of transient fatigue may also have important implications for tactical decisions made during match-play and the use of match-day strategies to enhance performance.
- Similar to previous research, study two demonstrates that muscle damage following match-play is to some extent predicted by the number of physical contacts induced

during performance for all players. Furthermore, it is the first to demonstrate that muscle damage is also determined by factors related to high-speed running and sprinting for backs.

- As an alternative to assessing individual recovery following each match, study two suggests performance markers may be used to tailor individual recovery strategies and subsequent training.
- CWI may impair the stimulus for adaptive physiology following strength training in elite athletes.
- During periods of physical development it is suggested that CWI and other postexercise strategies which seek to reduce inflammation and muscle damage are avoided in attempt to optimise adaptation from training.
- Study four suggests that sleep disruption is evident with rugby union players, particularly post-match, which may have detrimental effects to recovery and preparation for training/competition.
- Individual assessment should be conducted within a squad to identify those who may display evidence of sleep disruption, with consideration given to physiological and psychological monitoring data.
- Study four demonstrates actigraphy is a non-invasive, practical means of assessing sleep patterns in professional team sport athletes.

9.2. Recommendations for future research

• Despite exhibiting significant positional group differences in movement characteristics, further research is required to investigate the differences between individual positions. For example, open-side flankers were suggested to cover comparable high-speed distances to inside and outside backs, and cover greater distances than other loose forwards however due to sample sizes individual positions were not statistically analysed. Further research with greater sample sizes will therefore give a greater indication of specific positional movement patterns which will provide even greater understanding of physiological demands for performance.

- Temporal analysis of match-play demonstrated transient fatigue throughout each half however further research is required to investigate whether changes in movement patterns throughout each half vary between positions. Furthermore, research to investigate the effect of physical preparedness and fatigue on work rate and transient fatigue would provide a greater understanding of the effect of fatigue on performance.
- Studies comparing the effect of the opposition, possession, territory, success and playing standard on movement characteristics in rugby league (Gabbett, 2013a; Gabbett, 2013b; Gabbett 2013c; Gabbett et al., 2013) and Australian rules (Aughey et al., 2011b; Sullivan et al., 2014) have demonstrated trends which may be applied to further understand movement characteristics (Kempton et al., 2013a). For example, research has shown physical demands determined by GPS vary according to team playing standard (Gabbett, 2013a), the opposition (Gabbett, 2013b) and team success (Gabbett, 2013c) in rugby league. Research is therefore warranted to understand how movement characteristics may vary during match-play in rugby union. Furthermore, research is warranted to understand the influence of playing environment (e.g. conditions) on movement characteristics.
- Further research is also required using greater sample sizes to determine whether performance characteristics which relate to increases in muscle damage vary within positional groups. For example, moderate effect size correlations were found between muscle damage and high-speed running volume in back players however study one demonstrates that inside and outside backs cover significantly greater distances at high-speed than half backs. Furthermore, no correlations were found between high-speed running and muscle damage in forwards however open-side flankers cover much greater distances at high-speed than other forwards. Therefore despite the findings of the present study, further research may provide an even greater understanding of the movements and mechanisms of muscle damage relative to playing position which may enhance player management.
- Despite study two observing correlations between movement characteristics and muscle damage, it would be important to examine the correlation between performance characteristics and recovery of function as previous research suggests that there is a potential for neuromuscular function reduction to remain, despite normalised CK (West et al., 2014), and steroid hormone profiles (West et al., 2014).

- Although contact statistics may give an indication of the extent of muscle damage experienced by players, it does not account for the extent of contact or exertion. Therefore despite advances in GPS technology further research is required to quantify the extent of physical contact.
- With recent research suggesting physiological recovery may be modulated by the psychological response to intervention, further research investigating the effect of strategies on adaptation is required with consideration to individual perception of the strategies used.
- The results of study three have great implications for the use of post-exercise recovery strategies for athletes however it should be considered that CWI was the only strategy investigated, which may vary in its mechanisms and efficacy in enhancing recovery compared to other strategies. Research is therefore required to assess the effect of other recovery strategies on adaptation to exercise. Furthermore, research is also required to investigate whether CWI impairs adaptation to other modalities of exercise.
- Research demonstrates sleep may be disrupted post-match however further research is required to assess the factors which may contribute towards this, including performance demands. Furthermore, it is inferred from previous research that disrupted sleep may impair post-match recovery; however to what extent this may be has not been researched.

APPENDIX A

Applications for Ethical Committee approval of a research project

SPORT AND EXERCISE SCIENCE COLLEGE OF ENGINEERING, SWANSEA UNIVERSITY ETHICAL ADVISORY COMMITTEE

APPPLICATION FOR ETHICAL COMMITTEE APPROVAL OF A RESEARCH PROJECT

In accordance with Departmental Safety Policy, all research undertaken in the department must be approved by the Departmental Ethics Advisory Committee **prior to** data collection. **Applications for approval should be typewritten on this form using the template available in the Public Folders.** The researcher(s) should complete the form in consultation with the project supervisor. Where appropriate, the application must include the following appendices:

(A) subject information sheet;

(B) subject consent form;

(C) subject health questionnaire.

After completing sections 1-12 of the form, 1 copy of the form should be handed-in to the Department Administrator who will then submit copies of the application for consideration by the Departmental Ethics Advisory Committee. The applicant(s) will be informed of the decision of the Committee in due course.

1. DRAF'T TITLE OF PROJECT

Characterissation of Recovery from Professional Rugby Union

2. NAMES AND STATUS OF RESEARCH TEAM

Rhys Joness (PhD Student) Dr. Liam Kilduff (Supervisor, Swansea University) Mr. Nick Owen (Supervisor, Swansea University) Dr. Blair Crewther (Supervisor/Researcher Visitor, Swansea University) Dr. David Shearer (University of Glamorgan) Brad Harrimgton (Head of Strength & Conditioning, Scarlets Rugby)

3. RATIONALE

As a consequence of the demands of training and competition, rugby players are likely to experience high levels of physiological and psychological stress which may result in fatigue. Previous research (Shearer et al., under review) suggests that professional players may have not fully recovered three days following competition therefore compromising the extent to which a player can train, and with the added stress of training may compromise preparation for a following game. The recovery process following competition and training may be affected by multiple factors however functional recovery is strongly correlated to the extent of muscle damage induced through exercise and the subsequent inflammatory response (Avela et al., 1999; Horita et al., 2000; Highton et al., 2009).

Several strategies have been proposed to reduce exercise induced muscle damage (e.g. cold water immersion, contrast water therapy, compression garments) and thus enhance the rate of recovery; however, despite their prevalence equivocal evidence exists to support their application with variations in testing methodologies making comparison between studies and determination of strategy efficacy difficult.

Muscle darnage induced via mechanical insult and through increased oxidative stress in rugby union have produced levels of muscle damage which far exceed those reported in laboratory studies (Gill et al., 2006), therefore to assess whether recovery strategies may enhance recovery in rugby union it may be most appropriate to use competition as a means of muscle damage. However, to use competition as a model of assessing the efficacy of various methods, performance characteristics, muscle damage and the recovery process need to follow the same pattern at each game.

4. REFERENCES

Avela, J., Kyrolainen, H., Komi, P., Rama, D. (1999). Reduced reflex sensitivity persists several days after long lasting stretch shortening cycle exercise. *Journal of Applied Physiology*, **86**: 1292-1300.

Elloumi, M., Maso, F., Michaux, O., Robert, A., Lac, G. (2003). Behaviour of saliva cortisol (C), testosterone (T) and the T/C ratio during a rugby match and during the post-competition recovery days. *European Journal of Applied Physiology*, **90**: 23-28.

Gill, N., Beaven, C., Cook, C. (2006). Effectiveness of post-match recovery strategies in rugby players. British Journal of Sports Medicine. 40: 260-263. 2006

Hightom, J., Twist, C., Eston, R. (2009). The effects of exercise-induced muscle damage on agility and sprint running performance. *Journal of Exercise Science and Fitness*, 7: 24-30.

Horita, T. (2000). Stiffness regulation during stretch-shortening cycle exercise. Thesis (PhD). University of Jyvaskyla.

Shearer, D. (in review). Monitoring training and competition fatigue in elite athletes: a neuromuscular and endocrine validation of the brief assessment of mood (BAM). *Journal of Sports Sciences*.

Taylor, K-L., Barker, M., Cronin, J., Gill, N., Chapman, D., Sheppard, J. (2009). *The effect of an extended warm-up on diurnal performance differences in loaded counter-movement jumps*. Panel presented at the NSCA 32nd National Conference & Exhibition, Las Vegas, NV. Abstract retrieved from http://www.nsca-lift.org/Abstracts/Abstracts.pdf

5. AIMS and OBJECTIVES

The study aims to characterise the recovery process following three professional rugby union matches by assessing the recovery of markers of muscle damage (CK, CRP), hormone status (Testosterone and Cortisol), self-perceived recovery (modified profile of mood states), heart rate variability and functional recovery (countermovement and drop jumps) compared to pre-game values. In addition to monitoring the recovery of several physiological markers, match performance will be analysed using notational analysis and Global Positioning Systems (GPS) to determine the variations in performance characteristics.

In addition to characterising the recovery process and determining whether competition may be used as a means for assessing the efficacy of recovery strategies, the findings may give an indication to which markers are most sensitive to fatigue following competition which may improve monitoring procedures/future protocols. We will also assess the correlations between several variables which may provide a greater understanding of the recovery process.

6. METHODOLOGY

6.1. Study Design

Subjects are to be assigned from a male senior professional regional rugby union club (n=23, aged 19-34) who compete at European club level and contain several international players.

The study is to be performed during the 2011-2012 season around three competitive games. Tests performed will be part of the clubs monitoring procedures and will be conducted by club and university staff. Players will be required to report to training at the clubs training facility at their specified times and will the testing procedures should last no longer than 30 minutes. Players will be required to exert maximal effort on functional tests and are required to conform to pre-testing conditions stipulated.

6.2. Experimental Procedures

Other than match-days, testing procedures shall remain standard for all studies. Players will be given time allocations to arrive for testing at the clubs training facility which will remain the same for each testing day to minimize endocrine and neuromuscular fluctuations through intra-day variance (Elloumi et al., 2003; Taylor et al., 2009). Players will only be allowed to train at scheduled sessions and will not be allowed to perform any activity on the day prior to pre-competition testing. Players also restricted from drinking alcohol throughout the study period.

On non-match days, players will be required to attend in groups of 8. Bodyweight monitoring (Seca 770 Digital Scales, Seca Ltd., Birmingham, UK) and hydration analysis (Osmocheck Refractometer, Vitech Scientific, UK;

on non-match days only) will be performed prior to further testing.

Players will first be provided with heart rate monitors (Polar RS800CX; Polar Electro, Kempele, Finland) for analysis of heart rate variability. After attaching the heart rate belt so that the monitor is positioned at the base of the sternum (following application of conductive gel), players must lie in a supine position for 5mins to assess R-R intervals. From the last 3mins various vagal-related indices will be calculated to assess the parasympathetic activity of the nervous system.

Once heart rate variability analysis is completed, players will be required to provide a saliva sample by inserting a Salimetrics Oral Swab (SOS; Salimetrics Europe, Suffolk, UK) underneath their tongue for 3mins before removing and placing in plastic tube for further analysis of testosterone and cortisol. While waiting for the swab to become saturated by saliva, subjects will complete a modified profile of mood states (POMS) questionnaire which has previously been shown to correlate changes in function following competition (Shearer et al., under review).

Following saliva collection players will then be required to provide a ~400UL capillary blood sample from the fingertip for analysis of markers of muscle damage and inflammation (Creatine Kinase and C-Reactive Protein).

Players will then undergo a 5 minute dynamic warm up, followed by 3 practice countermovement jumps. Following warm up players will be reminded of the procedures before performing 2 maximal single countermovement jumps and 2 maximal drop jumps from a 40cm box. For countermovement jumps players will be required to stand still on the force plate (Kistler type 92866AA, Kistler Instruments Ltd., Farnborough, UK) with hands on hips to allow baseline vertical force to be determined. Once instructed to jump, players will be required to jump as high as possible while keeping their hands on their hips. Countermovement depth is to be self-selected for each player. For drop jumps players will stand on the box until asked to jump. Players are to perform the movement with hands on hips and will be required to jump as high as possible while minimising contact time on the force platform. Various performance markers will be calculated to assess functional recovery (Bioware type 2812A1-3, Kistler Instruments Ltd., Farnborough, UK).

Tests are to be performed the day prior to the game (approximately 36hrs pre-game), post-match, approximately 12hrs post-game and 36hrs post-game. Due to the variance in kick-off times, it will not be possible to minimize the fluctuations due to intra-day variance on match day. Furthermore, it will be difficult to limit the ingestion of fluid and food during the second half of the game and limit caffeine prior and during competition. Prior to the game players will be required to provide a saliva sample and complete the POMS questionnaire. Immediately following the completion of the game players will be required to provide a blood sample and perform jump tests as described above.

Following the game, individual and team statistics will be collected from the clubs performance analysis staff (SportsCode Gamebreaker; Sportstec, Warriewood, NSW). Performance characteristics will also be taken from data collected by Global Positioning Systems worn by the players (minimaxx v4 10Hz; Catapult Sports, Victoria, Australia).

6.3. Data Analysis Techniques

Using SPSS software (version 3; SPSS Inc., Chicago, IL.), a repeated measures analysis of variance (ANOVA) will be performed to establish the significance of changes between data collection points.

6.4. Storage and Disposal of Data and Samples

Saliva and plasma blood samples will be placed in an insulated box filled with ice and transferred to the exercise physiology laboratory at Swansea University where they will be frozen at a temperature of -60° C and stored for subsequent analysis. Access to samples will be supervised by the laboratory technician. Following analysis all saliva and blood samples will be disposed of.

Under the responsibility of the research team, the data collected will be stored during analysis with a password on a secure computer with access available to investigators and supervisors only. Feedback and interpretation of results will be provided to rugby club staff when requested. Data will be disposed of post analysis by the research team but will be retained by rugby club staff for future reference.

7. LOCATION OF THE PREMISES WHERE THE RESEARCH WILL BE CONDUCTED.

Testing will be conducted at the regional rugby training facility (Parc-Y-Scarlets, Pemberton, Llanelli) and at the Exercise Physiology laboratory at Swansea University. All tests will be performed under the supervision of Dr. Liam Kilduff.

8. SUBJECT RISKS AND DISCOMFORTS

Other than the blood capillary sampling, the testing procedure is non-invasive and should not induce physiological fatigue in the players. The blood capillary sample will require a finger prick to be administered which will be a small, sharp pain however very little discomfort will be experienced during collection. The warm up devised by University and club staff should reduce the likelihood of any injury from performing maximal jumps, however club first aid staff and doctors will be present at all testing sessions. Each individual will be required to complete an AHA/ACSM health screening questionnaire to ensure no risk factors for testing exist.

9. INFORMATION SHEET AND INFORMED CONSENT

The submission should be specific about the type of consent that will be sought:

Have you included a Subject Information Sheet for the participants of the study ? YES

Have you included a Subject Consent Form for the participants of the study? YES

10. COMPUTERS

Are computers to be used to store data? YES

If so, is the data registered under the Data Protection Act? YES

11. STUDENT DECLARATION

Please read the following declarations carefully and provide details below of any ways in which your project deviates from them. Having done this, each student listed in section 2 is required to sign where indicated.

- 1. I have ensured that there will be no active deception of participants.
- 2. I have ensured that no data will be personally identifiable.
- 3. I have ensured that no participant should suffer any undue physical or psychological discomfort
- 4. I certify that there will be no administration of potentially harmful drugs, medicines or foodstuffs.
- 5. I will obtain written permission from an appropriate authority before recruiting members of any outside institution as participants.
- 6. I certify that the participants will not experience any potentially unpleasant stimulation or deprivation.
- 7. I certify that any ethical considerations raised by this proposal have been discussed in detail with my supervisor.
- 8. I certify that the above statements are true with the following exception(s):

Student signature: (include a signature for each student in research team)

Date:

12. SUPERVISOR'S DECLARATION

In the supervisor's opinion, this project (delete those that do not apply):

• Raises some ethical issues, but I consider that appropriate steps and precautions have been taken and I have approved the proposal.

Supervisor's signature:

Date:

13. ETHICS COMMITTEE DECISION (COMMITTEE USE ONLY)

ETHICAL APPROVAL: GRANTED REJECTED (delete as appropriate) The ethical issues raised by this project have been considered by members of the Departmental Ethical Approval Committee who made the following comments: Please ensure that you take account of these comments and prepare a revised submission that should be shown to your supervisor/ resubmitted to the Department Ethical Approval Committee (delete as appropriate). Signed: Date:

(Chair, Departmental Ethics Advisory Committee)

SPORT AND EXERCISE SCIENCE COLLEGE OF ENGINEERING, SWANSEA UNIVERSITY ETHICAL ADVISORY COMMITTEE

APPLICATION FOR ETHICAL COMMITTEE APPROVAL OF A RESEARCH PROJECT

In accordance with Departmental Safety Policy, all research undertaken in the department must be approved by the Departmental Ethics Advisory Committee **prior to** data collection. **Applications for approval should be typewritten on this form using the template available in the Public Folders.** The researcher(s) should complete the form in consultation with the project supervisor. Where appropriate, the application must include the following appendices:

- (D) subject information sheet;
- (E) subject consent form;
- (F) subject health questionnaire.

After completing sections 1-12 of the form, 1 copy of the form should be handed-in to the Department Administrator who will then submit copies of the application for consideration by the Departmental Ethics Advisory Committee. The applicant(s) will be informed of the decision of the Committee in due course.

1. DRAFT TITLE OF PROJECT

Does cold water immersion impair adaptation to strength training in professional rugby union players?

2. NAMES AND STATUS OF RESEARCH TEAM

Rhys Jones (PhD Student) Dr. Liam Kilduff (Supervisor, Swansea University) Brad Harrington (Head of Athletic Performance, Scarlets Rugby)

3. RATIONALE

As a consequence of the physical demands of rugby union, players make take several days to fully recover following match-play (West et al., 2014). Insufficient recovery may compromise an individual's ability to train and may predispose an individual to a greater risk of injury (Lazarim et al., 2009). Furthermore, with the accumulation of subsequent training, preparation for subsequent matches may be compromised (McLean et al., 2010). Therefore, in an attempt to accelerate recovery following training and match-play, it is common practise to employ one or more post-exercise recovery strategies, such as cold water immersion (CWI).

Cold water immersion has been reported to reduce the extent of muscle damage, inflammation and contractile function impairment following exercise (Wilcock et al., 2006). Indeed a recent meta-analysis found that CWI is an effective strategy to enhance recovery of muscle power post-exercise (Leeder et al., 2012). Furthermore, research demonstrates that CWI (e.g. 2x5min at 10°C; Ingram et al., 2009) is an effective strategy in reducing blood markers of muscle damage (e.g. Vaile et al., 2008) and delayed onset of muscle soreness (DOMS) post-exercise (e.g. Vaile et al., 2008), and may enhance recovery of strength (e.g. Bailey et al., 2007), speed (e.g. Ingram et al., 2008), and parasympathetic nervous system activity (e.g. Buchheit et al., 2009). Therefore, CWI appears to be an integral component of recovery and preparation during periods of competition.

In addition to teams using CWI to speed up recovery from matches, CWI may be used post-training during periods of physical development such as pre-season in attempt to optimise training adaptation; by potentially allowing greater training loads (increased frequency, intensity or duration) to be performed in subsequent sessions, thus increasing the stimulus for adaptation (Versey et al., 2013). However, despite enhancing physiological recovery for subsequent sessions, the use of CWI to enhance adaptation during these periods may be counterintuitive. For example, it has been suggested that muscle damage and the subsequent inflammatory response is a vital precursor for the signalling mechanisms which initiate the repair and growth of cells (Carlson and Faulkner, 1983), therefore incorporating strategies which attempt to blunt the effects of exercise induced muscle damage may in fact reduce the potential for training adaptation to occur. Indeed research has demonstrated that CWI (Yamane et al., 2006; Frohlich et al., 2014) post-exercise may inhibit the stimulus for adaptive physiology. Therefore, during periods where physical development is of prime importance such as pre-season, the use of recovery strategies such as CWI may in fact impede adaptation.

Currently there is lack of literature examining the effect of CWI on adaptation in elite athletes; therefore best practice recovery protocols during periods of adaptation are unclear. In attempt to understand the role CWI has on adaptive physiology, further research is required to assess the effect of CWI on adaptation to training during periods where physiological adaptation is of prime importance in elite athletes.

4. REFERENCES

Baeche TR, Earle RW and Wathan D. Resistance training. In: *Essentials of strength training and conditioning*. Baeche TR and Earle RW eds. Champaign, IL: Human Kinetics, 407, 2008.

Bailey DM, Erith SJ, Griffin PJ, Dowson A, Brewer DS, Gant N and Williams C. Influence of cold-water immersion on indices of muscle damage following prolonged intermittent shuttle running. *J Sports Sci* 25: 1163-70, 2007.

Beckham G, Mizuguchi S, Carter C, Sato K, Ramsey M, Lamont H, Hornsby G, Haff G and Stone M. Relationships of isometric mid-thigh pull variables to weightlifting performance. *J Sports Med Phys Fitness* 53: 573-81, 2013.

Bemben MG, Clasey JL and Massey BH. The effect of the rate of contraction on the force-time curve parameters of male and female subjects. Res Q Exerc Sport 61: 96-9, 1990.

Buchheit M, Peiffer JJ Abbiss CR and Laursen PB. Effect of cold water immersion on post-exercise parasympatheric reactivation. Am J Physiol Heart Circ Physiol 296: H421-7, 2009.

Carlson BM and Faulkner JA. The regeneration of skeletal muscle fibres following injury: a review. Med Sci Sports Exerc 15: 187-98, 1983.

Cochrane D. Alternating hot and cold water immersion for athlete recovery: a review. *Phys Ther Sport* 5: 26-32, 2004.

Frohlich M, Faude O, Klein M, Peter A, Emrich E and Meyer T. Strength training adaptations after cold water immersion. *J Strength Cond Res* epub ahead of print, 2014.

Haff G, Carlock JM, Hartman MK, Lon Kilgore J, Kawamori N, Jackson JR, Morris RT, Sands WA and Stone MH. Force-time curve characteristics of dynamic and isometric muscle actions of elite women Olympic weightlifters. *J Strength Cond Res* 19: 741-8, 2005.

Ingram J, Dawson B, Goodman C, Wallman K and Beilby J. Effect of water immersion methods on postexercise recovery from simulated team sport exercise. *J Sci Med Sport* 12: 417-21, 2009.

Lazarim FL, Antunes-Neto JM, da Silva FO, Nunes LA, Bassini-Cameron A, Cameron LC, Alves AA, Brenzikofer R and de Macedo DV. The upper values of plasma creatine kinase of professional soccer players during the Brazilian National Championship. *J Sci Med Sport* 12: 85-90, 2009.

Leeder J, Gissane C, van Someren K, Gregson W and Howatson G. Cold water immersion and recovery from strenuous exercise: a meta-analysis. *Br J Sports Med* 46: 233-40, 2012.

McLean BD, Coutts AJ, Kelly V, McGuigan MR and Cormack SJ. Neuromuscular, endocrine, and perceptual fatigue responses during different length between-match microcycles in professional rugby league players. *Int J Sports Physiol and Perform* 5: 367-83, 2010.

Rahmani A, Viale F, Dalleau G and Lacour J-R. Force/velocity and power/velocity relationships in squat exercise. *Eur J Appl Physiol* 84: 227-32, 2001.

Siri WR. Body composition from fluid spaces and density: analysis of methods. In: *Techniques for measuring body composition*. Eds: Brozek J and Henschel A. Nat.Acad.Sci.Washington D.C, 223-44, 1961.

Stone MH, Sands WA, Carlock J, Callan S, Dickie D, Daigle K, Cotton J, Smith SL and Hartman M. The importance of isometric maximum strength and peak rate-of-force development in sprint cycling. J Strength

Cond Res 18: 878-84, 2004.

Vaile J, Halson S, Gill N and Dawson B. Effect of cold water immersion on repeat cycling performance and thermoregulation in the heat. J Sports Sci 26: 431-40, 2008.

Versey NG, Halson SL and Dawson BT. Water immersion recovery for athletes: effect on exercise performance and practical recommendations. *Sports Med* 43: 1101-30, 2013.

West DJ, Finn CV, Cunningham DJ, Shearer DA, Jones MR, Harrington BJ, Crewther BT, Cook CJ and Kilduff LP. Neuromuscular function, hormonal, and mood responses to professional rugby union match. *J Strength Cond Res* 28: 194-200, 2014.

West DJ, Owen NJ, Jones MR, Bracken RM, Cook CJ, Cunningam DJ, Shearer DA, Finn CV, Newton RU, Crewther BT and Kilduff LP. Relationships between force-time characteristics of the isometric midthigh pull and dynamic performance in professional rugby league players. *J Strength Cond Res* 25: 3070-5, 2011.

Wilcock IM, Cronin JB and Hing WA. Physiological response to water immersion: a method for sport recovery? *Sports Med* 36: 747-5, 2006.

Wilmore JH and Behnke MD. An anthropometric estimation of body density and lean body weight in young women. Am J Clin Nutr 23: 267-74, 1970.

Yamane M, Teruya H, Nakano M, Ogai R, Ohnishi N and Kosaka M. Post-exercise leg and forearm flexor muscle cooling in humans attenuates endurance and resistance training effects on muscle performance and on circulatory adaptation. *Eur J Appl Physiol* 96: 572-80, 2006.

5. AIMS and OBJECTIVES

The aim of this study will be to assess the effect of CWI on adaptation to training in professional rugby union players.

Following initial strength testing, individuals from a professional rugby union team will be allocated into two post-training recovery groups for the duration of a five week pre-season training phase. Individuals in one group will be immersed in cold water post-training (CWI) while the other group will not use any post-exercise recovery strategies (control; CON). Following the completion of the training phase, players will be re-tested to establish the effect CWI has on adaptation to strength training in professional rugby union players.

6. METHODOLOGY

6.1. Study Design

Subjects are to be assigned from a male senior professional regional rugby union club (n=22, age 18-35) who compete at European club level and contain several international players. To be included in the study, all players will have been engaged in a structured weight-training program for at least 2 years and will be able to complete an isometric mid-thigh pull (IMTP) with correct technique as assessed by a qualified strength and conditioning coach. Players will be informed of the aims and procedures of the study.

A randomised control trial research design will be used to investigate the effect of CWI on adaptation to strength training during a five week training block during pre-season. Following baseline strength testing, players will be assigned to two post-training groups for the five week period; CWI or CON, with consideration to strength results, playing position, training experience and key lift selection. To establish the efficacy of strength training on adaptation, strength tests will be repeated one week following the completion of the five week training period. All testing, training and recovery sessions are to be conducted and monitored at the team's training facilities.

6.2. Experimental Procedures

To determine baseline maximal strength, IMTP testing will be conducted on the first day of pre-season for the measurement of peak force (PF) and relative peak force (Rel. PF). The following day players will then be required to complete bench press and chin-up 3RM tests as part of their upper body strength session. Body

composition tests will also be conducted prior to baseline testing by an International Society for the Advancement of Kinanthropometry (ISAK) accredited technician.

Following testing, players will be pair-matched into two post-training recovery groups; CWI or a control group with consideration to strength results, playing position, training experience and key lift selection. Pre-season training will follow a weekly pattern for five weeks. Following the final training session of the day, players assigned to the CWI group are to be immersed in cold water to the level of the anterior superior iliac spine at 10-15°C (Comark PDT300; Comark Instruments, Norwich, UK) in a portable ice bath (White Gold Fitness, Bedford, UK). During weeks 1-3 players are to be immersed for 2x5min (Ingram et al., 2009), with 2min between immersions standing out of the bath. For the final two weeks of the training block players are to be immersed for 1x10min (Bailey et al., 2007). Players are permitted a maximum of 2min to shower clean prior to CWI however they are not permitted to return to the shower between or following immersions as this may be counterproductive to the vasoconstrictive effects of CWI (Cochrane, 2004). Players are required to attend for CWI immediately following their final session of the day however they are not restricted to nutritional supplements immediately post-exercise which will be prescribed during this period. Players assigned to the CON group will receive no post-exercise recovery intervention during the training period.

The IMTP testing was carried out with players standing on a portable force platform (type 92866AA, Kistler Instruments Ltd., Farnborough, United Kingdom), positioned on the floor centred underneath the bar of a power rack.

Prior to pre-training IMTP testing, players will be positioned so that they assume a body position similar to that of athletes in the initiation of the second pull of a clean. This position allows athletes to maintain a knee angle of approximately 120-140° as previously used (Haff et al., 2005; Stone et al., 2004). The pull-bar (Keiser UK, Tetbury, Gloucestershire, United Kingdom) can be fixed to various heights above the force platform, to accommodate different sized players. The height at which players pull from will be recorded so that players adopt the same position for post-training testing. The custom made pull-bar will prevent any bending of the bar during the pull and the rack will be anchored to the floor.

Prior to testing players will undertake a standardised warm up which will include 5min dynamic movement followed by sequential 60, 90 and 120kg mid-thigh clean pull or rack pulls (2-3 reps) depending on the players' preference. In preparation for maximal effort testing, one submaximal practice effort will then performed which also allows investigators to ensure starting position can be maintained during testing.

When testing, players are to stand on the force platform, with their hands strapped to the bar using lifting straps. Minimal pre-tension will be allowed to ensure that there is no slack in the subjects' body prior to the initiation of the pull (Beckham et al., 2013). The portable force platform with built-in charge amplifier will be used to measure the vertical component of the ground reaction force (GRF) of the subjects during performance of a maximal effort IMTP. A sample rate of 1,000 Hz and a vertical force range of 20 kN will be used for all trials. The force-time data will be recorded on a portable computer using a 16-bit analog to digital converter. A sample length of 10s will be used for all trials, consisting of a 5s quiet standing phase, and a 5s period when players perform the IMTP following the command to 'go'. The platform's calibration will be checked before and after each testing session. During each trial, subjects will be instructed to pull as hard and as fast as possible for a period of approximately 5s. These commands are based on previous research indicating that the use of these instructions produces optimal results for the attainment of maximal force (Bemben et al., 1990; Rahmani et al., 2001).

During upper body testing, players will be asked to build up to a 3RM on the bench press and the chin-up. Prior to testing players will complete a 5min dynamic warm up and 2-4 sets of 3-6 reps (~35-80% 1RM) of both bench press and chin-ups in preparation for maximal effort testing. When performing the bench press, players are requested to keep their hips on the bench and feet on the floor, and perform a full range of motion (i.e. to complete a repetition the bar is to be lowered to chest and returned to a 'locked-out' arms position). Hand width on the bar performing the bench press will be self-prescribed.

To add resistance to the chin-up, players may add weight to a belt supported around the waist. Using a shoulder width supinated grip, players will lower themselves to hang so that arms are 'locked-out', and pause before being instructed to start the test. Each repetition will be initiated from this position and will be completed once the shoulders reached the line of the bar. Players will be asked to work up to a 3RM however 2 and 4RM efforts will also accepted.
A period of familiarisation will not be required for IMTP, bench press or chin-up testing prior to the initiation of the study, as the players are used to the tests and procedures as part of their normal testing battery.

Calculation of peak force from GRF-time history will be determined using methods previously used in professional rugby league using similar testing procedures (West et al., 2011). The PF will be determined from the vertical component of the GRF-time history, defined as the peak produced during the IMTP minus the subject's body weight, which will be measured prior to testing (Seca 876; Seca, Birmingham, U.K.). Subsequent to the determination of PF, Rel. PF will then be calculated by dividing PF by the subject's body weight.

Calculation of bench Press and chin-up 1RMs from $3RM (\pm 1)$ testing will be determined by use of prediction tables (Baechle and Earle, 2008). Body fat will be measured as the sum of 8 calliper measurements (triceps, subscapular, bicep, iliac crest, supraspinale, abdominal, front thigh and calf; ISAK, 2007) and expressed as total millimetres (mm). Excluding iliac crest, the remaining 7 measures will then be used to provide an estimate of fat mass (Wilmore and Behnke, 1970; Siri, 1961) from which an estimate of fat free mass will be calculated.

6.3. Data Analysis Techniques

Statistical analyses are to be performed using the Statistical Package for the Social Sciences (SPSS for Windows, version 14.0; SPSS, Inc., Chicago, IL, USA). Within-group changes and between-group differences in strength measures are to be analysed using a two-way (group*time) repeated measures analysis of variance (ANOVA), with paired t-tests carried out where relevant.

6.4. Storage and Disposal of Data and Samples

Under the responsibility of the research team, the data collected will be stored during analysis with a password on a secure computer with access available to investigators and supervisors only. Feedback and interpretation of results will be provided to rugby club staff when requested. Data will be disposed of post analysis by the research team but will be retained by rugby club staff for future reference.

7. LOCATION OF THE PREMISES WHERE THE RESEARCH WILL BE CONDUCTED.

Testing will be conducted at the regional rugby training facility (Parc-Y-Scarlets, Pemberton, Llanelli).

8. SUBJECT RISKS AND DISCOMFORTS

Testing will require all individuals to exert maximal effort therefore there may a risk of injury, however all players will be familiar with test protocols and risk of injury should be offset by warm-up protocols established. Furthermore, prior to the study players will be provided with a two week preparatory programme to follow which is designed to re-introduce the exercises used in pre-season in attempt to reduce muscle soreness and injury. However should there be an injury, club staff and doctors will be present for all testing sessions.

Each individual will be required to complete an AHA/ACSM health screening questionnaire to ensure no risk factors for testing exist.

9. INFORMATION SHEET AND INFORMED CONSENT

The submission should be specific about the type of consent that will be sought:

Have you included a Subject Information Sheet for the participants of the study ? YES

Have you included a Subject Consent Form for the participants of the study? YES

10. COMPUTERS

Are computers to be used to store data? YES

If so, is the data registered under the Data Protection Act? YES

11. STUDENT DECLARATION

Please read the following declarations carefully and provide details below of any ways in which your project deviates from them. Having done this, each student listed in section 2 is required to sign where indicated.

- 1. I have ensured that there will be no active deception of participants.
- 2. I have ensured that no data will be personally identifiable.
- 3. I have ensured that no participant should suffer any undue physical or psychological discomfort
- 4. I certify that there will be no administration of potentially harmful drugs, medicines or foodstuffs.
- 5. I will obtain written permission from an appropriate authority before recruiting members of any outside institution as participants.
- 6. I certify that the participants will not experience any potentially unpleasant stimulation or deprivation.
- 7. I certify that any ethical considerations raised by this proposal have been discussed in detail with my supervisor.
- 8. I certify that the above statements are true with the following exception(s):

Student signature: (include a signature for each student in research team)

Date:

12. SUPERVISOR'S DECLARATION

In the supervisor's opinion, this project (delete those that do not apply):

• Raises some ethical issues, but I consider that appropriate steps and precautions have been taken and I have approved the proposal.

Supervisor's signature:

Date:

13. ETHICS COMMITTEE DECISION (COMMITTEE USE ONLY)

ETHICAL APPROVAL: GRANTED REJECTED (delete as appropriate)

The ethical issues raised by this project have been considered by members of the Departmental Ethical Approval Committee who made the following comments:

Please ensure that you take account of these comments and prepare a revised submission that should be shown

to your supervisor/ resubmitted to the Department Ethical Approval Committee (delete as appropriate).

Signed:

Date:

(Chair, Departmental Ethics Advisory Committee)

APPLICATON FOR ETHICAL COMMITTEE APPROVAL OF A RESEARCH PROJECT

In accordance withDepartmental Safety Policy, all research undertaken in the department must be approved by the Departmental Ethic Advisory Committee **prior to** data collection. **Applications for approval should be typewritten on this form using the template available in the Public Folders.** The researcher(s) should complete the form in consultation with the project supervisor. Where appropriate, the application must include the following appendices:

- (A) subject information sheet;
- (B) subject consent form;
- (C) subject health juestionnaire.

After completing ections 1-12 of the form, 1 copy of the form should be handed-in to the Department Administrator who will then submit copies of the application for consideration by the Departmental Ethics Advisory Committee. The applicant(s) will be informed of the decision of the Committee in due course.

1. DRAFT TITLI OF PROJECT

Characterisation of sleep patterns during training and competition periods in professional rugby union

2. NAMES AND STATUS OF RESEARCH TEAM

Rhys Jones (PhD Sudent) Dr. Liam Kilduff (Sipervisor, Swansea University) Dr. David Shearer (Jniversity of Glamorgan) Brad Harrington (H:ad of Athlete Performance, Scarlets Rugby)

3. RATIONALE

Sleep is a basic requirement for human health due to its physiological and psychological restorative effects (Leeder et al., 2012). During sleep, highest concentrations of growth hormones; which play a significant anabolic role in skettal muscle growth via an increase in protein synthesis and reduction in protein breakdown, are observed (Kraemer and Mazzetti 2003). Sleep, and the lack thereof, should therefore be stressed as contributing an important role in the process of muscle recovery (Dattilo et al., 2011). In addition sleep is also an important regulator of central nervous system and cognitive functions (Dattilo et al., 2011).

However, under corditions of sleep deprivation concentrations of anabolic and catabolic hormones are reduced and increased respectively (Dattilo et al., 2011), inflammation increases (Boudjelta et al., 2008), sympathetic nervous system activity is increased (Meerlo et al., 2010), and there are impairments to strength and power production (Edge et al., 2010), muscle glycogen repletion (Edge et al., 2010) and cognitive function (Meerlo et al., 2008).

Sleep is therefore a vital component of an athlete's recovery and preparation. Indeed, although only based on anecdotal evidence, sleep has been reported to be the single most efficacious recovery strategy following exercise in elite athletes (Halson, 2008). However, recent research has found that elite athletes may have poorer sleep quality when compared to age and sex matched controls (Leeder et al., 2012).

In rugby union, sleep deprivation prior to competition could have a negative impact on neuromuscular and cognitive performance. Furthermore, competition has been shown to induce large increases in muscle damage (Gill et al., 2006) and neuromuscular impairment (West et al., *in review*) which may remain for several days. Sleep deprivation following competition would therefore likely exacerbate the recovery process impairing the ability to train and prepare for subsequent performance.

4. **REFERENCES**

Boudjelta, K., Farau, B., Stenuit, P., Esposito, M., Dyzma, M., Brohee, D., Ducobu, J., Vanhaeverbeek, M., Kerkofs, M. Sleep restriction increases white blood cells, mainly neutrophil count, in young healthy men: A

pilot study. Vascular Health and Risk Management, 4: 1467-1470. 2008.

Dattilo, M., Antunes, H., Medeiros, A., Monico Neto, M., Souza, H., Tufik, S., de Mello, M. Sleep and muscle recovery: Endocrinology and molecular basis for a new and promising hypothesis. *Medical Hypotheses*, **77**: 220-222. 2011

Edge. The effects of sleep deprivation on muscle recovery and performance in athletes. Available at www.sportnz.org.nz/documents/research/the_effects_of_sleep_deprivation_on_muscle_recovery_and_performa nce_in_athletes.pdf (Accessed 30th December 2009).

Gill, N., Beaven, C., Cook, C. (2006). Effectiveness of post-match recovery strategies in rugby players. British Journal of Sports Medicine. 40: 260-263. 2006

Halson, S. Nutrition, sleep and recovery. European Journal of Sports Science. 8: 119-126. 2008

Leeder, J., Glaister, M., Pizzoferro, K., Dawson, J., Pedlar, C. Sleep duration and quality in elite athletes measured using wristwatch actigraphy. *Journal of Sports Sciences*. **30**: 541-545. 2012

Kraemer and Mazzetti (2003). Hormonal Mechanisms related to the expression of muscular strength and power. In: Strength and Power in Sport, 2nd Ed. Blackwell:UK

Meerlo, P., Sgoifo, A., Suchecki, D. Restricted and disrupted sleep: Effects on autonomic function, neuroendocrine stress systems and stress resposivity. *Sleep Medicine Reviews*. **12**: 197-210. 2008

West, D., Finn., C., Cunningham, D., Shearer, D., Jones, M., Harrington, B., Crewther, B., Cook, C., Kilduff, L. The neuromuscular function, hormonal and mood responses to a professional rugby union match. *Journal of Strength and Conditioning Research*, in review.

5. AIMS and OBJECTIVES

The study aims to observe the sleeping patterns of professional rugby players. Players will be assessed on different days with respect to preparation and recovery from performance to establish whether sleep is affected by previous or forthcoming exercise.

6. METHODOLOGY

6.1. Study Design

Subjects are to be assigned from a male senior professional regional rugby union club (n=15, age 18-35) who compete at European club level and contain several international players.

The study is to be performed during the 2012-2013 season. Players will be informed of the aims and procedures of the study. The study requires the players to wear an Actiwatch (Cambridge Neurotechnology Ltd. UK) the night following a full days training (weights and two rugby sessions), a rest day, the day before a match and the night following a match. Players will perform this for three home games of similar kick off time to get an average for each night's sleep. The study requires no physical exertion and requires and provided the watch is worn correctly there should be no risks associated with testing.

6.2. Experimental Procedures

On observation nights, players will be required to wear their Actiwatch on the non-dominant wrist from 8pm to the point of getting out of bed the following morning. Watches will be set with an epoch length of 1 minute, collecting data from 8:15pm until 2pm the following day.

Data from each recording period is automatically calculated using the Sleepwatch software (Actiwatch activity and sleep analysis version 5.5, Cambridge Neurotechnology Ltd., UK). Individual night's sleep will be analysed for the following range of variables: time in bed, sleep latency, time asleep, time awake, percent time sleeping while in bed (sleep efficiency), actual sleep percentage, moving minutes, percentage moving time and sleep restlessness (fragmentation index). Players will be given an information sheet which will contain sections for players to note time to bed, time of wake and any other notes which may influence sleep patterns (e.g. napping, illness). Sleep quality will be determined by measures of sleep efficiency and fragmentation index.

6.3. Data Analysis Techniques

Using SPSS software (version 3; SPSS Inc., Chicago, IL.), a repeated measures analysis of variance (ANOVA) will be performed to establish the significance of changes between data collection points.

6.4. Storage and Disposal of Data and Samples

Under the responsibility of the research team, the data collected will be stored during analysis with a password on a secure computer with access available to investigators and supervisors only. Feedback and interpretation of results will be provided to rugby club staff when requested. Data will be disposed of post analysis by the research team but will be retained by rugby club staff for future reference.

7. LOCATION OF THE PREMISES WHERE THE RESEARCH WILL BE CONDUCTED.

Testing will be conducted at the regional rugby training facility (Parc-Y-Scarlets, Pemberton, Llanelli) and at the Exercise Physiology laboratory at Swansea University.

8. SUBJECT RISKS AND DISCOMFORTS

Watches are only to be worn during the evening and will not be worn during any physical exercise. If players are required to perform additional training during the evening they will be informed that they should remove it prior to doing so. Provided the watch is worn correctly there should be no risks associated with wearing the watch during sleep.

9. INFORMATION SHEET AND INFORMED CONSENT

The submission should be specific about the type of consent that will be sought:

Have you included a Subject Information Sheet for the participants of the study ? YES

Have you included a Subject Consent Form for the participants of the study? YES

10. COMPUTERS

Are computers to be used to store data? YES

If so, is the data registered under the Data Protection Act? YES

11. STUDENT DECLARATION

Please read the following declarations carefully and provide details below of any ways in which your project deviates from them. Having done this, each student listed in section 2 is required to sign where indicated.

- 1. I have ensured that there will be no active deception of participants.
- 2. I have ensured that no data will be personally identifiable.
- 3. I have ensured that no participant should suffer any undue physical or psychological discomfort
- 4. I certify that there will be no administration of potentially harmful drugs, medicines or foodstuffs.
- 5. I will obtain written permission from an appropriate authority before recruiting members of any outside institution as participants.
- 6. I certify that the participants will not experience any potentially unpleasant stimulation or deprivation.
- 7. I certify that any ethical considerations raised by this proposal have been discussed in detail with my supervisor.
- 8. I certify that the above statements are true with the following exception(s):

Student signature: (include a signature for each student in research team)

Date:

12. SUPERVISOR'S DECLARATION

 In the supervisor's opinion, this project (delete those that do not apply): Raises some ethical issues, but I consider that appropriate steps and precautions have been taken and I have approved the proposal. 				
Supervisor's signature:		Date:		
13. ETHICS COMMITTEE DECISION (COMMITTEE USE ONLY)				
ETHICAL APPROVAL:	GRANTED	REJECTED	(delete as appropriate)	
The ethical issues raised by this project have been considered by members of the Departmental Ethical Approval Committee who made the following comments:				
		••••••		
		••••••		
Please ensure that you take account of these comments and prepare a revised submission that should be shown to your supervisor/ resubmitted to the Department Ethical Approval Committee (delete as appropriate).				
Signed:		Date:		
(Chair, Departmental Ethics Advisory Committee)				

APPENDIX B

Subject information sheets

SUBJECT INFORMATION SHEET

Date : 12th October 2011

Contact Details:

Rhys Jones 454297@swansea.ac.uk

Dr. Liam Kilduff l.kilduff@swansea.ac.uk

1. Study title

Characterisation of Recovery from Professional Rugby Union

2. Invitation paragraph

We would like to invite you to volunteer for this study that aims to determine the recovery response of professional rugby players following competition in elite rugby union. We would like to thank you for taking the time to read this information sheet and very much hope you choose to take part in what is an exciting study.

3. What is the purpose of this study?

A lack of appropriate recovery following competition can increase physiological and psychological fatigue or injury risk. With the accumulation of training the following week this may compromise preparation for the next game and over time may result in overtraining. It is therefore important that the expected recovery pattern following competition is understood to allow for better planning and management of players.

Furthermore, in order to accelerate post-exercise recovery process, several strategies have been proposed. However, despite their widespread use their effectiveness is unclear, largely due to variations in methods used. To assess the effect recovery strategies have on recovery in rugby union it may be most appropriate to use competition as a model of muscle damage. For us to perform future research investigating the effect of various recovery strategies we need to understand variations in the recovery process following each game.

Monitoring the match characteristics and recovery process from three professional rugby union games will therefore allow us to provide information which will improve your recovery and preparation between games. In addition, it will allow us to understand the recovery process from which we may be able to investigate the use of various recovery strategies which could accelerate your recovery.

4. Why have I been chosen?

As we wish to apply our research to elite rugby union, by taking part you will help us further understand the physiology of the sport improving the validity of our research. You are entirely free to withdraw from the study at any time should you wish to do so, without giving any reason for doing so or without giving prior notification of doing so.

5. What will happen to me if I take part?

You will be required to attend four testing session for each game that is selected for analysis. You will be allocated times to attend for testing the day prior to the game (36hrs pre-game), the day following the game (12hrs post-game) and two days following the game (36hrs post-game). In addition you will be required to perform several tests post-game. Testing will be performed at the training gym at Parc Y Scarlets. With exception to match day you are not allowed to drink, chew gum or brush teeth

30mins prior to saliva sampling. You must only train at scheduled sessions and are not allowed to perform any activity on the day prior to pre-competition testing. On each testing occasion you must attend with in training shorts and t-shirt and wearing the same footwear. Alcohol is strictly prohibited 48hrs prior and throughout the testing period.

On non-match days you will follow the same protocol. After going through your standard monitoring procedures on arrival (i.e. bodyweight and hydration) you are required to make your way to the gym for your allocated testing time.

On arrival you must first wear a heart rate monitor for 5mins whilst lying to provide a measure of heart rate variability. You will then be required to provide a saliva sample for hormone analysis by placing a small swab underneath your tongue for 3mins. While waiting for the swab to become saturated by saliva, you will complete a modified profile of mood states (POMS) questionnaire.

Following saliva collection players you will then be required to provide a capillary blood sample.

You will then undergo a 5 minute dynamic warm up, followed by 3 practice countermovement jumps. Following warm up you will be reminded of the procedures before performing 2 maximal single countermovement jumps and 2 maximal drop jumps from a 40cm box.

On match day you will be required to provide a saliva sample and complete a POMS questionnaire prior to the game. Immediately following the game you will be required to provide a blood sample and perform jump tests.

6. What are the possible disadvantages of taking part?

With the exception to the fingertip blood sample, the tests performed will be non-invasive and have low muscle fatigue risk therefore there is a low risk in taking part. A warm up will be conducted prior to any tests to further reduce any possibility of injury.

7. What are the possible benefits of taking part?

As well as providing feedback of multiple force plate measures to your club (e.g. rate of force development, power), the research taken from the test results will enable us to further understand the physiology of rugby which will educate staff on the recovery process in rugby union which will allow more effective monitoring, recovery and preparation of players. The findings from the study may also allow us to plan further studies determining the efficacy of several interventions to help enhance your recovery and preparation from competition/training.

8. Will my taking part in the study be kept confidential?

Yes. All data will be kept confidential and will be accessible only by the research team, your club staff and yourself during and after the study is completed.

SUBJECT INFORMATION SHEET

Date : 24th May 2013

Contact Details:

Rhys Jones <u>454297@swansea.ac.uk</u> PhD Swansea University and Scarlets Rugby

Dr. Liam Kilduff <u>l.kilduff@swansea.ac.uk</u> Swansea University

1. Study title

Does cold water immersion impair adaptation to strength training in professional rugby union players?

2. Invitation paragraph

We would like to invite you to volunteer for this study that aims to establish the effect cold water immersion (CWI) may have on adaptation to strength training. We would like to thank you for taking the time to read this information sheet and very much hope you choose to take part in what is an exciting study.

3. What is the purpose of this study?

Cold water immersion (CWI) is commonly used in elite sport due to the common belief that it may enhance recovery from exercise and therefore improve preparation for further exercise. Indeed, research demonstrates that CWI may enhance recovery of function and reduce muscle soreness; therefore CWI appears to be an important component of recovery during concentrated periods of training such as pre-season.

Despite CWI potentially allowing greater training volume to be performed, recent research suggests that the use of CWI during periods of training may actually impair adaptation which would therefore limit training gains. However, to date no research has been performed in elite athletes. Therefore the study aims to establish the effect CWI may have on adaptation to strength training during pre-season.

4. Why have I been chosen?

As we wish to apply our research to elite rugby union, by taking part you will help us further understand the physiology of the sport improving the validity of our research. You are entirely free to withdraw from the study at any time should you wish to do so, without giving any reason for doing so or without giving prior notification of doing so.

5. What will happen to me if I take part?

The study will be based around your training so there will be no training or testing requirements outside of your pre-season schedule. However, during the initial five week period of pre-season you will be allocated to either a CWI or a control group (CON). The CWI group will be immersed in cold water to the level of the top of the hip for a total of 10mins immediately following the final session of the day while the CON group will receive no post-exercise recovery strategies during the training period.

6. What are the possible disadvantages of taking part?

The only disadvantage to taking part in the study is the potential discomfort from cold water

immersion which will be monitored to stay between 10-15°C.

7. What are the possible benefits of taking part?

The research conducted will greatly further understanding of how CWI affects recovery and adaptation. This information may be used to improve the provision of post-recovery strategies which will help enhance your recovery and adaptation to training.

8. Will my taking part in the study be kept confidential?

Yes. All data will be kept confidential and will be accessible only by the research team, your club staff and yourself during and after the study is completed.

SUBJECT INFORMATION SHEET

Date : 24th September 2012

Contact Details:

Rhys Jones <u>454297@swansea.ac.uk</u> PhD Swansea University and Scarlets Rugby

Dr. Liam Kilduff <u>l.kilduff@swansea.ac.uk</u> Swansea University

1. Study title

Characterisation of sleep patterns during training and competition periods in professional rugby union

2. Invitation paragraph

We would like to invite you to volunteer for this study that aims to characterise sleep patterns of elite rugby union players. We would like to thank you for taking the time to read this information sheet and very much hope you choose to take part in what is an exciting study.

3. What is the purpose of this study?

Sleep promotes muscle repair and growth, and central nervous system and cognitive function restoration. Following training and competition sleep therefore plays a vital role in your recovery and preparation for training and your next game. However, under conditions when sleep quality is reduced cognitive function and strength and power may be reduced.

Last season's research showed some players may take several days to recover following a game. If players are suffering from impaired sleep following a game this may increase the time it takes to recover. Not only will this impair the ability to train, but with the accumulation of training the following week this may compromise preparation for the next game. Furthermore if sleep is impaired during the training week, in particular the night prior to playing, this will impact performance.

Evidence exists to suggest that elite athletes may have poorer sleep quality than non-elite athletes. By monitoring your sleep characteristics in the week prior to a match and in the days following, it will allow us to establish the sleep patterns of elite rugby players and establish whether sleep varies according to previous or forthcoming exercise. This may provide information which will allow us to try and enhance sleep quality; thus enhance individual recovery and preparation.

4. Why have I been chosen?

As we wish to apply our research to elite rugby union, by taking part you will help us further understand the physiology of the sport improving the validity of our research. You are entirely free to withdraw from the study at any time should you wish to do so, without giving any reason for doing so or without giving prior notification of doing so.

5. What will happen to me if I take part?

You will be required to where a sleep monitor watch on five nights, during three separate weeks. You will be required to wear the watch from 8pm until your rise from bed the following morning. You will be informed the weeks of testing and nights of testing, but we will test the following nights; the night following a full days training (weights and two rugby sessions), a rest day, the day before a match and the night following a match. For each weeks testing, you will be allocated a monitor and a separate

sheet which will provide you with sections for you to fill in time to bed and time out of bed, and any notes which may have affected your sleep (e.g. napping, alcohol, illness). You will then be required to hand in your monitor and sheet the first day back in training the following week.

6. What are the possible disadvantages of taking part?

The testing is non-invasive and requires little effort. The watches are fairly discrete and waterproof so shouldn't affect your evening; however we ask if you could remove the watch when exercising to reduce the likelihood of damage.

7. What are the possible benefits of taking part?

The research taken will enable us to further understand your sleep patterns and may allow more effective monitoring, recovery and preparation of players. The findings from the study may also allow us to plan further studies in particular assessing the efficacy of several interventions to help enhance sleep quality.

8. Will my taking part in the study be kept confidential?

Yes. All data will be kept confidential and will be accessible only by the research team, your club staff and yourself during and after the study is completed.

APPENDIX C

Raw data

REFERENCES

- 1. Abbiss CR and Laursen PB. Models to explain fatigue during prolonged endurance cycling. *Sports Med* 35: 865-98, 2005.
- Abedelmalek S, Souissi N, Chtourou H, Denguezli M, Aouichaoui C, Ajina M, Aloui A, Dogui M, Haddouck S and Tabka Z. Effects of partial sleep deprivation on proinflammatory cytokines growth hormone, and steroid hormone concentrations during repeated brief sprint interval exercise. *Chronobiol Int* 30:502-9, 2013.
- Akenhead R, Hayes PR, Thompson KG and French D. Dimunutions of acceleration and deceleration output during professional football match play. J Sci Med Sport 16: 556-61, 2013.
- Al Haddad H, Laursen PB, Ahmaidi S and Buchheit M. Influence of cold water face immersion on post-exercise parasympathetic reactivation. *Eur J Appl Physiol* 108: 599-606, 2010.
- 5. Al Haddad H, Parouty J and Buchheit M. Effect of daily cold water immersion on heart rate variability and subjective ratings of well-being in highly trained swimmers. *Int J Sports Physiol Perform* 7: 33-8, 2012.
- 6. Ali A and Farrally M. A computer-video aided time motion analysis technique for match analysis. J Sports Med Phys Fitness 31: 82-8, 1991.
- 7. Allen DG, Lamb GD, and Westerblad H. Skeletal muscle fatigue: cellular mechanisms. *Physiol Rev* 88: 287-332, 2008.
- Amsler F and Blattler W. Compression therapy for occupational leg symptoms and chronic venous disorders - a meta-analysis of randomised controlled trials. *Eur J Vasc Endovasc* 35: 366-72, 2008.
- 9. Ancoli-Israel S, Cole R, Alessi C, Chambers M, Moorcroft W and Pollak CP. The role of actigraphy in the study of sleep and circadian rhythms. *Sleep* 26: 342-92, 2003.
- 10. Argus CK. Performance effects of wearing compression garments (Skins) during exercise and recovery. *Unknown*, unknown.
- Argus CK, Gill ND, Keogh JW, Hopkins WG and Beaven CM. Changes in strength, power, and steroid hormones during a professional rugby union competition. J Strength Cond Res 23: 1583-92, 2009.

- Armstrong RB. Mechanisms of exercise-induced delayed onset muscular soreness: a brief review. *Med Sci Sport Exer* 16: 529-38, 1984.
- Arnold L, Henry A, Poron F, Baba-Amer Y, van Rooijen N, Plonquet A, Gherardi RK and Chazaud B. Inflammatory monocytes recruited after skeletal muscle injury switch into antiinflammatory macrophages to support myogenesis. *J Exp Med* 204: 1057-69, 2007.
- 14. Aughey RJ. Applications of GPS technologies to field sports. Int J Sports Physiol Perform 6: 295-310, 2011a.
- 15. Aughey RJ. Increased high-intensity activity in elite Australian football finals matches. Int J Sports Physiol Perform 6: 367-79, 2011b.
- Aughey RJ and Falloon C. Real-time versus post-game GPS data in team sports. J Sci Med Sport 13: 348-9, 2010.
- 17. Austin D, Gabbett T and Jenkins D. Repeated high-intensity exercise in professional rugby union. *J Sports Sci* 29: 1105-12, 2011a.
- Austin D, Gabbett T and Jenkins D. The physical demands of Super 14 rugby union. J Sci Med Sport 14: 259-63, 2011b.
- 19. Austin DJ and Kelly SJ. Positional differences in professional rugby league match play through the use of global positioning systems. *J Strength Cond Res* 27: 14-9, 2013.
- 20. Avela J, Kyrolainen H, Komi PV and Rama D. Reduced reflex sensitivity persists several days after long-lasting stretch-shortening cycle exercise. *J Appl Physiol* 86: 1292-300, 1999.
- 21. Baeche TR, Earle RW and Wathan D. Resistance training. In: Essentials of strength training and conditioning. Baeche TR and Earle RW eds. Champaign, IL: Human Kinetics, 407, 2008.
- 22. Bailey DM, Erith SJ, Griffin PJ, Dowson A, Brewer DS, Gant N and Williams C. Influence of cold-water immersion on indices of muscle damage following prolonged intermittent shuttle running. *J Sports Sci* 25: 1163-70, 2007.
- 23. Balson PD, Gaitanos GC, Soderlund K and Ekblom B. High-intensity exercise and muscle glycogen availability in humans. *Acta Physiol Scand* 165: 337-45, 1999.

- 24. Bambaeichi E, Reilly T, Cable NT and Giacomoni M. Influence of time of day and partial sleep loss on muscle strength in eumenorrheic females. *Ergonomics* 48:1499-511, 2005.
- 25. Banfi G, Melegati G, Barassi A, Dogliotti G, d'Eril GM, Dugue B ad Corsi MM. Effects of whole-body cryotherapy on serum mediators of inflammation andserum muscle enzymes in athletes. *J Therm Biol* 34: 55-9, 2009.
- 26. Barbero-Alvarez JC, Coutts A, Granda J, Barbero-Alvarez V and Castagna C. The validity and reliability of a global positioning satellite system device to assess speed and repeated sprint ability (RSA) in athletes. *J Sci Med Sport* 13: 232-35, 2010.
- 27. Barnett A. Using recovery modalities between training sessions in elite athletes: does it help? *Sports Med* 36: 781-96, 2006.
- Barris S and Button C. A review of vision-based motion analysis in sport. Sports Med 38: 1025-43, 2008.
- 29. Beaven CM, Cook CJ, Gray D, Downes P, Murphy I, Drawer S, Ingram JR, Kilduff LP and Gill N. Electrostimulation's enhancement of recovery during a rugby preseason. *Int J Sports Physiol Perform* 8: 92-8, 2013.
- Beaven CM, Cook CJ, Kilduff L, Drawer S and Gill N. Intermittent lower-limb occlusion enhances recovery after strenuous exercise. *Appl Physiol Nutr Metab* 37: 1132-9, 2012.
- 31. Beckham G, Mizuguchi S, Carter C, Sato K, Ramsey M, Lamont H, Hornsby G, Haff G and Stone M. Relationships of isometric mid-thigh pull variables to weightlifting performance. *J Sports Med Phys Fitness* 53: 573-81, 2013.
- 32. Belenky G, Wesensten N, Thorne D, Thomas ML, Sing HC, Redmond DP, Russo MB, Balkin TJ. Patterns of performance degradation and restoration during sleep restriction and subsequent recovery: a sleep dose-response study. J Sleep Res 12:1-12, 2003.
- Bemben MG, Clasey JL and Massey BH. The effect of the rate of contraction on the force-time curve parameters of male and female subjects. *Res Q Exerc Sport* 61: 96-9, 1990.

- 34. Bigland-Ritchie BR, Dawson NJ, Johansson RS and Lippold OC. Reflex origin for the slowing of motoneurone firing rates in fatigue of human voluntary contractions. J Physiol 379: 451-9, 1986.
- 35. Bird S. Sleep, recovery, and athletic performance: A brief review and recommendations. *Str Cond J* 35: 43-7, 2013.
- 36. Bloomer RJ. The role of nutritional supplements in the prevention and treatment of resistance exercise-induced skeletal muscle injury. *Sports Med* 37: 519-32, 2007.
- 37. Bonifazi M, Ginanneschi F, Della Volpe R and Rossi A. Effects of gonadal steroids on the input-output relationship of the corticospinal pathway in humans. *Brain Res* 1011: 187-94, 2004.
- Bonnet MH and Arand DL. Caffeine use as a model of acute and chronic insomnia. Sleep 15: 526-36, 1992.
- Boyd LJ, Ball K and Aughey RJ. The reliability of MinimaxX accelerometers for measuring physical activity in Australian football. Int J Sports Phys Perform 6: 311-21, 2011.
- 40. Bradley PS, Carling C, Diaz AG, Hood P, Barnes C, Ade J, Boddy M, Krustrup P and Mohr M. Match performance and physical capacity of players in the top three competitive standards of English professional soccer. *Hum Mov Sci* 32: 808-21, 2013.
- 41. Brandenberger G and Weibel L. The 24h growth hormone rhythm in men: sleep and circadian influences questioned. *J Sleep Res* 13: 251-5, 2004.
- 42. Bravo L. Polyphenols: chemistry, dierary sources, metabolism, and nutritional significance. *Nutr Rev* 56: 317-33, 1998.
- 43. Brophy-Williams N Landers G and Wallman K. Effect of immediate and delayed cold water immersion after a high intensity exercise session on subsequent run performance. *J Sports Sci Med* 10: 665-70, 2011.
- 44. Buchheit M, Al Haddad H, Simpson BM, Palazzi D, Bourdon PC, Di Salvo V, Mendez-Villanueva A. Monitoring accelerations with GPS in football: time to slow down? Int J Sports Phys Perform 9: 442-5, 2014.

- 45. Buchheit M, Peiffer JJ Abbiss CR and Laurser PB. Effect of cold water immersion on post-exercise parasympatheric reactivation. *Im J Physiol Heart Circ Physiol* 296: H421-7, 2009.
- 46. Buchheit M, Laursen PB and Ahmaidi S. Paras mpathetic reactivation after repeated sprint exercise. *Am J Physiol* 293:H133-41, 200'.
- 47. Burgess HJ, Trinder J, Kim Y and Luke D. Sleep and circadian influences on cardiac autonomic nervous system activity. *Am J Physid* 273: H1761-8, 1997.
- 48. Butterfield TA, Best TM and Merrick MA. The dual roles of neutrophils and macrophages in inflammation: a critical balancebetween tissue damage and repair. J Athl Training 41: 457-65, 2006.
- 49. Buysse DJ, Reynolds CF 3rd, Monk TH, Berman SR and Kupfer DJ. The Pittsburgh Sleep Quality Index: a new instrument fr psychiatric practice and research. *Psychiatry Res* 28: 193-213, 1989.
- 50. Byrnes WC and Clarkson PM. Delayed onset muscle soreness and training. *Clin Sports Med* 5: 605-14, 1986.
- 51. Cady EB, Jones DA, Lynn J and Newham DJ Changes in force and intracellular metabolites during fatigue of human skeletal mu:cle. *J Physiol* 418: 315-25, 1989.
- 52. Cahill N, Lamb K, Worsfold P, Headey F and Murray S. The movement characteristics of English Premiership rugby union players. *J Sports Sci* 31: 229-37, 2013.
- 53. Carlson BM and Faulkner JA. The regeneration of skeletal muscle fibres following injury: a review. *Med Sci Sports Exerc* 15: 187-93, 1983.
- 54. Carroll CC, Dickinson JM, LeMoine JK, Haus JM, Weinheimer EM, Hollon CJ, Aagaard P, Magnusson SP and Trappe TA. Influence of acetaminophen and ibuprofen on in vivo patellar tendon adaptations to knee extensor resistance exercise in older adults. *J Appl Phys* 111: 508-515, 2011.
- 55. Castellano J, Casamichana D, Calleja-Gonzalez J, Roman JS and Ostojic SM. Reliability and accuracy of 10Hz GPS devices for short distance exercise. *J Sports Sci Med* 10: 233-4, 2011.

- 56. Chen TC, Nosaka K, Lin MJ, Chen HL and Wu CJ. Changes in running economy at different intensities following downhill running. *J Sport Sci* 27: 1137-44, 2009.
- 57. Chen JL, Yeh DP, Lee JP, Chen CY, Huang CY, Lee SD, Chen CC, Kuo TB, Kao CL and Kuo CH. Parasympathetic nervous activity mirrors recovery status in weightlifting performance after training. *J Strength Cond Res* 25: 1546-52, 2011.
- 58. Cheung K, Hume P and Maxwell L. Delayed onset muscle soreness : treatment strategies and performance factors. *Sports Med* 33: 145-64, 2003.
- 59. Christian LM, Graham JE, Padgett DA, Glaser R and Kiecolt-Glaser JK. Stress and would healing. *Neuroimmunomodulation* 13: 337-46, 2006.
- 60. Chwalbinska-Moneta J. Influence of whole-body cryotherapy on results of selected exercse response. *Sport Wycz* 5-6:461-62, 2003.
- 61. Clarkson PM and Hubal MJ. Exercise-induced muscle damage in humans. Am J Phys Med Rehab 81: S52-69, 2002.
- 62. Cochrane D. Alternating hot and cold water immersion for athlete recovery: a review. *Phys Ther Sport* 5: 26-32, 2004.
- 63. Cohen S, Doyle WJ, Alper CM, Janicki-Deverts D and Turner RB. Sleep habits and susceptibility to the common cold. *Arch Intern Med* 169: 62-7, 2009.
- 64. Connolly DA, McHugh MP, Padilla-Zakour OI, Carlson L and Sayers SP. Efficacy of a tart cherry juice blend in preventing the symptoms of muscle damage. Br J Sports Med 40: 679-83, 2006.
- 65. Cook CJ and Beaven CM. Individual perception of recovery is related to subsequent sprint performance. *Br J Sports Med* 11: 705-9, 2013.
- 66. Cook CJ and Crewther BT. Changes in salivary testosterone concentrations and subsequent voluntary squat performance following the presentation of short video clips. *Horm Behav* 61: 17-22, 2012.
- 67. Cook CJ, Crewther BT, Kilduff LP, Drawer S and Gaviglio CM. Skill execution and sleep deprivation: effects of acute caffeine or creatine supplementation a randomised placebo-controlled trial. J Int Soc Sports Nutr 8:2, 2011.

- 68. Cook CJ, Kilduf? LP and Jones MR. Optmising athlete recovery. In: *High-performance training for sports*. Joyce, D and Lewindon D, eds. Oxford, Taylor and Francis, in press.
- 69. Cormack SJ. Neuromuscular fatigue and encocrine responses in elite Australian rules football players. PhD thesis, Edith CowanUniversity, W. Australia, 2008.
- 70. Cormack SJ, Mooney MG, Morgan W and McGuigan MR. Influence of neuromuscular fatigue on accelerometer load inelite Australian football players. Int J Sports Physiol Perform 8: 373-8, 2013.
- Cormack SJ, Newton RU, and McGuigan MR. Neuromuscular and endocrine responses of elite players to an Australian rule: football match. *Int J Sports Physiol* 3: 359-74, 2008.
- 72. Cote DJ, Prentic WE, Hooker DN and Shields EW. Comparison of three treatment procedures for minimising ankle sprain swelling *Physical Therapy* 68: 1072-6, 1988.
- Cote KA, McCormick CM, Geniole SN, Renn RP, MacAulay SD. Sleep deprivation lowers aggression and testosterone in men. *Biol Psychol* 92: 249-56, 2013.
- 74. Cote KA, Milner CE, Osip SL, Ray LB and Baxter KD. Walking quantitative electroencephalogram and auditory event-related potentials following experimentally induced sleep fragmentation. *Sleep* 26: 687-94, 2003.
- 75. Coughlan GF, Green BS, Pook PT, Toolan E and O'Connor SP. Physical game demands in elite rugby union: a global positioning system analysis and possible implications for rehabilitation. *J Orthop Sports Phys Ther* 41: 600-5, 2011.
- 76. Coutts AJ and Duffield R. Validity and reliability of GPS devices for measuring movement demands of team sports. *J Sci Med Sport* 13: 133-5, 2010.
- 77. Crewther BT and Cook CJ. Effects of different post-match recovery interventions on subsequent athlete hormonal state and game performance. *Physiol Behav* 106: 471-75, 2012a.
- 78. Crewther BT and Cook CJ. The effects of different pre-game motivational interventions on athlete free hormonal state and subsequent performance in professional rugby union matches. *Physiol Behav* 106: 683-8, 2012b.

- 79. Crewther BT, Kilduff LP, Cook CJ, Cunninghan DJ, Bunce P, Bracken RM and Gaviglio CM. Relationships between salivary free testosterone and the epression of force and power in elite athletes. J Sports Med Phys Fitness 52: 221-7, 2012.
- Crowe MJ, O'Connor D and Rudd D. Cold vater recovery reduces anaerobic performance. *Int J Sports Med* 28: 994-8, 2007
- Cunniffe B, Hore AJ, Whitcombe DM. Time course of changes in immuneoendocrine markers following an international rugby game. *Eur J Appl Physiol* 108: 113-22, 2010.
- 82. Cunniffe B, Proctor W, Baker JS and Davies B. An evaluation of the physiological demands of elite rugby union using Global Positioning System tracking software. J Strength Cond Res 23: 1195-203, 2009.
- Curl CL, Delbridge LM, Canny BJ and Wendt IR. Testosterone modulates cardiomyocyte Ca(2+) handingling and contactile function. *Physiol Rev* 58: 293-97, 2009.
- 84. Dattilo M, Antunes HKM, Medeiros A, Monico Neto M, Souza HS, Tufik S and de Mello MT. Sleep and muscle recovery: endorcrinological and molecular basis for a new and promising hypothesis. *Med Hypotheses* 77: 220-2, 2011.
- 85. Dawson B, Cow S, Modra S, Bishop D and Stewart G. Effects of immediate postgame recovery procedures on muscle soreness, power and flexiblity levels over the next 48 hours. *J Sci Med Sport* 8: 210-21, 2005.
- 86. Davies RC, Eston RG, Poole DC, Rowlands AV, DiMenna F, Wilkerson DP, Twist C and Jones AM. Effect of eccentric exercise-induced muscle damage on the dynamics of muscle oxygenation and pulmonary oxygen uptake. J Appl Physiol 105: 1413-21, 2008.
- Davies V, Thompson KG and Cooper SM. The effects of compression garments on recovery. J Strength Cond Res 23: 1786-94, 2009.
- 88. DeGroot M, Massie BM, Boska M, Gober J, Miller RG and Weiner MW. Dissociation of [H⁺] from fatigue in human muscle detected by high time resolution 31P-NMR. *Muscle Nerve* 16: 91-8, 1993.

- 89. Del Coso J, Fernandez D, Albian-Vicen J, Salinero JJ, Gonzalez-Millan C, Areces F, Ruiz D, Gallo C, Calleja-Gonzalez J and Perez-Gonzalez B. Running pace decrease during a marathon is positively related to blood markers of muscle damage. *PLoS One* 8: e57602, 2013.
- 90. Deutsch MU, Kearney GA and Rehrer NJ. Time-motion analysis of professional rugby union players during match-play. *J Sports Sci* 15: 461-72, 2007.
- 91. Deutsch MU, Maw GJ, Jenkins D and Reaburn P. Heart rate, blood lactate and kinematic data of elite colts (under-19) rugby union players during competition. J Sports Sci 16: 561-70, 1998.
- 92. di Prampero PE, Fusi S, Sepulcri L, Morin JB, Belli A and Antonutto G. Sprint running: a new energetic approach. *J Exp Biol* 208: 2809-16, 2005.
- 93. Dobson BP and Keogh JW. Methodological issues for the application of time motion analysis research. *Strength Cond J* 29: 48-55, 2007.
- 94. Dodd S, Powers SK, Callender T and Brooks E. Blood lactate disappearance at various intensities of recovery exercise. *J Appl Physiol* 57: 1462-5, 1984.
- 95. Dogramaci SN, Watsford ML and Murphy AJ. The reliability and validity of subjective notational analysis in comparison to global positioning system tracking to assess athlete movement patterns. *J Strength Cond Res* 25: 852-59, 2011.
- 96. Donnelly AE, Maughan RJ and Whiting PH. Effects of ibuprofen on exerciseinduced muscle soreness and indices of muscle damage. Br J Sports Med 24: 191-5, 1990.
- 97. Doran SM, Van Dongen HP and Dinges DF. Sustained attention performance during sleep deprivation: evidence of state instability. *Arch Ital Biol* 139: 253-67, 2001.
- 98. Driver HS and Taylor SR. Exercise and Sleep. Sleep Med Rev 4: 387-402, 2000.
- 99. Drust B, Waterhouse J, Atkinson G, Edwards B and Reilly T. Circadian rhythms in sports performance an update. *Chronobiol Int* 22: 21-44, 2005.
- 100. Duffield R, Cannon J and King M. The effects of compression garments on recovery of muscle performance following high-intensity sprint and plyometric exercise. J Sci Med Sport 13: 136-40, 2010a.

- 101. Duffield R, Murphy A, Kellett A and Reid M. Recovery from repeated on-court tennis sessions: combining cold water immersion, compression and sleep recovery interventions. *Int J Sports Physiol Perform* 9:273-82, 2014
- 102. Duffield R, Murphy A, Snape A, Minett GM and Skein M. Post-match changes in neuromuscular function and the relationship to match demands in amateur rugby league matches. *J Sci Med Sport* 15: 238-43, 2012.
- 103. Duffield R and Portus M. Comparison of three types of full-body compression garments on throwing and repeat-sprint performance in cricket players. Br J Sport Med 41: 409-14, 2007.
- 104. Duffield R, Reid M, Baker J and Spratford W. Accuracy and reliability of GPS devices for measurement of movement patterns in confined spaces for court-based sports. *J Sci Med Sport* 13: 523-5, 2010b.
- 105. Duthie GM, Pyne D and Hooper S. The applied physiology and game analysis of rugby union. *Sports Medicine* 33: 973-91, 2003a.
- 106. Duthie GM, Pyne D and Hooper S. The reliability of video based time motion analysis. J Hum Movement Stud 44: 259-72, 2003b.
- 107. Duthie G, Pyne D and Hooper S. Time motion analysis of 2001 and 2002 super 12 rugby. *J Sports Sci* 23: 523-30, 2005.
- 108. Dwyer DB and Gabbett TJ. Global positioning system data analysis: velocity ranges and a new definition of sprinting for field sport athletes. *J Strength Cond Res* 26: 818-24, 2012.
- 109. Eaton C and George K. Position specific rehabilitation for rugby union players. Part1: Empirical movement analysis data. *Phys Ther Sport* 7: 22-9, 2006.
- Ebbeling CB and Clarkson PM. Exercise-induced muscle damage and adaptation. Sports Med 7: 207-34, 1989.
- 111. Edge J, Mundel T and Short MJ. The effects of sleep deprivation on muscle recovery and performance in athletes.Available at: http://www.sportnz.org.nz/Documents/Research/The_effects_of_sleep_deprivation_o n_muscle_recovery_and_performance_in_athletes.pdf, 2010.

- 112. Eissen A, Fisman EZ, Rubenfire M, Freimark D, McKechnie R, Tenebaum A, Motro M and Adler Y. Ischemic preconditioning: nearly two decades of research. A comprehenesive review. *Atherosclerosis* 172: 201-10, 2004.
- 113. Ekstedt M, Akerstedt T and Soderstrom M. Microarousals during sleep are associated with increased levels of lipids, cortisol, and blood pressure. *Psychosom Med* 66: 925-31, 2004.
- 114. Elias M. Serum cortisol, testosterone, and testosterone-binding globulin responses to competitive fighting in human males. *Aggress Behav* 7: 215-24, 1981.
- 115. Elloumi M, Maso F, Michaux O, Robert A and Lac G. Behaviour of saliva cortisol (C), testosterone (T) and the T/C ratio during a rugby match and during the postcompetition recovery days. *Eur J Appl Physiol* 90: 23-8, 2003.
- 116. Erdmann WS. Gathering of kinematic data of sport event by televising the whole pitch and track. In: *Proceedings of 10th ISBS symposium*. Rodano R, ed. Milano, Italy: International Society of Biomechanics in Sports. 1992: 159-62.
- 117. Eston R and Peters D. Effects of cold water immersion on the symptoms of exercise-induced muscle damage. J Sport Sci 17: 231-38, 1999.
- 118. Estrada M, Espinosa A, Muller M and Jaimovich E. Testosterone stimulates intracellular calcium release and mitogen-activated protein kinases via a G protein-coupled receptor in skeletal muscle cells. *Endocrinology* 144: 3586-97, 2003.
- 119. Everson CA and Crowley WR. Reductions in circulating anabolic hormones induced by sustained sleep deprivation in rats. *Am J Physiol Endocrinol Metab* 286: E1060-70, 2004.
- 120. Fatouros IG, Chatznikolaou A, Douroudos II, Nikolaidis MG, Kypargos A, Margonis K, Michailidis Y, Vantarakis A, Taxildaris K, Katrabasas I, Mandalidis D, Kouretas D and Jamurtas AZ. Time-course of changes in oxidative stress and antioxidant status responses following a soccer game. J Strength Cond Res 24: 3278-87, 2010.
- 121. Feige B, Gann H, Brueck R, Hornyak M, Litsch S, Hohagen F and Riemann D. Effects of alcohol on polysomnographically recorded sleep in healthy subjects. *Alcohol Clin Exp Res* 30:1527-37, 2006.

- 122. Field A. Exploring data. In: *Discovering statistics using SPSS*. Field A. 2nd ed., Oxford, The Alden Press, 2005.
- 123. Fietze I, Strauch J, Holzhausen M, Glos M, Theobald C, Lehnkering H and Penzel T. Sleep quality in professional ballet dancers. *Chronobiol Int* 26: 1249-62, 2009.
- 124. Figueiro MG, Wood B, Plitnick B and Rea MS. The impact of light from computer monitors on melatonin levels in college students. *Neuro Endocrinol Lett* 32: 158-63, 2011.
- 125. Finberg M, Braham R, Goodman C, Gregory P and Peeling P. Effects of electrostimulation therapy on recovery from acute team sport activity. *Int J Sports Physiol Perform* 8: 293-9, 2013.
- 126. Fischer CP, Hiscock NJ, Penkowa M, Basu S, Vessby B, Kallner A, Sjoberg LB and Pedersen BK. Supplementation with vitamins C and E inhibits the release of interleukin-6 from contracting human skeletal muscle. *J Physiol* 558: 633-45, 2004.
- 127. Frohlich M, Faude O, Klein M, Peter A, Emrich E and Meyer T. Strength training adaptations after cold water immersion. *J Strength Cond Res* epub ahead of print, 2014.
- 128. Fry AC, Schilling BK, Fleck SJ, Kraemer WJ. Relationships between competitive wrestling success and neuroendocrine responses. J Strength Cond Res 25: 40-5, 2011.
- 129. Gabbett TJ. GPS analysis of elite women's field hockey training and competition. J Strength Cond 24: 1321-4, 2010.
- 130. Gabbett TJ. Influence of playing standard on the physical demands of professional rugby league. *J Sports Sci* 10: 1125-38, 2013a.
- 131. Gabbett TJ. Influence of the opposing team on the physical demands of elite rugby league match play. *J Strength Cond Res* 27: 1629-35, 2013b.
- 132. Gabbett TJ. Physical, technical and tactical factors affect final ladder position in semi-professional rugby league. Int J Sports Physiol Perform epub ahead of print, 2013c.
- 133. Gabbett TJ, Jenkins DG and Abernethy B. Physical collisions and injury during professional rugby league skills training. *J Sci Med Sport* 13: 578-83, 2010.

- 134. Gabbett TJ, Jenkins DG and Abernethy B. Physical demands of professional rugby league training and competition using microtechnology. J Sci Med Sport 15: 80-6, 2012.
- 135. Gabbett TJ, Jenkins DG and Abernethy B. Relative importance of physiological, anthropometric, and skill qualities to team selection in professional rugby league. J Sports Sci 29: 1453-61, 2011.
- 136. Gabbett TJ, Polley C, Dwyer DB, Kearney S and Corvo A. Influence of field position and phase of play on the physical demands of match-play in professional rugby league forwards. *J Sci Med Sport* epub ahead of print, 2013.
- 137. Gabbett TJ and Seibold AJ. Relationships between tests of physical qualities, team selection, and physical match performance in semiprofessional rugby league players. J Strength Cond Res 27: 3259-65, 2013.
- 138. Gabbett TJ and Ullah S. Relationship between running loads and soft-tissue injury in elite team sport athletes. *J Strength Cond Res* 26: 953-60, 2012.
- 139. Gais S, Plihal W, Wagner U and Born J. Early sleep triggers memory for early visual discrimination skills. *Nat Neurosci* 3: 1335-9, 2000.
- 140. Garet M, Tournaire N, Roche F, Laurent R, Lacour JR, Barthelemy JC and Pichot V. Individual interdependence between nocturnal ANS activity and performance in swimmers. *Med Sci Sports Exerc* 36: 2112-8, 2004.
- 141. Gill ND, Beaven CM and Cook C. Effectiveness of post-match recovery strategies in rugby players. *Br J Sports Med* 40: 260-3, 2006.
- 142. Gleeson M. Probiotics use in sports. In 13th Nutrition and Health Conference. Brussels 122-35, 2010.
- 143. Gleeson M. Immune function in sport and exercise. J Appl Physiol 103: 693-9, 2007.
- 144. Goel N, Rao H, Durmer JS and Dinges DF. Neurocognitive consequences of sleep deprivation. *Semin Neurol* 29: 320-39, 2009.
- 145. Gomez-Cabrera MC, Domenech E, Romagnoli M, Arduini A, Borras C, Pallardo FV, Sastre J and Vina J. Oral administration of vitamin C decreases muscle

mitochondrial biogenesis and hampers training-induced adaptations in endurance performance. Am J Clin Nutr 87: 142-9, 2008.

- 146. Goodall S and Howatson, G. The effects of multiple cold water immersions on indices of muscle damage J Sport Sci Med 7: 235-41, 2008.
- 147. Gounin JP, Kiecolt-Glaser JK, Malarkey WB and Glaser R. The influence of anger expression on would healing. *Brain Behav Immun* 22: 699-708, 2008.
- 148. Grant AC, Gow IF, Zammit VA and Shennan DB. Regulation of protein synthesis in lactating rat mammary tissue by cell volume. *BBA* 1475: 39-46, 2000.
- 149. Gregson W, Black MA, Jones H, Milson J, Morton J, Dawson B, Atkinson G and Green DJ. Influence of cold water immersion on limb and cutaneous blood flow at rest. Am J Sports Med 39: 1316-23, 2011.
- 150. Haff G, Carlock JM, Hartman MK, Lon Kilgore J, Kawamori N, Jackson JR, Morris RT, Sands WA and Stone MH. Force-time curve characteristics of dynamic and isometric muscle actions of elite women Olympic weightlifters. J Strength Cond Res 19: 741-8, 2005.
- 151. Hagner W, Smolka A and Rozanska J. Influence of whole-body cryotherapy of results of exercise test. *Balneol Pol* 115: 35-9, 2009.
- 152. HajSalem M, Chtourou H, Aloui A, Hammouda O and Souissi N. Effects of partial sleep deprivation at the end of the night on anaerobic performances in judokas. *Biol Rhythm Res* 44:815-21, 2013.
- 153. Halson SL. Nutrition, sleep and recovery. J Sports Sci 8: 119-26, 2008.
- 154. Halson S. Does the time frame between exercise influence the effectiveness of hydrotherapy for recovery? Int J Sports Physiol Perform 6: 147-59, 2011.
- 155. Halson SL, Bartram J, West N, Stephens J, Argus CK, Driller MW, Sargent C, Lastella M, Hopkins WG and Martin DT. Does hydrotherapy help or hinder adaptation to training in competitive cyclists? *Med Sci Sports Exerc* epub ahead of print, 2014.
- 156. Haneishi K, Fry AC, Moore CA, Schilling BK, Li Y and Fry MD. Cortisol and stress responses during a game and practice in female collegiate soccer players. *J Strength Cond Res* 21: 583-588, 2007.

- 157. Hausswirth C, Louis J, Aubry A, Bonnet G, Duffield R and Le Meur Y. Evidence of disturbed sleep and increased illness in overreached endurance athletes. *Med Sci Sports Exerc* 46: 1036-45, 2014.
- 158. Hausswirth C, Louis J, Bieuzen F, Pournot H, Fournier J, Fillard JR and Brisswalter J. Effects of whole-body cryotherapy vs. far-infared vs. passive modalities on recovery from exercise-induced muscle damage in highly-trained runners. *PLoS One* 6: e27749, 2011.
- 159. Hellhammer DH, Hubert W and Schurmeyer T. Changes in saliva testosterone aafter psychological stimulation in mean. *Psychoneuroendocrinology* 10: 77-81, 1985.
- 160. Highton JM, Twist, C and Eston RG. The effects of exercise induced muscle damage on agility and sprint running performance. *J Exerc Sci Fit* 7: 24-30, 2009.
- 161. Hopkins WG, Marshall SW, Batterham AM and Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sport Exer* 41: 3-13, 2009.
- 162. Holway FE and Spriet LL. Sport-specific nutrition: practical strategies for team sports. *J Sports Sci* 29: S115-25, 2011
- 163. Horita, T. Stiffness Regulation during Stretch-shortening Cycle Exercise. PhD thesis, University of Jyvaskyla, Jyvaskyla, 2000.
- 164. Howatson G, Bell PG, Tallent J, Middleton B, McHugh MP and Ellis J. Effect of tart cherry juice (Prunus cerasus) on melatonin levels and enhanced sleep quality. *Eur J Nutr* 51:909-16, 2012.
- 165. Howatson G, Goodall S and van Someren KA. The influence of cold water immersions on adaptation following a single bout of damaging exercise. *Eur J Appl Physiol* 105: 615-21, 2009.
- 166. Howatson G, McHugh MP, Hill JA, Brouner J, Jewell AP, van Someren KA, Shave RE and Howatson SA. Influence of tart cherry juice on indices of recovery following marathon running. *Scand J Med Sci Sports* 20: 843-52, 2010.
- 167. Howatson G and Milak A. Exercise-induced muscle damage following a bout of sport specific repeated sprints. *J Strength Cond Res* 23: 2419-24, 2009.

- 168. Howatson G and van Someren KA. The prevention and treatment of exerciseinduced muscle damage. *Sports Med* 38: 483-503, 2008.
- 169. Iliodromitis EK, Lazou A and Kremastinos DT. Ischemic preconditioning: protection against myocardial necrosis and apoptosis. *Vasc Health Risk Manag* 3: 629-37, 2007.
- 170. Ingram J, Dawson B, Goodman C, Wallman K and Beilby J. Effect of water immersion methods on post-exercise recovery from simulated team sport exercise. J Sci Med Sport 12: 417-21, 2009.
- 171. Irwin M, Thompson J, Miller C, Gillin JC and Ziegler M. Effects of sleep and sleep deprivation on catecholamine and interleukin-2 levels in humans: clinical implications. *J Clin Endocrinol Metab* 84: 1979-85, 1999.
- 172. Jacob RA, Spinozzi GM, Simon VA, Kelley DS, Prior RL, Hess-Pierce B and Kader AA. Consumption of cherries lowers plasma urate in healthy women. J Nutr 33: 1826-9, 2003.
- 173. Jae SY, Heffernan KS, Park SH, Jung SH, Yoon ES, Kim EJ, Ahn ES and FernhallB. Does an acute inflammatory response temporarily attenuate parasympathetic reactivation? *Clin Auton Res* 20: 229-33, 2010.
- 174. Jakeman JR, Byrne C and Eston RG. Lower limb compression garment improves recovery from exercise-induced muscle damage in young, active females. *Eur J Appl Physiol* 109: 1137-44, 2010.
- 175. Jakeman JR, Macrae R and Eston R. A single 10min bout of cold-water immersion therapy after strenuous plyometric exercise has no beneficial effect on recovery from the symptoms of exercise-induced muscle damage. *Ergonomics* 52: 456-60, 2009.
- 176. Jarraya S, Jarraya M, Chtourou H and Souissi N. Effect of time of day and partial sleep deprivation on the reaction time and the attentional capacities of the handball goalkeeper. Biol Rhythm Res 45: 183-91, 2014.
- 177. Jarvinen TA, Jarvinen TL, Kaariainen M, Kalimo H and Jarvinen M. Muscle injuries: biology and treatment. *Am J Sports Med* 33: 745-64, 2005.

- 178. Jean-Louis G, Zizi F, von Gizycki H and Hauri P. Actigraphic assessment of sleep in insomnia: application of the actigraph data analysis software (ADAS). *Physiol Behav* 65: 659-63, 1999.
- 179. Jennings D, Cormack S, Coutts AJ, Boyd L and Aughey RJ. The validity and reliability of GPS units for measuring distance in team sport specific running patterns. *Int J Sports Physiol Perform* 5: 328-41, 2010.
- 180. Johnston RD, Gabbett TJ, Jenkins DG and Hulin DT. Influence of physical qualities on post-match fatigue in rugby league players. J Sci Med Sport epub ahead of print, 2014a.
- 181. Johnston RJ, Watsford ML, Kelly SJ, Pine MJ and Spurrs RW. The validity and reliability of 10Hz and 15Hz GPS units for assessing athlete movement demands. *J Strength Cond Res* 28: 1649-55, 2014b.
- 182. Johnston RJ, Watsford ML, Pine MJ, Spurrs RW, Murphy A and Pruyn EC. Movement demands and match performance in professional Australian football. *Int J Sports Med* 33: 89-93, 2012.
- 183. Judelson DA, Maresh CM, Yamamoto LM, Farrell MJ, Armstrong LE, Kraemer WJ, Volek JS, Spiering BA, Casa DJ and Anderson JM. Effect of hydration state on resistance exercise-induced endocrine markers of anabolism, catabolism, and metabolism. J Appl Physiol 105: 816-24, 2008.
- 184. Jurimae J, Maestu J, Purge P and Jurimae T. Changes in stress and recovery after heavy training in rowers. J Sci Med Sport 7:335-9, 2004.
- 185. Jurimae J, Maetsu J, Purge P, Jurimae T and Soot T. Relations among heavy training stress, mood state, and performance for male junior rowers. *Percept Mot Skills* 95: 520-6, 2002.
- 186. Kant GJ, Genser SG, Thorne DR, Pfalser JL and Mougey EH. Effects of 72 hour sleep deprivation on urinary cortisol and indices of metabolism. *Sleep* 7: 142-6, 1984.
- 187. Kelley DS, Rasooly R, Jacob RA, Kader AA and Mackey BE. Consumption of bing sweet cherroes lowers circulating concentrations of inflammation markers in healthy men and women. J Nutr 136: 981-6, 2006.

- 188. Kellmann M. Preventing overtraining in athletes in high-intensity sports and stress/recovery monitoring. *Scan J Med Sci Sports* 20: S95-102, 2010.
- 189. Kempton T, Sirotic AC, Cameron M and Coutts AJ. Match-related fatigue reduces physical and technical performance during elite rugby league match-play: a case study. *J Sports Sci* 31: 1770-80, 2013a.
- 190. Kempton T, Sirotic AC and Coutts AJ. Between match variation in professional rugby league competition. *J Sci Med Sport* epub ahead of print, 2013b.
- 191. Kiecolt-Glaser JK, Page GG, Marucha PT, MacCallum RC and Glaser R. Psychological influences on surgical recovery: perspectives from psychoneuroimmunology. Am Psychologist 53: 1209-18, 1998.
- 192. Kiviniemi AM, Hautala AJ, Kinnunen H and Tulppo MP. Endurance training guided individually by daily heart rate variability measurements. *Eur J Appl Physiol* 101: 743-51, 2007.
- 193. Klimek AT, Lubkowska A, Szygula Z, Fraczek B and Chudecka M. The influence of single whole body cryostimulation treatment on the dynamics and the level of maximal anaerobic power. *Int J Occup Environ Health* 24: 184-91, 2011.
- 194. Knoblauch MA, O'Connor DP and Clarke MS. Capillary and venous samples of total creatine kinase are similar after eccentric exercise. J Strength Cond Res 24: 3471-5, 2010.
- 195. Kraemer WJ, Flanagan SD, Comstock BA, Fragala MS, Earp JE, Dunn-Lewis C, Ho JY, Thomas GA, Solomon-Hill G, Penwell ZR, Powell MD, Wolf MR, Volek JS, Denegar CR, and Maresh CM. Effects of a whole body compression garment on markers of recovery after a heavy resistance workout in men and women. J Strength Cond Res 24: 804-14, 2010.
- 196. Kraemer WJ and Mazzetti SA. Hormonal mechanisms related to the expression of muscular strength and power. In: Komi PV (ed) Strength and Power in Sports. Blackwell Science Ltd., London, 73-95, 2003.
- 197. Krustrup P, Ortenblad N, Nielsen J, Nybo L, Gunnarsson TP, Iaia FM, Madsen K, Stephens F, Greenhaff P and Bangsbo J. Maximal voluntary contraction force, SR function and glycogen resynthesis during the first 72 h after a high-level competitive soccer game. *Eur J Appl Physiol* 111: 2987-95, 2011.

- 198. Kushida CA, Chang A, Gadkary C, Guilleminault C, Carrillo O, Dement W. Comparison of actigraphic, polysomonographic and subjective assessment in sleepdisordered patients. *Sleep Med* 2: 389-96, 2001.
- 199. Lacome M, Piscione J, Hager JP and Bourdin M. A new approach to quantifying physical demands in rugby union. *J Sports Sci* 32: 290-300, 2014.
- 200. Lane KN and Wenger HA. Effect of selected recovery conditions on performance of repeated bouts of intermittent cycling separated by 24 hours. J Strength Cond Res 18: 855-60, 2004.
- 201. Lastella M, Lovell GP and Sargent C. Athletes' precompetitive sleep behaviour and its relationship with subsequent precompetitive mood and performance. *Eur J Sport Sci* 1-8, iFirst article, 2012.
- 202. Lazarim FL, Antunes-Neto JM, da Silva FO, Nunes LA, Bassini-Cameron A, Cameron LC, Alves AA, Brenzikofer R and de Macedo DV. The upper values of plasma creatine kinase of professional soccer players during the Brazilian National Championship. J Sci Med Sport 12: 85-90, 2009.
- 203. Lee C, Linkenauger SA, Bakdash JZ, Joy-Gaba JA and Profitt DR. Putting like a pro: the role of positive contagion in golf performance and perception. *PloS one* 6: e26016, 2011.
- 204. Leeder J, Gardner AS, Foley S, van Someren K and Pedlar CR. The effect of jet lag on parameters of sleep in elite divers quantified by actigraphy. *Med Sci Sport Exer* 41: 57-8, 2009.
- 205. Leeder J, Gissane C, van Someren K, Gregson W and Howatson G. Cold water immersion and recovery from strenuous exercise: a meta-analysis. *Br J Sports Med* 46: 233-40, 2012a.
- 206. Leeder J, Glaister M, Pizzoferro K, Dawson J and Pedlar C. Sleep duration and quality in elite athletes measured using wristwatch actigraphy. J Sports Sci 30: 541-45, 2012b.
- 207. Leger D, Elbaz M, Raffray T, Metlaine A, Bayon V and Duforez F. Sleep management and the performance of eight sailors in the Tour de France à la voile yacht race. *J Sports Sci* 26:21-8, 2008.

- 208. Lemaitre F, Buchheit M, Joulia F, Fontanari P and Tourny-Chollet C. Static apnea effect on heart rate and its variability in elite breath-hold divers. *Aviat Space Environ Med* 79: 99-104, 2008.
- 209. Lockey SW, Skene DJ and Arendt J. Comparison between subjective and actigraphic measurement of sleep and sleep rhythms. J Sleep Res 8: 175-84, 1999.
- 210. Lubkowska A. Cryotherapy: physiological considerations and applications to physical therapy. In Physical therapy perspectives in the 21st century – challenges and possibilites. Ed. Bettany-Saltikov J and Paz-Lourido, 155-76, 2010.
- 211. Luczak J and Michalik J. Wplyw skrajnie niskich temperatur na wybrane cechy motoryczne człowieka. *Fizjoterapia Pol* 3: 206-11, 2006.
- 212. MacLeod H, Morris J, Nevill A and Sunderland C. The validity of a nondifferential global positioning system for assessing player movement patterns in field hockey. J Sports Sci 27: 121-8, 2009.
- 213. Macquet P, Laureys S, Peigneux P, Fuchs S, Petiau C, Phillips C, Aerts J, Del Fiore G, Degueldre C, Meulemans T, Luxen A, Franck G, Van Der Linden M, Smith C and Cleeremans A. Experience-dependent changes in cerebral activation during human REM sleep. *Nat Neurosci* 3: 831-6, 2000.
- 214. Mah CD, Mah KE, Kezirian EJ and Dement WC. The effects of sleep extension on the athletic performance of collegiate basketball players. *Sleep* 34:943-50, 2011.
- 215. Maughan R, Shirrefs SM and Leiper JB. Blood sampling. In: Sport and Exercise Physiology Testing Guidelines: Volume 1 – Sport Testing. Winter EM, Jones AM, Davidson RC, Bromley PD and Mercer TH, eds. London, Routledge, 25-29, 2006.
- 216. Mazur A and Lamb TA. Testosterone, status and mood in human males. *Horm Behav* 14: 236-46, 1980.
- 217. McLean BD, Coutts AJ, Kelly V, McGuigan MR and Cormack SJ. Neuromuscular, endocrine, and perceptual fatigue responses during different length between-match microcycles in professional rugby league players. *Int J Sports Physiol and Perform* 5: 367-83, 2010.
- 218. McLellan CP and Lovell DI. Neuromuscular responses to impact and collision during elite rugby league match play. J Strength Cond Res 26: 1431-40, 2012.

- 219. McLellan CP, Lovell DI and Gass GC. Biochemical and endocrine responses to impact and collision during elite Rugby League match play. *J Strength Cond Res* 25: 1553-62, 2011a.
- 220. McLellan CP, Lovell DI and Gass GC. Performance analysis of elite rugby league match play using global positioning systems. *J Strength Cond Res* 25: 1703-10, 2011b.
- 221. McMaster DT, Gill N, Cronin J and McGuigan M. The development, retention and decay rates of strength and power in elite rugby union, rugby league and American football. *Sports* Med 43: 367-84, 2013.
- 222. Meerlo P, Koehl M, Van der Borght K and Turek FW. Sleep restriction alters the hypothalamic-pituitary-adrenal response to stress. *J Neuroendocrinol* 14: 1-11, 2002.
- 223. Meerlo P, Sgofio A and Suchecki D. Restricted and disrupted sleep: Effects on autonomic function, neuroendocrine stress systems and stress responsivity. *Sleep Med Rev* 12: 197-210, 2008.
- 224. Mejri MA, Hammouda O, Zouaoui K, Chaouachi A, Chamari K, Ben Rayana MC and Souissi N. Effect of two types of partial sleep deprivation on Taekwondo players performance during intermittent exercise. *Biol Rhythm Res* 45: 17-26, 2014.
- 225. Mezick EJ, Matthews KA, Hall M, Kamarck TW, Buysse DJ, Owens JF and Reis SE. Intra-individual variability in sleep duration and fragmentation: associations with stress. *Psychoneuroendocrinology* 34:1346-54, 2009.
- 226. Millar ID, Barber MC, Lomax MA, Travers MT and Shennan DB. Mammary protein synthesis is acutely regulated by the cellular hydration state. *Biochem Biophys Res Commun* 230: 351-5, 1997.
- 227. Mohr M, Krustrup P and Bangsbo J. Match performance of high-standard soccer players with special reference to development of fatigue. J Sports Sci 21:519-28, 2003.
- 228. Mohr M, Krustrup P, Nybo L, Nielsen JJ and Bangsbo J. Muscle temperature and sprint performance during soccer matches – beneficial effect of re-warm up at half time. Scan J Med Sci Sport 14: 156-62, 2004.
- 229. Moller-Levet C, Archer S, Bucca G et al. Effects of insufficient sleep on circadian rhythmicity and expression amplitude of the human blood transcriptome. *Proc Natl Acad Sci U S A*. 110:4431-2, 2013.
- 230. Mooney M, Cormack SJ, O'Brien B and Coutts AJ. Do physical capacity and interchange rest periods influence match exercise-intensity profile in Australian football? *Int J Sports Physiol Perform* 8:165-72, 2013.
- 231. Mourot L, Bouhaddi M, Gandelin E, Cappelle S, Dumoulin G, Wolf J, Rouillon J and Regnard J. Cardiovascular autonomic control during short-term thermoneutral and cool head out immersion. *Aviat Space Environ Med* 79: 14-20, 2008.
- 232. Nedelec M, McCall A, Carling C, Legall F, Berthoin S and Dupont G. The influence of soccer playing actions on the recovery kinetics after a soccer match. *J Strength Cond Res* 28: 1517-23, 2014.
- 233. Nedeltcheva AV, Kilkus JM, Imperial J, Schoeller DA and Penev PD. Insufficient sleep undermines dietary efforts to reduce adiposity. *Ann Intern Med* 153: 435-41, 2010.
- 234. Nemet D, Meckel Y, Bar-Sela S, Zaldivar F, Cooper DM and Eliakim A. Effect of local cold-pack application on systemic anabolic and inflammatory response to sprint-interval training: a prospective comparative trial. *Eur J Appl Physiol* 107: 411-7, 2009.
- 235. Nicol, C And Komi, PV. Stretch-shortening fatigue and its influence on force and power production. In: *Strength and power in sport*. Komi, P, eds. Oxford, Blackwell, 203-30, 2003.
- 236. Nosaka K, Chapman D, Newton M and Sacco P. Is isometric strength loss immediately after eccentric exercise related to changes in indirect markers of muscle damage? *Appl Physiol Nutr Metab* 31: 313-9, 2006.
- 237. Nunes LAS, Gandra PG, Alves AA, Kubota L and Vaz de Macedo D. Adequacies of skin puncture for evaluating biochemical and hematological blood parameters in athletes. *Clin J Sports Med* 16: 418-21, 2006.
- 238. Oliver SJ, Costa RJ, Laing SJ, Bilzon JL and Walsh NP. One night of sleep deprivation decreases treadmill endurance performance. *Eur J Appl Physiol* 107: 155-61, 2009.

- 239. O'Reilly KP, Warhol MJ, Fielding RA, Frontera WR, Meredith CN and Evans WJ. Eccentric exercise-induced muscle damage impairs muscle glycogen repletion. J Appl Physiol 63: 252-6, 1987.
- 240. Osgnach C, Poser S, Bernardini R, Rinaldo R and di Prampero PE. Energy cost and metabolic power in elite soccer: a new match analysis approach. *Med Sci Sports Exerc* 42: 170-8, 2010.
- 241. Paddon-Jones DJ and Abernethy PJ. Acute adaptation to low volume eccentric exercise. *Med Sci Sports Exerc* 33: 1213-9, 2001.
- 242. Paddon-Jones DJ and Quigley BM. Effect of cryotherapy on muscle soreness and strength following eccentric exercise. *Int J Sports Med* 18: 588-93, 1997.
- 243. Parkhouse WS and McKenzie DC. Possible contribution of skeletal muscle buffers to enhanced anerobic performance: a brief review. *Med Sci Sports Exerc* 16: 328-38, 1984.
- 244. Parouty J, Al Haddad H, Quod M, Lepretre PM, Ahmaidi S and Buchheit M. Effect of cold water immersion on 100m sprint performance in well trained swimmers. *Eur J Appl Physiol* 109: 483-90, 2010.
- 245. Partsch H, Winiger J and Lun B. Compression stockings reduce occupational leg swelling. *Dermatol Surg* 30: 737-43; 2004.
- 246. Passerlegue P and Lac G. Saliva cortisol, testosterone and T/C variations during a wrestling competition and during the post-competitive recovery period. *Int J Sports Med* 20: 109-13, 1999.
- 247. Pedersen BK and Bruunsgaard H. How physical exercise influences the establishment of infections. *Sports Med* 19: 393-400, 1995.
- 248. Peiffer JJ, Abbiss CR, Watson G, Nosaka K and Laursen PB. Effect of a 5min coldwater immersion recovery on exercise performance in the heat. Br J Sports Med 44: 461-5, 2010.
- 249. Peigneux P, Laureys S, Fuchs S, Collette F, Perrin F, Reggers J, Phillips C, Degueldre C, Del Fiore G, Aerts J, Luxen A and Maquet P. Are special memories strengthened in the human hippocampus during slow wave sleep? *Neuron* 44: 535-45.

- 250. Pejovic S, Basta M, Vgontzas AN, Kritikou I, Shaffer ML, Tsaoussoglou M, Stiffler D, Stefanakis Z, Bixler EO and Chrousos GP. Effects of recovery sleep after one work week of mild sleep restriction on interleukin-6 and cortisol secretion and daytime sleepiness and performance. *Am J Physiol Endocrinol Metab* 305:E890-6, 2013.
- 251. Penzel T and Canisius S. Polysomnography. In: *sleep apnea: current diagnosis and treatment*. Randerath WJ, Sanner BM and Somers VK, eds. Prog Respir Res. Basel, Karger vol 35: 51-60, 2006.
- 252. Perini R, Orizio C, Comande A, Castellano M, Beschi M and Veicsteinas A. Plasma norepinephrine and heart rate dynamics during recovery from submaximal exercise in man. *Eur J Appl Physiol Occup Physiol* 58: 879-83, 1989.
- 253. Petersen C, Pyne D, Portus M and Dawson B. Validity and reliability of GPS units to monitor cricket-specific movement patterns. *Int J Sports Physiol Perform* 4:381-93, 2009.
- 254. Pichot V, Roche F, Gaspoz J-M, Enjolras F, Antoniadis A, Minini P, Costes F, Busso T, Lacour J-R and Barthelemy JC. Relation between hear rate variability and training load in middle-distance runners. *Med Sci Sports Exerc* 32: 1729-36, 2000.
- 255. Pirke KM, Kockott G and Dittmar F. Psychosexual stimulation and plasma testosterone in man. Arch Sex Behav 3: 577-84, 1974.
- 256. Pizza FX, Cavender D, Stockard A, Baylies H and Beighle A. Anti-inflammatory doses of ibuprofen: effect on neutrophils and exericse-induced muscle injury. *Int J Sports Med* 20: 98-102, 1999.
- 257. Pointon M and Duffield R. Cold Water Immersion Recovery following Simulated Collision Sport Exercise. *Med Sci Sports Exer* 44: 206-16, 2011.
- 258. Pollak C, Tryon WW, Nagaraja H and Dzwonczyk R. How accurately does wrist actigraphy identify states of sleep and wakefulness? *Sleep* 24: 957-65, 2001.
- 259. Portas MD, Harley JA, Barnes CA and Rush CJ. The validity and reliability of 1Hz and 5Hz global positioning systems for linear, multidirectional and soccer-specific activities. *Int J Sports Physiol Perform* 5: 448-58, 2010.

- 260. Postolache TT, Hung TM, Rosenthal RN, Soriano JJ, Montes F and Stiller JW. Sports chronobiology consultation. From the lab to the arena. *Clin Sports Med* 24: 415-56, 2005.
- 261. Pournot H, Bieuzen F, Louis J, Mounier R, Fillard JR, Barbiche E and Hausswirth C. Time-course of changes in inflammatroy response after whole-body cryotherapy multi exposures following severe exercise. *PLoS One* 6: e22748, 2011.
- 262. Powers SK and Jackson MJ. Exercise-induced oxidative stress: cellular mechanisms and impact on muscle force production. *Physiol Revs* 88: 1243-76, 2008.
- 263. Quarrie KL, Hopkins WG, Anthony MJ and Gill ND. Positional demands of international rugby union: evaluation of player actions and movements. J Sci Med Sport 16: 353-9, 2013.
- 264. Quarrie KL and Wilson BD. Force production in the rugby union scrum. J Sports Sci 18: 237-46, 2000.
- 265. Rahmani A, Viale F, Dalleau G and Lacour J-R. Force/velocity and power/velocity relationships in squat exercise. *Eur J Appl Plhysiol* 84: 227-32, 2001.
- 266. Rampinini E, Bosio A, Ferraresi I, Petruolo A, Morelli A and Sassi A. Matchrelated fatigue in soccer players. *Med Sci Spiorts Exer* 43: 2161-70, 2011.
- 267. Reilly T and Ekblom B. The use of recovery methods post-exercise. J Sports Sci 23: 619-27, 2005.
- 268. Richmond L, Dawson B, Hillman DR and Eastwood PR. The effect of interstate travel on sleep patterns of elite Australian rules footballers. *J Sci Med Sport* 7: 186-96, 2004.
- 269. Roberts SP, Trewartha G, Higgitt RJ, Ell-Abd J and Stokes KA. The physical demands of elite English rugby union. J Sports Sci 26: 825-33, 2008.
- 270. Roberts S, Trewartha G and Stokes K. A comparison of time-motion analysis methods for field based sports. Int J Sports: Physiol Perform 1: 388-99, 2006.
- 271. Robertson MD, Russell-Jones D, Umpleby AM and Dijk DJ. Effects of three weeks of mild sleep restriction implemented im the home environment on multiple

metabolic and endocrine markers in healthy young men. *Metabolism* 62: 204-11, 2013.

- 272. Sadeh A. The role and validity of actigraphy in sleep medicine: An update. *Sleep* Med Rev 15: 259-67, 2011.
- 273. Sallinen M, Onninen J, Tirkkonen K, Haavisto ML, Harma M, Kubo T, Mutanen P, Virkkala J, Tolvanen A and Porkka-Heiskanen T. Effects of cumulative sleep restriction on self-perceptions while multitasking. J Sleep Res 22: 273-81, 2013.
- 274. Santos RV, Tufik S and De Mello MT. Exercise, sleep and cytokines: is there a relation. *Sleep Med Rev* 11: 231-9, 2007.
- 275. Sargent C, Halson S and Roach GD. Sleep or swim? Early-morning training severely restricts the amount of sleep obtained by elite swimmers. *Eur J Sport Sci* 14: S310-5, 2014.
- 276. Saxton JM, Clarkson PM, James R, Miles M, Westerfer M, Clark S and Donnelly AE. Neuromuscular dysfunction following eccentric exercise. *Med Sci Sports Exer* 27: 1185-93, 1995.
- 277. Sedeaud A, Marc A, Schipman J, Tafflet M, Hager JP and Toussaint JF. How they won Rugby World Cup through height, mass and collective experience. *Br J Sports Med* 46: 580-4, 2012.
- 278. Seiler S, Haugen O and Kuffel E. Autonomic recovery after exercise in trained athletes: intensity and duration effects. *Med Sci Sports Exerc* 39: 1366-73, 2007.
- 279. Sellwood KL, Brukner P, Williams D, Nicol A and Hinman R. Ice-water immersion and delayed-onset muscle soreness: a randomised controlled trial. *Br J Sports Med* 41: 392-7, 2007.
- 280. Seyle, H. The general adaptation syndrome and the diseases of adaptation. *J Clin Endocrinol* 6: 117-230, 1946.
- 281. Shirreffs SM, Taylor AJ, Leiper JB and Maughan RJ. Post-exercise rehydration in man: effects of volume consumed and drink sodium content. *Med Sci Sports Exer* 28: 1260-71, 1996.

- 282. Sinnerton S, Reilly T. Effects of sleep loss and time of day in swimmers. In: MacLaren D, Reily T, Lees A, eds. *Biomechanics and medicine in swimming: swimming science VI*. London: E and FN Spon. 399-405, 1992.
- 283. Siri WR. Body composition from fluid spaces and density: analysis of methods. In: *Techniques for measuring body composition*. Eds: Brozek J and Henschel A. Nat.Acad.Sci.Washington D.C, 223-44, 1961.
- 284. Skein M, Duffield R, Edge J, Short MJ and Mundel T. Intermittent-sleep performance and muscle glycogen after 30h of sleep deprivation. *Med Sci Sports Exerc* 43: 1301-11, 2011.
- 285. Skein M, Duffield R, Minett GM, Snape A and Murphy A. The effect of overnight sleep deprivation after competitive rugby league matches on post-match physiological and perceptual recovery. *Int J Sports Physiol Perform* 8: 556-64, 2013.
- 286. Smart DJ, Gill ND, Beaven CM, Cook CJ and Blazevich AJ. The relationship between changes in interstitial creatine kinase and match-related impacts in rugby union. *Br J Sports Med* 42: 198-201, 2008.
- 287. Smith DJ. A framework for understanding the training process leading to elite performance. *Sports Med* 33: 1103-1126, 2003.
- 288. Smith C, Kruger MJ, Smith RM and Myburgh KH. The inflammatory response to skeletal muscle injury: illuminating complexities. *Sports Med* 38: 947-69, 2008.
- 289. Souissi N, Chtourou H, Aloui A, Hammouda O, Dogui M, Chaouachi A and Chamari K. Effects of time-of-day and partial sleep deprivation on short-term maximal performances of judo competitors. *J Strength Cond Res* 27: 2473-80, 2013.
- 290. Souissi N, Sesboue B, Gauthier A, Larue J and Davenne D. Effects of one night's sleep deprivation on anaerobic performance the following day. *Eur J Appl Physiol* 89: 359-66, 2003.
- 291. Souissi N, Souissi M, Souissi H, Chamari K, Tabka Z, Dogui M, Davenne D. Effect of time of day and partial sleep deprivation on short-term high-power output. *Chronobiol Int* 25: 1062-76, 2008.

- 292. Spath-Schwalbe E, Gofferje M, Kern W, Born J and Fehm HL. Sleep disruption alters nocturnal ACTH and cortisol secretory patterns. *Biol Psychiatry* 29: 575-84, 1991.
- 293. Spiegel K, Leproult R, Colecchia EF, L'Hermite-Baleriaux N, Nie Z, Copinschi G and Van Cauter E. Adaptation of the 24h growth hormone profile to a state of sleep debt. *Am J Physiol Regul Integr Comp Physiol* 279: R874-83, 2000.
- 294. Spiegel K, Leproult R, Van Cauter E. Impact of sleep debt on metabolic and endocrine function. *Lancet* 354:1435-9, 1999.
- 295. Spencer M, Lawrence S, Rechichi C, Bishop D, Dawson B and Goodman C. Timemotion analysis of elite field hockey, with special reference to repeated-sprint activity. *J Sports Sci* 22: 843-50, 2004.
- 296. Sramek P, Simeckova M, Jansky L, Savlikova J and Vybiral S. Human physiological responses to immersion into water of different temperatures. *Eur J Appl Physiol* 81: 436-42, 2000.
- 297. Stanley J, Buchheit M and Peake JM. The effect of post-exercise hydrotherapy on subsequent exercise performance and heart rate variability. *Eur J Appl Physiol* 112: 951-61, 2012.
- 298. Stauber WT. Factors involved in strain-induced injury in skeletal muscles and outcomes of prolonged exposures. *J Electromyogr Kinesiol* 14: 61-70, 2004.
- 299. Stone MH, Sands WA, Carlock J, Callan S, Dickie D, Daigle K, Cotton J, Smith SL and Hartman M. The importance of isometric maximum strength and peak rate-of-force development in sprint cycling. *J Strength Cond Res* 18: 878-84, 2004.
- 300. Strobel NA, Peake JM, Matsumoto A, Marsh SA, Coombes JS, and Wadley GD. Antioxidant supplementation reduces skeletal muscle mitochondrial biogenesis. *Med Sci Sports Exer* 43: 1017-24, 2011.
- 301. Stoleru SG, Ennaji A, Cournot A and Spira A. LH pulsatile secretion and testosterone blood levels and influenced by sexual arousal in human males. *Psychoneuroendocrinology* 18: 205-18. 1993.

- 302. Sullivan C, Bilsborough JC, Cianciosi M, Hocking J, Cordy J and Coutts AJ. Match score affects activity profile and skill performance in professional Australian football players. *J Sci Med Sport* 17: 326-31, 2014.
- 303. Takahashi I, Umeda T, Mashiko T, Chinda D, Oyama T, Sugawara K and Nakaji S. Effects of rugby sevens matches on human neutrophil-related non-specific immunity. Br J Sports Med 41: 13-8, 2007.
- 304. Takarada Y. Evaluation of muscle damage after a rugby match with special reference to tackle plays. Br J Sports Med 37: 416-9, 2003.
- 305. Tassi P and Muzet A. Sleep inertia. Sleep Med Rev 4:341-53, 2000.
- 306. Taylor SR, Rogers GG and Driver HS. Effects of training volume on sleep, psychological, and selected physiological profiles of elite female swimmers. *Med Sci Sports Exerc* 29: 688-93, 1997.
- 307. Temesi J, Arnal PJ, Davranche K, Bonnefoy R, Levy R, Verges S and Millet GY. Does central fatigue explain reduced cycling after complete sleep deprivation? *Med Sci Sports Exerc* 45: 2243-53, 2013.
- 308. Thilers PP, Macdonald SW and Heriltz A. The association between endogenous free testosterone and cognitive performance: a population-based study in 35 to 90 year-old men and women. *Psychoneuroendocrinology* 31: 565-76, 2006.
- 309. Thorpe R and Sunderland C. Muscle damage, endocrine, and immune marker response to a soccer match. *J Strength Cond Res* 26: 2783-90, 2012.
- 310. Tiemeier H, Pelzer E, Jonck L, Moller HJ and Rao ML. Plasma catecholamines and selective slow wave sleep deprivation. *Neuropsychobiology* 45: 81-6, 2002.
- 311. Tofas T, Jamurtas AZ, Fatouros I, Nikolaidis MG, Koutedakis Y, Sinouris EA, Papageorgakopoulous N and Theocharis DA. Plyometric exercise increases serum indices of muscle and collagen breakdown. *J Strength Cond Res* 22: 490-6, 2008.
- 312. Trappe TA, Carroll CC, Dickinson JM, LeMoine JK, Haus JM, Sullivan BE, Lee JD, Jemiolo B, Weinheimer EM and Hollon CJ. Influence of acetaminophen and ibuprofen on skeletal muscle adaptations to resistance exercise in older adults. Am J Physiol Reg I 300: R655-62, 2011.

- 313. Trappe TA, White F, Lambert CP, Cesar D, Hellerstein M, and Evans WJ. Effect of ibuprofen and acetaminophen on postexercise muscle protein synthesis. Am J Physiol Endocrinol Metab 282: E551-6, 2002.
- 314. Trinder J, Paxton SJ, Montgomery I and Fraser G. Endurance as opposed to power trianing: their effect on sleep. *Psychophysiology* 22: 668-73, 1985.
- 315. Tucker AT, Maass A, Bain DS, Chen L-H, Axxam M, Dawson H and Johnston A. Augmentation of venous, arterial and microvascular blood supply in the leg by isometric neuromuscular stimulation via the peroneal nerve. *Int J Angiol* 19: e31-7, 2010.
- 316. Twist C and Eston R. The effects of exercise-induced muscle damage on maximal intensity intermittent exercise performance. *Eur J Appl Physiol* 94: 652-6, 2005.
- 317. Vaile J, Halson S, Gill N and Dawson B. Effect of cold water immersion on repeat cycling performance and thermoregulation in the heat. *J Sports Sci* 26: 431-40, 2008.
- 318. Vaile JM, Gill ND, and Blazevich AJ. The effect of contrast water therapy on symptoms of delayed onset muscle soreness. *J Strength Cond Res* 21: 697-702, 2007.
- 319. Van Dongen HP, Maislin G, Mullington JM and Dinges DF. The cumulative cost of additional wakefulness: dose-response effects on neurobehavioural functions and sleep physiology from chronic sleep restriction and total sleep deprivation. *Sleep* 26: 117-26, 2003.
- 320. Varley MC, Fairweather IH, and Aughey RJ. Validity and reliability of GPS for measuring instantaneous velocity during acceleration, deceleration, and constant motion. *J Sports Sci* 30: 121-7, 2012.
- 321. Venter RE. Perceptions of team athletes on the importance of recovery modalities. *Eur J Sport Sci* 1-8, iFirst article, 2012.
- 322. Versey N, Halson S, and Dawson B. Effect of contrast water therapy duration on recovery of cycling performance: a dose-response study. *Eur J Appl Physiol* 111: 37-46, 2011.
- 323. Versey NG, Halson SL and Dawson BT. Water immersion recovery for athletes: effect on exercise performance and practical recommendations. *Sports Med* 43: 1101-30, 2013.

- 324. Vgontzas AN, Chrousos GP. Sleep, the hypothalamic-pituitary-adrenal axis, and cytokines: multiple interactions and disturbances in sleep disorders. *Endocrinol Metab Clin North Am.* 31:15-36, 2002.
- 325. Walker MP and Stickgold R. Sleep-dependent learning and memory consolidation. *Neuron* 44: 121-33, 2004.
- 326. Waterhouse J, Atkinson G, Edwards B and Reilly T. The role of a short post-lunch nap in improving cognitive, motor, and sprint performance in participants with partial sleep deprivation. *J Sport Sci* 25: 1557-66, 2007.
- 327. Weiss AR, Johnson NL, Berger NA and Redline S. Validity of activity-based devices to estimate sleep. *J Clin Sleep Med* 15: 336-42, 2010.
- 328. West DJ, Finn CV, Cunningham DJ, Shearer DA, Jones MR, Harrington BJ, Crewther BT, Cook CJ and Kilduff LP. Neuromuscular function, hormonal, and mood responses to professional rugby union match. *J Strength Cond Res* 28: 194-200, 2014.
- 329. West DJ, Cook CJ, Stokes KA, Atkinson P, Drawer S, Bracken RM and Kilduff LP. Profiling the time-course changes in neuromuscular function and muscle damage over two consecutive tournament stages in elite rugby sevens players. *J Sci Med Sport* epub ahead of print, 2013.
- 330. West DJ, Owen NJ, Jones MR, Bracken RM, Cook CJ, Cunningam DJ, Shearer DA, Finn CV, Newton RU, Crewther BT and Kilduff LP. Relationships between force-time characteristics of the isometric midthigh pull and dynamic performance in professional rugby league players. J Strength Cond Res 25: 3070-5, 2011.
- 331. Wilcock IM, Cronin JB and Hing WA. Physiological response to water immersion: a method for sport recovery? *Sports Med* 36: 747-5, 2006.
- 332. Wilmore JH and Behnke MD. An anthropometric estimation of body density and lean body weight in young women. *Am J Clin Nutr* 23: 267-74, 1970.
- 333. Winchester JB. The use of endocrine markers to predict and monitor performance in strength and power activities. PhD thesis, Louisiana State University, Louisiana 2008.

- 334. Wisbey B, Montgomery PG, Pyne DB and Rattray B. Quantifying movement demands of AFL football using GPS tracking. J Sci Med Sport 13: 531-6, 2010.
- 335. Wisbey B, Rattray B and Pyne DB. Quantifying changes in AFL player game demands using GPS tracking: 2008 AFL season. Florey (ACT): FitSense Australia, 2008.
- 336. Wood B, Rea MS, Plitnick B and Figueiro MG. Light level and duration of exposure determine the impact of self-luminous tablets on melatonin suppression. *Appl Ergon* 44: 237-40, 2013.
- 337. Wozniak A, Wozniak B, Drewa G, Mila-Kierzenkowska C and Rakowski A. The effect of whole-body cryostimulation on the pro-oxidant-antioxidant balans in blood of elite kayakers after training. *Eur J Appl Physiol* 101: 533-537, 2007.
- 338. Yamane M, Teruya H, Nakano M, Ogai R, Ohnishi N and Kosaka M. Post-exercise leg and forearm flexor muscle cooling in humans attenuates endurance and resistance training effects on muscle performance and on circulatory adaptation. *Eur J Appl Physiol* 96: 572-80, 2006.
- 339. Young WB, Hepner J, and Robbins DW. Movement demands in Australian rules football as indicators of muscle damage. *J Strength Cond Res* 26: 492-6, 2012.
- 340. Youngstedt SD, O'Connor PJ, Crabbe JB and Dishman RK. The influence of acute exercise on sleep following high caffeine intake. *Physiol Behav* 68: 563-70, 2000.
- 341. Youngstedt SD, O'Connor PJ and Dishman RK. The effects of acute exercise on sleep: a quantitative synthesis. *Sleep* 20: 203-14, 1997.
- 342. Zagrobelny Z, Halawa B, Niedzielski C and Wawrowska A. The influence of a single whole body cryostimulation on selected hemodynamic indices and hormone concentrations in the serum of healthy individuals. *Pol Tyg Lek* 48: 303-305, 1993.
- 343. Zubillaga A. La actividad del jugador de fútbol en alta competición: análisis de variabilidad. Doctoral thesis, University of Malaga, 2006.