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Tapering strategies for elite endurance running performance

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Tapering Strategies for Elite Endurance Running Performance

by

Kate L. Spilsbury

A Doctoral Thesis submitted in partial fulfilment of the requirements for the award of

Doctor of Philosophy of Loughborough University

May 2016

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ABSTRACT

It is common practice for endurance athletes to manipulate training load prior to an important competition, known as tapering. An effective strategy aims to alleviate accumulated fatigue, whilst maximising physiological adaptation and facilitating a peak performance. Improvements in performance of 0.5 to 6.0% have been reported after a successful taper, a margin that could potentially have a dramatic influence on performance outcome at the elite level. This thesis explored the strategies currently employed by elite endurance athletes and investigated novel training manipulations during the taper to further enhance performance, to gain a more thorough understanding of the physiological mechanisms, and to identify a minimally invasive physiological biomarker capable of monitoring recovery status during the taper. Tapering strategies in elite endurance athletes were shown to be individualised and influenced by the preceding training load. Algorithms were developed, capable of explaining a large proportion of the variance (53-95%) in tapering strategy training variables (with the exception of interval volume), for a given pre-taper training load (Chapter III). A tapering strategy implemented using the algorithms was ‘most likely’ to improve 1,500 m treadmill performance ($ES = 0.53$). When the intensity of final interval session was increased from 100% to 115% race speed, the effect on treadmill performance was ‘unclear’ ($ES = 0.22$) and perhaps due to insufficient recovery to respond positively to the increased intensity interval session (Chapter IV). When continuous volume was reduced further (by 60%), the novel high intensity strategy was ‘very likely’ to improve 1,500 m track performance ($ES = 0.74$), compared to the algorithm-derived taper ($ES = 0.40$) (Chapter VI). In middle-distance runners, training above race speed in the final days of the taper might be more beneficial than current practice, although training volume must be further reduced to compensate. It was possible to measure plasma concentrations of interleukin-6 and soluble interleukin-6 receptor from capillary samples (Chapter II), although these markers in addition to C-reactive protein, testosterone and cortisol were not sensitive enough to detect changes in recovery status during tapering (Chapters IV and V). Measures of muscle maximum voluntary contraction force (algorithm-derived taper: 9%; $ES = 0.39$; novel taper: 6%; $ES = 0.29$), and rate of force development (algorithm-derived taper: $ES = 0.53$; novel taper: $ES = 0.26$) improved in response to tapering (Chapter IV), and could represent alternative non-invasive markers of recovery and taper effectiveness to facilitate peak performance.

KEYWORDS

TAPERING, PERFORMANCE, PEAKING, DISTANCE RUNNING, TRAINING LOAD, VOLUME, FREQUENCY, INTENSITY, ATHLETE, RECOVERY, PACING

PREFACE

The findings within this thesis have been peer reviewed and published as follows:

1. Spilsbury, K.L., Faulkner, S.H., Fudge, B.W., Pringle, J.S.M., Orme, M., & Nimmo, M.A. The effects of an increase in intensity during tapering on 1,500 m performance. *Journal of Sports Sciences*. In review.
2. Spilsbury, K.L., Fudge, B.W., Ingham, S.A., Faulkner, S.H., & Nimmo, M.A. (2015). Tapering strategies in elite British endurance runners. *European Journal of Sports Science*, 15(5), 367-73.
3. Faulkner, S.H., Spilsbury, K.L., Harvey, J., Jackson, A., Huang, J., Platt, M., Tok, A., & Nimmo, M.A. (2014). The detection and measurement of interleukin-6 in venous and capillary blood samples, and in sweat collected at rest and during exercise. *European Journal of Applied Physiology* 114(6), 1207-16.

Abstracts from the following studies have been peer reviewed and accepted for conference presentations as follows:

1. Spilsbury, K.L., Faulkner, S.H., Fudge, B.W., Pringle, J.S.M., Jackson, A., & Nimmo, M.A. Tapering in middle-distance runners: the effect of a final high intensity training bout on 1,500 m performance. European College of Sports Science (ECSS) Annual Congress, Amsterdam 2nd-5th July 2014. Oral presentation.
2. Spilsbury, K.L., Fudge, B.W., Ingham, S.A., Pringle, J.S.M., King, J.A., Saini, A., & Nimmo, M.A. Tapering strategies in elite British Endurance runners. European College of Sports Science (ECSS) Annual Congress, Barcelona 26-29th June 2013. Poster presentation.

DECLARATION

The work presented in this thesis was funded by the English Institute of Sport and Loughborough University. I hereby declare that this thesis has been composed by myself, and that the work it includes has been carried out by myself, except where specifically acknowledged below. I confirm that this thesis has not been submitted in any previous application for a higher degree and that all sources of information have been specifically acknowledged by means of references.

Chapter II was conducted with assistance from Dr J. King during data collection, to carry out the simultaneous collection of venous and capillary blood samples.

Chapter IV was conducted with assistance from undergraduate students Miss K. Addy, Miss S. Chambers and Miss N. Rawlinson during data collection in the performance assessments. Mr M. Orme assisted with physical activity data collection. Mr A. Jackson assisted with analysis of blood samples.

Chapter VI was conducted with assistance from Dr S. Faulkner during data collection.

ACKNOWLEDGEMENTS

I would like to thank my supervisors Dr Steve Faulkner, Dr Barry Fudge and Professor Myra Nimmo for their mentorship and guidance throughout my PhD studies. I am sincerely grateful for their encouragement and I am privileged to have worked with such a knowledgeable and expert team.

I am grateful to my sponsors, the English Institute of Sport and Loughborough University and in particular, to Dr Steve Ingham for the opportunity to undertake such a fantastic and applied PhD. I would also like to thank Dr Jamie Pringle, and the EIS physiology team for their support.

My gratitude extends to the athletes who volunteered their time to participate in my research. Without their willingness, commitment and dedication, this work would not have been possible.

To Gaz, Andy and Conor, as much as I hate to admit it, it's been fun to share this journey with you! I look forward to continuing to work with you all in the future. To everyone at SSEHS who helped me along the way, I couldn't have done it without you. In particular, thank you to Mel, Amo, James, Fry, Andy, Mark, Jess and Steve and also to my fellow PhD students for helping me through the challenges and for the memories of the good times.

Finally, I would like to thank my family and friends for their continued support and patience. You have always been there for me and inspired me to challenge myself and I hope that I have made you proud. To Deane, I will be forever grateful for your love, support and belief in me. You've got me through the hardest of times with unrelenting optimism, despite the time together that we've had to sacrifice so that I could follow my dream. Thank you for everything and I look forward to our time together on the other side.

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LIST OF ABBREVIATIONS

AMPK	5 Adenosine monophosphate-activated protein kinase
ATP	Adenosine triphosphate
BCS	Bovine calf serum
BMI	Body mass index (kg/m^2)
BPM	Beats per minute ($\text{b}\cdot\text{min}^{-1}$)
BSA	Bovine serum albumin
C	Cortisol
CK	Creatine kinase
CMJ	Counter movement jump
CoA	Coenzyme A
CO ₂	Carbon dioxide
CRP	C-reactive protein
CSA	Cross-sectional area
CV	Coefficient of variation
ES	Effect size
ELISA	Enzyme-linked immunosorbent assay
FCS	Foetal calf serum
GPS	Global positioning system
IL-10	Interleukin-10
IL-1ra	Interleukin-1 receptor agonist
IL-6	Interleukin-6
LH	Luteinising hormone
MHC	Myosin heavy chain
MRF4	Myogenic regulatory factor 4
mTOR	Mammalian target of rapamycin
MURF-1	Muscle ring-finger protein-1
MVC	Maximal voluntary contraction
MVPA	Moderate-vigorous physical activity
O ₂	Oxygen
PBS	Phosphate-buffered saline
RER	Respiratory exchange ratio
RFD	Rate of force development

rhIL-6	Recombinant human interleukin-6
RPE	Rating of perceived exertion
SD	Standard deviation
SEM	Standard error of the mean
sIL-6r	Soluble interleukin-6 receptor
T	Testosterone
T:C	Testosterone to cortisol ratio
TBS	Tris-buffered saline
TNF- α	Tumor necrosis factor-alpha
TU	Training units
$\dot{V}O_2$	Oxygen uptake ($\text{mL}\cdot\text{min}^{-1}$)
$\dot{V}O_{2\text{max}}$	Maximum oxygen uptake ($\text{mL}\cdot\text{kg}\cdot\text{min}^{-1}$)
$\dot{V}O_{2\text{peak}}$	Peak oxygen uptake ($\text{mL}\cdot\text{kg}\cdot\text{min}^{-1}$)

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Chapter I

General Introduction

1.1 Background

The physiological superiority of elite endurance athletes is determined partly by genetic predisposition (Bouchard & Lortie, 1984, Bouchard, 1998). Endurance training also has a profound effect on key physiological adaptations including; maximal oxygen uptake ($\dot{V}O_{2\max}$), fractional utilisation of $\dot{V}O_{2\max}$, running economy and oxygen uptake ($\dot{V}O_2$) kinetics, all of which are important determinants of endurance performance (Basset & Howley, 2000; Bransford & Howley, 1976; Coyle, 1999; Hill & Lupton, 1923; Jones, 2006; Jones & Carter, 2000; Joyner & Coyle, 1998; Whipp & Wasserman, 1972). Additionally, it is evident that the magnitude of the physiological response to training is subject to genetic influence (Bouchard et al., 1988). Therefore, favourable genetics combined with chronic adaptation from effective training, creates a phenotype with physiological characteristics optimised for elite endurance performance.

Winning a gold medal at a global competition such as the Olympic Games, is the pinnacle of sporting success in track and field athletics. However, a systematic training process over many years is critical in developing an endurance athlete to perform successfully at the elite level (Smith, 2003). For example, over 10 years of arduous training, underpinned by scientific principles, were required for Paula Radcliffe to reach her athletic potential and achieve the world record for the marathon (2:15:24 h:min:s) (Jones, 2006). The primary aims of endurance training are to cause adaptation to the pulmonary, cardiovascular and neuromuscular systems that improve the delivery of O_2 from atmospheric air to the mitochondria and therefore allow improved endurance performance by maintaining a higher speed over a specific race distance (Jones & Carter, 2000). Optimal physiological adaptation to training will only occur however, if the magnitude of training load is applied in an appropriate manner over time, providing stimuli large enough (i.e. overload or overreaching) to initiate the desired responses, in combination with recovery periods sufficient to allow 'supercompensation' (Halsen & Jeukendrup, 2004; Meeusen et al., 2013; Smith, 2003).

Whilst many elite athletes continually strive to push the limits of human performance, and epitomise the Olympic motto of '*Citius, Altius, Fortis*', failure to balance increases in

training load with adequate recovery and regeneration periods can result in prolonged maladaptation and underperformance (Meeusen et al., 2013). This negative response is known as overtraining syndrome and is characterised by a persistent reduction in performance, despite a decrease in training load, and has been associated with a multitude of physiological, psychological, immunological and biochemical symptoms, which vary between individuals (Fry et al., 1991). Nevertheless, a phase of intensified or ‘overloaded’ training is commonly built into the training plan prior to a competition period, in an attempt to optimise physiological adaptation for the benefit of performance (Aubry et al., 2014). Since physiological stress is high and between-session recovery is typically minimal during this time, temporary physiological disturbances are allowed to manifest, including; glycogen depletion, neuromuscular fatigue, decrements in red cell volume and hemoglobin, imbalance in anabolic and catabolic tissue activities, and ultimately an acute suppression of performance (Halsen & Jeukendrup, 2004; Halsen et al., 2002; Hellard et al., 2013; Mujika and Padilla, 2003). Consequently, the challenge for coaches is to implement a change in training load after this intensified period which is sufficient to alleviate accumulated fatigue, whilst maintaining or further enhancing physiological adaptation. This strategy is known as tapering and aims to facilitate the peaking of performance at the appropriate time for major competition (Mujika & Padilla 2003).

1.2 Introduction to Tapering

Tapering can be achieved by manipulating the training load variables of volume, frequency and intensity over a particular duration (Houmard, 1991). In relation to endurance runners, to which this review will focus on, the volume of training typically represents the distance covered (km) and frequency refers to how regularly the training is undertaken and reflects the recovery period between training sessions. Intensity describes the physiological demand of the training and can be expressed in several different ways. Endurance athletes commonly train at relative intensities, running at speeds based on percentages of their own individual maximal aerobic capacity, maximal heart rate or race pace (Jones, 2006). In an attempt to optimise athletic performance, a variety of different tapering strategies have been investigated. Such strategies include the manipulation of a single training variable, in addition to alterations of a combination of variables, over differing durations. The pattern of the taper describes the manner in which the training variables are manipulated leading into competition. Several tapering patterns have traditionally been employed in an attempt to establish the optimal strategy for performance (Figure 1.1). A linear pattern consists of a

progressive reduction in training of equal proportion. Although the term ‘tapering’ would imply that training is always reduced in a progressive manner, it can also be reduced in a non-progressive standardised approach, known as a step taper (Mujika, 1998). In contrast, an exponential pattern initially reduces training dramatically before beginning to plateau nearer to competition. An exponential pattern can take the form of a fast or slow decay of training reduction, with a slow decay maintaining more training than a fast decay. For example, at 7 d into a 14-d fast decay taper, the athlete would be completing approximately 25% of normal training compared to 35% in a slow decay taper. A higher proportion of normal training is completed using a linear pattern compared to an exponential pattern; at 7 d into a 14-d linear taper, the athlete would be undertaking 60% of their normal training, compared to 25% and 35% in fast and slow decay exponential patterns.

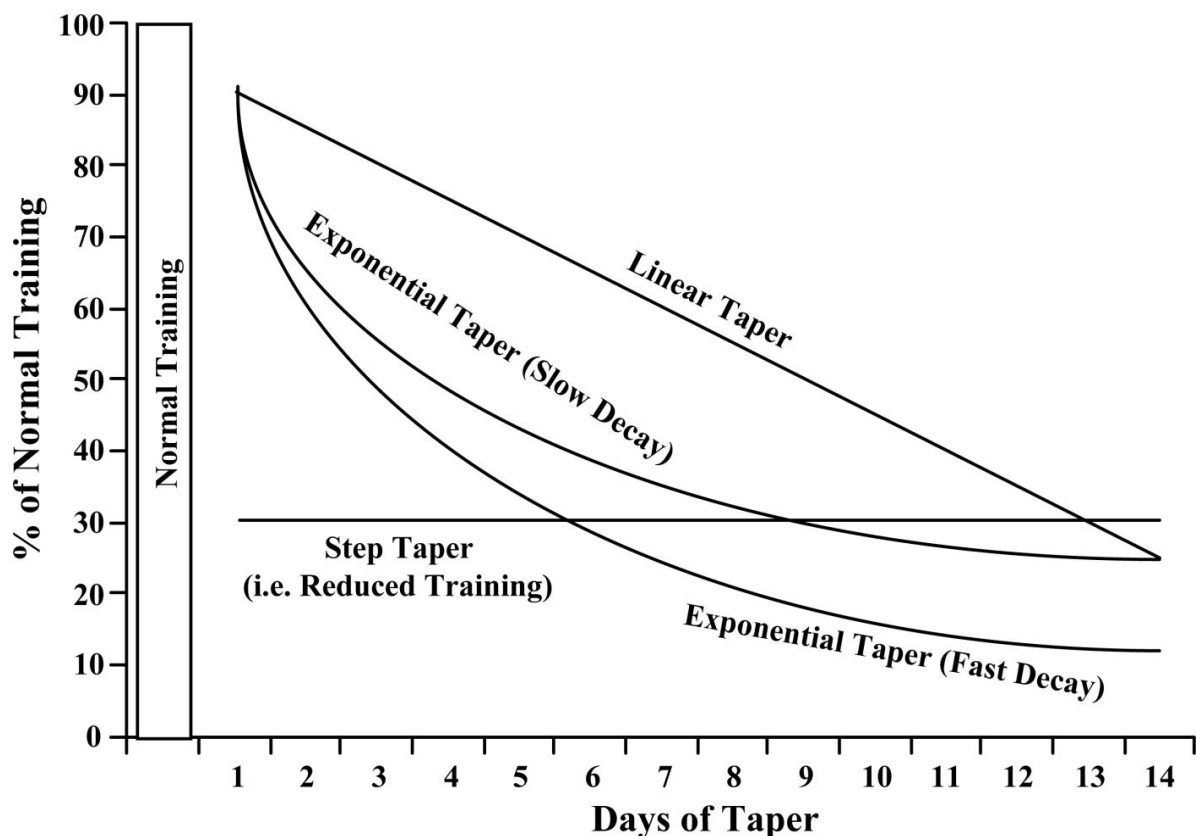


Figure 1.1. A schematic representation of the different tapering patterns: linear taper, exponential taper with slow or fast time constants of decay of the training, and step taper (figure from Mujika & Padilla, 2003).

1.3 Tapering Strategies and Performance

An optimal taper aims to maximise the elimination of accumulated fatigue from daily training, whilst retaining or further enhancing end-point performance (Bosquet et al., 2007; Mujika & Padilla, 2003). Previous research has demonstrated performance improvements of between 0.5 to 6.0% using competitive performance measures in response to tapering in running, swimming, cycling and triathlon (Mujika & Padilla, 2003). In a meta-analysis of the available literature, Bosquet et al. (2007) reported a mean performance improvement of 1.96% in competitive runners, swimmers and cyclists. Given this observation, the potential performance gain resulting from an optimal tapering strategy could prove extremely valuable for an athlete competing at the elite level. For example, the mean taper-induced improvement in swimming performance was 2.2% at the 2000 Sydney Olympic Games, in events ranging from 50 m freestyle to 400 m medley (Mujika et al., 2002). This was in excess of the differences between the gold medallist and the 4th placed swimmer (1.6%) and between the 3rd and 8th placed swimmers (2.0%) in these events.

The application of a taper in elite sport is largely the exclusive domain of the coach and athlete, and is predominantly based on their own empirical observations and experiences with little input from exercise scientists (Mujika et al., 2002). In the past, there has been a paucity of scientific literature attempting to predict the optimal taper and improve subsequent endurance running performance. However, there have been a number of studies (Houmard et al., 1990; Houmard et al., 1994; McConnell et al., 1993; Mujika et al., 2000; Mujika et al., 2002; Shepley et al., 1992; Wittig et al., 1989) that have intervened with various training manipulations in runners, as part of a structured taper (Table 1.1). Further tapering methods have been proposed by way of theoretical mathematical modelling (Thomas et al., 2009). It should be noted however, that few studies have isolated the effects of each training variable during the taper. This creates difficulty in identifying an optimal strategy, since the specific effect of 1 particular training variable on performance may be masked by the interaction with other training variables.

Table 1.1. Effects of tapering strategies on running performance.

Author (year)	Taper duration (d)	Participants	Training load variables	Performance measure	Performance outcome
Mujika et al. (2000)	6	Well-trained middle-distance runners	↓ volume 50% or 75%	800 m indoor time trial	No change
Mujika et al. (2002)	6	Well-trained middle-distance runners	↓ frequency 33% ↓ high intensity volume 80%	800 m indoor time trial	No change
Wittig et al. (1989)	21	Well-trained long-distance runners	↓ volume 70%	5 km race	No change
Houmard et al. (1990)	21	Well-trained distance runners ($\dot{V}O_{2\max}$ 61.8 ± 1.1 ml·kg·min ⁻¹)	↓ volume 70% ↓ frequency 17%	5 km indoor simulated race	No change
McConnell et al. (1993)	28	Well-trained distance runners ($\dot{V}O_{2\max}$ 63.5 ± 1.2 ml·kg·min ⁻¹)	↓ volume 66% ↓ frequency 50% ↓ intensity, all running <70% $\dot{V}O_{2\max}$	5 km indoor simulated race	Race time worsened by 1%
Houmard et al. (1994)	7	Trained distance runners ($\dot{V}O_{2\text{peak}}$ 55.3 ± 3.2 ml·kg·min ⁻¹)	↓ volume 85% ↑ intensity, all running as 400 m intervals at 5 km race pace	5 km treadmill time trial	3% improvement
Shepley et al. (1992)	7	Highly-trained middle-distance and cross-country runners ($\dot{V}O_{2\max}$ 66-71 ml·kg·min ⁻¹)	↓ volume 90% ↑ intensity, all running as 500 m intervals at pace equivalent to 115 – 120% $\dot{V}O_{2\max}$	Treadmill time to exhaustion at pace equivalent to season's best 1,500 m performance	22% improvement

1.3.1 Training Volume and Frequency

The work of Hickson et al., (1982) provided the first evidence that performance can be maintained with large reductions in training volume. After 10 weeks of continuous running and interval cycling training ($40 \text{ min}\cdot\text{d}^{-1}$, $6 \text{ d}\cdot\text{wk}^{-1}$) in recreationally active participants, daily volume was reduced by either by 33% (to $26 \text{ min}\cdot\text{d}^{-1}$) or 66% (to $13 \text{ min}\cdot\text{d}^{-1}$), with training intensity and frequency maintained for a further 15 weeks. In both groups, short-term endurance performance (time to exhaustion at $\dot{V}\text{O}_{2\text{max}}$) was maintained after the 15 week period, despite the reduction in volume. Prolonged endurance performance (time to exhaustion at 80% $\dot{V}\text{O}_{2\text{max}}$) was maintained in the 33% group, but declined by 10% in the 66% group. It is not clear however, how long prolonged endurance performance might have been maintained in the 66% group before the decline occurred.

Further literature on trained athletes suggests that dramatic reductions in volume during tapering do not compromise running performance for taper periods of up to 3 weeks. Using an extreme example, power output at the ventilatory threshold in response to an 8 week training programme was unchanged in cyclists after 4 d of complete rest (Neary et al., 1992). Mujika et al. (2000) found that 800 m running performance was unaffected by either a 50% or a 75% reduction in high and low intensity volume over 6 d. Over a longer 3 week taper period, training volume was reduced by 70% in a group of male distance runners (Wittig et al., 1989). Performance time was not different for a 5 km race following this strategy, suggesting that although performance can be maintained with a reduction in training volume, alterations to this training variable alone might not be the key to achieving an improvement in performance after a taper.

Performance time for a simulated 5 km indoor race was also unaffected by a reduction in training volume (70%) *and* frequency (17%) over 3 weeks in well-trained distance runners (Houmard et al., 1990). It was reported that 800 m and 1,600 m performance times were improved, however the results should be interpreted with caution, since the athletes only raced over 5 km and performance times for 800 m and 1,600 m were taken from 2- and 4-lap splits of the 5 km race. Since elapsed time was provided verbally every 400 m, athletes were likely to be motivated to improve on their time from the pre-taper 5 km and may have run the initial part of the race faster in an attempt to achieve this. This indicates that different pacing strategies were employed in the race and verbal indication of elapsed time may have confounded the effect of the tapering, as overall 5 km performance did not

improve. In addition, participants were homogenous for performance and were encouraged to race each other. Whilst this may increase the validity of the performance measure, it is not clear whether different tactical behaviours may have influenced the performance outcome.

Nevertheless, it was confirmed that a reduction in training volume and frequency during a 6-d taper maintains 800 m running performance in well-trained middle distance runners (Mujika et al., 2002). After 18 weeks of training, participants were assigned to either a moderate frequency taper (resting every third day) or a high frequency taper (daily training). Both tapers were characterised by a non-linear progressive reduction in high intensity training volume down to 20% of pre-taper levels. However, the moderate frequency taper represented a 33% reduction in training frequency through non-daily training. Time trial performance after the reduced volume, moderate frequency taper remained unchanged (126.6 ± 2.8 vs. 127.1 ± 2.1 s). However, performance time for 800 m improved by 1.27% following the reduced volume, high frequency taper (121.8 ± 4.7 vs. 124.0 ± 4.9 s; $P < 0.05$). Despite no difference in the overall volume reduction between the 2 groups, when the volume reduction was achieved by reducing training frequency, performance was not improved. This suggests that training frequency should not be manipulated as an alternative method to reduce training volume. In support, a 6-d taper involving a linear reduction in training volume down to 80% and incorporating a 17% reduction in frequency, did not improve 20 km cycling time trial performance (Neary et al., 2003). In addition, Bosquet et al. (2007) concluded from a meta-analysis that performance improvements are more sensitive to reductions in training volume, rather than to reductions in the other training load variables of intensity and frequency [effect size 0.72 ± 0.36 vs. 0.33 ± 0.17 and 0.35 ± 0.17 ($\pm 95\%$ confidence intervals)]. It was also suggested from the available literature on running, cycling and swimming that 41-60% represents the optimal reduction in training volume (Bosquet et al., 2007).

1.3.2 Training Intensity

Although performance can be maintained after a reduction in the volume of training, it seems that the preservation of intensity is fundamental to maintaining physiological adaptations and preventing a decline in performance. This was also demonstrated by the early work of Hickson et al. (1985), albeit in recreationally active individuals. After 10 weeks of continuous running and interval cycling training ($40 \text{ min} \cdot \text{d}^{-1}$, $6 \text{ d} \cdot \text{wk}^{-1}$), intensity

was reduced by either 33% or 66%, with training volume and frequency maintained for a further 15 weeks. The 33% group experienced a 9% reduction in $\dot{V}O_{2\max}$ by 10 weeks and a 21% reduction in prolonged endurance performance (time to exhaustion at 80% $\dot{V}O_{2\max}$) after 15 weeks. Although short-term endurance performance (time to exhaustion at $\dot{V}O_{2\max}$) was maintained in this group after 5 weeks, there was a marked reduction in the 66% group. The 66% group also experienced a greater reduction in $\dot{V}O_{2\max}$ than the 33% group and prolonged endurance performance was reduced by 30% after 15 weeks.

In 9 out of 10 well-trained distance runners, simulated 5 km race performance suffered a 1% decline (12 s) ($P < 0.05$) after 4 weeks of training at an intensity $< 70\%$ $\dot{V}O_{2\max}$ (McConnell et al., 1993). It was reported that the runners would usually complete approximately 76% of the total training distance at intensities $> 70\%$ $\dot{V}O_{2\max}$ in a normal training week, again suggesting that the maintenance of intensity is important during tapering. Weekly training volume was also reduced by approximately 66% and training frequency was reduced by 50% in this study. However, sub-maximal and maximal treadmill tests were performed the day before the 5 km performance tests and although this was also the case before the baseline performance test, it may have confounded the effects of the tapering strategy. Training was not supervised, although all training prescribed during the tapering phase was individualised. This finding recommends that training intensity must not be compromised during the tapering period and the maintenance of high intensity training is necessary protect performance from deteriorating when large reductions in volume and frequency are also implemented (McConnell et al., 1993).

Trained young swimmers underwent a training programme consisting of approximately 5,800 m·d⁻¹, over 6 d·wk⁻¹ for 8.5 weeks (Papoti et al., 2007). Daily training involved a combination of slow/regeneration (~10%), moderate (~70%), heavy (10%) and severe (~10%) intensities. The 11-d taper that followed was characterised by a non-linear progressive reduction in training volume (48%), without alteration to frequency and training intensity. Performance over a 200 m time trial was improved by 1.6% ($P < 0.05$), supporting the notion that an optimal reduction in training volume (Bosquet et al., 2007), combined with the maintenance of high training intensity can increase performance.

In further support of the inclusion of high intensity training during tapering, a 3% improvement in 5 km treadmill time trial performance ($P < 0.05$) was evident after a 7-d taper, whereby *all* running (85% reduction in volume) was completed as interval training

at $\sim 100\% \dot{V}O_{2\max}$ (Houmard et al., 1994, Figure 1.2). Intervals were 400 m unless other distances (100 m and 200 m) were required to meet the calculated training distance. Although it was stated that participants usually completed 6-10% of total weekly volume as high intensity training during normal training, it was not reported whether the intensity prescribed ($\sim 100\% \dot{V}O_{2\max}$) was higher than in normal training. It is also worth noting that a treadmill time trial may not mimic a legitimate race situation and lacks logical validity, despite controlling for environmental conditions that may influence results on an outdoor track. Participants were required to indicate to investigators when a change in speed was desired, which again limits validity of the test. Elapsed time was given regularly throughout the time trial which may have introduced bias toward the post-taper trial where athletes were likely to be more motivated to improve and may have altered their pacing strategy. Treadmill time trials also appear to be less reliable than field-based running time trials (Hopkins et al., 2001), perhaps since athletes are typically less familiar with treadmill running. Training during the tapering period was carried out on a 400 m track, heart rate was monitored and all training was supervised. This suggests that the athletes completed the exact training prescribed by the investigators with precision during the taper, which adds to rigor to this investigation.

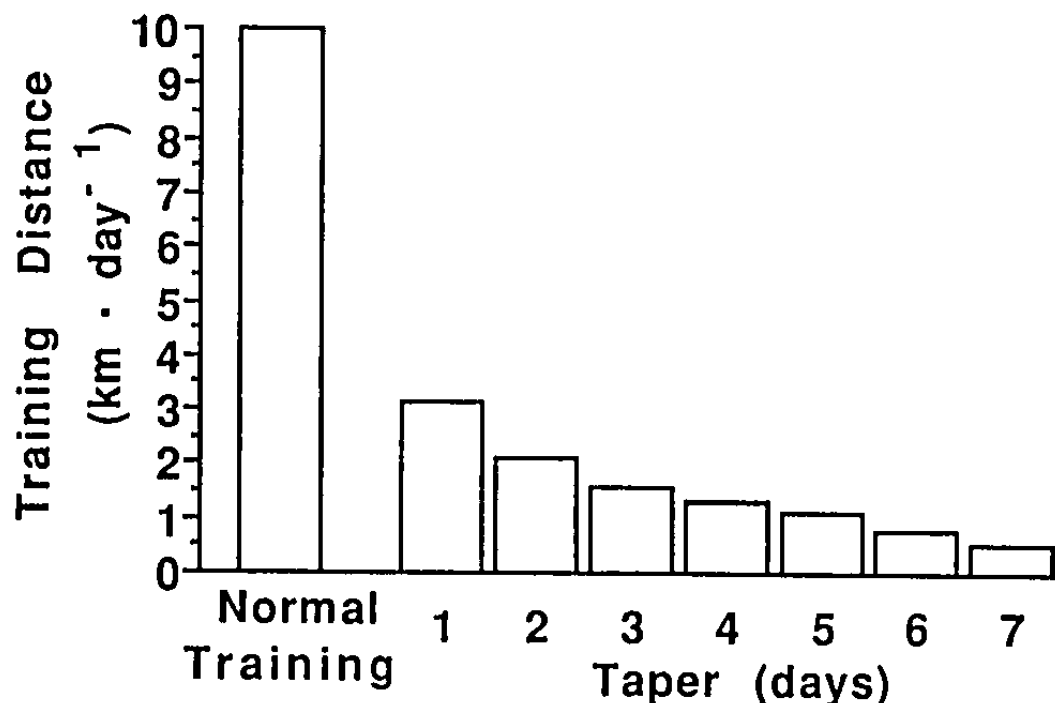


Figure 1.2. Example of taper training volume for an individual running $10 \text{ km} \cdot \text{d}^{-1}$ during normal training. Weekly training volume was reduced by 85% in total and all running volume during the taper was performed as 400 m intervals at 5 km pace or slightly faster ($\sim 100\% \dot{V}O_{2\max}$) (figure from Houmard et al., 1994).

Whilst the completion of high intensity training during the taper appears to be important, it was demonstrated that an increase in intensity during the taper compared to regular intensity, could have profound positive effects on performance (Shepley et al., 1992). In highly-trained cross-country runners, interval training intensity prior to tapering was approximately 95-100% $\dot{V}O_{2\max}$ ($\sim 25 \text{ km}\cdot\text{wk}^{-1}$) (Shepley et al., 1992). During the taper, intensity was elevated to 115-120% of $\dot{V}O_{2\max}$, in a low-volume strategy ($7.5 \text{ km}\cdot\text{wk}^{-1}$; $\sim 90\%$ reduction from regular training volume), which consisted of 3-5 500 m runs $\cdot\text{d}^{-1}$ with a recovery period of ~ 7 min. The 7-d taper improved running time to fatigue at 1,500 m speed by 22% (~ 70 s; $P < 0.05$), although the performance outcome measure lacks logical validity and is therefore recognised as a limitation to the study. Time to exhaustion protocols have also been found to be less reliable than time trials (Currell & Jeukendrup, 2008; Laursen et al., 2007). Despite this, the study also found that running time to fatigue was unaffected by a low intensity, moderate-volume taper and by a rest-only taper (+6% and -3%, respectively). The study employed a repeated-measures design and each participant performed each of the 3 tapering strategies in a random order, separated by 4 weeks of normal training. Randomisation removes potential for order effects on the performance outcome and the repeated measures design excludes the influence of individual differences that might occur with independent groups (e.g. age or performance standard). The coach of the athletes taking part was a co-investigator in the study and supervised all training to ensure it was completed as instructed. Participants did not receive any feedback regarding their performance times until the entire investigation was complete to avoid providing incentive to improve and confounding the effects of the taper.

It is clear that the preservation of high intensity training during the tapering period is crucial to prevent a decline in performance, but evidence points to an augmentation in intensity during this period to improve performance. It would seem that after large reductions in training volume, the remaining volume should be completed as high intensity intervals at a pace equivalent to, or above $\dot{V}O_{2\max}$ to enhance performance. It is questionable however, whether all volume during the taper must consist of high intensity intervals and the lower intensity continuous running discarded, since the majority of volume during regular training is usually comprised of this type in endurance runners (Smith, 2003). There is a need therefore, to further investigate high intensity strategies and to assess the outcome using a field-based performance measure.

Considering the evidence discussed above for differing manipulations to the component parts of training load (frequency, volume and intensity), it could be suggested that the definition of tapering in terms of a ‘reduction in training load’ is too simplistic. Whilst this definition could imply that *all* training load variables are reduced during the taper, in fact there are intricacies around how each variable should be manipulated (if at all), in order to optimise performance.

1.3.3 Pattern of the Taper

Several patterns of tapering have been investigated in an attempt to establish the optimal strategy to improve performance. Figure 1.3 provides an example of the pattern in which the constituent parts of the training load might be adjusted during the taper.

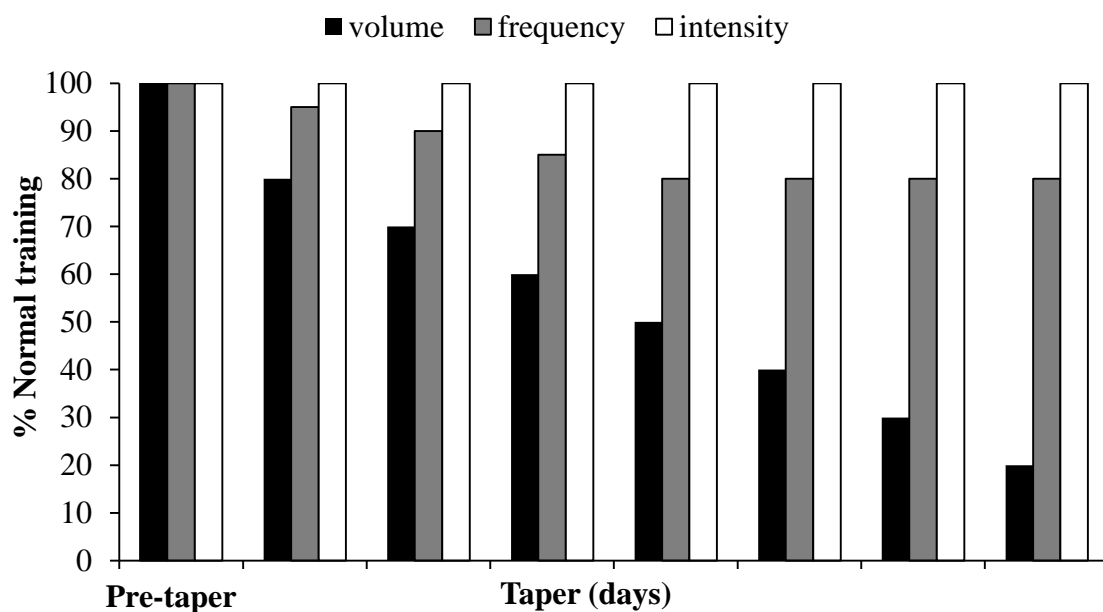


Figure 1.3. A schematic example of a tapering strategy, whereby training volume is reduced in a linear pattern, training frequency is reduced linearly initially before plateauing and training intensity is maintained throughout.

Investigations in the literature have implemented a number of different patterns to alter training load during tapering, however only 1 intervention study has compared the patterns in relation to performance. A systems model was first used to predict performance from simulations of different taper profiles: i) step taper vs. exponential decay taper, and ii) fast vs. slow exponential decay tapers (Banister et al., 1999). The simulations indicated that an exponential decay taper was superior to a step taper and a fast decay exponential taper was

more effective than a slow decay exponential taper, in optimising performance. The authors then attempted to validate the results in highly-trained triathletes. After a period of intensive training, participants tapered their training volume for 2 weeks either exponentially or via a step taper. Frequency was maintained, with the exception of 1 less training session in the final taper week. Intensity of training was maintained throughout the taper period. An exponential taper resulted in a 4% improvement in 5 km running performance, whereas a step taper produced a non-significant improvement of 1.2%. Participants then resumed training for a further 6 weeks of intensive training. A 13-d exponential taper was subsequently performed, in which the time constant of decay in training load was either fast ($\tau = 4$ d) or slow ($\tau = 8$ d). It was concluded that a fast exponential taper was a more effective pattern to reduce training volume than a slow decay exponential taper (6.3% vs. 2.4% improvement in 5 km time trial performance, respectively). Although the mechanisms are unknown, it is likely that a fast decay exponential taper provides more time for the athlete to overcome the fatigue accumulated during the last weeks of intensive and extensive training prior to the tapering period. Further validation is necessary however, since the performance measure was not well controlled. Participants selected their own outdoor 5 km running course and it is possible that environmental conditions may have differed between trials. It is also unknown as to whether the chosen courses were measured and timed accurately and if participants were supervised during the performance tests.

Contrary to traditional tapering patterns, an investigation using mathematical modelling of data from national and international-level swimmers studied a more complex ‘two-phase’ pattern (Thomas et al., 2009). Computer simulations were employed to investigate the potential performance benefit of an *increase* in training load during the final days of the tapering period (Figure 1.4). Training load was quantified in arbitrary units for work done, as described previously by Busso (2003). In brief, a criterion performance test to measure mean power during 5 min all-out exercise was carried out and arbitrarily ascribed 100 training units (TU). Each 5 min bout of exercise during training thereafter was weighted by intensity relative to the 5 min test (i.e. mean power output/5 min power $\times 100$). Training load for a session comprised of 5 bouts of exercise at 85% of 5 min mean power for example, would be calculated as 425 TU. Linear and two-phase tapers lasting approximately 5 weeks were compared after 28 d of training overload at 120% of normal training load. Both tapers were identical until the final 3 d of a two-phase taper, during

which the training load was significantly increased by $29 \pm 42\%$. Although the mathematical model predicted that the negative influence of fatigue was completely removed during both tapers and the positive influence of adaptation to training was enhanced slightly further during a two-phase tapering strategy, performance was improved similarly after both strategies ($\sim 4.0\%$, $P < 0.05$). It was suggested that the two-phase taper might further optimise performance if the 2nd phase contained more work specific to the requirements of the upcoming competition, rather than an increase in training load *per se*. For example, it has been observed anecdotally that performance often progressively improves from the first round of a major competition to the final (Thomas et al., 2009). However, this could not be considered using the modelling procedure in this instance, since the quantification of training load required aggregation of the individual components (volume, intensity, etc.) and therefore individual contribution to the increased training load in the 2nd phase was not clear.

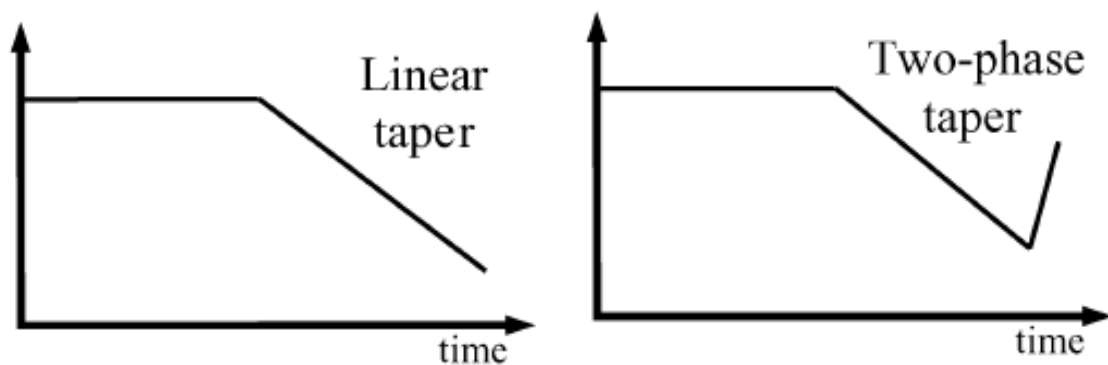


Figure 1.4. A schematic representation of linear and two-phase tapering patterns. Both protocols were characterised by a training overload at 120% of normal training for 28 d, followed by a linear taper for approximately 5 weeks. In a two-phase pattern, training load was abruptly increased in the final 3 d of the taper (figure from Thomas et al., 2009).

This supports the findings of Bosch & Mendonca (2008), who studied performance in competitive swimmers following 2 different tapering strategies in a crossover design. The traditional taper maintained pre-taper volume for the first 5 days, followed by a 50% reduction over 7 days prior to competition. The modified taper consisted of a 50% reduction in volume over the first 7 days, followed by a gradual increase back to pre-taper volume over the next 4 days and a low-volume recovery session the day before competition. Total training volume over the 12 days before competition was not different

between the two approaches (11,400 m). There was no difference in 200m swim performance between the two strategies, confirming that an increase in volume in the 2nd phase of the taper was not more beneficial than traditional tapering patterns.

Given the evidence for large reductions in training volume during tapering (Bosquet et al., 2007; Houmard et al., 1990; Mujika et al., 2000; Neary et al., 1992; Wittig et al., 1989) and the inclusion of high intensity training to improve performance (Houmard et al., 1994; Papoti et al., 2007; Shepley et al., 1992), it is possible that the two-phase taper might be more effective if volume remains reduced and intensity is increased in the final days before competition. Further research is required to validate such a novel tapering pattern in the field and to understand the underlying physiological mechanisms. In particular, the time-course of the stress-adaptation response during the 2nd phase of the taper in order to maximise performance on the day of competition.

1.4 Taper Duration

A taper period of 8-14 d appears to be optimal in endurance running, although strategies lasting from 6 d to 4 weeks might still be effective in some athletes (Bosquet et al., 2007). Identifying the optimal taper duration poses perhaps one of the greatest challenges for athletes and coaches, and anecdotally many feel insecure at the prospect of reducing training prior to competition. This anxiety is based on the premise that a prolonged reduction in training load during the taper may result in detraining. Detraining has been defined as the partial or complete loss of adaptations to training resulting from an insufficient training stimulus, which may take place during short periods of training cessation or from a significant reduction in habitual training load (Mujika & Padilla, 2001a). Athletes and coaches must take care not to tip the balance between tapering and detraining, since the latter is characterised by losses in cardiorespiratory, metabolic and muscular adaptations to endurance training which are likely to be detrimental to performance (Mujika & Padilla, 2001a; Mujika & Padilla, 2001b).

1.4.1 Risk of Detraining

It has been reported that $\dot{V}O_{2\max}$ is unaffected by 10 d of complete training cessation in competitive male distance runners (61.2 ± 5.6 vs. 61.3 ± 6.2 mL·kg·min⁻¹) (Cullinane et al., 1986). Training volume was at least 80 km·wk⁻¹ in the year preceding the study and was 112 km·wk⁻¹ in the 2 weeks immediately before. In contrast, $\dot{V}O_{2\max}$ declined by 7% after 12 d of inactivity in endurance athletes, compared to values obtained in the trained state

(57.7 ± 2.6 vs. 62.1 ± 3.3 mL·kg⁻¹·min⁻¹; $P < 0.05$) (Coyle et al., 1984). An association was evident between trained $\dot{V}O_{2\max}$ and percentage decline in $\dot{V}O_{2\max}$ with training termination, whereby participants with the highest $\dot{V}O_{2\max}$ values prior to training cessation experienced the greatest declines ($R^2 = 0.93$; $P < 0.001$). In support, previously sedentary individuals were trained for 12 weeks and then divided into 3 groups to either: i) maintain training; ii) reduce training frequency by approximately 50%; or iii) completely terminate training for 2 weeks (Houmard et al., 1996). All groups experienced improvements in $\dot{V}O_{2\max}$ and time to exhaustion (both $P < 0.05$) during an incremental treadmill test after the period of training. Furthermore they were able to retain the training induced gains in $\dot{V}O_{2\max}$ and performance, despite 2 weeks of reduced training or complete inactivity. This supports the findings of Hickson et al. (1982), whereby recreationally active individuals could maintain the improvements in $\dot{V}O_{2\max}$ (10-20%) gained from the previous 10 weeks of training for up to 15 weeks of reduced volume (33% and 66%) training. However, in a group of endurance-trained runners, 14 d of training cessation resulted in a 4.6% reduction ($P < 0.05$) in $\dot{V}O_{2\max}$ (Houmard et al., 1992). The period of inactivity was accompanied by a decline in performance, whereby time to exhaustion decreased during an incremental treadmill test (11.8 ± 0.5 min vs. 13.0 ± 0.5 min; $P < 0.001$). A concomitant decrease in resting plasma volume (5.1%) was also observed in the endurance-trained runners. This may somewhat explain the reduction in $\dot{V}O_{2\max}$, since a decline in plasma volume might cause a reduction in stroke volume. Maximal heart rate increased ($P < 0.001$) after training cessation, in a possible attempt to offset a fall in stroke volume and maintain cardiac output. Despite this, $\dot{V}O_{2\max}$ was still found to decline and therefore heart rate did not increase sufficiently to fully compensate for changes in stroke volume (Houmard et al., 1992).

It would seem that recently gained aerobic adaptations to training can be readily retained with reduced training or training cessation in previously sedentary individuals, compared to highly trained athletes whose extensive training history leads to more developed aerobic capabilities. The evidence presented suggests that 10 d could be the upper limit for retention of $\dot{V}O_{2\max}$ in highly trained athletes who have ceased training and that a reduction in plasma volume might be a contributing factor. However, the time course of cardiorespiratory adaptation-loss in highly trained individuals when training is sustained, but at a reduced load during tapering remains uncertain.

There is evidence for a shift in myosin heavy chain (MHC) isoform expression from type IIx and hybrid type IIa/IIx to type IIa, either through resistance or endurance training (Andersen et al., 1994; Andersen & Aagaard, 2000). Inactivity can reverse this process however, resulting in an increase type IIx MHC isoform expression (Staron et al., 1991). Short term training cessation did not result in changes in the muscle fibre distribution of 6 highly-trained endurance runners after wearing a plaster cast for 7 d and refraining from training for a further 8 d (Houston et al., 1979). In addition, Houmard et al. (1992) found that cross-sectional area (CSA) of type I and type II muscle fibres was not different after 14 d of training termination in endurance runners and fibre type distribution remained unchanged. This might suggest that tapering periods with severely reduced training of up to approximately 2 weeks will not induce changes in muscle fibre type, or cause a reduction in mean fibre CSA. After longer period of 4 weeks training cessation however, muscle power has been shown to decline (Neufer et al., 1987).

Metabolic changes have been noted after 14 d of training cessation, indicated by an increase in respiratory exchange ratio (RER) at submaximal (4.3%) and maximal (2.9%) exercise intensities (Houmard et al., 1992). Three weeks of training cessation can result in a decrease in skeletal muscle mitochondrial enzyme activities (Mujika & Padilla, 2001b). The activity of citrate synthase, succinate dehydrogenase, β -hydroxyacyl-CoA dehydrogenase and malate dehydrogenase declined by approximately 20% (Coyle et al., 1984; Coyle et al., 1985). Collectively, this evidence points to a shift toward increased carbohydrate metabolism after the removal of training stimuli, which is perhaps due to increased muscle glycogen storage. The potential impact on competition performance is unknown, although it could be speculated that reduced oxidative enzyme activity may have a negative impact on fractional utilisation of $\dot{V}O_{2\max}$.

Much of the detraining literature has investigated the effects of complete training cessation and even immobilisation on training-induced physiological adaptations. It is therefore unknown as to the time-course of detraining when the athlete remains training, but at a tapered level. Despite a decline in a small number of training-induced adaptations after 12-14 d of inactivity, it could be speculated that some level of training stimulus from the taper training load would attenuate or even negate such declines over this duration, whilst still allowing the positive outcome of fatigue elimination.

1.4.2 Prior Training Load

It is also necessary to consider whether the optimal taper duration is influenced by the preceding training load (Kubukeli et al., 2002; Smith, 2003) and the amount of fatigue carried into the taper (Bosquet et al., 2007). However, few experimental studies have attempted to investigate this question, perhaps due to the difficulty in quantifying the multifaceted manifestation of fatigue. Existing literature on overreaching and overtraining acknowledges a lack of physiological tools capable of accurate diagnosis and prevention (Halsen & Jeukendrup, 2004; Urhausen & Kindermann, 2002). Despite this, work by Thomas et al. (2008) using mathematical modelling offers theoretical insight into prior training load and implications for the design of the tapering strategy. Using computer simulations of training and performance data from elite 100 m and 200 m swimmers over 2 complete seasons, it was predicted that a 20% increase in training load for 28 d prior to the tapering period would require a longer taper duration to improve performance compared to after regular training (22.4 ± 13.4 d vs. 16.4 ± 10.3 d; $P < 0.05$). More recently however, Aubry et al. (2014) observed that peak performance was achieved within 2 weeks of tapering in triathletes, regardless of prior training load (i.e. overload or not) or whether they were diagnosed as functionally overreached or not prior to the taper. However, performance was assessed using an incremental cycling test to exhaustion, rather than a competition measure. It is still necessary therefore, to establish clarity on the time course of fatigue elimination in endurance runners for the peaking of performance.

Additionally, it is important to consider whether a taper is *always* necessary before a competition. The aim of a taper is to alleviate accumulated fatigue and maintain or further enhance physiological adaptation (Mujika & Padilla 2003). However, there may be circumstances in which athletes are not suffering from the level of fatigue that would cause an acute suppression in performance and warrant a taper period. For example, if an athlete is prevented from training to their maximum capacity through injury or illness prior to competition and residual fatigue from earlier heavy training has already been alleviated.

Alternatively, it may be counterproductive to implement an optimal taper before every competition of the season. Many sports now have busy competitive calendars before the season culminates with a major championship, due to an increase in commercial sponsorship and therefore increased opportunity for financial gain (Pyne et al., 2009). No study has experimentally investigated tapering strategies for multiple peaking, although it can be speculated that implementing optimal taper periods too frequently could comprise

long-term adaptation to training. Thus limiting the performance level that can be achieved at the most important competition at the end of the season. When competitions occur frequently, it may be necessary to implement short-duration ‘mini tapers’ before minor events (Tønnessen et al., 2014) and prioritise 2-3 important competitions to carry out an optimal taper (e.g. qualification event for major championships and major championships event) (Le Meur et al., 2012). Further research is required to determine how tapering strategies should be individualised and planned, with careful consideration of prior training load and peaking for the most important competition of the season.

1.5 Possible Physiological Mechanisms of Tapering on Performance

1.5.1 Insights from Mathematical Modelling

The physiological mechanisms underlying the observed improvements in performance after tapering are not yet well understood. Insight might be gained from mathematical modelling, whereby mathematical analysis of the relationship between training load and performance has been conducted to facilitate understanding. Banister et al. (1975) developed a complex systems model to understand the fluctuation of athletic performance throughout periods of heavy training and tapering, estimated from the difference between two antagonistic transfer functions; a negative function representing fatigue and a positive function to represent fitness. In theory, a period of intensified training would result in a decline in performance due to an increase in fatigue that exceeds the gain in adaptation and fitness. A reduction in training thereafter, would alleviate fatigue more quickly than fitness would decline, thus resulting in an improvement in performance. In brief, daily training load $[w(t)]$ is converted by the parameter vector of the model using two multiplying factors (k_1 and k_2) into fitness and fatigue impulses. Two time constants (τ_1 and τ_2) represent the decay in accumulated fitness and fatigue, respectively and occur at different rates. The following parameters of the model parameter vector have been suggested; $k_1 = 1$, $k_2 = 2$, $\tau_1 = 45$ d and $\tau_2 = 15$ d (Fitz-Clarke et al., 1991; Morton et al., 1990). The systems model can predict variations in performance at any time $p(t)$ based on training dose $[w(t)]$. More recent extensions of the model have contributed to greater understanding of certain aspects of the taper, including the optimal pattern of the taper (Banister et al., 1999, described in section 1.3.3). Mujika et al. (1996) investigated the effect of training and taper duration on performance in elite swimmers. The goodness of fit between the actual performance achieved by the swimmers at different times in the season and the modelled performance was statistically significant for 17 out of 18 individuals. The 3 and 4 week progressive

tapering periods resulted in 3% improvements in competition performance, which with the use of the model, the authors attributed to a significant reduction in negative influence of training, without comprising the positive influence of fitness, over this taper duration.

Banister's model however, assumes that fitness and fatigue impulses are always proportional to training load and that the parameters remain consistent over time. This concept fails to consider the impact of previous training on the magnitude and duration of fatigue induced by an acute training session. A recursive least squares algorithm was used to explore this and it was shown that the negative effect of a training bout is exacerbated when training intensity (Figure 1.5; Busso et al., 1997) and training frequency (Busso et al., 2002) are increased. The Variable Dose-Response Model was subsequently proposed by Busso (2003) and attempts to describe performance responses to varying training loads over time. The development in this model is represented by a gain term for the negative function of fitness that varies with training load according to a first order relationship. Using training and performance data from 6 healthy individuals, goodness of fit of performance was improved with the variable dose-response model, compared to previous models of Banister et al. (1975), Calvert et al., (1976) and Busso et al. (1991) which used time-invariant gain terms for the positive and negative influences of training. Model simulations showed that the transient decreases in performance with intensified training could be as a result of a change in the response to the training dose. For example, a particular training session could be more difficult for the athlete to manage when overall training load is intensified, but a progressive reduction in training load would allow the athlete to respond more effectively to the same session or complete a more intense or prolonged session. Improved performance as a result of the tapering period may therefore be attributed to both the recovery from previous training and restoration of the ability to respond to training. A combination of delayed physiological responses to previous training and early reaction to training done during the taper is likely to facilitate enhanced performance (Mujika et al., 2004). Experimental data on athletes and their responses to heavy training and tapering offers support for this theory (Halsen et al., 2002). Following 2 weeks of normal training, training was intensified for 2 weeks in endurance-trained cyclists. Power output and performance time in a simulated time-trial, whereby target amount of work was to be completed in the shortest possible time, declined ($P < 0.05$) after the onset of the intensified training period, and further fatigue and performance decrements occurred when the intensive training stimuli continued. When training load

was subsequently reduced, time-trial performance improved by 2% from baseline in the 2nd week of the recovery period. This may also help to explain the findings of Thomas et al (2009) where mathematical modelling of a two-phase taper based on elite swimmers resulted in significant improvement in performance.

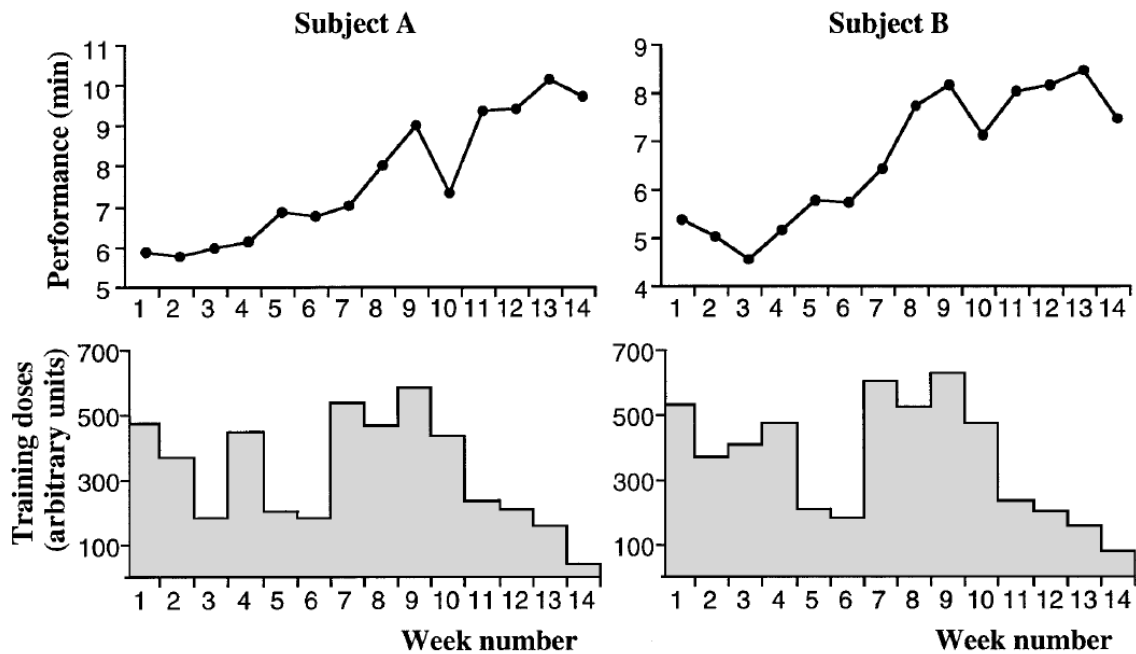


Figure 1.5. Variation in training dose (estimated from training intensity) and change in performance response over each week of training in 2 individuals (figure from Busso et al., 1997).

Whilst mathematical modelling offers insight into the effects of training on performance, there are several limitations to this approach and findings should be considered with caution. Firstly, existing models require a single system input to represent training load, typically calculated from volume weighted by intensity. This limits the scope of the model in not accounting for and over-simplifying the diversity of training completed by elite athletes (Hellard et al., 2006). It has been suggested that a sport-specific multifactorial model with several inputs would provide a clearer understanding of the effects of different types of training on performance, although this approach would be more complex, time consuming and perhaps not realistic for elite athletes (Busso & Thomas, 2006). Performance, the system's output, can be defined more easily. The challenge arises however, in achieving an adequate number of performance observations across all periods of training, which is not always feasible in elite athletes. For multiple linear regression, 15

observations per parameter is recommended (Stephens, 1986), but since the models of Banister and Busso are non-linear, more performances per parameter are required (Hellard et al., 2006). In the study by Mujika et al. (1996) on elite swimmers for example, the mean number of measured performances was 18. This perhaps explains the reason for many of the models being developed on small sample sizes, using data from non-elite participants and therefore limits application to elite athletes. Regardless, it is not recommended that models from the literature are applied to individuals and used to predict training (Busso and Thomas, 2006). Mathematical models are also unable to discern the complex physiological processes that are involved in training adaptation and insight from modelling is limited to understanding the dynamic and temporal changes in performance during and after intensification (Busso and Thomas, 2006).

1.5.2 Muscle Glycogen

The importance of muscle glycogen as a metabolic substrate for endurance exercise has been widely acknowledged since the 1960's, after the development of the muscle biopsy technique (Bergström & Hultman, 1966; Bergström et al., 1967). The rate of muscle glycogen utilisation increases with exercise intensity and is the primary source of fuel in the early stages of exercise and for intensities exceeding $\sim 75\%$ $\dot{V}O_{2\max}$ (Hargreaves, 1996; Hermansen et al., 1967; Romjin et al., 1993). The ability of muscle glycogen to produce a greater energy yield per unit of O_2 and a more rapid rate of ATP resynthesis compared to fat and protein perhaps explains why it remains the preferential fuel for the maintenance of moderate to intense exercise. Relative to fat and protein however, muscle glycogen stores are finite and become severely reduced or even depleted as a result of prolonged submaximal exercise, such as marathon running (Sherman et al., 1983). In response to high intensity intermittent exercise, it has also been demonstrated that rapid depletion of muscle glycogen occurs in type II fibres in particular (Gollnick et al., 1973). Although it has been suggested that periods of training with low muscle glycogen stores might stimulate other physiological adaptations beneficial to endurance performance (Hansen et al., 2005; Hawley & Burke, 2010; Yeo et al., 2008), it is well known that sub-optimal muscle glycogen is associated with a reduced capacity to perform both submaximal (Bergstrom et al., 1967) and supramaximal exercise (Maughan & Poole, 1981). As such, to ensure optimal endurance performance, athletes are encouraged to maximise the availability of muscle glycogen prior to competition.

During periods of heavy training however, athletes frequently undertake training sessions with low muscle glycogen availability either by intent (Stellingwerff, 2013) or by circumstance, often through multiple training sessions in a day (Philp et al., 2012). Successive days of intense prolonged training without adequate carbohydrate consumption can result in a gradual reduction of muscle glycogen content and deterioration in performance (Costill et al., 1971). Even with optimal carbohydrate intake, glycogen stores are replenished at a rate of approximately 5-7% per hour, therefore requiring up to 20 h to re-establish stores following a glycogen depleting training session (Coyle & Coyle, 1993). The tapering period may provide the opportunity for athletes to replenish muscle glycogen as a result of a reduced training load and consequently, lowered glycogen utilisation and greater recovery time between training sessions. In cyclists, muscle glycogen concentration increased by 17% and 25% after 4 and 8 d of tapering, suggesting that changes occur progressively with taper duration (Neary et al., 1992). The same group also found that a 7-d taper in cyclists, where training volume was gradually reduced (by ~60%) and intensity maintained (85-90% maximal heart rate), improved muscle glycogen and 40 km time trial performance to a greater extent (34% and 4.3%) than a 7-d taper where training volume was maintained and intensity was gradually reduced (to 55% maximal heart rate) (29% and 2.2%; Neary et al., 2003). Both performance time and muscle glycogen concentration were unchanged in the control condition, where regular training load was maintained (Neary et al., 2003). Similar findings were evident in endurance runners, where a low volume, high intensity 7-d taper resulted in a 15% increase in muscle glycogen and 22% improvement in treadmill time to exhaustion at 1,500 m race speed (Shepley et al., 1992). In comparison, a moderate volume, low intensity taper did not result in increased muscle glycogen and the margin of improvement in performance was much smaller (6%; Shepley, et al., 1992).

There is also evidence that muscle glycogen 'supercompensation' can occur with dietary manipulation (Bergstöm et al., 1967; Goforth et al., 1997; Madsen et al., 1990). The traditional carbohydrate loading strategy however, involved a muscle glycogen depleting exercise bout, followed by 2-3 d of low carbohydrate intake (depletion phase), before a similar period of high carbohydrate intake (supercompensation phase) (Bergstöm et al., 1967). Whilst muscle glycogen concentration has shown to supercompensate by up to 100-fold and improve exercise capacity compared to a mixed diet (Karlsson & Saltin, 1971), this strategy could limit the intensity and responses to training during the depletion phase

and could be a risk to athletes close to competition, due to potential increased susceptibility to illness from restricting carbohydrate after exhaustive exercise (Gleeson et al., 2004). However, it was subsequently evident that supercompensation could occur in trained athletes by merely reducing training load and consuming a high carbohydrate diet (> 50% total energy from carbohydrate) (Costill et al., 1981; Sherman et al., 1981). For example, muscle glycogen concentration in trained female athletes was 13% greater after a 7-d taper coupled with a high carbohydrate diet compared to a moderate carbohydrate diet (Walker et al., 2000). This was also associated with elevated carbohydrate oxidation, RER values and blood lactate concentration during cycling to volitional exhaustion at 80% $\dot{V}O_{2max}$, which was also improved (Walker et al., 2000). In addition, carbohydrate loading has also been shown to improve performance in time trial performance tests (Rauch et al., 1995; Williams et al., 1992).

Whilst high glycogen availability is desirable in the competition setting where it may contribute to optimal performance (Hargreaves, 2004; Hawley et al., 1997; Temesi et al., 2011), athletes should take care to match energy intake with the reduced energy expenditure consequential of a taper period, to avoid changes in body composition that might be detrimental to performance (Mujika et al. 2004). It was evident for example, that triathletes did not modify their total energy or carbohydrate intake during 2 weeks of tapering compared to 4 weeks of intensified training and percentage body fat increased from 11.5% to 12.1% (Margaritis et al., 1990). Whilst this may not have been large enough to impact on performance, more substantial changes could be expected after longer tapers.

Nevertheless, these findings suggest that the taper provides an opportunity for athletes to optimise muscle glycogen stores, which may subsequently contribute to increased performance. Although a high carbohydrate diet and high intensity training appear to be key to increasing/restoring muscle glycogen levels during tapering, a prior depletion phase is not recommended.

1.5.3 Neuromuscular Changes

Increased strength and power have been commonly observed as a result of tapering and such increases have been correlated with improved performance in swimmers (Cavanaugh & Musch, 1989; Costill et al., 1985; Johns et al., 1992; Raglin et al., 1996), and in endurance-trained runners (Luden et al., 2010; Shepley et al., 1992). Although, some studies have reported no change in muscular force after tapering (Hooper et al., 1998;

Houmard et al., 1994). Equivocal findings might be due to variances in participant training status, the type of tapering strategy undertaken and differences in the methodology implemented to measure force production and rate of force development.

Increased maximal voluntary isometric strength of the knee extensors after both high intensity-low volume and low intensity-moderate volume tapers was found in cross-country and middle distance runners (Shepley et al., 1992). Percutaneously nerve-stimulated evoked contractile properties of the right knee extensors also improved during both tapers and the gains in peak twitch torque were 13% and 19%, respectively. Similar positive changes occurred in a rest-only taper group, but performance in a treadmill test to exhaustion at approximately 1,500 m velocity declined by 3% in this group. This suggests that improvements in performance following a taper are influenced by a multiplicity of physiological factors, rather than muscular strength and power alone.

Single muscle fibre analysis indicated that highly trained collegiate swimmers exhibited a 30% higher peak isometric force, a 67% faster shortening velocity and a 250% higher absolute fibre power in type IIa fibres of the deltoid muscle after a 21-d low volume taper (Trappe et al., 2000). Type I fibres also increased their shortening velocity by 32%. In support, Luden et al. (2010) found that peak force (11%) and absolute power (9%) of type IIa fibres in the gastrocnemius muscle were improved in distance runners after 3 weeks of tapering. The reported changes in contractile properties at the single fibre level are perhaps due to an increased packing density of myofibrillar proteins (Metzger & Moss, 1987), altered myosin ATPase activity and/or changes in calcium sensitivity (Trappe et al., 2000) and are likely to be related to the observed post-taper improvements in whole muscle strength and power.

It has been suggested that reduced training during tapering may allow for an increase in maximal tension development through changes in contractile mechanisms and/or neural controls on fibre recruitment (Costill et al., 1985). Increases in strength were observed despite an unchanged percentage motor unit activation, which may be due to positive changes in muscle fibre size during the tapering period (Shepley et al., 1992). This is supported by a study in male collegiate swimmers, where type IIa fibre diameter and cross sectional area (CSA) increased by 11% and 24%, respectively following a 3 week reduction in training (Trappe et al., 2000). Furthermore, tapers of between 7 and 21 d have been linked to increased type II CSA (Neary et al., 2003) and type IIa fibre diameter

(Luden et al., 2010). Changes in both protein synthesis and breakdown during tapering may explain the marked type II muscle fibre growth. Trained muscle typically displays higher rates of protein synthesis at rest (Pikosky et al., 2006), suggesting that heavy phases of training suppress the rate of muscle protein synthesis, as has been shown in rats (Seene et al., 2004). Conceptually, when the training overload is removed in the form of taper, protein synthesis can proceed at high rates. Furthermore, overtraining can also elevate rates of protein catabolism (Seene et al., 2004). Therefore, altered protein balance might also be contributed to by a decrease in the rate of protein breakdown with the removal of heavy training during the taper.

Although protein turnover was not measured, Luden et al. (2010) observed an exaggerated post-exercise gene response following 3 weeks of tapering. The growth-related MRF4 gene displayed an augmented exercise response following the taper. Conversely, the MuRF-1 gene was attenuated after tapering and is associated with proteolysis of myofibrillar proteins (Reid, 2005). This supports the notion that that muscle is less susceptible to myofibrillar breakdown when training load is reduced. In combination, this gene response is compatible with muscle hypertrophy and may explain the observed increase in size and function of type II fibres after tapering (Luden et al., 2010). A possible explanation is that high volume training in the months prior to tapering, of low intensity exercise in particular, induces negative changes to type II muscle fibre size, particularly since the activity of AMPK is increased after long duration exercise and inhibits the activity of mTOR and thus suppresses the rate of protein synthesis (Baar, 2006). This is supported by Fitts et al. (1989) who observed a 15% decrease in the type IIa fibre size of swimmers, after training volume was doubled for 10 d. In contrast, type I fibre size was unaltered after this period. Tapering may therefore have the reverse effect on type II fibre size, given the removal of a large proportion of low intensity training volume, whilst high intensity training is continued. Further research is required to investigate protein turnover during tapering and to study the time-course of the acute up-regulation of genes to the formation of proteins that cause greater adaptation and might contribute to improved performance.

Other potential mechanisms responsible for improved performance after tapering may be related to local changes in enzyme activity, which have a positive impact on metabolic efficiency (Mujika et al., 2004). In the high intensity-low volume taper group, Shepley et al. (1992) observed an 18% increase in citrate synthase activity after 7 d. The improvement

in time-to-fatigue running performance at 1500 m speed in this study was therefore attributed to an increased capacity to maintain a high rate of oxidative energy production, despite the potentially performance-limiting effects of increasing intracellular temperature, hydrogen ion and lactate concentration and superoxide free radicals. In support, a significant increase in cytochrome oxidase activity was evident post-taper in cyclists, providing an indication of enhanced respiratory capacity of the muscle (Neary et al., 1992).

At the single fibre level, a high intensity-low volume taper caused myofibrillar ATPase and succinate dehydrogenase in type I fibres to increase by 11% and 12%, respectively (Neary et al., 2003). In type II fibres myofibrillar ATPase, succinate dehydrogenase, β -hydroxyacyl CoA dehydrogenase and cytochrome oxidase increased by 15-16%. After a high volume-low intensity taper however, cytochrome oxidase (10%) and β -hydroxyacyl CoA dehydrogenase (17%) increased in type I fibres, but only β -hydroxyacyl CoA dehydrogenase (18%) increased in type II fibres. The metabolic changes that occur at the single fibre level as a result of tapering are likely to contribute to the observed differences in performance at the whole-body level. Similar to the changes in muscle fibre size, it is evident that the metabolic properties in endurance athletes are more responsive to a high-intensity taper. This may be due to the specific contractile properties of type II fibres and greater potential of this fibre type to increase their oxidative enzyme capacity (Neary et al., 2003).

The evidence presented suggests that muscular strength and power production might be attenuated during periods of intensive training, due to a combination of factors including altered protein synthesis and shifts in fibre type volume. However, muscle function is able to recover during the tapering period when training load is reduced, and therefore appears to be improved compared to pre-taper levels.

1.5.4 Hormonal Changes

It has been suggested that the measurement of plasma testosterone (T) and cortisol (C) could be indicative of changes in anabolic and catabolic tissue activity, respectively. The T:C ratio may represent the balance between anabolic and catabolic activity and a significant decrease could represent overstrain and under recovery (Adlercreutz et al. 1986), although findings in response to tapering are equivocal (Mujika et al., 2004). Flynn et al. (1994) found no difference in total T, free T, and total T:C ratio (T:C) or free T:C in male cross-country runners after a 3 week taper, with no performance benefit following the

taper, which suggests that the tapering strategy was not adequate to reduce previously accumulated training stress. Following a 6-d taper, the hormonal markers; total T, free T, C, total T:C and free T:C remained stable and were not associated with any changes in performance (Mujika et al., 2000). However, total T was found to correlate inversely with the low intensity continuous training distance covered during taper and positively with high intensity interval distance. In support, total T was significantly increased after either a moderate or high frequency taper of 6 d, where low intensity continuous training was removed from the main part of the training sessions (Mujika et al., 2002). The authors suggested an increase in total T was a result of the exclusion of low intensity continuous running for main part of training sessions during the taper. The mechanism responsible for this observation is perhaps initiated by an enhanced pituitary response to the prior period of heavy training, bringing about a positive influence on androgenic-anabolic activity during the subsequent taper (Mujika et al., 2002). Previous investigations have shown a delay in the relationship between luteinising hormone (LH) concentration (an indication of enhanced pituitary activity) during intense training and T levels during reduced training or tapering, characterised by a reduction in physiological stress (Busso et al., 1992; Mujika et al., 1996). More specifically, Busso et al. (1992) found that variations in LH concentration during 4 weeks of heavy training in weightlifters were positively associated with changes in the T:sex-hormone-binding-globulin ratio during 2 weeks of reduced training that followed ($R = 0.90$; $P < 0.05$). This relationship was also found in highly trained swimmers, where the percentage change in LH throughout 12 weeks of heavy training was positively correlated with the plasma concentration of non-sex hormone binding globulin bound T (the sum of free T and albumin-bound T) after 4 weeks of tapering ($R = 0.72$; $P < 0.05$) (Mujika et al., 1996). It was initially thought that positive changes in testosterone during tapering may mediate the increases in muscle fibre size observed after the tapering period (Sinha-Hikim et al., 2002). However, more recent evidence suggests that the small and transient changes in circulating testosterone induced by exercise are not associated with increases in lean body mass, hypertrophy or strength (West & Phillips, 2012).

Resting C can be chronically elevated during heavy endurance training (Fry & Kraemer, 1997; Houmard et al., 1990; Luger et al., 1987), which might be explained by the metabolic action of this hormone. Cortisol increases substrate availability by mobilising amino acids in the muscle and stimulating gluconeogenesis in the liver, thereby increasing hepatic glucose output, in addition to limiting glucose uptake into the muscle (Orth et al.,

1998). As a result of increased C, glucose utilisation in muscle is reduced, perhaps in an attempt to prevent severe depletion of muscle glycogen during periods of high-volume training (Bonifazi et al., 2000). This potentially protective effect may be linked to interleukin-6 (IL-6) release from the muscle, since infusion of recombinant human IL-6 (rhIL-6) at a concentration elicited after strenuous exercise resulted in a significant increase in C (Steensberg et al., 2003). Changes in resting C may therefore be indicative of the progressive increase in muscle glycogen storage observed with tapering, when training volume is reduced (Neary et al., 1992; Shepley et al., 1992). In support, over 2 seasons, the 1.5-2.1% improvement in major competition performance after tapering in elite swimmers was positively related to corresponding 22-49% increases in post-competition peak lactate and negatively correlated ($R = -0.66$) with the 19-29% change in resting pre-competition plasma C (Bonifazi et al., 2000). A similar conclusion was drawn by Mujika et al. (2002) following a 6-day high frequency taper, where strong correlations were found between changes in peak blood lactate after an 800 m running race and performance, serum C ($R = -0.75$), total T:C and free T:C ratios ($R = 0.82$). A reduction in plasma C with tapering therefore, is perhaps a prerequisite for increased muscle glycogen concentration and improved performance.

Collectively, this evidence suggests that the tapering period can create a hormonal milieu favourable to anabolic processes and particularly muscle glycogen storage. It not clear however, how the anabolic and catabolic processes would be affected in long distance and marathon runners, since a large proportion of their training volume during the tapering period is likely to consist of lower intensity continuous running.

1.5.5 Haematology

Trained athletes typically display elevated blood volume, haemoglobin mass and red cell volume compared to untrained individuals (Kjellberg et al., 1949; Heinicke et al., 2001). These adaptations are beneficial to endurance performance given their close relationship with $\dot{V}O_{2\max}$ (Levine, 1993; Schmidt & Prommer, 2010; Vanoverschelde et al., 1993). Increased blood volume improves venous return, which is necessary for increased stroke volume and subsequently, a high cardiac output (Coyle et al., 1990; Hopper et al., 1988; Kanstrup & Ekblom, 1982). In addition, it widely accepted that elevated red cell volume and haemoglobin mass reflects a greater capacity for O_2 to be transported between the respiratory surfaces and metabolising tissues (Jensen, 2009).

Tapering has been shown to increase total blood volume and red cell volume after a high intensity-low volume strategy (Shepley et al., 1992). Despite the taper duration in this instance representing the minimal time-course for the maturation of erythrocytes (Israels & Israels, 2006), it is possible that the tapering period induced a positive balance between haemolysis and erythropoiesis. The sudden reduction in training load may have reduced the rate of intravascular haemolysis and stimulated the reticuloendothelial system's capacity for reuptake of dying erythrocytes (Hallberg & Magnusson, 1984). This view is supported by Mujika et al. (2002), who found that serum haptoglobin was significantly increased after 6 d of tapering in middle distance runners, in addition to an observed trend towards an increase in reticulocytes. This evidence once more reflects the anabolic environment that appears to be created by a period of tapered training.

1.5.6 Other Considerations

Whilst the taper is crucial to ensure athletes are ready to produce an optimal performance, they must also be able to regulate their rate of work output during the race. This distribution of work or energy expenditure over a particular distance is also referred to as 'pacing' or a 'pacing strategy' (Abiss & Laursen, 2008) and enables the athlete to cover the race distance in the shortest possible time, without catastrophic failure in any physiological system (St Clair Gibson et al., 2006). Whilst there has been greater attention on assessing the optimal pacing strategies for performance, little is known about the influence of tapering on pacing in competition.

It has been suggested that pacing is determined by a combination of central and peripheral regulation. According to Ulmer (1996), the teleoanticipation process creates a brain algorithm to initiate a starting pace via efferent neural commands, based on knowledge of the distance to be completed and previous experience. In addition, the environmental conditions, current health status (St Clair Gibson et al., 2006) and possibly muscle glycogen content (Rauch et al., 2005) might all provide input into the decision making process. The central governor model further develops the teleoanticipation theory and suggests that alterations are continually made to pace during the race, initiated by afferent input from the peripheral physiological systems and receptors that monitor change resulting from the current pace (Lambert et al., 2005; St Clair Gibson et al., 2006). Despite complex information processing between the brain and periphery in regulating pacing however, it is unknown whether the initial pace set in a feedforward manner might be

interfered with by the effects of tapering on recovery from accumulated fatigue. For example, athletes might train with overload prior to the taper to maximise adaptation (Aubry et al., 2014) and become unaccustomed to performing without the accumulated fatigue that is consequential of the normal training process (Halsen & Jeukendrup, 2004). This overload practice might influence the memory and negatively impact the decision making process in selecting race pace. It is possible therefore, that the change in fatigue status after tapering might need to be taken into account when informing the brain algorithm, to achieve an optimal pacing strategy and an improvement in performance.

1.5.7 Summary

The physiological mechanisms fundamental to the process of tapering have not yet been well defined in endurance runners and most of what is known is speculative (Child et al., 2000; Mujika et al., 2000). It appears that a myriad of physiological changes occur during the taper, including metabolic, neuromuscular, hormonal and haematological responses, which may all contribute to improving performance. This is perhaps due to the complex interaction of the physiological determinants associated with endurance performance and the influence of differing tapering strategies implemented in existing investigations. Adding to the confusion might also be the differing training status of participants in tapering studies. However, the available evidence appears to suggest that the tapering period allows for the restoration of physiological capacities that may have been previously suppressed by intensified training, which may then lead to amplified physiological responses to the training completed during the taper. Despite this, athletes may not always approach competition following the same training load or state of fatigue (e.g. due to illness or injury) and may require modification of their typical tapered training to fully recover and peak for competition, or to avoid detraining. This may also influence the selected pacing strategy and capability to deliver an optimal performance.

To optimise a tapering strategy in an elite population therefore, where the prior approach may not always be the same, it is fundamental to understand individual fatigue status preceding the taper and rate of recovery during the taper. Given the existing mechanistic literature and the available analysis methodologies and technologies, it may be possible to identify a biomarker of training stress and recovery to objectively monitor the effectiveness of a tapering strategy and readiness to compete on an individual level. This may inform coaches to apply real-time adjustments to the planned training load, which

could be necessary to achieve optimal restoration of physiological capacities and responses to training before competition. In an elite population however, it is not feasible to employ the invasive procedures such as muscle biopsies, necessary to monitor a number of physiological changes associated with a taper (e.g. muscle glycogen concentration). Therefore, it is necessary to identify biomarkers that are associated with the physiological changes during tapering, which can be measured both quickly and with minimal invasiveness.

1.6 Potential Biomarkers of Recovery during Tapering

1.6.1 Interleukin-6

It has been suggested that certain cytokines could represent markers of training stress in athletes (Jürimäe et al., 2011). Interleukin-6 is a pleiotropic cytokine with a variety of biological roles including exercise-induced immunoregulatory effects, generation of acute phase reactions and metabolic functions to maintain energy homeostasis during exercise. The polypeptide messenger molecule, IL-6, is predominantly produced in the working skeletal muscle during exercise (Ostrowski et al., 1998) and this production can explain the exercise-induced elevation in plasma IL-6 (Steensberg et al., 2000). It has been well-documented that IL-6 increases to a much greater extent than any other cytokine during exercise (Febbraio & Pedersen, 2002; Pedersen, 2009; Pedersen & Febbraio, 2008; Petersen & Pedersen, 2005) and more than 100 fold increases have been found after marathons and other prolonged bouts of running (Nieman et al., 2005; Ostrowski et al., 1999; Suzuki et al., 2000). Since skeletal muscle is the source of IL-6 production during exercise, it is not surprising that exercise modes involving large muscle groups, such as running, result in more dramatic increases in plasma IL-6. However, approximately 50% of the plasma IL-6 fold change from pre- to post-exercise can be explained by the duration of exercise (Fischer, 2006) and therefore a longer duration of exercise at the same relative intensity would result in a greater IL-6 response. Although muscle damage is not required to cause the elevation in plasma IL-6, eccentric loading of the muscle during prolonged running may lead to increased micro muscular damage, which may result in a delayed peak concentration and a slower return to resting levels (Hellsten et al., 1997; Macintyre, 2001; Toft et al., 2002).

Significantly, IL-6 release has been shown to be elevated in glycogen depleted states and therefore may serve as an indirect measure of muscle glycogen status (Gleeson & Bishop,

2000). Gleeson and Bishop reported that the plasma IL-6 response to exercise was elevated when participants had been following a low-carbohydrate diet, compared to a high-carbohydrate diet. In support, IL-6 release was significantly augmented after just 1 h of a 5 h 2-legged knee extensor exercise in a glycogen-depleted leg compared to a non-depleted control leg (Steensberg et al., 2001). Individual data indicated that the highest IL-6 mRNA expression was found in participants with the lowest muscle glycogen post exercise. Furthermore it has been shown that carbohydrate ingestion attenuates the exercise-induced elevation in IL-6. Immediately after 2.5 h of running at approximately 75% $\dot{V}O_{2max}$, the increase in IL-6 from baseline was lower in a carbohydrate feeding trial vs. the placebo trial (421% and 753%, respectively) (Nehlsen-Cannarella et al., 1997). Based on these data, it appears that low muscle glycogen acts as a trigger for a pro-inflammatory response, suggesting that muscle glycogen, and post exercise nutritional recovery are essential components in regulating the well documented post-exercise IL-6 response.

Despite attempts to maintain energy homeostasis, athletes may spend prolonged periods of time in heavy phases of training with sub-optimal muscle glycogen stores. It remains unclear whether this is as a result of repeatedly engaging in glycogen depleting training sessions without adequate carbohydrate intake and recovery time, as a consequence of muscle damage-impaired glycogen replenishment, or as a combination of both factors. It could be speculated therefore, that athletes may exhibit high plasma IL-6 concentrations. It has been shown that intensified training in athletes can cause a chronic increase in resting plasma IL-6 concentrations (Robson-Ansley et al., 2007; Ronsen et al., 2002). Robson-Ansley et al. (2007) found that 2 weeks of intensified running training in trained male triathletes caused a significant increase in resting plasma IL-6 16 h post-exercise. Plasma IL-6 also showed a tendency to remain elevated above levels measured prior to the intensified training period even when training returned to normal. Moreover, as the final blood sample was taken only 1 week after the termination of the intensified training, it is not clear as to how long resting plasma IL-6 could remain elevated. In contrast, Halson et al. (2003) found no difference in plasma IL-6 concentration during or after 2 weeks of intensified cycling training compared to normal training concentrations. However, this was likely due to the mode of exercise, as systemic increases in IL-6 appear to be attenuated in cycling compared to running, which recruits a greater muscle mass. In addition, endurance runners often train multiple times per day, which has been associated

with an augmented IL-6 response post-exercise, compared to just 1 daily bout (Ronsen et al., 2002). After a longer period of intensified training in elite cyclists (8 weeks), the IL-6 response to a 40 km time trial was 113% greater than before the training period (Farhangimaleki et al., 2009). A 3 week tapering period where training volume was reduced by 50% was necessary to reverse the elevated IL-6 response. Evidence suggests that prolonged intense training can significantly elevate both resting and post-exercise plasma IL-6 concentrations. The monitoring of IL-6 could therefore help to assess the effectiveness of a tapering strategy following a heavy period of training and potentially act as a peripheral indicator of muscle glycogen content.

An additional benefit of monitoring IL-6 status is its role in establishing the anti-inflammatory cascade (Steensberg et al., 2003). Infusion of rhIL-6, results in increased plasma interleukin-1 receptor agonist (IL-1ra), interleukin-10 (IL-10), C and C-reactive protein (CRP), independent of increases in tumor necrosis factor-alpha (TNF- α) (Steensberg et al., 2003). Interleukin-1 receptor agonist competitively blocks the binding of IL-1 to both type I and type II IL-1 receptors and in turn, prevents the protein from exerting its pro-inflammatory effects (Granowitz et al., 1991). It is possible that this process is facilitated by CRP, which may enhance the release of IL-1ra from monocytes during the latter stages of recovery from exercise (Pue et al., 1996). Interleukin-10 is another major anti-inflammatory cytokine and is known to hinder a number of immune pathways, including the inhibition of TNF- α production by monocytes (Petersen & Pedersen, 2005). Frequent intense exercise has been associated with alterations in leukocyte subpopulations and antimicrobial capacity, which may be partly explained by increases in C, as its anti-inflammatory actions have been well-documented (Barnes, 1998; Pedersen & Ullum, 1994; Steensberg et al., 2003). It is apparent that the release of IL-6 from contracting muscles may facilitate a broad anti-inflammatory response and athletes engaging in prolonged intense exercise on a regular basis exhibit increased susceptibility to illness, which is not desirable immediately prior to competition (Pedersen & Ullum, 1994). Regular monitoring of IL-6 could serve therefore as an indicator of stress or overtraining during normal training with an attenuation of the elevation during the tapering period.

Certain cytokines are able to cross the blood-brain barrier (Szelényi, 2001) and it has been suggested that exercise-induced IL-6 release at the muscle could initiate signalling at the

brain (Robson-Ansley et al., 2010), which may in turn influence perceptions of fatigue and effort. This may have the further effect of influencing a centrally mediated pacing strategy during exercise performance (Lambert et al., 2005, St Clair Gibson et al., 2006). Infusion of low-dose rhIL-6 was associated with greater feelings of fatigue and inactivity and an inability to concentrate (Späth-Schwalbe et al., 1998), supporting the potential for IL-6 to affect perceptions of fatigue. Robson-Ansley et al. (2004) observed that rhIL-6 administration prior to a 10 km time trial run significantly impaired performance compared to a placebo trial. Taken together, the evidence in response to systemically elevated IL-6 levels points to a centrally-mediated effect. However, despite the theory that raised plasma IL-6 levels may be involved in the severe fatigue experienced by athletes during heavy training, evidence to support this notion is equivocal (Halsen et al., 2003; Robson-Ansley et al., 2009).

Given the available evidence, there is a strong possibility that IL-6 could represent a potential biomarker for monitoring the effectiveness of a tapering strategy and physiological readiness to perform on an individual level. Providing that it can be detected with validity and reliability in a minimally/non-invasive manner, which is a necessity when working in the field with elite athletes. One aspect which potentially makes this problematic is the daily variability of IL-6 and the requirement to establish a significant change. However, the action of IL-6 is dependent on the availability of the soluble IL-6 receptor (sIL-6R), which is more stable on a day-to-day basis than its complimentary protein (Robson-Ansley et al., 2010) and may therefore provide a more sensitive route.

1.6.2 Soluble Interleukin-6 Receptor

Concentrations of the sIL-6R have been shown to be associated with prolonged endurance exercise and with general feelings of fatigue (Robson-Ansley et al., 2009). It is possible that increases in sIL-6R could cause a greater sensitivity to IL-6. When bound, the IL-6/sIL-6R complex acts as an agonist and is capable of stimulating cells that are not responsive to IL-6 alone. In rats, it was demonstrated that intracerebroventricular (ICV) infusion of recombinant human sIL-6R can enhance and prolong the decrements in locomotor activity and food intake experienced after ICV infusion of rhIL-6 (Schöbitz et al., 1995). Taken together, this suggests that the IL-6/sIL-6R complex acts antagonistically to heighten centrally mediated fatigue, which could exacerbate fatigue experienced during or after exercise. Increases in IL-6 in response to exercise generate acute phase reactions,

including the release of CRP. C-reactive protein has been shown to increase concentrations of sIL-6R *in vitro*, by inducing receptor shedding from neutrophils (Chalaris et al., 2007; Jones et al., 1999). This is supported *in vivo*, where both CRP and sIL-6R concentrations are significantly elevated at rest, during a 6-d cycling event (Robson-Ansley et al., 2009). The release of IL-6 in response to exercise is highly dependent on exercise duration and modality (Fischer, 2006). Therefore, during tapering when the volume (duration of exercise) is reduced, it is likely that IL-6 responses will be suppressed and in turn, reduce levels of CRP and sIL-6R and facilitate an improvement in performance due to reduced feelings of fatigue.

1.6.3 Creatine Kinase

Previous evidence has shown elevated CK values prior to tapering (Houmard et al., 1990). Creatine kinase is a muscle enzyme that often increases in the blood in response to strenuous or eccentric exercise, most likely due to increased cell membrane permeability, cellular disruption, damage or apoptosis (Brancaccio et al., 2010). Factors influencing the degree of CK entry into the blood include training volume (Millard et al., 1985) and exercise mode (Koller et al., 1998). It is possible that exercise induced muscle damage may be experienced during heavy training prior to tapering and monitoring of CK in the blood prior to, and during tapering may therefore reflect the mechanical-muscular strain of training and/or recovery. Although CK concentration in the blood has been shown to decline as a result of tapering in endurance runners (Child et al., 2000; Houmard et al., 1990), changes were not accompanied by an improvement in performance. After 2 different 6-d tapers in middle-distance runners, large inter-individual variation in blood CK response were also evident, with mean values unchanged from the pre-taper period (Mujika et al., 2000; Mujika et al., 2002). Despite this, there was a positive association between the volume of low intensity continuous running and plasma CK concentration during tapering, which indirectly implies that athletes can limit the exercise-induced muscle damage prior to competition by reducing the volume of this type of running during the taper (Mujika et al., 2000). Although assessing changes to CK in the blood may be useful to monitor recovery from an acute training bout, this biomarker does not appear to be a clear indicator of recovery during tapering and or indicative of performance in endurance runners.

1.6.4 Testosterone:Cortisol Ratio

The T:C ratio has been previously identified as a marker of training stress (Adlercreutz et al., 1986), however as previously discussed (chapter 1.7.4), T:C responses to tapering are equivocal (Flynn et al., 1994; Mujika et al., 2000; Mujika et al., 2002). This is perhaps due to differences in the magnitude of prior training load, the tapering strategies implemented or the training status of the participants. This index of training stress is considered valuable for identifying periods of heavy training and less intensive training during a full season of training in elite athletes (Vervoorn et al., 1991), and therefore might only be useful to monitor recovery during a period of tapering in conjunction with previous longitudinal data. However, it is important to consider expense when monitoring long-term variations (Lac & Maso, 2004), although it is advantageous that T and C concentrations can be measured in saliva, providing a simple, non-invasive sampling alternative to blood collection (Lewis, 2006). Further research is required to confirm the responses of T and C to tapering in endurance runners and to indicate suitability as biomarkers of recovery and readiness to perform.

1.6.5 Catecholamines

The plasma and urinary concentrations of catecholamines have been previously measured in athletes to indicate training stress and to recognise overreaching and overtraining (Halsen et al., 2002; Hooper et al., 1993; Lehmann et al., 1992; Mackinnon et al., 1997), and might offer another possible option for indicating recovery status prior to competition. Mujika et al. (1996) investigated the catecholamine responses in highly-trained swimmers to 12 weeks of intense training and during a 4 week taper involving a progressive reduction in training volume to ~25% of pre-taper levels. Plasma adrenaline, noradrenaline and dopamine concentration were unchanged throughout the study period, despite an improvement in performance following the taper. Furthermore, no changes in plasma adrenaline, noradrenaline or dopamine concentrations were observed in endurance trained cyclists over a 6 week period consisting of 2 weeks each of normal training, intensified training and recovery training (taper) (Halsen et al., 2002). A performance decline was evident during the intensified training period, where time to complete a target workload was increased compared to normal training. Although plasma adrenaline and noradrenaline concentrations were correlated with training volume, there was no change in plasma adrenaline and noradrenaline between early-season, mid-season, late-season and taper sampling points in highly trained swimmers (Hooper et al., 1993). This suggests that

performance changes in response to an acute period of intensified or reduced training are not reflected by changes in catecholamine concentration. In contrast, it was found that plasma noradrenaline could predict the change in performance associated with tapering in elite swimmers and could account for 82% of the variance (Hooper et al., 1999). However the performance improvement in this study was less than that required for a meaningful change and the findings may be explained by the swimmers remaining on heavy training loads, particularly in the first week of the 2 week taper. Evidence for the sensitivity of catecholamines to changes in training load is inconsistent therefore and might not represent a reliable biomarker of recovery during tapering. Furthermore, the measurement of these hormones is not practical on a longitudinal basis due to expense and complexity of analysis (Mujika et al., 2004).

1.7 Summary

Existing evidence from both performance and mechanistic literature points to a reduced-volume, high intensity taper, which is carried out in a progressive fast-decay pattern to achieve positive physiological responses and an improvement in performance. However, it is not clear as to how the duration required for the tapering period is affected by the preceding training load and fatigue status. The inclusion of high intensity training during tapering appears to be the key to improving performance, although further work is necessary to optimise the manipulation of training intensity during the taper and of particular importance, to assess the outcome using a field-based performance measure.

A number of positive physiological changes have been observed as a result of the pre-competition taper, including metabolic, neuromuscular, hormonal and haematological responses, all of which may contribute to some extent to improving endurance running performance. It would seem that the tapering period facilitates the restoration of physiological capacities that may have been previously suppressed by heavy training. This may also then lead to amplified physiological responses to the training completed during the taper, however a more thorough understanding of relationship between tapering and performance enhancement is essential to allow for further optimisation of strategies.

From an applied perspective, the ability to monitor training stress and recovery during the taper might be advantageous to coaches and athletes, allowing them to evaluate the efficacy of the chosen strategy and modify training load if deemed necessary. The longitudinal monitoring of variations in IL-6, sIL-6R, T and C concentrations might be

indicative of training stress and energy homeostasis throughout a full season, although further research is required to investigate whether these minimally-invasive physiological biomarkers are sensitive enough to indicate taper effectiveness on an individual level to facilitate the peaking of competition performance.

A summary of the components of tapering, current recommendations and observations from the literature is presented in Figure 1.6. Despite an increasing body of research investigating the effects of tapering on performance and the underlying physiological mechanisms, the tapering strategies currently employed by elite endurance runners prior to major competition remain unknown. The application of tapering strategies in elite sport appear to be predominantly determined by coach and athlete experiences, rather than from the scientific literature. This is perhaps due to the culture in track and field, with traditional views held by more experienced coaches and a resistance or fear to change, despite possible opportunities for optimisation.

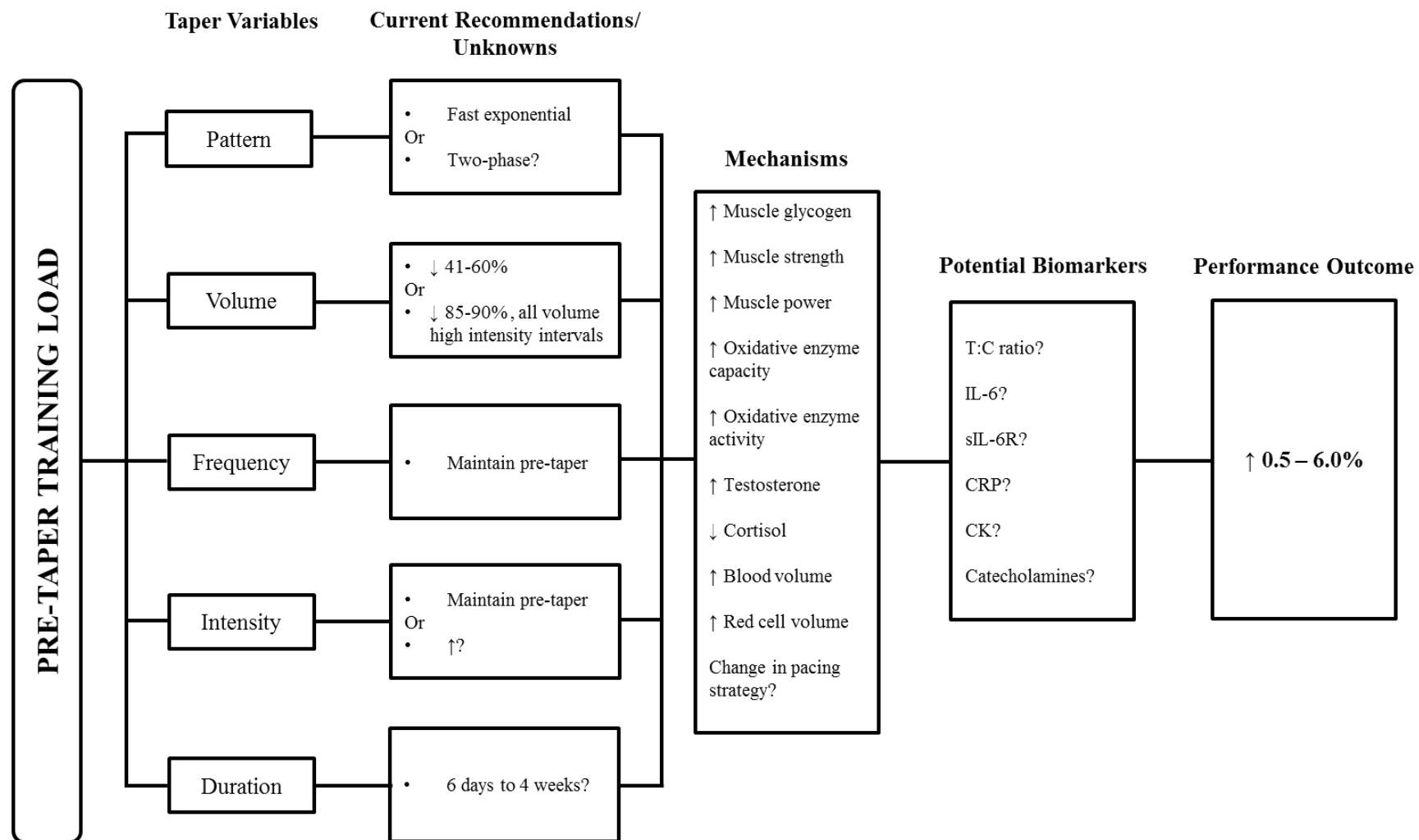


Figure 1.6. A model of the components of tapering and the current observations and recommendations from the scientific literature. Question marks denote unknowns or areas where further research is required.

1.8 Aims of the Thesis

The aims of the current thesis are:

1. To explore the strategies currently employed by elite athletes in order to inform future research directions.
2. To gain a more thorough understanding of the physiological mechanisms underpinning the relationship between tapering and enhancement of performance.
3. To optimise the manipulation of training intensity during the taper to enhance performance.
4. To identify a minimally invasive physiological biomarker capable of monitoring recovery status during the taper, to allow individualisation of strategies.

Specific hypotheses will be presented in experimental chapters after a more detailed introduction to the specific research question.

Chapter II

A Comparison of Capillary and Venous Blood Sampling Methods for the Detection of Plasma IL-6 and sIL-6R

2.0 Chapter Summary

The purpose of this chapter was to quantify the relationship between venous and capillary blood sampling methods for the measurement of plasma IL-6 and sIL-6R, to assess suitability for measuring less invasively in the athletic population. Twelve physically active healthy male participants were recruited for the measurement of IL-6 at rest and during exercise and an additional group of 12 physically active healthy male participants for the measurement of sIL-6R at rest. Venous and capillary blood samples were collected at rest in all trials, and after 45 min and 75 min of exercise at 60% $\dot{V}O_{2\max}$ in the cycling and running trials. Plasma IL-6 and sIL-6R concentrations were measured using ELISAs. Mean plasma IL-6 concentration was not different between venous and capillary blood sampling methods in the cycling trial and the running trial at rest or in response to exercise, although there was some disagreement in the reported values between the 2 sampling methods (bias \pm 95% limits of agreement; $-3.99 \text{ pg}\cdot\text{ml}^{-1}$ to $3.56 \text{ pg}\cdot\text{ml}^{-1}$ and $-3.88 \text{ pg}\cdot\text{ml}^{-1}$ to $3.72 \text{ pg}\cdot\text{ml}^{-1}$ after cycling and running trials, respectively). Mean resting plasma sIL-6R concentration was higher from venous blood sampling, compared to capillary sampling ($49.8 \pm 15.4 \text{ ng}\cdot\text{ml}^{-1}$ vs. $43.6 \pm 10.1 \text{ ng}\cdot\text{ml}^{-1}$ respectively; $P < 0.05$), despite a positive correlation between the 2 methods ($R^2 = 0.72$; $P < 0.05$). To ensure precision of data interpretation, venous and capillary blood sampling methods should not be implemented interchangeably for the detection of plasma IL-6 and sIL-6R concentration.

2.1 Introduction

The polypeptide messenger molecule IL-6 has a variety of biological roles including exercise-induced immunoregulation, generation of acute phase reactions and metabolic functions to maintain energy homeostasis during exercise. There has been an increasing interest in the effects of chronic exercise training on IL-6 in athletic populations and it has been suggested that this cytokine could represent a marker of training stress in athletes (Jürimäe, et al., 2011). It has been observed that prolonged intense training can significantly elevate both resting (Robson-Ansley et al., 2007) and post-exercise (Farhangimaleki et al., 2009) plasma IL-6 concentrations, and that limited recovery

between heavy endurance exercise bouts is associated with an augmented post-exercise plasma IL-6 response (Ronsen et al., 2002). This is not desirable for athletes prior to competition, since increased plasma IL-6 concentration has been associated with a worsening of 10 km running performance and increased fatigue sensation (Robson-Ansley et al., 2004).

Athletes may not exhibit excessively elevated plasma IL-6 values during heavy training, but rather have a greater sensitivity to IL-6, as is experienced in individuals with chronic fatigue syndrome (Arnold et al., 2002). It is possible that sIL-6R may be key in this process, as concentrations were elevated at rest during 6 d of prolonged cycling and were associated with general feelings of fatigue, despite no change in resting IL-6 on days 2-6 (Robson-Ansley et al., 2009). Regular monitoring of plasma IL-6 and sIL-6R could therefore provide an indication of recovery from previous exercise bouts, in terms of energy homeostasis and inflammation status. This may facilitate coaches in prescribing training appropriate to the recovery status of their athlete and could be particularly useful during the tapering period prior to competition to facilitate the peaking of performance.

It is of interest therefore, to explore alternative simple, minimally-invasive and inexpensive sampling techniques for measuring IL-6 and sIL-6R that can be used in the field for the athletic population. The gold standard for measurement of circulating IL-6 and sIL-6R is via venous blood sampling. However, this method is fairly invasive and requires training to attain competence in carrying out the procedure. Since the biochemical assays for detection of IL-6 and sIL-6R only require 25 µl and 3 µl of plasma in duplicate, respectively, there is also often an unnecessary surplus of blood remaining from a venepuncture sample.

Previously, research has attempted to detect certain cytokines in a variety of other body fluids using non-invasive methods. Despite the evidence that salivary C concentrations are highly correlated with serum levels in response to exercise (O'Connor & Corrigan, 1987), it appears that concentrations of IL-6 in saliva are not associated with those found in the blood at rest or in response to exercise (Cox et al., 2008; Minetto et al., 2005; Minetto et al., 2007). Using recycling immunoaffinity chromatography, IL-6 was detected in the sweat of healthy premenopausal women after a 24-h period (Marques-Deak et al., 2006). The IL-6 concentrations detected in the sweat and concentrations detected from venous plasma were significantly correlated and the concentrations appeared comparable.

However, our laboratory was unable to detect IL-6 in the sweat of healthy males or females after exercise in the heat (Faulkner et al., 2014) and contradict the findings of Marques-Deak et al. (2006).

Although slightly more invasive than sweat collection, capillary blood sampling is used regularly in the field with the athletic population for the measurement of lactate (Tanner et al., 2010) and plasma IL-6 has also been successfully detected using this blood sampling method (Smith et al., 2011). However the sampling method has not been validated against venous blood sampling and no study has investigated sIL-6R concentrations from capillary blood. While it is necessary to utilise techniques that are simple and as minimally invasive as possible, it also is important to ensure that the data reflect that of the systemic system.

The aim of the current study was to quantify the relationship between venous and capillary blood sampling methods for the detection of plasma IL-6 and sIL-6R concentrations at rest. The secondary aims were to examine the relationship between sampling methods for IL-6 after differing durations of exercise and in response to differing modes of exercise (cycling and running). It was hypothesised that plasma IL-6 and sIL-6R measured from capillary blood would be strongly correlated with that from venous blood.

2.2 *Methods*

2.2.1 *Participants*

Approval from the Loughborough University Ethical Advisory Committee was gained prior to the commencement of all studies in this thesis. Written information was provided to potential participants, outlining the purpose of the study, experimental procedures and potential risks or discomfort that may be experienced whilst taking part in the study. Participants were fully briefed and were offered the opportunity to ask questions before signing a statement of informed consent (appendix A) and in experimental studies, completing a health screening questionnaire (appendix B).

To minimise risk to participant health and to ensure current health status of the participant would not compromise study findings, participants were only recruited for the experimental studies if they met the following criteria;

- Were non-smokers
- Were free from any musculoskeletal injury
- Had no personal history of cardiovascular or metabolic disease

- Were weight stable and not on a weight reduction/increase diet
- Not taking anti-inflammatory medication
- Not taking medication that affects metabolism or digestion
- Had a body mass index (BMI) of less than 25 kg/m² (i.e. not classed as overweight)
- Had no known circulatory disorders (i.e. Raynaud's disease)

Twelve physically active healthy male participants volunteered to take part in the trials for the measurement of IL-6 at rest and during exercise (mean \pm SD): age 23.8 ± 3.5 y, body mass 72.9 ± 5.8 kg, height 176.7 ± 0.1 cm, body mass index 23.4 ± 1.3 kg·m⁻². An additional group of 12 physically active healthy male participants agreed to take part in the trial for the measurement of sIL-6R at rest (mean \pm SD): age 22.6 ± 3.1 y, body mass 72.7 ± 8.1 kg, height 181.2 ± 7.2 cm, body mass index 22.1 ± 1.7 kg·m⁻². Three participants took part in both the IL-6 and sIL-6R trials.

2.2.2 Preliminary Exercise tests

Preliminary submaximal and maximal ergometer tests were carried out at least 48 h prior to the main experimental trials to determine workloads equivalent to 60% $\dot{V}O_{2peak}$ for both cycling and running. This exercise intensity was chosen to ensure participants were able to exercise for the complete duration required in the experimental trials. Aerobic cycling tests were carried out on an electromagnetically braked cycle ergometer (Lode Excalibur, Groningen, The Netherlands) and aerobic running tests on a motorised treadmill (HP Cosmos, Nussdorf-Traunstein, Germany). Expired air was measured continuously using an online breath-by-breath gas analyser (Ultima CPX, MedGraphics, MN, USA). Prior to use, 2-point calibrations of both gas sensors were carried out using a known gas mixture (12% O₂, 5% CO₂) and a reference gas (21% O₂). A bi-directional differential pressure pneumotach was calibrated using a 3 L syringe over 5 flow rates. Participants were fitted with nose clip and breathed through a mouthpiece housing the pneumotach, which was connected via the umbilical to the analyser. Oxygen uptake and carbon dioxide production were quantified using the BreezeSuite software (MedGraphics, MN, USA). Heart rate was monitored using short-range telemetry (RS200, Polar Electro, Kempele, Finland) during the preliminary tests. A heart rate transmitter belt was fitted across the chest and aligned beneath the sternum. The belt was worn next to the skin and water was applied to the electrodes beforehand to enhance signal detection. Participants were asked to rate their perceived exertion using Borg's RPE scale (Borg, 1973) (appendix C). The scale is a

subjective measure of effort, ranging from 6 (indicating no exertion) to 20 (indicating maximal exertion). Participants were familiarised with the exercise equipment prior to the preliminary tests.

On arrival at the laboratory, measurements of body mass, height, BMI were made using the following techniques and equipment:

- Body mass – measured using a balance beam scales (Avery, Birmingham, UK) to the nearest 0.1 kg. Participants wore light clothing but footwear and items from pockets were removed.
- Height – measured using a wall-mounted precision stadiometer (Seca, Hamberg, Germany) to the nearest 0.1 cm. Footwear was removed and participants stood with their heels, buttocks and upper back against the stadiometer. Feet were together and flat on the floor. The head was aligned in the Frankfurt plane before the sliding scale was lowered to touch the top of the head. Participants were instructed to take a deep inhalation of breath before the measurement value was recorded.
- BMI – calculated by dividing body mass in kilograms by the square of height in meters.

Maximal Cycle Ergometer Test

An incremental cycling test to volitional exhaustion was used to determine peak $\dot{V}O_2$ ($\dot{V}O_{2peak}$). The test protocol began at 100 W and was increased by 35 W every 3 min thereafter. Expired air was measured as described above, and heart rate and RPE were recorded at the end of each stage and at the end of the test. Mean $\dot{V}O_2$ in the final 30-s period of the test was accepted as $\dot{V}O_{2peak}$. The power (W) that would elicit 60% of $\dot{V}O_{2peak}$ was estimated by plotting $\dot{V}O_2$ data against power (W) from the 3 min stages of the test.

Submaximal Treadmill Test

The relationship between $\dot{V}O_2$ and submaximal running speed was determined using a submaximal incremental test. The test was 16 min in duration and was divided into 4 stages. The initial running speed was between 8 and 11 km·h⁻¹ depending on the participant's fitness level and increased by 1 km·h⁻¹ every 4 minutes. The speed at which heart rate reached 160 bpm was chosen as the constant speed for the maximal treadmill test, to ensure participants reached volitional fatigue within 10 ± 2 min (Buchfuhrer et al. 1983).

Maximal Treadmill Test

Participants were given 20 min to recover from the submaximal test before beginning the maximal test. The test protocol consisted of a gradient increase of 1% every minute until the participants reached volitional exhaustion. Heart rate was monitored throughout the test and RPE at the end of the test. Expired air was measured as described above, and mean $\dot{V}O_2$ in the final 30-s period of the test was accepted as $\dot{V}O_{2peak}$. The speed that would elicit 60% of $\dot{V}O_{2peak}$ was estimated by plotting $\dot{V}O_2$ data from the submaximal treadmill test against the corresponding submaximal running speed.

Mean $\dot{V}O_{2peak}$ was $49.5 \pm 5.5 \text{ mL}\cdot\text{kg}\cdot\text{min}^{-1}$ for cycling and $59.5 \pm 9.2 \text{ mL}\cdot\text{kg}\cdot\text{min}^{-1}$ for running. Exercise intensity equivalent to 60% $\dot{V}O_{2peak}$ was $166 \pm 18 \text{ W}$ and $10.5 \pm 1.5 \text{ km}\cdot\text{h}^{-1}$ for cycling and running, respectively. The preliminary and experimental trials for cycling were separated by at least 7 d from the preliminary and experimental trials for running.

2.2.3 Experimental Protocol

Participants reported to the laboratory between 0700 h and 0800 h following a 10 h overnight fast, having remained inactive and refrained from caffeine and alcohol consumption in the previous 24 h. Body mass was measured, before participants were seated in a semi-supine position for resting venous and capillary blood sampling (described below). In the exercise trials, participants then cycled or ran at a workload equivalent to 60% $\dot{V}O_{2peak}$ for 45 min. At 45 min, participants remained on the cycle ergometer or treadmill in a stationary position for approximately 2 min while simultaneous venous and capillary blood samples were obtained. Exercise was resumed for a further 30 min and immediately after, final venous and capillary samples were taken simultaneously. Heart rate was monitored at 15-min intervals during the trials and participants consumed water *ad libitum* throughout.

2.2.4 Blood Sampling

Venous blood samples were collected by venepuncture from an antecubital vein in the right arm into a 10 mL vacutainer which had been pre-cooled and pre-treated with K^+EDTA (BD Biosciences, San Diego, USA). Immediately after, the remaining samples were centrifuged at $4000\times g$ for 10 min at 4°C and the separated plasma was pipetted in to multiple eppendorfs and stored at -80°C for subsequent analysis.

Capillary samples were obtained by fingerpick sampling from the left hand using an automated lancet. Prior to collecting the resting capillary sample, the hand and forearm were warmed for 5 min using a warm-air hot box to arterialise the blood at the sample site. Blood was collected into two pre-cooled 200 µl microvettes pre-treated with K⁺EDTA (Sarstedt, Leicester, UK). Immediately after, the remaining samples were centrifuged at 4000×g for 10 min at 4°C and the separated plasma was pipetted in to multiple eppendorfs and stored at -80°C for subsequent analysis.

2.2.5 *Blood Analysis*

Plasma IL-6 and sIL-6R concentrations were analysed using sandwich ELISAs (appendix D) and corrected for any changes in plasma volume (appendix E) during exercise trials.

2.2.6 *Statistical Analysis*

Data were analysed using SPSS (Statistical Package for Social Sciences Inc. v19.0; Chicago, IL, USA) and were initially tested for distribution using the Shapiro-Wilk test. Data are presented as mean ± SD, unless specified otherwise. Statistical significance was accepted at $P \leq 0.05$.

The IL-6 data were log-transformed to satisfy assumptions of parametric tests. The log-transformed levels of IL-6 in venous and capillary blood were compared between methods and across sampling points using repeated measures 2-factor ANOVA. The differences between capillary and venous concentrations for sIL-6R data were tested for distribution and consequently a paired-samples t-test was performed on the venous and capillary sIL-6R data. The strength of the relationship between sampling methods for IL-6 and sIL-6R data was assessed using the Pearson-product-moment correlation coefficient (data log-transformed and pooled for cycling and running trials). As the Pearson-product-moment correlation coefficient solely provides information on the strength of the relationship between measurements, Bland-Altman analysis (Bland & Altman, 1986) was performed to give an indication of the ‘agreement’ between variables. Data are presented as mean ± SEM.

2.3 *Results*

Mean plasma IL-6 concentration was not different between venous and capillary blood sampling methods during, or in response to cycling ($P = 0.75$ and $P = 0.88$, respectively; Figure 2.1A). There was however, a difference in venous compared to capillary-derived

IL-6 at rest in the cycling trial (0.48 ± 0.13 vs. 0.31 ± 0.16 ; $P < 0.05$; Figure 2.1A). The concentration of IL-6 in both venous and capillary samples increased in response to cycling exercise ($P < 0.05$) and the pattern of this response was not different between sampling methods (time \times sample site; $P = 0.08$). The investigator was unable to take a venous blood sample from 1 participant at 75 min in the cycling trial and the statistical analysis was therefore performed using data from the remaining 11 participants at this time-point. In the running trial, mean plasma IL-6 was not different between venous and capillary blood sampling methods ($P = 0.89$; Figure 2.1B). There was a main effect of time as plasma IL-6 concentration in both venous and capillary samples increased in response to running ($P < 0.05$). The response was not different between sampling methods (time \times sample site; $P = 0.75$). Mean resting plasma sIL-6R concentration was higher from venous blood sampling, compared to capillary sampling (49.8 ± 4.5 ng·ml⁻¹ vs. 43.6 ± 2.9 ng·ml⁻¹ respectively; $P < 0.05$; Figure 2.1C).

When IL-6 data was pooled from all time-points, there were positive associations between concentrations from venous and capillary blood sampling in the cycling trial ($R^2 = 0.87$; $P < 0.05$; Figure 2.2A) and in the running trial ($R^2 = 0.86$; $P < 0.05$; Figure 2.2B). A positive association between venous and capillary blood sampling for resting sIL-6R concentration was evident ($R^2 = 0.72$; $P < 0.05$; Figure 2.2C).

Bland-Altman plots (Figure 2.3) provide a schematic illustration of the agreement between venous and capillary sampling methods for the detection of IL-6 and sIL-6R. The 95% limits of agreement are shown in Table 2.1.

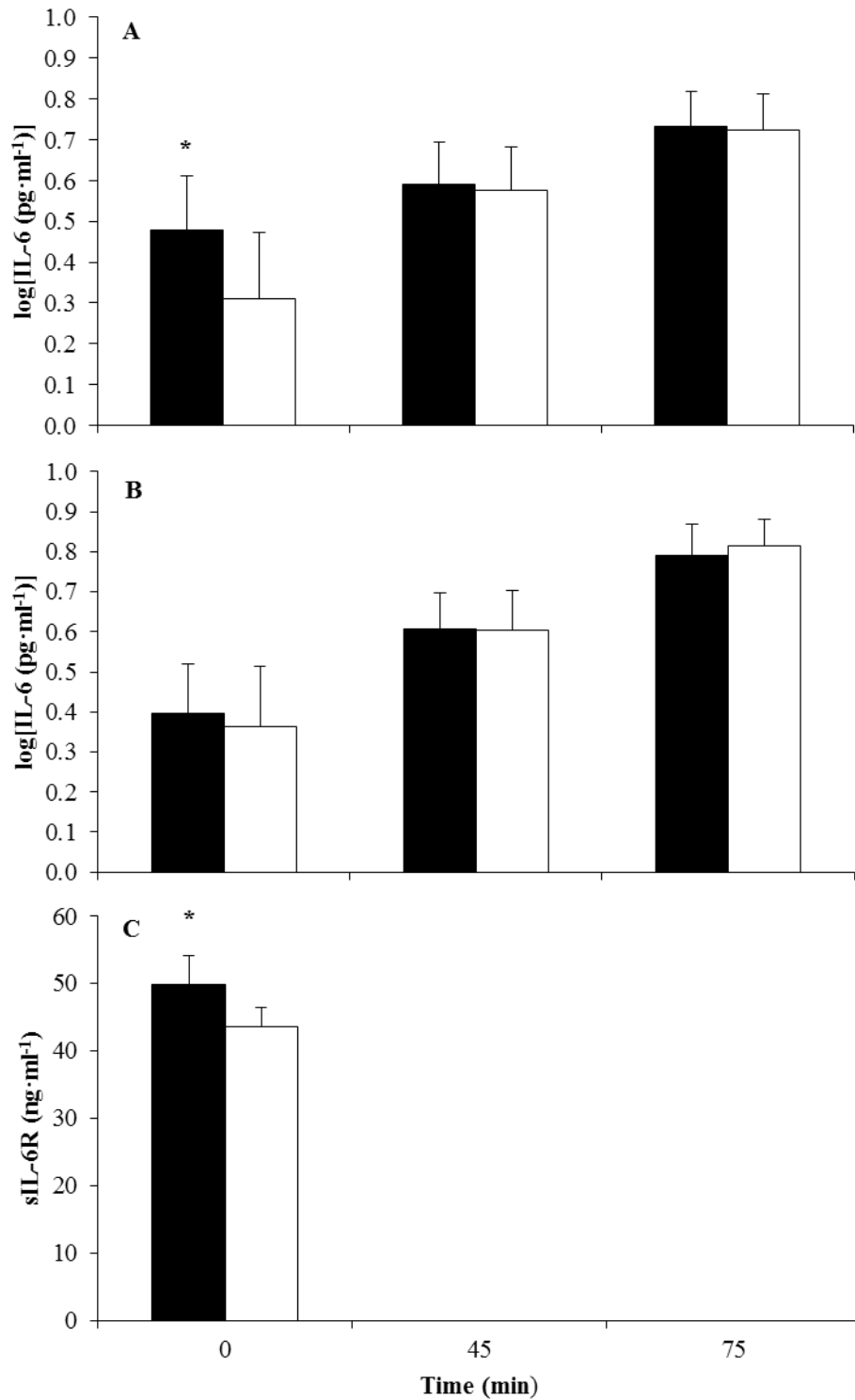


Figure 2.1. Plasma IL-6 concentration measured from venous blood sampling (■) and capillary blood sampling (□) in the cycling trial (A) and the running trial (B). Plasma sIL-6R concentration measured from venous blood sampling (■) and capillary blood sampling (□) at rest (C). * indicates venous higher than capillary ($P < 0.05$). Data shown as mean \pm SEM.

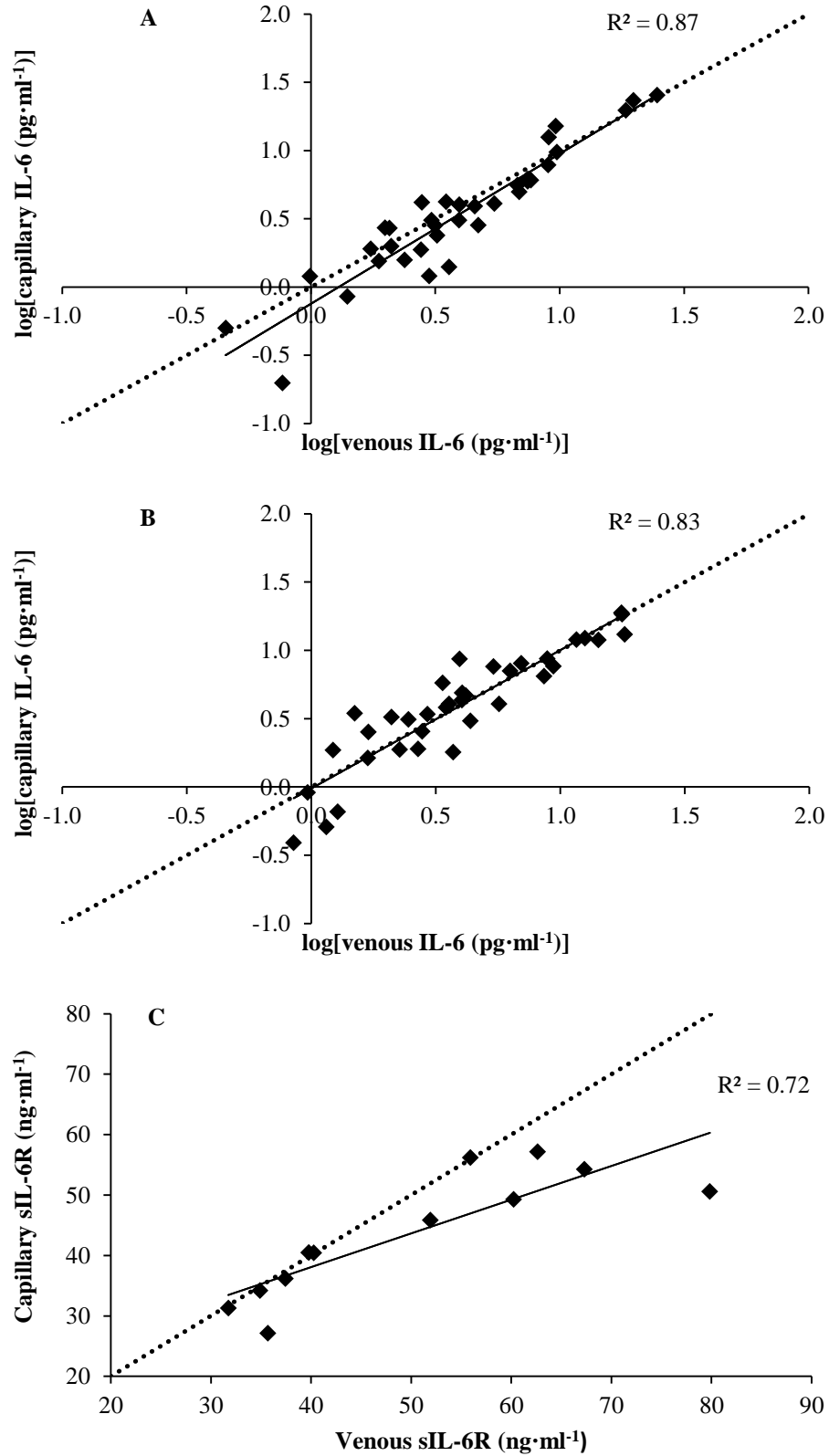


Figure 2.2. Association between venous and capillary blood sampling methods for the measurement of plasma IL-6 concentration in the cycling trial (A; $R^2 = 0.87$) and in the running trial (B; $R^2 = 0.83$) and for plasma sIL-6R at rest (C; $R^2 = 0.72$), all $P < 0.05$. Dashed line represents the line of identity.

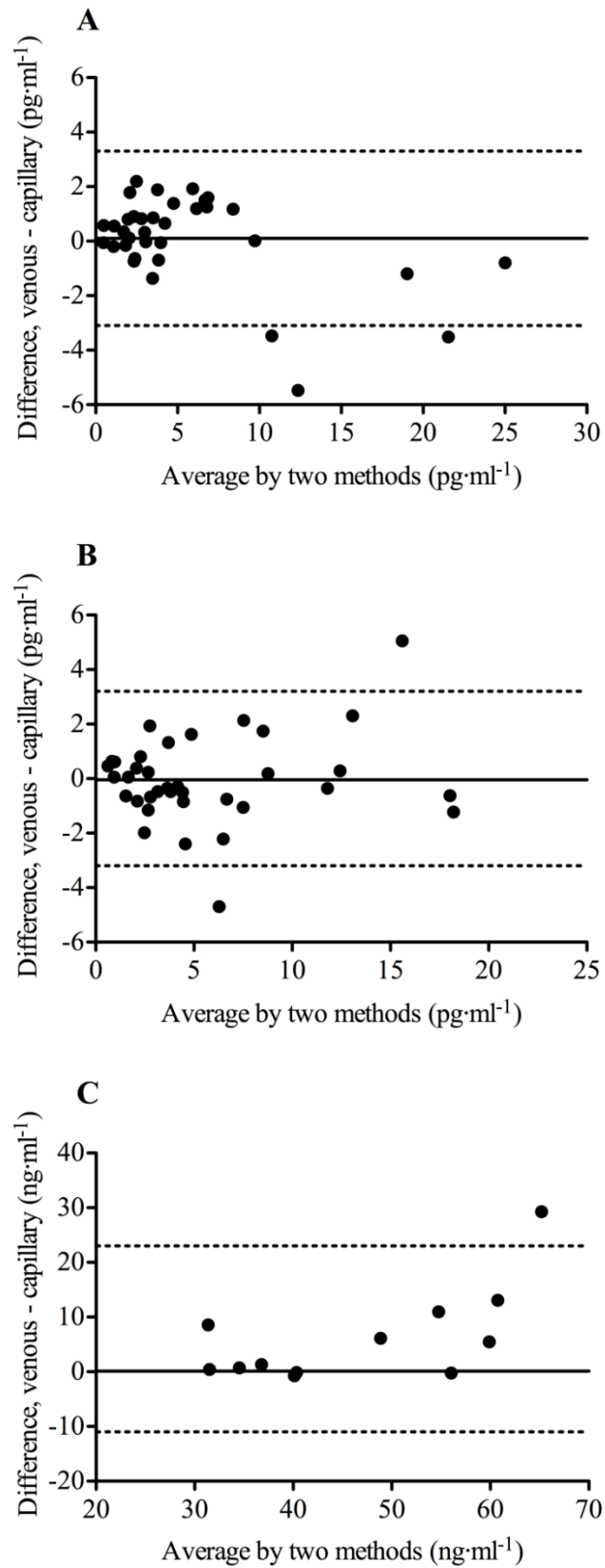


Figure 2.3. The 95% limits of agreement and mean difference between venous and capillary blood sampling for the measurement of plasma IL-6 in the cycling trial (A), in the running trial (B) and for plasma sIL-6R concentration at rest (C).

Table 2.1. Bland-Altman analysis of the venous and capillary blood sampling methods for plasma IL-6 concentrations measured in the cycling trial and the running trial and for resting Plasma sIL-6R concentration.

Trial	Time (min)	Bias	\pm SD	95% Limits of agreement
Cycling				
IL-6 (pg·ml ⁻¹)	0	0.67	0.94	-1.17 to 2.50
	45	-0.18	1.84	-3.79 to 3.43
	75	-0.22	1.92	-3.99 to 3.56
Running				
IL-6 (pg·ml ⁻¹)	0	0.13	1.73	-3.26 to 3.52
	45	-0.18	1.29	-2.71 to 2.34
	75	-0.08	1.94	-3.88 to 3.72
Rest				
sIL-6R (ng·ml ⁻¹)	0	6.24	8.66	-10.73 to 23.22

2.4 Discussion

The current study demonstrates that, when plasma IL-6 concentrations from either venous or capillary blood are compared using ANOVA, which considers the group mean and associated variance, there is no difference between the 2 sample sites during, or in response to cycling or running exercise. In view of the strong positive correlations between venous and capillary IL-6, it appears that capillary sampling may offer an alternative to venous sampling to quantify plasma IL-6 concentrations.

One unexpected finding was that despite high correlation, pre-exercise measurements of IL-6 differed between venous and capillary derived samples in the cycling trial. One possible explanation for this may be that the arterialisation protocol of the resting sample was not totally effective in fully arterialising the venous blood. Furthermore, it is possible that if the peripheral blood vessels were not sufficiently vasodilated in response to heating, then it would be expected that the venous to capillary delivery would be restricted (Gallen & Macdonald, 1990), resulting in a discrepancy and potential time-lag between venous and capillary appearance of IL-6. Although arterialisation protocols of 5-10 min heat exposure have been reported previously (Lobley et al., 2013, Pacy et al., 1991), some advocate 20 min of heating to arterialise samples (Stephens et al., 2008). Given that no differences were evident between IL-6 concentrations measured in either venous or capillary blood in response to exercise where peripheral blood flow would be elevated, it suggests that this is

the likely cause. In order to validate this speculation, when repeating the pre-exercise protocol in the running trial, it was ensured that the arterialisation protocol was tightly adhered to by participants. In this trial, it was demonstrated that there was no difference between plasma IL-6 when sampled from either venous or capillary sites following effective arterialisation. Since the bias of $0.13 \text{ pg}\cdot\text{ml}^{-1}$ was similar to that observed in response to, and after exercise, it is suggested with confidence, that the repeated measure during the running trial is a true reflection of the baseline IL-6 taken from both venous and capillary sites. Therefore, this highlights the importance of ensuring full arterialisation, with a minimum of 5 min of heating, when taking a measurement using capillary blood sampling at rest. However, despite the statistical difference at rest in the cycling trial, the bias between sampling sites was only $0.67 \text{ pg}\cdot\text{ml}^{-1}$, which is unlikely to be of clinical relevance when classifying inflammation status, given that chronic low-level inflammation is defined as a 2- to 4-fold increase in circulating pro- and anti-inflammatory cytokines (Bruunsgaard, 2005). With regards to the relevance in an athletic population, the lower threshold for plasma IL-6 concentration to result in performance impairment is currently unknown.

Despite ensuring the arterialisation protocol was tightly adhered to in the sIL-6R experiment, the mean resting concentration of plasma sIL-6R was higher from venous blood compared to capillary blood. Several previous investigations have reported discrepancies between capillary and venous blood samples for the measurement of blood glucose (Colagiuri et al., 2003), lactate (Foxdal et al., 1990), ionised magnesium (Matthiesen et al., 2002) and *ex vivo* stimulated and non-stimulated TNF- α and IL-10 production (Eriksson et al., 2007). It was suggested that the rate of glucose consumption in the tissues can account for the difference between blood glucose concentrations from venous and capillary samples (Kuwa et al., 2001). Likewise, lactate uptake and utilisation by both the muscles in the inactive forearm, and in the heart and liver might explain the difference between capillary and venous samples for the measurement of lactate (Foxdal et al., 1990). It was speculated that the difference in ionised magnesium between capillary and venous blood at actual pH was due to the higher pH of capillary blood, which may have been as result of greater exposure to air in capillary tubes (Matthiesen et al., 2002). None of these explanations are likely to apply to the observed heterogeneous distribution of sIL-6R in blood plasma at rest in the current study. However, it has been demonstrated that capillary blood contains a higher white cell count than venous blood, in particular of

monocytes and neutrophils (Daae et al., 1988; Yang et al., 2001), which may help explain the observed differences between capillary and venous samples for the measurement of TNF- α and IL-10 (Eriksson et al., 2007). It could be speculated that this evidence might also support the finding of a difference in sIL-6R between capillary and venous samples. When stimulated with CRP, neutrophils experience a shedding of the membrane-bound IL-6 receptor (Jones et al., 1999) and this process may be augmented in capillary blood where the neutrophil count could be higher than venous blood. However, the participants in the current study were healthy, active males and were therefore unlikely to display chronically elevated CRP levels. Despite the observed differences, the strong positive correlation between plasma sIL-6R concentrations from venous and capillary blood sampling methods would suggest it appropriate to apply a correction factor, allowing the use of capillary sampling to measure plasma sIL-6R concentrations.

Although it might be more feasible to monitor plasma IL-6 in athletes on a regular basis using a less invasive capillary sampling method, the lower threshold for resting plasma IL-6 concentration to result in performance impairment is unknown. Previously, when trained runners were infused with rhIL-6 to elicit plasma IL-6 values of $21.7 \pm 15.4 \text{ pg}\cdot\text{ml}^{-1}$, a significant decrement in 10 km time trial performance was observed compared to a placebo trial (Robson-Ansley et al., 2004). However, a wide range of resting plasma IL-6 concentrations have been reported in endurance athletes, from $3.43 \pm 3.75 \text{ pg}\cdot\text{ml}^{-1}$ in well-trained distance runners (Cox et al., 2008), to $136 \pm 16 \text{ pg}\cdot\text{ml}^{-1}$ in elite rowers during a normal training phase (Smith et al., 2011). This discrepancy is perhaps due to the large muscle volume recruited during rowing compared to running (Fischer, 2006). It might be necessary therefore, to routinely monitor the plasma IL-6 responses at rest in endurance athletes throughout a full competitive season, in order to identify the norm for an individual. Possible fluctuations should be monitored across a variety of training phases and deviations from the norm might be indicative of training stress and potential risk of performance impairment.

In addition, capillary blood sampling may also be useful to monitor the plasma IL-6 response to exercise in athletes. The current study observed a similar relationship between the 2 methods in response to exercise of differing durations and modes, despite exercise duration being a major determining factor in the amplitude of the IL-6 response, in addition to the volume of muscle mass involved. The measurement of plasma IL-6 in response to a single exercise session could be used to characterise recovery status, as the

cytokine response may actually reflect the concurrent and/or existing energy deficit (Jürimäe, et al, 2011). For example, increases of up to 100-fold have been observed after marathon running (Ostrowski et al., 1999). This is supported by the finding that the post-exercise IL-6 response is dependent on pre-exercise muscle glycogen content (Steensberg et al., 2001) and it appears that increasing recovery time between training sessions attenuates the post-exercise elevation in IL-6 (Ronsen et al., 2002).

Despite the potential advantages of capillary blood sampling in terms of practicality in the field and minimal discomfort for the athlete, it should be noted that the bias \pm 95% limits of agreement indicated that venous and capillary sampling sites were not consistently in complete agreement. This suggests that it is inadvisable to use the 2 sampling methods interchangeably and one sampling method should be utilised consistently to avoid error in interpreting plasma IL-6 concentration at rest or in response to exercise. A further limitation is the need for an effective arterialisation protocol when sampling at rest and lengthy ELISA analysis required to quantify plasma concentrations of IL-6 or sIL-6. In addition the inter-assay coefficient of variation (CV) for IL-6 was 8.0%, which limits the sensitivity for detecting changes between samples run on different ELISA plates. However, the development of a new real-time biosensor for the detection of IL-6 (Huang et al., 2013), presents the possibility of instant feedback for coaches and athletes in the elite population.

2.5 Conclusion

It was possible to determine plasma IL-6 at rest and in response to different exercise modes and durations in both venous and capillary blood samples. Strong correlations were evident between capillary and venous IL-6 concentrations and there were no differences between methods at rest when blood was effectively arterialised and in response to exercise, suggesting that capillary blood can be used as an alternative sampling method to monitor plasma IL-6. A difference between methods was observed for the measurement of resting sIL-6R, although the observed positive correlation suggests that a correction factor can be applied to allow the use of capillary blood sampling. However, a consistent sampling method must be implemented and it is inadvisable to use venous and capillary sampling interchangeably for the measurement of both plasma IL-6 and sIL-6R concentrations.

Chapter III

Tapering Strategies in Elite Endurance Runners

3.0 Chapter Summary

The aim of this chapter was to describe and compare the pre-competition training practices of elite British and Kenyan endurance runners. Training details from elite British middle-distance, long-distance and marathon runners were collected via survey for 7 d in a regular training phase, and throughout a pre-competition taper. Data were also analysed retrospectively from diaries of Kenyan long-distance runners, which detailed training undertaken 1 week prior to the 2004 and 2005 Kenyan national trials. It was found that tapering methods differ between event groups, specifically that marathon runners reported a longer taper duration (14 d) than long-distance and middle-distance runners (both 6 d). The peak interval training intensity during tapering was faster than race-speed ($P < 0.05$) in British marathon (114%) and long-distance (112%) runners and also in Kenyan long-distance runners (115%). In contrast, British middle-distance runners did not train faster than race-speed during tapering (100%). The extent to which training was manipulated during the taper was associated with training load prior to the taper in elite British endurance runners. Algorithms were generated to predict and potentially prescribe taper content based on regular training.

3.1 Introduction

The selection of a tapering strategy in elite sport is largely determined by the daily interaction and decision making between coach and athlete, based upon their own observations and experience with little or no scientific data input (Mujika et al., 2002). Previously, studies have manipulated the various training load variables as part of a structured taper (Houmard et al., 1994; McConell et al., 1993; Mujika et al., 2000; Mujika et al., 2002), or by way of theoretical mathematical modelling (Thomas & Busso, 2005). It appears that dramatic reductions in training volume can be implemented during this period (Mujika et al., 2000; Wittig et al., 1989), however it is clear that training intensity must not be compromised (McConell et al., 1993) and the inclusion of high intensity interval training during the taper is necessary to enhance performance (Houmard et al., 1994; Shepley et al., 1992). Since the tapering strategies of elite athletes have not been widely documented without experimental intervention, it is unclear whether this strategy is adopted in practice. The aim of the present study was to investigate the current tapering

practices of elite endurance runners and to explore relationships between regular and tapered training phases. Survey data was gathered on training during both regular training and tapering periods, across a number of endurance running events in elite British athletes in the 2012 Olympic year. The secondary aim was to compare the training practices in the final days before competition of British runners with highly successful Kenyan runners.

3.2 Methods

3.2.1 Survey

A survey (Bristol Online Surveys, University of Bristol, UK) was made available to the athletes for a 5 month period, or alternatively a paper version was adopted (appendix F). The survey collected details of the main event contested at championships and personal lifetime best time for this event, in addition to training information specific to regular training (RT). RT was defined as the last week of full training before load modification as part of a taper prior to competition. Participants provided details of the duration and distance of training completed each day during this period, which was categorised into continuous or interval running. Interval running represented division of the training session into high intensity repetitions (\geq lactate turnpoint speed) interspersed with recovery periods (light jogging or rest) (Billat, 2001). Lactate turnpoint speed was estimated from season's best 10 km performance (Jones, 2006; Petit et al., 1997) or prediction of this speed if athletes did not possess a 10 km performance (Daniels, 2005). Continuous running described the remaining training sessions, where running was uninterrupted and of lower intensity. Although endurance athletes have been shown to accurately self-report training (Sylta et al., 2014), training data was cross-validated against global positioning system (GPS) data from the corresponding RT week for participants who were already habitual users of GPS. The above training information was also gathered for the entire tapered training period (TT) prior to major competition and was not limited to 1 week. Participants referred to training diaries to detail the required training data.

3.2.2 Participants

The survey inclusion criteria specified that participants were current senior British athletes and had competed at national, international and/or Olympic level in at least 1 of the following events; 800 m, 1,500 m, 3,000 m steeplechase, 5,000 m, 10,000 m or the marathon. Thirty-seven participants (male: $n = 21$; female: $n = 16$; age: 26.0 ± 4.6 y) met the criteria and completed the survey. Personal lifetime best performance times for the

specified competition event were within 2-12% and 1-14% of the British records in 2012 for male and female athletes, respectively (Table 3.1). Middle distance events (MD) were defined as 800 m and 1,500 m and long distance events (LD) as 3,000 m steeplechase, 5,000 m and 10,000 m (Winter et al., 2007). Where appropriate, marathon runners were considered as a separate group (MAR).

Table 3.1. Personal best performance time (mean \pm SD) and performance level expressed as percentage of the 2012 British record time for male and female participants in each event distance.

Event	Male			Female		
	<i>n</i>	Performance time (min:s.ms or h:min:s)	Percentage of 2012 British record	<i>n</i>	Performance time (min:s.ms or h:min:s)	Percentage of 2012 British record
800 m	3	1:48.7 \pm 0.8	93	3	2:02.4 \pm 3.2	95
1,500 m	7	3:41.3 \pm 3.4	94	5	4:07.7 \pm 5.3	96
3,000 m steeplechase	2	8:45.5 \pm 13.4	92	1	9:30.2 \pm 0.0	99
5,000 m	3	14:10.1 \pm 25.2	94	1	15:55.8 \pm 0.0	90
10,000 m	2	28:34.5 \pm 3.5	93	0	n/a	n/a
Marathon	4	2:15:46 \pm 1:43.0	95	6	2:31:54 \pm 2:48.8	88

Table 3.2. Personal best performance time (mean \pm SD) in 2004 and 2005 and performance level expressed as percentage of the 2012 Kenyan record time for participants in each event distance.

Event	2004			2005		
	<i>n</i>	Performance time (min:s.ms)	Performance level (%) Kenyan record)	<i>n</i>	Performance time (min:s.ms)	Percentage of 2012 Kenyan record
3,000 m SC	0	n/a	n/a	1	08:04.2 \pm 0.0	98
5,000 m	4	13:01.4 \pm 11.2	97	5	13:21.7 \pm 21.1	94
10,000 m	2	27:59.2 \pm 27.8	94	1	28:09.0 \pm 0.0	94

3.2.3 *Survey Data Analysis*

Training frequency was calculated as the number of continuous running and interval training sessions. Volume was the sum of distance reported for continuous running and interval training. Mean speed was determined from the volume divided by duration of each continuous run or interval repetition and expressed as a percentage of personal best race speed to represent training intensity. Frequency, volume and intensity from TT were calculated as a percentage of the corresponding values from RT. The interval session that elicited the highest average speed during TT was defined as the peak intensity interval training session. The volume of this session was calculated, in addition to the number of days from competition. Taper duration represented the number of days prior to competition that athletes began to modify training load from RT.

3.2.4 *Training Diaries and Analysis*

As part of previous research investigating energy balance and hydration status in elite Kenyan athletes, information was gathered on training practice 1 week prior to the 2004 Kenyan Olympic trials and the 2005 Kenyan World Championship trials (Fudge et al., 2006; Fudge et al., 2008). The participants included Kenyan athletes (mean \pm SD: age 20.1 \pm 1.8 y) competing in 3,000 m SC, 5,000 m or 10,000 m. Personal best times in 2004 and 2005 were within 1-7% and 1-8%, of the Kenyan records in 2012 respectively (Table 4.2). Information was provided on frequency and duration (min) of continuous running and frequency of interval running, in addition to volume (km) and duration (s) of repetitions completed. The mean speed of each participant's personal best race performance was calculated. The mean speed of each interval training session was determined from volume divided by duration for each repetition completed and recorded as a percentage of race speed. Training data in preparation for the races in 2004 and 2005 were pooled for further analysis, with 2 runners providing data from both years.

3.2.5 *Statistical Analysis*

Data were analysed using SPSS (Statistical Package for Social Sciences Inc. v19.0; Chicago, IL, USA) and were initially tested for distribution using the Shapiro-Wilk test. Data are presented as mean \pm SD or median (interquartile range). Statistical significance was accepted at $P \leq 0.05$.

Paired t-tests were used to compare self-reported training volume with GPS data and RT training variables with TT for each event group. One-way analysis of variance was used to

make event comparisons for TT variables, with Bonferroni *post-hoc* analysis. Pearson product moment correlation coefficient was used to assess the association between training in RT and in TT. Non-normally distributed data were analysed using the Wilcoxon signed-rank test, the Kruskal-Wallis test with Bonferroni *post-hoc* analysis and Spearman's correlation co-efficient. Multiple linear regression of RT training variables was used to predict taper duration and the proportion of RT training undertaken during TT for each variable. A correlation matrix (Table 3.3) was produced to determine RT predictors with the highest correlation with each TT dependant variable. Backward elimination was utilised as the method of entry in each regression analysis and the optimal model with a maximum of 3 predictor variables selected, due to the number of observations ($n = 37$). Normality of regression residuals was investigated by agreement of their frequency distributions with the superimposed normality curve. Homoscedasticity and linearity of the data was explored by plotting the standardised predicted values and the standardised residuals. Comparisons between British LD and Kenyan LD data were made using a one-way MANOVA.

Table 3.3. Correlation matrix of the associations between regular training (RT) and the proportion undertaken during tapering (TT) for all training variables (pooled data).

Regular training	Tapered training						
	Taper duration	Continuous volume	Interval volume	Continuous frequency	Interval frequency	Continuous intensity	Interval intensity
Continuous volume	0.35*	-0.70*	0.16	-0.39*	0.20	0.67*	0.42*
Interval volume	0.31	0.09	-0.15	0.33*	-0.08	0.37*	0.23
Continuous frequency	0.47*	-0.70*	0.15	-0.63*	0.18	0.60*	0.20
Interval frequency	-0.36*	0.53*	-0.35*	0.35*	-0.41*	-0.50*	-0.29*
Continuous intensity	0.62*	-0.41*	0.13	-0.22	0.05	0.97*	0.60*
Interval intensity	0.31	-0.16	0.14	-0.12	0.15	0.69*	0.81*

* $P < 0.05$

3.3 Results

3.3.1 Training

Self-reported training data reported in the survey were cross-validated with GPS data for RT (volume: 129 ± 53 km vs. 132 ± 49 km; continuous intensity: 64 ± 10 vs. $64 \pm 12\%$ race speed; interval intensity: 99 ± 8 vs. $100 \pm 7\%$ race speed, respectively; all $P > 0.05$) and subsequently the self-report data were used for analysis. Differences between RT and

TT for volume, frequency and intensity of training confirmed the presence of a taper (Table 3.3). The MAR group reported a longer taper duration and a greater reduction in continuous running volume during TT than both MD and LD (all $P < 0.05$; Table 3.3). The proportion of RT interval running intensity during TT was greater in MD runners compared to MAR runners ($P < 0.05$; Table 3.4).

3.3.2 Peak Intensity Interval Session during TT

The interval training session that elicited the peak intensity during TT was completed at speeds exceeding personal best race speed in LD and MAR runners (112 (27)% and 114 (3)%, respectively; both $P < 0.05$). The MD group did not elevate the speed of the peak intensity interval session above personal best race speed (100 (2)%). The MAR runners completed a higher volume of running during the peak intensity interval session than either MD or LD (5 (2) km vs. 2 (1) km and 2 (4) km; $P < 0.05$) and the session was further from competition (10 (7) d vs. 3 (2) d and 3 (2) d; $P < 0.05$).

Table 3.4. Training completed during regular training (RT) and tapered training (TT) for middle distance (MD: $n = 18$), long distance (LD: $n = 9$) and marathon (MAR: $n = 10$) groups (male and female data combined). RT training volume is calculated as kilometres per week ($\text{km}\cdot\text{wk}^{-1}$), frequency as runs per week ($\text{run}\cdot\text{wk}^{-1}$) and intensity as a percentage of personal best race speed (%RS). For TT, all values are calculated as a percentage of the corresponding RT variable. Statistical comparisons between cohorts performed on the TT percentage data. Data presented as median (interquartile range).

Training Variables	M D		L D		MAR	
	RT	TT	RT	TT	RT	TT
Taper duration (d)	-	6 (3) [†]	-	6 (1) [†]	-	14 (8)
Training volume (RT; $\text{km}\cdot\text{wk}^{-1}$, TT; %RT)						
Continuous running	64 (41)	70 (16) ^{*†}	101 (60)	71 (24) ^{*†}	158 (40)	53 (12) [*]
Interval running	9 (5)	53 (45) [*]	13 (3)	67 (23) [*]	10 (4)	64 (34) [*]
Training frequency (RT; $\text{run}\cdot\text{wk}^{-1}$, TT; %RT)						
Continuous running	7 (2)	85 (33) [*]	9 (3)	88 (24) ^{*†}	11 (1)	73 (14) [*]
Interval running	3 (1)	100 (50)	2 (2)	100 (25)	1 (0)	100 (14)
Training intensity (RT; %RS, TT; %RT)						
Continuous running	60 (6)	96 (5) [*]	72 (6)	96 (6) [*]	84 (8)	97 (9) [*]
Interval running	96 (9)	104 (7) ^{*†}	108 (14)	100 (1)	110 (9)	99 (7)

^{*}different to RT ($P \leq 0.05$), [†]different to MAR group ($P \leq 0.01$)

3.3.3 RT and TT Relationships

Higher weekly RT continuous running volumes and frequencies were associated with a greater reduction in continuous running volume during TT (both $R = -0.70$; $P < 0.05$; Figures: 3.1A, 3.1B). Similarly, there was a greater reduction in training frequency during the TT period when there was a high frequency of continuous running ($R = -0.63$; $P < 0.05$; Figure 3.1C) and interval training during RT ($R = -0.41$; $P < 0.05$). When expressed as a percentage of race speed, the intensity for both continuous and interval training during RT was positively associated with intensity during TT (continuous intensity: $R = 0.97$ and interval intensity: $R = 0.81$; both $P < 0.05$; Figures: 3.1D, 3.1E). Taper duration was longer when the intensity of continuous running was a greater percentage of race speed ($R = 0.62$; $P < 0.05$; Figure 3.1F). Based on these data, prediction models for taper duration and taper training load variables were created from the pooled data (Table 3.5). Regression residuals were normally distributed for all models presented, however heteroscedasticity was evident for the model predicting interval volume. The algorithms generated had strong correlations to TT variables and the highest prediction accuracy was demonstrated for the continuous intensity prediction model.

3.3.4 British vs. Kenyan Comparison

In both groups, peak interval intensity was significantly higher than race speed ($P < 0.05$; Table 3.5), but not different between groups ($P = 0.26$). The volume of the peak session was higher in Kenyan runners compared to British runners ($P < 0.05$; Table 3.6). The proximity of the peak interval intensity session to competition was not different for British and Kenyan runners.

Frequency and total duration of continuous running was lower in Kenyan runners compared to British runners (5 ± 2 runs \cdot wk $^{-1}$ and 246 ± 62 min vs. 7 ± 2 runs \cdot wk $^{-1}$ and 333 ± 94 min, respectively; $P < 0.05$). Total interval frequency and interval volume were not different between British runners (2 ± 1 runs \cdot wk $^{-1}$ and 11 ± 3 km \cdot wk $^{-1}$) and Kenyan runners (2 ± 1 runs \cdot wk $^{-1}$ and 9 ± 4 km \cdot wk $^{-1}$). Average interval session intensity was higher in Kenyan runners compare to British runners ($112 \pm 4\%$ vs. $105 \pm 9\%$, respectively; $P < 0.05$).

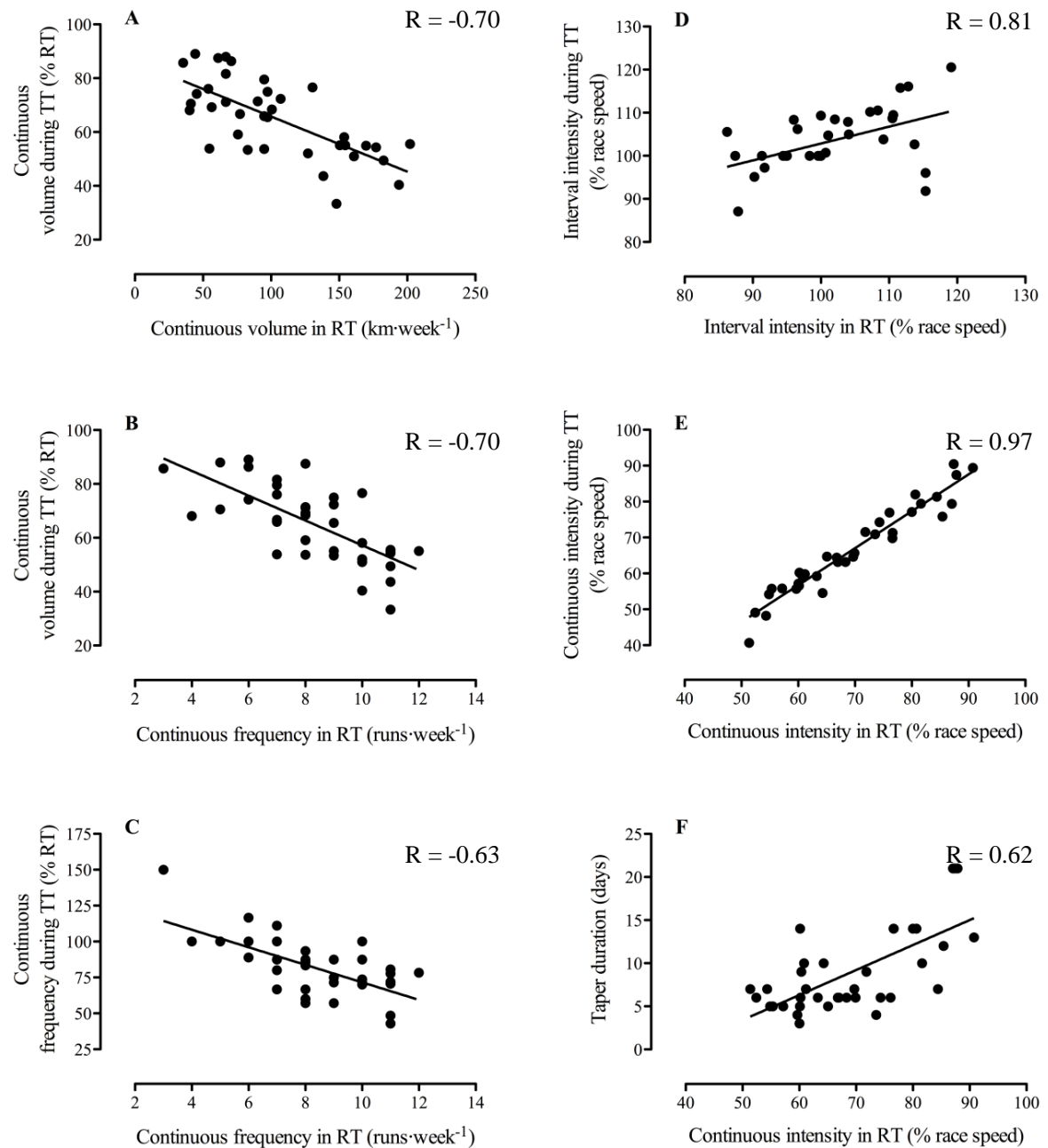


Figure 3.1. Associations between continuous running volume and frequency during the regular training phase (RT) and continuous running volume undertaken during the tapered training phase (TT) (expressed as a percentage of the RT phase) (A: $R = -0.70$; B: -0.70 ; $P < 0.05$) and between RT continuous running frequency and the proportion undertaken in TT (C: $R = -0.63$; $P < 0.05$). Associations between RT and TT for the intensity of interval running (D: $R = 0.81$; $P < 0.05$) and the intensity of continuous running (E: $R = 0.97$; $P < 0.05$), when expressed as a percentage of race speed. Association between continuous intensity in RT and taper duration (F: $R = 0.62$; $P < 0.05$).

Table 3.5. Prediction models for tapered training with regular training phase volume, frequency and intensity as predictors using pooled data.

Tapered training variable	Prediction models	R ²	SEE
Taper duration	Taper duration = 4.102 + (0.426*RT continuous intensity) + (-0.241*RT interval intensity)	0.54	3.4
Continuous volume	% of RT continuous volume in TT = 97.153 + (-0.106*RT continuous volume) + (-2.547*RT continuous frequency)	0.53	10.1
Interval volume	% of RT interval volume in TT = 131.585 + (-0.238*RT continuous volume) + (-19.106*RT interval frequency)	0.24	25.7
Continuous frequency	% of RT continuous frequency in TT = 130.800 + (0.211*RT continuous volume) + (1.059*RT interval volume) + (-10.016*RT continuous frequency)	0.60	13.5
Interval frequency	No difference between RT and TT for any event group. Maintain RT interval frequency during TT	-	-
Continuous intensity	% of race speed in TT = -13.443 + (-0.070*RT continuous volume) + (0.946*RT continuous frequency) + (1.141*RT continuous intensity)	0.95	2.9
Interval intensity	% of race speed in TT = 34.356 + (0.684*RT interval intensity)	0.66	4.4

Key: TT (tapered training), RT (regular training), RT predictors: taper duration (d), volume (weekly km), frequency (weekly training occurrences), intensity (% race speed). SEE (standard error of the estimate); taper duration (d), volume, frequency, intensity (%)

Table 3.6. Peak interval training intensity during the tapered training phase, volume of this interval session and proximity to competition for both Kenyan and British LD groups.

Group	Peak interval training intensity (% personal best race speed)	Volume of peak intensity interval session (km)	Proximity to competition (d)
Kenyan athletes	115 ± 4*	4.5 ± 1.4	3 ± 2
British athletes	111 ± 13*	2.9 ± 2.0†	3 ± 1

*significantly different to personal best race speed ($P \leq 0.05$), †significantly different to Kenyan athletes ($P \leq 0.05$)

3.4 Discussion

To the author's knowledge, the present study is the first to document the tapering strategies implemented by elite endurance runners in events from 800 m to marathon. The study also demonstrates for the first time that the volume, frequency and intensity of training undertaken during RT are indicative of the training characteristics of TT and that there are event-specific differences between tapering strategies. Furthermore, LD and MAR runners reported peak interval training intensity above race speed during TT, whereas MD runners trained at race speed. The main outcome of the present study is the development of algorithms which are capable of explaining a large proportion of the variance (53-95%) in tapering strategy training variables (with the exception of interval volume) for a given RT training load in endurance runners. This could provide a useful tool for coaches and athletes to aid tapering strategy design, based on the current practices of elite British athletes.

A meta-analysis of the effects of tapering on performance suggests that the optimal tapering strategy consists of a 41-60% reduction in training volume, whilst maintaining frequency and intensity for 2 weeks (Bosquet et al., 2007), whilst an insufficient reduction in training volume undermines the beneficial effects of a taper (Houmard et al., 1989; Neary et al., 2003). In contrast to the recommended common principles for tapering (Bosquet et al., 2007), our data suggest that despite general reductions in overall volume, tapering strategies are individualised, with the content based on the training undertaken in RT. For example, high RT weekly running volume was associated with a lower percentage of continuous running volume during TT, compared to athletes undertaking a low weekly running volume in RT. A possible explanation is that athletes undertaking a higher total running volume prior to the taper require a greater reduction in continuous volume during

this period in an attempt to alleviate an accumulated and chronic fatigue. This is supported by evidence that more superior performances in the careers of elite swimmers were achieved following an increase in training load leading up to the taper, and a sharp decrease in the taper period (Hellard et al., 2013). It has been suggested previously that the degree of fatigue prior to the taper might influence tapering strategy delivery (Bosquet et al., 2007), with greater fatigue requiring a longer taper in order to improve performance (Thomas & Busso, 2005; Thomas et al., 2008). This might explain why the MAR runners reported a longer taper and a greater reduction in continuous running volume during TT compared to MD and LD, potentially owing to greater residual fatigue at TT onset. It was also apparent that taper duration was extended when continuous running intensity was closer to race speed in RT, which was evident in the MAR group compared to LD and MD (84% vs. 72% and 60% of race speed, respectively).

Evidence suggests that performance is not compromised after taper periods of up to 3 to 4 weeks in endurance runners (Harber et al., 2004; Houmard et al., 1990; Mujika et al., 2000; Wittig et al., 1989), if intense training is included to maintain aerobic fitness (Luden et al., 2010; McConell et al., 1993) and prevent a decline in performance. An increase in interval intensity from regular training during the taper appears to have profound positive effects on performance (Shepley et al., 1992). Consistent with this, all groups maintained (LD and MAR) or slightly increased (MD) the average intensity of interval training during TT compared to RT, and a strong positive association was evident between RT and TT for interval training intensity. Despite an increase in interval intensity compared to regular training, MD athletes did not run faster than race speed in the interval session that elicited the peak intensity during TT. In contrast, LD and MAR runners performed their peak intensity interval session above race speed (median: 112% and 114%, respectively). It is uncertain why MD athletes do not exceed race speed in the same way that other groups do during taper interval training. The volume of the peak intensity session was greater than race distance for the MD group, which may be a contributing factor to the absence of intensity above race speed. Furthermore, it may be that MD athletes complete intervals at a more race-specific speed in this session, in order to practice race pacing prior to competition. Moreover, 100% of race speed in middle distance events equates to higher absolute speeds than the same percentage of long distance event race speeds. Therefore, MD athletes could be reluctant to execute training close to competition at the absolute

speeds of 112-114% of race speed similar to LD and MAR runners, for fear of potential increased injury risk.

The present study confirmed that both British and Kenyan LD runners performed the peak intensity interval session before competition at speeds above race pace, this session was completed the same number of days before competition and the total interval session frequency and volume was not different between nations during this period. However, the volume of the peak intensity session was higher in Kenyan runners with the frequency and total duration of continuous running being lower compared to British runners.

Since no difference was found between Kenyan and British long distance runners, it can be speculated that completing the peak intensity interval session above race speed in the final days of the taper is common practice among elite athletes in this event group. A question remains as to whether this strategy would be beneficial to middle distance runners. Conceptually, it is possible that a single training session performed faster than race speed in the final days of the taper could better prepare the athlete for performance on race-day. Considering insight from the Variable Dose-Response Model (Busso, 2003), the peak interval intensity during TT may be completed at intensities above race speed because the athlete is more fully recovered from previous overload and has greater capacity to respond effectively to training during the taper, without significant additional fatigue (Mujika, et al., 2004). In support, computer simulations displayed a positive effect on performance when training load was increased in the final days of the taper (Thomas et al., 2009). This may increase confidence and perceptually, race speed could feel more comfortable, having run much faster in the days before. Acute physiological responses following intensified training during a taper are evident, including increased buffering capacity (Houmard et al., 1994), increases in oxidative enzyme activity, red blood cell volume and muscle glycogen (Shepley et al., 1992). However, it is unknown whether a single profound increase in training intensity in the final 3 days of the taper would result in such adaptation and improve subsequent running performance in middle-distance runners, when incorporated into existing tapering strategies.

An additional aspect to consider is whether British runners should implement a greater reduction in continuous running volume and frequency and complete a greater volume in the peak intensity interval session. Kenyan runners reported a higher volume for the peak intensity session compared to British runners, despite no difference between nations for the

relative intensity of the session. This might be explained by the lower volume and frequency of continuous running in Kenyan athletes in the final week before competition, which translates into greater recovery time between training sessions than in British runners. It can be speculated therefore, that Kenyan athletes are able to cope with a greater training volume in the peak session and also a higher average intensity of interval running over the 7-d period before competition.

The current British data allowed the formation of equations that linked tapering strategy to RT load. Taper duration, in addition to taper training volume, frequency and intensity, can be established based on predetermined RT load characteristics, with particularly good accuracy for most models. Variance is limited for interval volume (SEE: 25.7%), although the validity of this prediction model can be questioned since the assumption of homoscedasticity of residuals was violated. The size of the error term differed across values from the independent variable of interval volume and standard error may be biased. For this variable, the average value reported by British athletes in the applicable event group should be implemented.

As an example, if the RT continuous running of a 1,500 m athlete consists of a frequency of eight runs, totalling 70 km of volume at 60% mean 1,500 m race speed, the current work would predict that their respective TT continuous running would be comprised of 69% RT volume (48 km), 76% RT frequency (6 runs) at 58% mean 1,500 m race speed. If the interval training in RT of the athlete consists of 3 sessions, totalling 10 km volume at 90% mean 1,500 m race speed, their respective interval training in TT would be predicted as 53% RT volume (5.3 km), 100% RT frequency (3 sessions) at 96% mean 1,500 m race speed. For the given RT training load, our data would predict a taper duration of 8 d.

Although the practices of elite athletes are mimicked throughout sport, it is not clear whether the current strategies of these runners are optimal for performance. It is possible however, that the reported practices have been refined through trial and error over many years of competition in the sport. Further research is required to investigate the influence of RT training load on tapering strategy and subsequent performance in order to fully validate the algorithms. For the elite athlete who is travelling to international competition, it is not always possible to follow a prescriptive regimen due to exposure to heat or altitude during the taper or long-haul travel, and it might be necessary therefore to adapt the taper to the specific circumstances (Pyne et al., 2009). The algorithms should be implemented

with caution however, since only previous training is considered in prescribing the taper and there is the assumption therefore, that physiological rate of recovery for a given training load is similar among individuals. In addition, outliers were evident in the associations between RT and TT, most notably between continuous intensity in RT and taper duration.

3.5 *Conclusion*

The present data suggest that training undertaken in RT is correlated with the TT strategy. From an applied perspective, it may not be optimal to implement a standardised taper before every major competition, since athletes may not always approach competition having completed the same training load, although performance data is required to support this notion. It is clear that British LD and MAR runners increase interval intensity above race speed in the final days of the taper, as do Kenyan LD runners, but the possibility of a performance benefit from this strategy warrants further investigation. In particular in MD runners, for whom this strategy is not current practice.

Chapter IV

The Effects of a Higher Intensity Training Bout during Tapering in Middle-Distance Runners

4.0 Chapter Summary

In chapter III, middle-distance runners did not report training at an intensity above race speed during the taper. This chapter examined the influence of an increase in intensity in the final interval session within a tapering strategy on middle-distance running performance and physiological responses in well-trained runners. Ten runners completed 2 conditions, each involving a 7-d period of regular training and a 7-d period of tapering, separated by 3 weeks of regular training. In first condition, the taper was prescribed using the algorithm developed in chapter III, where interval intensity was equal to 1,500 m race speed (RP). In the other, the final interval training session was completed at 115% of 1,500 m race speed (HI). Performance assessments, including a 1,500 m treadmill time trial were completed before and after the regular training period and after the tapering period. Venous blood samples were collected on 8 occasions in each condition for the measurement of plasma IL-6, sIL-6R, CRP, T and C concentration. 1,500 m time trial performance was faster after the RP taper compared to pre-taper (3.4%; 288 ± 19 s vs. 298 ± 19 s; $P < 0.05$; ES = 0.53), but the effects of the HI taper were unclear (1.4%; 292 ± 19 s vs. 296 ± 18 s; $P = 0.53$; ES = 0.22). The data suggested that a tapering strategy prescribed based on current practices of elite middle-distance runners was *most likely* to improve performance, whereas the same strategy with an increase in intensity is not recommended. The potential biomarkers of plasma IL-6, sIL-6R, CRP, and the T:C ratio were not sensitive enough to indicate changes in recovery and performance across a 7-d period of tapering.

4.1 Introduction

The inclusion of high intensity training during the taper appears to be the key to improving performance (Houmard et al., 1994; Shepley et al., 1992). In combination with a large reduction in training volume, high intensity training during the taper has led to amplified physiological responses including; increased buffering capacity (Houmard et al., 1994), increases in oxidative enzyme activity, red blood cell volume and muscle glycogen content (Shepley et al., 1992) in endurance runners. In practice, elite Kenyan 3,000 m steeplechase, 5,000 m and 10,000 m runners performed their final interval sessions within the last 3 d of

the taper at an average running speed significantly above race pace ($15 \pm 4\%$), when preparing for the 2004 Olympic trials and 2005 World Championship trials (chapter III). On collating this data in elite British endurance runners, it became clear that long-distance and marathon runners also train at intensities above race speed within the final days of the taper period before competition, but this was not evident in middle-distance runners. Theoretically, an interval session completed at intensities above race speed late in the taper when the athlete is more fully recovered, might allow greater capacity to respond effectively to this type of training stimulus and further improve subsequent performance (Busso, 2003; Mujika, et al., 2004). In support, theoretical models have shown that a moderate increase in training load at the end of taper might further improve performance as the athlete can capitalise on additional adaptation, after initially overcoming accumulated fatigue from previous training (Thomas et al., 2009). Despite evidence of this practice in long-distance and marathon runners, it is not clear whether it would be of benefit to the performance of middle-distance runners.

It is possible that individual athletes recover at different rates during the taper and it would be beneficial therefore, for coaches and athletes to have an objective measure of their readiness for competition. This would allow further refinement of training load during the taper, if deemed necessary. In the elite population it is not feasible to employ the invasive procedures such as muscle biopsies, necessary to monitor a number of the physiological changes associated with the taper, so any such biomarker would need to be minimally invasive, with rapid sample analysis to allow prompt feedback. The longitudinal monitoring of variations in inflammatory cytokines; IL-6, sIL-6R, CRP, and the T:C ratio might be indicative of training stress and energy homeostasis throughout different phases of training (Adlercreutz et al., 1986; Jürimäe et al., 2011, Robson-Ansley et al., 2007), although their sensitivity to detect taper effectiveness in middle-distance runners requires investigation.

The aims of the current study were: i) to investigate the effectiveness of an algorithm-derived tapering strategy on 1,500 m time trial performance; and ii) to establish whether an increase in the intensity (115% race speed) of the final interval session during this tapering strategy can further enhance 1,500 time trial performance. Lastly, the aim was to explore potential biomarkers that might provide an objective indication of physiological readiness to perform. It was hypothesised that increasing the intensity of the final interval session to

115% of race speed will further improve post-taper performance in middle-distance runners.

4.2 Methods

4.2.1 Pilot Study

Six well-trained male middle distance runners: (mean \pm SD) age 21.4 ± 2.9 y, height 177.9 ± 7.7 cm, body mass 68.2 ± 2.8 kg, were recruited to test the reliability of 1,500 m time trial performance on an ‘on-response’ treadmill (MTC climb 2000, Runner, Italy). Personal best 1,500 m time was 251.9 ± 14.0 s (range: 231.3 – 265.7 s). Treadmill sensors allow the detection of user position on the belt and control belt velocity in accordance with user position relative to these sensors. Distance from the sensor results in acceleration, deceleration or maintenance of a constant speed, thus allowing the controller to initiate an autonomous speed adjusting system. Participants underwent familiarisation to the lab environment, equipment and testing protocol at least 7 d prior to testing. A standardised warm up was performed, consisting of 10 min running at a speed equivalent to 60% of individual personal best time for 1,500 m, followed by 2 sets of 10 s at 90% and 20 s at 60% and a 1-min rest period (Wiles et al., 1992). After a rolling start of 30 s at 60% of personal best time, the participant completed a 1,500 m time trial at self-selected speed. The treadmill was set to a 1% gradient throughout the trial (Jones & Doust, 1996). Speed and time indicators were concealed from the subject, but distance remained visible. The treadmill time trial was performed in a fasted state on 2 separate occasions, 7 d apart, after controlling for physical activity and diet in the 24 h before. Individual participant data for time to complete each 1,500 m time trial are presented in Table 5.1. The mean CV for time to completion of the treadmill time trial was 0.9%, similar to the variation reported for 1,500 m track time trial performance (0.8%) in well-trained runners (Hodges et al., 2006). Given that performance measures require a CV similar to, or less than the typical variation in competitive performance ($\sim 1\%$ in middle-distance runners) in order to detect the smallest worthwhile change (Hopkins, 2005), the ‘on response’ treadmill time trial was considered sensitive enough for use in the main experimental trials.

Table 4.1. Time to complete (s) 2 repeated 1,500 m time trials on an ‘on-response’ treadmill.

Participant	1	2	3	4	5	6	Mean	SD
Trial 1	265.4	281.9	267.0	265.2	287.2	321.0	281.3	21.6
Trial 2	259.8	292.7	266.4	263.5	287.3	323.5	282.2	24.3
Mean	262.6	287.3	266.7	264.4	287.3	322.3	281.7	22.8
SD	4.0	7.6	0.4	1.2	0.1	1.8		
CV (%)	1.5	2.7	0.2	0.5	0.0	0.5	0.9	

4.2.2 Participants

A separate group of 10 well-trained male middle distance runners: age 21.7 ± 3.0 y, height 182.9 ± 7.0 cm, body mass 73.4 ± 6.8 kg, volunteered to take part in the main experimental trials. This group included 2 participants who also completed the pilot study. Inclusion criteria specified that participants were competitive middle distance runners (800 m & 1,500 m) with a training history of at least 2 years and had trained consistently without interruption for the previous 2 months. Personal best 1,500 m time was 257.5 ± 26.9 s (range: 231.3 – 316.7 s).

4.2.3 Experimental Design

The study employed a repeated-measures cross-over design. Each of the 2 conditions involved a 7-d period of regular training (control) and a 7-d period of tapering (Figure 4.1). Conditions were separated by at least 3 weeks of regular training. Performance assessments were carried out on the day before the control period (day 1: baseline), on the day after the control period (day 9: post-control) and on the day after the taper period (day 17: post-taper). This structure was completed for both conditions, with 6 separate performance trials in total. At least 1 week prior to the first condition, participants became accustomed to the laboratory environment, equipment and testing protocol, including completion of the performance measures. Participants were not informed about the precise differences between the conditions, but could not be blinded to the manipulation of training load.

Training during the control period was determined by the participant and recorded objectively from their own GPS data. Participants were instructed to replicate the training performed in the control period of the first condition in the control week of the second condition. Training was categorised into continuous or interval running (described

previously: chapter 3.2.1) and quantified for mean weekly volume (km), frequency and intensity (% personal best 1,500 m race speed). During the taper period in the race-pace condition (RP), participants completed individualised training prescribed by the algorithms that were developed in chapter III, whereby the speed of the final interval session (described below in 4.2.4) was equivalent to the average speed of season’s best 1,500 m time. In the high intensity condition (HI), training during the taper period was the same as RP, except the final interval session was performed at 115% of the speed in RP. Training load was confirmed throughout all periods using GPS data.

As an example, if continuous running in the control period consisted of a frequency of eight runs, totalling 70 km of volume at 60% mean 1,500 m race speed, the algorithms would predict that continuous running during the taper period would consisted of 69% volume (48 km), 76% frequency (6 runs) at 58% mean 1,500 m race speed. If the interval training in the control period consisted of 3 sessions, totalling 10 km volume at 90% mean 1,500 m race speed, interval training during the taper would be predicted as 53% volume (5.3 km), 100% frequency (3 sessions) at 96% mean 1,500 m race speed. The 3rd and final interval session during the taper period would be completed at 100% race speed in RP and 115% race speed in HI.

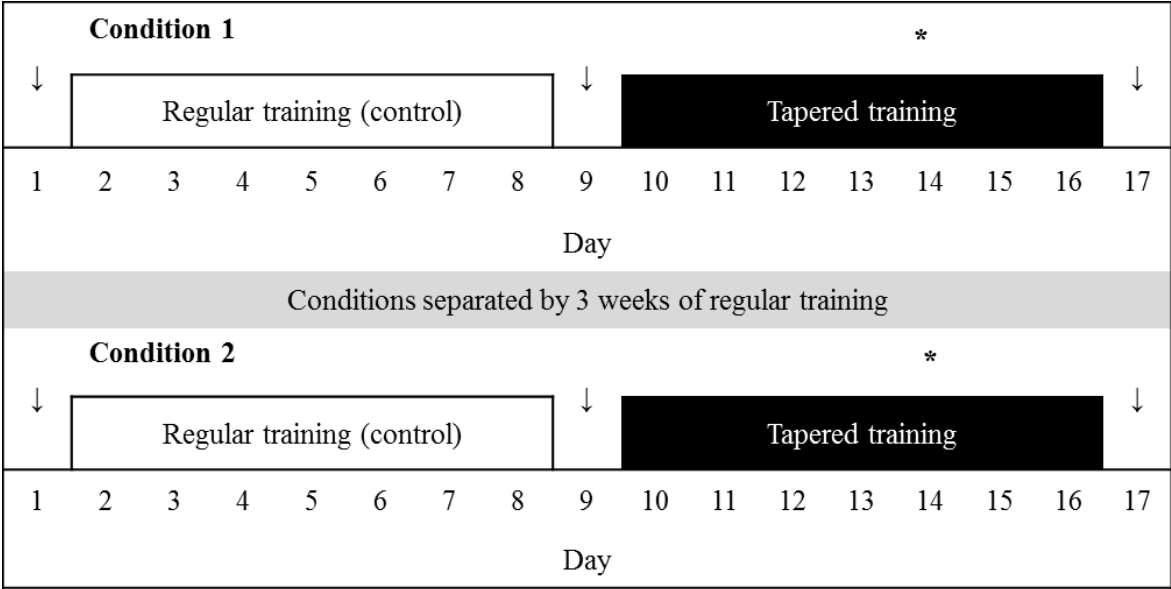


Figure 4.1. Experimental design illustrated by 2 experimental conditions, separated by at least 3 weeks of regular training. Arrows represent performance assessments and asterisk indicates laboratory interval session.

4.2.4 Laboratory Interval Session within Taper Period

A controlled interval running session was completed on a motorised treadmill (Woodway, Germany) on day 14, 3 d prior to the final performance test. Participants arrived at the laboratory between 0700 h and 0900 h in a fasted state. A standardised warm up was performed, as described above (Wiles et al., 1992). This was followed by a series of 300 m interval repetitions with 90 s recovery. The individualised number of repetitions was dependent upon interval volume calculated from the algorithm (chapter III). Intensity of the effort was equivalent to season's best 1,500 m race speed in the RP condition and 115% of season's best 1,500 m race speed in the HI condition. Heart rate was recorded in the 5 s of each repetition and participants indicated their RPE (appendix C) immediately after each repetition. A cool down of 10 min running at a speed equivalent to 60% of 1,500 m personal best race speed was performed after completion of the session.

4.2.5 Performance Assessment

Participants arrived at the laboratory in a fasted state between 0700 h and 0900 h on days 1, 9 and 17 of each condition. On arrival, body mass and height were recorded (described previously: chapter 2.2.2). Participants provided a venous blood sample (described previously: chapter 2.2.4), which was followed by assessments of muscle function. After a 30 min rest period, a 1,500 m treadmill time trial was completed.

4.2.5.1 Force Measurement

Participants were strapped into a custom built isometric strength rig, in a seated position in order to measure peak isometric voluntary knee extension (MVC) force. Knee angle was fixed at 60° flexion, using the centre of the knee joint as the fulcrum in relation to the greater trochanter at the top of the femur and the lateral malleolus in the ankle. The ankle brace was adjusted to 1 cm above the lateral malleolus on the right tibia. Participants placed their hands across their chest to further isolate the quadriceps contraction measurement and minimise contribution of the upper body. Eight sub-maximal contractions at intensities relative to perceived maximal force were performed as a warm-up (3x 25%, 2x 50%, 2x 75% and 1x 90%), followed by a series of 3-4 maximum contractions of ~3-5 seconds duration. A rest period of 30 s was enforced between contractions. Contraction force was displayed in real time on a computer monitor positioned in front of the participant in order to give feedback. Force was recorded using a calibrated S-beam strain gauge (0–1,500 N linear range; Force Logic, Swallowfield, UK)

strapped to the distal region of the tibia. Force data were sampled and recorded at 5,000 Hz using an external A/D converter (Micro 1401, CED, Cambridge, UK) and a PC utilising Spike 2 software (CED, Cambridge, UK). Force data were low pass-filtered at 500 Hz (4th order Butterworth digital filter) and notch filtered at 50 Hz (infinite impulse response digital filter [q-factor of 50]) to remove mains frequency noise and the peak force was used in data analysis. In the same position, explosive isometric voluntary knee extensor contractions were then performed to measure rate of force development (RFD). Approximately 10 attempts at explosive contractions of ~1 s duration were required. Participants were instructed to develop force as “fast and hard” as possible from rest. Approximately 20 s rest was taken between contractions. Only the explosive contractions that exceeded 80% of peak knee extensor force, and were performed without countermovement or pre-tension (<0.5 N deviation from baseline) were considered for analysis. Of those contractions, the 3 displaying the highest peak rate of force development were selected for further analysis, and the results averaged across these 3 contractions. RFD was assessed by measuring force at 50, 100 and 150 ms following force onset (F_{50} , F_{100} and F_{150} , respectively). Onset was defined as the last peak or trough before force exceeded the limits of the noise during the preceding 500 ms (Tillin et al., 2010). This systematic, manual identification of force onset has been shown to be both highly accurate (Allison 2003; Tillin et al. 2013) and reliable (Buckthorpe et al. 2012; Tillin et al. 2013).

4.2.5.2 1,500 m Performance Assessment

A 1,500 m treadmill time trial was completed after a standardised warm up (described above, Wiles, et al., 1992) on an ‘on-response’ treadmill (MTC climb 2000, Runner, Italy). After a rolling start of 30 s at 60% of personal best 1,500 speed, participants completed a 1,500 m time trial at self-selected speed. The treadmill was set to a 1% gradient throughout the trial (Jones & Doust, 1996). Speed and time indicators were concealed, but distance remained visible to participants. Heart rate was monitored at 30 s intervals. Participants did not receive any verbal encouragement or performance feedback.

4.2.6 Dietary Intake and Physical Activity Level

Dietary intake and physical activity were monitored throughout both conditions to assess consistency between control and taper and between RP and HI. Subjects were instructed to eat and drink *ad libitum* during the control and tapering period of each condition and to weigh all food and fluid consumed using scales (Salter Arc, Kent, UK). Dietary intake was

recorded in a food record diary (appendix G). Total energy intake and carbohydrate intake was calculated for each condition using nutritional analysis software (CompEat Pro 5.8.0, Grantham, UK).

Physical activity was monitored using ActiGraph GT3X+ triaxial accelerometer (ActiGraph, Pensacola, FL) during the control and taper periods of both HI and RP. Sampling frequency was set at 60 Hz, analysed in 60 s epochs. Devices were fitted at the midline of the right anterior hip and a total of 6 monitors were used throughout the study. Inter-unit reliability has been found to be acceptable (Santos-Lozano et al., 2012) but each participant wore the same accelerometer during all wear period to further reduce inter-device variability. Participants were instructed to wear the device each day from waking until sleep; removing for water-based activities such as bathing or swimming (appendix H). Days with fewer than 600 min of wear time data were classified as invalid and not included in the analysis. Non-wear time was defined as continuous runs of zeros lasting \geq 60 min, with no allowance for counts greater than zero. Cut-points (counts per minute) to classify sedentary, light and moderate to vigorous physical activity (MVPA) were 0 - 99, 100 - 1951 and 1952 - 9498, respectively (Freedson et al., 1998). Overall levels of physical activity were calculated using total average counts per minute for the control and taper periods in RP and HI. Time spent in sedentary, light and MVPA was calculated as a percentage of total wear time for the control and taper periods in RP and HI. Mean energy expenditure (calories per hour of wear time) was estimated from physical activity in the control and taper periods of both RP and HI.

4.2.7 Blood Sampling

In addition to days 1, 9 and 17, participants arrived at the laboratory in a fasted state to provide a venous blood sample on days 3, 7, 11, 14 and 15.

After the treadmill time trials and controlled laboratory interval session, capillary blood samples were obtained from the fingertip using an automated lancet at 0 min, 1 min and 2 min post-completion. An end-to-end capillary tube was used to collect 20 μ l of blood for each sample. The tube was transferred into an eppendorf prefilled with 1 ml of haemolysis solution, inverted and analysed for lactate using an automated analyser (Biosen C-Line, EKF Diagnostics, Barleben, Germany).

4.2.8 Blood Analysis

Venous blood samples were analysed for plasma IL-6, sIL-6R, CRP, T and C concentration via sandwich ELISAs (appendix D). Although chapter II concluded that capillary blood sampling could be used in the field to quantify plasma IL-6 and sIL-6R, venous samples were taken in the current study to allow for the additional measurement of CRP, T and C, and for comparison with previous findings from the literature.

4.2.9 Statistical Analysis

Data were analysed using SPSS (Statistical Package for Social Sciences Inc. v22.0; Chicago, IL, USA) and were initially tested for distribution using the Shapiro-Wilk test. Data are presented as mean \pm SD, unless specified otherwise. Statistical significance was accepted at $P \leq 0.05$.

Non-parametric tests were used where the data were not normally distributed, specifically energy intake in the final 3 d of the taper. Plasma IL-6 and CRP concentrations were log-transformed and included in analysis for normal data. All other data were confirmed to be normally distributed. Data gathered at the laboratory interval sessions were compared using paired samples *t*-tests. Performance measures from day 1 and 9 were also compared using a paired-samples *t*-test to ensure no-learning effect was present. No differences were evident, so the mean of the results from day 1 and day 9 were calculated to represent a control performance (no taper). Performance measures data, dietary intake and physical activity data were analysed via a 2-way repeated-measures ANOVA, with Bonferroni *post-hoc* analysis. Magnitude-based inferences about the true (population) effect of the RP taper and HI taper on 1,500 m running performance were calculated. The uncertainty in the effect was expressed as 90% confidence limits and as the likelihood that the true value of the effect represents substantial change; harm or benefit (Batterham & Hopkins, 2006). The smallest meaningful change in 1,500 m performance was assumed to be a reduction or increase in running time of 0.5% (Hopkins, 2005). Effect size (ES) was calculated and the magnitude was considered either trivial (0–0.19), small (0.20–0.49), medium (0.50–0.79) or large (≥ 0.80) (Cohen, 1992). To ensure accelerometer wear time was consistent across both trials, wear time was analysed using a 1-way repeated-measures ANOVA. Mean daily physical activity and carbohydrate intake from the final 3 d of the taper in RP and HI were compared using paired samples *t*-tests. Energy intake during this period was analysed using the Wilcoxon Signed Rank test. Plasma IL-6, sIL-6R, CRP concentrations and T:C

ratio were compared throughout RP and HI trials using a 1-way repeated measures ANOVA. *Post-hoc* analysis was carried out using the Bonferroni method, with adjustment for multiple comparisons.

4.3 *Results*

4.3.1 *Training*

Training completed during control and taper periods in both conditions is presented in Table 4.2. Outcome variables of the laboratory interval session are presented in Table 4.3.

Table 4.2. Volume, frequency and intensity of training in the control and taper periods (mean \pm SD; $n = 10$). Warm up and cool down data is not shown.

Training Variables	RP			HI		
	Control	Taper	% Δ	Control	Taper	% Δ
Training volume						
Continuous running (km \cdot wk ⁻¹)	45 \pm 13	33 \pm 9 [*]	-26%	41 \pm 15	30 \pm 9 ^{**}	-26%
Interval running (km \cdot wk ⁻¹)	10 \pm 4	6 \pm 2 [*]	-39%	9 \pm 4	6 \pm 2 ^{**}	-39%
Training frequency						
Continuous running (run \cdot wk ⁻¹)	4 \pm 1	4 \pm 0 [*]	-16%	4 \pm 1	4 \pm 0 ^{**}	-17%
Interval running (run \cdot wk ⁻¹)	2 \pm 0	2 \pm 0	0%	2 \pm 1	2 \pm 1	0%
Training intensity						
Continuous running (% race speed)	61 \pm 8	57 \pm 9 [*]	-6%	62 \pm 8	62 \pm 8 ^{**}	-6%
Interval running (% race speed)	89 \pm 8	95 \pm 5 [*]	+7%	94 \pm 8	98 \pm 6 ^{**}	+5%
Laboratory interval session (% race speed)	-	100 \pm 0	-	-	115 \pm 0 [†]	-

* Different to RP control, ** different to HI control, [†] different to RP taper, all $P < 0.05$.

Table 4.3. Average speed, heart rate, rate of perceived exertion (RPE) and peak lactate during the controlled laboratory interval session in the race-pace (RP) condition and in the high intensity condition (HI) (mean \pm SD).

Variable	RP	HI
Speed (km·h ⁻¹)	20.8 \pm 1.8	23.9 \pm 2.1*
Volume (km)	2.7 \pm 0.7	2.7 \pm 0.7
Heart rate (b.min ⁻¹)	169 \pm 9	178 \pm 7*
RPE	14 \pm 1	17 \pm 1*
Peak lactate (mmol·L ⁻¹)	3.8 \pm 1.6	9.9 \pm 3.4*

* Different to RP, $P < 0.05$.

4.3.2 1,500 m Performance Assessment

There was a main effect of taper (control training vs. taper training) on 1,500 m time trial performance ($P < 0.05$). An interaction effect ($P < 0.05$) suggests that time trial performance responded differently to the RP and HI tapers. There was no difference in control performance time between RP and HI ($P = 1.00$), however, there was a 3.4% improvement in performance time after the RP taper compared to control (288 \pm 19 s vs. 298 \pm 19 s; $P < 0.05$; Figure 4.2). Performance time after the taper in HI was not different to control (292 \pm 19 s vs. 296 \pm 18 s; $P = 0.53$). Individual responses to RP and HI conditions are shown in Figure 4.2. When considered relative to the smallest worthwhile change in performance, qualitative inference suggests that the tapering strategy in RP condition was *most likely* to be beneficial to 1,500 m time, whereas the HI condition was *unclear* (Table 4.4). Physiological responses to the 1,500 m time trial are shown in Table 4.5.

Table 4.4. Differences in pre- and post-taper 1500 m time trial performance improvements in the RP and HI conditions.

Taper condition	Mean improvement (s) and 90% confidence limits	Qualitative inference ^a	Effect size
RP	10.0; \pm 1.6	Most likely beneficial	0.53
HI	4.2; \pm 12.0	Unclear	0.22

^a with reference to a smallest worthwhile change of 0.5%.

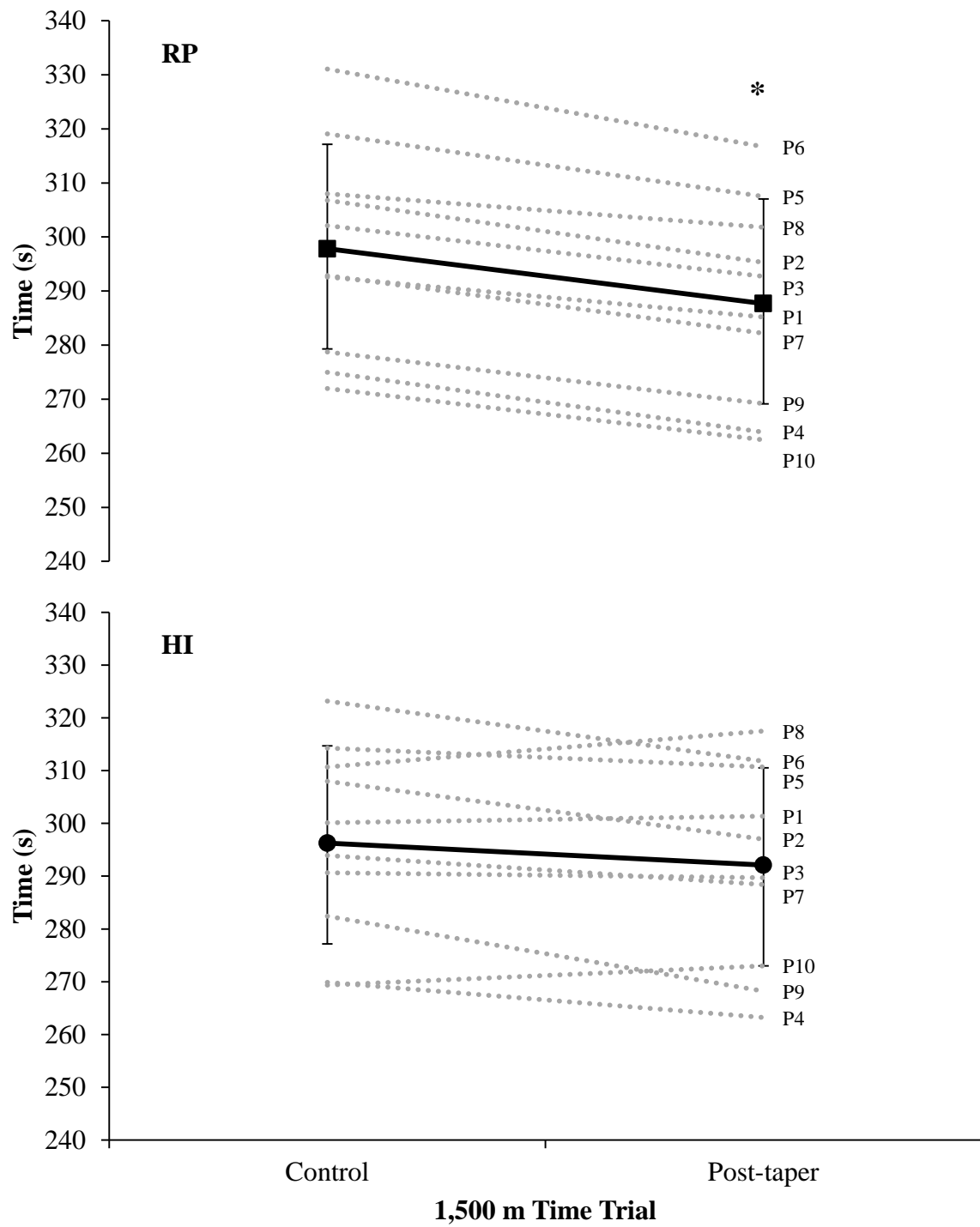


Figure 4.2. 1,500 m time trial performance after the control period and post-taper in the RP condition and in the HI condition. Individual times for each participant (P1-10) are represented by dashed lines and group mean \pm SD by solid line. * Different to control ($P < 0.05$).

Table 4.5. Pre and post-taper physiological responses to a 1,500 m time trial in the RP and HI conditions and results of the 2-way repeated measures ANOVA (mean \pm SD; $n = 10$).

Variable	RP			HI			<i>P</i> value		
	Pre-taper	Post-taper	Effect size	Pre-taper	Post-taper	Effect size	Strategy	Condition	Interaction
Peak blood lactate (mmol·L ⁻¹)	7.1 \pm 3.1	10.1 \pm 2.6	1.08	7.7 \pm 2.4	9.3 \pm 2.5	0.63	$P < 0.05$	NS	NS
Peak heart rate (bpm)	182 \pm 7	184 \pm 9	0.20	184 \pm 8	183 \pm 7	0.13	NS	NS	NS

(Strategy: control vs. tapering; condition: RP vs. HI; NS: non-significant).

4.3.3 Force Measurement

A main effect of tapering was evident for MVC force ($P < 0.05$) and for RFD ($P < 0.05$; Figure 4.3). There was a main effect of time on RFD (F_{50} vs. F_{100} vs. F_{150} ; $P < 0.05$). No interaction effect on MVC force was observed (RP: 722.3 ± 149.9 vs. 663.4 ± 153.1 N; +9%; ES = 0.39; HI: 721.7 ± 143.3 vs. 682.3 ± 130.2 N; +6%; ES = 0.29; $P = 0.40$), or a condition \times time interaction effect for RFD (RP: ES = 0.54, 0.50, 0.56; HI: ES = 0.22, 0.34, 0.22 for F_{50} , F_{100} , F_{150} , respectively; $P = 0.06$).

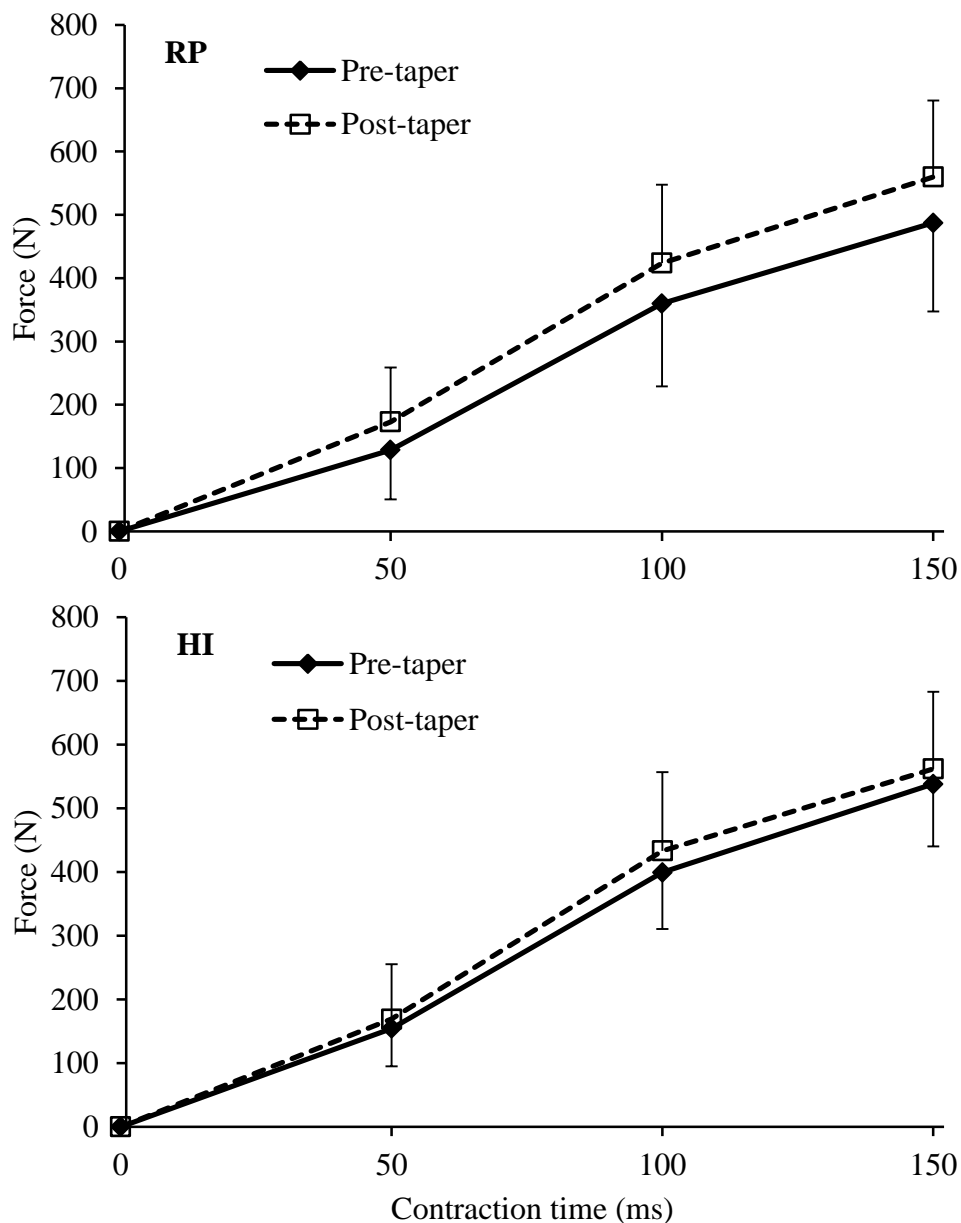


Figure 4.3. Explosive force production in the first 150 ms of explosive voluntary contractions, measured; pre-taper (filled diamonds) and post-taper (open squares) in RP and HI.

4.3.4 Energy Intake and Physical Activity

Mean daily energy intake remained consistent throughout both conditions (RP: 2907 ± 419 vs. 2812 ± 506 kcal; ES = 0.21; HI: 2815 ± 366 vs. 2728 ± 456 kcal; ES = 0.21; $P > 0.05$), as did carbohydrate consumption (RP: ES = 0.05; HI: ES = 0.09; $P > 0.05$). A main effect of tapering was evident ($P < 0.05$) when mean daily carbohydrate consumption was expressed relative to mean daily running volume (km), suggesting a daily carbohydrate excess during tapering compared to control, but without differences between conditions (RP: 1.1 ± 0.3 vs. 0.7 ± 0.2 g·kg bw·km⁻¹; ES = 1.50; HI: 1.1 ± 0.4 vs. 0.7 ± 0.2 g·kg bw·km⁻¹; ES = 1.01; $P > 0.05$). There was no change in body mass throughout both conditions (RP: 71.9 ± 7.0 kg vs. 72.0 ± 6.9 kg; ES = 0.01; HI: 73.1 ± 6.5 kg vs. 72.9 ± 6.8 kg; ES = 0.04; $P > 0.05$).

Daily accelerometer wear time was consistent throughout both conditions and included training completed (RP: ES = 0.14; HI: ES = 0.33; $P > 0.05$). Daily physical activity (counts·min⁻¹) was lower during tapering compared to the control period (main effect of tapering; $P < 0.05$). Time spent in MVPA was lower during tapering compared to control (main effect of tapering; -1.6%; $P < 0.05$). Time spent sedentary or in light physical activity was not different between strategies or conditions (all $P > 0.05$). Mean energy expenditure from physical activity was lower during tapering compared to control (main effect of tapering; -8 kcal; $P < 0.05$).

In the last 3 d of the taper after the laboratory interval session, there were no differences in physical activity (counts·min⁻¹; ES = 0.48; $P = 0.57$) or in time spent sedentary (ES = 0.10), in light (ES = 0.18), or in MVPA (ES = 0.25; all $P > 0.05$) between RP and HI. Mean daily energy intake was higher in HI compared to RP (+5%; 2704 ± 358 kcal vs. 2576 ± 447 kcal respectively; ES = 0.32; $P < 0.05$), but mean daily carbohydrate intake was consistent in both conditions (ES = 0.18; $P = 0.34$).

4.3.5 Blood Plasma Measurements

No differences in the inflammatory markers IL-6, sIL-6R and CRP were evident at any time point across the control and tapering periods in either RP or HI. Mean values for the control and taper periods in RP and HI are shown in Table 4.6. In HI, the T:C ratio was higher on days 1 and 17 compared to day 7 (18 ± 5 and 18 ± 4 vs. 14 ± 3 ; $P < 0.05$) and higher on day 9 compared to day 3 (16 ± 3 vs. 15 ± 4 ; $P < 0.05$; Figure 4.4). No differences in T:C ratio were evident in the RP trial.

Table 4.6. Mean concentration of inflammatory markers; IL-6, sIL-6R and CRP, during the control and taper periods in the RP and HI conditions.

Inflammatory Biomarker	RP Condition		Effect Size	HI Condition		Effect Size
	Control	Taper		Control	Taper	
IL-6 (pg·ml ⁻¹)	2.5 ± 3.1	3.1 ± 3.5	0.18	4.2 ± 5.1	5.1 ± 6.0	0.16
sIL-6R (ng·ml ⁻¹)	34.1 ± 9.7	33.1 ± 9.2	0.11	30.7 ± 6.8	31.2 ± 7.2	0.07
CRP (µg·ml ⁻¹)	1.0 ± 1.2	0.6 ± 0.6	0.44	0.9 ± 0.9	1.1 ± 2.1	0.13

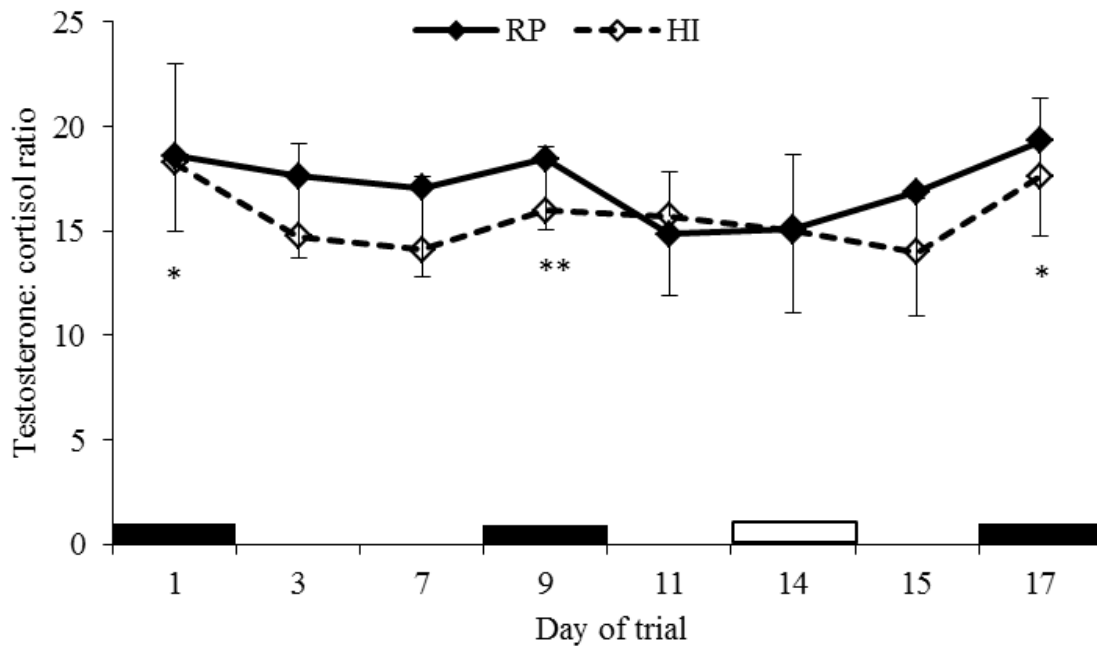


Figure 4.4. Testosterone to cortisol ratio in the RP condition (solid line) and in the HI condition (dashed line) (Mean ± SD; shaded blocks: performance measures on days 1, 9 and 17; open block: laboratory interval session). * Different to day 7 ($P < 0.05$), ** different to day 3 ($P < 0.05$).

4.4 Discussion

The main finding of the present study was that 1,500 m performance time improved by 3.4% after a race-pace tapering strategy, but contrary to the hypothesis, the effect of an increase in interval intensity above race speed on performance was *unclear*.

Improvements of between 0.5-6.0% can be expected in response to a successful taper (Mujika & Padilla, 2003). The observed improvement in performance from the RP taper (3.4%) falls within this range and was *most likely* to have a positive effect on performance.

As suggested by 90% confidence limits, negative results were experienced by some athletes implementing the HI taper. The 1st, 6th and 8th fastest participants experienced a worsening in performance time after the HI condition, which also demonstrates that individual responses did not appear to be related to performance standard. Nevertheless, performance time after the HI strategy was improved in 6 out of 10 individuals in excess of the smallest worthwhile change in performance (0.5%) for competitive 1,500 m athletes (Hopkins, 2005) and in excess of the time between the 1st and 4th placed athletes (0.6%) and between the 3rd and 8th placed athletes (0.7%) in the men's 1,500 m final at the London 2012 Olympic Games. The medium effect reported here from RP therefore, likely represents a large performance benefit.

The higher intensity interval session within the final 3 d of the HI taper did not result in a further improvement in running performance compared to RP. Although a greater capacity to respond effectively to high intensity training during the taper has been suggested previously (Houmard et al., 1994; Mujika et al., 2004; Shepley et al., 1992), the conservative reduction in training volume resulting from the algorithms may not allow sufficient recovery to respond positively to the increased intensity of this session. The overall volume reduction was ~30%, which represents a comparatively small adjustment compared to other studies where volume is reduced by up to 90% and wholly dedicated to high intensity training (Houmard et al., 1994; Shepley et al., 1992). The algorithm-derived taper was not designed for manipulation of training intensity above race speed, therefore a concomitant decrease in volume might be necessary to enhance performance.

Furthermore, the volume of the final interval session itself was greater than 1,500 m race distance (2.7 ± 0.7 km), due to tapered training being prescribed relative to regular training, which was uncontrolled prior to the study. Whilst this was the case for both conditions, a session of this volume completed faster than race speed may have exacerbated fatigue, with insufficient recovery time before the performance assessment. It is also possible that the lack of control over training load prior to the study may have contributed to the worsening in performance after HI in some individuals. It has been observed previously that deliberate overload/overreaching can result in greater performance super-compensation compared to habitual training, providing that the training stress from overload does not exceed capacity to recover during the taper (Aubry et al., 2014; Le Meur et al., 2013). Only 1 week of control training data was collected for the algorithm taper prescription and this may not have been representative of the extent to which athletes were

undergoing habitual/sub-optimal or overloaded training prior to the study. The addition of a higher intensity interval session may have influenced capacity to recover during the taper in some individuals in HI if they were overreaching beforehand, particularly given the volume and intensity of this session. In the days after the final interval session, participants did not modify their overall physical activity or training in HI compared to RP. This suggests they did not spend more time resting to compensate for the increased training stress compared to RP. It may be that some athletes require longer to reach peak performance after the final interval session in HI, perhaps due to the additional training stress and in light of insufficient recovery. It was not possible however, to explore the amplitude of the performance rebound between the RP and HI strategies, since participants completed one performance assessment after each taper condition.

Differences in the performance changes from each condition (RP vs. HI) cannot be explained by the measured physiological indices. However, there were several main effects of tapering (control vs. taper). Peak blood lactate after the performance trial increased in both conditions after tapering compared to control. This suggests that a greater contribution to energy production from glycolysis occurred after tapering and supports the important role of glycolytic metabolism in middle-distance running performance (Stellingwerff et al., 2011). A consistently reported physiological response to tapering is an increase in muscle glycogen concentration (Neary et al., 1992; Shepley et al., 1992) which may facilitate increased contribution from glycolysis and maximal performance capability (Houmard et al., 1994; Mujika et al., 2004). Although muscle glycogen was not measured in the current study, carbohydrate consumption remained consistent despite a reduction in overall physical activity and a lower proportion of time spent undertaking MVPA during the taper. It is possible therefore, that the participants in the current study might have experienced an increase in muscle glycogen. It has been suggested that during a period of heavy training before tapering, athletes have low muscle glycogen content, which is replenished over a 7-d taper, resulting in a 15% increase in muscle glycogen (Shepley et al., 1992). Furthermore, a 7-d taper coupled with a high carbohydrate diet increased muscle glycogen concentration and was associated with elevated carbohydrate oxidation, RER values and blood lactate concentration during cycling to volitional exhaustion at 80% $\dot{V}O_{2max}$ (Walker et al., 2000). In the present study, there was no change in carbohydrate consumption in the final 3 d of HI compared to RP, despite an increase in intensity of the final interval session. Since glycogen is the main energy source for high intensity exercise

(Hargreaves, 1996; Hermansen et al., 1967; Romjin et al., 1993; Stellingwerff et al., 2011) and there is evidence of muscle glycogen depletion in type II fibres after high intensity intermittent exercise (Gollnick et al., 1973), a more direct intervention to optimise carbohydrate consumption after the intensified interval session in HI might have influenced the performance outcome.

A large difference in performance improvement after a high intensity taper (22%) and a low intensity taper (6%) has been shown previously (Shepley et al., 1992). These large differences in performance were paralleled by modest differences in peak blood lactate (7% and 10% respectively), and suggests that although substrate availability may contribute to improved performance after tapering, the cause is multifactorial. Conversely, athletes should also take care to match energy intake with the reduced energy expenditure consequential of a taper period, to avoid changes in body composition that might be detrimental to performance (Margaritis et al., 2003; Mujika et al. 2004). In the present study, energy intake and body mass was not different between the control and taper periods, although body composition was not measured.

Peak isometric leg extension force and the rate of force development were improved after tapering, supporting previous research (Cavanaugh & Musch, 1989; Costill et al., 1985; Johns et al., 1992; Raglin et al., 1996). Improvements in peak force and absolute power of the single muscle fibre have also been observed alongside improvements in performance after tapering in endurance runners (Luden et al., 2010). These parameters have been shown to respond to the reduced training load during the taper, potentially due to attenuation in function during periods of intensive training rather than an improvement *per se*. The mechanisms suggested for this have included altered protein synthesis and shifts in fibre type, (Neary et al., 2003), particularly after longer tapers (Johns et al., 1992; Trappe et al., 2000). However, improvements in performance after tapering are likely influenced by many physiological factors, rather than muscular force and power in isolation. Similar beneficial changes to maximal voluntary isometric strength were reported in middle distance runners after tapering and after complete rest, however endurance performance capacity declined by 3% after the complete rest condition (Shepley et al., 1992).

Although it has been suggested previously that certain cytokines could represent markers of training stress in athletes (Jürimäe et al., 2011; Robson-Ansley et al., 2007), the inflammatory markers IL-6, sIL-6R and CRP were not sensitive enough to indicate

changes in recovery and readiness to perform across a 7-d period of tapering in middle-distance runners in the present study. Monitoring IL-6 in long distance and marathon runners may be more useful however, as it could be speculated that their heavier training loads (chapter IV) increase the likelihood of undertaking training sessions with low muscle glycogen availability, often through multiple training sessions in a day (Philp et al., 2012). IL-6 may therefore be indicative of muscle glycogen status more so in this group, as its release has been shown to be elevated in glycogen depleted states (Gleeson & Bishop, 2000).

It is not clear why there were also no changes in plasma concentrations of sIL-6R and CRP, as these markers have been shown to be elevated during heavy training (Robson-Ansley et al., 2009). However this could be related to the lack of influence of training and tapering on IL-6. Increases in IL-6 in response to exercise generate acute phase reactions, including the release of CRP. C-reactive protein has been shown to increase concentrations of sIL-6R *in vitro*, by inducing receptor shedding from neutrophils (Chalaris et al., 2007; Jones et al., 1999). The lack of response suggests that these biomarkers are not sensitive to detect the subtle changes associated with performance enhancement of ~1%, which could be the difference between gold and 4th position in major championships.

Similarly, although it has been suggested previously that the tapering period might create a hormonal milieu favourable to anabolic processes (Mujika et al., 2002), the performance difference induced by tapering was not reflected by clear changes in the T:C ratio. This has been found previously in runners (Mujika et al., 2000) and swimmers (Flynn et al., 1994; Mujika et al., 1996), where improvements in performance after tapering were not accompanied by changes in the T:C ratio.

As opposed to blood biomarkers, it might be more useful to monitor changes in muscle function to evaluate the effectiveness of the taper, given the improvements in peak isometric leg extension force and rate of force development evident in the present study, in addition to previous literature (Cavanaugh & Musch, 1989; Costill et al., 1985; Johns et al., 1992; Shepley et al., 1992; Raglin et al., 1996). Whilst it may not be practical to utilise ‘gold standard’ laboratory measurements, an alternative non-invasive, field-based measure such as counter movement jump (CMJ) in conjunction with session-RPE, may indicate readiness to perform in elite endurance athletes. After measuring these indices across the season, significantly higher CMJ scores and significantly lower session-RPE scores were

observed in the week before the season's best performance time compared to the season's worst performance time (Balsalobre-Fernandez et al., 2014). These variables could be measured simply on a regular basis without interfering with an athlete's training programme and may be sensitive enough to indicate athlete readiness to perform.

4.5 Conclusion

A 7-d taper prescribed using an algorithm based on the current practices of elite British middle-distance runners is most likely to improve performance. In contrast, the addition of intensity above race speed in the final interval session of this taper would not be recommended due to unclear effects on performance. The measured physiological indices could not explain the difference in performance, although it is possible that a greater reduction in training volume may be required during this strategy to facilitate a positive response to the increase in intensity and improve subsequent performance. The biomarkers; IL-6, sIL-6R, CRP, and T:C ratio do not appear to objectively indicate the physiological status for performance following a 7-d taper in middle-distance runners, although the monitoring of muscle function may offer an alternative.

Chapter V

Case Study: Training and Tapering in an Elite Female Marathon Runner

5.0 Chapter Summary

The aim of this chapter was to explore the training and tapering strategy of an elite female marathon runner in preparation for a marathon race. In addition, to investigate whether plasma IL-6 and sIL-6R concentrations were indicative of recovery status for performance following a marathon taper. The athlete underwent a 16 week training program (including the taper) followed by 2 weeks of recovery post-race. The main findings were that training load in the specific preparation phase (weeks 7-13) was similar to other elite marathon runners and that the tapering strategy partly reflected the optimal approach suggested from the scientific literature and that of marathon runners in chapter III, although aspects of the taper predicted from the algorithm were somewhat different. Resting plasma IL-6 and sIL-6R concentrations did not appear to be indicative of recovery during tapering for the marathon.

5.1 Introduction

Despite considerable scientific interest in the physiological determinants of marathon running performance, in particular the possibility of a sub 2 h marathon (Joyner et al., 2011), few studies have characterised the training and tapering strategies of elite marathon runners. To the author's knowledge, chapter III was also the first to comprehensively document the tapering strategies implemented by elite endurance runners in events ranging from 800 m to marathon. The primary aim of this case study therefore, was to assess whether the tapering strategy of an elite female marathon runner reflects the findings of chapter III and whether a scientific approach to training is implemented throughout preparation for a marathon race.

It would still be of benefit to identify a minimally invasive biomarker of taper effectiveness and readiness to perform in elite athlete. It has been suggested previously that certain cytokines could represent markers of training stress in athletes (Jürimäe et al., 2011; Robson-Ansley et al., 2007), although resting plasma IL-6 and sIL-6R concentrations were not sensitive enough to indicate changes in readiness to perform across a 7-d period of tapering in middle-distance runners in chapter IV. It is unknown

however, whether monitoring IL-6 and sIL-6R concentrations in marathon runners may be more informative.

Interleukin-6 is produced in the working muscle during exercise (Ostrowski et al., 1998) and a large proportion of the increase can be explained by the duration of exercise (Fischer, 2006). Furthermore, IL-6 release has been shown to be elevated in glycogen depleted states (Gleeson & Bishop, 2000; Steensberg et al., 2001). Taken together, this suggests that marathon runners may produce significant levels of IL-6 in heavy periods of training, due to a large volume of prolonged training close to marathon pace (chapter III), without adequate time for replenishment (Coyle & Coyle, 1993). For example, marathon pace can occur at a high fractional utilisation of $\dot{V}O_{2\max}$ (75-85%) in highly trained athletes (Jones, 2006; Maughan & Leiper, 1983; Sjödín & Svedenhag, 1985) and muscle glycogen becomes the primary source of fuel for intensities exceeding $\sim 75\%$ $\dot{V}O_{2\max}$ (Hermansen et al., 1967; Romjin et al., 1993). The secondary aim was therefore, to determine if plasma IL-6 and sIL-6R concentrations were elevated at rest in an elite marathon runner and whether changes during the taper may be indicative of readiness to perform.

5.2 Methods

An elite female marathon runner (personal best: 2:30:56 h:min:s; height: 1.60 m; body mass: 47.3 kg; age: 35 y), with a training history of over 15 y, undertook a 16 week training program, targeting a competitive marathon in week 16, followed by 2 weeks of complete rest and a further 1 week of recovery training. The athlete trained under the supervision of their personal coach and followed their own individual training programme, which was not influenced by the case study.

5.2.1 Training

All running during the 19 week period was recorded objectively from the participant's own GPS device. Training mesocycles outlined by the personal coach included the general preparation phase (weeks 1-6), specific preparation phase (weeks 7-13) and taper (weeks 14-16). During weeks 5-8 the athlete undertook an altitude sojourn to Iten, Kenya, to train at $\sim 2,400$ m above sea level. In week 11, the athlete competed in an International half marathon race (performance time; 1:14:20 h:min:s).

5.2.2 *Training Analysis*

Training was categorised into continuous or interval running (described previously: chapter 3.2.1) and quantified for mean weekly volume (km), frequency and intensity (% personal best marathon race speed and in relation to personal best 10 km speed). The session that elicited the highest average speed during the taper was defined as the peak intensity interval training session. The volume of this session was also calculated, in addition to the number of days from the marathon race. Mean weekly volume, frequency and intensity for both continuous and interval running were entered into the algorithm (chapter III) for comparison.

5.2.3 *Blood Sampling and Analysis*

The athlete reported to the laboratory between 0730 h and 0830 h following a 10 h overnight fast on days; 1, 5, 10, 15, and 17 of the 3 week taper and 22 d after the marathon. After being seated in a semi-supine position, a resting venous blood sample was collected by venepuncture (described previously: chapter 2.2.4). Plasma IL-6 and sIL-6R concentrations were determined using commercially available ELISA kits (R&D Systems, Inc., Mineappolis, USA, supplied by Bio-Techne, Abingdon, UK) with the aid of a plate reader (Optica, Mikura, West Sussex, UK).

5.3 *Results*

Performance time was 2:31:46 h:min:s in the 4th marathon of the athlete's career, 50 s slower than personal best performance time.

Training data in the 16 week programme prior to the marathon were based on 180 sessions, at a frequency of 11 ± 1 sessions per week. Highest weekly training volume was 182 km in the final week of the specific preparation phase, compared to 121 km in race week which included the marathon itself. Average weekly volume, frequency and intensity of both continuous and interval running during each mesocycle are shown in Table 5.1, in addition to a predicted taper using the algorithm (chapter III). Further non-running training such as strength training, drills and stability work was completed twice per week, but not included in the analysis. In the recovery training week after 2 weeks of complete rest, the athlete completed a total of 52 km of continuous running during 5 training sessions, at an intensity of $82 \pm 2\%$ of race speed.

Table 5.1. Training load during each mesocycle in the 16 week marathon programme (not including the marathon race) and prediction of the taper using the algorithm (chapter III).

Training Load Variables	General	Specific	%Δ from Specific Preparation			
	Preparation (weeks 1-6)	Preparation (weeks 7-13)	Taper Week 14	Taper Week 15	Taper Week 16	Algorithm (13 d taper)
Training volume						
Continuous running (km·wk ⁻¹)	144 ± 1	164 ± 12	90	74	46	54
Interval running (km·wk ⁻¹)	9 ± 5	4 ± 3	97	58	60	74
Training frequency						
Continuous running (run·wk ⁻¹)	11 ± 1	10 ± 0	100	90	90	61
Interval running (run·wk ⁻¹)	1 ± 1	1 ± 0	100	100	100	100
Training intensity						
Continuous running (% race speed)	84 ± 1	86 ± 3	102	101	100	96
Interval running (% race speed)	113 ± 3	115 ± 4	99	103	99	98

The peak intensity training session of the taper was completed 11 d before the marathon race at an average intensity of 115% race speed. The first section of the session was 6.4 km at 111% race speed, followed by 2 km of high intensity intervals at 119% race speed.

Plasma IL-6 was $0.20 \pm 0.07 \text{ pg}\cdot\text{ml}^{-1}$ during the taper (range: $0.12\text{-}0.29 \text{ pg}\cdot\text{ml}^{-1}$) vs. $0.43 \text{ pg}\cdot\text{ml}^{-1}$ after 2 weeks of complete rest and 1 week of recovery training. Plasma sIL-6R was $33.9 \pm 2.9 \text{ pg}\cdot\text{ml}^{-1}$ during the taper (range: $30.8\text{-}37.3 \text{ pg}\cdot\text{ml}^{-1}$) vs. $30.3 \text{ pg}\cdot\text{ml}^{-1}$ after 2 weeks of complete rest and one week of recovery training (Figure 5.1).

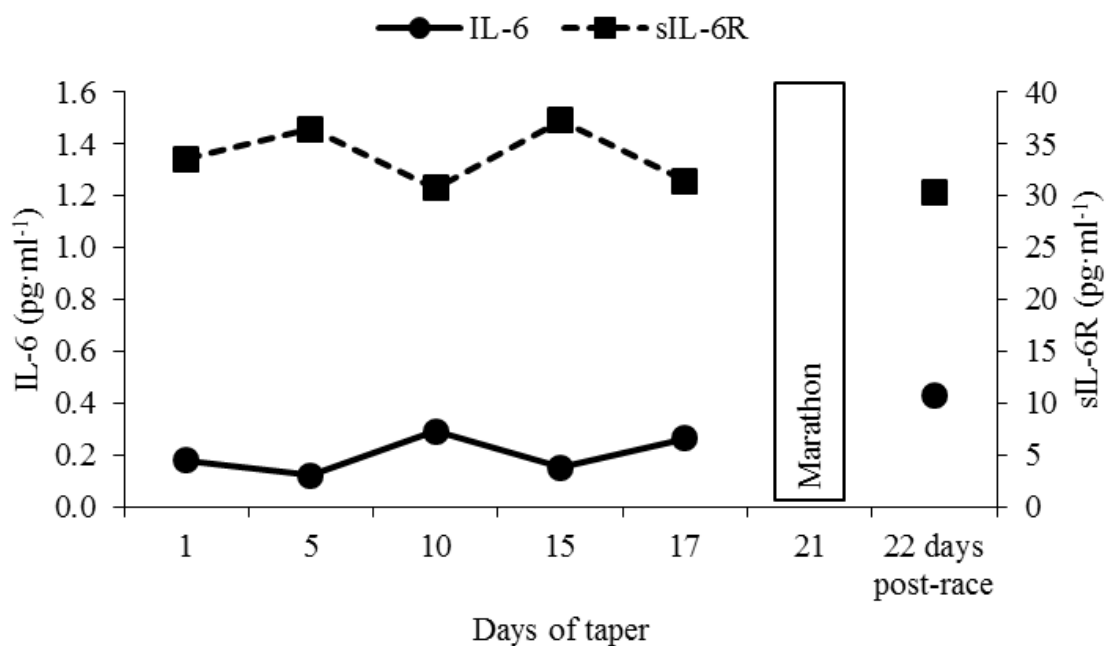


Figure 5.1. Plasma IL-6 (filled circles) and sIL-6R (open squares) concentration during the pre-marathon taper phase and 22 d post-race.

5.4 Discussion

The purpose of this case study was to characterise the training and tapering strategy of an elite female marathon runner preparing for a marathon race and to explore the responses of plasma IL-6 and sIL-6R concentration to tapering and increased recovery. The main findings were that training load in the specific preparation phase was similar to other elite marathon runners and that the tapering strategy partly reflected the optimal approach suggested from the scientific literature and that of marathon runners in chapter III, although aspects of the algorithm taper were somewhat different. In addition, resting plasma IL-6 and sIL-6R concentrations did not appear to be indicative of recovery during tapering for the marathon.

5.4.1 Training

Training volume during the specific preparation phase (mean: $\sim 168 \text{ km}\cdot\text{wk}^{-1}$) was comparable to that of elite French/Portuguese female marathon runners (Billat et al., 2001) and to that reported by other elite British marathon runners (chapter III). Elite female American marathon runners also reported a similar peak training volume in their build up to the U.S. Olympic Trials ($180 \text{ km}\cdot\text{wk}^{-1}$ vs. $182 \text{ km}\cdot\text{wk}^{-1}$) (Karp, 2007). Training frequency of 10-12 sessions per week appears to be typical in elite female marathon runners completing such volumes (Billat et al., 2001; Karp, 2007; chapter III). Despite high training volumes however, elite marathon runners complete a relatively small proportion of training at specific marathon pace and most of their training volume at a slower pace. In this case, the mean percentage of training at marathon pace was 8%, with a very large proportion run at a slower pace (83%) and small proportions run at tempo pace (between marathon pace and 10 km pace, i.e. between lactate threshold and lactate turnpoint; Jones, 2006; Smith and Jones, 2001) (6%) and as high intensity intervals (faster than 10 km pace, i.e. $>$ lactate turnpoint; Jones, 2006) (3%). A similar intensity distribution is described as a ‘polarised’ pattern (Seiler & Kjerland, 2006) and has been observed previously in elite marathon runners (Billat et al., 2001; Karp, 2007; Stellingwerff, 2012) and other Olympic-standard endurance athletes (Tønnesson et al., 2014).

5.4.2 Taper

It has been suggested that the most effective tapering strategy to improve performance in competitive athletes consists of a reduction in training volume of 41-60%, whilst training frequency and intensity are maintained over a duration of ~ 2 weeks (Bosquet et al., 2007). The duration of the taper period in this case was ~ 3 weeks (20 d), 6 d longer than the median reported by other elite British marathon runners (14 d) and 7 d longer than the algorithm prediction. Since peak training volume occurred in the final week of the specific preparation phase ($182 \text{ km}\cdot\text{wk}^{-1}$) and was greater than the median reported prior to the taper in other British marathon runners, a longer taper might have been necessary to alleviate fatigue from previous overload and improve performance (Thomas & Busso, 2005; Thomas et al., 2008). However, the first week of the taper for the current athlete involved only a 10% reduction in continuous volume and 3% reduction in interval volume and may not have contributed a great deal to recovery therefore. Nevertheless, positive physiological adaptations and/or performance improvements have been reported as a result

of tapering strategies lasting from 6 d to 4 weeks (Houmard et al., 1990; McConnell et al., 1993; Mujika et al., 2002). Compared to the specific preparation phase, continuous running volume was progressively reduced during the taper, by 10% in the first week and by 26% and 54% in the second and third weeks of the taper, not including the marathon race itself. The average continuous volume reduction predicted from the algorithm taper was 46% over 13 d, compared to a 40% reduction over the last 13 d in the current case. Interval volume remained similar in the first week of the taper and was reduced by ~40% in week 2 and 3, more so than the algorithm predicted. Whilst there has been limited research exploring the optimal pattern to reduce training load, a systems model predicted that an exponential fast-decay pattern was the most effective at improving performance (Banister et al., 1999). In contrast to the current case, this pattern is characterised by a sharp decrease in training load early in the taper, followed by a levelling off closer to competition. A limitation to the algorithm is that it does not indicate the pattern to which training should be altered over the taper duration. Interval training frequency remained consistent throughout the taper, whilst continuous running frequency was reduced by one session per week (~10%) in taper weeks 2 and 3. Despite the general consensus that frequency should be maintained during the taper (Bosquet et al., 2007), a 10% reduction in this case is unlikely to have had a negative impact on performance (Mujika & Padilla, 2003). The algorithm suggested a much larger continuous frequency reduction of 39% however, which would equate to 4 fewer runs per week. There was no substantial change in continuous or interval training intensity during the taper compared to the specific preparation phase, in agreement with the suggested scientific approach (Bosquet et al., 2007) and with other elite marathon runners (Stellingwerff, 2012). It is unknown whether this was a deliberate practice by the athlete and coach or coincidental. A slight reduction in continuous (4%) and interval (2%) were predicted from the algorithm. The peak intensity training session of the taper was completed 11 d from the marathon race at an average intensity of 115% race speed, similar to other elite British marathon runners (chapter III).

5.4.3 Inflammatory Markers

It was expected that plasma IL-6 concentration would be highest on day 1 of the taper and exhibit a gradual reduction throughout the taper, particularly after peak training volume occurred in the final week of the specific preparation phase. The rationale was based on the finding that resting plasma IL-6 concentration was elevated in response to an acute period of intensified training in triathletes, which did not return to baseline after 1 week of

their regular training load (Robson-Ansley, 2007). After a longer period of intensified training in elite cyclists (8 weeks), the IL-6 response to a 40 km time trial was 113% greater than before the training period (Farhangimaleki et al., 2009). A 3 week tapering period where training volume was reduced by 50% was necessary to reverse the elevated IL-6 response. It is possible that the accumulation of training during intensified periods might lead to muscle glycogen depletion, a potent stimulus for IL-6 release (Steensberg, 2000). This could also be exacerbated in marathon runners, who may consciously opt to train with low carbohydrate availability (Burke, 2010) or simply due to multiple training sessions in a day (Philp et al., 2012). In the current case however, resting plasma IL-6 concentration remained similar throughout the taper and after 3 weeks of recovery. This supports the findings of Halson et al. (2003), who observed no change in resting plasma IL-6 concentration in response to periods of intensified and reduced cycling training. In addition, plasma IL-6 concentration was transiently elevated post-exercise during a 6-d endurance cycling event, but had returned to pre-exercise concentrations each following morning (Robson-Ansley et al., 2009). Although muscle glycogen and dietary intake were not measured in this case, it could be speculated that the athlete was not muscle glycogen depleted prior to the taper and therefore resting plasma IL-6 concentration was not chronically elevated. This could in part, be due to the large proportion of training volume in the specific phase (83%) that was completed slower than marathon pace and therefore muscle glycogen is unlikely to be the primary source of fuel at this intensity. In addition, strategies to train the gut to absorb more carbohydrate for exogenous oxidation (Jeukendrup, 2011) are common among marathon runners in the specific preparation phase (Stellingwerff, 2012) and may reduce reliance on muscle glycogen stores. Furthermore, trained athletes may exhibit an attenuated inflammatory response to exercise, compared to non-athletes (Gokale et al., 2007).

Whilst elevated plasma IL-6 concentrations have been shown to be detrimental to athletic performance and heighten feelings of fatigue (Robson-Ansley et al., 2004), concentrations as low as $\sim 5 \text{ pg}\cdot\text{ml}^{-1}$ have been associated with an increased perception of effort (Robson-Ansley et al., 2007). The measured concentrations in the current case are considerably lower than this ($0.20 \text{ pg}\cdot\text{ml}^{-1}$ vs. $5 \text{ pg}\cdot\text{ml}^{-1}$) and therefore unlikely to have negatively impacted performance. Elevated sIL-6R however, can increase sensitivity to IL-6 and concentrations were found to be persistently elevated at rest ($\sim 75 \text{ pg}\cdot\text{ml}^{-1}$ vs. $\sim 50 \text{ pg}\cdot\text{ml}^{-1}$) and associated with feelings of fatigue in response to a 6-d endurance cycling event

(Robson-Ansley et al., 2009). In this case however, plasma sIL-6R was subtly different (~ 2 pg \cdot ml $^{-1}$) from day 1 of the taper compared to day 17 of the taper.

These findings confirm those of chapter IV and suggest that resting plasma IL-6 and sIL-6R are not useful markers of recovery and readiness to perform during a 3 week taper in an elite marathon runner.

Chapter VI

Influence of a Novel 7- Day Tapering Strategy on 1,500 m Track Performance and Pacing in Middle-Distance Runners

6.0 Chapter Summary

Chapter IV suggested that a greater reduction in training volume might be required when intensity of the final interval session is increased, to facilitate a positive response and improve subsequent performance. This chapter examined the influence of a 60% reduction in continuous running volume and an increase in intensity in the final interval session within a taper on 1,500 m track running performance and pacing strategy. Eight well-trained middle-distance runners completed 2 conditions, each involving a 7-d period of tapering. In 1 condition, the taper was prescribed using the algorithm developed in chapter III, where continuous running volume was reduced by 28% and interval intensity was equal to 1,500 m race pace (RP). In the other condition, continuous running volume was reduced by 60% and intensity of the final interval training session was prescribed at 115% of 1,500 m race pace (HI). Conditions were separated by 3 weeks of regular training and 1,500 m performance assessments were completed on an indoor 200 m track 1 day before and after each taper condition. Qualitative inference suggested that the tapering strategy in HI was *very likely* to be beneficial to 1,500 m time (259.0 ± 6.3 s vs. 253.8 ± 7.7 s) compared to *likely* beneficial in the RP condition (258.9 ± 7.4 s vs. 255.7 ± 8.7 s). After both RP and HI tapers, the first and second 300 m segments of the 1,500 m time trial were faster than pre-taper (both $P < 0.05$). It was recommended that pacing after a taper should be practiced to avoid an over-fast start.

6.1 Introduction

The inclusion of intense training during the taper is key to enhancing performance (Mujika, 2010), however information regarding optimal practice in the final days of the taper is lacking (Tønnesson et al., 2014). In chapter III it was observed that long-distance runners train at intensities above race speed within the final days of the taper period before competition, which was not noted in middle-distance runners. This led to an investigation on the influence of an increase in intensity (115% of race speed) in the final interval session of a taper on 1,500 m treadmill time trial performance in chapter IV. However, the effects on performance from this strategy were unclear and some athletes may experience a worsening in performance. In contrast, a strategy that was prescribed from the algorithms

based on the practices of elite middle-distance runners lowered performance time by 3.4% on a self-paced treadmill. Since the average training volume reduction in the high intensity strategy (~30%) was less than the recommendations from the literature (Bosquet et al., 2007), it is possible that this was insufficient to overcome prior fatigue and respond positively to the more intense final interval session. In addition, the total volume of this session was greater than 1,500 m race distance itself and may have exacerbated fatigue. As a result, the potential augmented responses to the higher intensity session may have been prevented. There is a need therefore, to reassess this strategy with a further reduction in continuous volume and a lower final interval session volume.

Whilst the taper is crucial to ensure athletes are ready to produce a peak performance, they must also be able to deliver this performance through optimal pace selection. It is thought that complex information processing between the brain and periphery regulates pacing, to avoid catastrophic failure in any physiological system (Lambert et al., 2005; St Clair Gibson et al., 2006; Ulmer, 1996). It is unknown however, whether the initial pace set in a feedforward manner might be interfered with by the effects of tapering on recovery from prior training overload. It is possible therefore, that the change in fatigue status after tapering might need to be taken into account to achieve an optimal pacing strategy.

It appears that no study has investigated how tapering influences track performance over 1,500 m, with chapter IV and the study by Shepley et al. (1992) previously based on laboratory treadmill performance. The aims of the current study are therefore: i) to investigate the effectiveness of an algorithm-derived tapering strategy on 1,500 m time trial track performance; ii) to establish whether the addition of higher intensity interval session (115% of race speed) and a greater overall reduction in continuous volume (60%), can further enhance 1,500 time trial track performance. Lastly: iii) to explore whether tapering influences the selected pacing strategy for 1,500 m performance. It is hypothesised that, the novel 7-d tapering strategy will result in a greater improvement in 1,500 m track performance than the algorithm-derived strategy alone.

6.2 Methods

6.2.1 Participants

A group of 8 highly-trained male middle distance runners: (mean \pm SD) age 21.4 ± 4.2 y, height 182.8 ± 7.2 cm, body mass 67.4 ± 8.0 kg, volunteered to take part in the experimental trials. Inclusion criteria specified that participants were competitive middle distance runners (800 m & 1,500 m) who had a training history of at least 2 years and had trained consistently without interruption for the previous 2 months. Mean personal best 1,500 m time was 241.4 ± 9.2 s (range: 227.6 s – 251.7 s).

6.2.2 Experimental Design

The study employed a repeated-measures cross-over design. Each condition involved a 7-d period of tapering, with a 1,500 m time trial performance assessment on the day before (pre-taper, day 0) and the day after (post-taper, day 8) (Figure 6.1). Conditions were separated by at least 3 weeks of regular training. Participants were not informed about the precise differences between the 2 conditions, but could not be blinded to the manipulation of training load.

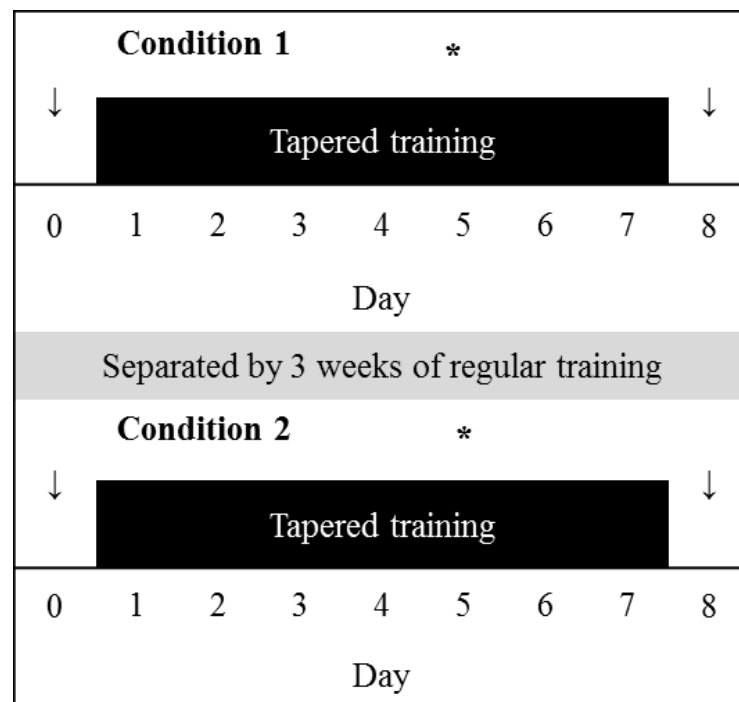


Figure 6.1. Experimental design illustrated by 2 experimental conditions, separated by 3 weeks of regular training. Arrows represent 1,500 m time trial performance assessments and asterisk indicates final interval session of the taper on day 5.

6.2.3 Training and Taper

The investigation began in the 16th week of the winter training season. Participants trained under the supervision of their personal coaches and followed their own individual training programmes, which were not manipulated prior to the experimental conditions. Training in the 4 weeks prior to the first condition was recorded objectively from the participant's own GPS device. Training was categorised into continuous or interval running (described previously: chapter 3.2.1) and quantified for mean weekly volume (km), frequency and intensity (% personal best 1,500 m race speed). During the taper period in the race-pace condition (RP), participants completed individualised training relative to the mean of the 4 weeks preceding the experimental conditions, which was prescribed using the algorithms from chapter III. Although prior training load was uncontrolled (as in chapter IV), the mean of the 4 weeks preceding was used in this instance to ensure the taper was more accurately prescribed. In RP, the speed of the final interval training session was equivalent to the average speed of personal best 1,500 m time (described below) and continuous running volume was reduced by 28%. In the high intensity condition (HI), continuous running volume was reduced by 60% and the intensity of the final interval session of the taper was prescribed at 115% of the speed in RP. Training load was confirmed throughout both conditions using GPS data.

6.2.4 Final Interval Session within Taper Period

In each condition, a standardised interval running session was completed on day 5, 3 d prior to the final performance assessment. Both were carried out on an outdoor 400 m track, at the same time of day. Participants were instructed to perform the same warm up procedure before each session, consisting of a 10 min self-paced jog, 10 min of mobility drills and 5-6 progressive 80 m stride outs. Interval volume during the taper was distributed so that the final interval session in each condition involved 5 repetitions of 300 m with 90 s recovery. Intensity of each repetition was instructed to be equivalent to personal best 1,500 m race speed in the RP condition and 115% of personal best 1,500 m race speed in the HI condition. A cool down of 15 min self-paced jogging was performed after completion of the session.

6.2.5 Performance Assessment

Participants completed 2 1,500 m time trial runs in each condition, before (pre-taper) and after (post-taper) the 7-d tapering period. All performance assessments were carried out on

an indoor 200 m track at the same time of day to control for consistency between conditions. Dietary intake in the 24 h before each performance assessment was repeated and caffeine consumption was prohibited during this period. Participants were allocated to 1 of 3 separate heats based on performance standard, to avoid tactical competition among athletes with similar personal best times. This also ensured that participants were able to run on the inner lane of the track. Participants were instructed to run the time trial as an all-out effort and to avoid drafting strategies or tactics. Overall performance time and split times at 100 m intervals were recorded using a radio frequency identification timing system (ULTRA 4, RFID Race Timing Systems, West Midlands, UK), whereby participants were required to wear a small transponder on their left ankle. No feedback was provided on split times or overall performance times until both experimental conditions had been completed. Mean running speed was calculated for each 300 m segment of the time trials using the 100 m split times.

6.2.6 *Statistical Analysis*

Data were analysed using SPSS (Statistical Package for Social Sciences Inc. v22.0; Chicago, IL, USA) and were initially tested for distribution using the Shapiro-Wilk test. Data are presented as mean \pm SD, unless specified otherwise. Statistical significance was accepted at $P \leq 0.05$.

Non-parametric tests were used where the data were not normally distributed, specifically training frequency data. All other data were confirmed to be normally distributed. Training frequency data from the pre-experimental period, RP and HI were compared using the Mann-Whitney U test. All other training load data, including 300 m repetition session data were compared using paired samples t-tests. Pre-taper time trial performance data were compared using paired samples t-tests, to confirm no difference in baseline performance in RP and HI conditions. Time trial data from the performance assessments were analysed via a 2-way repeated-measures ANOVA, with taper (no taper *vs.* taper) and condition (RP *vs.* HI) as within-subject factors and Bonferroni post hoc analysis. Magnitude-based inferences about the true (population) effect of the RP taper and HI taper on 1,500 m running performance were calculated. The uncertainty in the effect was expressed as 90% confidence limits and as the likelihood that the true value of the effect represents substantial change; harm or benefit (Batterham & Hopkins, 2006). The smallest meaningful change in 1,500 m performance was assumed to be a reduction or increase in

running time of 0.5% (Hopkins, 2005). Effect size (ES) was calculated and the magnitude was considered either trivial (0–0.19), small (0.20–0.49), medium (0.50–0.79) or large (≥ 0.80) (Cohen, 1992). Mean running speed was compared in the corresponding 300 m segment of pre- and post-taper time trials for both RP and HI conditions using the Wilcoxon Signed Rank test.

6.3 Results

6.3.1 Training

Confirmation of training completed during the pre-taper period and in both conditions is presented in Table 6.1. Mean 300 m repetition time in the final interval session was faster in the HI condition compared to the RP condition (43.8 ± 2.0 s vs. 48.0 ± 2.1 s, respectively; $P < 0.05$), but slower in HI than prescribed (43.8 ± 2.0 s vs. 41.0 ± 1.5 s; $P < 0.05$).

6.3.2 1,500 m Performance

Pre-taper 1,500 m performance times were not different in RP and HI conditions (258.9 ± 7.4 s vs. 259.0 ± 6.3 s; $P = 1.00$). There was a main effect of taper (no taper vs. 7-d tapered training) on 1,500 m time trial performance ($P < 0.05$). There was no main effect of condition (RP vs. HI, $P = 0.39$) or an interaction effect ($P = 0.40$), suggesting that time trial performance responded similarly to the both types of taper in RP and HI (RP: 255.7 ± 8.7 s vs. 258.9 ± 7.4 s; HI: 253.8 ± 7.7 s vs. 259.0 ± 6.3 s; Figure 6.2). When considered relative to the smallest worthwhile change in performance, qualitative inference suggested that the tapering strategy in HI condition was *very likely* to be beneficial to 1,500 m time, compared to *likely* beneficial in the RP condition (Table 6.2).

Table 6.1. Weekly volume, frequency and intensity of training in the pre-taper and taper periods (mean \pm SD; $n = 8$). Warm up and cool down data is not shown.

Training Variables	Pre-taper	RP Taper	% Δ	HI Taper	% Δ
Training volume					
Continuous running (km \cdot wk ⁻¹)	54 \pm 14	37 \pm 8*	-28%	21 \pm 5*†	-60%
Interval running (km \cdot wk ⁻¹)	13 \pm 4	8 \pm 2*	-41%	8 \pm 2*	-41%
Training frequency					
Continuous running (run \cdot wk ⁻¹)	5 \pm 1	4 \pm 0*	-23%	4 \pm 0*	-23%
Interval running (run \cdot wk ⁻¹)	2 \pm 0	2 \pm 0	0%	2 \pm 0	0%
Training intensity					
Continuous running (% race speed)	65 \pm 4	62 \pm 5*	-5%	62 \pm 4*	-5%
Interval running (% race speed)	95 \pm 3	99 \pm 2*	5%	100 \pm 2*	5%
Final interval session (% race speed)	-	101 \pm 1	-	110 \pm 3†	-

* Different to pre-taper, † different to RP taper, all $P < 0.05$.

Table 6.2. Differences in pre- and post-taper 1,500 m time trial performance improvements in the RP and HI conditions.

Taper	Mean improvement (s) and 90% confidence limits	Qualitative inference ^a	Effect size
RP	3.2; \pm 3.8	Likely beneficial	0.40
HI	5.2; \pm 3.7	Very likely beneficial	0.74

^a with reference to a smallest worthwhile change of 0.5%.

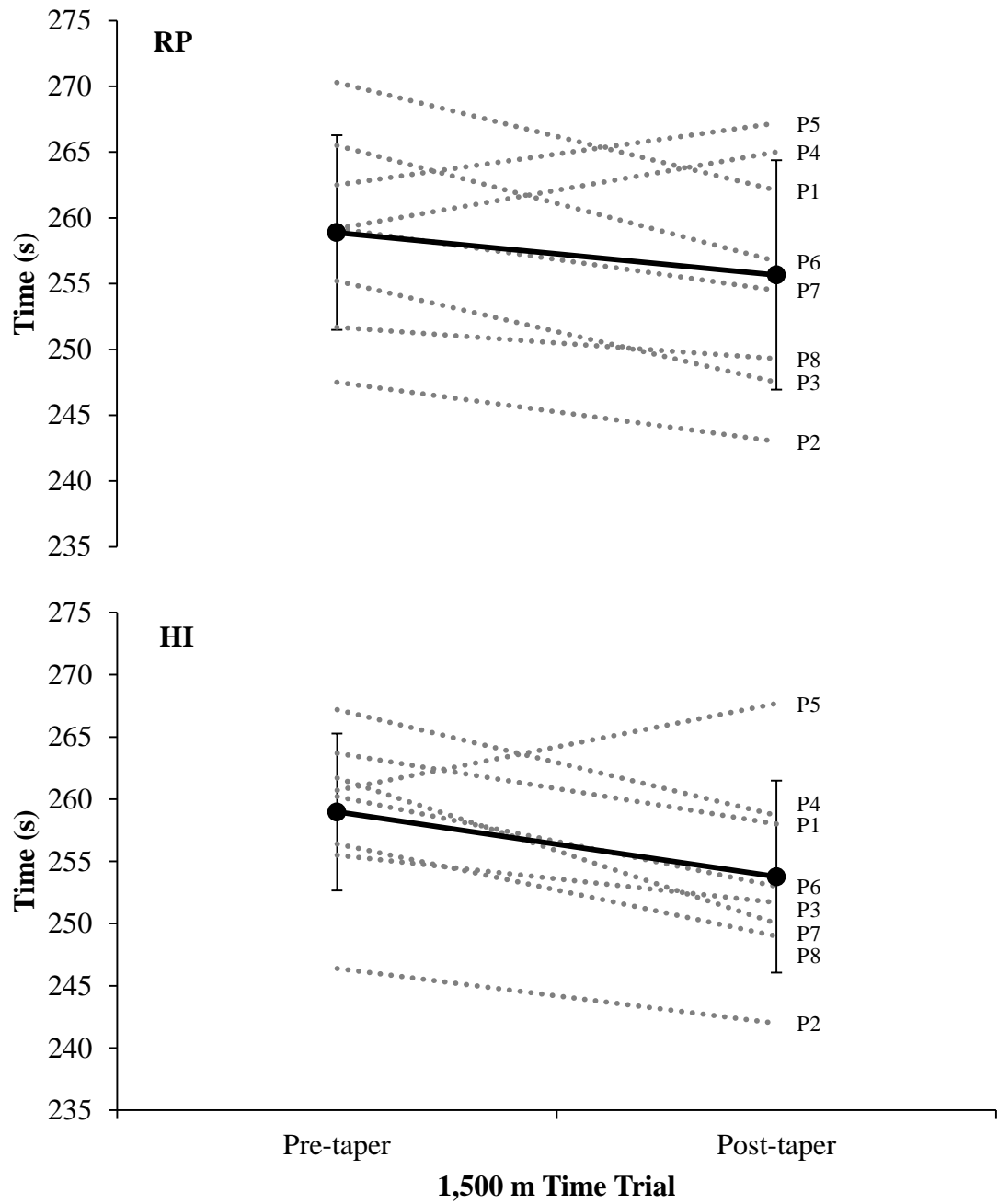


Figure 6.2. 1,500 m time trial performance time pre-taper and post-taper in the RP condition and the HI condition. Individual times for each participant (P1-8) are represented by dashed lines and group mean \pm SD by solid line.

6.3.3 Pacing

Split times indicated that participants ran faster in the first (0-300 m) and second (300-600 m) segments of the post-taper time trial compared to the pre-taper time trial in both the RP and HI conditions (RP: ES = 1.14 and 1.56; HI: ES = 0.97 and 2.01; all $P < 0.05$; Figure 6.3).

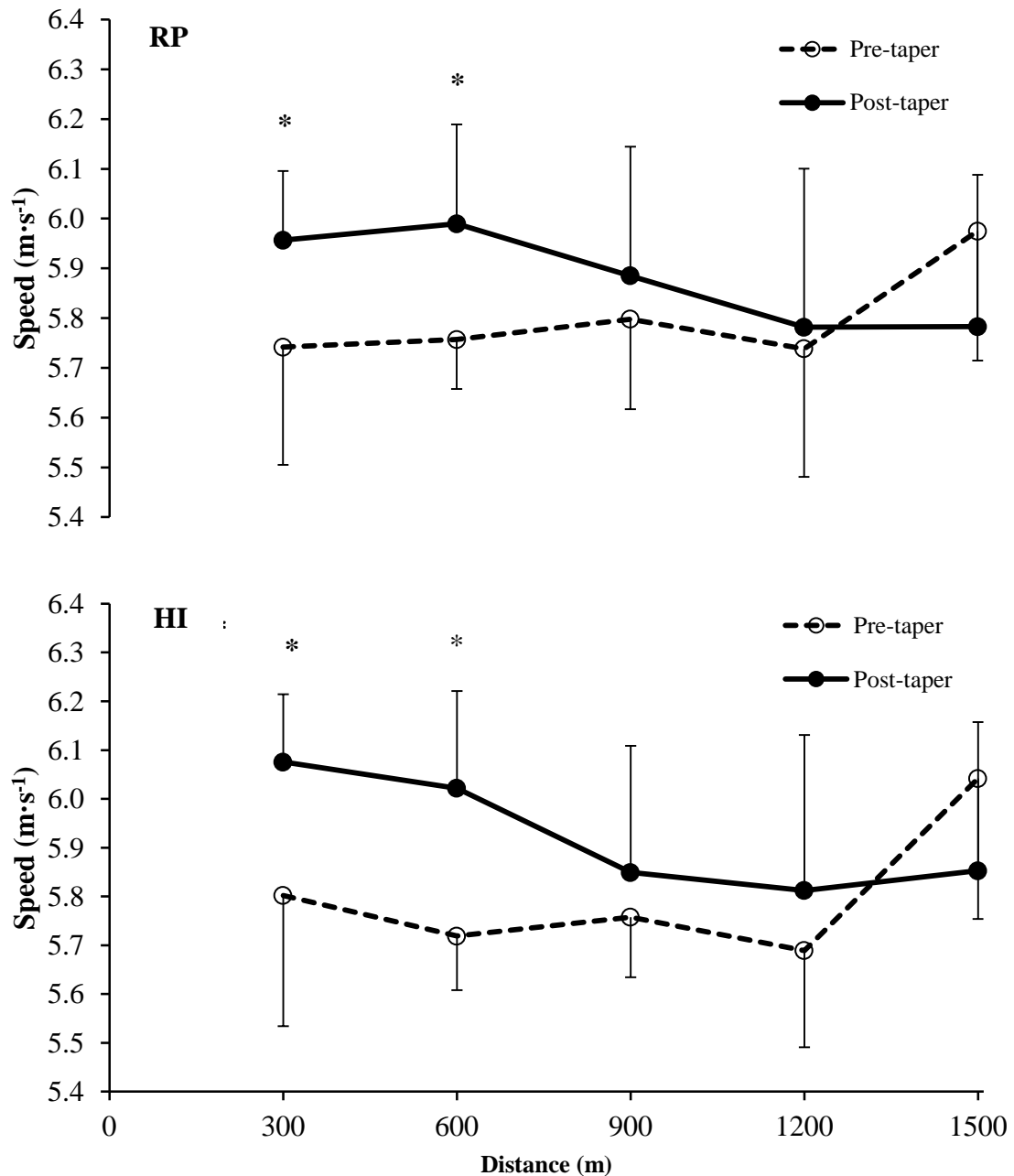


Figure 6.3. Mean speed for each 300 m segment of the 1,500 m time trial in RP and HI. Pre-taper time trial data is represented by the dashed line and post-taper time trial data by the solid line. * Different to corresponding pre-taper 300 m segment ($P \leq 0.05$).

6.3.4 Individual Responses

Six of 8 participants improved performance after tapering in the RP condition (range 2.4-8.8 s) and 2 participants showed a decreased performance (4.7 s and 5.8 s). Seven of 8 participants improved performance after tapering in the HI condition (range 3.8-11.7 s) and 1 participant showed a decreased performance (7.0 s). The same participant showed a decline in performance in both conditions. Mean 300 m segment speeds are shown for this individual in Figure 6.4.

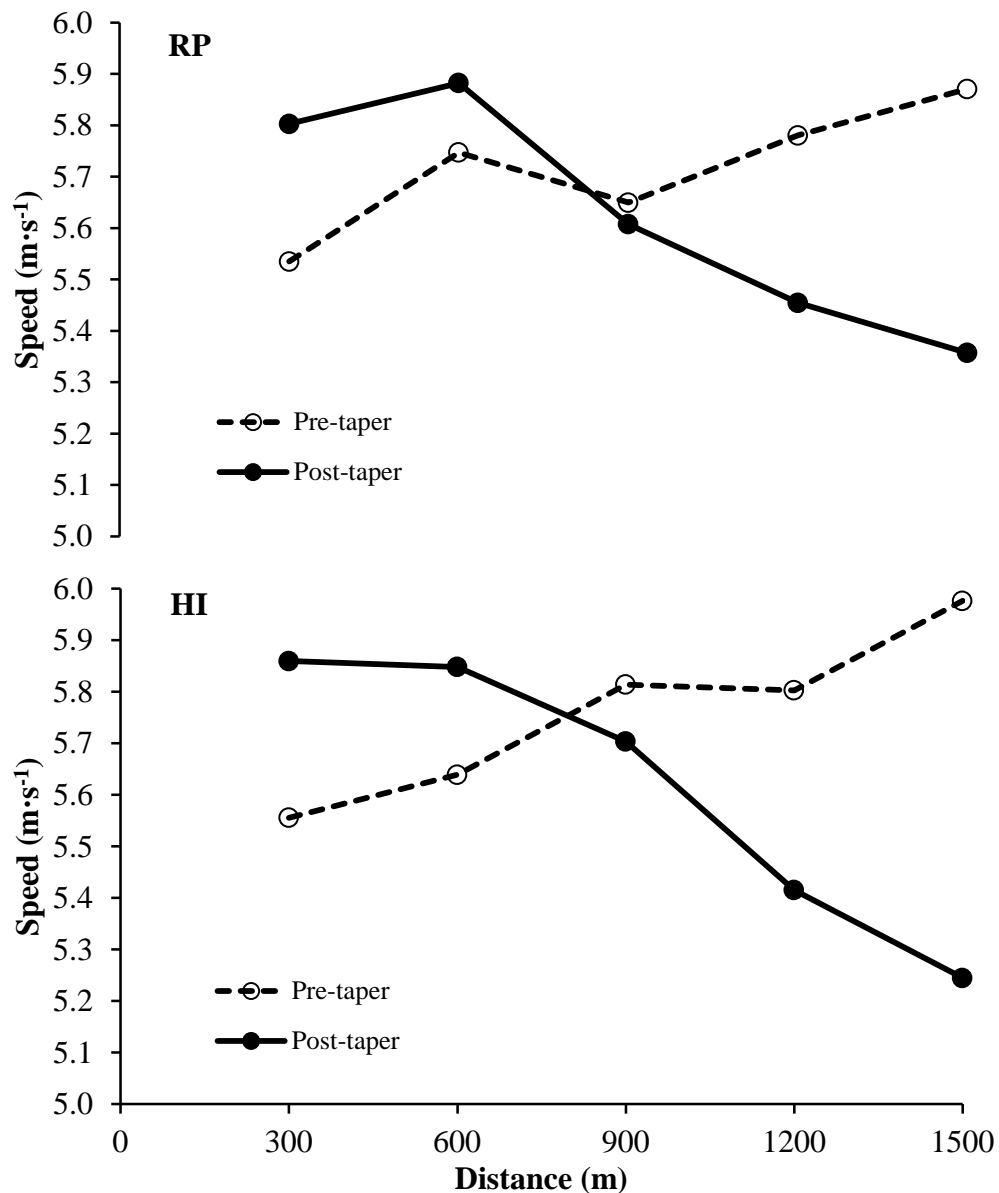


Figure 6.4. Mean running speed for each 300 m segment of the 1,500 m time trial in RP and HI for the individual participant with a decline in performance times post-taper. The pre-taper time trial is represented by the dashed line/open circles and post-taper time trial by the solid line/filled circles.

6.4 Discussion

The main finding of the current study was that 1,500 m track performance improved after tapering. The improvement in performance from both RP (1.3%; small effect) and HI (2.0%; medium effect) tapers, albeit non-significant, fall within the expected range of 0.5 to 6% (Mujika & Padilla, 2003) and are in excess of the smallest worthwhile change in performance (0.5%) for competitive 1,500 m athletes (Hopkins, 2005).

6.4.1 1,500 m Performance

Despite no interaction effect, magnitude-based inferences (Batterham & Hopkins, 2006) suggested that the HI taper was ‘very likely beneficial’ to 1,500 m time trial performance compared to ‘likely beneficial’ for the RP taper. In theory, this could be due to a higher intensity interval session completed late in the taper when the athlete is more fully recovered, allowing a greater capacity to respond positively (Busso, 2003; Mujika et al., 2004) and thus lead to a further improvement in subsequent performance. Previously, theoretical models have shown that a moderate increase in training at the end of a taper may further improve performance as the athlete can capitalise on additional adaptations, after initially overcoming accumulated fatigue from previous training (Thomas et al., 2009). These data would indirectly support this, although likely an effect of insufficient statistical power to detect such marginal differences in performance between RP and HI. Chapter IV did not demonstrate enhanced performance after HI however, perhaps due to the conservative reduction in training volume prescribed via the algorithm, derived from athletes who did not implement this higher intensity session (chapter III). In the present study, continuous running volume was reduced by 60% in HI, as recommended by a meta-analysis on the available literature (Bosquet et al., 2007), which might have increased the likelihood of improved performance and a larger effect, although not to a level of statistical significance.

The intensity of the final interval session in HI might further explain an increased likelihood of improved performance in the present study. In the present study, the session was completed on an outdoor 400 m running track and although participants were instructed to run at a pace equal to 115% of race-speed, training data revealed that mean intensity of the session was in fact 110% in the HI condition. Although faster than RP, this was significantly slower than prescribed. In comparison, the final interval session in chapter IV was carried out on a treadmill and speed was therefore fixed at 115% of race-

speed for each individual in the HI condition. Since efforts at > 100% of race speed in middle-distance events equate to considerably higher absolute speeds than the same percentages of long-distance event race speeds, it is possible that 115% of race-speed was too intense for this event group, particularly so close to ‘competition’. Participants may have been unaccustomed to running these speeds at this time of year and may have held back through fear of injury. Elite marathon runners are able to complete their peak intensity interval session during the taper at ~114% of race speed, but this is a slower absolute speed than for 1,500 m runners and typically this session occurs 10 d from competition (chapter III). Considering the practices of marathon runners and the recommendations from the literature that the optimal taper duration is approximately 2 weeks (Bosquet et al., 2007), it is possible that the 1 week taper was not sufficient to overcome fatigue from previous training. Therefore, athletes may have self-selected to run slightly slower than instructed on the track as a result of afferent feedback from the periphery, to avoid premature fatigue (St Clair Gibson et al., 2006). This may have provided protection from potentially exacerbated fatigue in HI, whilst still allowing a positive response to the session and enhanced subsequent time trial performance compared to RP.

6.4.2 Pacing

Participants completed the first 2 300 m segments of the time trial quicker after tapering in both RP and HI, followed by a similar pace in the remaining segments compared to pre-taper. In closed-loop events, such as 1,500 m, where the aim is to complete a fixed distance in the shortest possible time, athletes must regulate their rate of work output to optimise overall race performance and prevent catastrophic changes to physiological homeostasis (Tucker & Noakes, 2009). The faster start in the present study suggests that the brain-pacing algorithm (St Clair Gibson et al., 2006, Ulmer et al., 1996) was able to detect and take into account the differing recovery status after tapering and perhaps athletes’ previous experience of racing after tapering. After the faster first 2 300 m segments, pace was adjusted similar to pre-taper time trials in the remaining 3 segments to potentially avoid premature physiological catastrophe (Noakes et al., 2005; St Clair Gibson et al., 2006). A positive pacing strategy such as this, is typically selected in events lasting < 4 min, whilst pace becomes more evenly distributed in longer events (Tucker & Noakes, 2009). In support, 1,500 m performance might be enhanced using a fast-start strategy due to a speeding of $\dot{V}O_2$ kinetics (Bailey et al. 2011). However, it may also be harmful to

performance if not judged correctly (Hanon et al. 2007) and such interventions may require practice prior to important competition. In support, Mauger et al. (2009) found that cyclists were able to adopt a successful pacing strategy once prior experience of the 4 km time trial distance was gained, even with no distance or time feedback.

This could explain the response of 2 individuals whose performance declined after tapering in the RP condition, 1 of whom also experienced a decline after tapering in HI. This may have contributed to the differing qualitative inferences based on the mean performance improvements and 90% confidence limits. In both pre-taper time trials, the individual with worsened performance in both conditions executed a negative pacing strategy, shown by a slower start and an increase in speed in the final 300 m segments, but opted for a positive pacing strategy in the post-taper time trials, which resulted in slower overall times. It had been anticipated that, as all athletes in the present study were experienced in racing the 1,500 m distance, this would have been avoided. On recording this result, it was clear from subsequent discussion with this individual that they had limited experience and memory of racing after implementing an optimal taper and therefore had unrealistic expectations about the level of performance they could achieve. The brain algorithmic process involved in initiating pace (St Clair Gibson et al., 2006, Ulmer et al., 1996) might have been provided with inaccurate information due to a lack of previous experience of an optimal taper and therefore the efferent neural commands sent to dictate starting pace might have generated inappropriate power output and pace for the individual's current performance capacity. Anecdotal evidence suggests that many athletes feel insecure at the prospect of reducing training load prior to competition and are unlikely to implement large reductions in training volume, as recommended in the literature (Bosquet et al. 2007). In support, the individual athlete in this case was completing the highest training volume prior to tapering ($84 \text{ km} \cdot \text{wk}^{-1}$) and was therefore prescribed the largest absolute reduction in training volume in both RP and HI. The unfamiliarity of implementing a large reduction such as this, likely led to heightened expectations, due to limited memory of competing in a tapered physiological state. It appears to be rare for athletes and coaches to practice different pacing strategies in low-key competition and it has been suggested that the learning implications from experiencing a range of pacing patterns may be beneficial to performance (Foster et al. 1993). It is therefore recommended that athletes take the opportunity to practice pacing after tapering ahead of less important competition or during periods of reduced volume in their normal training cycle.

6.5 Conclusion

In conclusion, a novel 7-d tapering strategy where continuous training volume is reduced by 60% and the final interval session is completed at 110% of race pace, results in a greater likelihood of increased 1,500 m track performance compared to a 7-d taper based on the practices of elite middle-distance runners. After tapering, athletes appear to adopt a positive pacing strategy during 1,500 m track performance. To avoid an over-fast start when employing this pacing strategy however, pacing in a tapered physiological state should be practiced and close attention should be paid to split times early in the race to facilitate an optimal pacing strategy.

Chapter VII

General Discussion and Application of Research

7.0 *Thesis Summary*

Tapering has been established as a fundamental strategy incorporated into the overall training process, to enable athletes to reach peak performance at the right time for important competition (Mujika & Padilla, 2003; Smith, 2003). Despite general recommendations from previous literature regarding optimal tapering practices (Bosquet et al., 2007; Houmard et al., 1990; Houmard et al., 1994; McConell et al., 1993; Mujika, 2010; Mujika et al., 2000; Mujika et al., 2002; Shepley et al., 1992; Wittig et al., 1989), the application of these recommendations into elite endurance running appear to be limited by: 1) knowledge of the current practices; 2) understanding of the relationship between training, tapering and performance enhancement; and therefore, 3) the ability to identify potential opportunities for strategy optimisation. The current thesis intended to enhance tapering strategies for highly trained endurance runners by exploring current practice and identifying potential areas for optimisation, through a more thorough understanding of the physiological mechanisms underpinning improved performance. Finally, the current thesis intended to investigate the possibility of a biomarker to monitor recovery status during the taper.

The main findings of the current thesis are as follows:

1. Middle-distance, long-distance and marathon runners reduce frequency, intensity and volume of continuous running and volume of interval running during the taper compared to regular training. Marathon runners implement a greater reduction in continuous volume, over a longer taper duration (47% and 14 d) than both middle- (30% and 6 d) and long-distance runners (29% and 6 d). Only middle-distance runners increase interval intensity during the taper (96% vs. 100% race speed), but do not typically run faster than race-speed in the peak intensity session of the taper, which occurred 3 d before competition. Long-distance and marathon runners run faster than race speed during the taper, a practice also reported in elite Kenyan long-distance runners (111%, 114% and 115% race speed, respectively; chapter III).

2. Algorithms were capable of explaining a large proportion of the variance (53-95%) in tapering strategy training variables (with the exception of interval volume) for a given regular training load in elite endurance runners (chapter III).
3. A tapering strategy implemented using the algorithms was *most likely* to improve 1,500 m treadmill performance in well-trained middle-distance runners. When the intensity of final interval session was increased to 115% race speed, the effect on treadmill performance was *unclear* and some athletes may experience a worsening in 1,500 m time (chapter IV).
4. When the algorithm-derived taper was implemented with a further reduction in continuous volume (60% reduction) and the final interval session intensity was 110% race speed, this tapering strategy was *very likely* to improve 1,500 m track performance and had a medium effect (chapter VI).
5. The improvement in 1,500 m performance time after tapering appears to be achieved by adopting a positive pacing strategy and running at a faster speed in the first 600 m of the race distance. An over-fast start may occur after tapering in some individuals and result in a worsening in performance time (chapter VI).
6. Peak leg extension force and RFD were improved in response to tapering, in addition to a higher peak lactate post-1,500 m time trial, but neither could explain performance differences between race-pace and higher intensity strategies. Well-trained middle-distance runners maintain a consistent total energy intake and carbohydrate intake despite a reduced training load during tapering (chapter IV).
7. Strong relationships were evident between capillary and venous blood samples for the measurement of plasma IL-6 and sIL-6R concentration, suggesting that the cytokines can be measured less invasively in the field from capillary samples (chapter II).
8. Plasma IL-6 and sIL-6R do not appear to be biomarkers sensitive enough to detect changes in recovery status and readiness to perform during tapering in middle-distance (chapter IV) or marathon runners (chapter V). This was also the case for CRP, T and C and therefore the measurement of these biomarkers is not recommended to facilitate further real-time adjustment of tapering strategies (chapter IV).

7.1 *Current Tapering Strategies in Elite Endurance Runners*

Previously, it has been demonstrated that dramatic reductions in training volume can be implemented during tapering (Mujika et al., 2000; Wittig et al., 1989), however training intensity must not be compromised (McConnell et al., 1993) and high intensity interval training during the taper is key to enhancing performance (Houmard et al., 1994; Shepley et al., 1992). Chapter III found that whilst in general, elite British endurance athletes reduced training volume (albeit more conservatively) and retained high intensity interval sessions during the taper, there were notable differences between event groups. Specifically, marathon runners reported a greater reduction in continuous training volume, over a longer taper duration than both middle- and long-distance runners. This was in agreement with previous speculation, that a higher prior training load and subsequent degree of fatigue may require a more aggressive and prolonged taper (Bosquet et al., 2007; Kubukeli et al., 2002; Smith, 2003; Thomas et al., 2008). This strategy was also confirmed in chapter V, where the pre-taper training volume in an elite marathon runner was higher than the average reported by British marathon runners ($182 \text{ km} \cdot \text{wk}^{-1}$ vs. $168 \text{ km} \cdot \text{wk}^{-1}$) and therefore the taper duration was longer (20 d vs. 14 d). Analysis of the relationship between prior training load and the extent to which training load variables were manipulated during the taper was subsequently carried out, using data pooled from all events.

Strong relationships were evident between regular training load and the magnitude of manipulation during the taper, allowing for the development of algorithms. Thus, providing a useful tool for coaches and athletes to aid design of the tapering strategy based on individual training load in the preceding training phase. Given these findings, athletes would be discouraged from implementing the same training programme during tapering for different competitions, as the same training load may not have been completed beforehand (e.g. through injury or illness). There may be a need therefore, to re-address the tapering strategy for each competition to maximise performance. The event-specific differences between tapering strategies implemented by elite endurance athletes can also be utilised to facilitate taper design and might be applicable to other sports such as cycling and swimming, where a range of event distances with different physiological determinants and training practices exist.

7.2 *Optimisation of Current Practice*

In chapter III, marathon runners and both British and Kenyan long-distance runners reported training at intensities well above race speed during the taper (114%, 111% and 115% race speed, respectively; $P < 0.05$). Middle-distance runners however, typically trained at race speed. Although previous research has suggested that high intensity training during the taper is key to improving performance (Houmard et al., 1994) and training intensity should perhaps be increased during this period (Shepley et al., 1992), the strategies investigated were extreme and do not reflect the typical training practices of elite endurance athletes. For example, training volume was reduced by 85-90% and all running was completed as high intensity intervals (Houmard et al. 1994; Shepley et al., 1992). Given the anxiety and insecurity that athletes anecdotally report regarding a reduction in training load prior to competition and the large proportion of lower intensity continuous running volume that is completed habitually (chapter III), it was unlikely that such a severe strategy would be readily accepted by elite athletes. However, it was unknown whether a single profound increase in training intensity in the final days of the taper would improve subsequent running performance, when incorporated into the existing tapering strategies of middle-distance runners. For example, an interval session completed at intensities above race speed late in the taper when the athlete has become more fully recovered, might allow greater capacity to respond effectively to this type of training stimulus and further improve subsequent performance (Mujika, et al., 2004). In support of this concept, a theoretical model suggests that a moderate increase in training load at the end of taper might further improve performance as the athlete can capitalise on additional adaptation, after initially overcoming accumulated fatigue from previous training (Thomas et al., 2008).

Chapters IV and VI therefore focused on investigating the effect of the algorithm-derived taper (from chapter III) on middle-distance running performance and manipulating the intensity of the final interval session of this taper. It was found that the current tapering strategy prescribed using the algorithms was *most likely* to improve 1,500 m treadmill performance in well-trained middle-distance runners. When the intensity of final interval session was increased to 115% race speed in this strategy however, the effect on treadmill performance was unclear and some athletes ran slower following the taper. The hypothesis predicting a greater performance improvement from the higher intensity taper was therefore rejected. It may be that some athletes require longer to reach peak performance after the high intensity session, in agreement with Thomas et al. (2009), but it was not

possible to explore the time course of the performance rebound between the 2 strategies, since only 1 performance assessment was completed after each taper. Alternatively, it was speculated that a conservative reduction in training volume resulting from the algorithm, compared to that of the previous high intensity strategies (Houmard et al, 1994; Shepley et al., 1992) or even general recommendations (Bosquet et al., 2007), may have prevented sufficient recovery to respond positively to the increased intensity interval session. Chapter VI thereafter assessed the effect of the high intensity taper, where continuous volume was also reduced by 60%, as per the recommendations of Bosquet et al., (2007). Since participants completed the high intensity session on a track rather than a treadmill, pace could not be fixed and was in fact 110% of race speed, suggesting that 115% might have been too intense in chapter IV and participants self-selected to run slower, albeit still faster than race speed. In chapter VI, 1,500 m performance was assessed using a more ecologically valid indoor track time trial. Whilst the inclusion of an ‘above race-speed session’ within the algorithm-derived taper initially resulted in equivocal effects on performance, the strategy was *very likely* to improve performance ($ES = 0.74$) if a greater concomitant reduction in continuous volume was implemented and the peak intensity interval session was completed at 110% race speed. The hypothesis in chapter VI was accepted due to a greater likelihood of improved performance and a larger effect from the novel high intensity strategy compared to race-speed strategy.

Collectively, these findings suggest that coaches and athletes need to achieve a balance between the manipulations of volume and intensity during the taper. To maximise the responses to high intensity training during the taper and ultimately improve performance, volume should be reduced enough to alleviate the temporary physiological disturbances associated with heavy training such as glycogen depletion, neuromuscular fatigue, hematological decrements and hormonal imbalances (Halsen & Jeukendrup, 2004; Halsen et al., 2002; Hellard et al., 2013; Mujika and Padilla, 2003), whilst intensity should not be increased excessively. The current findings can be readily applied to middle-distance runners and have also been disseminated among high performance coaches in cycling, triathlon and swimming. Furthermore, the findings suggest that it is not necessary to implement extreme volume reduction strategies (Houmard et al., 1994; Shepley et al., 1992) that are far removed from an athlete’s typical training routine (chapter III) to achieve a meaningful improvement in performance. Although more research investigating

this type of strategy is required, employing more ecologically valid performance measures, such as track-based time trials.

7.3 *Physiological Mechanisms*

A number of positive physiological changes have been reported as a result of the pre-competition taper, including increases in muscle strength and power (Luden et al., 2010; Shepley et al., 1992), muscle glycogen (Neary et al., 1992; Neary et al., 2003; Shepley et al., 1992; Walker et al., 2000), oxidative enzyme volume and activity (Neary et al., 1992; Neary et al., 2003; Shepley et al., 1992), blood and red cell volume (Mujika et al., 2002; Shepley et al., 1992) and hormonal changes (Mujika et al., 2002), all of which may contribute to improving endurance running performance. Given these responses, it has been suggested that the tapering period facilitates the restoration of physiological capacities that may have been previously suppressed by heavy training (Halsen & Jeukendrup, 2004). This may also lead to amplified physiological responses to the training completed during the taper (Mujika et al., 2004; Thomas et al., 2008). The complexity arises however, when differing performance outcomes are accompanied by similar physiological changes, thus emphasising the multitude of factors that may contribute to performance.

Chapters IV and VI attempted to gain a more thorough understanding of some of the physiological mechanisms underpinning the relationship between tapering and enhancement of performance, to facilitate the optimisation of strategies. Measures of peak leg extension force and rate of force development improved in response to tapering (chapter IV), although no differences were evident between race-speed and high intensity tapering strategies, despite a difference in the performance outcome. In a more extreme example, similar beneficial changes to maximal voluntary isometric strength were reported in middle distance runners after tapering and after a period of complete rest, however endurance performance capacity declined by 3% after the complete rest condition (Shepley et al., 1992). The current work perhaps confirms therefore, that improved muscle function occurs as part of a restorative process throughout the taper, rather than as an amplified response to the training done during the taper itself. Although not measured in the current thesis, possible mechanisms for improved strength and rate of force development might include; altered protein synthesis and shifts in fibre type (Luden et al., 2010; Neary et al., 2003; Trappe et al., 2000), changes in contractile geometry (Metzger & Moss, 1987),

increased muscle enzyme activity (Neary et al., 1992; Neary et al., 2003; Shepley et al., 1992), calcium sensitivity (Trappe et al., 2000) and neural control on fibre recruitment (Costill et al., 1985). Although, relative contribution to increased muscle performance from each these mechanisms is likely to be highly dependent on taper duration and strategy.

Muscle glycogen has been recognised as an important determinant of performance capacity in both prolonged submaximal (Bergstrom et al., 1967) and supramaximal (Maughan & Poole, 1981) exercise. It is possible that pre-taper muscle glycogen stores become somewhat depleted through inadequate carbohydrate consumption, coupled with multiple training sessions in 1 day (Costill et al., 1972; Philp et al., 2012). Increases in muscle glycogen concentration have been reported after tapering previously (Neary et al., 1992; Neary et al., 2003; Shepley et al., 1992; Walker et al., 2000), suggesting that the taper may provide an opportunity for athletes to replenish glycogen stores as a result of a reduced training load and consequently, lowered glycogen utilisation and greater recovery time between training sessions. Additionally, supercompensation of muscle glycogen stores can be achieved through a reduced training load and concomitant high carbohydrate diet (Costill et al., 1981; Sherman et al., 1981). Although muscle glycogen was not measured in the current work, it was found in chapter IV that carbohydrate consumption remained unchanged from pre-taper in both conditions despite a reduction in overall physical activity and a lower proportion of time spent undertaking MVPA during the taper. This habitual behaviour perhaps increases the likelihood of augmented muscle glycogen during the taper. There was no difference in carbohydrate consumption in the final 3 d of the high intensity taper compared to the race-speed taper, despite an increase in intensity of the final interval session. Since glycogen is the main energy source for high intensity exercise (Stellingwerff et al., 2011) and there is evidence of rapid muscle glycogen depletion in type II fibres after high intensity intermittent exercise (Gollnick et al., 1973), a more direct intervention to optimise carbohydrate consumption after the intensified interval session might have influenced the performance outcome.

Although positive responses, the restoration of muscle strength and power and increased muscle glycogen pose a potential challenge for the determination of an athlete's pacing strategy after the taper. For example, athletes might train with overload prior to the taper to maximise adaptation (Aubry et al., 2014) and become unaccustomed to performing

without the accumulated fatigue that is consequential of the normal training process (Halsen & Jeukendrup, 2004). This overload practice might influence the memory and negatively impact the decision making process in selecting the initial race pace (St Clair Gibson et al., 2006; Rauch et al., 2005). The current work found that middle-distance runners adopt a more positive pacing strategy after tapering in order to improve performance, although some athletes may succumb to an over-fast start, which results in a worsening in performance. This finding should encourage athletes to become accustomed to racing in a tapered state to ensure an optimal pacing strategy is executed, perhaps utilising low-key events and paying close attention to split times early in the race.

The physiological mechanisms fundamental to the process of tapering are still not yet fully understood in endurance runners. It appears that a myriad of physiological changes occur during the taper, all of which may contribute, in some degree, to improving performance. However, the complex interaction of physiological determinants, coupled with recovery status and the influence of other factors on performance, means that physiological responses to tapering cannot always exclusively explain performance differences between strategies.

7.4 *Potential Physiological Biomarkers*

A final aim of the current thesis was to identify a physiological biomarker capable of monitoring recovery status during the taper, to allow further individualisation and optimisation of strategies in real-time. In the athlete population however, it is not feasible to employ the invasive procedures such as muscle biopsies, necessary to monitor a number of the physiological changes associated with the taper. Biomarkers are required to be minimally invasive, with fast sample analysis to allow prompt feedback. Previously, it has been suggested that the longitudinal monitoring of variations in inflammatory cytokines; IL-6, sIL-6R, CRP, and the T:C ratio, may be indicative of training stress and energy homeostasis throughout different phases of training (Adlercreutz et al., 1986; Jürimäe et al., 2011, Robson-Ansley et al., 2007), although their sensitivity to monitor taper effectiveness and recovery acutely in endurance runners required investigation.

The hypothesis in chapter II predicted that plasma IL-6 and sIL-6R measured from capillary blood would be strongly correlated with that from venous blood. This was accepted and it was deemed possible that the cytokines could be measured less invasively using capillary samples in the field. However, these markers did not appear to be sensitive

enough to detect or indicate the subtle changes in recovery status associated with tapering and performance in middle-distance or marathon runners. This was also the case for CRP, and the T:C ratio and these measures perhaps are more useful when monitored on a more longitudinal basis throughout different phases of training.

Whilst the blood biomarkers measured in this thesis did not represent useful indicators of recovery and taper effectiveness, other non-invasive measures could be considered. In chapter IV for example, peak isometric leg extension force and rate of force development improved in response to tapering. These changes could not explain the difference in running performance after two different tapering strategies and it appears therefore, that improved muscular performance occurs as part of a restorative process throughout the taper, rather than as a result of further adaptation. For this reason, measurements of muscular performance might offer insight into readiness to perform and indicate further opportunities for real-time fine-tuning of tapered training. Whilst laboratory measurements are not practical, non-invasive, field-based measures such as a CMJ in conjunction with session-RPE have been shown to indicate readiness to perform in elite endurance athletes (Balsalobre-Fernandez et al., 2014). Alternatively, post-exercise heart rate recovery might provide insight into readiness to perform in elite endurance athletes (Hug et al., 2014). These variables can be measured simply on a regular basis and at a lower cost, as part of an athlete's regular training programme. Rapid feedback could be obtained from such measures and it would be of interest to carry out further investigation in response to tapering in elite endurance athletes.

7.5 Tapering Summary

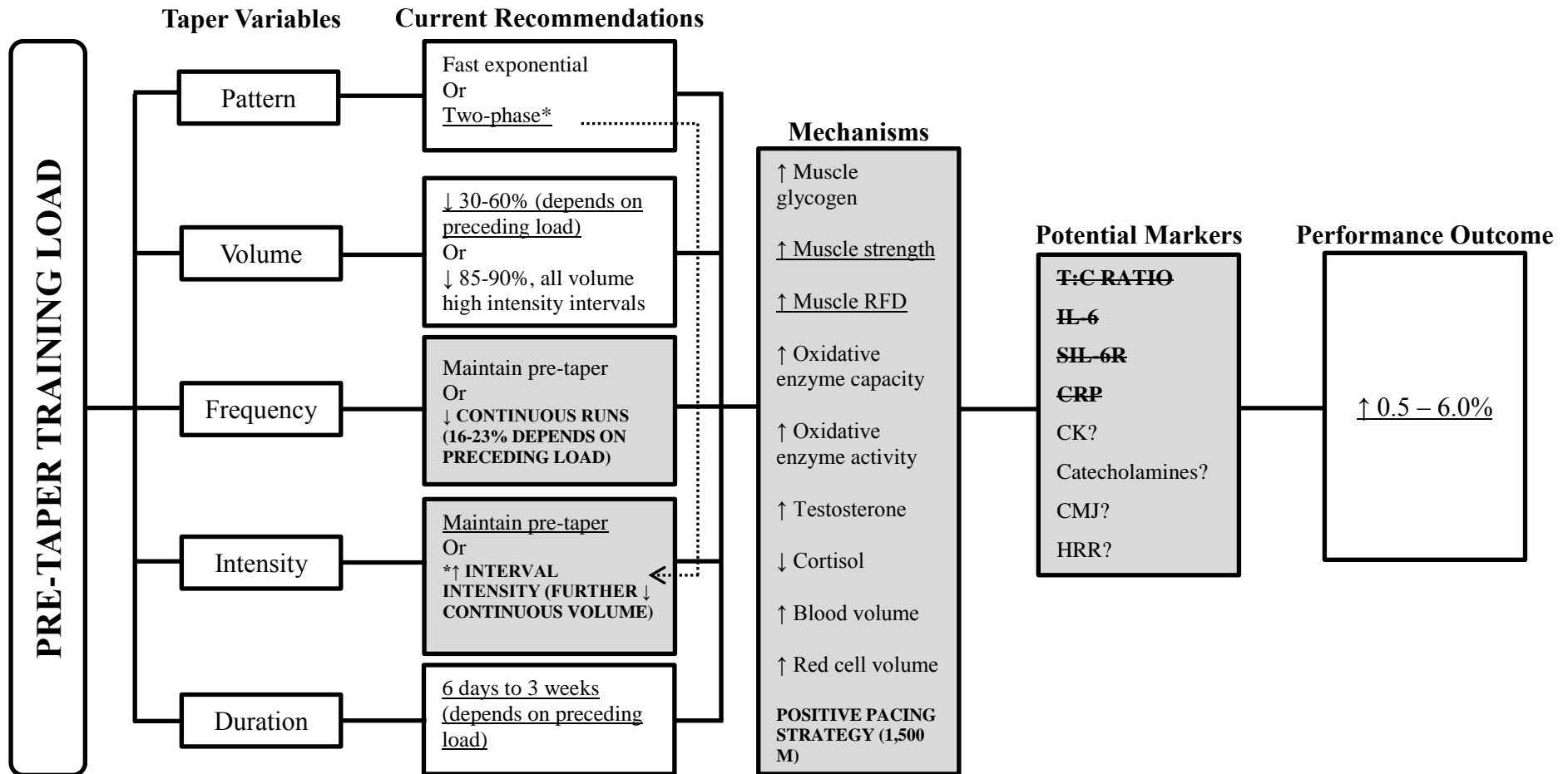


Figure 7.1. A model of the components of tapering, recommendations from the scientific literature and observations from the current work. Underlined text refers to knowledge that the current work has confirmed. Bold capitalised text in shaded boxes highlights novel findings from the current work. Question marks denote unknowns, where further research is required.

7.6 Recommendations for Coaches and Athletes

Table 7.1. Recommendations for coaches and athletes, based on observations from the scientific literature and findings from the current work.

Taper Component	Recommendation
General	<p>Do not complete the same taper before different competitions; plan the taper relative to the preceding training load</p> <p>Address the component parts of training load separately (volume, frequency, intensity, etc.)</p>
Pattern	Consider the pattern in which training is manipulated during the taper (fast exponential or two-phase)
Volume	The first part of the taper should focus on reducing accumulated fatigue, via a rapid reduction in running volume of up to 60%
Frequency	Training frequency should be maintained, although continuous running can be slightly reduced if necessary (up to 23% if previous training volume and frequency are high)
Intensity	<p>High intensity interval training should not be compromised throughout the taper</p> <p>The intensity of interval training can be increased later in the taper, providing volume has been reduced adequately and there is enough recovery time before competition</p>
Duration	Duration of the taper is typically 6 d to 14 d, but should be individualised depending on preceding training load and rate of recovery, if known
Performance Implications	<p>Athletes should possess a clear pacing strategy, to prevent an over-fast start to the race after tapering</p> <p>Pacing in a tapered state could be practiced during less important competition or in training during periods of reduced volume in the normal cycle</p>

7.7 *Limitations*

The limitations of the current work are acknowledged. Firstly, it is important to highlight that performance can be influenced by a multitude of factors, including physiological, psychological, lifestyle and tactical components (Smith, 2003) and therefore it is difficult to isolate the effects of tapering on performance. This perhaps explains the paucity of literature investigating tapering strategies, despite the importance of this training phase prior to competition. Attempts were made to control confounding variables where possible in the current work (e.g. standardising warm up and prior dietary intake), in addition to selection of appropriate performance measures (Currell & Jeukendrup, 2008).

The ultimate main aim of the current work was to optimise tapering strategies for elite endurance runners. However, given the nature of tapering in reducing training volume and the multiple performance assessments that were required, elite athletes were unwilling to participate in the experimental studies in chapters IV and VI. Attempts were made to preserve a high performance standard, although there was reluctance to manipulate training load through fear of compromising performance and sample sizes were therefore restricted. This resulted in a cohort heterogeneous for performance in chapter IV and perhaps limits application of the results to the elite population.

In recruiting athletes of as high standard as possible, it was also not feasible to prescribe the same pre-taper training programme to all participants in chapters IV and VI and therefore prior training load was individualised and uncontrolled. This means that although the taper was implemented relative the prior training load as per studies in previous literature, the absolute training load completed during the taper was different between individuals. However, this could also be a strength of this experimental work, as each individual's physiology and training history may vary and therefore to prescribe a standardised and unfamiliar training programme prior to the taper may have confounded the effects of the taper on performance. It would have been unrealistic to recruit participants with similar physiology, current training programmes and of the same performance level for the experimental chapters. Efforts were also made in chapter VI to collect prior training load data over a longer period (4 weeks vs. 1 week in chapter IV) in order to prescribe the taper more accurately.

It was not possible to conduct the investigations during the competitive summer season, and since all training during the taper was relative to prior training, it is possible that

differences between winter and summer training loads may have confounded the effects of the algorithm-derived taper. However, a number of the participants in chapter VI were targeting late-winter indoor middle-distance races after the completion of the investigation and their training was therefore specifically focused towards this.

It was also not feasible to assess the physiological characteristics of the elite athletes in chapter III prior to collecting data on training and tapering practices. This is perhaps a limitation, as training intensity was quantified relative to race pace, rather than to individual physiological landmarks (lactate threshold, lactate turnpoint, $\% \dot{V}O_{2\max}$ or $\%v\dot{V}O_{2\max}$). It is likely that between-athlete variability exists in how performance is generated, particularly in middle-distance running (i.e. % energy contributions from aerobic vs. anaerobic sources). Two athletes with the same 1,500 m performance time for example, might therefore elicit different responses to a fixed intensity session at 115% of race speed, owing to potential differences in maximal speed, aerobic and anaerobic capacities. It is possible that assessing the physiological characteristics of the cohort in chapters IV and VI prior to the investigation might therefore have facilitated understanding of individual responses to the tapering strategies. Additionally, a more detailed method to monitor training load prior to and during the taper, might have added to understanding of individual responses. Although daily external load can be relatively easily quantified in terms of volume and intensity of training, no insight is provided into internal load using this method, and a combination of both might be useful to track the cumulative effects of training and to indicate fatigue and recovery status (Halsen, 2014).

Whilst the taper aims to ensure athletes arrive at competition in peak condition for performance, a final appreciation is the magnitude to which performance improvements from tapering investigations are transferred into actual competition, especially given the tactical nature of races at major championships.

7.8 Recommendations for Future Research

The research conducted in this thesis was of a highly applied and exploratory nature. The findings therefore, lead to a number of further important questions, in addition to those already discussed.

Despite evidence for the inclusion of high intensity training during the taper, details regarding best practice during the final days prior to competition remain elusive. In the

current thesis, the effect of the taper was assessed in a single time trial performance. In practice however, middle-distance runners complete 3 rounds of competition in a major championship (heats, semi-finals and final). At the Rio 2016 Olympic Games, male 1,500m runners will compete on days 5, 7 and 9. This poses the question as to whether athletes should begin their taper duration on count back from the heat or from the final and whether the early rounds provide opportunity for low volume, high intensity bouts that are beneficial to performance in the final. Conversely, these rounds can manifest as slow, tactical races and could possibly interfere with the planned taper.

Competing at major championship also implies that the athlete will have been required to achieve a qualification standard and finish in a qualification position in a trial race, typically held several weeks beforehand. This might require the athlete to perform optimally in a number of races over an extended period of time and therefore the optimal tapering strategy for a single performance may no longer be valid. This may be exacerbated by the fact that athletics is not a professional sport and therefore athletes rely on prize money from races such as the Diamond League (series of 14 events) to support their income. Further investigation into the tapering process for an extended competition period is required, particularly since this issue also applies to a number of Olympic sports.

The focus in the current thesis was on manipulation of running training during tapering, however, elite endurance athletes also incorporate aspects on non-running training into their schedules, such as strength training, drills and stability training (Ingham et al., 2012; Tjelta, 2013). Further research is therefore required to investigate the influence of non-running training on recovery during tapering and the optimal modifications, if necessary, for performance.

Finally, major championships for many sports typically take place in different host countries with each edition. Some locations can therefore pose environmental challenges, such as high temperatures or altitude. Often athletes travel to the venue for a holding camp in the weeks preceding the event to undergo an acclimatisation process, however it is not clear how the additional environmental stress would affect the tapering strategy and whether adjustments would be required to facilitate the peaking of performance. Additionally, altitude training is now incorporated into the training programmes of many elite endurance athletes, particularly prior to competition. Whilst the body of literature concerning the optimal time to compete after an altitude sojourn is expanding, little is

known about the influence of altitude on the tapering strategy and whether further modifications to training are required to optimise subsequent performance.

7.9 Conclusion

The findings of this thesis suggest that tapering strategies in elite endurance athletes are individualised and influenced by the preceding training load. In middle-distance runners, training above race speed in the final days of the taper might be more beneficial than current practice, although training volume must be further reduced to compensate. It appears that the physiological mechanisms underpinning improved performance after tapering are complex and multifaceted, although a novel finding is the change to pacing strategy in 1,500 m track performance. It is proposed that future studies continue to investigate non-invasive indicators of recovery and taper effectiveness, including measures of muscle function, to allow further real-time adjustment of training load to facilitate the peaking of performance.

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Appendices

Appendix A: Informed consent

Appendix B: Participant health screening questionnaire

Appendix C: Rating of perceived exertion (RPE) scale

Appendix D: Analysis of Blood Samples

Appendix E: Estimation of Plasma Volume

Appendix F: Training record

Appendix G: Food diary

Appendix H: ActiGraph record



Optimising Tapering Strategies in Middle-Distance Runners

INFORMED CONSENT FORM

(to be completed after Participant Information Sheet has been read)

The purpose and details of this study have been explained to me. I understand that this study is designed to further scientific knowledge and that all procedures have been approved by the Loughborough University Ethics Approvals (Human Participants) Sub-Committee.

Yes ☐ No ☐

I have read and understood the information sheet and this consent form.

Yes ☐ No ☐

I have had an opportunity to ask questions about my participation.

Yes ☐ No ☐

I understand that I am under no obligation to take part in the study.

Yes ☐ No ☐

I understand that I have the right to withdraw from this study at any stage for any reason, and that I will not be required to explain my reasons for withdrawing.

Yes ☐ No ☐

I understand that all the information I provide will be treated in strict confidence and will be kept anonymous and confidential to the researchers unless (under the statutory obligations of the agencies which the researchers are working with), it is judged that confidentiality will have to be breached for the safety of the participant or others.

Yes ☐ No ☐

I agree to participate in this study.

Yes ☐ No ☐

Your name _____

Your signature _____

Signature of investigator _____

Date _____



Code

Health Screen Questionnaire for Study Volunteers

As a volunteer participating in a research study, it is important that you are currently in good health and have had no significant medical problems in the past. This is (i) to ensure your own continuing well-being and (ii) to avoid the possibility of individual health issues confounding study outcomes.

Please complete this brief questionnaire to confirm your fitness to participate:

1. At present, do you have any health problem for which you are:

(a) on medication, prescribed or otherwise	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>
(b) attending your general practitioner	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>
(c) on a hospital waiting list	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>

2. In the past two years, have you had any illness which required you to:

(a) consult your GP	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>
(b) attend a hospital outpatient department	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>
(c) be admitted to hospital	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>

3. Have you ever had any of the following:

(a) Convulsions/epilepsy	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>
(b) Asthma	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>
(c) Eczema	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>
(d) Diabetes	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>
(e) A blood disorder	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>
(f) Head injury	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>

(g) Digestive problems	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>
(h) Heart problems	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>
(i) Problems with bones or joints	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>
(j) Disturbance of balance/coordination	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>
(k) Numbness in hands or feet	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>
(l) Disturbance of vision	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>
(m) Ear / hearing problems	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>
(n) Thyroid problems	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>
(o) Kidney or liver problems	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>
(p) Allergy to nuts	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>

4. Has any, otherwise healthy, member of your family under the

age of 35 died suddenly during or soon after exercise?	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>
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If YES to any question, please describe briefly if you wish (eg to confirm problem was/is short-lived, insignificant or well controlled.)

.....

.....

.....

5. Allergy Information

(a) are you allergic to any food products?	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>
(b) are you allergic to any medicines?	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>
(c) are you allergic to plasters?	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>

If YES to any of the above, please provide additional information on the allergy

.....

.....

6. Please provide contact details of a suitable person for us to contact in the event of any incident or emergency.

Name:
.....

Telephone
Number:
.....

Work ☐ Home ☐ Mobile ☐

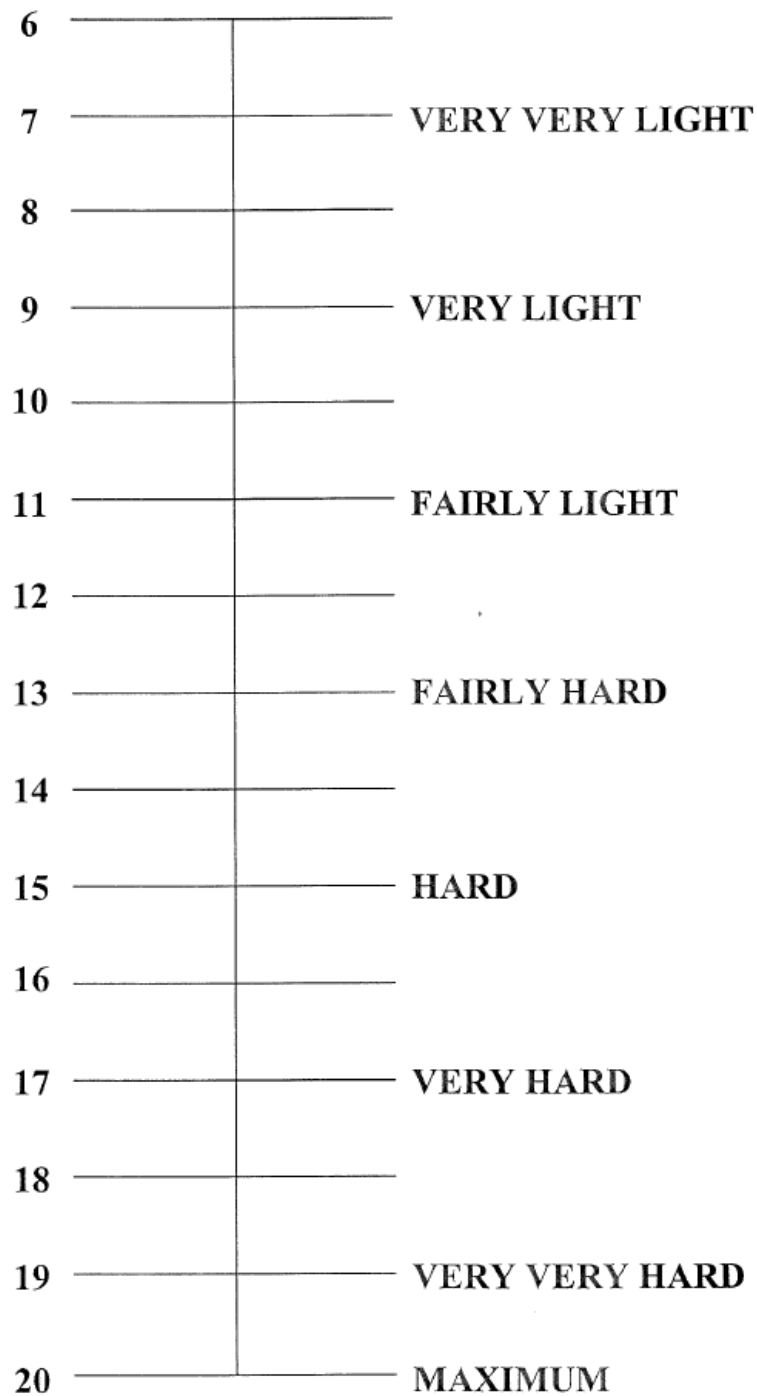
Relationship to
Participant:.....
.....

7. Are you currently involved in any other research studies at the University or elsewhere?

Yes ☐ No ☐

If yes, please provide details of the study

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

RATE OF PERCEIVED EXERTION

Analysis of Blood Samples

Plasma IL-6, sIL-6R, CRP, T and C concentrations were analysed using sandwich enzyme-linked immunosorbent assays (ELISAs). All materials and chemical reagents were obtained from Sigma-Aldrich Ltd. (Poole, UK) unless otherwise specified. All samples from the same participant were analysed on the same plate and in consecutive wells in each study. All incubation periods were at room temperature and during each incubation stage the plate was placed on a horizontal orbital plate shaker (Mini Orbital Shaker SOB, Stuart Scientific, UK) at 60 rpm unless otherwise stated. Concentrations were determined in relation to a 4-parameter standard curve (GraphPad Prism, Version 4.00; San Diego California, USA). Concentrations were corrected for any changes in plasma volume (appendix E).

Plasma Interleukin-6

The plates were coated overnight with anti-human IL-6 monoclonal capture antibody (OptEIA, BD Biosciences, Oxford, UK) diluted 1:250 in 0.1 M sodium carbonate. The following day, the plates were washed and then blocked with 5% bovine serum albumin (BSA: Probumin, Millipore, Illinois, USA) in Tris-buffered saline (TBS). After an incubation period of 1 h, the plates were washed and the standards and samples were added to the wells. The samples were diluted 1:4 in TBS with 10% foetal calf serum (FCS). After a further 2 h incubation period, the plates were washed and 100 µl detection antibody (OptEIA, BD, Biosciences, Oxford, UK) diluted 1:250 TBS-T with 1% BSA was added in each well. Plates were incubated for a further 1 h and subsequently washed. Streptavidin alkaline phosphatase enzyme was diluted 1:2,000 in TBS with 1% BSA and 100 µl of the solution was added to each well and incubated for 45 min, then washed before an ELISA amplification system was used (Invitrogen, Paisley, UK). The reaction was stopped with 50 µl of 10% sulphuric acid and the absorbance of the wells was read at 490 nm with a correction of 690 nm (Varioskan Flash, Thermo Scientific, Vantaa, Finland).

Plasma Soluble Interleukin-6 Receptor

Plasma sIL-6R concentration was measured using commercially available antibody pairs (M5 capture antibody and M182 detection antibody, BD Biosciences, Oxford, UK). The plate was coated with capture antibody diluted 1:250 in 0.1 M sodium carbonate for

incubation overnight. The following day, the plate was washed and blocked with assay diluent (phosphate-buffered saline (PBS) with 10% FCS). After 1 h incubation, the plate was washed and the standards and samples were added to the wells. The samples were diluted 1:200 in assay diluent. After a further 2 h incubation period, the plate was washed and 100 µl detection antibody diluted 1:1000 in assay diluent was added to each well. The plate was incubated for 1 h before washing. The enzyme streptavidin horse radish peroxidase was diluted 1:4000 in assay diluent and added to the wells. After 45 min incubation, the plate was washed and a substrate solution was added for 30 min before adding a stop solution (10% sulphuric acid). The absorbance of the wells was read at 450 nm with a correction of 570 nm (Varioskan Flash, Thermo Scientific, Vantaa, Finland).

C-Reactive Protein

The plates were coated overnight with anti-human CRP rabbit polyclonal capture antibody (Calbiochem, Merck Chemicals, Nottingham, UK) diluted 1:1,000 in 0.05 M sodium carbonate. The following day, the plates were washed and then blocked with 1% bovine BSA (Probumin, Millipore, Illinois, USA) in PBS. After an incubation period of 1 h, the plates were washed and the standards and samples were added to the wells. The samples were diluted 1:100 in PBS with 1% BSA. After An overnight incubation period, the plates were washed and 50 µl secondary antibody (anti-human CRP mouse monoclonal antibody, Abcam, Cambridge, UK) diluted 1:750 in PBS was added in each well. Plates were incubated for a further 2 h and subsequently washed. Horseradish peroxidase conjugated anti-mouse immunoglobulins rabbit polyclonal antibody was diluted 1:1,000 in PBS and 50 µl of the solution was added to each well and incubated for 1 h. After washing, 50 µl of substrate solution was added. The reaction was stopped with 75 µl of 10% sulphuric acid and the absorbance of the wells was read at 490 nm with a correction of 640 nm (Varioskan Flash, Thermo Scientific, Vantaa, Finland).

Testosterone and Cortisol

Plasma T and C concentrations were determined using commercially available ELISA kits, with the aid of a plate reader (Varioskan Flash, Thermo Scientific, Vantaa, Finland). For testosterone (R&D Systems, Inc., Mineappolis, USA, supplied by Bio-Techne, Abingdon, UK), plasma samples were initially diluted 10-fold. The plates were coated with 50 µl primary anti-body solution and incubated for 1 h. After washing, 100 µl of standards, controls and samples were added, followed by 50 µl of testosterone conjugate to each well.

After 3 h incubation, plates were washed and 200 µl substrate solution added to each well. Plates were protected from light during a final 30 min incubation, after which 50 µl stop solution was added. Absorbance of the wells was read at 450 nm with a correction of 570 nm. For cortisol (DRG Instruments GmbH, Marburg, Germany, supplied by Immunodiagnostic Systems, Tyne & Wear, UK), plasma samples were undiluted. Initially, 20 µl of standards, controls and samples were added to the appropriate wells, followed by 200 µl enzyme conjugate. Plates were thoroughly mixed for 10 s and incubated for 1 h. After washing, 100 µl substrate solution was added and plates were incubated for a further 15 min. After the addition of 100 µl stop solution, absorbance of the wells was read at 450 nm. Quality controls supplied with the assay kits were run on each plate to ensure precision of analysis.

Precision of Analysis

All samples from the same participant were analysed on the same assay plate to eliminate inter-assay variation. All samples were measured in duplicate. In chapter II, the inter-assay CV for IL-6 was 8.0% and the intra-assay CVs were; IL-6 5.8% and sIL-6R 2.6%. In Chapter IV, the IL-6 inter-assay CV was 5.4% and the intra-assay CV 3.2%. The sIL-6R inter-assay CV was 3.5% and the intra-assay CV 3.7%. For CRP, the inter-assay CV was 9.4% and the intra-assay CV 5.4%. The inter-assay CVs were 6.2% and 6.5%, and the intra-assay CVs were 5.0% and 3.9% for T and C, respectively. In chapter V, the intra-assay CV was 8.3% for IL-6 and 8.0% for sIL-6R. Quality controls supplied with commercial assay kits were run on each plate to ensure precision of analysis.

Estimation of Plasma Volume

In chapters II and IV, estimations of plasma volume change were calculated. Haematocrit was measured in triplicate using the microcapillary technique and the mean was calculated. Whole blood was collected into non-heparinised microhaematocrit tubes, which were sealed with Cristaseal capillary tube sealant (Hawksley, Sussex, UK). Samples were centrifuged for 4 min (Haematospin 1300, Hawksley, Sussex, UK) and haematocrit content was determined using a microhaematocrit tube reader (Hawksley, Sussex, UK). Haemoglobin concentration was measured by a colorimetric method, using a commercially available kit (Randox, Co Antrim, UK). Samples were measured in duplicate and absorbance of cyanmethaemoglobin was read at 540 nm using an ultra-violet spectrophotometer (CECIL CE1011, Cecil Instruments Ltd., Cambridge, UK). Changes in plasma volume were estimated according to (Dill & Costill, 1974) using the following formula:

Blood volume (BV)

- $BV_A = BV_B (Hb_B / Hb_A)$

Red cell volume (CV)

- $CV_B = BV_B (Hct_B / 100)$
- $CV_A = BV_A (Hct_A)$

Plasma volume (PV)

- $PV_B = BV_B - CV_B$
- $PV_A = BV_A - CV_A$
- $\Delta PV\% = 100 (PV_A - PV_B) / PV_B$

Where:

- The subscripts B and A refer to 'baseline' and 'after' (i.e. subsequent samples over time), respectively
- $BV_B = 100$
- Hb is haemoglobin concentration (g/dL)
- Hct is haematocrit (%)

Appendix F

Normal Week	Monday		Tuesday		Wednesday		Thursday	
	am	pm	am	pm	am	pm	am	pm
Type of Exercise	Easy <input type="checkbox"/> Steady <input type="checkbox"/> Tempo <input type="checkbox"/> Session <input type="checkbox"/>	Easy <input type="checkbox"/> Steady <input type="checkbox"/> Tempo <input type="checkbox"/> Session <input type="checkbox"/>	Easy <input type="checkbox"/> Steady <input type="checkbox"/> Tempo <input type="checkbox"/> Session <input type="checkbox"/>	Easy <input type="checkbox"/> Steady <input type="checkbox"/> Tempo <input type="checkbox"/> Session <input type="checkbox"/>	Easy <input type="checkbox"/> Steady <input type="checkbox"/> Tempo <input type="checkbox"/> Session <input type="checkbox"/>	Easy <input type="checkbox"/> Steady <input type="checkbox"/> Tempo <input type="checkbox"/> Session <input type="checkbox"/>	Easy <input type="checkbox"/> Steady <input type="checkbox"/> Tempo <input type="checkbox"/> Session <input type="checkbox"/>	Easy <input type="checkbox"/> Steady <input type="checkbox"/> Tempo <input type="checkbox"/> Session <input type="checkbox"/>
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Pacemin/mile orrace pacemin/mile orrace pacemin/mile orrace pacemin/mile orrace pacemin/mile orrace pacemin/mile orrace pacemin/mile orrace pacemin/mile orrace pace
Heart rate								
Supplementary Exercise	Resistance <input type="checkbox"/> Technical <input type="checkbox"/> Strides <input type="checkbox"/>	Resistance <input type="checkbox"/> Technical <input type="checkbox"/> Strides <input type="checkbox"/>	Resistance <input type="checkbox"/> Technical <input type="checkbox"/> Strides <input type="checkbox"/>	Resistance <input type="checkbox"/> Technical <input type="checkbox"/> Strides <input type="checkbox"/>	Resistance <input type="checkbox"/> Technical <input type="checkbox"/> Strides <input type="checkbox"/>	Resistance <input type="checkbox"/> Technical <input type="checkbox"/> Strides <input type="checkbox"/>	Resistance <input type="checkbox"/> Technical <input type="checkbox"/> Strides <input type="checkbox"/>	Resistance <input type="checkbox"/> Technical <input type="checkbox"/> Strides <input type="checkbox"/>
Durationminsminsminsminsminsminsminsmins

	Friday		Saturday		Sunday	
	am	pm	am	pm	am	pm
Type of Exercise	Easy <input type="checkbox"/> Steady <input type="checkbox"/> Tempo <input type="checkbox"/> Session <input type="checkbox"/>	Easy <input type="checkbox"/> Steady <input type="checkbox"/> Tempo <input type="checkbox"/> Session <input type="checkbox"/>	Easy <input type="checkbox"/> Steady <input type="checkbox"/> Tempo <input type="checkbox"/> Session <input type="checkbox"/>	Easy <input type="checkbox"/> Steady <input type="checkbox"/> Tempo <input type="checkbox"/> Session <input type="checkbox"/>	Easy <input type="checkbox"/> Steady <input type="checkbox"/> Tempo <input type="checkbox"/> Session <input type="checkbox"/>	Easy <input type="checkbox"/> Steady <input type="checkbox"/> Tempo <input type="checkbox"/> Session <input type="checkbox"/>
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Pacemin/mile orrace pacemin/mile orrace pacemin/mile orrace pacemin/mile orrace pacemin/mile orrace pacemin/mile orrace pace
Heart rate						
Supplementary Exercise	Resistance <input type="checkbox"/> Technical <input type="checkbox"/> Strides <input type="checkbox"/>	Resistance <input type="checkbox"/> Technical <input type="checkbox"/> Strides <input type="checkbox"/>	Resistance <input type="checkbox"/> Technical <input type="checkbox"/> Strides <input type="checkbox"/>	Resistance <input type="checkbox"/> Technical <input type="checkbox"/> Strides <input type="checkbox"/>	Resistance <input type="checkbox"/> Technical <input type="checkbox"/> Strides <input type="checkbox"/>	Resistance <input type="checkbox"/> Technical <input type="checkbox"/> Strides <input type="checkbox"/>
Durationminsminsminsminsminsmins

Please use the box to describe details of your sessions and strides i.e. rep distance/time and recovery:

Target weekly mileage (if known).....

CONFIDENTIAL

Code

FOOD DIARY

**OPTIMISING TAPERING STRATEGIES IN MIDDLE DISTANCE
RUNNERS**

Information will be treated in confidence.

If you have any problems, please contact: K.L.Spilsbury@lboro.ac.uk

School of Sport and Exercise Sciences
Loughborough University
Loughborough
Leicestershire
LE11 3TU

INSTRUCTION FOR USING THE FOOD DIARY

Everything that you eat and drink over the course of the day should be weighed and the weight and type of food or drink recorded.

For solid foods, the food should be placed on the scale on a plate or container. The plate or container must be weighed empty first and the scales can then be zeroed. Each item of food can then be added to the plate and weighed individually, returning the scales to zero between each item.

e.g. Plate	150g zero scale
Roast Beef	100g zero scale
Potato	150g zero scale
Gravy	30g zero scale

For drinks, a cup or glass must first be weighed and then the scale can be returned to zero and the drink added. Please remember to record separately the weight of tea, milk and sugar put into a drink.

Do not forget to weigh and record second helpings and between meal snacks.

Any leftovers (eg. apple cores) should also be weighted and recorded in the leftovers column.

Eating Out – Most people eat foods away from home each day, please do not forget to record these. Take your diary and scales with you where ever it is possible. If this is too inconvenient just record the type of food eaten with an estimated weight – but please say when a weight has been estimated.

Most snack foods will have the weight of the food on the packet so they do no need weighing if you eat the whole packet yourself.

Names and descriptions of foods should be as detailed as possible, including the brand name and any other information available.

e.g.	Cheese – is insufficient information.
	Cheese, cheddar (Shape reduced fat) – is sufficient information.

Start a new page in your diary for each day, and record each item on a separate line. Record the time of day in the first column of each line.

e.g. 10:30 am McVities Digestive Biscuits (2) 50g

The space provided at the foot of each page for general comments is for you to give any further information about your diet

e.g. Missed lunch due to stomach pains

.DAY

Date: / /

Please use a separate line for each item eaten; write in weight of plate; leave a line between different 'plate' entries.

[illegible]

ActiGraph Participant Information and Diary Sheet

The ActiGraph activity monitor is a small red plastic box worn around the waist on an elastic belt. It measures human movement. It does not interfere with daily activities and does not pose any risk to the person wearing it.

You should wear the monitor every day for the study duration. You should put it on first thing in the morning and continue wearing it until you go to bed.

Wearing the monitor

The monitor should be worn on the right hip with the black button at the top (as shown in the picture) or, if it does not have a black button, with the word *ActiGraph* the right way up.



It is important that the belt is fastened tightly so it fits snugly to the body. If it is loose, the measurements will be inaccurate.

The monitor is not waterproof.

Please remove it when you take a bath or shower or go swimming. Whenever you need to remove the monitor, please re-attach it as soon as you can, and record the times using the log on the other side of this sheet.

When to put the monitor on

The device should be fastened around your waist as soon as you wake up. It may be that you take it off again soon after in order to take a shower etc but following this instruction means the data is as accurate as possible. We appreciate your help with this.

When to take the monitor off

The device should be taken off when you plan to go to *sleep*. If you lie in bed watching TV, for example, please keep the monitor on until you have finished. Again, this is in the interest of collecting accurate data.

Returning the monitor

Please put your activity monitor and log sheet in the envelopes provided and return the package to Kate on your final visit to the lab. Please return the package as soon as possible so the data on your monitor are not lost and so other people can use the monitor and take part in the study.

Questions or problems

If you have any questions or problems, please do not hesitate to contact:
Kate Spilsbury (K.L.Spilsbury@lboro.ac.uk)

Thank you very much for taking part in this study.

Activity monitor log

When did you start wearing the monitor?

Date: _____ **Time:** _____

Sometimes you will have to remove the activity monitor. If you take it off during the day (for example when you take a bath or shower or go swimming), please write down when you took the monitor off, when you put it back on, and why you took it off. Please circle whether each time listed was a.m. or p.m.

	Time taken off	Time put back on	Reason taken off
Example	12.45 AM / PM	2.15 AM / PM	Swimming
Day 1	Lab testing day – don't need to wear monitor		
Day 2	AM / PM	AM / PM	
Day 3	AM / PM	AM / PM	
Day 4	AM / PM	AM / PM	
Day 5	AM / PM	AM / PM	
Day 6	AM / PM	AM / PM	
Day 7	AM / PM	AM / PM	
Day 8	AM / PM	AM / PM	
Day 9	Lab testing day – don't need to wear monitor		

	Time taken off	Time put back on	Reason taken off
Day 10			
	AM / PM	AM / PM	
Day 11			
	AM / PM	AM / PM	
Day 12			
	AM / PM	AM / PM	
Day 13			
	AM / PM	AM / PM	
Day 14			
	AM / PM	AM / PM	
Day 15			
	AM / PM	AM / PM	
Day 16			
	AM / PM	AM / PM	
Day 17	Lab testing day – don't need to wear monitor (please return it)		